No. 23-16026

IN THE UNITED STATES COURT OF APPEALS
FOR THE NINTH CIRCUIT

HELEN DOE, parent and next friend of Jane Doe; et al.,

Plaintiffs-Appellees,

v.

THOMAS C. HORNE, in his official capacity as State Superintendent of Public Instruction; et al.,

Defendants-Appellants,

and

WARREN PETERSEN, Senator, President of the Arizona State Senate; BEN TOMA, Representative, Speaker of the Arizona House of Representatives,

Intervenor-Defendants-Appellants.

On Appeal from the United States District Court
for the District of Arizona

EXHIBITS TO INTERVENOR-DEFENDANTS-APPELLANTS' EMERGENCY MOTION UNDER CIRCUIT RULE 27-3 FOR A STAY PENDING APPEAL

VOLUME 1

TABLE OF CONTENTS

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA

Helen Doe, et al.,

Plaintiffs,

v.

Thomas C Horne, et al.,

Defendants.

No. CV-23-00185-TUC-JGZ

ORDER ON MOTION FOR STAY PENDING APPEAL AND REQUEST FOR ADMINISTRATIVE STAY

Before the Court is Intervenor-Defendants' Motion for Stay Pending Appeal and Request for Administrative Stay. (Doc. 132.) Intervenor-Defendants request that the Court stay its July 20, 2023 preliminary injunction. In the alternative, they request an administrative stay of the injunction for seven days to allow time for the United States Court of Appeals for the Ninth Circuit to consider an emergency motion to stay and request for administrative stay.[1] The preliminary injunction at hand enjoins Defendant Horne from enforcing A.R.S. § 15-120.02 (Save Women's Sports Act) as to 11-year-old Jane Doe and 15-year-old Megan Roe. The injunction allows Plaintiffs to participate in girls' sports at their schools when athletics begin in July 2023. Neither school opposes the injunction.

"The bar for obtaining a stay of a preliminary injunction is higher than the *Winter* standard for obtaining injunctive relief." *Index Newspapers LLC v. U.S. Marshals Serv.*, 977 F.3d 817, 824 (9th Cir. 2020). In deciding whether to grant a stay, "a court considers

[1] Intervenor-Defendants request a ruling on their Motion by Monday, July 31, 2023, to allow them time to seek prompt appellate relief, if necessary. (Doc. 132 at 15.)

four factors: (1) whether the stay applicant has made a strong showing that he is likely to succeed on the merits; (2) whether the applicant will be irreparably injured absent a stay; (3) whether issuance of the stay will substantially injure the other parties interested in the proceeding; and (4) where the public interest lies." *Nken v. Holder*, 556 U.S. 418, 426 (2009) (cleaned up). "The first two factors are the most critical; the last two are reached only once an applicant satisfies the first two factors." *Al Otro Lado v. Wolf*, 952 F.3d 999, 1007 (9th Cir. 2020) (cleaned up). Applying the *Nken* factors here, the Court denies Intervenor-Defendants' Motion for Stay.

Failure to Demonstrate Strong Showing of Success on Merits

Applicants for a stay pending appeal must make a strong showing that they are likely to succeed on the merits. *Al Otro Lado*, 952 F.3d at 1010. Under the Ninth Circuit's sliding scale approach to preliminary injunctions, "the elements of the preliminary injunction test are balanced, so that a stronger showing of one element may offset a weaker showing of another." *All. for the Wild Rockies v. Cottrell*, 632 F.3d 1127, 1131 (9th Cir. 2011). For example, where there is a weak irreparable harm showing, the applicant must make a strong showing of a likelihood of success on the merits. *Al Otro Lado*, 952 F.3d at 1010. This sliding scale approach also applies to stays pending appeal. *Id.* at 1007. It is insufficient that the chance of success is better than negligible; the applicant must demonstrate "more than a mere possibility of relief." *Nken*, 556 U.S. at 434. Intervenor-Defendants fail to make the required showing.

Intervenor-Defendants argue they are likely to succeed on the merits for four reasons. (Doc. 132 at 2.) They argue that the Act is subject to rational basis review "[f]or the reasons stated" in their prior briefing. *Id.* at 9. But binding precedent holds that laws that discriminate against transgender persons are sex-based classifications subject to heightened scrutiny. *See Karnoski v Trump*, 926 F.3d 1180, 1201 (9th Cir. 2019) ("We conclude that the 2018 Policy on its face treats transgender persons differently than other persons, and consequently something more than rational basis but less than strict scrutiny applies."). Therefore, rational-basis review does not apply.

Intervenor-Defendants assert that the Court's finding that transgender females who do not undergo male puberty have no competitive advantage over female athletes is clearly erroneous because "all the competent evidence in the record suggests the opposite." (Doc. 132 at 7.) They also argue that "[t]he evidence of male competitive advantage pre-puberty is overwhelming and effectively uncontradicted." (*Id.*) These arguments misstate the record and the evidence. Experts cited by both parties agree that male physiological advantages are largely the result of circulating testosterone levels in men post-puberty. (Doc. 127 at ¶¶ 97, 100, 112-117.) In addition, Plaintiffs' expert provided persuasive evidence that any prepubertal differences between boys and girls in various athletic measurements are minimal or nonexistent. (*Id.* at ¶¶ 109-110.) Defendants' data regarding differences in prepubescent girls' and boys' physical fitness performance was not credited because the data is observational, does not determine a cause for what is observed, and fails to account for other factors which could explain the data. (*Id.* at ¶¶ 101, 103-106, 109-110.)

Intervenor-Defendants argue that the Court misapplied heightened scrutiny. (Doc. 132 at 3-7.) To withstand heightened scrutiny, a classification by sex "must serve important governmental objectives and must be substantially related to achievement of those objectives." (Doc. 127 at ¶ 145) (quoting *Craig v. Boren*, 429 U.S. 190, 197 (1976)). According to Intervenor-Defendants, the Court required perfect tailoring of the Act to Plaintiffs rather than assessing the validity of the classification as a whole. (Doc. 132 at 5-7.) Intervenor-Defendants argue that the Court disregarded extensive evidence of the competitive advantages for the large majority of transgender–female athletes, *i.e.*, those that transition after undergoing male puberty, simply because the individual Plaintiffs claim they did not, or will not, undergo male puberty.[2] (*Id.* at 4.)

This argument is unpersuasive. First, it imagines facts that were not presented. Intervenor-Defendants did not introduce any evidence, let alone extensive evidence, that

[2] Although Intervenor-Defendants disparage Plaintiffs' "claims" that they have not, and will not, undergo male puberty, Plaintiffs provide evidentiary support for their statements. *See* Doc. 127 at ¶¶ 24-27, 48-51.

the majority of transgender-female athletes have undergone male puberty. The evidence at the hearing showed only that in the past ten to twelve years, the Arizona Interscholastic Association (AIA) fielded twelve requests and approved seven students to play on a team consistent with their gender identity. (Doc. 127 at ¶ 66.) No evidence was presented as to whether any of those seven students were transgender females, and no evidence was presented as to whether any of those seven students had undergone puberty. This lack of evidence suggests that the Act's categorical bar against transgender female athletes is unrelated to the purpose of the Act.

In addition, Intervenor-Defendants' argument disregards much of the Court's heightened scrutiny analysis. In applying heightened scrutiny, the Court examined the Act's "actual purposes and carefully consider[ed] the resulting inequality." *SmithKline Beecham Corp. v. Abbott Lab'ys*, 740 F.3d 471, 483 (9th Cir. 2014). The Court found that Defendant Horne and Intervenors failed to produce "persuasive evidence at the preliminary injunction stage to show that the Act is substantially related to the legitimate goals of ensuring equal opportunity for girls to play sports and to prevent safety risks," and cited the breadth of the Act and its effect on individuals other than Plaintiffs as support. (*Id.* at ¶¶ 158-161.) Intervenor-Defendants claim in their Motion for Stay that the State's purpose is to regulate unfair advantages caused by transgender-female athletes who have undergone male puberty, but the Act broadly and categorically prohibits all transgender athletes, including prepubescent transgender athletes. The Act bans all education levels of transgender athletes—from kindergarten through college—although there is no evidence of injuries or unfair competitive advantages occurring at the kindergarten level. And despite the State's claim that the Act is intended to protect girls, the Act only bans "biological boys" from girls' teams, without prohibiting "biological girls" from playing on boys' teams, including teams made up of boys who have undergone puberty. (Doc. 127 at ¶¶ 157-160.) Given the Act's overbreadth, it cannot be said that the Court required a "perfect fit." Rather, the State failed to show "an exceedingly persuasive justification" for its discriminatory treatment, *United States v. Virginia*, 518 U.S. 515, 531 (1996), or a

justification that is genuine and not reliant on overbroad generalizations, *id.* at 533.

Finally, Intervenor-Defendants argue that the Court's conclusion that the Act violates Title IX is unlikely to be upheld on appeal because Title IX specifically authorizes separation of sports teams based on biological sex which *Bostock v. Clayton County*, 140 S. Ct. 1731 (2020), and *Grabowski v. Arizona Board of Regents*, 69 F.4th 1110 (9th Cir. 2023), do not change. (Doc. 132 at 10.) Whether legislation that prohibits all transgender athletes from participating in competitive sports violates Title IX is currently subject to debate. A mere "possibility of relief," however, fails to demonstrate a strong showing of likely success on the merits, particularly in light of Plaintiffs' equal protection claim.

The Court concludes that Intervenor-Defendants fail to make a strong showing that they are likely to succeed on the merits of their claim. This failure is particularly detrimental because, as discussed below, Intervenor-Defendants' showing of irreparable harm is weak. *See Al Otro Lado*, 952 F.3d at 1010 (where there is a weak irreparable harm showing, the applicant must make a strong showing of a likelihood of success on the merits). Thus, the first *Nken* factor favors Plaintiffs.

Intervenor-Defendants Will Not Suffer Irreparable Harm Absent Stay

An applicant for stay pending appeal must demonstrate that a stay is necessary to avoid likely irreparable injury to the applicant while an appeal is pending. *Al Otro Lado*, 952 F.3d at 1007. Showing a possibility of irreparable injury is insufficient. *Id.* The applicant is required to show that irreparable harm is likely to occur before the appeal is decided. *Id.* The applicant's irreparable harm burden "is higher than it is on the likelihood of success prong, as [it] must show that an irreparable injury is the more probable or likely outcome." *Id.*

In its Order granting the preliminary injunction, the Court concluded, "There is no evidence that any Defendant will be harmed by allowing Plaintiffs to continuing playing with their peers as they have done until now." (Doc. 127 at ¶ 184.) Intervenor-Defendants advance little argument as to their irreparable harm, citing only "the sovereign interest of the State of Arizona in enforcing its valid statutes." (Doc. 132 at 14.). Clearly, however,

there is no irreparable harm if the statute is not valid. Intervenor-Defendants "cannot suffer harm from an injunction that merely ends an unlawful practice." *Rodriguez v. Robbins*, 715 F.3d 1127, 1145 (9th Cir. 2013). The second *Nken* factor favors Plaintiffs.

Substantial Injury to Other Parties

Because Intervenor-Defendants fail to establish the first two *Nken* factors, the Court need not address the last two factors. *See Al Otro Lado*, 952 F.3d at 1007 ("The first two factors are the most critical; the last two are reached only once an applicant satisfies the first two factors.") (cleaned up). However, factors three and four also do not support Intervenor-Defendants' request for stay.

The third factor, "whether issuance of the stay will substantially injure the other parties interested in the proceeding," weighs against granting a stay. Plaintiffs will suffer injury in the absence of a stay. Prior to the Act, there were no bars to Plaintiffs participating in girls' sports at their schools. If a stay is granted, Plaintiffs will suffer severe and irreparable mental, physical, and emotional harm if the Act applies to them because they cannot play on boys' sports teams; the Act will effectively exclude Plaintiffs from school sports and deprive them of the social, educational, physical, and emotional health benefits that both sides acknowledge come from school sports; and Plaintiffs will suffer the shame and humiliation of being unable to participate in a school activity simply because they are transgender—a personal characteristic over which they have no control. (Doc. 127 at ¶¶ 174-176.) The school year has started, and Plaintiffs want to participate in girls' sports. The issuance of a stay would deprive Plaintiffs the opportunity to participate in girls' first quarter sports—which are currently in progress—including the first cross-country meet scheduled for August 14, 2023. (Doc. 127 at ¶¶ 32, 35, 38, 41, 55, 57-60.)

Intervenor-Defendants argue that the preliminary injunction imposes irreparable harm on other interested parties. (Doc. 132 at 12-14.) They argue that, absent a stay, "biological girls" will be unfairly displaced from participation in girls' sports by Plaintiffs, whose involvement will necessarily exclude "biological girls" who try out for the team, and that Plaintiffs' involvement will reduce the other girls' playing time and success. (*Id.*

at 12-13.) However, there is no evidence that Plaintiffs' participation will cause such harms to other participants. There is no evidence that the schools limit the number of girls who participate in any of the sports at issue and there is no evidence that either Plaintiff would present an advantage, let alone an unfair advantage, if allowed to participate.

Public Interest Lies in Plaintiffs' Favor

Intervenor-Defendants argue that the public interest favors a stay because the public has an interest in upholding the laws passed by their elected officials. (Doc. 132 at 15.) However, as discussed above, a state cannot suffer harm from an injunction that merely ends a discriminatory practice. *Rodriguez*, 715 F.3d at 1145. Thus, it follows that, "it is always in the public interest to prevent the violation of a party's constitutional rights." (Doc. 127 at ¶ 180) (quoting *Melendres v. Arpaio*, 695 F.3d 990, 1002 (9th Cir. 2012)). The fourth *Nken* factor supports denial of the Motion for Stay.

Administrative Stay Would Disrupt Status Quo

As an alternative to their request for a stay pending appeal, Intervenor-Defendants request a seven-day administrative stay to allow the Circuit Court of Appeals time to consider their emergency motion to stay the preliminary injunction order. (Doc. 132 at 15.) An administrative stay "is only intended to preserve the status quo until the substantive motion for a stay pending appeal can be considered on the merits." *Doe #1 v. Trump*, 944 F.3d 1222, 1223 (9th Cir. 2019). The *Nken* factors do not support imposition of an administrative stay. Moreover, prohibiting Plaintiffs from participating in girls' athletics would disrupt the status quo. Accordingly,

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IT IS ORDERED that Intervenor-Defendants' Motion for Stay Pending Appeal and Request for Administrative Stay (Doc. 132) is DENIED.

Dated this 31st day of July, 2023.

Honorable Jennifer G. Zipps
United States District Judge

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IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA

Helen Doe, et al.,

Plaintiffs,

v.

Thomas C Horne, et al.,

Defendants.

No. CV-23-00185-TUC-JGZ

ORDER ON MOTION FOR PRELIMINARY INJUNCTION AND FINDINGS OF FACT AND CONCLUSIONS OF LAW

INTRODUCTION

Plaintiffs filed this action on April 17, 2023, seeking preliminary and permanent injunctive relief related to the implementation of A.R.S. § 15-120.02, the Save Women's Sports Act ("the Act"), which Plaintiffs allege precludes them from playing on girls' sports teams because they are transgender girls. Plaintiffs assert that they have not undergone male puberty and do not have a competitive or physiological advantage over their non-transgender peers on these teams. Plaintiffs ask the Court for declaratory relief that enforcement by Defendants of Ariz. Rev. Stat. § 15-120.02 violates Plaintiffs' rights under the Equal Protection Clause of the Fourteenth Amendment to the United States Constitution, Title IX, 20 U.S.C. § 1681 et seq., the Americans with Disabilities Act, 42 U.S.C. § 12101, et seq., and Section 504 of the Rehabilitation Act, 29 U.S.C. § 794, et seq.

The Arizona legislature adopted A.R.S. § 15-120.02, effective September 24, 2022, as follows: "Each interscholastic or intramural athletic team or sport that is sponsored by a public school or a private school whose students or teams compete against a public school shall be expressly designated as one of the following based on the biological sex of the students who participate on the team or in the sport: 1) 'males,' 'men' or 'boys'; 2) 'females,' 'women' or 'girls,' and 3) 'coed' or 'mixed'." "Athletic teams or sports designated for 'females,' 'women' or 'girls' may not be open to students of the male sex." The statute does not apply to "restrict the eligibility of any student to participate in any . . . athletic team or sport designated as being for males, men or boys or designated as coed or mixed." The statute creates a private cause of action for injunctive relief and damages for any student for a deprivation of an athletic opportunity or who has suffered any direct or indirect harm as a result of a school knowingly violating this section.

The Motion for Preliminary Injunction asks the Court to enjoin enforcement of A.R.S. § 15-120.02 by Defendant Horne and enjoin implementation of and compliance with the Act by Defendants Kyrene Middle School and The Gregory School (TGS) as to Plaintiffs. The Court has granted intervenor status to the legislators who adopted the Act. The Motion for Preliminary Injunction was fully briefed by all parties and the Intervenor Legislators ("Intervenors"). The Court will grant the Motion for Preliminary Injunction pursuant to the Findings of Fact and Conclusions of Law set out below. Defendant Arizona Interscholastic Association, Inc.'s ("AIA") transgender policy, allowing transgender girls to play on teams consistent with their gender identity, complies with the terms of the preliminary injunction.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

On July 10, 2023, the Court heard oral argument and took evidence pertaining to Plaintiffs' Motion for Preliminary Injunction. Having heard oral argument, having examined the proofs[1] offered by the parties, and having heard the arguments of counsel

[1] By stipulation, the parties offer proof by way of expert declarations filed in advance of the hearing and by supplement thereafter. Accordingly, the Court references the evidence herein by CM/ECF document number, not as a trial exhibit.

and being fully advised herein, the Court now finds generally in favor of Plaintiffs and against the Defendants, and hereby makes the following special Findings of Fact and Conclusions of Law pursuant to the Federal Rules of Civil Procedure, Rule 52(a) and (c) which constitutes the decision of the Court herein:

I. <u>Findings of Fact</u>

To the extent these Findings of Fact are also deemed to be Conclusions of Law, they are hereby incorporated into the Conclusions of Law that follow.

A. <u>Gender identity and gender dysphoria</u>.

1. "Gender identity" is the medical term for a person's internal, innate, deeply held sense of their own gender. (Dr. Daniel Shumer ("Shumer Decl.") (Doc. 5) ¶ 18.) Everyone has a gender identity. (*Id.*)

2. "Gender identity" differs from "gender role," which are behaviors, attitudes, and personality traits that a particular society considers masculine or feminine, or associates with male or female social roles. For example, the convention that girls wear pink and have longer hair, or that boys wear blue and have shorter hair, are socially constructed gender roles. Gender identity does not refer to socially contingent behaviors, attitudes, or personality traits; it is an internal and largely biological phenomenon. (Shumer Decl. (Doc. 5) ¶¶ 19-22.)

3. There is a consensus among medical organizations that gender identity is innate and cannot be changed through psychological or medical treatments. (Dr. Stephanie Budge Rebuttal ("Budge Decl. (Rebuttal)") (Doc. 65-1) ¶ 31; Dr. Stephanie Budge ("Budge Decl.") (Doc. 4) ¶ 21; Daniel Shumer Rebuttal ("Shumer Decl. (Rebuttal)") (Doc. 65-2) ¶¶ 54–58; Shumer Decl. (Doc. 5) ¶ 23.)

4. When a child is born, a health care provider identifies the child's sex based on the child's observable anatomy. (Budge Decl. (Doc. 4) ¶ 18; Shumer Decl. (Doc. 5) ¶ 27.) This identification is known as an "assigned sex," and in most cases turns out to be consistent with the person's gender identity. (Budge Decl. (Doc. 4) ¶ 18; Shumer Decl. (Doc. 5) ¶ 27.)

5. The term "biological sex" is not defined in the Act, but the Court finds that as used by Defendants it is synonymous with the term "assigned sex." (*See* Declaration of Dr. James

M. Cantor ("Cantor Decl.") (Doc. 82-2; Doc. 92-2) ¶¶ 105-107; Declaration of Dr. Gregory A. Brown ("Brown Decl.") (Doc. 82-1; 92-1) ¶ 1; Dr. Emma Hilton ("Hilton Decl.") (Doc. 92-8) ¶¶1.8, ¶ 3.1-3.2 (explaining sex is an objective feature determined at the moment of conception; infants are born male or female, ascertainable by chromosomal analysis or visual inspection at birth).)[2]

6. For a transgender person, that initial designation does not match the person's gender identity. (Budge Decl. (Doc. 4) ¶ 18; Shumer Decl. (Doc. 5) ¶ 27.)

7. Gender dysphoria is a serious medical condition characterized by significant and disabling distress due to the incongruence between a person's gender identity and assigned sex. (Budge Decl. (Doc. 4) ¶ 23; Shumer Decl. (Doc. 5) ¶ 28.) Defendant Horne and Intervenors accept that gender dysphoria is a medical condition. (Preliminary Injunction, Oral Argument: July 10, 2023).

8. Gender dysphoria is highly treatable. Every major medical association in the United States agrees that medical treatment for gender dysphoria is necessary, safe, and effective. (Budge Decl. (Doc. 4) ¶ 25; Shumer Decl. (Doc. 5) ¶ 30.)

9. "Transgender individuals may experience 'gender dysphoria,' which is 'characterized by significant and substantial distress as result of their birth-determined sex being different from their gender identity.' 'In order to be diagnosed with gender dysphoria, the incongruence must have persisted for at least six months and be accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning.' If left untreated, symptoms of gender dysphoria can include severe anxiety and depression, suicidality, and other serious mental health issues. Attempted suicide rates in the transgender community are over 40%." *Hecox v. Little*, 479 F. Supp. 3d 930, 945-46 (D. Idaho 2020) (cleaned up), *aff'd* No. 20-35813, 2023 WL 1097255 (9th Cir. Jan. 30,

[2] From a medical perspective, the terms "biological sex," "biological male," and "biological female" are imprecise terms because a person's sex encompasses several different biological attributes, including sex chromosomes, certain genes, gonads, sex hormone levels, internal and external genitalia, other secondary sex characteristics, and gender identity, which may or may not be in alignment. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶44 (citing Joshua D. Safer, *Care of Transgender Persons*, 381 N. Engl. J. Med. 2451 (2019)).

2023).

10. The major associations of medical and mental health providers in the United States, including the American Medical Association, the American Academy of Pediatrics, the American Psychiatric Association, the American Psychological Association, and the Pediatric Endocrine Society, have endorsed medical standards of care for treating gender dysphoria in adolescents, which were developed by the World Professional Association for Transgender Health ("WPATH") and the Endocrine Society. (Shumer Decl. (Doc. 5) ¶ 31.)

11. The goal of medical treatment for gender dysphoria is to alleviate a transgender patient's distress by allowing them to live consistently with their gender identity. (Budge Decl. (Doc. 4) ¶ 27; Shumer Decl. (Doc. 5) ¶ 30.)

12. Undergoing treatment to alleviate gender dysphoria is commonly referred to as "transition" and includes one or more of the following components: (i) social transition, including adopting a new name, pronouns, appearance, and clothing, and correcting identity documents; (ii) medical transition, including puberty-delaying medication and hormone-replacement therapy; and (iii) for adults, surgeries to alter the appearance and functioning of primary- and secondary-sex characteristics. (Budge Decl. (Doc. 4) ¶¶ 26–27; Shumer Decl. (Doc. 5) ¶ 34.)

13. For social transition to be clinically effective, it must be respected consistently across all aspects of a transgender individual's life. (Budge Decl. (Doc. 4) ¶ 27.)

14. At the onset of puberty, adolescents with gender dysphoria may be prescribed puberty-delaying medications to prevent the distress of developing physical characteristics that conflict with the adolescent's gender identity. (Budge Decl. (Doc. 4) ¶ 28; Shumer Decl. (Doc. 5) ¶ 35.)

15. For older adolescents, doctors may also prescribe hormone therapy to induce the puberty associated with the adolescent's gender identity. (Budge Decl. (Doc. 4) ¶ 28; Shumer Decl. (Doc. 5) ¶ 36.)

16. When transgender adolescents are provided with appropriate medical treatment and have parental and societal support, they can thrive. (Shumer Decl. (Doc. 5) ¶ 29.)

17. Untreated gender dysphoria can cause serious harm, including anxiety, depression, eating disorders, substance abuse, self-harm, and suicide. (Budge Decl. (Doc. 4) ¶ 33; Shumer Decl. (Doc. 5) ¶ 28.)

18. Being denied recognition and support can cause significant harm, exacerbate gender dysphoria, and expose transgender adolescents to the risk of discrimination and harassment. (Budge Decl. (Doc. 4) ¶¶ 33–34; Shumer Decl. (Doc. 5) ¶ 28.)

19. Attempts to "cure" transgender individuals by forcing their gender identity into alignment with their birth sex are harmful and ineffective. Those practices have been denounced as unethical by all major professional associations of medical and mental health professionals, such as the American Medical Association, the American Academy of Pediatrics, the American Psychiatric Association, and the American Psychological Association, among others. (Shumer Decl. (Doc. 5) ¶ 25.)

B. <u>Plaintiffs are transgender girls who have not and will not experience male puberty</u>.

20. Plaintiff Jane Doe is an 11-year-old transgender girl who will attend Kyrene Aprende Middle School beginning on July 19, 2023. (Jane Doe ("J. Doe Decl.") (Doc. 6) ¶ 1; Helen Doe (Second) ("H. Doe 2nd Decl.") (Doc. 78) ¶ 3.)

21. Jane has lived as a girl in all aspects of her life since she was five years old. (J. Doe Decl. (Doc. 6) ¶ 2; Helen Doe ("H. Doe Decl.") (Doc. 7) ¶¶ 3, 5.)

22. Jane was diagnosed with gender dysphoria when she was seven years old. (H. Doe Decl. (Doc. 7) ¶ 7.)

23. Jane has changed her name through a court order to a more traditional female name and has a female gender marker on her passport. (Pls. Exs. 13 (Doc. 90-1), 15 (Doc. 90-3).)

24. Jane has been monitored by her doctor for signs of the onset of puberty as part of her medical treatment for gender dysphoria. (H. Doe Decl. (Doc. 7) ¶ 11.)

25. At an appointment on June 27, 2023, Jane's doctor prescribed a Supprelin implant, which is a puberty-blocking medication. (Helen Doe (Third) ("H. Doe 3rd Decl.")

(Doc. 97-1) ¶ 4.)

26. Jane is in the process of scheduling the implant procedure for as soon as possible. (*Id.*)

27. Accordingly, Jane has not and will not experience any of the physiological changes that increased testosterone levels would cause in a pubescent boy. (Shumer Decl. (Doc. 5) ¶ 45; Budge Decl. (Doc. 4) ¶ 28.)

28. Sports are very important to Jane and her parents. (J. Doe Decl. (Doc. 6) ¶ 5; H. Doe Decl. ¶ 12.)

29. Jane particularly loves playing soccer and has played soccer on girls' club and recreational sports teams for nearly five years. (J. Doe Decl. (Doc. 6) ¶¶ 6–8; H. Doe Decl. (Doc. 7) ¶ 12.)

30. Aside from its physical and emotional health benefits, soccer has helped Jane make new friends and connect with other girls. (J. Doe Decl. (Doc. 6) ¶ 7; H. Doe Decl. (Doc. 7) ¶ 13.)

31. Jane's teachers, coaches, friends, and members of her soccer team have all been supportive of Jane's identity. (H. Doe Decl. (Doc. 7) ¶ 9; Stipulation in Lieu of Answer ("Kyrene/Toenjes Stip.") (Doc. 59) ¶ 1.)

32. When Jane enters Kyrene Aprende Middle School this July, she intends to participate and compete with the cross-country team and try out for the girls' soccer and basketball teams. (J. Doe Decl. (Doc. 6) ¶ 9; H. Doe 2nd Decl. (Doc. 78) ¶ 4.)

33. Both the soccer and basketball teams at Kyrene Aprende Middle School have separate teams for boys and girls. (J. Doe Decl. (Doc. 6) ¶ 9.)

34. The cross-country team trains together, but boys and girls compete separately. (*Id.*)

35. Registration for the cross-country team began on July 1, 2023. (H. Doe 2nd Decl. (Doc. 78) ¶ 6.)

36. The registration occurs online and involves the submission of registration forms and supporting documents, such as a physical report signed by a doctor. (*Id.*)

37. Typically, a student's registration takes at least two to three days to process after it is submitted. (*Id.*)

38. The first practice for cross country is on July 31, 2023, and the first cross-country competitive meet will occur the week of August 14, 2023. (*Id.* ¶ 7.)

39. Jane is excited to participate and compete on the girls' teams with her friends and peers. (J. Doe Decl. (Doc. 6) ¶¶ 8–9.)

40. If not for the Act, the Kyrene School District would permit Jane Doe to play on girls' sports teams. (Kyrene/Toenjes Stip. (Doc. 59) ¶ 1.)

41. However, if the Act is applied to Jane, she will not be able to play on the girls' soccer and basketball teams or compete with the girls' cross-country team. (*Id.*)

42. Plaintiff Megan Roe is a 15-year-old transgender girl who attends TGS. (Megan Roe ("M. Roe Decl.") (Doc. 8) ¶¶ 2, 5.)

43. Megan has always known she is a girl. (Kate Roe ("K. Roe Decl.") (Doc. 9) ¶ 3.)

44. Megan has lived as a girl in all aspects of her life since she was seven years old. (M. Roe Decl. (Doc. 8) ¶ 3; K. Roe Decl. (Doc. 9) ¶¶ 4–5.)

45. Through a court order, Megan has changed her name to a more traditional female name and her gender to female. (Pls.' Ex. 14 (Doc. 90-2).) She also has a female gender marker on her passport. (Pls.' Ex. 16 (Doc. 90-4).)

46. Megan was diagnosed with gender dysphoria when she was ten years old. (K. Roe Decl. (Doc. 9) ¶ 6.)

47. Before starting school at TGS, Megan's parents shared with administrators and teachers at the school that Megan is a transgender girl. (M. Roe Decl. (Doc. 8) ¶ 5.) TGS has been very supportive of Megan and her identity. (*Id.*; Defendant TGS Motion to Dismiss ("TGS Mot. to Dismiss") (Doc. 37) at 3.)

48. Megan has been taking puberty blockers since she was 11 years old as part of her medical treatment for gender dysphoria. (M. Roe Decl. (Doc. 8) ¶ 6; K. Roe Decl. (Doc. 9) ¶ 6.) This prevented Megan from undergoing male puberty. (K. Roe Decl. (Doc. 9) ¶ 6.)

49. Megan began receiving hormone therapy when she was 12 years old. (M. Roe Decl. ¶ 6; K. Roe Decl. (Doc. 9) ¶ 6.)

50. As a result of the puberty blockers and hormone therapy, Megan has not experienced the physiological changes that increased testosterone levels would cause in a pubescent boy. (K. Roe Decl. (Doc. 9) ¶ 6; Shumer Decl. (Doc. 5) ¶ 47; Budge Decl. (Doc. 4) ¶ 29.)

51. The hormone treatment that she has received has caused Megan to develop many of the physiological changes associated with puberty in females. (Shumer Decl. (Doc. 5) ¶ 47; see also Budge Decl. (Doc. 4) ¶ 29.)

52. Sports have always been a part of Megan's life. (M. Roe Decl. (Doc. 8) ¶ 4.)

53. When she was about seven years old, Megan joined a swim team. (K. Roe Decl. (Doc. 9) ¶ 7.)

54. The coach of the swim team was supportive of Megan and her gender identity. (*Id.*)

55. Megan intends to try out for the girls' volleyball team at TGS for this year's fall season. (M. Roe Decl. (Doc. 8) ¶ 7.)

56. Volleyball is an important part of the TGS community and many students attend the games. (M. Roe Decl. (Doc. 8) ¶ 8; K. Roe Decl. (Doc. 9) ¶ 8.)

57. Megan is excited to play on the girls' volleyball team with her friends. (M. Roe Decl. (Doc. 8) ¶ 7; K. Roe Decl. (Doc. 9) ¶ 8.)

58. Megan's teammates, coaches, and school are highly supportive of her and would welcome her participation on the girls' volleyball team. (M. Roe Decl. (Doc. 8) ¶ 5; K. Roe Decl. (Doc. 9) ¶ 5; TGS Mot. to Dismiss (Doc. 37) at 3; Dr. Julie Sherrill ("Sherrill Decl.") (Doc. 37-1) ¶ 5.)

59. If not for the Act, TGS would permit Megan to play on the girls' volleyball team. (Sherrill Decl. (Doc. 37-1) ¶ 5.)

60. If the Act is applied to Megan, she will not be able to compete with the girls' volleyball team. (*Id.*)

C. <u>Prior to enactment of A.R.S. § 15-120.02, Plaintiffs would have been allowed to play on girls' sports teams</u>.

61. Defendant AIA sets rules for governing interscholastic sports, grades 9-12, and cutoff age of 19, for member schools, with membership being voluntary, but compliance with AIA rules being mandatory for all membership schools. (AIA Constitution; Article 2. Membership (Doc. 51-1).)

62. Each school or school district set its own rules on transgender students' participation in intramural sports. (*Id.* ¶¶ 2.5.2–3 (vesting "[f]inal authority and ultimate responsibilities in all matters pertaining to interscholastic activities of each school shall be vested in the school principal," with school administration assuming responsibility for verification of all student eligibility rules).)

63. Prior to the enactment of the Act, A.R.S. § 15-120.02, transgender girls in Arizona were permitted to play on girls' sports teams, under the AIA Constitution, Bylaws, Policies and Procedures § 41.9, as follows: "[A]ll students should have the opportunity to participate in Arizona Interscholastic Association activities in a manner that is consistent with their gender identity, irrespective of the sex listed on a student's eligibility for participation in interscholastic athletics or in a gender that does not match the sex at birth." (AIA Resp., Ex. 1 (Doc. 51-1).)

64. By December 2018, the AIA formalized its policy to permit transgender students to play on teams consistent with their gender identity so long as they had a letter of support from their parent or guardian explaining when they realized they were transgender. (Compl. (Doc. 1) ¶ 21; AIA Answer (Doc. 50) ¶ 21; AIA Transgender Policy § 41.9 (Doc. 51-1).)

65. Under the AIA policy, a student request to play on a team consistent with his or her gender identity is reviewed by a committee of medical and psychiatric experts, and consistent with AIA health and safety policy and if not motivated by an improper purpose, the request is approved or denied. (AIA Resp., Ex. 1 (Doc. 51-1) § 41.9.3; Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2d

Reg. Sess. 50:12–52 (Ariz. 2022).)

66. In the past 10 to 12 years, the AIA fielded approximately 12 requests consistent with their policy and approved seven students to play on a team consistent with their gender identity. Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2d Reg. Sess. 52:10 (Ariz. 2022).

67. The parties do not provide the Court with a breakdown of the gender identity for these seven transgender students but even assuming they were all transgender girls, the Court finds that seven students over 10 to 12 years is not a substantial number, particularly when compared to the "roughly 170,000 students playing sports in Arizona." (Preliminary Injunction, Oral Argument: July 10, 2023).[3]

68. Less than one percent of the population is transgender, with male and female transgender people being roughly the same in number. *Hecox*, 479 F. Supp. 3d at 977–78. "Presumably, this means approximately one half of one percent of the population is made up of transgender females. It is inapposite to compare the potential displacement allowing approximately half of the population (cisgender[4] men) to compete with cisgender women, with any potential displacement one half of one percent of the population (transgender women) could cause cisgender women. It appears untenable that allowing transgender women to compete on women's teams would substantially displace female athletes." *Id.* at 977-978.

69. The Arizona Bill Summary for the Act, SB 1165 transmitted to the Governor on May 11, 2022, expressly cites the AIA's "policy allowing transgender students to participate in activities in a manner consistent with their gender identity. (AIA Policies and Procedure, Art. 41 § 9)." (2022 Reg. Sess. S.B. 1165, Bill Summary).

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[3] The record is missing the relevant number of participants in girls' sports and in sports generally over this same 10-to-12-year period. Based on its independent research, the Court accepts the 170,000 number as representing the total number of students playing sports per year because in 2018-19, there were 52,817 girls and 68,520 boys playing sports in Arizona. https://www.statista.com/statistics/202219/us-high-school-athletic-participation-in-arizona.

[4] "The term 'cisgender' refers to a person who identifies with the sex that person was determined to have at birth." *Hecox*, 479 F. Supp. 3d at 945 (relying on *Doe v. Boyertown*, 897 F.3d 518, 522 (3rd Cir. 2018)).

70. Despite enactment of the Act, the AIA has not changed its transgender policy. (AIA Resp. (Doc. 51) at 5.) Yet, organizations like the AIA do not have discretion to disregard validly enacted laws of the State of Arizona. (AIA Resp. (Doc. 51) at 4.)

71. The Act prohibits "any licensing or accrediting organization or any athletic association or organization," including the AIA, from "entertain[ing] a complaint, open[ing] an investigation or tak[ing]any other adverse action against a school for maintaining separate interscholastic or intramural athletic teams or sports for students of the female sex." A.R.S. § 15-120.02(D).

72. The Act creates a private cause of action for students or schools to sue schools or organizations like the AIA if the school or organization violates the ban or retaliates in response to the reporting of a violation of the Act. A.R.S. § 15-120.02(F)-(G).

D. A.R.S. § 15-120.02 prevents Plaintiffs from playing on girls' sports teams at their schools.

73. On March 30, 2022, Arizona enacted the Act (S.B. 1165), with an effective date of September 24, 2022. Ariz. Rev. Stat. § 15-120.02.

74. As of the effective date of the Act, School Year 2022-23, first quarter (July-September) sports, including volleyball, were almost over. Second quarter (October-December) girls' sports are softball and soccer. The Third quarter (January-March) sports for girls, includes basketball. The Fourth quarter (March-May) sport is track and field.

75. In School Year 2022-23, Megan was allowed to practice as a member of the team, but not allowed to participate in TGS interscholastic competitions (games). (TGS Mot. to Dismiss (Doc. 37) at 3, n3.)

76. In School Year 2022-23, Jane played soccer but not at her elementary school because it did not have a school team; she will attend Kyrene Middle School for the first time this year. (Preliminary Injunction, Oral Argument: July 10, 2023).

77. The Court finds that the challenged conduct, passage of the Act precluding transgender girls from playing on girls' sports teams, occurred at a time when the Plaintiffs had an opportunity to play on girls' sports teams consistent with their gender identity.

78. Unlike the prior case-by-case basis used to approve a transgender girl's request to play on a team consistent with her gender identity, which considered among other things the age and competitive level relevant to the request, the Act categorically bans all transgender girls' participation by requiring each team that is sponsored by a public school or a private school team that competes against a public-school team to be designated as "male," "female," or "coed," based on the "biological sex of the students who participate." Ariz. Rev. Stat. § 15-120.02(A).

79. The Act applies equally to kindergarten through college teams although the problems identified as being addressed by the Act-- opportunity and safety-- are limited to high school and college sports. *See e.g.* Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2d Reg. Sess., 0:9:56 (Ariz. 2022) (Sharp testimony explaining problem being addressed is AIA policy that allows males in a matter of weeks to dominate a sport, break a girl's record, and cause a girl to lose her championship or scholarship opportunity); same at 1:24:00 (Sen. Burley explanation for vote "yea" to protect integrity of high school sports by preventing victimization of girls that are trying to compete for sports scholarships).[5]

80. "Biological sex" is not defined in the statute. Ariz. Rev. Stat. § 15-120.02. However, the S.B. 1165 Legislative Findings state that for purposes of school sports, a student's sex is determined at "fertilization and revealed at birth, or, increasingly, in utero." S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2022), § 2.

81. The Act states that "athletic teams or sports designated for 'females', 'women' or 'girls' may not be open to students of the male sex." Ariz. Rev. Stat. § 15-120.02 (B).

82. The Act was adopted for the purpose of excluding transgender girls from playing on girls' sports teams. *See, e.g*. Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2d Reg. Sess., 1:17:32–39 (Ariz. 2022) (statement of Sen. Vince Leach, Member, S. Comm. on Judiciary) (explaining his vote for the bill by stating, "if we allow transgenders to take over female sports, you will not have

[5] http: https://www.azleg.gov/videoplayer/?clientID=6361162879&eventID=2022011057

females participating"); 1:28:28–55 (statement of Sen. Warren Petersen, Chairman, S. Comm. on Judiciary) (questioning whether those opposing the bill would "be opposed to having just a trans league, so that they can all compete in their own league"); (Pls.' Ex. 25, Gov. Douglas Ducey Signing Letter) ("S.B. 1165 creates a statewide policy to ensure biologically female athletes at Arizona public schools, colleges, and universities have a level playing field to compete….This legislation simply ensures that the girls and young women who have dedicated themselves to their sport do not miss out… due to unfair competition.")

83. Precluding transgender girls, who have not experienced male puberty, from playing girls sports, treats transgender boys and transgender girls differently and treats boys' and girls' sports differently, with only girls' teams facing potential challenges, including litigation, related to suspected transgender players. *Compare* Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. On Judiciary, Jan. 20, 2022, 55th Leg., 2d Reg. Sess., 0:18:16 (inviting legislators to come see purported transgender girl on a team and describing need to challenge suspected transgender girls on opposing teams) *with Hecox*, 479 F. Supp. 3d at 988 (explaining all biological women are subject, in the event of a challenge, to the statutory verification process in order to play on a team, and this creates a different, more onerous set of rules for women's sports when compared to men's sports).

84. Contrary to the asserted safety goal, the Act does not protect transgender boys—identified by Defendant Horne and Intervenors as "biological girls." In fact, the Act allows "biological girls" to play on boys' sports teams, subjecting them to the alleged risks of that association. This is allowed prepuberty and without regard for whether the transgender boy is receiving testosterone enhancements.

85. The Act's creation of a private cause of action against a school for any student who is deprived of an athletic opportunity or suffers any harm, whether direct or indirect, related to a schools' failure to preclude participation of a transgender girl on a girls' team places an onerous burden on girls' sports programs, not faced by boys' athletic programs.

86. The record does not support a finding that prior to the Act's enactment, there was a problem in Arizona related to transgender girls replacing non-transgender girls on sports teams. Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2d Reg. Sess., 1:15:30–36 (Ariz. 2022) (statement of Sen. Warren Petersen, Chairman, S. Comm. on Judiciary) (acknowledging to another Senator that "we're not aware of a specific instance" where any cisgender girl had lost a place on a team to a transgender girl).

87. The record does not support a finding that during the 10 to 12 years prior to passage of the Act there was a risk of any physical injury to or missed athletic opportunity by any girl as a result of allowing seven transgender girls to play on sports teams consistent with their gender identity.

E. Excluding Plaintiffs from school sports causes very serious injury to Plaintiffs

88. School sports offer social, emotional, physical, and mental health benefits. (Budge Decl. (Doc. 4) ¶¶ 35–38.)

89. The social benefits of school sports include the opportunity to make friends and become part of a supportive community of teammates and peers. (*Id.* ¶ 35.)

90. School sports provide an opportunity for youth to gain confidence and reduce the effects of risk factors that lead to increases in depression. (*Id.* ¶ 36.)

91. Students who play school sports have fewer physical and mental health concerns than those that do not. (*Id.* ¶ 37.)

92. Students who participate in high school sports are more likely to finish college and participation in high school sports has a positive impact on academic achievement. (*Id.* ¶ 38.)

93. It would be psychologically damaging for a transgender girl to be banned from playing school sports on equal terms with other girls. (*Id.* ¶ 39; Budge Decl. (Rebuttal) (Doc. 65-1) ¶ 10.)

94. Transgender girls will internalize the shame and stigma of being excluded for a

personal characteristic (being transgender) over which they have no control and which already subjects them to prejudice and social stigma. (Budge Decl. (Doc. 4) ¶ 40.)

95. For transgender girls who are already playing on girls' teams, a law that requires them to be excluded from continued participation on girls' teams would have a further negative impact on their health and well-being, causing them to feel isolated, rejected, and stigmatized, and thereby putting them at high risk for severe depression and/or anxiety. (*Id.*)

96. For transgender girls, who are gender transitioning to address gender dysphoria, the benefits from playing sports on teams compatible with their gender identity is important because to be clinically effective, gender transitioning must be respected consistently across all aspects of her life.

F. <u>Transgender girls who have not undergone male puberty do not have an athletic advantage over other girls</u>.

97. The Plaintiffs' experts' opinions are based on the scientific consensus that the biological cause of average differences in athletic performance between men and women is caused by the presence of circulating levels of testosterone beginning with male puberty. (Shumer Decl. (Rebuttal) (Doc 65-2) ¶ 8 (citing Brown Decl. ¶¶ 127–30 relying on Handelsman (2018) at 823 ("summarizing evidence makes it highly likely that the sex difference in circulating testosterone of adults explains most, if not all, of the sex differences in sporting performance.")); (Brown Hecox Decl. ¶¶ 20a, 25–28, 77–85).

98. A large part of the record created by the Defendants is not relevant to the question before the Court: whether transgender girls like Plaintiffs, who have not experienced male puberty, have performance advantages that place other girls at a competitive disadvantage or at risk of injury. For example, Defendants submit evidence that girls have more body fat than boys at birth. (Brown Decl. (Doc. 82-1; 92-1) ¶ 79.) Without more, this evidence is not relevant to the question before the Court.

99. Defendant Horne and the Intervenors submit expert declarations, including the declaration by Dr. James Cantor, which in large part are not relevant criticisms of medical

treatments for gender dysphoria. The appropriateness of medical treatment for gender dysphoria is not at issue in this case. (Pls Ex. (Doc. 88-3) at 39-40 (dated March 30, 2022, describing purpose of Act to ensure a level playing by preventing unfair competition in women's sports).) Protecting transgender girls from any such risk is not a rationale or purpose of the Act.

100. Defendants' expert Dr. Brown admits that many of the specific male physiological advantages he describes are a result of testosterone levels in men post-puberty. This evidence is not relevant because the Plaintiffs have not and never will experience male puberty. The Court is not concerned with Dr. Brown's opinion that such advantages are not reversed by testosterone suppression after puberty or are reduced only modestly, leaving a large advantage over female athletes. Dr. Brown agrees it is well documented that the large increases in physiological and performance advantages for men result from increases in circulating testosterone levels that males experience in puberty, "or generally between the ages of about 12 through 18." (Brown Decl. (Doc. 82-1; 92-1) ¶¶ 163-164.)[6]

101. Defendants rely on school-based fitness testing of boys and girls, comparisons between $10^{th}/50^{th}/90^{th}$ percentile scores for girl and boy students ages 6 through 11 reflecting, for example, that 50% of 6-year-old boys completed more laps in the 20-meter shuttle (14) than girls (12). (Brown Decl. (Doc. 82-1; 92-1) ¶ 84.) Other fitness data reflects differences between 9 through 17-year-old boys and girls, with 9-year-old boys always exceeding girls' running times by various percentages ranging from 11.1-15.2%, *id.* ¶ 89; arm hang fitness scores (7.48 boys, 5.14 girls), *id.* ¶ 92; standing broad jump (128.3 boys, 118.0 girls), *id.* ¶ 99. (*See also* Brown Decl. (Doc. 82-1; 92-1) ¶106 (quoting Thomas 1985 study at 266) ("Boys exceed girls in throwing velocity by 1.5 standard deviation units as early as 4 to 7 years of age . . ." and throwing distance by 1.5 standard deviation units as

[6] A categorical bar to girls and women who are transgender stands in "stark contrast to the policies of elite athletic bodies that regulate sports both nationally and globally—including the National Collegiate Athletic Association ("NCAA") and the International Olympic Committee ("IOC")—which allow transgender women to participate on female sports teams once certain specific criteria are met," primarily specified levels of circulating testosterone. *Hecox*, 479 F. Supp. 3d at 944.

early as 2 to 4 years of age).)[7] (*But see* Shumer Decl. (2nd Rebuttal) (Doc. 65-2) ¶12 (opining clear scientific consensus that athletic ability does not diverge significantly until puberty (citing e.g., David Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clinical Endocrinology 68, 70–71 (2017) ("The gender divergence in athletic performance begins at the age of 12–13 years"); Ps Motion for PI, Jonathon W. Senefeld et al., *Sex Differences in Youth Elite Swimming*, 14 PLOS ONE 1, 1–2 (2019) (Doc. 88-2) at 42-43 (studying child and youth swimmers and concluding that the data suggests "girls are faster, or at least not slower, than boys prior to the performance-enhancing effects of puberty"); M.J. McKay, *Normative reference values for strength and flexibility of 1000 children and adults* (Doc. 88-3) at 12 (finding no significant ($p<0.05$) differences between the strength measures of boys or girls aged 3-9, except for shoulder internal rotators where boys were stronger).

102. The World Rugby Transgender Women's Guidelines 2020 , which Dr. Brown cites throughout his declaration, allow transgender girls and women to participate in women's rugby if they did not experience endogenous male puberty, stating: "Transgender women who transitioned pre-puberty and have not experienced the biological effects of testosterone during puberty and adolescence can play women's rugby." (Pls.' Ex. 24 (Doc. 88-3); Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶ 35.)

103. The physical fitness data relied on by Defendant Horne and Intervenors merely observes phenomena across a population sample in isolated areas and does not determine a cause for what is observed. There is no basis for these experts to attribute those small

[7] The Court does not know whether Dr. Brown's opinion that hormone and testosterone suppression cannot fully eliminate physiological advantages once an individual experienced male puberty has been revised since the peer review of the Handelsman study. *See Hecox* 479 F. Supp. 3d at 980 (criticizing Brown's opinion because not updated subsequent to peer review and noting some of the studies Dr. Brown relied on "actually held the opposite").This evidence, relating to transgender girls/women who have experienced male puberty, is not directly relevant in this case, except to the extent the Court might extrapolate that if testosterone suppression in transgender females who have experienced male puberty, can bring them into athletic alignment with other girls/women, then preventing transgender girls from experiencing male puberty in the first place would result in even greater equity. The Court does not draw such a conclusion for purposes of deciding the request for preliminary injunction.

differences to physiology or anatomy instead of to other factors such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or differences in the preferences of the boys and girls surveyed. (Dr. Linda Blade ("Blade Decl.") at 7–9; Hilton Decl. (Doc. 92-8) ¶¶ 7.3–7.5; Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶¶ 21, 46.) The Court finds that transgender girls, who are being raised in conformance with their gender identity, will be subject to the same social/cultural factors that girls face that correlates to lower physical fitness scores.

104. There is no evidence to support Dr. Hilton's opinion that girls have "delicate brain structures" making them prone to injury; brain MRIs reveal no differences based on sex, except for size. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶ 40.) Evidence suggests the difference between male and female sports' concussions occurs because girls, post-puberty, have weaker neck muscles than boys. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶ 41 (citing Abigail C. Bretzin et al., Association of Sex with Adolescent Soccer Concussion Incidence and Characteristics, 4 JAMA Network Open 4, 6 (2021); Ryan T. Tierney et al., Gender Differences in Head-Neck Segment Dynamic Stabilization During Head Acceleration, 37 Med. & Sci. Sports & Exercise 272, 272 (2005)).

105. The Court rejects Dr. Hilton's idea that "sporty-girls" will be "as well-trained as their male peers" and, therefore, higher win scores at Kyrene Middle School for boys cannot be explained by social cultural factors and must be biological. (Hilton Decl. (Doc. 92-8) (citing Thomas and French, 1985, *Gender differences across age in motor performance a meta-analysis:* Psychol Bull 98(2): 260-282)).

106. Height differences in babies are negligible, with differences disappearing altogether between ages 6 and 8 but reappearing when girls enter puberty and overtake boys in height and weight for a few years until boys experience puberty and grow taller on average than girls/women. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶¶ 12-15.)

107. The Plaintiffs do not challenge the existence of separate teams for girls and boys. Defendants do not explain why the minor differences in physical fitness scores for prepuberty boys compared to girls reflect a significant athletic advantage of boys over girls,

prepuberty. There are many other reasons why boys' and girls' sports teams are separated: (1) women historically were deprived of athletic opportunities in favor of men; (2) as a general matter, men had equal athletic opportunities to women; and (3) according to stipulated facts, average physiological differences meant that "males would displace females to a substantial extent" if permitted to play on women's teams. *See Hecox*, 479 F. Supp. 3d at 976 (distinguishing *Clark by and Through Clark v. Arizona Interscholastic Ass'n*, 695 F.2d 1126 (9th Cir. 1982) finding these factors do not apply for transgender women).

108. Defendants ask the Court to rely on evidence they allege supports separating sports teams by sex to conclude that transgender girls, who have not experienced puberty, should not play on girls' teams solely because they are boys, regardless of whether they have experienced puberty.

109. The Court will not make this leap because Plaintiffs present expert evidence that any prepubertal differences between boys and girls in various athletic measurements are minimal or nonexistent. (Shumer Decl. (Rebuttal) (Doc. 65-2) ¶ 5) (citing Alison McManus & Neil Armstrong, *Physiology of elite young female athletes*, 56 Medicine & Science Sports & Exercise 23, 24 (2011) ("Prior to 11 years of age differences in average speed are minimal"); *id.* at 27 ("[S]mall sex difference in fat mass and percent body fat are evident from mid-childhood"); *id.* at 29 ("[B]one characteristics differ little between boys and girls prior to puberty"); *id.* at 32 ("There is little evidence that prior to puberty pulmonary structure or function limits oxygen uptake"); *id.* at 34 ("[N]o sex differences in arterial compliance have been noted in pre- and early- pubertal children")).

110. Based on the evidence, transgender girls' physical characteristics, especially in terms of height, weight, and strength, overlap with those of other girls. In other words, some girls may be taller than average, and some transgender girls may be taller than average. The rationale for excluding transgender girls with above average physical characteristics is equally applicable to excluding taller than average girls, but height, weight, or strength factors are not used at any level of competition to protect girls or women

athletes. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶¶ 42-43; *see also Hecox*, 479 F. Supp. 3d at 980 (describing evidence of similar bell curve differences for transgender women, who have gone through male puberty and are using gender affirming interventions, including lowering testosterone as "a transgender woman who performed 80% as well as the best performer among men of that age before transition would also perform at about 80% as well as the best performer among women of that age after transition.")

111. The categorical preclusion of transgender women, especially girls who have not experienced male puberty, appears unrelated to the interests the Act purportedly advances. A "justification must be genuine, not hypothesized." *United States v. Virginia,* 518 U.S. 515, 533 (1996). The proponents of the Act fail to provide persuasive evidence of any genuine, not hypothesized problem. *Hecox*, 479 F. Supp. 3d at 979.

112. Before puberty, there are no significant differences in athletic performance between boys and girls. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶ 16; Shumer Decl. (Rebuttal) (Doc. 65-2) ¶¶ 9–13; Shumer Decl. (Doc. 5) ¶ 38; Pls.' Exs. 19–20, 22–23 (Doc. 88-2).)

113. After puberty, adolescent boys begin to produce higher levels of testosterone, which over time causes them to become, on average, stronger and faster than adolescent girls. (Shumer Decl. (Doc. 5) ¶ 39; Pls.' Exs. 18–19 (Doc. 88-2).)

114. The biological driver of average group differences in athletic performance between adolescent boys and girls is the difference in their respective levels of testosterone, which only begin to diverge significantly after the onset of puberty. (Shumer Decl. (Rebuttal) ¶¶ 4, 8; Shumer Decl. (Doc. 5) ¶ 39; Pls.' Exs. 18–19.)

115. Transgender girls who receive puberty-blocking medication do not have an athletic advantage over other girls because they do not undergo male puberty and do not experience the physiological changes caused by the increased production of testosterone associated with male puberty. (Shumer Decl. (Rebuttal) (Doc. 65-2) ¶¶ 15–16; Shumer Decl. (Doc. 5) ¶¶ 35, 38–42.)

116. Transgender girls who receive hormone therapy after receiving puberty-

blocking medication will develop the skeletal structure, fat distribution, and muscle and breast development typical of other girls. (Budge Decl. (Doc. 4) ¶ 29; Shumer Decl. (Rebuttal) (Doc. 65-2) ¶ 22; Shumer Decl. (Doc. 5) ¶¶ 35–36.)

117. A transgender girl who receives hormone therapy will typically have the same levels of circulating estrogen and testosterone as other girls. (Shumer Decl. (Doc. 5) ¶ 36.)

118. Knowing that a girl is transgender, if she has not gone through male puberty, reveals nothing about her athletic ability. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶ 31, 48; Shumer Decl. (Rebuttal) (Doc. 65-2) ¶¶ 26–27; Shumer Decl. (Doc. 5) ¶ 42.)

119. Similarly, transgender girls who have not yet undergone male puberty or who have received puberty-blocking medication at the onset of puberty do not present any unique safety risk to other girls. (Shumer Decl. (2nd Rebuttal) (Doc. 113) ¶¶ 25, 36; Shumer Decl. (Rebuttal) ¶ 41.)

120. In short, transgender girls, who have not experienced male puberty, play like girls. There is no logical connection between prohibiting them from playing on girls' sports teams and the goals of preventing unfair competition in girls' sports or protecting girls from being physically injured by boys.

G. Plaintiffs cannot play on boys' sports teams.

121. Jane cannot play on boys' teams or compete with the boys because she is a girl, with athletic capabilities like other girls her age and different from boys her age who are beginning to experience puberty and increased testosterone levels. Jane will not experience male puberty and will experience female puberty. Assuming there are safety issues created if girls compete with boys, Jane would be subjected to such risks by playing on boys' teams.

122. Jane's medical health depends on her ability to live her life fully as a girl, and playing on a boys' sports team and competing against boys would directly contradict her medical treatment for gender dysphoria and jeopardize her health. (H. Doe Decl. (Doc. 7) ¶ 15; Budge Decl. (Doc. 4) ¶¶ 33–34.)

123. "Participating in sports on teams that contradict one's gender identity 'is

equivalent to gender identity conversion efforts, which every major medical association has found to be dangerous and unethical.'" *Hecox*, 479 F. Supp. 3d at 977.

124. Jane would find it humiliating and embarrassing to play on a boys' team because everyone at school knows her as a girl. (J. Doe Decl. (Doc. 6) ¶ 11; H. Doe Decl. (Doc. 7) ¶ 15.)

125. If she is not allowed to play sports on a girls' team, Jane will be very upset. (J. Doe Decl. (Doc. 6) ¶ 10; H. Doe Decl. (Doc. 7) ¶ 16.)

126. Jane will not participate in sports at all if she is forced to be on a boys' team. (J. Doe Decl. (Doc. 6) ¶ 11; H. Doe Decl. (Doc. 7) ¶ 15.) The last thing she wants to do is draw attention to herself by drawing into question her gender identity. She wants to go to school like other girls. (Jane Decl. (Doc. 6) ¶ 11.)

127. Jane will also lose the opportunity to receive the physical, social, and emotional benefits that school sports provide. (H. Doe Decl. (Doc. 7) ¶ 16).

128. Megan cannot play on boys' teams or compete with the boys because she is a girl, with athletic capabilities like other girls her age and different from boys her age, who have experienced puberty and increased testosterone levels. Megan has not experienced male puberty and has experienced female puberty. Assuming there are safety issues created if girls compete with boys, Jane would be subjected to such risks by playing on boys' teams.

129. Playing on a boys' team would directly conflict with Megan's medical treatment for gender dysphoria, and her medical health depends on her ability to live her life fully as a girl. Playing on a boys' team would be emotionally painful and humiliating for her. (M. Roe Decl. (Doc. 8) ¶ 9; K. Roe Decl. (Doc. 9) ¶ 10.)

130. "Participating in sports on teams that contradict one's gender identity 'is equivalent to gender identity conversion efforts, which every major medical association has found to be dangerous and unethical.'" *Hecox*, 479 F. Supp. 3d at 977.

131. If she is not allowed to play on the girls' volleyball team, Megan will not compete on the boys' volleyball team. (M. Roe Decl. (Doc. 8) ¶ 9; K. Roe Decl. (Doc. 9)

¶ 10.)

132. Megan will be distraught if she loses the opportunity to try out for the girls' volleyball team. (K. Roe Decl. (Doc. 9) ¶ 11.)

133. Megan will also lose the opportunity to receive the physical, social, and emotional benefits that school sports provide. (*Id.* ¶ 9.)

II. Conclusions of Law

To the extent these Conclusions of Law are also deemed to be Findings of Fact, they are hereby incorporated into the preceding Findings of Fact.

134. A preliminary injunction is an "extraordinary and drastic remedy" that is "never awarded as of right." *Munaf v. Geren*, 553 U.S. 674, 689-90 (2008) (citations omitted). Instead, in every case, the court must balance competing claims of injury and must consider the effect on each party of granting or withholding relief. *Winter v. Natural Resources Defense Council, Inc.*, 555 U.S. 7 (2008).

135. A preliminary injunction may take one of two forms: 1) a prohibitory injunction prohibits a party from taking action and "preserve[s] the status quo pending a determination of the action on the merits." *Chalk v. United States Dist. Court,* 840 F.2d 701, 704 (9th Cir. 1988). A mandatory injunction goes beyond simply maintaining the status quo and requires a heightened burden of proof and is particularly disfavored. *Marlyn Nutraceuticals, Inc. v. Mucos Pharma GmbH & Co.*, 571 F.3d 873, 879 (9th Cir. 2009) (citing *Anderson v. United States*, 612 F.2d 1112, 1114 (9th Cir. 1980)).

136. "Status quo" for the purpose of an injunction "refers to the legally relevant relationship between the parties before the controversy arose." *Arizona Dream Act Coal. v. Brewer*, 757 F.3d 1053, 1061 (9th Cir. 2014) (emphasis in original); *see also Regents of Univ. of California v. Am. Broad. Companies, Inc.*, 747 F.2d 511, 514 (9th Cir. 1984) (for purposes of injunctive relief, the status quo means "the last uncontested status which preceded the pending controversy") (cleaned up).

137. For the purpose of issuing a preliminary injunction, the Court's findings that both Jane and Megan could have played on girls' sports teams last year prior to passage of

the Act, cannot play on sports teams consistent with their gender identity now, and want to participate in girls' sports programs at Kyrene Middle School and TGS this year, warrant issuance of a mandatory prohibitory injunction to preserve the status quo.

138. The purpose of a preliminary injunction or temporary restraining order is to preserve the status quo if the balance of equities so heavily favors the moving party that justice requires the court to intervene to secure the positions until the merits of the action are ultimately determined. *University of Texas v. Camenisch*, 451 U.S. 390, 395 (1981).

139. A party seeking a preliminary injunction must establish that: (1) they are likely to succeed on the merits of their claims; (2) they are likely to suffer irreparable harm in the absence of preliminary relief; (3) the balance of equities tips in their favor; and (4) an injunction is in the public interest. *Alliance for the Wild Rockies v. Cottrell*, 632 F.3d 1127, 1131 (9th Cir. 2011).

140. When the government is a party, the third and fourth factors merge. *Nken v. Holder*, 556 U.S. 418, 435 (2009); *Porretti v. Dzurenda*, 11 F.4th 1037, 1050 (9th Cir. 2021).

A. <u>Likelihood of success on the merits.</u>

Equal Protection Clause Claim

141. There is a strong presumption that gender classifications are invalid and the burden rests on the state to justify the classification. *Virginia*, 518 U.S. at 533. This burden tracks for purposes of considering the likelihood of the merits of the Plaintiffs' claim. Defendants must show that it is "more likely than not" that the Act is constitutional. *Gonzales v. O Centro Espirita Beneficente Uniao de Vegetal*, 546 U.S. 418, 429–30 (2006) (finding evidentiary equipoise insufficient and issuing a preliminary injunction).

142. The Supreme Court has addressed the Defendants' concern that legislation must be written for the population generally, therefore, "most legislation classifies for one purpose or another, with resulting disadvantage to various groups or persons." *Hecox*, 479 F. Supp. 3d at 972); (Preliminary Injunction, Oral Argument: July 10, 2023). There are

three tiers of judicial scrutiny depending on the characteristics of the disadvantaged group or the rights implicated by the classification. *Hecox*, 479 F. Supp. 3d at 972.

143. When the state restricts an individual's access to a fundamental right, the policy must withstand the strictest of scrutiny. *San Antonio Indep. Sch. District. v. Rodriguez*, 411 U.S. 1, 16-17 (1973). Access to interscholastic sports is not a constitutionally recognized fundamental right. *Walsh v. La High Sch. Athletic Ass'n*, 616 F.2d 152, 159-60 (5th Cir. 1980). Strict scrutiny also applies if a government policy discriminates against a suspect class such as race, alienage, and national origin because government policies that discriminate based on race or national origin typically reflect prejudice. *City of Cleburn v. Cleburn Living Center*, 473 U.S. 432, 440 (1985).

144. The least stringent level of scrutiny is rational basis review, which is applied to laws that impose a difference in treatment between groups but do not infringe upon a fundamental right or target a suspect or quasi-suspect class. *Heller v. Dow,* 509 U.S. 312, 319-321 (1993).

145. Heightened scrutiny is an intermediate scrutiny, a slightly less stringent standard than strict scrutiny, but greater than rational basis review. *Craig v. Boren*, 429 U.S. 190, 197 (1976); *Virginia*, 518 U.S. at 533. Heightened scrutiny applies to statutes that discriminate on the basis of sex, a quasi-suspect classification. "'The purpose of this heightened level of scrutiny is to ensure quasi-suspect classifications do not perpetuate unfounded stereotypes or second-class treatment.'" *Hecox*, 479 F. Supp. 3d at 973 (quoting *Latta v. Otter* (*Latta I*), 19 F. Supp. 3d 1054, 1073 (D. Idaho), *aff'd*, 771 F.3d 456 (9th Cir. 2014) (citing *Virginia*, 518 U.S. at 533)). To withstand heightened scrutiny, a classification by sex "must serve important governmental objectives and must be substantially related to achievement of those objectives." *Craig*, 429 U.S. at 197.

146. Laws that discriminate against transgender people are sex-based classifications and, as such, warrant heightened scrutiny. *See Karnoski v. Trump*, 926 F.3d 1180, 1200–01 (9th Cir. 2019) (analyzing a policy barring transgender people from military service as sex-based discrimination and applying heightened scrutiny); *see also D.T. v. Christ*, 552 F.

Supp. 3d 888, 896 (D. Ariz. 2021) ("Discrimination against transgender people is discrimination based on sex; as such, heightened scrutiny applies.").

147. Defendant Horne's and Intervenors' argument that the Act does not mention transgender girls and, therefore, does not discriminate based on transgender status or gender identity fails. The Act's disparate treatment of transgender girls because they are transgender is clear on the face of the statute and makes it facially discriminatory even if the statute does not expressly employ the term "transgender". *See e.g. Hecox*, 479 F. Supp. 3d at 975 (rejecting defendants' argument that similar Idaho statute "does not ban athletes on the basis of transgender status, but rather on the basis of the innate physiological advantages males generally have over females"); *A.M.*, 617 F. Supp. 3d at 965–66 (holding that a virtually identical Indiana statute discriminated against transgender individuals despite not using the term "transgender"); *B.P.J. v. W. Va. State Bd. of Educ.*, 550 F. Supp. 3d 347, 353–54 (S.D. W. Va. 2021) (holding that a virtually identical West Virginia statute "discriminates on the basis of transgender status"), *B. P. J. v. W. Virginia State Bd. Of Educ.,* No. 2:21-CV-00316, 2023 WL 111875, at *6 (S.D.W. Va. Jan. 5, 2023) (cleaned up), *stayed pending appeal B.P.J. v. W. Virginia State Bd. of Educ.*, No. 23-1078, 2023 WL 2803113, at *1 (4th Cir. Feb. 22, 2023).

148. The Arizona legislature intentionally created a classification, specifically "biological girls," that necessarily excludes transgender girls, and expressly allowed only that exclusive classification to play girls sports to the exclusion of transgender girls.

149. The legislative history demonstrates that the purpose of the Act is to exclude transgender girls from girls' sports teams. Therefore, the Court applies heightened scrutiny to the Act, does not make a presumption of constitutionality, and does not defer to legislative judgment. *SmithKline Beecham Corp. v. Abbott Laboratories,* 740 F.3d 471, 483 (9th Cir. 2014).

150. Plaintiffs Jane and Megan are transgender girls, members of a quasi-protected class. The Court applies heightened scrutiny in this case, placing the burden on the government to show "an exceedingly persuasive justification" for the alleged

discriminatory treatment, *Virginia*, 518 U.S. at 531, which must not be based on "generalizations" or "stereotypes," *id.* at 549–50, 565. "The justification 'must be genuine, not hypothesized or invented post hoc in response to litigation,' and 'must not rely on overbroad generalizations about the different talents, capacities, or preferences of males and females." *Karnoski*, 926 F.3d at 1200 (quoting *Virginia*, 518 U.S. at 533).

151. In applying heightened scrutiny review, the Court must examine the Act's "'actual purposes and carefully consider any resulting inequality to ensure that our most fundamental institutions neither send nor reinforce messages of stigma or second-class status.'" *Latta II,* 771 F.3d at 468 (quoting *SmithKline,* 740 F.3d at 483).

152. According to Defendants, the Act is to protect girls from physical injury in sports and promote equality and equity in athletic opportunities, which are, in addition to redressing past discrimination against women in athletics, considered legitimate and important governmental interests justifying rules excluding males from participating on female teams. *Clark,* 695 F.2d at 1131.

153. However, the well-established scientific consensus is that, before puberty, there are no significant physiological differences in athletic performance between boys and girls. Instead, there is overlap between the sexes, with some boys being better athletically than some girls and some girls outplaying some boys. There is also no evidence that transgender girls who do not undergo male puberty because they have taken puberty suppressing medication at the onset of male puberty have an athletic advantage over other girls. There are no studies that have documented any such advantage, and there is no medical reason to posit that any such advantage would exist. (*Id*. ¶ 26.)

154. The testimony by Drs. Brown and Hilton that boys have some biological advantages related to physical fitness before puberty does not support a conclusion that Plaintiffs, who have not experienced male puberty, have any athletic advantage over other girls or pose a safety risk to other girls by playing on girls' sports teams.

155. Defendant Horne and Intervenors discuss *Clark,* 695 F.2d at 1131, throughout their briefs but *Clark* strongly supports Plaintiffs. In *Clark*, the Ninth Circuit held that it

was lawful to exclude boys from girls' volleyball teams because: (1) women had historically been deprived of athletic opportunities in favor of men; (2) as a general matter, men had equal athletic opportunities compared to women; and (3) according to the stipulated facts in the case, average physiological differences meant that males would displace females to a substantial extent if permitted to play on women's volleyball teams. *Hecox*, 479 F.Supp. 3d at 1131.

156. None of the *Clark* premises hold true for girls who are transgender: (1) far from being favored in athletics, "women who are transgender have historically been discriminated against;" (2) transgender women—unlike the boys in Clark—would not be able to participate in any school sports; and (3) based on the very small numbers of transgender girls in the population, "transgender women have not and could not 'displace' cisgender women in athletics 'to a substantial extent.'" *Hecox*, 479 F. Supp. 3d at 977 (quoting *Clark*, 695 F.2d at 1131). Hecox's analysis of *Clark* is more compelling here, where Plaintiffs have not experienced male puberty and will experience female puberty. *See Hecox*, 479 F. Supp. 3d at 981 (transgender girls who do not experience male puberty "do not have an ascertainable advantage over cisgender female athletes").

157. Under *Clark,* the legislature need not pick the wisest alternative for addressing a problem, but it must show that the policy is "substantially related to the goals of providing fair and equal playing opportunities for girls and protections to ensure the safety of girls playing sports. *Clark,* 695 F.2d at 1132.

158. The Court finds that Defendant Horne and Intervenors fail to produce persuasive evidence at the preliminary injunction stage to show that the Act is substantially related to the legitimate goals of ensuring equal opportunities for girls to play sports and to prevent safety risks:

- There is no evidence in the record that transgender girls who have not experienced male puberty, have presented an actual problem of unfair competition or created safety risks to other girls.
- There is no empirical evidence in the record that transgender girls who have not experienced puberty, have any physiological advantages over other girls that create unfair competition for positions on girls' sports teams and other athletic opportunities, or pose a safety risk to other girls.

- The Act is overly broad, reaching sports at all grade levels, including grades when athletes are prepuberty; it bans transgender girls, who have not experienced male puberty and who, instead, will or have experienced female puberty. "The Supreme Court has long viewed with suspicion laws that rely on overbroad generalizations about the different talents, capacities, or preferences of males and females." *B. P. J.* 2023 WL 111875, at *6. Laws that discriminate based on sex must be backed by an "exceedingly persuasive justification." *Virginia,* 518 U.S. at 531.

- The Act treats transgender boys and transgender girls and boys' and girls' sports differently. Transgender boys who, according to Defendants' reasoning and classifications are "biological girls", are allowed to play on boys' sports teams, subject to the alleged risks of that association which the Act proports to address. The Act creates a private cause of action that burdens only girls' sports programs with transgender challenges, investigations, and litigation. The Act subjects only female athletes, transgender and otherwise, to gender challenges and investigations. Boys playing on boys' teams do not have to worry about any gender challenge or investigation.

159. Defendant Horne and Intervenors have not established that categorically banning all transgender girls from playing girls' sports is substantially related to an important government interest. *Virginia*, 518 U.S. at 524.

160. Defendant Horne's and Intervenors' argument that the Act is necessary to protect girls' sports by barring transgender girls, who purportedly have an unfair athletic advantage over other girls and/or pose a safety risk to other girls, is based on overbroad generalizations and stereotypes that erroneously equate transgender status with athletic ability. *See Hecox*, 479 F. Supp. 3d at 982 (holding that the asserted advantage between transgender and non-transgender female athletes "is based on overbroad generalizations without factual justification"). Therefore, the Act does not withstand heightened scrutiny. *Karnoski*, 926 F.3d at 1200 (citing *Virginia*, 518 U.S. at 533).

161. Because the Court's findings of fact reflect that the Act's categorical bar against transgender girls' participation on girls' sports teams is not a genuine justification, the Plaintiffs are likely to prevail on the merits. Heightened scrutiny requires more than a hypothesized problem. *Virginia,* 518 U.S. at 533.

162. In fact, the Act fails even under the rational basis test because it is not related to any important government interest. "[I]f the constitutional conception of 'equal protection of the laws' means anything, it must at the very least mean that a bare

congressional desire to harm a politically unpopular group cannot constitute a legitimate governmental interest." *United States Dep't of Agric. v. Moreno*, 413 U.S. 528, 534 (1973).

Title IX Claim

163. Title IX provides, in relevant part, that no person "shall, on the basis of sex, be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any education program or activity receiving Federal financial assistance[.]" 20 U.S.C. § 1681(a).

164. Defendants Kyrene School District (administered and overseen by Defendant Toenjes) and the AIA receive federal financial assistance, and Defendant Horne is a grant recipient of federal funds. All Defendants must comply with Title IX's requirements. (See Compl. ¶¶ 9–13.)[8]

165. Discriminating against an individual on the basis of transgender status is discrimination based on sex. *See Bostock v. Clayton Cnty*., 140 S. Ct. 1731, 1741 (2020) ("[I]t is impossible to discriminate against a person for being . . . transgender without discriminating against that individual based on sex.").

166. The Ninth Circuit has held that discrimination based on transgender status also constitutes impermissible discrimination under Title IX. *See Grabowski v. Ariz. Bd. of Regents*, 69 F.4th 1110, 1116 (9th Cir. 2023) (holding that *Bostock* Title VII case applies to Title IX); *Doe v. Snyder*, 28 F.4th 103, 114 (9th Cir. 2022).

167. The Act discriminates against Plaintiffs based on their status as transgender girls by providing that for purposes of school sports a student's sex is fixed "at birth." S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2002), § 2.

168. The Act's classification of all transgender girls as male and its prohibition of students who are "male" from playing on girls' teams, Ariz. Stat. § 15-120.02(B), intentionally excludes all transgender girls, including Plaintiffs, from participating on girls' teams.

[8] TGS has filed a motion to dismiss on the basis that it does not receive federal financial assistance and therefore is not required to comply with Title IX requirements. The Court will address this motion by separate order.

169. Exclusion from athletics on the basis of sex is a cognizable harm under Title IX because it deprives Plaintiffs of the benefits of sports programs and activities that their non-transgender classmates enjoy. *See Grabowski*, 69 F.4th 1121–22 (holding that being removed from the team was an adverse action under Title IX); *see also A.M. by E.M. v. Indianapolis Pub. Sch.*, 617 F. Supp. 3d 950 (S.D. Ind. 2022), appeal dismissed sub nom. *A.M. by E.M. v. Indianapolis Pub. Sch. & Superintendent*, No. 22-2332, 2023 WL 371646 (7th Cir. Jan. 19, 2023) (granting a preliminary injunction of a similar Indiana law that banned transgender girls from playing on girls' sports teams based on Title IX).

170. The Court rejects Defendant Horne's and Intervenors' arguments that Plaintiffs' schools offer teams for both boys and girls and, therefore, Plaintiffs are not excluded from participating in sports on teams consistent with their "biological sex." The Court's findings of fact reflect that Plaintiffs, who are transgender girls, cannot play on boys' teams because they are transgender girls who have not and will not go through male puberty and will go through female puberty. Moreover, playing on a boys' team would be shameful and humiliating for Plaintiffs as well as in direct conflict with ongoing treatment for gender dysphoria, a serious medical condition.

B. Plaintiffs Will Suffer Irreparable Harm if Relief Is Not Granted.

171. Plaintiffs face irreparable harm if this Court does not enjoin the Act as to them.

172. Enforcement of the Act in violation of the Equal Protection Clause in and of itself is sufficient to presume irreparable harm to justify a preliminary injunction. *Hernandez v. Sessions*, 872 F.3d 976, 994–95 (9th Cir. 2017) ("It is well established that the deprivation of constitutional rights unquestionably constitutes irreparable injury.") (internal quotation marks and citation omitted); *Hecox,* 479 F. Supp. 3d at 987 (noting this "dispositive presumption").

173. A violation of Title IX also causes irreparable harm. *See Anders v. Cal. State Univ., Fresno*, 2021 WL 1564448, at *18 (E.D. Cal. Apr. 21, 2021) (finding irreparable harm under Title IX given the "presumption of irreparable injury where plaintiff shows violation of a civil rights statute" and in light of "the insult that comes from unequal

treatment"); *Portz v. St. Cloud State Univ.*, 196 F. Supp. 3d 963, 973 (D. Minn. 2016) ("Plaintiffs have a fair chance of succeeding on their Title IX claim, and Congress passed Title IX pursuant to its power to enforce the Fourteenth Amendment. Plaintiffs' expectation that they may be treated unequally in violation of Title IX's terms is an irreparable harm.") (cleaned up).

174. Plaintiffs will also suffer severe and irreparable mental, physical, and emotional harm if the Act applies to them because they cannot play on boys' sports teams. Playing on a boys' team would directly contradict Plaintiffs' medical treatment for gender dysphoria and would be painful and humiliating. Plaintiffs' mental health is dependent on living as girls in all aspects of their lives.

175. Enforcing the Act against Plaintiffs will effectively exclude Plaintiffs from school sports and deprive them of the social, educational, physical, and emotional health benefits that both sides acknowledge come from school sports. This exclusion is a cognizable harm. *Grabowski*, 69 F.4th at 1121.

176. Plaintiffs will also suffer the shame and humiliation of being unable to participate in a school activity simply because they are transgender—a personal characteristic over which they have no control. *Grimm v. Gloucester Cnty. Sch. Bd.*, 972 F.3d 586, 625 (4th Cir. 2020) (explaining that the stigma of exclusion "publicly brand[s] all transgender students with a scarlet 'T'") (internal quotation marks and citation omitted).

177. In addition, Plaintiffs will suffer the cognizable and irreparable "dignitary wounds" associated with the passage of a law expressly designed to communicate the state's moral disapproval of their identity, wounds that "cannot always be healed with the stroke of a pen." *Obergefell v. Hodges*, 576 U.S. 644, 678 (2015); *Hecox,* 479 F. Supp. 3d at 987 (finding such wounds constitute irreparable harm).

178. Plaintiffs have established that they will suffer irreparable harm if the Act is enforced against them.

C. <u>The Public Interest and Balance of Equities Favor Injunctive Relief.</u>

179. When an injunction is sought against a governmental entity, the public interest

and balance-of-the-hardships factors merge. *Nken*, 556 U.S. at 435–36.

180. As an initial matter, "it is always in the public interest to prevent the violation of a party's constitutional rights." *Melendres v. Arpaio*, 695 F.3d 990, 1002 (9th Cir. 2012).

181. The balance of equities favors Plaintiffs as well. Defendant Horne and Intervenors "cannot suffer harm from an injunction that merely ends an unlawful practice." *Rodriguez v. Robbins*, 715 F.3d 1127, 1145 (9th Cir. 2013). Plaintiffs, however, face serious and ongoing harm if the Act is enforced against them.

182. The alleged harm to Defendants and Intervenors—"that biological girls will be forced to compete against transgender girls who allegedly have an athletic advantage"—is unsupported by the record. *A.M.,* 617 F. Supp. 3d at 968. Moreover, there is no evidence in the record "that allowing [Plaintiffs] to play on the girls' [teams] will make this [purported] harm a reality." *Id.* On the contrary, the record suggests the opposite. Based on the record for the preliminary injunction, the Court has found that Plaintiffs do not have a competitive advantage over other girls, and they do not pose a safety risk.

183. But for the Act, Defendants TGS, Kyrene School District, Superintendent Toenjes, and the AIA would all permit Plaintiffs to play on girls' teams.

184. There is no evidence that any Defendant will be harmed by allowing Plaintiffs to continue playing with their peers as they have done until now. *Hecox*, 479 F. Supp. 3d at 988 ("[A] preliminary injunction would not harm Defendants because it would merely maintain the status quo while Plaintiffs pursue their claims.").

185. Accordingly, the public interest and balance of equities favor a preliminary injunction.

CONCLUSION

The Court's findings of fact support Plaintiffs' assertions that very serious damages will result from a change in the status quo, and as a matter of law and fact, this is not a doubtful case. *See Anderson*, 612 F.2d at 1114 (generally, mandatory injunctions require extreme or very serious damage and not issued in doubtful cases). Because Plaintiffs have

satisfied all elements necessary to obtain a preliminary injunction, the Court grants Plaintiffs' motion for a preliminary injunction.

The Court has the discretion to determine whether the moving party is required to post a bond as a condition for the granting of a preliminary injunction. *Diaz v. Brewer*, 656 F.3d 1008, 1015 (9th Cir. 2011) (citing *Johnson v. Couturier*, 572 F.3d 1067, 1086 (9th Cir. 2009)). Here, a bond is not required because "there is no realistic likelihood of harm to the defendant from enjoining his or her conduct." *Jorgensen v. Cassiday*, 320 F.3d 906, 919 (9th Cir. 2003).

Accordingly,

IT IS ORDERED that the Motion for Preliminary Injunction (Doc. 3) is GRANTED.

IT IS FURTHER ORDERED that Defendant Horne is enjoined from enforcing A.R.S. § 15-120.02 as to Plaintiffs.

IT IS FURTHER ORDERED that the Act shall not prevent Plaintiffs from participating in girls' sports and, as agreed by Kyrene School District and Laura Toenjes, in her official capacity, pursuant to the Stipulation in Lieu of an Answer (Doc. 59), and by TGS in open Court at the hearing for the Preliminary Injunction, the Plaintiffs shall be allowed to play girls' sports at their respective schools.

IT IS FURTHER ORDERED that the AIA transgender policy, § 41.9, complies with the terms of this preliminary injunction.

Dated this 20th day of July, 2023.

Honorable Jennifer G. Zipps
United States District Judge

EXHIBIT 15

WILENCHIK & BARTNESS
A PROFESSIONAL CORPORATION

Senate Engrossed

interscholastic; intramural athletics; biological sex

State of Arizona
Senate
Fifty-fifth Legislature
Second Regular Session
2022

SENATE BILL 1165

AN ACT

AMENDING TITLE 15, CHAPTER 1, ARTICLE 1, ARIZONA REVISED STATUTES, BY ADDING SECTION 15-120.02; RELATING TO ATHLETICS.

(TEXT OF BILL BEGINS ON NEXT PAGE)

Be it enacted by the Legislature of the State of Arizona:

Section 1. Title 15, chapter 1, article 1, Arizona Revised Statutes, is amended by adding section 15-120.02, to read:

15-120.02. Interscholastic and intramural athletics; designation of teams; biological sex; cause of action; definition

A. EACH INTERSCHOLASTIC OR INTRAMURAL ATHLETIC TEAM OR SPORT THAT IS SPONSORED BY A PUBLIC SCHOOL OR A PRIVATE SCHOOL WHOSE STUDENTS OR TEAMS COMPETE AGAINST A PUBLIC SCHOOL SHALL BE EXPRESSLY DESIGNATED AS ONE OF THE FOLLOWING BASED ON THE BIOLOGICAL SEX OF THE STUDENTS WHO PARTICIPATE ON THE TEAM OR IN THE SPORT:

1. "MALES", "MEN" OR "BOYS".

2. "FEMALES", "WOMEN" OR "GIRLS".

3. "COED" OR "MIXED".

B. ATHLETIC TEAMS OR SPORTS DESIGNATED FOR "FEMALES", "WOMEN" OR "GIRLS" MAY NOT BE OPEN TO STUDENTS OF THE MALE SEX.

C. THIS SECTION DOES NOT RESTRICT THE ELIGIBILITY OF ANY STUDENT TO PARTICIPATE IN ANY INTERSCHOLASTIC OR INTRAMURAL ATHLETIC TEAM OR SPORT DESIGNATED AS BEING FOR "MALES", "MEN" OR "BOYS" OR DESIGNATED AS "COED" OR "MIXED".

D. A GOVERNMENT ENTITY, ANY LICENSING OR ACCREDITING ORGANIZATION OR ANY ATHLETIC ASSOCIATION OR ORGANIZATION MAY NOT ENTERTAIN A COMPLAINT, OPEN AN INVESTIGATION OR TAKE ANY OTHER ADVERSE ACTION AGAINST A SCHOOL FOR MAINTAINING SEPARATE INTERSCHOLASTIC OR INTRAMURAL ATHLETIC TEAMS OR SPORTS FOR STUDENTS OF THE FEMALE SEX.

E. ANY STUDENT WHO IS DEPRIVED OF AN ATHLETIC OPPORTUNITY OR SUFFERS ANY DIRECT OR INDIRECT HARM AS A RESULT OF A SCHOOL KNOWINGLY VIOLATING THIS SECTION HAS A PRIVATE CAUSE OF ACTION FOR INJUNCTIVE RELIEF, DAMAGES AND ANY OTHER RELIEF AVAILABLE UNDER LAW AGAINST THE SCHOOL.

F. ANY STUDENT WHO IS SUBJECT TO RETALIATION OR ANOTHER ADVERSE ACTION BY A SCHOOL OR AN ATHLETIC ASSOCIATION OR ORGANIZATION AS A RESULT OF REPORTING A VIOLATION OF THIS SECTION TO AN EMPLOYEE OR REPRESENTATIVE OF THE SCHOOL OR THE ATHLETIC ASSOCIATION OR ORGANIZATION, OR TO ANY STATE OR FEDERAL AGENCY WITH OVERSIGHT OF SCHOOLS IN THIS STATE, HAS A PRIVATE CAUSE OF ACTION FOR INJUNCTIVE RELIEF, DAMAGES AND ANY OTHER RELIEF AVAILABLE UNDER LAW AGAINST THE SCHOOL OR THE ATHLETIC ASSOCIATION OR ORGANIZATION.

G. ANY SCHOOL THAT SUFFERS ANY DIRECT OR INDIRECT HARM AS A RESULT OF A VIOLATION OF THIS SECTION HAS A PRIVATE CAUSE OF ACTION FOR INJUNCTIVE RELIEF, DAMAGES AND ANY OTHER RELIEF AVAILABLE UNDER LAW AGAINST THE GOVERNMENT ENTITY, THE LICENSING OR ACCREDITING ORGANIZATION OR THE ATHLETIC ASSOCIATION OR ORGANIZATION.

H. ALL CIVIL ACTIONS MUST BE INITIATED WITHIN TWO YEARS AFTER THE ALLEGED VIOLATION OF THIS SECTION OCCURRED. A PERSON OR ORGANIZATION THAT

PREVAILS ON A CLAIM BROUGHT PURSUANT TO THIS SECTION IS ENTITLED TO MONETARY DAMAGES, INCLUDING DAMAGES FOR ANY PSYCHOLOGICAL, EMOTIONAL OR PHYSICAL HARM SUFFERED, REASONABLE ATTORNEY FEES AND COSTS AND ANY OTHER APPROPRIATE RELIEF.

I. FOR THE PURPOSES OF THIS SECTION, "SCHOOL" MEANS EITHER:

1. A SCHOOL THAT PROVIDES INSTRUCTION IN ANY COMBINATION OF KINDERGARTEN PROGRAMS OR GRADES ONE THROUGH TWELVE.

2. AN INSTITUTION OF HIGHER EDUCATION.

Sec. 2. Legislative findings and purpose

The legislature finds that:

1. "With respect to biological sex, one is either male or female." Arnold De Loof, Only Two Sex Forms but Multiple Gender Variants: How to Explain?, 11(1) COMMUNICATIVE & INTEGRATIVE BIOLOGY (2018), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5824932.

2. A person's "sex is determined at [fertilization] and revealed at birth or, increasingly, *in utero*." Lucy Griffin et al., Sex, gender and gender identity: a re-evaluation of the evidence, 45(5) BJPSYCH BULLETIN 291 (2021), https://www.cambridge.org/core/journals/bjpsych-bulletin/article/sex-gender-and-gender-identity-a-reevaluation-of-the-evidence/76A3DC54F3BD91E8D631B93397698B1A.

3. "[B]iological differences between males and females are determined genetically during embryonic development." Stefanie Eggers & Andrew Sinclair, Mammalian sex determination—insights from humans and mice, 20(1) CHROMOSOME RES. 215 (2012), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3279640.

4. "Secondary sex characteristics that develop during puberty . . . generate anatomical divergence beyond the reproductive system, leading to adult body types that are measurably different between sexes." Emma N. Hilton & Tommy R. Lundberg, Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage, 51 SPORTS MED. 199 (2021), https://doi.org/10.1007/s40279-020-01389-3.

5. There are "'[i]nherent differences' between men and women," and that these differences "remain cause for celebration, but not for denigration of the members of either sex or for artificial constraints on an individual's opportunity." United States v. Virginia, 518 U.S. 515, 533 (1996).

6. In studies of large cohorts of children from six years old, "[b]oys typically scored higher than girls on cardiovascular endurance, muscular strength, muscular endurance, and speed/agility, but lower on flexibility." Konstantinos Tambalis et al., Physical fitness normative values for 6-18-year-old Greek boys and girls, using the empirical distribution and the lambda, mu, and sigma statistical method, 16(6) EUR J. SPORT SCI. 736 (2016), https://pubmed.ncbi.nlm.nih.gov/26402318. See also, Mark J Catley & Grant R Tomkinson, Normative Health-related fitness values for children: analysis of 85347 test results on 9-17 year

old Australians since 1985, 47(2) BRIT. J. SPORTS MED. 98 (2013), https://pubmed.ncbi.nlm.nih.gov/22021354.

7. Physiological differences between males and females relevant to sports performance "include a larger body size with more skeletal-muscle mass, a lower percentage of body fat, and greater maximal delivery of anaerobic and aerobic energy." Øyvind Sandbakk et al., Sex Differences in World-Record Performance: The Influence of Sport Discipline and Competition Duration, 13(1) INT'L J. SPORTS PHYSIOLOGY & PERFORMANCE 2 (2018), https://pubmed.ncbi.nlm.nih.gov/28488921.

8. Men also have higher natural levels of testosterone, which affects traits such as hemoglobin levels, body fat content, the storage and use of carbohydrates, and the development of Type 2 muscle fibers, all of which result in men being able to generate higher speed and power during physical activity. Doriane Lambelet Coleman, Sex in Sport, 80 LAW & CONTEMP. PROBS. 63, 74 (2017) (quoting Gina Kolata, Men, Women and Speed. 2 Words: Got Testosterone?, N.Y. TIMES (Aug. 21, 2008).

9. There is a sports performance gap between males and females, such that "the physiological advantages conferred by biological sex appear, on assessment of performance data, insurmountable." Hilton, *supra* at 200.

10. While classifications based on sex are generally disfavored, the Supreme Court has recognized that "sex classifications may be used to compensate women for particular economic disabilities [they have] suffered, . . . to promote equal employment opportunity, . . . [and] to advance full development of the talent and capacities of our Nation's people." United States v. Virginia, 518 U.S. 515, 533 (1996) (internal citations and quotation marks omitted).

11. One place where sex classifications allow for the "full development of the talent and capacities of our Nation's people" is in the context of sports and athletics.

12. Courts have recognized that the inherent, physiological differences between males and females result in different athletic capabilities. See, e.g., Kleczek v. Rhode Island Interscholastic League, Inc., 612 A.2d 734, 738 (R.I. 1992) ("Because of innate physiological differences, boys and girls are not similarly situated as they enter athletic competition."); Petrie v. Ill. High Sch. Ass'n, 394 N.E.2d 855, 861 (Ill. App. Ct. 1979) (noting that "high school boys [generally possess physiological advantages over] their girl counterparts" and that those advantages give them an unfair lead over girls in some sports like "high school track").

13. The benefits that natural testosterone provides to male athletes is not diminished through the use of testosterone suppression. A recent study on the impact of such treatments found that policies like those of the International Olympic Committee requiring biological males to undergo at least one year of testosterone suppression before competing in

women's sports do not create a level playing field. "[T]he reduction in testosterone levels required by [policies like those of the International Olympic Committee] is insufficient to remove or reduce the male advantage, in terms of muscle mass and strength, by any meaningful degree." The study concluded that "[t]he data presented here demonstrate that superior anthropometric, muscle mass and strength parameters achieved by males at puberty, and underpinning a considerable portion of the male performance advantage over females, are not removed by the current regimen of testosterone suppression" permitted by the International Olympic Committee and other sports organizations. Rather, the study found that male performance advantage over females "remains substantial" and "raises obvious concerns about fair and safe competition." Hilton, *supra* at 207, 209.

14. Having separate sex-specific teams furthers efforts to promote sex equality by providing opportunities for female athletes to demonstrate their skill, strength and athletic abilities while also providing them with opportunities to obtain recognition, accolades, college scholarships and the numerous other long-term benefits that flow from success in athletic endeavors.

Sec. 3. Severability

If a provision of this act or its application to any person or circumstance is held invalid, the invalidity does not affect other provisions or applications of the act that can be given effect without the invalid provision or application, and to this end the provisions of this act are severable.

Sec. 4. Short title

This act may be cited as the "Save Women's Sports Act".

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IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, *et al.*, Plaintiffs, v. Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction, *et al.*, Defendants.	Case No. 4:23-cv-00185-JGZ **Declaration of Dr. Gregory A. Brown, Ph.D., FACSM, in Support of [Intervenors' Proposed] Opposition to Plaintiffs' Motion for a Preliminary Injunction**

TABLE OF CONTENTS

Personal Qualifications and Disclosure

I serve as Professor of Exercise Science in the Department of Kinesiology and Sport Sciences at the University of Nebraska Kearney, where I teach classes in Exercise Physiology among other topics. I am also the Director of the General Studies program. I have served as a tenured (and nontenured) professor at universities since 2002.

In August 2002, I received a Doctor of Philosophy degree from Iowa State University, where I majored in Health and Human Performance, with an emphasis in the Biological Bases of Physical Activity. In May 1999, I received a Master of Science degree from Iowa State University, where I majored in Exercise and Sport Science, with an emphasis in Exercise Physiology.

I have received many awards over the years, including the Mortar Board Faculty Excellence Honors Award, College of Education Outstanding Scholarship / Research Award, and the College of Education Award for Faculty Mentoring of Undergraduate Student Research. I have authored more than 50 refereed publications and more than 70 refereed presentations in the field of Exercise Science. I have authored chapters for multiple books in the field of Exercise Science. And I have served as a peer reviewer for over 30 professional journals, including *The American Journal of Physiology*, the *International Journal of Exercise Science*, the *Journal of Strength and Conditioning Research*, *Therapeutic Advances in Endocrinology and Metabolism, Sports Medicine*, and *The Journal of Applied Physiology*.

My areas of research have included the endocrine response to testosterone prohormone supplements in men and women, the effects of testosterone prohormone supplements on health and the adaptations to strength training in men, the effects of energy drinks on the physiological response to exercise, assessment of various athletic training modes in males and females, and sex-based differences in athletic performance. Articles that I have published that are closely related to topics that I discuss in this expert report include:

- Studies of the effect of ingestion of a testosterone precursor on circulating

testosterone levels in young men. Douglas S. King, Rick L. Sharp, Matthew D. Vukovich, Gregory A. Brown, et al., *Effect of Oral Androstenedione on Serum Testosterone and Adaptations to Resistance Training in Young Men: A Randomized Controlled Trial*, JAMA 281: 2020-2028 (1999); G. A. Brown, M. A. Vukovich, et al., *Effects of Anabolic Precursors on Serum Testosterone Concentrations and Adaptations to Resistance Training in Young Men*, Int J Sport Nutr Exerc Metab 10: 340-359 (2000).

- A study of the effect of ingestion of that same testosterone precursor on circulating testosterone levels in young women. G. A. Brown, J. C. Dewey, et al., *Changes in Serum Testosterone and Estradiol Concentrations Following Acute Androstenedione Ingestion in Young Women*, Horm Metab Res 36: 62-66 (2004.)
- A study finding (among other things) that body height, body mass, vertical jump height, maximal oxygen consumption, and leg press maximal strength were higher in a group of physically active men than comparably active women, while the women had higher percent body fat. G. A. Brown, Michael W. Ray, et al., *Oxygen Consumption, Heart Rate, and Blood Lactate Responses to an Acute Bout of Plyometric Depth Jumps in College-Aged Men And Women*, J. Strength Cond Res 24: 2475-2482 (2010).
- A study finding (among other things) that height, body mass, and maximal oxygen consumption were higher in a group of male NCAA Division 2 distance runners, while women NCAA Division 2 distance runners had higher percent body fat. Furthermore, these male athletes had a faster mean competitive running speed (~3.44 min/km) than women (~3.88 min/km), even though the men ran 10 km while the women ran 6 km. Katherine Semin, Alvah C. Stahlnecker, Kate A. Heelan, G. A. Brown, et al, *Discrepancy Between Training, Competition and Laboratory Measures of Maximum Heart Rate in NCAA Division 2 Distance Runners*, Journal of Sports Science and Medicine 7: 455-460 (2008).
- A presentation at the 2021 American Physiological Society New Trends in Sex and

Gender Medicine Conference entitled "Transwomen Competing in Women's Sports: What We Know and What We Don't".

- I have also authored an August 2021 entry for the American Physiological Society Physiology Educators Community of Practice Blog (PECOP Blog) titled "The Olympics, Sex, and Gender in the Physiology Classroom, and a May 2023 entry for the PECOP Blog titled "The Olympics, sex, and gender in the physiology classroom (part 2): Are there sex based differences in athletic performance before puberty?" I have also authored an April 17, 2023 post for the Center on Sport Policy and Conduct titled "Should Transwomen be allowed to Compete in Women's Sports? A view from an Exercise Physiologist."
- A presentation at the 2022 annual meeting of the American College of Sports Medicine titled "Comparison of Running Performance Between Division and Sex in NCAA Outdoor Track Running Championships 2010-2019." And a presentation at the 2023 annual meeting of the American College of Sports Medicine titled "Boys and Girls Differ in Running and Jumping Track and Field Event Performance Before Puberty."

A list of my published scholarly work for the past 10 years appears as an Appendix.

Purpose of this Declaration

I have been asked by counsel for Proposed Intervenors Senator Warren Petersen, President of the Arizona Senate, and Representative Ben Toma, Speaker of the Arizona House of Representatives in the matter of *Doe and Roe v. Horne et al.* to offer my opinions about the following: (a) whether males have inherent advantages in athletic performance over females, and if so the scale and physiological basis of those advantages, to the extent currently understood by science and (b) whether the sex-based performance advantage enjoyed by males is eliminated if feminizing hormones are administered to male athletes who identify as transgender (and in the case of prepubertal children, whether puberty blockers eliminate the advantage). In this declaration, when I use the terms "boy" or "male," I am referring to biological males based on the individual's reproductive biology and genetics as determined at birth. Similarly, when I use the terms "girl" or "female," I am referring to biological females based on the individual's reproductive biology and genetics as determined at birth. When I use the term transgender, I am referring to persons who are males or females, but who identify as a member of the opposite sex.

I have previously provided expert information in cases similar to this one in the form of written declarations and depositions in the cases of *Soule vs. CIAC* in the state of Connecticut, *B.P.J. vs. West Virginia State Board of Education* in the state of West Virginia, and *L.E. vs. Lee* in the state of Tennessee, and in the form of a written declaration in the case of *Hecox vs. Little* in the state of Idaho. I have not previously testified as an expert in any trials.

The opinions I express in this declaration are my own, and do not necessarily reflect the opinions of my employer, the University of Nebraska.

I have been compensated for my time serving as an expert in this case at the rate of $200 per hour. My compensation does not depend on the outcome in the case.

Overview

In this declaration, I explore three important questions relevant to current discussions and policy decisions concerning inclusion of transgender individuals in women's athletic competitions. Based on my professional familiarity with exercise physiology and my review of the currently available science, including that contained in the many academic sources I cite in this report, I set out and explain three basic conclusions:

- At the level of (a) elite, (b) collegiate, (c) scholastic, and (d) recreational competition, men, adolescent boys, or male children, have an advantage over equally aged, gifted, and trained women, adolescent girls, or female children in almost all athletic events;
- Biological male physiology is the basis for the performance advantage that men, adolescent boys, or male children have over women, adolescent girls, or female children in almost all athletic events; and
- The administration of androgen inhibitors and cross-sex hormones to men or adolescent boys after the onset of male puberty does not eliminate the performance advantage that men and adolescent boys have over women and adolescent girls in almost all athletic events. Likewise, there is no published scientific evidence that the administration of puberty blockers to males before puberty eliminates the pre-existing athletic advantage that prepubertal males have over prepubertal females in almost all athletic events.

In short summary, men, adolescent boys, and prepubertal male children perform better in almost all sports than equally aged, trained, and gifted women, adolescent girls, and prepubertal female children because of their inherent physiological advantages. In general, men, adolescent boys, and prepubertal male children, can run faster, output more muscular power, jump higher, and possess greater muscular endurance than equally aged, trained, and gifted women, adolescent girls, and prepubertal female children. These advantages become greater during and after male puberty, but they exist before puberty.

Further, while after the onset of puberty males are on average taller and heavier than females, a male performance advantage over females has been measured in weightlifting competitions even between males and females matched for body mass.

Male advantages in measurements of body composition, tests of physical fitness, and athletic performance have also been shown in children before puberty. These advantages are magnified during puberty, triggered in large part by the higher testosterone concentrations in men, and adolescent boys, after the onset of male puberty. Under the influence of these higher testosterone levels, adolescent boys and young men develop even more muscle mass, greater muscle strength, less body fat, higher bone mineral density, greater bone strength, higher hemoglobin concentrations, larger hearts and larger coronary blood vessels, and larger overall statures than women. In addition, maximal oxygen consumption (VO_2max), which correlates to ~30-40% of success in endurance sports, is higher in both elite and average men and boys than in comparable women and girls when measured in regard to absolute volume of oxygen consumed and when measured relative to body mass.

Although androgen deprivation (that is, testosterone suppression) may modestly decrease some physiological advantages that men and adolescent boys have over equally aged, trained, and gifted women and adolescent girls, it cannot fully or even largely eliminate those physiological advantages once an individual has passed through male puberty.

Evidence and Conclusions

I. The scientific reality of biological sex

1. The scientific starting point for the issues addressed in this report is the biological fact of dimorphic sex in the human species. It is now well recognized that dimorphic sex is so fundamental to human development that, as stated in a recent position paper issued by the Endocrine Society, it "must be considered in the design and analysis of human and animal research. . . . Sex is dichotomous, with sex determination in the fertilized zygote stemming from unequal expression of sex chromosomal genes." (Bhargava et al. 2021 at 220). As stated by Sax (2002 at 177), "More than 99.98% of humans are either male or female." All humans who do not suffer from some genetic or developmental disorder are unambiguously male or female.

2. Although sex and gender are used interchangeably in common conversation, government documents, and in the scientific literature, the American Psychological Association defines sex as "physical and biological traits" that "distinguish between males and females" whereas gender "implies the psychological, behavioral, social, and cultural aspects of being male or female (i.e., masculinity or femininity)" (https://dictionary.apa.org, accessed May 5, 2023). The concept that sex is an important biological factor determined at conception is a well-established scientific fact that is supported by statements from a number of respected organizations including, but not limited to, the Endocrine Society (Bhargava et al. 2021 at 220), the American Physiological Society (Shah 2014), the Institute of Medicine, and the National Institutes of Health (Miller 2014 at H781-82). Collectively, these and other organizations have stated that every cell has a sex and every system in the body is influenced by sex. Indeed, "sex often influences gender, but gender cannot influence sex." (Bhargava 2021 at 228.)

3. To further explain: "The classical biological definition of the **2 sexes** is that females have ovaries and make larger female gametes (eggs), whereas males have testes and make smaller male gametes (sperm) … the definition can be extended to the ovaries

and testes, and in this way the categories—female and male—can be applied also to individuals who have gonads but do not make gametes … sex is dichotomous because of the different roles of each sex in reproduction.” (Bhargava 2021 at 221.) Furthermore, “sex determination begins with the inheritance of XX or XY chromosomes” (Bhargava 2021 at 221.) And, “Phenotypic sex differences develop in XX and XY embryos as soon as transcription begins. The categories of X and Y genes that are unequally represented or expressed in male and female mammalian zygotes … cause phenotypic sex differences” (Bhargava 2021 at 222.)

4. Although disorders of sexual development (DSDs) are sometimes confused with discussions of transgender individuals, the two are different phenomena. DSDs are disorders of physical development. Many DSDs are “associated with genetic mutations that are now well known to endocrinologists and geneticists.” (Bhargava 2021 at 225) By contrast, a sense of transgender identity is usually not associated with any physical disorder, and “a clear biological causative underpinning of gender identity remains to be demonstrated.” (Bhargava 2021 at 226.) The importance of distinguishing between the two is exemplified by the World Athletics Council updating “…the eligibility regulations for transgender and DSD athletes to compete in the female category” in March 2023. (World Athletics)

5. Further demonstrating the biological importance of sex, Gershoni and Pietrokovski (2017) detail the results of an evaluation of “18,670 out of 19,644 informative protein-coding genes in men versus women” and reported that “there are over 6500 protein-coding genes with significant S[ex]D[ifferential] E[xpression] in at least one tissue. Most of these genes have SDE in just one tissue, but about 650 have SDE in two or more tissues, 31 have SDE in more than five tissues, and 22 have SDE in nine or more tissues” (Gershoni 2017 at 2-3.) Some examples of tissues identified by these authors that have SDE genes include breast mammary tissue, skeletal muscle, skin, thyroid gland, pituitary gland, subcutaneous adipose, lung, and heart left ventricle. Based on these observations the authors state “As expected, Y-linked

genes that are normally carried only by men show SDE in many tissues" (Gershoni 2017 at 3.) A stated by Heydari et al. (2022, at 1), "Y chromosome harbors male-specific genes, which either solely or in cooperation with their X-counterpart, and independent or in conjunction with sex hormones have a considerable impact on basic physiology and disease mechanisms in most or all tissues development." As stated out by O'Connor (2023, at 2, quoting Institute of Medicine) "not every difference observed between male and female cells can be attributed to differences in exposure to sex hormones."

6. In a review of 56 articles on the topic of sex-based differences in skeletal muscle, Haizlip et al., (2015) state that "More than 3,000 genes have been identified as being differentially expressed between male and female skeletal muscle." (Haizlip 2015 at 30.) Furthermore, the authors state that "Overall, evidence to date suggests that skeletal muscle fiber-type composition is dependent on species, anatomical location/function, and sex" (Haizlip 2015 at 30.) The differences in genetic expression between males and females influence the skeletal muscle fiber composition (i.e. fast twitch and fast twitch sub-type and slow twitch), the skeletal muscle fiber size, the muscle contractile rate, and other aspects of muscle function that influence athletic performance. As the authors review the differences in skeletal muscle between males and females they conclude, "Additionally, all of the fibers measured in men have significantly larger cross-sectional areas (CSA) compared with women." (Haizlip 2015 at 31.) The authors also explore the effects of thyroid hormone, estrogen, and testosterone on gene expression and skeletal muscle function in males and females. One major conclusion by the authors is that "The complexity of skeletal muscle and the role of sex adding to that complexity cannot be overlooked." (Haizlip 2015 at 37.) The evaluation of SDE in protein coding genes helps illustrate that the differences between men and women are intrinsically part of the chromosomal and genetic makeup of humans which can influence many tissues that are inherent to the athletic competitive advantages of men compared to women.

II. Biological men, or adolescent boys, have large, well-documented performance advantages over women and adolescent girls in almost all athletic contests.

7. It should scarcely be necessary to invoke scientific experts to "prove" that men are on average larger, stronger, and faster than women. All of us, along with our siblings and our peers and perhaps our children, have passed through puberty, and we have watched that differentiation between the sexes occur. This is common human experience and knowledge.

8. Nevertheless, these differences have been extensively studied and measured. I cited many of these studies in the first paper on this topic that I prepared, which was submitted in litigation in January 2020. Since then, in light of current controversies, several authors have compiled valuable collections or reviews of data extensively documenting this objective fact about the human species, as manifest in almost all sports, each of which I have reviewed and found informative. These include Coleman (2020), Hilton & Lundberg (2021), World Rugby (2020), Harper (2021), Hamilton (2021), and a "Briefing Book" prepared by the Women's Sports Policy Working Group (2021). The important paper by Handelsman et al. (2018) also gathers scientific evidence of the systematic and large male athletic advantage.

9. These papers and many others document that men, adolescent boys, and prepubertal male children, substantially outperform comparably aged, gifted, and trained women, adolescent girls and prepubertal female children, in competitions involving running speed, swimming speed, cycling speed, jumping height, jumping distance, and strength (to name a few, but not all, of the performance differences). As I discuss later, it is now clear that these performance advantages for men, adolescent boys, and prepubertal male children, are inherent to the biological differences between the sexes.

10. In fact, I am not aware of any scientific evidence today that disproves that after puberty men possess large advantages in athletic performance over women–so large that they are generally insurmountable for comparably gifted and trained athletes at

every level (i.e. (a) elite, (b) collegiate, (c) scholastic, and (d) recreational competition). And I am not aware of any scientific evidence today that disproves that these measured performance advantages are at least largely the result of physiological differences between men and women which have been measured and are reasonably well understood.

11. My use of the term "advantage" in this paper must not be read to imply any normative judgment. The adult female physique is simply different from the adult male physique. Obviously, it is optimized in important respects for the difficult task of childbearing. On average, women require far fewer calories for healthy survival. Evolutionary biologists can and do theorize about the survival value or "advantages" provided by these and other distinctive characteristics of the female physique, but I will leave that to the evolutionary biologists. I use "advantage" to refer merely to performance advantages in athletic competitions.

12. I find in the literature a widespread consensus that the large performance and physiological advantages possessed by males–rather than social considerations or considerations of identity–are precisely the *reason* that most athletic competitions are separated by sex, with women treated as a "protected class." To cite only a few statements accepting this as the justification:

- Handelsman et al. (2018) wrote, "Virtually all elite sports are segregated into male and female competitions. The main justification is to allow women a chance to win, as women have major disadvantages against men who are, on average, taller, stronger, and faster and have greater endurance due to their larger, stronger, muscles and bones as well as a higher circulating hemoglobin level." (803)
- Millard-Stafford et al. (2018) wrote "Current evidence suggests that women will not swim or run as fast as men in Olympic events, which speaks against eliminating sex segregation in these individual sports" (530) "Given the historical context (2% narrowing in swimming over 44 y), a reasonable

assumption might be that no more than 2% of the current performance gap could still potentially be attributed to sociocultural influences.", (533) and "Performance gaps between US men and women stabilized within less than a decade after federal legislation provided equal opportunities for female participation, but only modestly closed the overall gap in Olympic swimming by 2% (5% in running)." (533) Dr. Millard-Stafford, a full professor at Georgia Tech, holds a Ph.D. in Exercise Physiology and is a past President of the American College of Sports Medicine.

- In 2021, Hilton et al. wrote, "most sports have a female category the purpose of which is the protection of both fairness and, in some sports, safety/welfare of athletes who do not benefit from the physiological changes induced by male levels of testosterone from puberty onwards." (204)
- In 2020 the Swiss High Court ("Tribunal Fédéral") observed that "in most sports . . . women and men compete in two separate categories, because the latter possess natural advantages in terms of physiology."[1]
- The members of the Women's Sports Policy Working Group wrote that "If sports were not sex-segregated, female athletes would rarely be seen in finals or on victory podiums," and that "We have separate sex sport and eligibility criteria based on biological sex because this is the only way we can assure that female athletes have the same opportunities as male athletes not only to participate but to win in competitive sport. . . . If we did not separate athletes on the basis of biological sex–if we used any other physical criteria–we would never see females in finals or on podiums." (WSPWG Briefing Book 2021 at 5, 20.)
- In 2020, the World Rugby organization stated that "the women's category exists to ensure protection, safety and equality for those who do not benefit from the

[1] "dans la plupart des sports . . . les femmes et les hommes concourent dans deux catégories séparées, ces derniers étant naturellement avantagés du point de vue physique." Tribunal Fédéral decision of August 25, 2020, Case 4A_248/2019, 4A_398/2019, at §9.8.3.3.

biological advantage created by these biological performance attributes." (World Rugby Transgender Women Guidelines 2020.)

- In 2021 Harper et al. stated "…the small decrease in strength in transwomen after 12–36 months of GAHT [Gender Affirming Hormone Therapy] suggests that transwomen likely retain a strength advantage over cisgender women." (7) and "…observations in trained transgender individuals are consistent with the findings of the current review in untrained transgender individuals, whereby 30 months of GAHT may be sufficient to attenuate some, but not all, influencing factors associated with muscular endurance and performance." (8)
- Hamilton et al (2021), "If a biologically male athlete self-identifies as a female, legitimately with a diagnosis of gender dysphoria or illegitimately to win medals, the athlete already possesses a physiological advantage that undermines fairness and safety. This is not equitable, nor consistent with the fundamental principles of the Olympic Charter and could be a potential danger to the health and safety of athletes." (840)
- Hamilton et al. (2021), in a consensus statement for the International Federation of Sports Medicine (FIMS) concluded that "Transwomen have the right to compete in sports. However, cisgender women have the right to compete in a protected category." (1409)

13. While the sources I mention above gather more extensive scientific evidence of this uncontroversial truth, I provide here a brief summary of representative facts concerning the male advantage in athletic performance.

A. Men are stronger.

14. Males exhibit greater strength throughout the body. Both Handelsman et al. (2018) and Hilton & Lundberg (2021) have gathered multiple literature references that document this fact in various muscle groups.

15. Men have in the neighborhood of 60%-100% greater **arm strength** than women.

(Handelsman 2018 at 812.)[2] One study of elbow flexion strength (basically, bringing the fist up towards the shoulder) in a large sample of men and women found that men exhibited 109% greater isometric strength, and 89% higher strength in a single repetition. (Hilton 2021 at 204, summarizing Hubal (2005) at Table 2.)

16. **Grip strength** is often used as a useful proxy for strength more generally. In one study, men showed on average 57% greater grip strength than women. (Bohannon 2019.) A wider meta-analysis of multiple grip-strength studies not limited to athletic populations found that 18- and 19-year-old males exhibited in the neighborhood of 2/3 greater grip strength than females. (Handelsman 2017 Figure 3, summarizing Silverman 2011 Table 1.)[3]

17. Liguori et al. (2021), in the *ACSM's Guidelines for Exercise Testing and Prescription* which is the flagship textbook for the American College of Sports Medicine and is considered the industry standard for information on evaluating physical fitness in adults, demonstrates that across all age groups and percentiles when comparing males and females, male handgrip strength is 66.2% higher than females (Table 3.10 at 95). To help illustrate this sex-based difference in handgrip strength, a 20–24-year-old male who ranks in the 95th percentile has 55 kg for handgrip strength in the dominant hand while a 20–24-year-old female who ranks in the 95th percentile has 34 kg for handgrip strength in the dominant hand. For comparison, a 20–24-year-old male with a handgrip strength of 34 kg would be in the 10th percentile for males.

18. In an evaluation of maximal isometric handgrip strength in 1,654 healthy men, 533

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[2] Handelsman expresses this as women having 50% to 60% of the "upper limb" strength of men. Handelsman cites Sale, *Neuromuscular function*, for this figure and the "lower limb" strength figure. Knox et al., *Transwomen in elite sport* (2018) are probably confusing the correct way to state percentages when they state that "differences lead to decreased trunk and lower body strength by 64% and 72% respectively, in women" (397): interpreted literally, this would imply that men have **almost 4x as much** lower body strength as do women.

[3] Citing Silverman, *The secular trend for grip strength in Canada and the United States*, J. Ports Sci. 29:599-606 (2011).

healthy women aged 20-25 years and 60 "highly trained elite female athletes from sports known to require high hand-grip forces (judo, handball)," Leyk et al. (2007) observed that, "The results of female national elite athletes even indicate that the strength level attainable by extremely high training will rarely surpass the 50th percentile of untrained or not specifically trained men." (Leyk 2007 at 415.)

19. Liguori et al. (2021), in the *ACSM's Guidelines for Exercise Testing and Prescription* indicates that when measuring upper body strength using bench press and expressing strength as the maximal weight lifted relative to body weight, males exhibit 64% greater strength (Table 3.11 at 96-97). To help illustrate this sex-based difference in upper body strength, an under 20-year-old male who ranks in the 95th percentile can bench press 1.76 kg for every kg of body mass while an under 20-year-old female who ranks in the 95th percentile can bench press 0.88 kg for every kg of body mass. For comparison, an under 20-year-old male with a bench press strength of 0.88 kg per kg of body mass would be between the 15th and 20th percentile for males.

20. Men have in the neighborhood of 25%-60% greater **leg strength** than women. (Handelsman 2018 at 812.) In another measure, men exhibit 54% greater knee extension torque and this male leg strength advantage is consistent across the lifespan. (Neder 1999 at 120-121.)

21. Liguori et al. (2021), in the *ACSM's Guidelines for Exercise Testing and Prescription* (Table 3.12 at 98), across all age groups and percentiles when comparing males and females, when measuring leg press strength as the maximal weight lifted relative to body weight, males exhibit 39% greater strength. To help illustrate this sex-based difference in lower body strength, a 20–29-year-old male who ranks in the 90th percentile can leg press 2.27 kg for every kg of body mass while a 20–29-year-old female who ranks in the 90th percentile can leg press 1.82 kg for every kg of body mass. For comparison, a 20–29-year-old male who can leg press 1.82 kg for every kg of body mass would be between the 30th and 40th

percentiles for males.

22. When male and female Olympic weightlifters of the same body weight are compared, the top males lift weights between 30% and 40% greater than the females of the same body weight. But when top male and female performances are compared in powerlifting, without imposing any artificial limitations on bodyweight, the male record is 65% higher than the female record. (Hilton 2021 at 203.)

23. In another measure that combines many muscle groups as well as weight and speed, moderately trained males generated 162% greater punching power than females even though men do not possess this large an advantage in any single bio-mechanical variable. (Morris 2020.) This objective reality was subjectively summed up by women's mixed-martial arts fighter Tamikka Brents, who suffered significant facial injuries when she fought against a biological male who identified as female and fought under the name of Fallon Fox. Describing the experience, Brents said:

> "I've fought a lot of women and have never felt the strength that I felt in a fight as I did that night. I can't answer whether it's because she was born a man or not because I'm not a doctor. I can only say, I've never felt so overpowered ever in my life, and I am an abnormally strong female in my own right."[4]

B. Men run faster.

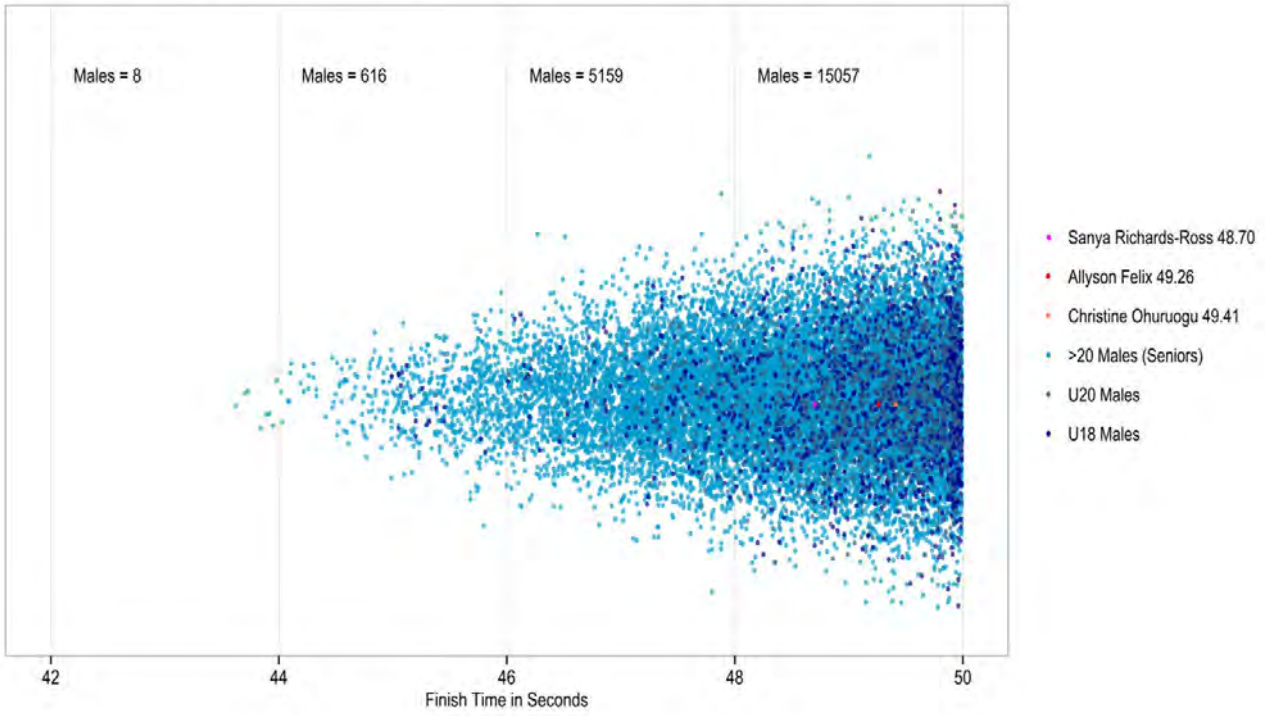
24. Many scholars have detailed the wide performance advantages enjoyed by men in running speed. One can come at this reality from a variety of angles.

25. Multiple authors report a male speed advantage in the neighborhood of 10%-13% in a variety of events, with a variety of study populations. Handelsman et al. 2018 at 813 and Handelsman 2017 at 70 both report a male advantage of about 10% by age 17. Thibault et al. 2010 at 217 similarly reported a stable 10% performance

[4] http://whoatv.com/exclusive-fallon-foxs-latest-opponent-opens-up-to-whoatv/ (last accessed May 5, 2023).

advantage across multiple events at the Olympic level. Tønnessen et al. (2015 at 1-2) surveyed the data and found a consistent male advantage of 10%-12% in running events after the completion of puberty. They document this for both short sprints and longer distances. One group of authors found that the male advantage increased dramatically in ultra-long-distance competition (Lepers & Knechtle 2013.)

26. A great deal of current interest has been focused on track events. It is worth noting that a recent analysis of publicly available sports federation and tournament records found that men enjoy the *least* advantage in running events, as compared to a range of other events and metrics, including jumping, pole vaulting, tennis serve speed, golf drives, baseball pitching speed, and weightlifting. (Hilton 2021 at 201-202.) Nevertheless, as any serious runner will recognize, the approximately 10% male advantage in running is an overwhelming difference. Dr. Hilton calculates that "approximately 10,000 males have personal best times that are faster than the current Olympic 100m female champion." (Hilton 2021 at 204.) Professors Doriane Coleman, Jeff Wald, Wickliffe Shreve, and Richard Clark dramatically illustrated this by compiling the data and creating the figure below (last accessed on May 5, 2023, at https://bit.ly/35yOyS4), which shows that the *lifetime best performances* of three female Olympic champions in the 400m event—including Team USA's Sanya Richards-Ross and Allyson Felix—would not match the performances of "literally thousands of boys and men, including thousands who would be considered second tier in the men's category" *just in 2017 alone*: (data were drawn from the International Association of Athletics Federations (IAAF) website which provides complete, worldwide results for individuals and events, including on an annual and an all-time basis).



27. Professor Coleman and her colleague Wicklyffe Shreve also created the table below (last accessed on May 5, 2023, at https://bit.ly/37E1s2X), which "compares the number of men—males over 18—competing in events reported to the International Association of Athletics Federation whose results in each event in 2017 would have ranked them above the very best elite woman that year."

TABLE 2 – World's Best Woman v. Number of Men Outperforming

Event	Best Women's Result	Best Men's Result	# of Men Outperforming
100 Meters	10.71	9.69	2,474
200 Meters	21.77	19.77	2,920
400 Meters	49.46	43.62	4,341
800 Meters	1:55.16*	1:43.10	3,992+
1500 Meters	3:56.14	3:28.80	3,216+
3000 Meters	8:23.14	7:28.73	1307+
5000 Meters	14:18.37	12:55.23	1,243
High Jump	2.06 meters	2.40 meters	777
Pole Vault	4.91 meters	6.00 meters	684
Long Jump	7.13 meters	8.65 meters	1,652
Triple Jump	14.96 meters	18.11 meters	969

28. The male advantage becomes insuperable well before the developmental changes of puberty are complete. Dr. Hilton documents that even "schoolboys"–defined as age 15 and under–have beaten the female world records in running, jumping, and throwing events. (Hilton 2021 at 204.)

29. Similarly, Coleman and Shreve created the table below (last accessed on May 5, 2023, at https://bit.ly/37E1s2X), which "compares the number of boys—males under the age of 18—whose results in each event in 2017 would rank them above the single very best elite [adult] woman that year:" data were drawn from the International Association of Athletics Federations (IAAF) website

TABLE 1 – World's Best Woman v. Under 18 Boys

Event	Best Women's Result	Best Boys' Result	# of Boys Outperforming
100 Meters	10.71	10.15	124+
200 Meters	21.77	20.51	182
400 Meters	49.46	45.38	285
800 Meters	1:55.16*	1:46.3	201+
1500 Meters	3:56.14	3:37.43	101+
3000 Meters	8:23.14	7:38.90	30
5000 Meters	14:18.37	12:55.58	15
High Jump	2.06 meters	2.25 meters	28
Pole Vault	4.91 meters	5.31 meters	10
Long Jump	7.13 meters	7.88 meters	74
Triple Jump	14.96 meters	17.30 meters	47

30. In an analysis I have performed of running events (consisting of the 100 m, 200 m, 400 m, 800 m, 1500 m, 5000 m, and 10000 m) in the Division I, Division II, and Division III NCAA Outdoor track championships for the years of 2010-2019, the average performance across all events of the 1st place man was 14.1% faster than the 1st place woman, with the smallest difference being a 10.2% advantage for men in the Division I 100 m race. The average 8th place man across all events (the last place to earn the title of All American) was 11.2% faster than 1st place woman, with the smallest difference being a 6.5% advantage for men in the Division I 100 m race. Importantly, the only overlap between men's and women's performance occurred only when a male performed exceptionally poorly (Brown et al. presented at the 2022 Annual Meeting of the American College of Sports Medicine.)

31. Athletic.net® is an internet-based resource providing "results, team, and event management tools to help coaches and athletes thrive." Among the resources available on Athletic.net are event records that can be searched nationally or by state age group, school grade, and state. Higerd (2021) in an evaluation of high school

track running performance records from five states (CA, FL, MN, NY, WA), over three years (2017 – 2019) observed that males were 14.38% faster than females in the 100M (at 99), 16.17% faster in the 200M (at 100), 17.62% faster in the 400M (at 102), 17.96% faster in the 800M (at 103), 17.81% faster in the 1600M (at 105), and 16.83% faster in the 3200M (at 106).

C. Men jump higher and farther.

32. Jumping involves both leg strength and speed as positive factors, with body weight of course a factor working against jump height. Despite their substantially greater body weight, males enjoy an even greater advantage in jumping than in running. Handelsman 2018 at 813, looking at youth and young adults, and Thibault 2010 at 217, looking at Olympic performances, both found male advantages in the range of 15%-20%. See also Tønnessen 2015 (approximately 19%); Handelsman 2017 (19%); Hilton 2021 at 201 (18%). Looking at the vertical jump called for in volleyball, research on elite volleyball players found that males jumped on average 50% higher during an "attack" at the net than did females. (Sattler 2015; see also Hilton 2021 at 203 (33% higher vertical jump).)

33. Higerd (2021) in an evaluation of high school high jump performance available through the track and field database athletic.net®, which included five states (CA, FL, MN, NY, WA), over three years (2017 – 2019) (at 82) observed that in 23,390 females and 26,843 males, females jumped an average of 1.35 m and males jumped an average of 1.62 m, for an 18.18% performance advantage for males (at 96). In an evaluation of long jump performance in 45,705 high school females and 54,506 high school males, the females jumped an average of 4.08 m and males jumped an average of 5.20 m, for a 24.14% performance advantage for males (at 97).

34. The combined male advantage of body height and jump height means, for example, that a total of seven women in the WNBA have ever dunked a basketball in the

regulation 10 foot hoop,[5] while the ability to dunk appears to be almost universal among NBA players: "Since the 1996–97 season (the earliest data is available from Basketball-Reference.com), 1,801 different [NBA] players have combined for 210,842 regular-season dunks, and 1,259 out of 1,367 players (or 92%) who have played at least 1,000 minutes have dunked at least once."[6]

D. Men throw, hit, and kick faster and farther.

35. Strength, arm-length, and speed combine to give men a large advantage over women in throwing. This has been measured in a number of studies.

36. One study of elite male and female baseball pitchers showed that men throw baseballs 35% faster than women—81 miles/hour for men vs. 60 miles/hour for women. (Chu 2009.) By age 12, "boys' throwing velocity is already between 3.5 and 4 standard deviation units higher than the girls'." (Thomas 1985 at 276.) By age seventeen, the *average* male can throw a ball farther than 99% of seventeen-year-old females. (Lombardo 2018; Chu 2009; Thomas 1985 at 268.) Looking at publicly available data, Hilton & Lundberg found that in both baseball pitching and the field hockey "drag flick," the *record* ball speeds achieved by males are more than 50% higher than those achieved by females. (Hilton 2021 at 202-203.)

37. Men achieve serve speeds in tennis more that 15% faster than women; and likewise in golf achieve ball speeds off the tee more than 15% faster than women. (Hilton 2021 at 202.)

38. More specifically, Marshall and Llewellyn (at 957) reported that female collegiate golfers at an NCAA Division III school have an average drive distance that is 46 yards (16.5%) fewer than males, a maximal drive distance of 33.2 yards (11.1%) fewer, an average club head speed that is 21.9 mph (20.4%) slower, and a maximum

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[5] https://www.espn.com/wnba/story/_/id/32258450/2021-wnba-playoffs-brittney-griner-owns-wnba-dunking-record-coming-more.

[6] https://www.si.com/nba/2021/02/22/nba-non-dunkers-patty-mills-tj-mcconnell-steve-novak-daily-cover

club head speed that is 18 mph (15.3%) slower. Using 3D motion analysis to evaluate the kinematics of 7 male and 5 female golfers with a mean handicap of 6, Egret (at 463) concluded that "The results of this study show that there is a specific swing for women." Horan used 3D motion analysis to evaluate the kinematics of 19 male and 19 female golfers with a handicap less than or equal to 4 and concluded "the results suggest that male and female skilled golfers have different kinematics for thorax and pelvis motion" and "What might be considered optimal swing characteristics for male golfers should not be generalized to female golfers." (at 1456).

39. Males are able to throw a javelin more than 30% farther than females. (Lombardo 2018 Table 2; Hilton 2021 at 203.)

40. Men serve and spike volleyballs with higher velocity than women, with a performance advantage in the range of 29-34%. (Hilton 2021 at 204 Fig. 1.)

41. Men are also able to kick balls harder and faster. A study comparing collegiate soccer players found that males kick the ball with an average 20% greater velocity than females. (Sakamoto 2014.)

E. Males exhibit faster reaction times.

42. Interestingly, men enjoy an additional advantage over women in reaction time–an attribute not obviously related to strength or metabolism (e.g. $V0_2max$). "Reaction time in sports is crucial in both simple situations such as the gun shot in sprinting and complex situations when a choice is required. In many team sports this is the foundation for tactical advantages which may eventually determine the outcome of a game." (Dogan 2009 at 92.) "Reaction times can be an important determinant of success in the 100m sprint, where medals are often decided by hundredths or even thousandths of a second." (Tønnessen 2013 at 885.)

43. The existence of a sex-linked difference in reaction times is consistent over a wide range of ages and athletic abilities. (Dykiert 2012.) Even by the age of 4 or 5, in a ruler-drop test, males have been shown to exhibit 4% to 6% faster reaction times

than females. (Latorre-Roman 2018.) In high school athletes taking a common baseline "ImPACT" test, males showed 3% faster reaction times than females. (Mormile 2018.) Researchers have found a 6% male advantage in reaction times of both first-year medical students (Jain 2015) and world-class sprinters (Tønnessen 2013).

44. Most studies of reaction times use computerized tests which ask participants to hit a button on a keyboard or to say something in response to a stimulus. One study on NCAA athletes measured "reaction time" by a criterion perhaps more closely related to athletic performance–that is, how fast athletes covered 3.3 meters after a starting signal. Males covered the 3.3 meters 10% faster than females in response to a visual stimulus, and 16% faster than females in response to an auditory stimulus. (Spierer 2010.)

45. Researchers have speculated that sex-linked differences in brain structure, as well as estrogen receptors in the brain, may be the source of the observed male advantage in reaction times, but at present this remains a matter of speculation and hypothesis. (Mormile at 19; Spierer at 962.)

III. Men have large measured physiological differences compared to women which demonstrably or likely explain their performance advantages.

46. No single physiological characteristic alone accounts for all or any one of the measured advantages that men enjoy in athletic performance. However, scientists have identified and measured a number of physiological factors that contribute to superior male performance.

A. Men are taller and heavier than women

47. In some sports, such as basketball and volleyball, height itself provides competitive advantage. While some women are taller than some men, based on data from 20 countries in North America, Europe, East Asia, and Australia, the 50th percentile for body height for women is 164.7 cm (5 ft 5 inches) and the 50th percentile for body height for men is 178.4 cm (5 ft 10 inches). Helping to illustrate the inherent height

difference between men and women, from the same data analysis, the 95th percentile for body height for women is 178.9 cm (5 feet 10.43 inches), which is only 0.5 cm taller than the 50th percentile for men (178.4 cm; 5 feet 10.24 inches), while the 95th percentile for body height for men is 193.6 cm (6 feet 4.22 inches). Thus, while some women are taller than some men, the tallest men are taller than the tallest women (Roser 2013.)

48. To look at a specific athletic population, an evaluation of NCAA Division I basketball players compared 68 male guards and 59 male forwards to 105 female guards and 91 female forwards, and found that on average the male guards were 187.4 ± 7.0 cm tall and weighed 85.2 ± 7.4 kg while the female guards were 171.6 ± 5.0 cm tall and weighed 68.0 ± 7.4 kg. The male forwards were 201.7 ± 4.0 cm tall and weighed 105.3 ± 5.9 kg while the female forwards were 183.5 ± 4.4 cm tall and weighed 82.2 ± 12.5 kg. (Fields 2018 at 3.)

B. Males have larger and longer bones, stronger bones, and different bone configuration.

49. Obviously, males on average have longer bones. "Sex differences in height have been the most thoroughly investigated measure of bone size, as adult height is a stable, easily quantified measure in large population samples. Extensive twin studies show that adult height is highly heritable with predominantly additive genetic effects that diverge in a sex-specific manner from the age of puberty onwards." (Handelsman 2018 at 818.) "Pubertal testosterone exposure leads to an ultimate average greater height in men of 12–15 centimeters, larger bones, greater muscle mass, increased strength and higher hemoglobin levels." (Gooren 2011 at 653.)

50. "Men have distinctively greater bone size, strength, and density than do women of the same age." (Handelsman 2018 at 818.)

51. "[O]n average men are 7% to 8% taller with longer, denser, and stronger bones, whereas women have shorter humerus and femur cross-sectional areas being 65% to 75% and 85%, respectively, those of men." (Handelsman 2018 at 818.)

52. Greater height, leg, and arm length themselves provide obvious advantages in several sports. But male bone geometry also provides less obvious advantages. "The major effects of men's larger and stronger bones would be manifest via their taller stature as well as the larger fulcrum with greater leverage for muscular limb power exerted in jumping, throwing, or other explosive power activities." (Handelsman 2018 at 818.)

53. Male advantage in bone size is not limited to length, as larger bones provide the mechanical framework for larger muscle mass. "From puberty onwards, men have, on average, 10% more bone providing more surface area. The larger surface area of bone accommodates more skeletal muscle so, for example, men have broader shoulders allowing more muscle to build. This translates into 44% less upper body strength for women, providing men an advantage for sports like boxing, weightlifting and skiing. In similar fashion, muscle mass differences lead to decreased trunk and lower body strength by 64% and 72%, respectively in women. These differences in body strength can have a significant impact on athletic performance, and largely underwrite the significant differences in world record times and distances set by men and women." (Knox 2019 at 397.)

54. Meanwhile, distinctive aspects of the female pelvis geometry cut against athletic performance. "[T]he widening of the female pelvis during puberty, balancing the evolutionary demands of obstetrics and locomotion, retards the improvement in female physical performance." (Handelsman 2018 at 818.) "[T]he major female hormones, oestrogens, can have effects that disadvantage female athletic performance. For example, women have a wider pelvis changing the hip structure significantly between the sexes. Pelvis shape is established during puberty and is driven by oestrogen. The different angles resulting from the female pelvis leads to decreased joint rotation and muscle recruitment ultimately making them slower." (Knox 2019 at 397.)

55. There are even sex-based differences in foot size and shape. Wunderlich &

Cavanaugh (2001) observed that a "foot length of 257 mm represents a value that is … approximately the 20th percentile men's foot lengths and the 80th percentile women's foot lengths." (607) and "For a man and a woman, both with statures of 170 cm (5 feet 7 inches), the man would have a foot that was approximately 5 mm longer and 2 mm wider than the woman." (608). Based on these, and other analyses, they conclude that "female feet and legs are not simply scaled-down versions of male feet but rather differ in a number of shape characteristics, particularly at the arch, the lateral side of the foot, the first toe, and the ball of the foot." (605) Further, Fessler et al. (2005) observed that "female foot length is consistently smaller than male foot length" (44) and concludes that "proportionate foot length is smaller in women" (51) with an overall conclusion that "Our analyses of genetically disparate populations reveal a clear pattern of sexual dimorphism, with women consistently having smaller feet proportionate to stature than men." (53)

56. Beyond simple performance, the greater density and strength of male bones provide higher protection against stresses associated with extreme physical effort: "[S]tress fractures in athletes, mostly involving the legs, are more frequent in females, with the male protection attributable to their larger and thicker bones." (Handelsman 2018 at 818.)

C. Males have much larger muscle mass.

57. The fact that, on average, men have substantially larger muscles than women is as well known to common observation as men's greater height. But the male advantage in muscle size has also been extensively measured. The differential is large.

58. "On average, women have 50% to 60% of men's upper arm muscle cross-sectional area and 65% to 70% of men's thigh muscle cross-sectional area, and women have 50% to 60% of men's upper limb strength and 60% to 80% of men's leg strength. Young men have on average a skeletal muscle mass of >12 kg greater than age-matched women at any given body weight." (Handelsman 2018 at 812. See also Gooren 2011 at 653, Thibault 2010 at 214.)

59. "There is convincing evidence that the sex differences in muscle mass and strength are sufficient to account for the increased strength and aerobic performance of men compared with women and is in keeping with the differences in world records between the sexes." (Handelsman 2018 at 816.)

60. As stated in the National Strength and Conditioning Association's *Guide to Tests and Assessments* "Sport performance is highly dependent on the health- and skill-related components of fitness (power, speed, agility, reaction time, balance, and Body Composition coordination) in addition to the athlete's technique and level of competency in sport-specific motor skills. All fitness components depend on body composition to some extent. An increase in lean body mass contributes to strength and power development. … Thus, an increase in lean body mass enables the athlete to generate more force in a specific period of time. A sufficient level of lean body mass also contributes to speed, quickness, and agility performance (in the development of force applied to the ground for maximal acceleration and deceleration)." (https://www.nsca.com/education/articles/kinetic-select/sport-performance-and-body-composition/ last accessed May 10, 2023)

61. Once again, looking at specific and comparable populations of athletes, an evaluation of NCAA Division I basketball players consisting of 68 male guards and 59 male forwards, compared to 105 female guards and 91 female forwards, reported that on average the male guards had 77.7 ± 6.4 kg of fat free mass and 7.4 ± 3.1 kg fat mass while the female guards had 54.6 ± 4.4 kg fat free mass and 13.4 ± 5.4 kg fat mass. The male forwards had 89.5 ± 5.9 kg fat free mass and 15.9 ± 5.6 kg fat mass while the female forwards had 61.8 ± 5.9 kg fat free mass and 20.5 ± 7.7 kg fat mass. (Fields 2018 at 3.)

D. Females have a larger proportion of body fat.

62. While women have smaller muscles, they have proportionately more body fat, in general a negative for athletic performance. "Oestrogens also affect body composition by influencing fat deposition. Women, on average, have higher

percentage body fat, and this holds true even for highly trained healthy athletes (men 5%–10%, women 8%–15%). Fat is needed in women for normal reproduction and fertility, but it is not performance-enhancing. This means men with higher muscle mass and less body fat will normally be stronger kilogram for kilogram than women." (Knox 2019 at 397.)

63. Looking once again to Liguri (2021) in the *ACSM's Guidelines for Exercise Testing and Prescription* (Tables 3.4 and 3.5 at 73 and 74), a 20–29-year-old male in the 99th percentile will have 4.2% body fat, while a 20–29-year-old female in the 99th percentile will have 11.4% body fat, meaning the female has 170% more fat relative to body mass than the male. Comparing a 20–29-year-old male and female in the 50th percentile (that is "average") the male will have 16.7% body fat and the female will have 21.8% body fat, meaning that the female has 30% more fat relative to total body mass than the male.

64. "[E]lite females have more (<13 vs. <5 %) body fat than males. Indeed, much of the difference in [maximal oxygen uptake] between males and females disappears when it is expressed relative to lean body mass. . . . Males possess on average 7–9 % less percent body fat than females." (Lepers 2013 at 853.)

65. Knox et al. observe that both female pelvis shape and female body fat levels "disadvantage female athletes in sports in which speed, strength and recovery are important," (Knox 2019 at 397), while Tønnessen et al. describe the "ratio between muscular power and total body mass" as "critical" for athletic performance. (Tønnessen 2015 at 7.)

E. Males are able to metabolize and release energy to muscles at a higher rate due to larger heart and lung size, and higher hemoglobin concentrations.

66. While advantages in bone size, muscle size, and body fat are easily perceived and understood by laymen, scientists also measure and explain the male athletic advantage at a more abstract level through measurements of metabolism, or the ability to deliver energy to muscles throughout the body.

67. Energy release at the muscles depends centrally on the body's ability to deliver oxygen to the muscles, where it is essential to the complex chain of biochemical reactions that make energy available to power muscle fibers. Men have multiple distinctive physiological attributes that together give them a large advantage in oxygen delivery.

68. Oxygen is taken into the blood in the lungs. Men have greater capability to take in oxygen for multiple reasons. "**[L]ung capacity** [is] larger in men because of a lower diaphragm placement due to Y-chromosome genetic determinants." (Knox 2019 at 397.) Supporting larger lung capacity, men have "greater cross-sectional area of the trachea"; that is, they can simply move more air in and out of their lungs in a given time. (Hilton 2021 at 201.)

69. More, male lungs provide superior oxygen exchange even for a given volume: "The greater lung volume is complemented by testosterone-driven **enhanced alveolar multiplication** rate during the early years of life. Oxygen exchange takes place between the air we breathe and the bloodstream at the alveoli, so more alveoli allows more oxygen to pass into the bloodstream. Therefore, the greater lung capacity allows more air to be inhaled with each breath. This is coupled with an improved uptake system allowing men to absorb more oxygen." (Knox 2019 at 397.)

70. "Once in the blood, oxygen is carried by haemoglobin. **Haemoglobin concentrations** are directly modulated by testosterone so men have higher levels and can carry more oxygen than women." (Knox 2019 at 397.) "It is well known that levels of circulating hemoglobin are androgen-dependent and consequently higher in men than in women by 12% on average…. Increasing the amount of hemoglobin in the blood has the biological effect of increasing oxygen transport from lungs to tissues, where the increased availability of oxygen enhances aerobic energy expenditure." (Handelsman 2018 at 816.) (See also Lepers 2013 at 853; Handelsman 2017 at 71.) "It may be estimated that as a result the average maximal oxygen transfer will be ~10% greater in men than in women, which has a direct

impact on their respective athletic capacities." (Handelsman 2018 at 816.)

71. But the male metabolic advantage is further multiplied by the fact that men are also able to **circulate more blood per second** than are women. "Oxygenated blood is pumped to the active skeletal muscle by the heart. The left ventricle chamber of the heart is the reservoir from which blood is pumped to the body. The larger the left ventricle, the more blood it can hold, and therefore, the more blood can be pumped to the body with each heartbeat, a physiological parameter called 'stroke volume'. The female heart size is, on average, 85% that of a male resulting in the stroke volume of women being around 33% less." (Knox 2018 at 397.) Hilton cites different studies that make the same finding, reporting that men on average can pump 30% more blood through their circulatory system per minute ("cardiac output") than can women. (Hilton 2021 at 202.)

72. Finally, at the cell where the energy release is needed, men appear to have yet another advantage. "Additionally, there is experimental evidence that testosterone increases . . . **mitochondrial biogenesis**, myoglobin expression, and IGF-1 content, which may augment energetic and power generation of skeletal muscular activity." (Handelsman 2018 at 811.)

73. "Putting all of this together, men have a much more efficient cardiovascular and respiratory system." (Knox 2019 at 397.) A widely accepted measurement that reflects the combined effects of all these respiratory, cardiovascular, and metabolic advantages is referred to as "$V0_2$max," which refers to the maximum rate at which an individual can consume oxygen during aerobic exercise.[7] Looking at 11 separate studies, including both trained and untrained individuals, Pate et al. concluded that men have a 50% higher $V0_2$max than women on average, and a 25% higher $V0_2$max

[7] $V0_2$max is "based on hemoglobin concentration, total blood volume, maximal stroke volume, cardiac size/mass/compliance, skeletal muscle blood flow, capillary density, and mitochondrial content." International Statement, *The Role of Testosterone in Athletic Performance* (January 2019), available at https://law.duke.edu/sites/default/files/centers/sportslaw/Experts_T_Statement_2019.pdf.

in relation to body weight. (Pate 1984 at 92. See also Hilton 2021 at 202.)

IV. The role of testosterone in the development of male advantages in athletic performance.

74. The following tables of reference ranges for circulating testosterone in males and females are presented to help provide context for some of the subsequent information regarding athletic performance and physical fitness in children, youth, and adults, and regarding testosterone suppression in transwomen and athletic regulations. These data were obtained from the Mayo Clinic Laboratories (available at https://www.mayocliniclabs.com/test-catalog/overview/83686#Clinical-and-Interpretive, accessed May 5, 2023).

Reference ranges for serum testosterone concentrations in males and females.

Age	Males	Females
0 – 5 months	2.6 – 13.9 nmol/l	0.7 – 2.8 nmol/l
6 months – 9 years	0.2 – 0.7 nmol/l	0.2 – 0.7 nmol/l
10 – 11 years	0.2 – 4.5 nmol/l	0.2 – 1.5 nmol/l
12 -13 years	0.2 – 27.7 nmol/l	0.2 – 2.6 nmol/l
14 years	0.2 – 41.6 nmol/l	0.2 – 2.6 nmol/l
15 – 16 years	3.5 – 41.6 nmol/l	0.2 – 2.6 nmol/l
17 – 18 years	10.4 – 41.6 nmol/l	0.7 – 2.6 nmol/l
19 years and older	8.3 – 32.9 nmol/l	0.3 – 2.1 nmol/l

Please note that testosterone concentrations are sometimes expressed in units of ng/dl, and 1 nmol/l = 28.85 ng/dl.

75. Tanner Stages can be used to help evaluate the onset and progression of puberty and may be more helpful in evaluating normal testosterone concentrations than age in adolescents. "Puberty onset (transition from Tanner stage I to Tanner stage II) occurs for boys at a median age of 11.5 years and for girls at a median age of 10.5 years. . . . Progression through Tanner stages is variable. Tanner stage V (young adult) should be reached by age 18." (https://www.mayocliniclabs.com/test-

catalog/overview/83686#Clinical-and-Interpretive, accessed May 5, 2023).

Reference Ranges for serum testosterone concentrations by Tanner stage

Tanner Stage	Males	Females
I (prepubertal)	0.2 – 0.7 nmol/l	0.7 – 0.7 nmol/l
II	0.3 – 2.3 nmo/l	0.2 – 1.6 nmol/l
III	0.9 – 27.7 nmol/l	0.6 – 2.6 nmol/l
IV	2.9 – 41.6 nmol/l	0.7 – 2.6 nmol/l
V (young adult)	10.4 – 32.9 nmol/	0.4 – 2.1 nmol/l

76. Senefeld et al. (2020 at 99) state that "Data on testosterone levels in children and adolescents segregated by sex are scarce and based on convenience samples or assays with limited sensitivity and accuracy." They therefore "analyzed the timing of the onset and magnitude of the divergence in testosterone in youths aged 6 to 20 years by sex using a highly accurate assay" (isotope dilution liquid chromatography tandem mass spectrometry). Senefeld observed a significant difference beginning at age 11, which is to say about fifth grade.

Serum testosterone concentrations (nmol/L) in youths aged 6 to 20 years measured using isotope dilution liquid chromatography tandem mass spectrometry (Senefeld et al. ,2020, at 99)

	Boys			Girls		
Age (y)	**5th**	**50th**	**95th**	**5th**	**50th**	**95th**
6	0.0	0.1	0.2	0.0	0.1	0.2
7	0.0	0.1	0.2	0.0	0.1	0.3
8	0.0	0.1	0.3	0.0	0.1	0.3
9	0.0	0.1	0.3	0.1	0.2	0.6
10	0.1	0.2	2.6	0.1	0.3	0.9
11	0.1	0.5	11.3	0.2	0.5	1.3
12	0.3	3.6	17.2	0.2	0.7	1.4
13	0.6	9.2	21.5	0.3	0.8	1.5
14	2.2	11.9	24.2	0.3	0.8	1.6
15	4.9	13.2	25.8	0.4	0.8	1.8
16	5.2	14.9	24.1	0.4	0.9	2.0
17	7.6	15.4	27.0	0.5	1.0	2.0
18	9.2	16.3	25.5	0.4	0.9	2.1
19	8.1	17.2	27.9	0.4	0.9	2.3
20	6.5	17.9	29.9	0.4	1.0	3.4

A. Boys exhibit advantages in athletic performance even before puberty.

77. It is often said or assumed that boys enjoy no significant athletic advantage over girls before puberty. However, this is not true. Writing in their seminal work on the physiology of elite young female athletes, McManus and Armstrong (2011) reviewed the differences between boys and girls regarding bone density, body composition, cardiovascular function, metabolic function, and other physiologic factors that can influence athletic performance. They stated, "At birth, boys tend to

have a greater lean mass than girls. This difference remains small but detectable throughout childhood with about a 10% greater lean mass in boys than girls prior to puberty." (28) "Sexual dimorphism underlies much of the physiologic response to exercise," and most importantly these authors concluded that, "Young girl athletes are not simply smaller, less muscular boys." (23)

78. Certainly, boys' physiological and performance advantages increase rapidly from the beginning of puberty until around age 17-19. But much data and multiple studies show that significant physiological differences, and significant male athletic performance advantages in certain areas, exist before significant developmental changes associated with male puberty have occurred.

79. Starting at birth, girls have more body fat and less fat-free mass than boys. Davis et al. (2019) in an evaluation of 602 infants reported that at birth and age 5 months, infant boys have larger total body mass, body length, and fat-free mass while having lower percent body fat than infant girls. In an evaluation of 20 boys and 20 girls ages 3-8 years old, matched for age, height, and body weight Taylor et al. (Taylor 1997) reported that the "boys had significantly less fat, a lower % body fat and a higher bone-free lean tissue mass than the girls" when "expressed as a percentage of the average fat mass of the boys", the girls' fat mass was 52% higher than the boys "…while the bone-free lean tissue mass was 9% lower" (at 1083.) In an evaluation of 376 prepubertal [Tanner Stage 1] boys and girls, Taylor et al. (2010) observed that the boys had 21.6% more lean mass, and 13% less body fat (when expressed as percent of total body mass) than did the girls. In an evaluation of bone mineral density in 1,432 boys and 1,483 girls who were an average of 6.2 years old Medina-Gomez (2016) observed that the boys had 7.6% more lean body mass, 15.6% less fat mass, and ~5% higher bone mineral density than the girls (Table 1, at 1102), and concluded that (at 1099), "bone sexual dimorphism is already present at 6 years of age, with boys having stronger bones than girls, the relation of which is influenced by body composition." In a review of 22 peer reviewed publications

on the topic, Staiano and Katzmarzyk (2012) conclude that "… girls have more T[otal]B[ody]F[at] than boys throughout childhood and adolescence." (at 4.)

80. In the seminal textbook, *Growth, Maturation, and Physical Activity*, Malina et al. (2004) present a summary of data from Gauthier et al. (1983) which present data from "a national sample of Canadian children and youth" demonstrating that from ages 7 to 17, boys have a higher aerobic power output than do girls of the same ages when exercise intensity is measured using heart rate (Malina at 242.) That is to say, that at a heart rate of 130 beats per minute, or 150, or 170, a 7 to 17 year old boy should be able to run, bike, or swim faster than a similarly aged girl.

81. Considerable data from school-based fitness testing exists showing that prepubertal boys outperform comparably aged girls in tests of muscular strength, muscular endurance, and running speed. These sex-based differences in physical fitness are relevant to the current issue of sex-based sports categories because, as stated by Lesinski et al. (2020), in an evaluation "of 703 male and female elite young athletes aged 8–18" (1) "fitness development precedes sports specialization" (2) and further observed that "males outperformed females in C[ounter]M[ovement]J[ump], D[rop]J[ump], C[hange]o[f]D[irection speed] performances and hand grip strength." (5).

82. Tambalis et al. (2016) states that "based on a large data set comprising 424,328 test performances" (736) using standing long jump to measure lower body explosive power, sit and reach to measure flexibility, timed 30 second sit ups to measure abdominal and hip flexor muscle endurance, 10 x 5 meter shuttle run to evaluate speed and agility, and multi-stage 20 meter shuttle run test to estimate aerobic performance (738). "For each of the fitness tests, performance was better in boys compared with girls ($p < 0.001$), except for the S[it and] R[each] test ($p < 0.001$)." (739) In order to illustrate that the findings of Tambalis (2016) are not unique to children in Greece, the authors state "Our findings are in accordance with recent studies from Latvia [ ] Portugal [ ] and Australia [Catley & Tomkinson

(2013)]."(744).

83. The 20-m multistage fitness test is a commonly used maximal running aerobic fitness test used in the Eurofit Physical Fitness Test Battery and the FitnessGram Physical Fitness test. It is also known as the 20-meter shuttle run test, PACER test, or beep test (among other names; this is not the same test as the shuttle run in the Presidential Fitness Test). This test involves continuous running between two lines 20 meters apart in time to recorded beeps. The participants stand behind one of the lines facing the second line and begin running when instructed by the recording. The speed at the start is quite slow. The subject continues running between the two lines, turning when signaled by the recorded beeps. After about one minute, a sound indicates an increase in speed, and the beeps will be closer together. This continues each minute (level). If the line is reached before the beep sounds, the subject must wait until the beep sounds before continuing. If the line is not reached before the beep sounds, the subject is given a warning and must continue to run to the line, then turn and try to catch up with the pace within two more 'beeps'. The subject is given a warning the first time they fail to reach the line (within 2 meters) and eliminated after the second warning.

84. To illustrate the sex-based performance differences observed by Tambalis, I have prepared the following table showing the number of laps completed in the 20 m shuttle run for children ages 6-18 years for the low, middle, and top decile (Tambalis 2016 at 740 & 742), and have calculated the percent difference between the boys and girls using the same equation as Millard-Stafford (2018).

Performance difference between boys and girls ÷ Girls performance

Number of laps completed in the 20m shuttle run for children ages 6-18 years

	Male			Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile
6	4	14	31	4.0	12.0	26.0	0.0%	16.7%	19.2%
7	8	18	38	8.0	15.0	29.0	0.0%	20.0%	31.0%
8	9	23	47	9.0	18.0	34.0	0.0%	27.8%	38.2%
9	11	28	53	10.0	20.0	40.0	10.0%	40.0%	32.5%
10	12	31	58	11.0	23.0	43.0	9.1%	34.8%	34.9%
11	15	36	64	12.0	26.0	48.0	25.0%	38.5%	33.3%
12	15	39	69	12.0	26.0	49.0	25.0%	50.0%	40.8%
13	16	44	76	12.0	26.0	50.0	33.3%	69.2%	52.0%
14	19	50	85	12.0	26.0	50.0	58.3%	92.3%	70.0%
15	20	53	90	12.0	25.0	47.0	66.7%	112.0%	91.5%
16	20	54	90	11.0	24.0	45.0	81.8%	125.0%	100.0%
17	18	50	86	10.0	23.0	50.0	80.0%	117.4%	72.0%
18	13	48	87	8.0	23.0	39.5	62.5%	108.7%	120.3%

85. The Presidential Fitness Test was widely used in schools in the United States from the late 1950s until 2013 (when it was phased out in favor of the Presidential Youth Fitness Program and FitnessGram, both of which focus on health-related physical fitness and do not present data in percentiles). Students participating in the Presidential Fitness Test could receive "The National Physical Fitness Award" for performance equal to the 50th percentile in five areas of the fitness test, "while performance equal to the 85th percentile could receive the Presidential Physical Fitness Award." Tables presenting the 50th and 85th percentiles for the Presidential Fitness Test for males and females ages 6 – 17, and differences in performance

between males and females, for curl-ups, shuttle run, 1 mile run, push-ups, and pull-ups appear in the Appendix.

86. For both the 50th percentile (The National Physical Fitness Award) and the 85th percentile (Presidential Physical Fitness Award), with the exception of curl-ups in 6-year-old children, boys outperform girls. The difference in pull-ups for the 85th percentile for ages 7 through 17 are particularly informative with boys outperforming girls by 100% – 1200%, highlighting the advantages in upper body strength in males.

87. A very recent literature review commissioned by the five United Kingdom governmental Sport Councils concluded that while "[i]t is often assumed that children have similar physical capacity regardless of their sex, . . . large-scale data reports on children from the age of six show that young males have significant advantage in cardiovascular endurance, muscular strength, muscular endurance, speed/agility and power tests," although they "score lower on flexibility tests." (UK Sports Councils' Literature Review 2021 at 3.)

88. Hilton et al., also writing in 2021, reached the same conclusion: "An extensive review of fitness data from over 85,000 Australian children aged 9–17 years old showed that, compared with 9-year-old females, 9-year-old males were faster over short sprints (9.8%) and 1 mile (16.6%), could jump 9.5% further from a standing start (a test of explosive power), could complete 33% more push-ups in 30 [seconds] and had 13.8% stronger grip." (Hilton 2021 at 201, summarizing the findings of Catley & Tomkinson 2013.)

89. The following data are taken from Catley & Tomkinson (2013 at 101) showing the low, middle, and top decile for 1.6 km run (1.0 mile) run time for 11,423 girls and boys ages 9-17.

1.6 km run (1.0 mile) run time for 11,423 girls and boys ages 9-17

	Male			Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile
9	684	522	423	769.0	609.0	499.0	11.1%	14.3%	15.2%
10	666	511	420	759.0	600.0	494.0	12.3%	14.8%	15.0%
11	646	500	416	741.0	586.0	483.0	12.8%	14.7%	13.9%
12	621	485	408	726.0	575.0	474.0	14.5%	15.7%	13.9%
13	587	465	395	716.0	569.0	469.0	18.0%	18.3%	15.8%
14	556	446	382	711.0	567.0	468.0	21.8%	21.3%	18.4%
15	531	432	373	710.0	570.0	469.0	25.2%	24.2%	20.5%
16	514	423	366	710.0	573.0	471.0	27.6%	26.2%	22.3%
17	500	417	362	708.0	575.0	471.0	29.4%	27.5%	23.1%

90. Tomkinson et al. (2018) performed a similarly extensive analysis of literally millions of measurements of a variety of strength and agility metrics from the "Eurofit" test battery on children from 30 European countries. They provide detailed results for each metric, broken out by decile. Sampling the low, middle, and top decile, 9-year-old boys performed better than 9-year-old girls by between 6.5% and 9.7% in the standing broad jump; from 11.4% to 16.1% better in handgrip; and from 45.5% to 49.7% better in the "bent-arm hang." (Tomkinson 2018.)

91. The Bent Arm Hang test is a measure of upper body muscular strength and endurance used in the Eurofit Physical Fitness Test Battery. To perform the Bent Arm Hang, the child is assisted into position with the body lifted to a height so that the chin is level with the horizontal bar (like a pull up bar). The bar is grasped with the palms facing away from body and the hands shoulder width apart. The timing starts when the child is released. The child then attempts to hold this position for as

long as possible. Timing stops when the child's chin falls below the level of the bar, or the head is tilted backward to enable the chin to stay level with the bar.

92. Using data from Tomkinson (2018; table 7 at 1452), the following table sampling the low, middle, and top decile for bent arm hang for 9- to 17-year-old children can be constructed:

Bent Arm Hang time (in seconds) for children ages 9 - 17 years

	Male			Female			Male-Female % Difference		
Age	10th %ile	50th %ile	90th %ile	10th %ile	50th %ile	90th %ile	10th %ile	50th %ile	90th %ile
9	2.13	7.48	25.36	1.43	5.14	16.94	48.95%	45.53%	49.70%
10	2.25	7.92	26.62	1.42	5.15	17.06	58.45%	53.79%	56.04%
11	2.35	8.32	27.73	1.42	5.16	17.18	65.49%	61.24%	61.41%
12	2.48	8.79	28.99	1.41	5.17	17.22	75.89%	70.02%	68.35%
13	2.77	9.81	31.57	1.41	5.18	17.33	96.45%	89.38%	82.17%
14	3.67	12.70	38.39	1.40	5.23	17.83	162.14%	142.83%	115.31%
15	5.40	17.43	47.44	1.38	5.35	18.80	291.30%	225.79%	152.34%
16	7.39	21.75	53.13	1.38	5.63	20.57	435.51%	286.32%	158.29%
17	9.03	24.46	54.66	1.43	6.16	23.61	531.47%	297.08%	131.51%

93. Evaluating these data, a 9-year-old boy in the 50th percentile (that is to say a 9-year-old boy of average upper body muscular strength and endurance) will perform better in the bent arm hang test than 9 through 17-year-old girls in the 50th percentile. Similarly, a 9-year-old boy in the 90th percentile will perform better in the bent arm hang test than 9 through 17-year-old girls in the 90th percentile.

94. Using data from Tomkinson et al. (2017; table 1 at 1549), the following table sampling the low, middle, and top decile for running speed in the last stage of the 20 m shuttle run for 9- to 17-year-old children can be constructed.

20 m shuttle Running speed (km/h at the last completed stage)

	Male			Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile
9	8.94	10.03	11.13	8.82	9.72	10.61	1.36%	3.19%	4.90%
10	8.95	10.13	11.31	8.76	9.75	10.74	2.17%	3.90%	5.31%
11	8.97	10.25	11.53	8.72	9.78	10.85	2.87%	4.81%	6.27%
12	9.05	10.47	11.89	8.69	9.83	10.95	4.14%	6.51%	8.58%
13	9.18	10.73	12.29	8.69	9.86	11.03	5.64%	8.82%	11.42%
14	9.32	10.96	12.61	8.70	9.89	11.07	7.13%	10.82%	13.91%
15	9.42	11.13	12.84	8.70	9.91	11.11	8.28%	12.31%	15.57%
16	9.51	11.27	13.03	8.71	9.93	11.14	9.18%	13.49%	16.97%
17	9.60	11.41	13.23	8.72	9.96	11.09	10.09%	14.56%	19.30%

95. Evaluating these data, a 9-year-old boy in the 50th percentile (that is to say a 9-year-old boy of average running speed) will run faster in the final stage of the 20 m shuttle run than 9 through 17-year-old girls in the 50th percentile. Similarly, a 9-year-old boy in the 90th percentile will run faster in the final stage of the 20-m shuttle run than 9 through 15, and 17-year-old girls in the 90th percentile and will be 0.01 km/h (0.01%) slower than 16-year-old girls in the 90th percentile.

96. Just using these two examples for bent arm hang and 20-m shuttle running speed (Tomkinson 2107, Tomkinson 2018) based on large sample sizes (thus having tremendous statistical power) it becomes apparent that a 9-year-old boy will be very likely to outperform similarly trained girls of his own age and older in athletic events involving upper body muscle strength and/or running speed.

97. Another report published in 2014 analyzed physical fitness measurements of 10,302 children aged 6 -10.9 years of age, from the European countries of Sweden,

Germany, Hungary, Italy, Cyprus, Spain, Belgium, and Estonia. (De Miguel-Etayo et al. 2014.) The authors observed "… that boys performed better than girls in speed, lower- and upper-limb strength and cardiorespiratory fitness." (57) The data showed that for children of comparable fitness (i.e. 99th percentile boys vs. 99th percentile girls, 50th percentile boys vs. 50th percentile girls, etc.) the boys outperform the girls at every age in measurements of handgrip strength, standing long jump, 20-m shuttle run, and predicted VO_2max (pages 63 and 64, respectively). For clarification, VO_2max is the maximal oxygen consumption, which correlates to 30-40% of success in endurance sports.

98. The standing long jump, also called the Broad Jump, is a common and easy to administer test of explosive leg power used in the Eurofit Physical Fitness Test Battery and in the NFL Combine. In the standing long jump, the participant stands behind a line marked on the ground with feet slightly apart. A two-foot take-off and landing is used, with swinging of the arms and bending of the knees to provide forward drive. The participant attempts to jump as far as possible, landing on both feet without falling backwards. The measurement is taken from takeoff line to the nearest point of contact on the landing (back of the heels) with the best of three attempts being scored.

99. Using data from De Miguel-Etayo et al. (2014, table 3 at 61), which analyzed physical fitness measurements of 10,302 children aged 6 -10.9 years of age, from the European countries of Sweden, Germany, Hungary, Italy, Cyprus, Spain, Belgium, and Estonia, the following table sampling the low, middle, and top decile for standing long jump for 6- to 9-year-old children can be constructed:

Standing Broad Jump (cm) for children ages 6-9 years

Male				Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	**%ile**	**%ile**	**%ile**	**%ile**	**%ile**	**%ile**	**%ile**	**%ile**	**%ile**
6-<6.5	77.3	103.0	125.3	69.1	93.8	116.7	11.9%	9.8%	7.4%
6.5-<7	82.1	108.0	130.7	73.6	98.7	121.9	11.5%	9.4%	7.2%
7-<7.5	86.8	113.1	136.2	78.2	103.5	127.0	11.0%	9.3%	7.2%
7.5-<8	91.7	118.2	141.6	82.8	108.3	132.1	10.7%	9.1%	7.2%
8-<8.5	96.5	123.3	146.9	87.5	113.1	137.1	10.3%	9.0%	7.1%
8.5-<9	101.5	128.3	152.2	92.3	118.0	142.1	10.0%	8.7%	7.1%

100. Another study of Eurofit results for over 400,000 Greek children reported similar results. "[C]ompared with 6-year-old females, 6-year-old males completed 16.6% more shuttle runs in a given time and could jump 9.7% further from a standing position." (Hilton 2021 at 201, summarizing findings of Tambalis et al. 2016.)

101. Silverman (2011) gathered hand grip data, broken out by age and sex, from a number of studies. Looking only at the nine direct comparisons within individual studies tabulated by Silverman for children aged 7 or younger, in eight of these the boys had strength advantages of between 13 and 28 percent, with the remaining outlier recording only a 4% advantage for 7-year-old boys. (Silverman 2011 Table 1.)

102. To help illustrate the importance of one specific measure of physical fitness in athletic performance, Pocek (2021) stated that to be successful, volleyball "players should distinguish themselves, besides in skill level, in terms of above-average body height, upper and lower muscular power, speed, and agility. Vertical jump is a fundamental part of the spike, block, and serve." (8377) Pocek further

stated that "relative vertical jumping ability is of great importance in volleyball regardless of the players' position, while absolute vertical jump values can differentiate players not only in terms of player position and performance level but in their career trajectories." (8382)

103. Using data from Ramírez-Vélez (2017; table 2 at 994) which analyzed vertical jump measurements of 7,614 healthy Colombian schoolchildren aged 9 - 17.9 years of age the following table sampling the low, middle, and top decile for vertical jump can be constructed:

Vertical Jump Height (cm) for children ages 9 - 17 years

	Male			Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile
9	18.0	24.0	29.5	16.0	22.3	29.0	12.5%	7.6%	1.7%
10	19.5	25.0	32.0	18.0	24.0	29.5	8.3%	4.2%	8.5%
11	21.0	27.0	32.5	19.5	25.0	31.0	7.7%	8.0%	4.8%
12	22.0	27.5	34.5	20.0	25.5	31.5	10.0%	7.8%	9.5%
13	23.0	30.5	39.0	19.0	25.5	32.0	21.1%	19.6%	21.9%
14	23.5	32.0	41.5	20.0	25.5	32.5	17.5%	25.5%	27.7%
15	26.0	35.5	43.0	20.2	26.0	32.5	28.7%	36.5%	32.3%
16	28.0	36.5	45.1	20.5	26.5	33.0	36.6%	37.7%	36.7%
17	28.0	38.0	47.0	21.5	27.0	35.0	30.2%	40.7%	34.3%

104. Similarly, using data from Taylor (2010; table 2, at 869) which analyzed vertical jump measurements of 1,845 children aged 10 -15 years in primary and secondary schools in the East of England, the following table sampling the low, middle, and top decile for vertical jump can be constructed:

Vertical Jump Height (cm) for children 10 -15 years

	Male			Female			Male-Female % Difference		
	10th	50th	90th	10th	50th	90th	10th	50th	90th
Age	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile	%ile
10	16.00	21.00	29.00	15.00	22.00	27.00	6.7%	-4.5%	7.4%
11	20.00	27.00	34.00	19.00	25.00	32.00	5.3%	8.0%	6.3%
12	23.00	30.00	37.00	21.00	27.00	33.00	9.5%	11.1%	12.1%
13	23.00	32.00	40.00	21.00	26.00	34.00	9.5%	23.1%	17.6%
14	26.00	36.00	44.00	21.00	28.00	34.00	23.8%	28.6%	29.4%
15	29.00	37.00	44.00	21.00	28.00	39.00	38.1%	32.1%	12.8%

105. As can be seen from the data from Ramírez-Vélez (2017) and Taylor (2010), males consistently outperform females of the same age and percentile in vertical jump height. Both sets of data show that an 11-year-old boy in the 90th percentile for vertical jump height will outperform girls in the 90th percentile at ages 11 and 12, and will be equal to girls at ages 13, 14, and possibly 15. These data indicate that an 11-year-old would be likely to have an advantage over girls of the same age and older in sports such as volleyball where "absolute vertical jump values can differentiate players not only in terms of player position and performance level but in their career trajectories." (Pocek 2021 at 8382.)

106. Boys also enjoy an advantage in throwing well before puberty. "Boys exceed girls in throwing velocity by 1.5 standard deviation units as early as 4 to 7 years of age. . . The boys exceed the girls [in throwing distance] by 1.5 standard deviation units as early as 2 to 4 years of age." (Thomas 1985 at 266.) This means that the average 4- to 7-year-old boy can out-throw approximately 87% of all girls of his age.

107. Record data from USA Track & Field indicate that boys outperform girls in

track events even in the youngest age group for whom records are kept (age 8 and under).[8]

American Youth Outdoor Track & Field Record times in age groups 8 and under (time in seconds)

Event	**Boys**	**Girls**	**Difference**
100M	13.65	13.78	0.95%
200M	27.32	28.21	3.26%
400M	62.48	66.10	5.79%
800M	148.59	158.11	6.41%
1500M	308.52	314.72	2.01%
Mean			3.68%

108. Looking at the best times within a single year shows a similar pattern of consistent advantage for even young boys. I consider the 2018 USATF Region 8 Junior Olympic Championships for the youngest age group (8 and under).[9]

2018 USATF Region 8 Junior Olympic Championships for the 8 and under age group

Event	**Boys**	**Girls**	**Difference**
100M	15.11	15.64	3.51%
200M	30.79	33.58	9.06%
400M	71.12	77.32	8.72%
800M	174.28	180.48	3.56%
1500M	351.43	382.47	8.83%
Mean			6.74%

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[8] http://legacy.usatf.org/statistics/records/view.asp?division=american&location=outdoor%20track%20%26%20field&age=youth&sport=TF

[9] https://www.athletic.net/TrackAndField/meet/384619/results/m/1/100m

[9] https://www.athletic.net/CrossCountry/Division/List.aspx?DivID=62211

109. Using Athletic.net[9], for 2021 Cross Country and Track & Field data for boys and girls in the 7-8, 9-10, and 11-12 year old age group club reports, and for 5th, 6th, and 7th grade for the whole United States I have compiled the tables for 3000 m events, and for the 100-m, 200-m, 400-m, 800-m, 1600-m, 3000-m, long jump, and high jump Track and Field data to illustrate the differences in individual athletic performance between boys and girls, all of which appear in the Appendix. The pattern of males outperforming females was consistent across events, with rare anomalies, only varying in the magnitude of difference between males and females.

110. Similarly, using Athletic.net, for 2022 Track & Field data for boys and girls in the 6th grade for the state of Arizona, I have compiled tables, which appear below, comparing the performance of boys and girls for the 100-m, 200-m, 400-m, 800-m, 1600-m, and 3200-m running events in which the 1st place boy was consistently faster than the 1st place girl (with the exception of the 1600-m in which the first place girl was 0.9% faster) and the average performance of the top 10 boys was consistently faster than the average performance for the top 10 girls. Based on the finishing times for the 1st place boy and the 1st place girl in the 6th grade in Arizona in the 400-m race, the boy was 7.1 seconds (10.9%) faster than the girl. Extrapolating the running time to a running pace, the boy would be expected to finish 49 m in front of the fastest girl in a single lap race on a standard 400-m track, or almost the length of ½ of a football field. In comparison, the 1st place boy would finish 8 m in front of the 2nd place boy, and the 1st place girl would finish 10 m in front of the 2nd place girl.

Top 10 Arizona boys and girls 6th grade outdoor track for 2022 (time in seconds)

	100 m			200 m			400 m		
	Boys	**Girls**		**Boys**	**Girls**		**Boys**	**Girls**	
1	12.60	12.71	Difference	25.53	26.01	Difference	58.40	65.54	Difference
2	13.14	13.44	between #1	26.84	28.20	between #1	59.59	67.04	between #1
3	13.35	13.60	boy and # 1	27.30	28.77	boy and # 1	61.74	68.27	boy and # 1
4	13.44	14.14	girl	27.44	29.10	girl	62.32	68.64	girl
5	13.44	14.15	0.9%	28.61	29.52	1.8%	63.14	69.87	10.9%
6	13.47	14.4		28.68	30.06		66.38	70.12	
7	13.54	14.41	Average	29.04	30.15	Average	66.46	80.22	Average
8	13.59	14.44	difference	29.14	30.17	difference	66.50	70.73	difference
9	13.78	14.50	boys vs girls	29.17	30.19	boys vs girls	67.35	72.09	boys vs girls
10	13.84	14.53	4.4%	29.59	30.34	3.8%	67.36	72.43	9.3%

	800 m			1600 m			3200 m		
	Boys	**Girls**		**Boys**	**Girls**		**Boys**	**Girls**	
1	146.67	154.55	Difference	333.71	331.01	Difference	793.27	835.76	Difference
2	149.47	157.70	between #1	335.23	340.22	between #1	816.60	904.96	between #1
3	150.70	159.31	boy and # 1	338.70	351.70	boy and # 1	818.87	947.81	boy and # 1
4	151.29	165.49	girl	340.97	360.44	girl	840.17	1064.43	girl
5	152.56	167.00	5.1%	344.90	362.47	-0.9%	842.58	1090.2	5.1%
6	153.70	169.89		350.19	369.10		859.92		
7	158.30	170.00	Average	352.20	371.88	Average	861.74		Average
8	158.45	172.40	difference	360.30	375.66	difference	866.30		difference
9	158.70	173.64	boys vs girls	361.31	382.29	boys vs girls	Only 8	Only 5	boys vs girls
10	159.83	173.90	7.5%	364.00	384.00	4.1%	times	times	13.5%
							listed	listed	

111. As serious runners will recognize, differences of 3%, 5%, or 8% are not easily overcome. During track competition the difference between first and second place, or second and third place, or third and fourth place (and so on) is often 0.5 - 0.7%, with some contests being determined by as little as 0.01%.

112. I performed an analysis of running events (consisting of the 100-m, 200-m, 400-m, 800-m, 1500-m, 5000-m, and 10,000-m) in the Division I, Division II, and Division III NCAA Outdoor championships for the years of 2010-2019: the mean difference between 1st and 2nd place was 0.48% for men and 0.86% for women. The mean difference between 2nd and 3rd place was 0.46% for men and 0.57% for women. The mean difference between 3rd place and 4th place was 0.31% for men and 0.44% for women. The mean difference between 1st place and 8th place (the last place to earn the title of All American) was 2.65% for men and 3.77% for women. (Brown et al. Unpublished observations, presented at the 2022 Annual Meeting of the American College of Sports Medicine.)

113. A common response to empirical data showing pre-pubertal performance advantages in boys is the argument that the performance of boys may represent a social–cultural bias for boys to be more physically active, rather than representing inherent sex-based differences in pre-pubertal physical fitness. However, the younger the age at which such differences are observed, and the more egalitarian the culture within which they are observed, the less plausible this hypothesis becomes. Eiberg et al. (2005) measured body composition, VO_2max, and physical activity in 366 Danish boys and 332 Danish girls between the ages of 6 and 7 years old. Their observations indicated that VO_2max was 11% higher in boys than girls. When expressed relative to body mass the boys' VO_2max was still 8% higher than the girls. The authors stated that "…no differences in haemoglobin or sex hormones[10] have been reported in this age group," yet "… when children with the

[10] This term would include testosterone and estrogens.

same VO_2max were compared, boys were still more active, and in boys and girls with the same P[hysical] A[ctivity] level, boys were fitter." (728). These data indicate that in pre-pubertal children, in a very egalitarian culture regarding gender roles and gender norms, boys still have a measurable advantage in regards to aerobic fitness when known physiological and physical activity differences are accounted for.

114. And, as I have mentioned above, even by the age of 4 or 5, in a ruler-drop test, boys exhibit 4% to 6% faster reaction times than girls. (Latorre-Roman 2018.)

115. When looking at the data on testosterone concentrations previously presented, along with the data on physical fitness and athletic performance presented, boys have advantages in athletic performance and physical fitness before there are marked differences in testosterone concentrations between boys and girls.

116. For the most part, the data I review above relate to pre-pubertal children. Today, we also face the question of inclusion in female athletics of males who have undergone "puberty suppression." The UK Sport Councils Literature Review notes that, "In the UK, so-called 'puberty blockers' are generally not used until Tanner maturation stage 2-3 (i.e. after puberty has progressed into early sexual maturation)." (9.) While it is outside my expertise, my understanding is that current practice with regard to administration of puberty blockers is similar in the United States. Tanner stages 2 and 3 generally encompass an age range from 10 to 14 years old, with significant differences between individuals. Like the authors of the UK Sports Council Literature Review, I am "not aware of research" directly addressing the implications for athletic capability of the use of puberty blockers. (UK Sport Councils Literature Review at 9.) As Handelsman documents, the male advantage begins to increase rapidly–along with testosterone levels–at about age 11, or "very closely aligned to the timing of the onset of male puberty." (Handelsman 2017.) It seems likely that males who have undergone puberty suppression will have physiological and performance advantages over females somewhere between those

possessed by pre-pubertal boys, and those who have gone through full male puberty, with the degree of advantage in individual cases depending on that individual's development and the timing of the start of puberty blockade.

117. Tack et al. (2018) observed that in 21 transgender-identifying biological males, administration of antiandrogens for 5-31 months (commencing at 16.3 ± 1.21 years of age), resulted in nearly, but not completely, halting of normal age-related *increases* in muscle strength. Importantly, muscle strength did not decrease after administration of antiandrogens. Rather, despite antiandrogens, these individuals retained higher muscle mass, lower percent body fat, higher body mass, higher body height, and higher grip strength than comparable girls of the same age. (Supplemental tables).

118. Klaver et al. (2018 at 256) demonstrated that the use of puberty blockers did not eliminate the differences in lean body mass between biological male and female teenagers. Subsequent use of puberty blockers combined with cross-sex hormone use (in the same subjects) still did not eliminate the differences in lean body mass between biological male and female teenagers. Furthermore, by 22 years of age, the use of puberty blockers, and then puberty blockers combined with cross sex hormones, and then cross hormone therapy alone for over 8 total years of treatment still had not eliminated the difference in lean body mass between biological males and females.

119. Nokoff et al. (2021) observed that teenage natal males who identified as female, (average of 13.7 ± 1.7 years) and who were on puberty blockers for an average of 11.3 ± 7 months, had numerically higher percent lean body mass and lower percent body fat than the comparison group of natal females (figure 1 at 116). (These authors did not statistically compare the natal males who identified as female to the natal females).

120. Navabi et al. (2021) observed that teenage natal males who identify as female (average of 15.4 ± 2.0 years), had 9.5 kg more lean body mass than did teenage natal

females (15.2 ± 1.8 years) who identified as male (at 4). After 355.2 ± 96.7 days of puberty blockers the natal males who identified as female still had 5.7 kg more lean body mass than did the natal females who identified as male (at 5). It is worth noting that the natal males lost 2.57 kg lean body mass and the natal females gained 1.21 kg lean body mass.

121. Nokoff et al. (2020) observed that in 14 teenage natal males who identified as female (average of 16.3 ± 1.4 years) and "were taking an average estradiol dose of 1.5 ± 1.0 mg/day with an average treatment duration of 12.3 ± 9.9 months (5 on oral, 9 on sublingual). Four were on a GnRHa at the time of the study visit and a total of 6 had been on a GnRHa in the past. Seven were on spironolactone for androgen blockade and 1 was on IM medroxyprogesterone acetate for puberty suppression." (at e707) the natal males had higher lean body mass and lower body fat than the comparison group of natal females (at e708).

122. The effects of puberty blockers on growth and development, including muscle mass, fat mass, or other factors that influence athletic performance, have been minimally researched. As stated by Roberts and Carswell (2021), "No published studies have fully characterized the impact of [puberty blockers on] final adult height or current height in an actively growing TGD youth." (1680). Likewise, "[n]o published literature provides guidance on how to best predict the final adult height for TGD youth receiving GnRHa and gender- affirming hormonal treatment." (1681). Thus, the effect of prescribing puberty blockers to a male child before the onset of puberty on the physical components of athletic performance is largely unknown. There is not any scientific evidence that such treatment eliminates the pre-existing performance advantages that prepubertal males have over prepubertal females.

123. Schulmeister et al. (2022) evaluated natal males with an average age of 11.9 (range 10.2 – 14.5) years at the start of puberty blockade and concluded that "youth treated with GnRHa for 12 months have growth rates similar to those of prepubertal

youth" (at 5).

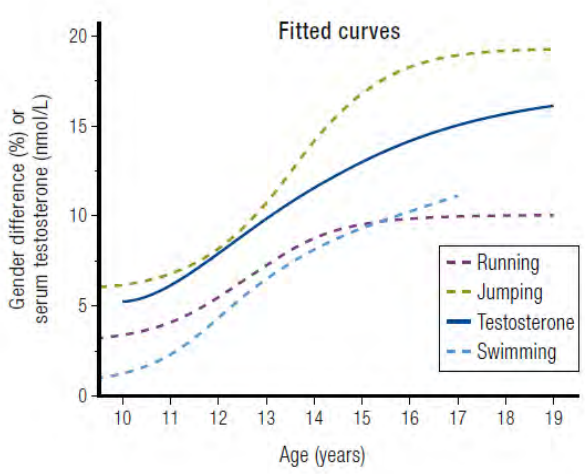
124. In Boogers et al. (2022), the researchers studied the effects of puberty suppression followed by cross-sex hormone therapy on the adult height of natal males who identify as female. Analyzing retrospective data collected from 1972 to 2018, they concluded that "although P[uberty] S[upression] and [cross-sex hormones] alter the growth pattern, they have little effect on adult height." (9) In other words, natal males who followed a normal course of puberty suppression followed by cross-sex hormone therapy reached an adult height at or near their predicted height in the absence of such therapy.

125. The findings from Schulmeister et al. (2022) and Boogers et al. (2022) are relevant to the question of whether puberty suppression eliminates sex-based performance advantages because these finding provide evidence that an important component of that advantage - male vs. female height - is not eliminated, or even meaningfully affected, by an ordinary course of puberty suppression or puberty suppression followed by cross-sex hormone therapy.

B. The rapid increase in testosterone across male puberty drives characteristic male physiological changes and the increasing performance advantages.

126. While boys exhibit some performance advantage even before puberty, it is both true and well known to common experience that the male advantage increases rapidly, and becomes much larger, as boys undergo puberty and become men. Empirically, this can be seen by contrasting the modest advantages reviewed immediately above against the large performance advantages enjoyed by men that I have detailed in Section II.

127. Multiple studies (along with common observation) document that the male performance advantage begins to increase during the early years of puberty, and then increases rapidly across the middle years of puberty (about ages 12-16). (Tønnessen 2015; Handelsman 2018 at 812-813.) Since it is well known that testosterone levels increase by more than an order of magnitude in boys across puberty, it is unsurprising that Handelsman finds that these increases in male performance advantage correlate to increasing testosterone levels, as presented in his chart reproduced below. (Handelsman 2018 at 812-13.)



128. Handelsman further finds that certain characteristic male changes including boys' increase in muscle mass do not begin at all until "circulating testosterone concentrations rise into the range of males at mid-puberty, which are higher than in women at any age." (Handelsman 2018 at 810.)

129. Knox et al. (2019) agree that "[i]t is well recognised that testosterone contributes to physiological factors including body composition, skeletal structure, and the cardiovascular and respiratory systems across the life span, with significant influence during the pubertal period. These physiological factors underpin strength, speed, and recovery with all three elements required to be competitive in almost all sports." (Knox 2019 at 397.) "High testosterone levels and prior male physiology provide an all-purpose benefit, and a substantial advantage. As the IAAF says, 'To

the best of our knowledge, there is no other genetic or biological trait encountered in female athletics that confers such a huge performance advantage.'" (Knox 2019 at 399.)

130. However, the undisputed fact that high (that is, normal male) levels of testosterone drive the characteristically male physiological changes that occur across male puberty does not at all imply that artificially *depressing* testosterone levels after those changes occur will reverse all or most of those changes so as to eliminate the male athletic advantage. This is an empirical question. As it turns out, the answer is that while some normal male characteristics can be changed by means of testosterone suppression, others cannot be, and all the reliable evidence indicates that males retain large athletic advantages even after long-term testosterone suppression.

V. The available evidence shows that suppression of testosterone in a male after puberty has occurred does <u>not</u> substantially eliminate the male athletic advantage.

131. The 2011 "NCAA Policy on Transgender Student-Athlete Participation" requires only that males who identify as transgender be on unspecified and unquantified "testosterone suppression treatment" for "one calendar year" prior to competing in women's events. In supposed justification of this policy, the NCAA's Office of Inclusion asserts that, "It is also important to know that any strength and endurance advantages a transgender woman arguably may have as a result of her prior testosterone levels dissipate after about one year of estrogen or testosterone-suppression therapy." (NCAA 2011 at 8.)

132. Similarly, writing in 2018, Handelsman et al. could speculate that even though some male advantages established during puberty are "fixed and irreversible (bone size)," "[t]he limited available prospective evidence . . . suggests that the advantageous increases in muscle and hemoglobin due to male circulating testosterone concentrations are induced or reversed during the first 12 months."

(Handelsman 2018 at 824.)

133. But these assertions or hypotheses of the NCAA and Handelsman are now strongly contradicted by the available science. In this section, I examine what is known about whether suppression of testosterone in males can eliminate the male physiological and performance advantages over females.

A. Empirical studies find that males retain a strong performance advantage even after lengthy testosterone suppression.

134. As my review in Section II indicates, a very large body of literature documents the large performance advantage enjoyed by males across a wide range of athletics. To date, only a limited number of studies have directly measured the effect of testosterone suppression and the administration of female hormones on the athletic performance of males. These studies report that testosterone suppression for a full year (and in some cases much longer) does not come close to eliminating male advantage in strength (hand grip, leg strength, and arm strength) or running speed.

Hand Grip Strength

135. As I have noted, hand grip strength is a well-accepted proxy for general strength. Multiple separate studies, from separate groups, report that males retain a large advantage in hand strength even after testosterone suppression to female levels.

136. In a longitudinal study, Van Caenegem et al. reported that males who underwent standard testosterone suppression protocols lost only 7% hand strength after 12 months of treatment, and only a cumulative 9% after two years. (Van Caenegem 2015 at 42.) As I note above, on average men exhibit in the neighborhood of 60% greater hand grip strength than women, so these small decreases do not remotely eliminate that advantage. Van Caenegem et al. document that their sample of males who elected testosterone suppression began with less strength than a control male population. Nevertheless, after one year of suppression, their study population still had hand grip only 21% less than the control male population, and

thus still far higher than a female population. (Van Caenegem 2015 at 42.)

137. Scharff et al. (2019) measured grip strength in a large cohort of male-to-female subjects from before the start of hormone therapy through one year of hormone therapy. The hormone therapy included suppression of testosterone to less than 2 nml/L "in the majority of the transwomen," (1024), as well as administration of estradiol (1021). These researchers observed a small decrease in grip strength in these subjects over that time (Fig. 2), but mean grip strength of this group remained far higher than mean grip strength of females—specifically, "After 12 months, the median grip strength of transwomen [male-to-female subjects] still falls in the 95th percentile for age-matched females." (1026).

138. Still a third longitudinal study, looking at teen males undergoing testosterone suppression, "noted no change in grip strength after hormonal treatment (average duration 11 months) of 21 transgender girls." (Hilton 2021 at 207, summarizing Tack 2018.)

139. A fourth study (Auer et al. 2016) reported no change in handgrip strength in 13 transwomen below the age of 45 years following 12 months of cross sex hormone therapy (Table 1, at 3).

140. A fifth study (Yun et al. 2021) observed that handgrip strength in the right hand decreased from 31.5 ± 5.8 kg to 29.9 ± 7.4 kg and in the left hand decreased from 31.8 ± 6.5 kg to 30.1 ± 6.9 kg during 6 months of cross sex hormone therapy in 11 males aged 28.5 ± 8.1 years who identify as women or nonbinary (Table 4, at 63). It is worth noting that the reduced grip strength in these male bodied individuals would rate in 75th percentile for females (Liguri, at 95).

141. Lapauw et al. (2008) looked at the extreme case of testosterone suppression by studying a population of 23 biologically male individuals who had undergone at least two years of testosterone suppression, followed by sex reassignment surgery that included "orchidectomy" (that is, surgical castration), and then at least an additional three years before the study date. Comparing this group against a control

of age- and height-matched healthy males, the researchers found that the individuals who had gone through testosterone suppression and then surgical castration had an average hand grip (41 kg) that was 24% weaker than the control group of healthy males. But this remains at least 25% *higher* than the average hand-grip strength of biological females as measured by Bohannon et al. (2019).

142. Alvares et al (2022) is a cross-sectional study on cardiopulmonary capacity and muscle strength in biological males who identify as female and have undergone long-term cross-sex hormone therapy. All of the study subjects that were biological males who identify as female had testosterone suppressed through medication (cyproterone acetate) or gonadectomy. (Supplementary materials) And they had taken exogenous estrogen for an average of 14.4 years with a standard deviation of 3.5 years. Compared to a control group of cisgender women, the study subjects exhibited 18% higher handgrip strength, confirming the findings of previous studies but extending the information to a longer time period. It is worth noting that the grip strength in these male bodied individuals would rate between the 90th and 95th percentile for females (Liguri, at 95).

143. Summarizing these and a few other studies measuring strength loss (in most cases based on hand grip) following testosterone suppression, Harper et al. (2021) conclude that "strength loss with 12 months of [testosterone suppression] . . . ranged from non-significant to 7%. . . . [T]he small decrease in strength in transwomen after 12-36 months of [testosterone suppression] suggests that transwomen likely retain a strength advantage over cisgender women." (Hilton 2021 at 870.)

Arm Strength

144. Lapauw et al. (2008) found that 3 years after surgical castration, preceded by at least two years of testosterone suppression, biologically male subjects had 33% less bicep strength than healthy male controls. (Lapauw (2008) at 1018.) Given that healthy men exhibit between 89% and 109% greater arm strength than healthy women, this leaves a very large residual arm strength advantage over biological

women.

145. Roberts et al. have published an interesting longitudinal study, one arm of which considered biological males who began testosterone suppression and cross-sex hormones while serving in the United States Air Force. (Roberts 2020.) One measured performance criterion was pushups per minute, which, while not exclusively, primarily tests arm strength under repetition. *Before* treatment, the biological male study subjects who underwent testosterone suppression could do 45% more pushups per minute than the average for all Air Force women under the age of 30 (47.3 vs. 32.5). *After* between one and two years of testosterone suppression, this group could still do 33% more pushups per minute. (Table 4.) Further, the body weight of the study group did not decline at all after one to two years of testosterone suppression (in fact rose slightly) (Table 3), and was approximately 24 pounds (11.0 kg) higher than the average for Air Force women under the age of 30. (Roberts 2020 at 3.) This means that the individuals who had undergone at least one year of testosterone suppression were not only doing 1/3 more pushups per minute, but were lifting significantly more weight with each pushup.

146. After two years of testosterone suppression, the study sample in Roberts et al. was only able to do 6% more pushups per minute than the Air Force female average. But their weight remained unchanged from their pre-treatment starting point, and thus about 24 pounds higher than the Air Force female average. As Roberts et al. explain, "as a group, transwomen weigh more than CW [cis-women]. Thus, transwomen will have a higher power output than CW when performing an equivalent number of push-ups. Therefore, our study may underestimate the advantage in strength that transwomen have over CW." (Roberts 2020 at 4.)

147. Chiccarelli et al. (2022) also published a longitudinal study which considered biological males who began testosterone suppression and cross-sex hormones while serving in the United States Air Force and concluded "Transgender females'

performance … remained superior in push-ups at the study's 4-year endpoint." (at 1) with the transwomen completing 16% more pushups than comparable women after 4 years of GAHT.

148. It is interesting that Roberts et al. (2020) and Chiccarelli et al. (2022) were comparing the same performance measurements in the same population and came to differing conclusions, which may be due to different sample sizes and study durations

Leg Strength

149. Wiik et al. (2020), in a longitudinal study that tracked 11 males from the start of testosterone suppression through 12 months after treatment initiation, found that isometric strength levels measured at the knee "were maintained over the [study period]."[11] (808) "At T12 [the conclusion of the one-year study], the absolute levels of strength and muscle volume were greater in [male-to-female subjects] than in . . . CW [women who had not undergone any hormonal therapy]." (Wiik 2020 at 808.) In fact, Wiik et al. reported that "muscle strength after 12 months of testosterone suppression was comparable to baseline strength. As a result, transgender women remained about 50% stronger than . . . a reference group of females." (Hilton 2021 at 207, summarizing Wiik 2020.)

150. Lapauw et al. (2008) found that 3 years after surgical castration, preceded by at least two years of testosterone suppression, subjects had peak knee torque only 25% lower than healthy male controls. (Lapauw 2008 at 1018.) Again, given that healthy males exhibit 54% greater maximum knee torque than healthy females, this leaves these individuals with a large average strength advantage over females even years after sex reassignment surgery.

Running and Swimming speed

151. The most striking finding of the recent Roberts et al. study concerned running

[11] Isometric strength measures muscular force production for a given amount of time at a specific joint angle but with no joint movement.

speed over a 1.5 mile distance–a distance that tests midrange endurance. Before suppression, the MtF study group ran 21% faster than the Air Force female average. After at least 2 year of testosterone suppression, these subjects still ran 12% faster than the Air Force female average. (Roberts 2020 Table 4.)

152. Chiccarelli (2022) reported that "Transgender females' performance showed statistically significantly better performance than cisgender females until 2 years of GAHT in run times…" (at 1) and yet the 1.5 mile run time was, on average, 45 seconds (5%) faster in the transwomen at years 2 and 3 than the Air Force female average.

153. The specific experience of the well-known case of NCAA athlete Cece Telfer is consistent with the more statistically meaningful results of Roberts et al., further illustrating that male-to-female transgender treatment does not negate the inherent athletic performance advantages of a post-pubertal male. In 2016 and 2017 Cece Telfer competed as Craig Telfer on the Franklin Pierce University men's track team, being ranked 200th and 390th (respectively) against other NCAA Division II men. "Craig" Telfer did not qualify for the National Championships in any events. Telfer did not compete in the 2018 season while undergoing testosterone suppression (per NCAA policy). In 2019 Cece Telfer competed on the Franklin Pierce University *women's* team, qualified for the NCAA Division II Track and Field National Championships, and placed 1st in the women's 400 meter hurdles and placed third in the women's 100 meter hurdles. (For examples of the media coverage of this please see https://www.washingtontimes.com/news/2019/jun/3/cece-telfer-franklin-pierce-transgender-hurdler-wi/ (last accessed May 5, 2023). https://triblive.com/sports/biological-male-wins-ncaa-womens-track-championship/ (last accessed May 25, 2023.)

154. The table below shows the best collegiate performance times from the combined 2015 and 2016 seasons for Cece Telfer when competing as a man in men's events, and the best collegiate performance times from the 2019 season when

competing as a woman in women's events. Comparing the times for the running events (in which male and female athletes run the same distance) there is no statistical difference between Telfer's "before and after" times. Calculating the difference in time between the male and female times, Telfer performed an average of 0.22% *faster* as a female. (Comparing the performance for the hurdle events (marked with H) is of questionable validity due to differences between men's and women's events in hurdle heights and spacing, and distance for the 110m vs. 100 m.) While this is simply one example, and does not represent a controlled experimental analysis, this information provides some evidence that male-to-female transgender treatment does not negate the inherent athletic performance advantages of a postpubertal male. (These times were obtained from https://www.tfrrs.org/athletes/6994616/Franklin_Pierce/CeCe_Telfer.html and https://www.tfrrs.org/athletes/5108308.html, last accessed May 5, 2023).

As Craig Telfer (male athlete)		**As Cece Telfer (female athlete)**	
Event	Time (seconds)	Event	Time (seconds)
55	7.01	55	7.02
60	7.67	60	7.63
100	12.17	100	12.24
200	24.03	200	24.30
400	55.77	400	54.41
55 H †	7.98	55 H†	7.91
60 H †	8.52	60 H†	8.33
110 H†	15.17	100 H†	13.41*
400 H‡	57.34	400 H‡	57.53**

* women's 3rd place, NCAA Division 2 National Championships

** women's 1st place, NCAA Division 2 National Championships

† men's hurdle height is 42 inches with differences in hurdle spacing between men and women

‡ men's hurdle height is 36 inches, women's height is 30 inches with the same spacing between hurdles

155. Harper (2015) has often been cited as "proving" that testosterone suppression eliminates male advantage. And indeed, hedged with many disclaimers, the author in that article does more or less make that claim with respect to "distance races," while emphasizing that "the author makes no claims as to the equality of performances, pre and post gender transition, in any other sport." (Harper 2015 at 8.) However, Harper (2015) is in effect a collection of unverified anecdotes, not science. It is built around self-reported race times from just eight self-selected transgender runners, recruited "mostly" online. How and on what websites the subjects were recruited is not disclosed, nor is anything said about how those not recruited online were recruited. Thus, there is no information to tell us whether these eight runners could in any way be representative, and the recruitment pools and methodology, which could bear on ideological bias in their self-reports, is not disclosed.

156. Further, the self-reported race times relied on by Harper (2015) *span 29 years*. It is well known that self-reported data, particularly concerning emotionally or ideologically fraught topics, is unreliable, and likewise that memory of distant events is unreliable. Whether the subjects were responding from memory or from written records, and if so what records, is not disclosed, and does not appear to be known to the author. For six of the subjects, the author claims to have been able to verify "approximately half" of the self-reported times. Which scores these are is not disclosed. The other two subjects responded only anonymously, so nothing about their claims could be or was verified. In short, neither the author nor the reader knows whether the supposed "facts" on which the paper's analysis is based are true.

157. Even if we could accept them at face value, the data are largely meaningless. Only two of the eight study subjects reported (undefined) "stable training patterns," and even with consistent training, athletic performance generally declines with age.

As a result, when the few data points span 29 years, it is not possible to attribute declines in performance to asserted testosterone suppression. Further, distance running is usually not on a track, and race times vary significantly depending on the course and the weather. Only one reporting subject who claimed a "stable training pattern" reported "before and after" times on the same course within three years' time," which the author acknowledges would "represent the best comparison points."

158. Harper (2015) to some extent acknowledges its profound methodological flaws, but seeks to excuse them by the difficulty of breaking new ground. The author states that, "The first problem is how to formulate a study to create a meaningful measurement of athletic performance, both before and after testosterone suppression. No methodology has been previously devised to make meaningful measurements." (2) This statement was not accurate at the time of publication, as there are innumerable publications with validated methodology for comparing physical fitness and/or athletic performance between people of different ages, sexes, and before and after medical treatment, any of which could easily have been used with minimal or no adaptation for the purposes of this study. Indeed, well before the publication of Harper (2015), several authors that I have cited in this review had performed and published disciplined and methodologically reliable studies of physical performance and physiological attributes "before and after" testosterone suppression.

159. More recently, and to her credit, Harper has acknowledged the finding of Roberts (2020) regarding the durable male advantage in running speed in the 1.5 mile distance, even after two years of testosterone suppression. She joins with co-authors in acknowledging that this study of individuals who (due to Air Force physical fitness requirements) "could at least be considered exercise trained," agrees that Roberts' data shows that "transwomen ran significantly faster during the 1.5 mile fitness test than ciswomen," and declares that this result is "consistent with the

findings of the current review in untrained transgender individuals" that even 30 months of testosterone suppression does not eliminate all male advantages "associated with muscle endurance and performance." (Harper 2021 at 8.) The Harper (2021) authors conclude overall "that strength may be well preserved in transwomen during the first 3 years of hormone therapy," and that [w]hether transgender and cisgender women can engage in meaningful sport [in competition with each other], even after [testosterone suppression], is a highly debated question." (Harper 2021 at 1, 8.)

160. Higerd (2021) "[a]ssess[ed] the probability of a girls' champion being biologically male" by evaluating 920,11 American high school track and field performances available through the track and field database Athletic.net in five states (CA, FL, MN, NY, WA), over three years (2017 – 2019),in eight events; high jump, long jump, 100M, 200M, 400M, 800M, 1600M, and 3200M and estimated that "there is a simulated 81%-98% probability of transgender dominance occurring in the female track and field event" and further concluded that "in the majority of cases, the entire podium (top of the state) would be MTF [transgender athletes]" (at xii).

161. The well-publicized case of Lia Thomas is also worth noting. University of Pennsylvania swimmer Lia Thomas began competing in the women's division in the fall of 2021, after previously competing for U. Penn. in the men's division. Thomas has promptly set school, pool, and/or league women's records in 200-yard freestyle, 500 yard freestyle, and 1650 yard freestyle competitions, beating the nearest female in the 1650 yard by an unheard-of 38 seconds.

162. Senefeld et al. (2023) compared "the performance times of a transgender woman (male sex, female gender identity) who competed in both men's and women's NCAA freestyle swimming and contextualized her performances relative to the performances of both world class and contemporary NCAA swimmers" (at 1035) and observed that this athlete [presumably Lia Thomas based on performance

times and the timing of this article] was unranked in 2018-2019 in the 100-yard, ranked 551st in the 200-yard, 65th in the 500-yard 32nd in the 1650-yards men's freestyle. After following the NCAA protocol for testosterone suppression and competing as a woman in 2021-2022, this swimmer was ranked 13th in the 100-yard, 3rd in the 200-yard, 1st in the 500-yard, and 13th in the 1650-yard women's freestyle. The performance times swimming as a female, when compared to swimming as a male, were 0.5% slower in the 100-yard, 2.6% slower in the 200-yard, 5.6% slower in the 500-yard, and 7.3% slower in the 1650-yard events than when swimming as a male (at 1034). The authors concluded "…these data suggest there may be a prolonged "legacy effect" (greater than 2 yr) associated with endogenous male testosterone concentrations or male puberty on freestyle swimming performances after feminizing GAHT, particularly for shorter event distances (100, 200, and 500 yards), which are closely associated with anthropometrics and maximal skeletal muscle strength and power" (at 1036).

B. Testosterone suppression does not reverse important male physiological advantages.

163. We see that, once a male has gone through male puberty, later testosterone suppression (or even castration) leaves large strength and performance advantages over females in place. It is not surprising that this is so. What is now a fairly extensive body of literature has documented that many of the specific male physiological advantages that I reviewed in Section II are not reversed by testosterone suppression after puberty, or are reduced only modestly, leaving a large advantage over female norms still in place.

164. Handelsman has well documented that the large increases in physiological and performance advantages characteristic of men develop in tandem with, and are likely driven by, the rapid and large increases in circulating testosterone levels that males experience across puberty, or generally between the ages of about 12 through 18. (Handelsman 2018.) Some have misinterpreted Handelsman as suggesting that

all of those advantages are and remain entirely dependent–on an ongoing basis–on *current* circulating testosterone levels. This is a misreading of Handelsman, who makes no such claim. As the studies reviewed above demonstrate, it is also empirically false with respect to multiple measures of performance. Indeed, Handelsman himself, referring to the Roberts et al. (2020) study which I describe below, has recently written that "transwomen treated with estrogens after completing male puberty experienced only minimal declines in physical performance over 12 months, substantially surpassing average female performance for up to 8 years." (Handelsman 2020.)

165. As to individual physiological advantages, the more accurate and more complicated reality is reflected in a statement titled "The Role of Testosterone in Athletic Performance," published in 2019 by several dozen sports medicine experts and physicians from many top medical schools and hospitals in the U.S. and around the world. (Levine et al. 2019.) This expert group concurs with Handelsman regarding the importance of testosterone to the male advantage, but recognizes that those advantages depend not only on *current* circulating testosterone levels in the individual, but on the "exposure in biological males to much higher levels of testosterone during growth, development, and throughout the athletic career." (*Emphasis added*.) In other words, both past and current circulating testosterone levels affect physiology and athletic capability.

166. Available research enables us to sort out, in some detail, which specific physiological advantages are immutable once they occur, which can be reversed only in part, and which appear to be highly responsive to later hormonal manipulation. The bottom line is that very few of the male physiological advantages I have reviewed in Section II above are largely reversible by testosterone suppression once an individual has passed through male puberty.

Skeletal Configuration

167. It is obvious that some of the physiological changes that occur during

"growth and development" across puberty cannot be reversed. Some of these irreversible physiological changes are quite evident in photographs that have recently appeared in the news of transgender competitors in female events. These include skeletal configuration advantages including:

- Longer and larger bones that give height, weight, and leverage advantages to men;
- More advantageous hip shape and configuration as compared to women.

Cardiovascular Advantages

168. Developmental changes for which there is no apparent means of reversal, and no literature suggesting reversibility, also include multiple contributors to the male cardiovascular advantage, including diaphragm placement, lung and trachea size, and heart size and therefore pumping capacity.[12]

169. In what is, to date, the only evaluation of VO_2max is a cross-sectional study on cardiopulmonary capacity and muscle strength in biological males who identify as female and have undergone long-term cross-sex hormone therapy (Alvares 2022). All of the study subjects that were biological males who identify as female had testosterone suppressed through medication (cyproterone acetate) or gonadectomy. (Supplementary materials) And they had taken exogenous estrogen for an average of 14.4 years with a standard deviation of 3.5 years. Compared to a control group of cisgender women, even after 14 years of testosterone suppression and estrogen administration the biological males who identify as female exhibited advantages in cardio-respiratory capacity measured as higher VO_2 peak and higher O_2 pulse, which suggests that male advantages are retained in events that are influenced by cardio-respiratory endurance (e.g. distance running, cycling, swimming, etc.).

170. On the other hand, the evidence is mixed as to hemoglobin concentration,

[12] "[H]ormone therapy will not alter … lung volume or heart size of the transwoman athlete, especially if [that athlete] transitions postpuberty, so natural advantages including joint articulation, stroke volume and maximal oxygen uptake will be maintained." (Knox 2019 at 398.)

which as discussed above is a contributing factor to $V0_2$ max. Harper (2021) surveyed the literature and found that "Nine studies reported the levels of Hgb [hemoglobin] or HCT [red blood cell count] in transwomen before and after [testosterone suppression], from a minimum of three to a maximum of 36 months post hormone therapy. Eight of these studies. . . found that hormone therapy led to a significant (4.6%–14.0%) decrease in Hgb/HCT ($p<0.01$), while one study found no significant difference after 6 months," but only one of those eight studies returned results at the generally accepted 95% confidence level. (Harper 2021 at 5-6 and Table 5.)

171. I have not found any study of the effect of testosterone suppression on the male advantage in mitochondrial biogenesis.

Muscle mass

172. Multiple studies have found that muscle mass decreases modestly or not at all in response to testosterone suppression. Knox et al. report that "healthy young men did not lose significant muscle mass (or power) when their circulating testosterone levels were reduced to 8.8 nmol/L (lower than the 2015 IOC guideline of 10 nmol/L) for 20 weeks." (Knox 2019 at 398.) Gooren found that "[i]n spite of muscle surface area reduction induced by androgen deprivation, after 1 year the mean muscle surface area in male-to- female transsexuals remained significantly greater than in untreated female-to-male transsexuals." (Gooren 2011 at 653.) An earlier study by Gooren found that after one year of testosterone suppression, muscle mass at the thigh was reduced by only about 10%, exhibited "no further reduction after 3 years of hormones," and "remained significantly greater" than in his sample of untreated women. (Gooren 2004 at 426-427.) Van Caenegem et al. found that muscle cross section in the calf and forearm decreased only trivially (4% and 1% respectively) after two years of testosterone suppression. (Van Caenegem 2015 Table 4.)

173. Taking measurements one month after start of testosterone suppression in

male-to-female (non-athlete) subjects, and again 3 and 11 months after start of feminizing hormone replacement therapy in these subjects, Wiik et al. found that total lean tissue (i.e. primarily muscle) did not decrease significantly across the entire period. Indeed, "some of the [subjects] did not lose any muscle mass at all." (Wiik 2020 at 812.) And even though they observed a small decrease in thigh muscle mass, they found that isometric strength levels measured at the knee "were maintained over the [study period]." (808) "At T12 [the conclusion of the one-year study], the absolute levels of strength and muscle volume were greater in [male-to-female subjects] than in [female-to-male subjects] and CW [women who had not undergone any hormonal therapy]." (808)

174. Alvares et al. (2022) In a cross-sectional study of 15 natal males aged 34.2 ± 5.2 years who had taken exogenous estrogen for an average of 14.4 ± 3.5 years, and compared to a control group of comparably aged females, the transwomen exhibited a 40% advantage in skeletal muscle mass confirming the findings of previous studies regarding the minimal reduction in muscle mass due to transgender hormone therapy, but extending the information to a longer time period (Table 3 at 5).

175. Other papers including Auer. et al (2016), Auer et al. (2018), Elbers et al. (1999), Gava et al. (2016), Haraldsen et al. (2007), Klaver et al. (2018), Klaver et al. (2017), Lapauw et al. (2008), Mueller et al. (2018), Wiercks (et al. (2014), and Yun et al. (2021) have evaluated the changes in body composition in males undergoing transgender hormone therapy with a common finding that there are large retained male advantages in lean body mass.

176. Hilton & Lundberg summarize an extensive survey of the literature as follows:

> "12 longitudinal studies have examined the effects of testosterone suppression on lean body mass or muscle size in transgender women. The collective evidence from these studies suggests that 12 months, which is the most commonly

> examined intervention period, of testosterone suppression to female typical reference levels results in a modest (approximately− 5%) loss of lean body mass or muscle size. . . .
>
> "Thus, given the large baseline differences in muscle mass between males and females (Table 1; approximately 40%), the reduction achieved by 12 months of testosterone suppression can reasonably be assessed as small relative to the initial superior mass. We, therefore, conclude that the muscle mass advantage males possess over females, and the performance implications thereof, are not removed by the currently studied durations (4 months, 1, 2 and 3 years) of testosterone suppression in transgender women. (Hilton 2021 at 205-207.)

177. When we recall that "women have 50% to 60% of men's upper arm muscle cross-sectional area and 65% to 70% of men's thigh muscle cross-sectional area" (Handelsman 2018 at 812), it is clear that Hilton's conclusion is correct. In other words, biologically male subjects possess substantially larger muscles than biologically female subjects after undergoing a year or even three years of testosterone suppression.

178. I note that outside the context of transgender athletes, the testosterone-driven increase in muscle mass and strength enjoyed by these male-to-female subjects would constitute a disqualifying doping violation under all league anti-doping rules with which I am familiar.

C. Responsible voices internationally are increasingly recognizing that suppression of testosterone in a male after puberty has occurred does not substantially reverse the male athletic advantage.

179. The previous very permissive NCAA policy governing transgender participation in women's collegiate athletics was adopted in 2011, and the previous

IOC guidelines were adopted in 2015. At those dates, much of the scientific analysis of the actual impact of testosterone suppression had not yet been performed, much less any wider synthesis of that science. In fact, a series of important peer-reviewed studies and literature reviews have been published only very recently, since I prepared my first paper on this topic, in early 2020.

180. These new scientific publications reflect a remarkably consistent consensus: once an individual has gone through male puberty, testosterone suppression does not substantially eliminate the physiological and performance advantages that that individual enjoys over female competitors.

181. Importantly, I have found no peer-reviewed scientific paper, nor any respected scientific voice, that is now asserting the contrary–that is, that testosterone suppression can eliminate or even largely eliminate the male biological advantage once puberty has occurred.

182. I excerpt the key conclusions from important recent peer-reviewed papers below.

183. Roberts 2020: "In this study, we confirmed that . . . the pretreatment differences between transgender and cis gender women persist beyond the 12-month time requirement currently being proposed for athletic competition by the World Athletics and the IOC." (6)

184. Wiik 2020: The muscular and strength changes in males undergoing testosterone suppression "were modest. The question of when it is fair to permit a transgender woman to compete in sport in line with her experienced gender identity is challenging." (812)

185. Harper 2021: "[V]alues for strength, LBM [lean body mass], and muscle area in transwomen remain above those of cisgender women, even after 36 months of hormone therapy." (1)

186. Hilton & Lundberg 2021: "evidence for loss of the male performance advantage, established by testosterone at puberty and translating in elite athletes to

a 10–50% performance advantage, is lacking. . . . These data significantly undermine the delivery of fairness and safety presumed by the criteria set out in transgender inclusion policies . . ." (211)

187. Hamilton et al. 2021, "Response to the United Nations Human Rights Council's Report on Race and Gender Discrimination in Sport: An Expression of Concern and a Call to Prioritize Research": "There is growing support for the idea that development influenced by high testosterone levels may result in retained anatomical and physiological advantages . . . . If a biologically male athlete self-identifies as a female, legitimately with a diagnosis of gender dysphoria or illegitimately to win medals, the athlete already possesses a physiological advantage that undermines fairness and safety. This is not equitable, nor consistent with the fundamental principles of the Olympic Charter." (840)

188. Hamilton et al. 2021, "Consensus Statement of the Fédération Internationale de Médecine du Sport" (International Federation of Sports Medicine, or FIMS), signed by more than 60 sports medicine experts from prestigious institutions around the world: The available studies "make it difficult to suggest that the athletic capabilities of transwomen individuals undergoing HRT or GAS are comparable to those of cisgender women." The findings of Roberts et al. "question the required testosterone suppression time of 12 months for transwomen to be eligible to compete in women's sport, as most advantages over ciswomen were not negated after 12 months of HRT."

189. Heather (2022) is another peer-reviewed literature review examining the evidence to date on whether testosterone suppression eliminates the physiological building blocks of male athletic advantage. In this review, Dr. Heather studied the existing literature on male advantages in brain structure, muscle mass, bone structure, and the cardio-respiratory system, and the effects of testosterone suppression on those advantages. She concluded:

Given that the percentage difference between medal placings

at the elite level is normally less than 1%, there must be confidence that an elite transwoman athlete retains no residual advantage from former testosterone exposure, where the inherent advantage depending on sport could be 10-30%. Current scientific evidence can not provide such assurances and thus, under abiding rulings, the inclusion of transwomen in the elite female division needs to be reconsidered for fairness to female-born athletes. (8)

190. Nokoff et al. (2023) is another peer-reviewed literature review examining the evidence to date on whether Gender Affirming Hormone Therapy in transwomen eliminates male sex-based athletic advantages and concludes that "reductions of lean body mass and muscle cross-sectional area in the first 12 to 36 months of GAHT … are associated with small reductions or no change in limb strength assessed by hand grip or knee flexion/extension." And "After pubertal change begin, sex segregation for sports involving endurance, power, and strength, … allow adolescent girls and women to excel."

191. Outside the forum of peer-reviewed journals, respected voices in sport are reaching the same conclusion.

192. The **Women's Sports Policy Working Group** identifies among its members and "supporters" many women Olympic medalists, former women's tennis champion and LGBTQ activist Martina Navratilova, Professor Doriane Coleman, a former All-American women's track competitor, transgender athletes Joanna Harper and Dr. Renee Richards, and many other leaders in women's sports and civil rights. I have referenced other published work of Joanna Harper and Professor Coleman. In early 2021 the Women's Sports Policy Working Group published a "Briefing Book" on the issue of transgender participation in women's sports,[13] in

[13] https://womenssportspolicy.org/wp-content/uploads/2021/02/Congressional-Briefing-WSPWG-Transgender-Women-Sports-2.27.21.pdf

which they reviewed largely the same body of literature I have reviewed above, and analyzed the implications of that science for fairness and safety in women's sports.

193. Among other things, the Women's Sports Policy Working Group concluded:

- "[T]he evidence is increasingly clear that hormones do not eliminate the legacy advantages associated with male physical development" (8) due to "the considerable size and strength advantages that remain even after hormone treatments or surgical procedures." (17)
- "[T]here is convincing evidence that, depending on the task, skill, sport, or event, trans women maintain male sex-linked (legacy) advantages even after a year on standard gender-affirming hormone treatment." (26, citing Roberts 2020.)
- "[S]everal peer-reviewed studies, including one based on data from the U.S. military, have confirmed that trans women retain their male sex-linked advantages even after a year on gender affirming hormones. . . . Because of these retained advantages, USA Powerlifting and World Rugby have recently concluded that it isn't possible fairly and safely to include trans women in women's competition." (32)

194. As has been widely reported, in 2020, after an extensive scientific consultation process, the **World Rugby** organization issued its Transgender Guidelines, finding that it would not be consistent with fairness or safety to permit biological males to compete in World Rugby women's matches, no matter what hormonal or surgical procedures they might have undergone. Based on their review of the science, World Rugby concluded:

- "Current policies regulating the inclusion of transgender women in sport are based on the premise that reducing testosterone to levels found in biological females is sufficient to remove many of the biologically-based performance advantages described above. However, peer-reviewed evidence suggests that this is not the case."
- "Longitudinal research studies on the effect of reducing testosterone to female

levels for periods of 12 months or more do not support the contention that variables such as mass, lean mass and strength are altered meaningfully in comparison to the original male-female differences in these variables. The lowering of testosterone removes only a small proportion of the documented biological differences, with large, retained advantages in these physiological attributes, with the safety and performance implications described previously."

- ". . . given the size of the biological differences prior to testosterone suppression, this comparatively small effect of testosterone reduction allows substantial and meaningful differences to remain. This has significant implications for the risk of injury . . . ."
- " . . . bone mass is typically maintained in transgender women over the course of at least 24 months of testosterone suppression, ... Height and other skeletal measurements such as bone length and hip width have also not been shown to change with testosterone suppression, and nor is there any plausible biological mechanism by which this might occur, and so sporting advantages due to skeletal differences between males and females appear unlikely to change with testosterone reduction.

195. In September 2021 the government-commissioned Sports Councils of the United Kingdom and its subsidiary parts (the five Sports Councils responsible for supporting and investing in sport across England, Wales, Scotland and Northern Ireland) issued a formal "Guidance for Transgender Inclusion in Domestic Sport" (UK Sport Councils 2021), following an extensive consultation process, and a commissioned "International Research Literature Review" prepared by the Carbmill Consulting group (UK Sport Literature Review 2021). The UK Sport Literature Review identified largely the same relevant literature that I review in this paper, characterizes that literature consistently with my own reading and description, and based on that science reaches conclusions similar to mine.

196. The UK Sport Literature Review 2021 concluded:

- "Sexual dimorphism in relation to sport is significant and the most important determinant of sporting capacity. The challenge to sporting bodies is most evident in the inclusion of transgender people in female sport." "[The] evidence suggests that parity in physical performance in relation to gender-affected sport cannot be achieved for transgender people in female sport through testosterone suppression. Theoretical estimation in contact and collision sport indicate injury risk is likely to be increased for female competitors." (10)
- "From the synthesis of current research, the understanding is that testosterone suppression for the mandated one year before competition will result in little or no change to the anatomical differences between the sexes, and a more complete reversal of some acute phase metabolic pathways such as haemoglobin levels although the impact on running performance appears limited, and a modest change in muscle mass and strength: The average of around 5% loss of muscle mass and strength will not reverse the average 40-50% difference in strength that typically exists between the two sexes." (7)
- "These findings are at odds with the accepted intention of current policy in sport, in which twelve months of testosterone suppression is expected to create equivalence between transgender women and females." (7)

197. Taking into account the science detailed in the UK Sport Literature Review 2021, the UK Sports Councils have concluded:

- "[T]he latest research, evidence and studies made clear that there are retained differences in strength, stamina and physique between the average woman compared with the average transgender woman or non-binary person registered male at birth, with or without testosterone suppression." (3)
- "Competitive fairness cannot be reconciled with self-identification into the female category in gender-affected sport." (7)
- "As a result of what the review found, the Guidance concludes that the inclusion of transgender people into female sport cannot be balanced regarding

transgender inclusion, fairness and safety in gender-affected sport where there is meaningful competition. This is due to retained differences in strength, stamina and physique between the average woman compared with the average transgender woman or non-binary person assigned male at birth, with or without testosterone suppression." (6)

- "Based upon current evidence, testosterone suppression is unlikely to guarantee fairness between transgender women and natal females in gender-affected sports. . . . Transgender women are on average likely to retain physical advantage in terms of physique, stamina, and strength. Such physical differences will also impact safety parameters in sports which are combat, collision or contact in nature." (7)

198. On January 15, 2022 the American Swimming Coaches Association (ASCA) issued a statement stating, "The American Swimming Coaches Association urges the NCAA and all governing bodies to work quickly to update their policies and rules to maintain fair competition in the women's category of swimming. ASCA supports following all available science and evidenced-based research in setting the new policies, and we strongly advocate for more research to be conducted" and further stated "The current NCAA policy regarding when transgender females can compete in the women's category can be unfair to cisgender females and needs to be reviewed and changed in a transparent manner." (https://swimswam.com/asca-issues-statement-calling-for-ncaa-to-review-transgender-rules/; Accessed January 16, 2022.)

199. On January 19, 2022, the NCAA Board of Governors approved a change to the policy on transgender inclusion in sport and stated that "…the updated NCAA policy calls for transgender participation for each sport to be determined by the policy for the national governing body of that sport, subject to ongoing review and recommendation by the NCAA Committee on Competitive Safeguards and Medical Aspects of Sports to the Board of Governors. If there is no

N[ational]G[overning]B[ody] policy for a sport, that sport's international federation policy would be followed. If there is no international federation policy, previously established IOC policy criteria would be followed" (https://www.ncaa.org/news/2022/1/19/media-center-board-of-governors-updates-transgender-participation-policy.aspx; Accessed January 20, 2022.)

200. On February 1, 2022, because "…a competitive difference in the male and female categories and the disadvantages this presents in elite head-to-head competition … supported by statistical data that shows that the top-ranked female in 2021, on average, would be ranked 536th across all short course yards (25 yards) male events in the country and 326th across all long course meters (50 meters) male events in the country, among USA Swimming members," USA Swimming released its Athlete Inclusion, Competitive Equity and Eligibility Policy. The policy is intended to "provide a level-playing field for elite cisgender women, and to mitigate the advantages associated with male puberty and physiology." (USA Swimming Releases Athlete Inclusion, Competitive Equity and Eligibility Policy, available at https://www.usaswimming.org/news/2022/02/01/usa-swimming-releases-athlete-inclusion-competitive-equity-and-eligibility-policy.) The policy states:

- For biologically male athletes seeking to compete in the female category in certain "elite" level events, the athlete has the burden of demonstrating to a panel of independent medical experts that:
    - "From a medical perspective, the prior physical development of the athlete as Male, as mitigated by any medical intervention, does not give the athlete a competitive advantage over the athlete's cisgender Female competitors" and
    - There is a presumption that the athlete is not eligible unless the athlete "demonstrates that the concentration of testosterone in the athlete's serum has been less than 5 nmol/L . . . continuously for a period of at least thirty-six (36) months before the date of the Application." This

presumption may be rebutted "if the Panel finds, in the unique circumstances of the case, that [the athlete's prior physical development does not give the athlete a competitive advantage] notwithstanding the athlete's serum testosterone results (e.g., the athlete has a medical condition which limits bioavailability of the athlete's free testosterone)." (USA Swimming Athlete Inclusion Procedures at 43.)

201. FINA, the international aquatics (swimming and diving) federation, issued a new policy in June 2022 allowing biological males to compete in the female category of aquatics only if they can establish that they "had male puberty suppressed beginning at Tanner Stage 2 or before age 12, whichever is later, and they have since continuously maintained their testosterone levels in serum (or plasma) below 2.5 nmol/L." FINA Policy on Eligibility for the Men's and Women's Categories § F.4.b.ii. A biologically male athlete who cannot meet these criteria is prohibited from competing in the female category. Id.

- This policy is based on the review of the scientific literature conducted by an independent panel of experts in physiology, endocrinology, and human performance, including specialists in transgender medicine. This panel concluded:

> [I]f gender-affirming male-to-female transition consistent with the medical standard of care is initiated after the onset of puberty, it will blunt some, but not all, of the effects of testosterone on body structure, muscle function, and other determinants of performance, but there will be persistent legacy effects that will give male-to-female transgender athletes (transgender women) a relative performance advantage over biological females. A biological female athlete cannot overcome that advantage through training or nutrition.

> Nor can they take additional testosterone to obtain the same advantage, because testosterone is a prohibited substance under the World Anti-Doping Code. (2)

202. In June 2022, British Triathlon adopted a new policy limiting competition in the female category to "people who are the female sex at birth." British Triathlon Transgender Policy § 7.2.

- This policy is based on its review of the scientific literature and conclusions that "the scientific community broadly agrees that the majority of the physiological/biological advantages brought about by male puberty are retained (either wholly or partially) by transwomen post transition" and that testosterone suppression does not "sufficiently remove[] the retained sporting performance advantage of transwomen." British Triathlon Transgender Policy § 2 (emphasis in original).

203. In June 2022, UCI, the world cycling federation, changed its eligibility criteria for males who identify as female competing in the female category from 12 months of testosterone suppression to the level of 5 nmol/L to 24 months of testosterone suppression to the level of 2.5 nmol/L. UCI Rules § 13.5.015.

- In releasing the new policy, UCI cited a position paper by Prof. Xavier Bigard (2022), which concluded that the "potential [male] advantage on muscle strength / power cannot be erased before a period of 24 months." (15) Notably, Prof. Bigard did not assert that the best available evidence shows that male advantage is actually erased after 24 months; he merely asserted that the evidence shows that male advantage is not erased before 24 months.
- It was reported by Sean Ingle in the Guardian on Thursday, May 4, 2023, that UCI may reconsider its transgender participation policy after a male who identifies as a female won the Tour of the Gila in New Mexico “The UCI also hears the voices of female athletes and their concerns about an equal playing field for competitors, and will take into account all elements, including the

evolution of scientific knowledge."

204. In July 2022, England's Rugby Football Union and Rugby Football League both approved new policies limiting the female category to players whose sex recorded at birth is female for contact rugby for the under 12 age group and above. Rugby Football League Gender Participation Policy § 4.2(d); Rugby Football Union Gender Participation Policy § 4.2(d).

- In August 2022, the Irish Rugby Football Union adopted the same policy. Irish Rugby Football Union Gender Participation Policy §§ 4.5(b) & (f).
- In September 2022, the Welsh Rugby Union also adopted the same policy.
- These bodies based their policy on a review of the scientific research, which showed that male advantage "cannot be sufficiently addressed even with testosterone suppression." Rugby Football Union Gender Participation Policy § 3.4; see also Rugby Football League Gender Participation Policy § 3.4; Irish Rigby Football Union Gender Participation Policy § 4.3.

205. In August 2022, the World Boxing Council issued a new policy requiring athletes to compete in accordance with their natal sex. World Boxing Council Statement/Guidelines Regarding Transgender Athletes Participation in Professional Combat Sports. The WBC concluded that any other policy would raise "serious health and safety concerns." Id.

206. In August 2022, World Triathlon issued a new policy limiting the female category to biological females and to biological males who have suppressed circulating testosterone to 2.5 nmol/L for at least 24 months and have not competed in the male category in at least 48 months. World Triathlon Transgender Policy Process § 3. Previously, it had followed the old IOC guidelines of requiring testosterone suppression to 10 nmol/L for at least 12 months.

- In issuing this policy, World Triathlon stated that "the potential advantage in muscle strength/power of Transgender women cannot be erased before two years of testosterone suppression." World Triathlon Transgender Policy Process § 3.

Notably, World Triathlon did not assert that two years of testosterone suppression actually erases male performance advantage, nor did it cite any evidence that would support such a proposition.

- Although World Triathlon listed sports scientists Drs. Emma Hilton and Ross Tucker as consultants in developing the new policy, both immediately criticized the policy as allowing male advantage into female triathlon competitions.
- Another sports scientist listed as a consultant to World Triathlon, Dr. Alun Williams, has opined that basing eligibility on circulating testosterone levels is not evidence-based policymaking because of the lack of evidence that testosterone suppression eliminates male performance advantage.

207. In March 2023, the World Athletics Council, the governing body for world class track & field competition issued new transgender and DSD (Disorders of Sex Development) regulations. The transgender participation policy is very similar to the policies of World Rugby, World Boxing, and FINA by stating "In regard to transgender athletes, the Council has agreed to exclude male-to-female transgender athletes who have been through male puberty from female World Rankings competition from 31 March 2023." And "For DSD athletes, the new regulations will require any relevant athletes to reduce their testosterone levels below a limit of 2.5 nmol/L for a minimum of 24 months to compete internationally in the female category in any event."

- These policies are particularly noteworthy as there is a clear separation of the concerns regarding athletes who are transgender and those who have a DSD.

Conclusions

The research and actual observed data show the following:

- At the level of (a) elite, (b) collegiate, (c) scholastic, and (d) recreational competition, men, adolescent boys, or male children, have an advantage over equally gifted, aged and trained women, adolescent girls, or female children in almost all athletic events;

- Biological male physiology is the basis for the performance advantage that men, adolescent boys, or male children have over women, adolescent girls, or female children in almost all athletic events; and
- The administration of androgen inhibitors and cross-sex hormones to men or adolescent boys after the onset of male puberty does not eliminate the performance advantage that men and adolescent boys have over women and adolescent girls in almost all athletic events. Likewise, there is no published scientific evidence that the administration of puberty blockers to males before puberty eliminates the pre-existing athletic advantage that prepubertal males have over prepubertal females in almost all athletic events.

For over a decade sports governing bodies (such as the IOC and NCAA) have wrestled with the question of transgender inclusion in female sports. The previous polices implemented by these sporting bodies had an underlying "premise that reducing testosterone to levels found in biological females is sufficient to remove many of the biologically-based performance advantages." (World Rugby 2020 at 13.) Disagreements centered around what the appropriate threshold for testosterone levels must be–whether the 10nmol/liter value adopted by the IOC in 2015, or the 5nmol/liter value adopted by the IAAF.

But the science that has become available within just the last few years contradicts that premise. Instead, as the UK Sports Councils, World Rugby, the FIMS Consensus Statement, and the Women's Sports Policy Working Group have all recognized the science is now sharply "at odds with the accepted intention of current policy in sport, in which twelve months of testosterone suppression is expected to create equivalence between transgender women and females" (UK Sports Literature Review 2021 at 7), and it is now "difficult to suggest that the athletic capabilities of transwomen individuals undergoing HRT or GAS are comparable to those of cisgender women." (Hamilton, FIMS Consensus Statement 2021.) It is important to note that while the 2021 "IOC Framework on Fairness, Inclusion, and Non-Discrimination on the Basis of Gender Identity and Sex Variations"

calls for an "evidence-based approach," that Framework does not actually reference *any* of the now extensive scientific evidence relating to the physiological differences between the sexes, and the inefficacy of hormonal intervention to eliminate male advantages relevant to most sports. Instead, the IOC calls on other sporting bodies to define criteria for transgender inclusion, while demanding that such criteria simultaneously ensure fairness, safety, and inclusion for all. The recently updated NCAA policy on transgender participation also relies on other sporting bodies to establish criteria for transgender inclusion while calling for fair competition and safety.

But what we currently know tells us that these policy goals—fairness, safety, and full transgender inclusion—are irreconcilable for many or most sports. Long human experience is now joined by large numbers of research papers that document that males outperform females in muscle strength, muscular endurance, aerobic and anaerobic power output, VO_2max, running speed, swimming speed, vertical jump height, reaction time, and most other measures of physical fitness and physical performance that are essential for athletic success. The male advantages have been observed in fitness testing in children as young as 3 years old, with the male advantages increasing immensely during puberty. To ignore what we know to be true about males' athletic advantages over females, based on mere hope or speculation that cross sex hormone therapy (puberty blockers, androgen inhibitors, or cross-sex hormones) might neutralize that advantage, when the currently available evidence says it does not, is not science and is not "evidence-based" policy-making.

Because of the recent research and analysis in the general field of transgender athletics, many sports organizations have revised their policies or are in the process of doing so. As a result, there is not any universally recognized policy among sports organizations, and transgender inclusion policies are in a state of flux, likely because of the increasing awareness that the goals of fairness, safety, and full transgender inclusion are irreconcilable.

Sports have been separated by sex for the purposes of safety and fairness for a

considerable number of years. The values of safety and fairness are endorsed by numerous sports bodies, including the NCAA and IOC. The existing evidence of durable physiological and performance differences based on biological sex provides a strong evidence-based rationale for keeping rules and policies for such sex-based separation in place (or implementing them as the case may be).

As set forth in detail in this report, there are physiological differences between males and females that result in males having a significant performance advantage over similarly gifted, aged, and trained females in nearly all athletic events before, during, and after puberty. There is not scientific evidence that any amount or duration of cross sex hormone therapy (puberty blockers, androgen inhibitors, or cross-sex hormones) eliminates all physiological advantages that result in males performing better than females in nearly all athletic events. Males who have received such therapy retain sufficient male physiological traits that enhance athletic performance vis-à-vis similarly aged females and are thus, from a physiological perspective, more accurately categorized as male and not female.

I swear or affirm under penalty of perjury that the foregoing is true and correct.

Dated: May 18, 2023 Signed: */s/ Dr. Gregory A. Brown, Ph.D., FACSM*

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Appendix 1 – Data Tables

Presidential Physical Fitness Results[14]

Curl-Ups (# in 1 minute)

	Male		Female			Male-Female Difference	
Age	50th %ile	85th %ile	50th %ile	85th %ile	Age	50th %ile	85th %ile
6	22	33	23	32	**6**	-4.3%	3.1%
7	28	36	25	34	**7**	12.0%	5.9%
8	31	40	29	38	**8**	6.9%	5.3%
9	32	41	30	39	**9**	6.7%	5.1%
10	35	45	30	40	**10**	16.7%	12.5%
11	37	47	32	42	**11**	15.6%	11.9%
12	40	50	35	45	**12**	14.3%	11.1%
13	42	53	37	46	**13**	13.5%	15.2%
14	45	56	37	47	**14**	21.6%	19.1%
15	45	57	36	48	**15**	25.0%	18.8%
16	45	56	35	45	**16**	28.6%	24.4%
17	44	55	34	44	**17**	29.4%	25.0%

[14] This data is available from a variety of sources. including: https://gilmore.gvsd.us/documents/Info/Forms/Teacher%20Forms/Presidentialchallengetest.pdf

Shuttle Run (seconds)

	Male		Female		Male-Female Difference		%
Age	50th %ile	85th %ile	50th %ile	85th %ile	Age	50th %ile	85th %ile
6	13.3	12.1	13.8	12.4	**6**	3.6%	2.4%
7	12.8	11.5	13.2	12.1	**7**	3.0%	5.0%
8	12.2	11.1	12.9	11.8	**8**	5.4%	5.9%
9	11.9	10.9	12.5	11.1	**9**	4.8%	1.8%
10	11.5	10.3	12.1	10.8	**10**	5.0%	4.6%
11	11.1	10	11.5	10.5	**11**	3.5%	4.8%
12	10.6	9.8	11.3	10.4	**12**	6.2%	5.8%
13	10.2	9.5	11.1	10.2	**13**	8.1%	6.9%
14	9.9	9.1	11.2	10.1	**14**	11.6%	9.9%
15	9.7	9.0	11.0	10.0	**15**	11.8%	10.0%
16	9.4	8.7	10.9	10.1	**16**	13.8%	13.9%
17	9.4	8.7	11.0	10.0	**17**	14.5%	13.0%

1 mile run (seconds)

	Male		Female		Male-Female Difference		%
Age	50th %ile	85th %ile	50th %ile	85th %ile	Age	50th %ile	85th %ile
6	756	615	792	680	**6**	4.5%	9.6%
7	700	562	776	636	**7**	9.8%	11.6%
8	665	528	750	602	**8**	11.3%	12.3%
9	630	511	712	570	**9**	11.5%	10.4%

10	588	477	682	559	**10**	13.8%	14.7%
11	560	452	677	542	**11**	17.3%	16.6%
12	520	431	665	503	**12**	21.8%	14.3%
13	486	410	623	493	**13**	22.0%	16.8%
14	464	386	606	479	**14**	23.4%	19.4%
15	450	380	598	488	**15**	24.7%	22.1%
16	430	368	631	503	**16**	31.9%	26.8%
17	424	366	622	495	**17**	31.8%	26.1%

Pull Ups (# completed)

					Male-Female		**%**
Male			**Female**		**Difference**		
	50th	**85th**	**50th**	**85th**		**50th**	**85th**
Age	**%ile**	**%ile**	**%ile**	**%ile**	**Age**	**%ile**	**%ile**
6	1	2	1	2	**6**	0.0%	0.0%
7	1	4	1	2	**7**	0.0%	100.0%
8	1	5	1	2	**8**	0.0%	150.0%
9	2	5	1	2	**9**	100.0%	150.0%
10	2	6	1	3	**10**	100.0%	100.0%
11	2	6	1	3	**11**	100.0%	100.0%
12	2	7	1	2	**12**	100.0%	250.0%
13	3	7	1	2	**13**	200.0%	250.0%
14	5	10	1	2	**14**	400.0%	400.0%
15	6	11	1	2	**15**	500.0%	450.0%

16	7	11	1	1	**16**	600.0%	1000.0%	
17	8	13	1	1	**17**	700.0%	1200.0%	

Data Compiled from Athletic.Net

2021 National 3000 m cross country race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	691.8	728.4	Difference	607.7	659.8	Difference	608.1	632.6	Difference
2	722.5	739.0	#1 boy vs #	619.6	674.0	#1 boy vs #	608.7	639.8	#1 boy vs #
3	740.5	783.0	1 girl	620.1	674.7	1 girl	611.3	664.1	1 girl
4	759.3	783.5	5.0%	643.2	683.7	7.9%	618.6	664.4	3.9%
5	759.6	792.8		646.8	685.0		619.7	671.6	
6	760.0	824.1		648.0	686.4		631.2	672.1	
7	772.0	825.7	Average	648.8	687.0	Average	631.7	672.3	Average
8	773.0	832.3	difference	658.0	691.0	difference	634.9	678.4	difference
9	780.7	834.3	boys vs girls	659.5	692.2	boys vs girls	635.0	679.3	boys vs girls
10	735.1	844.4	6.2%	663.9	663.3	5.6%	635.1	679.4	6.3%

2021 National 100 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	13.06	14.24	Difference	10.87	12.10	Difference	11.37	12.08	Difference
2	13.54	14.41	#1 boy vs #	10.91	12.24	#1 boy vs #	11.61	12.43	#1 boy vs #
3	13.73	14.44	1 girl	11.09	12.63	1 girl	11.73	12.51	1 girl
4	14.10	14.48	8.3%	11.25	12.70	10.2%	11.84	12.55	5.9%
5	14.19	14.49		11.27	12.75		11.89	12.57	
6	14.31	14.58		11.33	12.80		11.91	12.62	
7	14.34	14.69	Average	11.42	12.83	Average	11.94	12.65	Average
8	14.35	14.72	difference	11.43	12.84	difference	11.97	12.71	difference
9	14.41	14.77	boys vs girls	11.44	12.88	boys vs girls	12.08	12.71	boys vs girls
10	14.43	14.86	3.6%	11.51	12.91	11.1%	12.12	12.75	5.7%

2021 National 200 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	24.02	28.72	Difference	21.77	25.36	Difference	20.66	25.03	Difference
2	24.03	28.87	#1 boy vs #	22.25	25.50	#1 boy vs #	22.91	25.18	#1 boy vs #
3	28.07	29.92	1 girl	22.48	25.55	1 girl	23.14	25.22	1 girl
4	28.44	29.95	16.4%	22.57	25.70	14.2%	23.69	25.49	17.5%
5	28.97	30.04		22.65	26.08		23.84	25.78	
6	29.26	30.09		22.77	26.22		24.23	25.89	
7	29.34	30.27	Average	23.11	26.79	Average	24.35	26.03	Average
8	29.38	30.34	difference	23.16	26.84	difference	24.58	26.07	difference
9	29.65	30.41	boys vs girls	23.28	26.91	boys vs girls	24.59	26.10	boys vs girls
10	29.78	30.54	6.1%	23.47	26.85	13.1%	24.61	26.13	7.9%

2021 National 400 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	66.30	67.12	Difference	49.29	56.80	Difference	51.96	55.70	Difference
2	66.88	67.67	#1 boy vs #	50.47	58.57	#1 boy vs #	55.52	57.08	#1 boy vs #
3	67.59	67.74	1 girl	52.28	60.65	1 girl	55.58	57.60	1 girl
4	68.16	68.26	1.2%	52.44	61.45	13.2%	55.59	57.79	6.7%
5	68.51	68.37		53.31	61.81		55.72	58.02	
6	69.13	71.02		53.65	62.03		55.84	58.25	
7	69.75	72.73	Average	53.78	62.32	Average	55.92	59.25	Average
8	69.80	73.25	difference	54.51	62.33	difference	57.12	59.27	difference
9	69.81	73.31	boys vs girls	55.84	62.34	boys vs girls	57.18	59.40	boys vs girls
10	70.32	73.48	2.4%	55.90	62.40	13.0%	57.22	59.49	4.2%

2021 National 800 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	152.2	157.9	Difference	120.8	141.4	Difference	127.8	138.5	Difference
2	155.2	164.6	#1 boy vs #	124.0	142.2	#1 boy vs #	129.7	143.1	#1 boy vs #
3	161.0	164.9	1 girl	125.1	148.8	1 girl	130.5	144.2	1 girl
4	161.1	165.9	3.6%	125.6	151.3	14.5%	133.2	144.2	7.7%
5	161.2	168.5		126.5	151.6		136.2	144.9	
6	161.6	169.9		136.5	152.5		136.5	145.0	
7	161.8	171.5	Average	137.1	153.1	Average	136.7	145.2	Average
8	162.2	173.1	difference	138.5	153.7	difference	136.7	145.6	difference
9	165.3	173.4	boys vs girls	139.5	153.8	boys vs girls	137.0	145.6	boys vs girls
10	166.9	174.7	4.5%	140.2	154.2	12.6%	137.9	145.8	6.9%

2021 National 1600 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	372.4	397.6	Difference	307.4	319.3	Difference	297.3	313.8	Difference
2	378.3	400.9	#1 boy vs #	313.7	322.2	#1 boy vs #	298.4	317.1	#1 boy vs #
3	378.4	405.6	1 girl	315.0	322.6	1 girl	307.0	319.9	1 girl
4	402.0	435.2	6.3%	318.2	337.5	3.7%	313.9	323.3	5.2%
5	406.4	445.0		318.4	345.2		319.2	325.3	
6	413.4	457.0		320.5	345.7		320.4	326.2	
7	457.4	466.0	Average	327.0	345.9	Average	321.1	327.0	Average
8	473.3	466.8	difference	330.3	347.1	difference	321.9	330.0	difference
9	498.3	492.3	boys vs girls	333.4	347.5	boys vs girls	325.5	331.1	boys vs girls
10	505.0	495.0	4.0%	347.0	355.6	4.7%	327.1	332.5	2.9%

2021 National 3000 m Track race time in seconds

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	794.2	859.9	Difference	602.3	679.2	Difference	556.6	623.7	Difference
2	856.3		#1 boy vs #	644.9	709.7	#1 boy vs #	591.6	649.5	#1 boy vs #
3			1 girl	646.6	714.2	1 girl	600.8	651.6	1 girl
4			7.6%	648.2	741.9	11.3%	607.1	654.9	10.8%
5	No further data	No Further Data		648.4	742.7		609.1	662.9	
6				652.8	756.6		611.5	664.1	
7			Average	658.9	760.2	Average	615.7	666.3	Average
8			difference	660.1	762.5	difference	617.3	666.8	difference
9			boys vs girls	662.7	780.2	boys vs girls	618.4	673.2	boys vs girls
10			NA%	671.6	792.3	12.7%	620.6	674.4	8.2%

2021 National Long Jump Distance (in inches)

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	156.0	176.0	Difference	256.8	213.8	Difference	224.0	201.3	Difference
2	156.0	163.8	#1 boy vs #	247.0	212.0	#1 boy vs #	222.5	197.3	#1 boy vs #
3	155.0	153.0	1 girl	241.0	210.8	1 girl	220.5	195.8	1 girl
4	154.3	152.0	-11.4%	236.3	208.8	20.1%	210.3	193.5	11.3%
5	154.0	149.5		231.5	207.0		210.0	193.3	
6	152.8	146.0		225.0	204.8		206.8	192.5	
7	151.5	144.5	Average	224.0	194.5	Average	206.0	192.3	Average
8	150.8	137.5	difference	224.0	192.5	difference	205.5	192.0	difference
9	150.5	137.0	boys vs girls	221.8	192.3	boys vs girls	205.0	191.3	boys vs girls
10		No	1.4%			13.2%			9.1%
		Further							
	150.5	Data		219.0	187.5		204.5	189.0	

2021 National High Jump Distance (in inches)

	7-8 years old			9-10 years old			11-12 year old		
Rank	Boys	Girls		Boys	Girls		Boys	Girls	
1	38.0	37.5	Difference	72.0	58.0	Difference	63.0	56.0	Difference
2	38.0	34.0	#1 boy vs #	70.0	58.0	#1 boy vs #	61.0	56.0	#1 boy vs #
3	36.0	32.0	1 girl	65.8	57.0	1 girl	60.0	57.0	1 girl
4	36.0	32.0	1.3	62.0	56.0	24.1%	59.0	56.0	12.5%
5	35.8	32.0		62.0	56.0		59.0	56.0	
6	35.5			62.0	55.0		59.0	55.0	
7	34.0		Average	61.0	54.0	Average	59.0	54.0	Average
8	32.0	No	difference	60.0	54.0	difference	58.0	54.0	difference
9	59.0	further	boys vs girls	59.0	No	boys vs girls	57.8	56.0	boys vs girls
10		Data	21.6%		Further	12.5%			6.9%
	56.0			56.0	Data		57.8	56.0	

Appendix 2 – Scholarly Publications

Refereed Publications

1. Shaw BS. Breukelman G, Millard L, Moran J, Brown G, & Shaw I. Effects of a maximal cycling all-out anaerobic test on visual performance. Clin Exp Optom. https://doi.org/10.1080/08164622.2022.2153583, 2022
2. Brown GA, Shaw BS, Shaw I. How much water is in a mouthful, and how many mouthfuls should I drink? A laboratory exercise to help students understand developing a hydration plan. Adv Physiol Educ 45: 589–593, 2021.
3. Schneider KM and Brown GA (as Faculty Mentor). What's at Stake: Is it a Vampire or a Virus? International Journal of Undergraduate Research and Creative Activities. 11, Article 4. 2019.
4. Christner C and Brown GA (as Faculty Mentor). Explaining the Vampire Legend through Disease. UNK Undergraduate Research Journal. 23(1), 2019. (*This is an on-campus publication.)
5. Schneekloth B and Brown GA. Comparison of Physical Activity during Zumba with a Human or Video Game Instructor. 11(4):1019-1030. International Journal of Exercise Science, 2018.
6. Bice MR, Hollman A, Bickford S, Bickford N, Ball JW, Wiedenman EM, Brown GA, Dinkel D, and Adkins M. Kinesiology in 360 Degrees. International Journal of Kinesiology in Higher Education, 1: 9-17, 2017
7. Shaw I, Shaw BS, Brown GA, and Shariat A. Review of the Role of Resistance Training and Musculoskeletal Injury Prevention and Rehabilitation. Gavin Journal of Orthopedic Research and Therapy. 1: 5-9, 2016
8. Kahle A, Brown GA, Shaw I, & Shaw BS. Mechanical and Physiological Analysis of Minimalist versus Traditionally Shod Running. J Sports Med Phys Fitness. 56(9):974-9, 2016
9. Bice MR, Carey J, Brown GA, Adkins M, and Ball JW. The Use of Mobile Applications to Enhance Learning of the Skeletal System in Introductory Anatomy &

Physiology Students. Int J Kines Higher Educ 27(1) 16-22, 2016

10. Shaw BS, Shaw I, & Brown GA. Resistance Exercise is Medicine. Int J Ther Rehab. 22: 233-237, 2015.
11. Brown GA, Bice MR, Shaw BS, & Shaw I. Online Quizzes Promote Inconsistent Improvements on In-Class Test Performance in Introductory Anatomy & Physiology. Adv. Physiol. Educ. 39: 63-6, 2015
12. Brown GA, Heiserman K, Shaw BS, & Shaw I. Rectus abdominis and rectus femoris muscle activity while performing conventional unweighted and weighted seated abdominal trunk curls. Medicina dello Sport. 68: 9-18. 2015
13. Botha DM, Shaw BS, Shaw I & Brown GA. Role of hyperbaric oxygen therapy in the promotion of cardiopulmonary health and rehabilitation. African Journal for Physical, Health Education, Recreation and Dance (AJPHERD). Supplement 2 (September), 20: 62-73, 2014
14. Abbey BA, Heelan KA, Brown, GA, & Bartee RT. Validity of HydraTrend™ Reagent Strips for the Assessment of Hydration Status. J Strength Cond Res. 28: 2634-9. 2014
15. Scheer KC, Siebrandt SM, Brown GA, Shaw BS, & Shaw I. Wii, Kinect, & Move. Heart Rate, Oxygen Consumption, Energy Expenditure, and Ventilation due to Different Physically Active Video Game Systems in College Students. International Journal of Exercise Science: 7: 22-32, 2014
16. Shaw BS, Shaw I, & Brown GA. Effect of concurrent aerobic and resistive breathing training on respiratory muscle length and spirometry in asthmatics. African Journal for Physical, Health Education, Recreation and Dance (AJPHERD). Supplement 1 (November), 170-183, 2013
17. Adkins M, Brown GA, Heelan K, Ansorge C, Shaw BS & Shaw I. Can dance exergaming contribute to improving physical activity levels in elementary school children? African Journal for Physical, Health Education, Recreation and Dance (AJPHERD). 19: 576-585, 2013
18. Jarvi MB, Brown GA, Shaw BS & Shaw I. Measurements of Heart Rate and

Accelerometry to Determine the Physical Activity Level in Boys Playing Paintball. International Journal of Exercise Science: 6: 199-207, 2013

19. Brown GA, Krueger RD, Cook CM, Heelan KA, Shaw BS & Shaw I. A prediction equation for the estimation of cardiorespiratory fitness using an elliptical motion trainer. West Indian Medical Journal. 61: 114-117, 2013.

20. Shaw BS, Shaw I, & Brown GA. Body composition variation following diaphragmatic breathing. African Journal for Physical, Health Education, Recreation and Dance (AJPHERD). 18: 787-794, 2012.

Refereed Presentations

1. Steinman PM, Steinman PC, Brown GA. Knowledge Of The Female Athlete Triad In Female High School Athletes In Rural Nebraska. Accepted for presentation at the 70th Annual Meeting of the American College of Sports Medicine. Denver CO. May 30 – June 2, 2023.
2. Steinman PC, Steinman PM, Brown GA. Female Athlete Triad Knowledge Among Sports Medicine Rehabilitation Clinicians In Nebraska. Accepted for presentation at the 70th Annual Meeting of the American College of Sports Medicine. Denver CO. May 30 – June 2, 2023.
3. Brown GA, Brown CJ, Shaw I, Shaw B. Boys And Girls Differ In Running And Jumping Track And Field Event Performance Before Puberty. Accepted for presentation at the 70th Annual Meeting of the American College of Sports Medicine. Denver CO. May 30 – June 2, 2023.
4. Brown GA, Orr T, Shaw BS, Shaw I. Comparison of Running Performance Between Division and Sex in NCAA Outdoor Track Running Championships 2010-2019. 54(5), 2146. 69th Annual Meeting of the American College of Sports Medicine. San Diego, CA. May 31 - June 4, 2022.
5. Shaw BS, Lloyd R, Da Silva M, Coetzee D, Millard L, Breukelman G, Brown GA, Shaw I. Analysis Of Physiological Determinants During A Single Bout Of German Volume Training. 54(5), 886. 69th Annual Meeting of the American College of

Sports Medicine. San Diego, CA. May 31 - June 4, 2022.

6. Shaw I, Turner S, Brown GA, Shaw BS. Effects Of Resistance Exercise Modalities On Chest Expansion, Spirometry And Cardiorespiratory Fitness In Untrained Smokers. Med Sci Sport Exerc. 54(5), 889. 69th Annual Meeting of the American College of Sports Medicine. San Diego, CA. May 31 - June 4, 2022.
7. Elton D, Brown GA, Orr T, Shaw BS, Shaw I. Comparison Of Running Performance Between Division And Sex In NCAA Outdoor Track Running Championships 2010-2019. Northland Regional Meeting of the American College of Sports Medicine. Held Virtually. April 8, 2022
8. Brown GA. Transwomen competing in women's sports: What we know, and what we don't. American Physiological Society New Trends in Sex and Gender Medicine conference. Held virtually due to Covid-19 pandemic. October 19 - 22, 2021, 2021.
9. Shaw BS, Boshoff VE, Coetzee S, Brown GA, Shaw I. A Home-based Resistance Training Intervention Strategy To Decrease Cardiovascular Disease Risk In Overweight Children Med Sci Sport Exerc. 53(5), 742. 68th Annual Meeting of the American College of Sports Medicine. Held virtually due to Covid-19 pandemic. June 1-5, 2021.
10. Shaw I, Cronje M, Brown GA, Shaw BS. Exercise Effects On Cognitive Function And Quality Of Life In Alzheimer's Patients In Long-term Care. Med Sci Sport Exerc. 53(5), 743. 68th Annual Meeting of the American College of Sports Medicine. Held virtually due to Covid-19 pandemic. June 1-5, 2021.
11. Brown GA, Escalera M, Oleena A, Turek T, Shaw I, Shaw BS. Relationships between Body Composition, Abdominal Muscle Strength, and Well Defined Abdominal Muscles. Med Sci Sport Exerc. 53(5), 197. 68th Annual Meeting of the American College of Sports Medicine. Held virtually due to Covid-19 pandemic. June 1-5, 2021.
12. Brown GA, Jackson B, Szekely B, Schramm T, Shaw BS, Shaw I. A Pre-Workout

Supplement Does Not Improve 400 M Sprint Running or Bicycle Wingate Test Performance in Recreationally Trained Individuals. Med Sci Sport Exerc. 50(5), 2932. 65th Annual Meeting of the American College of Sports Medicine. Minneapolis, MN. June 2018.

13. Paulsen SM, Brown GA. Neither Coffee Nor A Stimulant Containing "Pre-workout" Drink Alter Cardiovascular Drift During Walking In Young Men. Med Sci Sport Exerc. 50(5), 2409. 65th Annual Meeting of the American College of Sports Medicine. Minneapolis, MN. June 2018.

14. Adkins M, Bice M, Bickford N, Brown GA. Farm to Fresh! A Multidisciplinary Approach to Teaching Health and Physical Activity. 2018 spring SHAPE America central district conference. Sioux Falls, SD. January 2018.

15. Shaw I, Kinsey JE, Richards R, Shaw BS, and Brown GA. Effect Of Resistance Training During Nebulization In Adults With Cystic Fibrosis. International Journal of Arts & Sciences' (IJAS). International Conference for Physical, Life and Health Sciences which will be held at FHWien University of Applied Sciences of WKW, at Währinger Gürtel 97, Vienna, Austria, from 25-29 June 2017.

16. Bongers M, Abbey BM, Heelan K, Steele JE, Brown GA. Nutrition Education Improves Nutrition Knowledge, Not Dietary Habits In Female Collegiate Distance Runners. Med Sci Sport Exerc. 49(5), 389. 64th Annual Meeting of the American College of Sports Medicine. Denver, CO. May 2017.

17. Brown GA, Steele JE, Shaw I, Shaw BS. Using Elisa to Enhance the Biochemistry Laboratory Experience for Exercise Science Students. Med Sci Sport Exerc. 49(5), 1108. 64th Annual Meeting of the American College of Sports Medicine. Denver, CO. May 2017.

18. Brown GA, Shaw BS, and Shaw I. Effects of a 6 Week Conditioning Program on Jumping, Sprinting, and Agility Performance In Youth. Med Sci Sport Exerc. 48(5), 3730. 63rd Annual Meeting of the American College of Sports Medicine. Boston, MA. June 2016.

19. Shaw I, Shaw BS, Boshoff VE, Coetzee S, and Brown GA. Kinanthropometric Responses To Callisthenic Strength Training In Children. Med Sci Sport Exerc. 48(5), 3221. 63rd Annual Meeting of the American College of Sports Medicine. Boston, MA. June 2016.
20. Shaw BS, Shaw I, Gouveia M, McIntyre S, and Brown GA. Kinanthropometric Responses To Moderate-intensity Resistance Training In Postmenopausal Women. Med Sci Sport Exerc. 48(5), 2127. 63rd Annual Meeting of the American College of Sports Medicine. Boston, MA. June 2016.
21. Bice MR, Cary JD, Brown GA, Adkins M, and Ball JW. The use of mobile applications to enhance introductory anatomy & physiology student performance on topic specific in-class tests. National Association for Kinesiology in Higher Education National Conference. January 8, 2016.
22. Shaw I, Shaw BS, Lawrence KE, Brown GA, and Shariat A. Concurrent Resistance and Aerobic Exercise Training Improves Hemodynamics in Normotensive Overweight and Obese Individuals. Med Sci Sport Exerc. 47(5), 559. 62nd Annual Meeting of the American College of Sports Medicine. San Diego, CA. May 2015.
23. Shaw BS, Shaw I, McCrorie C, Turner S., Schnetler A, and Brown GA. Concurrent Resistance and Aerobic Training in the Prevention of Overweight and Obesity in Young Adults. Med Sci Sport Exerc. 47(5), 223. 62nd Annual Meeting of the American College of Sports Medicine. San Diego, CA. May 2015.
24. Schneekloth B, Shaw I, Shaw BS, and Brown GA. Physical Activity Levels Using Kinect™ Zumba Fitness versus Zumba Fitness with a Human Instructor. Med Sci Sport Exerc. 46(5), 326. 61st Annual Meeting of the American College of Sports Medicine. Orlando, FL. June 2014.
25. Shaw I, Lawrence KE, Shaw BS, and Brown GA. Callisthenic Exercise-related Changes in Body Composition in Overweight and Obese Adults. Med Sci Sport Exerc. 46(5), 394. 61st Annual Meeting of the American College of Sports Medicine. Orlando, FL June 2014.

26. Shaw BS, Shaw I, Fourie M, Gildenhuys M, and Brown GA. Variances In The Body Composition Of Elderly Woman Following Progressive Mat Pilates. Med Sci Sport Exerc. 46(5), 558. 61st Annual Meeting of the American College of Sports Medicine. Orlando, FL June 2014.

27. Brown GA, Shaw I, Shaw BS, and Bice M. Online Quizzes Enhance Introductory Anatomy & Physiology Performance on Subsequent Tests, But Not Examinations. Med Sci Sport Exerc. 46(5), 1655. 61st Annual Meeting of the American College of Sports Medicine. Orlando, FL June 2014.

28. Kahle, A. and Brown, G.A. Electromyography in the Gastrocnemius and Tibialis Anterior, and Oxygen Consumption, Ventilation, and Heart Rate During Minimalist versus Traditionally Shod Running. 27th National Conference on Undergraduate Research (NCUR). La Crosse, Wisconsin USA. April 11-13, 2013

29. Shaw, I., Shaw, B.S., and Brown, G.A. Resistive Breathing Effects on Pulmonary Function, Aerobic Capacity and Medication Usage in Adult Asthmatics Med Sci Sports Exerc 45 (5). S1602 2013. 60th Annual Meeting of the American College of Sports Medicine, Indianapolis, IN USA, May 26-30 3013

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IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, *et al.*, Plaintiffs, v. Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction, *et al.*, Defendants.	Case No. 4:23-cv-00185-JGZ **Rebuttal Declaration of Dr. Gregory A. Brown, Ph.D., FACSM, in Further Support of Intervenor-Defendants' Opposition to Plaintiffs' Motion for a Preliminary Injunction**

I, Gregory A. Brown, declare as follows:

1. I have submitted an initial declaration to this Court dated May 18, 2023.

2. I now submit this expert rebuttal declaration based on my personal knowledge, and it reflects my expert opinions.

3. In preparing this rebuttal declaration, I have reviewed the expert declarations

filed by Plaintiffs, submitted by Dr. Shumer and Dr. Budge.

4. In Dr. Shumer's rebuttal to my expert declaration, at paragraph 4, he states "the studies and findings discussed throughout Dr. Brown's declaration support the scientific consensus that the biological cause of average group differences in athletic performance between males and females is the rise in circulating levels of testosterone beginning in endogenous male puberty." This statement seems to completely ignore paragraphs 77-115 of my declaration and the data tables contained therein along with the data tables included in the appendix (pages 99-107), all of which are drawn from 16 separate sources, which document numerous differences in physical fitness and athletic performance between boys and girls before the onset of puberty.

5. In Dr. Shumer's rebuttal to my expert declaration, at paragraph 5, he states that I have misrepresented the writing of McManus and Armstrong (2011) when I wrote (in paragraph 77) "It is often said or assumed that boys enjoy no significant athletic advantage over girls before puberty. However, this is not true. Writing in their seminal work on the physiology of elite young female athletes, McManus and Armstrong (2011) reviewed the differences between boys and girls regarding bone density, body composition, cardiovascular function, metabolic function, and other physiologic factors that can influence athletic performance. They stated, 'At birth, boys tend to have a greater lean mass than girls. This difference remains small but detectable throughout childhood with about a 10% greater lean mass in boys than girls prior to puberty.' (28) 'Sexual dimorphism underlies much of the physiologic response to exercise,' and most importantly these authors concluded that, 'Young girl athletes are not simply smaller, less muscular boys.' (23)." Dr. Shumer faults me for not noting that the McManus paper found no difference between the sexes in measures of *some other physical characteristics*. But I never claimed that prepubertal boys and girls are physically different in *every* respect. What I claimed—and what the McManus citation supports—is that prepubertal boys and girls are different in *some* areas that contribute to athletic performance. McManus found measurable differences between prepubertal boys and girls in body fat mass, percent body fat, lean

body mass, peak oxygen uptake, maximal pulmonary ventilation, blood volume, cardiac function.

6. I would therefore like to provide the following further quotations from McManus and Armstrong supporting my reading of the paper that boys enjoy a significant athletic advantage over girls before puberty.

7. "Small sex differences in fat mass and percent body fat are evident from mid-childhood…" (at 27) – Mid childhood is considered to be ages 6-12. This statement is used by Dr. Shumer in an endeavor to discredit my expert report, when indeed it supports my report. "Small differences" is an ambiguous term, yet athletic advantages are often the sum of many small differences (as pointed out in my declaration, differences of 3-5% are often more than the difference between a gold medal and no medal, see paragraphs 111-112). Furthermore, the magnitude of an advantage is not a deciding factor in whether that advantage is or is not allowed in sports. Anabolic-Androgenic steroids provide a 5-20% advantage in muscle *strength* (Hartgens and Kuipers, 2004) and are almost universally banned as *performance* enhancing substances. Androstenedione was sold as a testosterone enhancing nutritional supplement in the late 1990s and early 2000s and was banned as a performance enhancing substance even though research shows that androstenedione intake does not enhance the adaptations to resistance training (King et al. 1997, Brown et al. 2000). Fastskin swimming suits provide a 3.2 ± 2.4% performance benefit in swimming (Chatard and Wilson, 2008), and are banned from use by FINA.

8. "At birth, boys tend to have a greater lean mass than girls. This difference remains small but detectable throughout childhood with about a 10% greater lean mass in boys than girls prior to puberty." (at 28)

9. "In comparison to boys, girls are characterised with a smaller absolute peak VO_2. Predicted values range from 1.5 to 2.2 litres•min^{-1} in 10- to 16-year-old girls and are lower than boys by 11, 19, 23 and 27% at ages 10, 12, 14 and 16 years of age, respectively." (at 30) Peak VO_2 is an estimation of maximal oxygen consumption (called VO_2max), which accounts for 30-40% of performance in endurance exercise. Puberty is not typically

experienced by boys or girls by 10 years of age.

10. "In children, like adults, exercise pulmonary gas exchange depends on pulmonary ventilation (VE) and at maximal work rates high rates of ventilation are usual. Maximal values of 49– 95 litres•min^{-1} have been recorded for girls between the ages of 9 and 16 years [] and there is a consistent sex difference with values somewhat higher in boys (58– 105 litres • min^{-1}) for the same age span." "Maximum ventilation remains higher in boys, whether controlled for body size using a ratio standard or allometric adjustment with either stature and/or body mass []. Thus, the higher peak VO_2 in boys is indeed supported by a higher VE." (at 31)

11. When describing differences in blood volume per unit of body mass: "When normalised using a ratio standard with body mass, differences between girls and boys were apparent from about 6 years of age, with values lower in the girls." (at 32)

12. "There are clear differences in cardiac function at rest and during exercise between girls and boys, with differences apparent even prior to puberty. The electrical conduction system is influenced by sex steroid hormones, with girls normally having higher resting heart rates than boys – somewhere in the magnitude of 90 beats per minute at around 10-12 years of age []. This is thought to relate to intrinsic differences in the sinus node pacemaker [], a difference notable at birth with newborn boys displaying lower baseline heart rates than girls []. The higher resting heart rate in girls is often explained as an artefact of differences in cardiac dimensions, and indeed the ratio of heart mass to body mass has been found to be higher in boys than girls at birth, remaining so through adolescence []. Heart volume has also been found to be greater in boys with values of 342 and 403 ml for pre-pubertal girls and boys, respectively…" (at 32)

13. "Data recently published from a thoracic impedance measure of peak C[ardiac]I[ndex] and MRI markers of cardiac size [] demonstrated that pre- pubertal boys had a 16.7% higher (a- v O_2) difference than girls." (at 34) – Cardiac index is an assessment of the cardiac output value based on the patient's size. Cardiac output is the volume of blood the heart pumps per minute. (a-v O_2) difference is the arterio-venous oxygen

difference, and measures how well the tissues extract oxygen from the blood stream. (a-v O_2) difference accounts for roughly 40-50% of maximal oxygen consumption.

14. "Results showed phase II pVO_2 kinetics were approximately 20% slower in pre- pubertal girls compared to boys … This is suggestive of a lower tolerance of fatigue in the girls" (at 35) – pVO_2 stand for Pulmonary Oxygen Uptake, and pVO_2 kinetics provides an insight into the integrated capacity of an organism to transport and utilize oxygen to support an increased rate of energy turnover in contracting muscle cells.

15. "To summarise, there are differences between boys and girls in the aerobic responses to exercise which cannot be accounted for solely by size." (at 35)

16. Dr. Shumer states (at paragraph 5) that the article by McManus and Armstrong is published in the journal *Medicine and Science in Sports and Exercise* (which is the flagship journal for the American College of Sports Medicine). The referenced article by McManus & Armstrong is actually published in *Medicine and Sport Science*, which is a book series (not a journal) and is not in any way affiliated with the American College of Sports Medicine.

17. At paragraph 6, Dr. Shumer states "Dr. Brown gives the false impression that all 22 of the peer-reviewed publications demonstrated differences on total body fat. Instead, Staiano and Katzmarzyk expressly note that 'not all studies demonstrate sex differences in T[otal]B[ody]F[at] before puberty.'" Dr. Shumer contends that my report is deceptive because Staiano's conclusion—that prepubertal girls tend to have more body fat (which is exactly what the article says: "In prepubertal children, girls typically have more T[otal] B[ody] F[at] than boys.")—was not based on unanimous evidence, but rather on the weight of the evidence. Staiano noted that, of the 22 studies reviewed, four of them found similar body fat between boys and girls. Staiano suggested that these studies were influenced by a failure to control for "other influences like age, maturational status and obesity status." In any event, I did not claim that the evidence was unanimous; I simply cited the peer-reviewed conclusion reached by Staiano based on 18 of the 22 studies Staiano reviewed. That isn't deceptive. And experts do not need unanimity to reach a reliable conclusion;

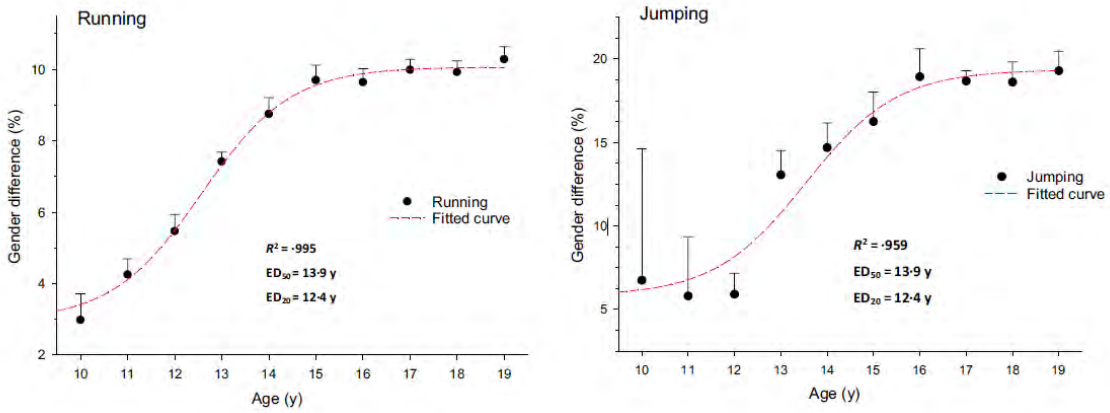
rather, they are to look to the great weight of the evidence, which is exactly what I did.

18. In paragraphs 7 and 8, Dr. Shumer criticizes my partial use of a statement from Handelsman: "Dr. Brown further misrepresents Handelsman (2018)'s findings, notably omitting key portions from the study he cites. Dr. Brown writes, '[t]here is convincing evidence that the sex differences in muscle mass and strength are sufficient to account for the increased strength and aerobic performance of men compared with women and is in keeping with the differences in world records between the sexes.' (Brown Decl. ¶ 59; Brown Hecox Decl. ¶ 88.) But Dr. Brown omits the following sentence from Handelsman which explains that '[t]he basis for the sex difference in muscle mass and strength is the sex difference in circulating testosterone.' David Handelsman, et al. Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance, 39 Endocrine Revs. 803, 816 (2018) (emphasis added)." The second half of that sentence is purposefully omitted as I do not agree with the proposition that testosterone is the only factor responsible for sex-based differences in athletic performance. Indeed, I dedicate many pages of my expert report to demonstrating that there are sex-based differences in athletic performance before puberty citing numerous sources and providing many tables of data. In many places within my report, I acknowledge that puberty driven increases in testosterone in males causes large increases in the differences in athletic performance between males and females (for example, see paragraphs 126-130), so to omit a partial sentence from a single source is hardly misleading.

19. I would like to point out that after paragraph 127, I include the lower panel of figure 2 from Handelsman (2017) which shows "Fitted sigmoidal curve plot of gender differences in performance (in percentage) according to age (in years) in running, jumping and swimming events as well as serum testosterone. Data shown as mean and standard error of the mean of the pooled gender differences by age."

20. I would like to add the upper panel to figure 2 from Handelsman (2017) (see below), which shows Gender differences in performance (in percentage) according to age (in years) in running events including 50 m, 60 m, 100 m, 200 m, 300 m, 400 m, 500 m,

600 m, 800 m, 1000 m, 1500 m, 1 mile, 2000 m, 3000 m and 2 miles (upper left panel) and in jumping events including high jump, pole vault, triple jump, long jump and standing long jump (upper right panel). This figure demonstrates an average male performance advantage of ~3% in running at age 10, ~4% at age 11, and ~5% at age 12, and this figure also demonstrates an ~6% male advantage in jumping at age 10, and ~5% at ages 11 and 12. Extrapolating the error bars in these graphs (which represent the standard deviation, it is very reasonable to expect that a majority of boys at ages 10, 11, and 12 will outperform girls of the same age.



21. In paragraphs 9-12, Dr. Shumer refers to an article written for "The Conversation" (a network of not-for-profit media outlets publishing news stories and research reports online, with accompanying expert opinion and analysis written by academics and researchers) summarizing "research published in American Academy of Neurology Journal." It is important to note that the research published in the journal *Neurology*, published by the American Academy of Neurology, referenced by Dr. Shumer evaluated "Twelve functional outcome measures were collected from 1,000 healthy individuals aged 3-101 years", and did not specifically focus on children or adolescents. In The Conversation, it is explained that "As part of wider research to assess people's physical capabilities across the lifespan, we tested 300 children and adolescents between the ages of 3 and 19."[1]

[1] https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328

22. In contrast to the above citation by Dr. Shumer to a single study with "300 children and adolescents between the ages of 3 and 19," my report cited peer-reviewed research publications of a range of sample sizes focusing on children, not children as "part of wider research to assess people's physical capabilities across the lifespan."

23. Here are some of the studies I cited:

a. In a test of reaction time in Spanish Preschool children (1,845 girls and 1,896 boys with a mean age of 55.93 ± 11.14 months) boys performed better than girls (paragraphs 43 and 114 referencing Latorre-Roman et al. 2018)

b. A summary of data from "a national sample of Canadian children and youth" ages 7-17 years demonstrating that boys have higher aerobic power than girls of the same age (Paragraph 80, Citing Malina et al. 2004, and Gauthier et al. 1983).

c. In an evaluation "of 703 male and female elite young German athletes aged 8-18" (1) "fitness development precedes sports specialization" (2) and "males outperformed females in C[ounter]M[ovement]J[ump], D[rop]J[ump], C[hange]o[f]D[irection speed] performances and hand grip strength." (Paragraph 81 Citing Lesinski et al. 2020).

d. A total of 424,328 Greek boys and girls aged 6-18 years using standing long jump to measure lower body explosive power, sit and reach to measure flexibility, timed 30 second sit ups to measure abdominal and hip flexor muscle endurance, 10 x 5 meter shuttle run to evaluate speed and agility, and multi-stage 20 meter shuttle run test to estimate aerobic performance. For each of the fitness tests, performance was better in boys compared with girls, except for the sit and reach test. (Paragraphs 82 -84, 100, Citing Tambalis 2016).

e. USA Presidential Fitness Testing data for 85th and 50th percentile demonstrating that boys perform better on tests of muscle strength and running endurance (Paragraph 85-86).

f. An evaluation of 85,000 Australian children aged 9-17 years old showed that, compared with 9-year-old females, 9-year-old males were faster over

short sprints (9.8%) and 1 mile (16.6%), could jump 9.5% further from a standing start (a test of explosive power), could complete 33% more push-ups in 30 [seconds] and had 13.8% stronger grip. (Paragraphs 88-89, citing Catley & Tomkinson, 2013).

g. Evaluation of the "Eurofit" test battery on children from 30 European countries and 2,779,165 test performances in 9-17 year old boys and girls showed that boys performed better than similarly aged girls at each age on tests of muscular strength, muscular endurance, and aerobic fitness (Paragraphs 90-93, citing Tomkinson 2018).

h. An evaluation of 20m shuttle run performance in 1,142,026 children aged 9-17 in 50 countries showing that boys performed better than girls of the same age (paragraphs 94-95, citing Tomkinson et al., 2017).

i. An evaluation of 10,302 children aged 6-10.9 years of age, from the European countries of Sweden, Germany, Hungary, Italy, Cyprus, Spain, Belgium, and Estonia demonstrating that boys performed better than girls in speed, lower- and upper-limb strength and cardiorespiratory fitness. (Paragraphs 97-99, citing De Miguel-Etayo et al. 2014).

j. An evaluation of 18 studies for males (N=5676 in total) and 17 studies for females (N=5489 in total) in the United States and Canada demonstrating the boys had strength advantages of between 13 and 28 percent, with the remaining outlier recording only a 4% advantage for 7-year-old boys (Paragraph 101, citing Silverman 2011).

k. An analysis of vertical jump measurements of 7,614 healthy Colombian schoolchildren aged 9 -17.9 years of age, showing that boys jump higher than girls of the same age (Paragraph 103, citing Ramírez-Vélez et al, 2017).

l. An analysis of vertical jump measurements of 1,845 children aged 10-15 years in primary and secondary schools in the East of England, showing that boys jump higher than girls of the same age (Paragraph 104, citing Taylor 2010).

m. Data from USA Track & Field (Paragraphs 107, 108).

n. Data from Athletic.net for the USA (Paragraph 109, 110).

o. Data from 366 Danish boys and 332 Danish girls between the ages of 6 and 7 years old showing that boys have higher measurements of aerobic fitness, even if the boys and girls engage in the same amount of physical activity (Paragraph 113, citing Eiberg 2005).

24. In Paragraph 10, Dr. Shumer contends that age, location, or socioeconomic factors have not been controlled for in the above-referenced studies. This is quite simply not so, as the vast majority of these papers compared the performance of children of the same age (as demonstrated in the normative data presented in paragraphs 77-115 and the appendix of my declaration), and, as explained above, the male advantages have been documented in a wide range of countries.

25. To further demonstrate that prepubertal boys exhibit advantages in measure of physical fitness and motor control which give them advantages in sports compared to girls of the same age, here are even more papers:

a. A Systematic Review and Meta-Analysis of 38 articles studies were carried out in 19 different countries (Australia, Belgium, Brazil, Britain, China, Croatia, Germany, Iran, Indonesia, Ireland, Japan, Korea, Myanmar, Poland, Portugal, Puerto Rico, Singapore, South Africa, and the USA representing data for 8394 children ages 3-6 years old who were assessed for object control skills. Significant differences were found, favoring boys vs. girls at ages 3, 4, 5, and 6 with at least some of the differences attributable to biology (Zheng et al, 2022).

b. 1,682 children and adolescent aged 6-17 years from central Spain, divided into prepubertal and pubertal groups based on Tanner stages demonstrating that pre-pubertal boys had more muscle mass, less fat mass, and performed better girls on tests of countermovement jump, handgrip strength, and 20 m shuttle run (Manzano-Carrasco et al. 2022).

c. 3,179 preschool children (1678 boys) ages 2.8-6.4 years from 10 different cities and towns in Spain and found boys outperformed girls in the 20 m

shuttle run, handgrip strength, standing long jump, and 4X 10 m shuttle run (Cadenas-Sanchez, 2019).

d. 31,484 children (16,023 boys and 15,461 girls) ages 6-11 years old from a representative sample of the French population with boys performing better on tests of Cardiorespiratory fitness, muscular endurance, and speed (Vanhelst et al. 2020).

e. 341 young Nigerian children (ages 3 to 5) At each age level the boys consistently performed better than the girls tests of catching, standing long jump, tennis ball throw and speed run (Toriola and Ingokwe, 1986).

f. 434 low-income preschool children from Santiago Chile (246 boys; 5.48 ± 0.31 years) showing that boys were heavier and taller thang girls, with boys performing better on handgrip strength test, standing long jump. and 20 m sprint (Cadenas-Sanchez, 2015).

26. It is also important to note that sports do not take into account socioeconomic factors or location. For example, at a youth wrestling tournament the athletes may be categorized based on sex, age, and body weight, but not socioeconomic status or location. In a youth soccer tournament, the athletes may be categorized based on sex, age, or possibly the team skill rating, but not socioeconomic status or location.

27. In Paragraph 12, Dr. Shumer claims that there has been wide replication of the lack of difference in sporting performance between prepubertal boys and girls and states that there is a general consensus that there are no sex-based differences in athletic performance before puberty, and yet cites only two sources. Neither of these sources professes to present a scientific consensus statement on the presence or lack of sex-based difference in performance before puberty.

28. In reading Senefeld et al., Sex Differences in Youth Elite Swimming, 14 PLOS ONE 1, 1–2 (2019), these authors cite only two sources regarding the sex-based differences in sporting performance in 10 to 12-year-olds, one of which is the Handelsman (2018) paper and the other is a paper by Tonnessen et al. (Performance development in

adolescent track and field athletes according to age, sex and sport discipline. PloS one. 2015;10(6):e0129014). Indeed, Senefeld et al. state "However, the sex-based differences in performance prior to age 10 are unknown …" (at page 2) and "However it is clear that these data provide one of the only examples of faster (or at least not slower) sports performance for girls than boys." (at page 8)

29. In paragraph 13, Dr. Shumer describes these data as "Demographic Data", which is incorrect. Demographic data are used to help understand the statistical characteristics of human populations. Demographic data can contain specific information about the characteristics of a given population, such as the following: age range, race and ethnicity, sex, gender, level of education, income, employment status, occupation, homeownership, birth rates, death rates, marriage rates, religious affiliation, political affiliation, spoken language, geographic location, or hobbies and interests. Many of the studies which Dr. Shumer calls demographic data are normative data, which are information from a population of interest that establishes a baseline distribution of results for that particular population (Lee & Schuele, Abdi & Williams, Encyclopedia of Research Design, Sage Publications, 2010).

30. The competition data presented in my report represent the under-8 and 9 to 10-year-old records from USA Track & Field, and annual performance data gleaned from Athletic.net for the State of Arizona in 2022, and for the entire United States in 2021.

31. I recently (June 2, 2023) presented research at the 2023 annual meeting of the American College of Sports Medicine using nationwide results from Athletic.net demonstrating that over the years 2017-2021, the top 10 boys ages 7-8 and 9-10 ran faster than girls of the same ages and jumped higher and father in 100m, 200m, 400m, 800m, 600m, high jump and long jump by 3-10% than the girls in every event every year (Brown GA, Brown CJ, Shaw I, Shaw B. Boy and Girls Differ in Track and Field Event Performance Before Puberty. 70th Annual Meeting of the American College of Sports Medicine. Denver CO. Presentation 2577. May 30 – June 2, 2023). There was another presentation in the same session in which the authors used data from Athletic.net for the

top 10 male and female athletes for the years 2019, 2020, and 2021 for ages 7-18 years, and observed that prepubertal males outperformed females of the same age by 3-10% in the 100m, 200m, 400m, 800, high jump and long jump every year and overall (Atkinson MA, Linde JJ, Hunter SK. Sex Differences in Performance of Elite Youth Track and Field Athletes. 70th Annual Meeting of the American College of Sports Medicine. Denver CO. Presentation 2572. May 30 – June 2, 2023). This demonstrates that (1) data from Athletic.net are considered sufficiently reliable for scholarly endeavors, and (2) prepubertal male advantages in running and jumping are consistently demonstrated in elite youth.

32. Additionally, The Motivational Times, from USA Swimming, show under 10-year-old boys consistently swimming faster than under 10-year-old girls.[2]

33. In paragraphs 14-27, Dr. Shumer contends that "Transgender girls who receive puberty suppressing medication at the onset of puberty have no athletic advantage over other girls." While Dr. Shumer is correct to state that there is no research showing puberty suppression and or cross sex hormones does not eliminate male athletic advantages, similarly there is no research showing it does. Dr. Shumer does not cite any studies showing that puberty suppression results in transgender girls exhibiting athletic performance that is the same as equally aged, gifted, and trained females. Dr. Shumer attempts to deflect the research showing the males who take puberty blockers and/or cross sex hormones retain male pattern advantages in lean body mass, muscle strength, body height, and so forth, by stating that this research does not demonstrate athletic advantages. In this he ignores the commonly held tenet in the professions of exercise physiology and strength & conditioning that lean body mass is one the major factors driving athletic performance overall, and driving the sex-based differences in athletic performance (see my declaration, paragraphs 61 and 81 for explanation of this tenet).

34. Overall, athletes spend an inordinate amount of time in the weight room, on the track, in the pool, etc. trying to improve their physical fitness, because improved

[2] https://swimswam.com/usa-swimming-releases-age-group-motivational-times-for-2021-2024/

physical fitness translates to improved athletic performance. Whether it is measured as a higher VO_2max in an endurance athlete, a higher 1-repetion maximum for a thrower or wrestler, or a larger amount of lean body mass in almost any athlete, these measures of improved physical fitness are indicators of a greater potential for successful athletic performance. The differences in physical fitness between males and females before and after puberty predispose males to a winning performance if they were to compete against females of the same age who have the same training and sports background.

35. Lean body mass is a significant determinant of muscle strength and sports performance. As demonstrated by Almiray-Stot et al. (2022), in healthy children ages 5 to 19-years-old, lean body mass is significantly correlated to muscle strength in both boys and girls "Highly positive correlations of muscle strength with lean mass in upper limbs were found r-values 0.87-0.92 for boys and r = 0.80-0.86 for girls. High and moderate positive correlations for lower limbs were also noted for upper limbs: r = 0.74-0.86 for boys and r = 0.67-0.82 for girls." (at 597). And, as observed by Zaras et al. (2020) in well trained adult female weightlifters: "Very large to nearly perfect correlations were found between snatch and clean and jerk for trunk lean body mass (r = 0.959 and 0.929) (at 1)." The connection between lean body mass and muscle strength is quite clear, and the muscle strength is very important to sports performance as stated by Comfort et al. (2023) in the *National Strength and Conditioning Association Position Statement on Weightlifting for Sports Performance*, "strength underpins performance in athletic tasks." (at 1165)

36. In paragraph 19, Dr. Shumer cites the paper by Harper on Race Times for Transgender Athletes as evidence that testosterone suppression and/or cross sex hormones eliminates male advantages. Please see my report, paragraphs 155-159, for an explanation of some of the numerous problems with the data from Harper. Also see my report paragraphs 151-152 for analysis of the papers by Roberts et al. and Chicarelli et al. regarding running times in transgender air force personnel, in which there is at least objective evaluation of endurance performance in transwomen. Also see paragraph 169 for an explanation of the work by Alvares on VO_2max in transwomen.

37. In paragraphs 28-31, Dr. Shumer claims that the pre-pubertal male athletic advantages are not due to "minipuberty". At no point in my declaration are the male athletic advantages differences ascribed to "minipuberty" (indeed, the term "minipuberty" is not found within my expert report).

38. It is important to note that in their initial declarations, and in their rebuttal statements, neither Dr. Stephanie Budge nor Dr. Daniel Shumer cited any peer reviewed publications or presented any data demonstrating that the use of gonadotropin-releasing hormone (GnRH) analogues (aka puberty blockers) prevent juvenile males from developing male sex-based advantages in sports performance. Specifically, neither Dr. Budge nor Dr. Shumer showed that the administration of puberty blockers causes males to cease developing sex-based differences in lean body mass, body height, muscle strength, muscle endurance, aerobic fitness, or any measure of sports-specific performance that gives males large athletic advantages over comparably aged, gifted and trained females before and after puberty.

39. In contrast, I presented considerable data and cited numerous peer reviewed publications demonstrating that males have advantages in physical fitness and sports performance before (see paragraphs 77-115, and the appendix, and the additional information in my rebuttal to Dr. Shumer) and after puberty (see paragraphs 7-73). I also cited and briefly summarized peer-reviewed publications demonstrating that administering puberty blockers does not erase male sex-based advantages in lean body mass (see my declaration paragraphs 117-121) and body height (see my declaration paragraphs 124 & 125).

I swear or affirm, under penalty of perjury, that the foregoing is true and correct.

Dated: June 29, 2023 */s/ Dr. Gregory A. Brown, Ph.D., FACSM*

Exhibit 18

Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance

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ABSTRACT Elite athletic competitions have separate male and female events due to men's physical advantages in strength, speed, and endurance so that a protected female category with objective entry criteria is required. Prior to puberty, there is no sex difference in circulating testosterone concentrations or athletic performance, but from puberty onward a clear sex difference in athletic performance emerges as circulating testosterone concentrations rise in men because testes produce 30 times more testosterone than before puberty with circulating testosterone exceeding 15-fold that of women at any age. There is a wide sex difference in circulating testosterone concentrations and a reproducible dose-response relationship between circulating testosterone and muscle mass and strength as well as circulating hemoglobin in both men and women. These dichotomies largely account for the sex differences in muscle mass and strength and circulating hemoglobin levels that result in at least an 8% to 12% ergogenic advantage in men. Suppression of elevated circulating testosterone of hyperandrogenic athletes results in negative effects on performance, which are reversed when suppression ceases. Based on the nonoverlapping, bimodal distribution of circulating testosterone concentration (measured by liquid chromatography mass spectrometry) and making an allowance for women with mild hyperandrogenism, notably women with polycystic ovary syndrome (who are overrepresented in elite athletics) the appropriate eligibility criterion for female athletic events should be a circulating testosterone of <5.0 nmol/L. This would include all women other than those with untreated hyperandrogenic disorders of sexual development and noncompliant male-to-female transgender as well as testosterone-treated female-to-male transgender or androgen dopers. ***(Endocrine Reviews 39: 803 – 829, 2018)***

Virtually all elite sports are segregated into male and female competitions. The main justification is to allow women a chance to win, as women have major disadvantages against men who are, on average, taller, stronger, and faster and have greater endurance due to their larger, stronger muscles and bones as well as a higher circulating hemoglobin level. Hence, elite female competition forms a protected category with entry that must be restricted by an objective eligibility criterion related, by necessity, to the relevant sex specific physical advantages. The practical need to establish an eligibility criterion for elite female athletic competition led the International Association of Athletic Federations (IAAF) to establish a rule in 2011, endorsed by the International Olympic Committee (IOC) in 2012, for hyperandrogenic women. That IAAF regulation stated that for athletes to be eligible to compete in female events, the athlete must be legally recognized as a female and, unless she has complete androgen insensitivity, maintain serum testosterone <10 nmol/L. That IAAF eligibility rule was challenged by an athlete to the Court for Arbitration in Sports, which ruled in 2015 that, although an eligibility criterion was justified, there was insufficient evidence of the extent of the competitive advantage enjoyed by hyperandrogenic athletes who had circulating testosterone >10 nmol/L over female athletes with circulating testosterone in the normal female range. The Court for Arbitration in Sports suspended the rule pending receipt of such evidence. In that context, the present review presents the available evidence on the hormonal basis for the sex difference

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ESSENTIAL POINTS

- It is widely accepted that elite athletic competitions should have separate male and female events
- The main justification is that men's physical advantages in strength, speed, and endurance mean that a protected female category, with objective entry criteria, is required
- Prior to puberty, there is no sex difference in circulating testosterone concentrations and athletic performance
- From male puberty onward, the sex difference in athletic performance emerges as circulating testosterone concentrations rise as the testes produce 30 times more testosterone than before puberty, resulting in men having 15- to 20-fold greater circulating testosterone than children or women at any age
- This wide, bimodal sex difference in circulating testosterone concentrations and the clear dose-response relationships between circulating testosterone and muscle mass and strength, as well as the hemoglobin level, largely account for the sex differences in athletic performance
- Based on the nonoverlapping, bimodal distribution of circulating testosterone concentration (measured by liquid chromatography mass spectrometry) with 95% references ranges of 7.7 to 29.4 nmol/L in healthy men and 0 to 1.7 nmol/L in healthy premenopausal women making an allowance for women with the mild hyperandrogenism of polycystic ovary syndrome, who are overrepresented in elite athletics the eligibility criterion for female athletic events should be a circulating testosterone concentration of <5.0 nmol/L

in athletic performance. It concludes that the evidence justifies a revised eligibility criterion of a threshold circulating testosterone concentration of 5 nmol/L (measured by a mass spectrometry method).

Sex, Fairness, and Segregation in Sport

If sports are defined as the organized playing of competitive games according to rules (1), fixed rules are fundamental in representing the boundaries of fair sporting competition. Rule breaking, whether by breaching eligibility or competition rules, such as use of banned drugs, illegal equipment, or match fixing, creates unfair competitive advantages that violate fair play. Cheating constitutes a fraud against not just competitors but also spectators, sponsors, the sport, and the public. In the absence of genuine fair competition, elite sports would lose their wide popular appeal and ability to captivate and inspire with the authentic attraction of genuine contest between highly trained athletes.

Nevertheless, fairness is an elusive, subjective concept with malleable boundaries that may change over time as social concepts of fairness evolve. For example, until the late 19th century when organized sports trainers emerged, training itself was considered a breach of fairness because competition was envisaged at that time as a contest based solely on natural endowments. Similarly, sports once distinguished between amateurs and professionals. The concept of fairness has deep and complex philosophical roots mainly focused on notions of distributive justice. These considerations affect sports through the universal application of antidiscrimination and human rights legislation. Less attention is given to the philosophical basis of fair competition in elite sports, where the objectives are not egalitarian but aim to discover a hierarchy of achievement derived from a mixture of unequal natural talent and individual training effort. Excellent, insightful discussion of the legal and moral complexities of sex and fair competition in elite sports from a legal scholar and former elite female athlete is available (2).

The terms *sex* and *gender* are often confused and used as if interchangeable. *Sex* is an objective, specific biological state, a term with distinct, fixed facets, notably genetic, chromosomal, gonadal, hormonal, and phenotypic (including genital) sex, each of which has a characteristic defined binary form. Whereas all facets of biological sex are almost always aligned so that assignment of sex at birth is straightforward, rare instances in which two or more facets of biological sex conflict constitute an intersex state, now referred to as disorders (or differences) of sex development (DSDs) (3). In contrast, *gender* is a subjective, malleable, self identified social construct that defines a person's individual gender role and orientation. Prompted by biological, personal, and societal factors, volitional expression of gender can take on virtually any form limited only by the imagination, with some individuals asserting they have not just a single natal gender but two genders, none, a distinct third gender, or gender that varies (fluidly) from time to time. Hence, whereas gender is usually consistent with biological sex as assigned at birth, in a few it can differ during life. For example, if gender were the basis for eligibility for female sports, an athlete could conceivably be eligible to compete at the same Olympics in both female and male events. These features render the unassailable personal assertion of gender identity incapable of forming a fair, consistent sex classification in elite sports.

The strongest justification for sex classification in elite sports is that after puberty men produce 20 times more testosterone than women (4 7), resulting in circulating testosterone concentrations 15 fold higher than in children or women of any age. Age grade competitive sporting records show no sex differences prior to puberty, whereas from the age of male puberty onward there is a strong and ongoing male advantage (8). The striking male postpubertal increase in cir culating testosterone provides a major, ongoing, cu mulative, and durable physical advantage in sporting contests by creating larger and stronger bones, greater muscle mass and strength, and higher circulating hemoglobin as well as possible psychological (be havioral) differences. In concert, these render women, on average, unable to compete effectively against men in power based or endurance based sports.

Sex classification in sports therefore requires proof of eligibility to compete in the protected (female) category. This deceptively simple requirement for fairness is taken for granted by peer female compet itors who regard participation by males, or athletes with physical features closely resembling males, as unfair. This makes policing of eligibility inescapable for sports, to avoid unfair male participation in female events. However, such policing inevitably intrudes into highly personal matters so that it must be achieved with respect for dignity and privacy, demanding use of the least invasive, scientifically reliable means. Un surprisingly, this dilemma has always been highly contentious since it first entered international elite sports in the early 20th century, and it has become increasingly prominent and contentious in recent decades; nevertheless, the requirement to maintain fair play in female events will not disappear as long as separate female competitions exist. During recent decades, there has been progressively better un derstanding of the complex biology of genetic sex determination and the impact of pubertal sexual maturation in establishing phenotypic sex ual dichotomy in physical capabilities. These sex dichotomous physical features form the basis of, but remain quite distinct from, adult gender roles and identity. During the last century, as knowledge grew, the attempts to formalize a scientific basis for the unavoidable necessity of policing eligibility for the female category have been continually challenged. Most recently, the increasing assertion of gender self identification as a social criterion has further chal lenged the hegemony of biology for determining "sports sex," Coleman's apt term (2). Allowing sub jective gender self identification to become the sole criterion of sports sex would allow for gaming and perceptions of systematic unfairness to grow. The case for women's sports being defined by sex rather than gender, including the consequences of acceding to gender based classification, has been outlined (9) in arguing the importance of proper medical management of athletes intending to compete in female events.

Separate male and female events in sports is a dominant form of classification that is superimposed on other graduated age group and weight classifica tions (*e.g.*, in weightlifting, power lifting, wrestling, boxing, rowing), which reflect differences in strength, power, and speed to ensure fairness in terms of op portunity to win and, additionally, safety in contact sports. Age and weight classifications rely on objective criteria (birth date, weigh in weight) for eligibility, and so should sex classification. Nevertheless, some power sports dependent on explosive strength and power (*e.g.*, throwing events, sprinting) do not segregate weight classes, whereas other sports where height is an advantage (*e.g.*, basketball, jockeys) do not have height classifications. These sports disproportionately attract athletes with greater weight and/or power to weight ratio or advantageous stature, respectively. If sex classification were eliminated, such open or mixed competitions would be dominated almost exclusively by men. It therefore seems highly unlikely that sex classification would ever be discarded, despite calls on philosophical or sociological grounds to end "gender" classification in sport (10).

Sex Difference in Circulating Testosterone Levels

Testosterone biosynthesis, secretion, and regulation in men and women

An androgen is a hormone capable of developing and maintaining masculine characteristics in reproductive tissues (notably the genital tract, as well as in other tissues and organs associated with secondary sexual characteristics and fertility) and contributing to the anabolic status of nonreproductive body tissues (11). The two dominant bioactive androgens circulating in mature mammals, including humans testosterone and its more potent metabolite DHT account for the development and maintenance of all androgen dependent characteristics, and their circulating levels in men and nonpregnant women arise from steroids synthesized *de novo* in the testes, ovary, or adrenals (12).

The sexually undifferentiated gonads in the em bryo develop into either ovaries or testes according to whether a Y chromosome (or at least the *sry* gene) is present. After birth and until puberty commences, circulating testosterone concentrations are essentially the same in boys and girls, other than briefly in the neonatal period of boys when higher levels prevail. The onset of male puberty, a brain driven process triggered by a still mysterious hypothalamic or higher cerebral mechanism (13), initiates a hormonal cascade. In males, this leads to enhanced pituitary LH secretion that stimulates the 500 million Leydig cells in the testes

to secrete 3 to 10 mg (mean, 7 mg) of testosterone daily (4, 6, 7, 14, 15). This creates a very high local concentration of testosterone within the testis as well as a steep downhill concentration gradient into the bloodstream that maintains circulating testosterone levels at adult male levels, which are tightly regulated by strong negative hypothalamic feedback of circulating testosterone. In the absence of testes, these mechanisms do not function in females. In girls, serum testosterone increases during puberty (16), peaking at age 20 to 25 years before declining gradually with age (17, 18), but it remains <2 nmol/L at all ages, as determined by a reliable method (see below).

In adult women, circulating testosterone is derived from three roughly equal sources: direct secretion from the adrenal gland or the ovary and indirect extraglandular conversion (in liver, kidney, muscle, fat, skin) from testosterone precursors secreted by the adrenal and ovary. Only when circulating testosterone concentrations rise in male adolescents above the prepubertal concentrations does the virilization characteristic of men commence, progress, and endure throughout adult life, at least until old age (18). In combination, these different sources produce ~0.25 mg of testosterone daily so that throughout life women maintain circulating testosterone levels of <2 nmol/L. Circulating testosterone concentrations in women are subject to little dynamic physiological regulation. As a result, circulating testosterone concentrations in healthy premenopausal women are stable (nonfluctuating) and not subject to strong negative feedback by exogenous testosterone (as happens in men). Even the small rise (50%) at the time of the mid cycle LH surge triggering ovulation (19) remains within the physiological range for premenopausal females.

Male and female reference ranges for circulating testosterone

A reliable threshold for circulating testosterone must be set using measurement by the reference method of liquid chromatography mass spectrometry (LC MS) rather than using one of the various available commercial testosterone immunoassays. The necessary reliance on steroid mass spectrometry for clinical applications in endocrinology, reproductive medicine, and sports medicine is widely recognized. It has been standard for decades in antidoping science (20), and the growing consensus is that it is required for high quality clinical research and practice recognized by cognate professional societies (21, 22) and editorials in leading clinical endocrinology (23) and reproductive medicine (24) journals. The inherently limited specificity of testosterone immunoassays arises from antibody cross reactivity with structurally related steroids (such as precursors and metabolites) other than the intended target. As a result, all steroid immunoassays, including for testosterone, display method specific bias whereby, for example, the lower limit of a testosterone reference range in healthy young men varies from 7.3 to 12.6 nmol/L according to the immunoassay used, so that no consensus definition of a lower limit could be obtained independent of the commercial immunoassay method used (25). Furthermore, testosterone immunoassays are optimized for circulating levels in men but display increasing inaccuracy at the lower, by an order of magnitude, circulating testosterone concentrations in women or children. In contrast to immunoassays, LC MS based methods are highly specific and do not depend on proprietary antibodies. Using LC MS based measurements, method specific bias can be avoided and a fixed consensus lower reference limit defined (Table 1). Hence, for the precision required in sports medicine, whether for eligibility criteria or antidoping applications, testosterone in serum must be measured by LC MS methods.

Prior to puberty, levels of circulating testosterone as determined by LC MS are the same in boys and girls (16). They remain lower than 2 nmol/L in women of all ages. However, from the onset of male puberty the testes secrete 20 times more testosterone resulting in circulating testosterone levels that are 15 times greater in healthy young men than in age similar women. Using LC MS measurement, circulating testosterone in adults has a strikingly nonoverlapping bimodal distribution with wide and complete separation between men and women. Table 1 (25 36) summarizes data from appropriate reported studies using mass spectrometry based methods to measure serum testosterone in healthy men and women. Based on a number weighted pooling with conventional 95% two sided confidence limits of the eight available studies using LC MS measurements of serum testosterone, the reference range for healthy young men (18 to 40 years) is 7.7 nmol/L to 29.4 nmol/L. Similarly, summarizing the nine available studies for healthy menstruating women under 40 years, the 95% (two sided) reference range is 0 to 1.7 nmol/L. These reference limits do not control for factors such as oral contraceptive use (35, 36), menstrual phase (19), SHBG (37, 38), overweight (39, 40), fasting and smoking (41), diet (40), and physical activity (42, 43) in women and men, all of which have small effects on circulating testosterone but without materially influencing the divergence between the nonoverlapping bimodal distribution of male and female reference ranges of circulating testosterone.

In creating a threshold for eligibility for female events it is also necessary to make allowance for women with polycystic ovary syndrome (PCOS) and nonclassical adrenal hyperplasia. PCOS is a relatively common disorder among women of reproductive ages with a prevalence of 6% to 10%, depending on the diagnostic criteria used (44), in which mild hyperandrogenism is a key clinical feature and has higher than expected prevalence among elite female athletes

Table 1. Serum Testosterone Measurements by LC-MS Methods in Studies of Healthy Men and Women

Study	Sample (Age 18 40 y)	N	Lower 95% CL (nmol/L)	Upper 95% CL (nmol/L)
Men				
Sikaris *et al.*, 2005 (25)	Elite, eugonadal	124	10.4	30.1
Turpeinen *et al.*, 2008 (26)	Convenience	30	10.1	31.2
Kushnir *et al.*, 2010 (27)	Convenience	132	7.2	24.2
Salameh *et al.*, 2010 (28)	Convenience	264	7.1	39.0
Neale *et al.*, 2013 (29)	Convenience	67	10.6	31.9
Kelsey *et al.*, 2014 (30)	Secondary pooled analysis	1058	7.2	25.3
Hart *et al.*, 2015 (31)	Birth cohort	423	7.4	28.0
Travison *et al.*, 2017 (32)	Pooled two cohorts	1656	7.9	31.1
Number weighted mean			7.7	29.4
Women				
Turpeinen *et al.*, 2008 (26)	Convenience	32	0.8	2.8
Kushnir *et al.*, 2010 (27)	Convenience	104	0.3	2.0
Salameh *et al.*, 2010 (28)	Convenience	235	0.03	1.5
Haring *et al.*, 2012 (33)	Population based	263	0.04	2.0
Neale *et al.*, 2013 (29)	Convenience	90	0	1.7
Bui *et al.*, 2013 (34)	Convenience	25	0.30	1.69
Rothman *et al.*, 2013 (19)	Convenience	31	0.4	0.92
Bermon and Garnier, 2017 (35)	Elite athletes	1652	0	1.62
Eklund *et al.*, 2017 (36)	Elite athletes and controls	223	0.26	1.73
Number weighted mean			0.06	1.68

Abbreviation: CL, confidence limit.

(36, 45 47). Nonclassical adrenal hyperplasia is a milder and later (adult) onset variant of classical congenital adrenal hyperplasia (48) with a much higher but still rare population prevalence (1:1000 vs 1:16,000 for the classical variant) (49). Table 2 (50 64) summarizes clinical studies (n = 16, ≥40 women) reporting serum testosterone concentrations measured by LC MS in samples from women with PCOS.

The pooled data reveal that the upper limit of serum testosterone in women with PCOS is 3.1 nmol/L (95% CI, one sided) or 4.8 nmol/L (using a 99.99% CI, one sided) (Table 3). Hence, a conservative threshold for circulating testosterone of 5 nmol/L measured by LC MS would identify <1:10,000 women with PCOS as false positives, based on circulating testosterone measurement alone. Circulating testosterone higher than this threshold is likely to be due to testosterone secreting adrenal or ovarian tumors, intersex/DSD, badly controlled or noncompliant male to female (M2F) transgender athletes, or testosterone doping.

The physiological effects of testosterone depend on the circulating testosterone, not its source (endogenous or exogenous)

Testosterone, whether of a natural endogenous or manufactured exogenous source, has an identical chemical structure and biological effects, aside from minor differences in isotopic composition, which are biologically insignificant. At equivalent doses and circulating levels, exogenous testosterone exerts the same biological and clinical effects on every known androgen responsive tissue or organ as endogenous testosterone, apart from effects on spermatogenesis, which as discussed below is only a matter of degree. Consequently, exogenous testosterone is a fully effective substitute for endogenous testosterone in therapeutic use, countering the effects of testosterone deficiency due to hypogonadism (reproductive system disorders). Any purported differences between endogenous and exogenous testosterone are due to corresponding differences in the endogenous production rate or exogenous dose. Such differences in

Data taken directly from paper or interpolated from other data (*e.g.*, median, quartiles, ranges, sample size) supplied as described by Wan *et al.*, 2014 (Estimating the sample mean and standard deviation from the sample size, median, range and/or interquartile range. BMC Med Res Methodol 14: 135) are shown in italics.

Table 2. Summary of Serum Testosterone (nmol/L) by LC-MS in Women With PCOS From 16 Studies

Study	N	Mean	SD
Moran *et al.*, 2017 (50)	92	*0.24*	*0.08*
Münzker *et al.*, 2017 (51)	274	*0.93*	*0.19*
O'Reilly *et al.*, 2017 (52)	114	*0.55*	*0.19*
Handelsman *et al.*, 2017 (53)	152	*0.38*	*0.25*
Pasquali *et al.*, 2016 (54)	156	*1.17*	*0.47*
Yang *et al.*, 2016 (55)	1159	*2.2*	*1.44*
Tosi *et al.*, 2016 (56)	116	*1.33*	*0.55*
Daan *et al.*, 2015 (57)	170	*1.64*	*0.53*
Bui *et al.*, 2015 (58)	44	*0.85*	*0.3*
Keefe *et al.*, 2014 (59)	52	*1.7*	*0.97*
Yasmin *et al.*, 2013 (60)	165	*1.99*	*1.02*
Janse *et al.*, 2011 (61)	200	*1.12*	*0.47*
Jedel *et al.*, 2011 (62)	72	*0.23*	*0.08*
Legro *et al.*, 2010 (Mayo) (63)	596	*2.12*	*0.89*
Legro *et al.*, 2010 (Quest) (63)	596	*1.98*	*0.97*
Stener Victorin *et al.*, 2010 (64)	74	*1.53*	*0.62*
Sum	4032		
Number weighted mean		*1.69*	*0.87*

effective exposure lead to corresponding differences in circulating testosterone levels and its effects according to the dose response curves for testosterone.

Similar to all hormones and drugs, over their effective range of biological activity the dose response relationship for testosterone is usually a sigmoidal curve with lower and upper plateaus joined by a monotonically rising middle region, which may be linear in the natural scale but more often log linear (linear on the log or similar transformed scale). In the middle portion of the typical sigmoidal dose response curve for the same increase in testosterone dose (or concentration), the response would be increased in simple proportional (*i.e.*, linear) but more often on a logarithmic scale. In contrast, at the lower and upper plateaus of dose or concentrations, changes in testosterone exposure may evoke minimal or no response on the endpoint. For example, in women of any age circulating testosterone concentrations are along the lower plateau of the dose response curve, so that increases in circulating testosterone concentrations within that lower plateau may have minimal or no effect. In female athletes with the mild hyperandrogenism of PCOS, higher performance has been shown (47), with their muscle mass and power performance correlating with androgen levels (36). However, beyond these effects where endogenous testosterone concentrations are in the high normal adult female range, it is only when the increases in circulating testosterone concentrations substantially and consistently exceed those prevailing in childhood (<2 nmol/L) and among women including those with PCOS (<5 nmol/L) that the effects would replicate the effects of rising testosterone concentrations of boys in middle to late puberty (typically >8 nmol/L), that is, the masculinizing effects of increased muscle, bone, and hemoglobin characteristics of men. As shown above, the circulating testosterone of most women never reaches consistently >5 nmol/L, a level that boys must sustain for some time to exhibit the masculinizing effects of male puberty.

In addition, the effects of testosterone are modulated in a form of fine tuning by the patterns of exposure, such as whether the circulating testosterone is delivered in the unphysiological steady state format (*e.g.*, quasi steady state delivery by implant or transdermal products) or by the peak and trough delivery of injections, as opposed to the natural state of endogenous fluctuations in serum testosterone around the average adult male levels. However, these latter pattern effects are subtle and the dominant effect remains that of dose and average testosterone

concentrations in blood, however they arise. Furthermore, there is evidence that the androgen sensitivity of responsive tissues differs and may be optimal at different circulating testosterone concentrations (65).

Male sexual function is maintained by endogenous testosterone at adult male circulating concentrations. These effects can be replicated by exogenous testosterone if and only if it achieves comparable circulating testosterone concentrations. For example, in a well controlled prospective study of older men with prostate cancer (66), androgen deprivation achieving castrate levels of circulating testosterone sustained during 12 months markedly suppressed sexual desire and function, whereas those effects did not occur in age matched men having nonhormonal treatment of prostate cancer or those without prostate cancer. In healthy younger men whose endogenous testosterone was fully suppressed, sexual function completely recovered when circulating testosterone was restored to the physiological male range by administration of exogenous testosterone (67). Similar effects were also observed in healthy, middle aged men in whom male sexual function was fully maintained (compared with placebo) during 2 years of treatment with an exogenous androgen (DHT) despite that treatment causing sustained, complete suppression of endogenous testosterone (68). This further supports the key interpretation that the biological effects of exogenous or endogenous testosterone are the same at comparable circulating levels.

Clinically, exogenous testosterone replicates fully all effects of endogenous testosterone on every reproductive and nonreproductive organ or tissue, with the sole exception of the testis. Sperm production in the testis requires a very high concentration of testosterone (typically 100 fold greater than in the general bloodstream), which is produced in nature only by the action of the pituitary hormone LH. LH stimulates the Leydig cells in the interstitial space of the testis between seminiferous tubules to produce high intratesticular concentrations of testosterone, which are necessary and sufficient to initiate and maintain sperm production in the adjacent seminiferous tubules. This high concentration of testosterone also provides a downhill gradient to supply the rest of the body, where circulating testosterone acts on androgen responsive tissues to produce and maintain masculine patterns of androgenization. When exogenous testosterone (or any other androgen) is administered to men, pituitary LH is suppressed by negative feedback and the sperm production halts for as long as exogenous testosterone or androgen exposure continues, after which it recovers (69). However, even the reduction in spermatogenesis and testis size when men are treated with exogenous testosterone is only a matter of degree. It is well established in rodents (70, 71) that spermatogenesis is induced by exogenous testosterone when the testosterone concentrations in the testis are high enough to replicate what occurs naturally via LH stimulation (72). However, direct replication that high dose testosterone also initiates and maintains spermatogenesis in humans is not feasible, as these testosterone doses are 10 to 100 fold higher than could be safely given to humans. Nevertheless, confirmatory evidence in humans is available from rare cases of men with an activating mutation of the chorionic gonadotropin/LH receptor (73, 74). This mutation causes autonomous testicular testosterone secretion leading to precocious puberty arising from the premature adult male circulating testosterone concentrations that lead to complete suppression of circulating gonadotropin (LH, FSH) secretion. In this illustrative case the testis was exposed to non physiologically high testosterone concentrations (but without any gonadotropin stimulation) that induced sperm production and allowed for natural paternity (73). This indicates that even for spermatogenesis, exogenous testosterone can replicate all biological effects of endogenous testosterone in accordance with the relevant dose response characteristics.

The most realistic view is that increasing circulating testosterone from the childhood or female range to the adult male range will have the same physiological effects whether the source of the additional testosterone is endogenous or exogenous. This is strongly supported by well established knowledge about the relationship of circulating testosterone concentrations

Table 3. Upper Confidence Limits on Serum Testosterone in Women With PCOS

Confidence Interval	Likelihood[a]	SD[b]	One-Sided[c]	Two-Sided[c]
95%	1:20	1.96	3.13	3.39
99%	1:100	2.35	3.47	3.73
99.9%	1:1000	3.10	4.21	4.39
99.99%	1:10,000	3.72	4.77	4.95

[a]Likelihood that a woman with PCOS would exceed that limit by chance.
[b]Number of SDs for each confidence limit.
[c]Two-sided CIs are conventional for a result that could exceed or fall below confidence limits, but here we focus only on values exceeding the upper limit, so that one-sided confidence limits are appropriate.

with the timing and manifestations of male puberty. The characteristic clinical features of masculinization (*e.g.*, muscle growth, increased height, increased he moglobin, body hair distribution, voice change) appear only if and when circulating testosterone concentra tions rise into the range of males at mid puberty, which are higher than in women at any age even after the rise in circulating testosterone in female puberty. If and only if the pubertal rise in circulating testosterone fails will the males affected be clinically considered hypogonadal. Such a failure of male puberty may occur for genetic reasons (arising from mutations that in activate any of the cascade of proteins whose activity is critical in the hypothalamus to trigger male puberty) or as a result of acquired conditions, caused by patho logical disorders of the hypothalamus or pituitary or functional defects arising from severe deficits of en ergy or nutrition (*e.g.*, extreme overtraining, un dernutrition), with the latter being comparable with hypothalamic amenorrhea or anorexia nervosa in female athletes/ballet dancers. If male puberty fails, testosterone replacement therapy is fully effective in replicating all of the distinctive masculine features apart from spermatogenesis.

Elevated circulating testosterone concentration caused by DSDs

Rare genetic intersex conditions known as DSDs can lead to markedly increased circulating testosterone in women. When coupled with ambiguous genitalia at birth, they may appear as undervirilized males or virilized females. This can cause athletes who were raised and identify as women to have circulating testosterone levels comparable to those of men and greatly exceeding those of non DSD (and nondoped) women, including those with PCOS. Key congenital disorders in this category are 46,XY DSDs, namely 5α reductase deficiency (75), 17β hydroxysteroid de hydrogenase type 3 deficiency (76), and androgen insensitivity (77, 78), as well as congenital adrenal hyperplasia (79), which is a 46,XX DSD. There is evidence that the first three conditions, components of 46,XY DSDs, are 140 fold more prevalent among elite female athletes than expected in the general pop ulation (80).

Genetic 5α reductase deficiency is due to an inactivating mutation in the 5α reductase type II enzyme (75). This leads to a deficit of DHT during fetal life when DHT is required for converting the sex undifferentiated embryonic and fetal tissue to form the sex differentiated masculine form external genitalia. Although genetic males (46,XY) with 5α reductase deficiency will develop testes, they usually remain undescended and labial fusion to form a scrotum and phallic growth does not occur. Hence, at birth the external genitalia may appear feminine, leading to a female assigned natal sex. Thus, individuals with 5α reductase deficiency may have male chromosomal sex (46,XY), gonadal sex (testes), and hormonal sex (adult male testosterone concentrations), but such severely undervirilized genitalia that affected individuals may be raised from birth as females rather than as undervirilized males. However, from the onset of male puberty, testicular Leydig cells start producing large amounts of testosterone, and the steep rise in circu lating testosterone to adult male levels (with the permissive role of 5α reductase activity) leads to masculine virilization, including male patterns of muscle and bone growth, hemoglobin levels, and other masculine body habitus features (hair growth pattern, voice change), as well as phallic growth (80). Such changes of male puberty prompt around half affected individuals who had female sex assigned at birth and developed as girls prior to puberty to adopt a male gender identity and role at puberty (81). Sperm are formed in the testes so that, using *in vitro* fertilization, these individuals may father children (82).

17β Hydroxysteroid dehydrogenase type 3 de ficiency (76) has a natural history similar to that of 5α reductase deficiency. This disorder is due to inacti vating mutations in a steroidogenic enzyme expressed only in the testis and that is essential for testosterone formation in the fetus. In the absence of a functional enzyme, the testis makes little testosterone but instead secretes large amounts of androstenedione, the steroid immediately prior to the enzymatic block. In the circulation, the excess of androstenedione is converted to testosterone (mainly by the enzyme AKR1C3) (12). Although the circulating testosterone is then con verted to circulating DHT, insufficient DHT is formed locally within the urogenital sinus to virilize genitalia at birth. This causes the same severe undervirilization of the external genitalia of genetically male individuals, leading to ambiguous genitalia at birth despite male chromosomal, gonadal, and hormonal sex. When puberty arrives, the testes start producing the adult male testosterone output. Again, this leads to marked virilization and subsequent assumption of a male gender identity by some affected individuals, con flicting with a female assigned natal sex and childhood upbringing.

Androgen insensitivity, which arises from muta tion in the androgen receptor (AR), poses different but complex challenges for eligibility for female athletic events. As the AR is located on the X chromosome, genetic males (46,XY) are hemizygous, so that an inactivating mutation in the AR can be partially or fully insensitive to androgen action. Affected in dividuals have male internal genitalia (testes in the inguinal canal or abdomen with Wolffian ducts) and consequently adult male circulating testosterone concentrations after puberty. These nonlethal muta tions have a wide spectrum of functional effects, ranging from full resistance to all androgen action in complete androgen insensitivity syndrome (CAIS) where individuals have a full female phenotype with

normal female external genitalia, to partial androgen insensitivity syndrome (PAIS) where some androgen action is still exerted, leading to various degrees of ambiguous genitalia, or to mild androgen insensitivity, which produces a very mild, undervirilized male phenotype (normal male genital and somatic development but with little body hair and no male pattern balding) (77). Testosterone (and dihydrotestosterone) have no consistent effect of inducing normal nitrogen retention (anabolic) responses in patients with CAIS (83 86), although some reduced androgen responsiveness is retained by patients with PAIS (84, 87 90). Athletes with CAIS can compete fairly as females because the circulating testosterone, although at adult male levels, has no physiological effect so that, in terms of androgen action and the ensuing physical somatic advantages of male sex, affected individuals are indistinguishable from females and gain no benefits of the sex difference arising from unimpeded testosterone action. A more complex issue arises with athletes having PAIS reflecting the degree of incomplete impairment of AR function. Residual androgen action in such AR mutations is harder to characterize quantitatively, as there is no standardized, objective *in vitro* test to quantify AR functionality. Hence, individuals with PAIS may have adult male circulating testosterone concentrations but variable androgen sensitivity. At present, determination of eligibility to compete in the female category requires a case by case evaluation, primarily based on the degree of virilization. The current best available clinical approach to determining the functional impact (degree of functionality/sensitivity) of an AR mutation is based on the degree of somatic, primarily genital, virilization assessed according to the Quigley classification of grade of androgen sensitivity (91).

Congenital adrenal hyperplasia (CAH) is a relatively common defect in adrenal steroidogenesis in the enzymatic pathway, leading to synthesis of cortisol, aldosterone, and sex steroid precursors. The disease varies in severity from life threatening (adrenal failure) to mild (hirsutism and menstrual irregularity), or even asymptomatic and undiagnosed. The most common mutations causing CAH occur in the 21 hydroxylase enzyme, accounting for 95% of cases (79). The defect leads to a bottleneck, creating a major backing up of precursor steroids that then overflow into other steroid pathways, leading to diagnostic high levels of 17 hydroxyprogesterone and, in female patients, excessive circulating testosterone or other adrenal source androgen precursors (*e.g.*, androstenedione, dehydroepiandrosterone) that may be converted to testosterone in tissues. A common clinical problem with management of CAH is that glucocorticoid/mineralocorticoid treatment is not always fully effective partly due to variable compliance, which may leave high circulating testosterone, including well into or even above the normal male range (92). It is unlikely that mild nonclassical congenital adrenal hyperplasia is a major contributor to the mild hyperandrogenism prevalent among elite female athletes. The prevalence of PCOS (6% to 16%) is about 100 fold higher than mild nonclassical congenital adrenal hyperplasia (0.1%) (49), whereas a disproportionately high number of elite female athletes (especially in power sports) have PCOS (45). In one study of hyperandrogenic female athletes, even mild nonclassic adrenal hyperplasia was ruled out by normal 17 hydroxyprogesterone (36) and, in another (47), reported serum androstenedione and cortisol did not differ from controls, ruling out significant congenital adrenal hyperplasia.

Sex Difference in Muscle, Hemoglobin, Bone, and Athletic Performance Relating to Adult Circulating Testosterone Concentrations

Following puberty, testosterone production increases (16) but remains <2 nmol/L in women, whereas in men testosterone production increases 20 fold (from 0.3 mg/d to 7 mg/d), leading to 15 fold higher circulating testosterone concentrations (15 vs 1 nmol/L). The greater magnitude of sex difference in testosterone production (20 fold) compared with circulating levels (15 fold) is due to women's higher circulating SHBG, which retards testosterone clearance, creating a slower circulating half time of testosterone. This order of magnitude difference in circulating testosterone concentrations is the key factor in the sex difference in athletic performance due to androgen effects principally on muscle, bone, and hemoglobin.

"Sex differences in height, where they exist, are largely dependent on postpubertal differences in circulating testosterone."

Muscle

Biology

It has been known since ancient times that castration influences muscle function. Modern knowledge of the molecular and cellular basis for androgen effects on skeletal muscle involves effects due to androgen (testosterone, DHT) binding to the AR that then releases chaperone proteins, dimerizes, and translocates into the nucleus to bind to androgen response elements in the promoter DNA of androgen sensitive genes. This leads to increases in (1) muscle fiber numbers and size, (2) muscle satellite cell numbers, (3) numbers of myonuclei, and (4) size of motor neurons (93). Additionally, there is experimental evidence that testosterone increases skeletal muscle myostatin expression (94), mitochondrial biogenesis (95), myoglobin expression (96), and IGF 1 content (97), which may augment energetic and power generation of skeletal muscular activity.

Customized genetic mouse models can provide unique experimental insight into mammalian physiology that is unobtainable by human experimentation.

The tight evolutionary conservation of the mammalian reproductive system explains why genetic mouse models have provided consistent, high fidelity replication of the human reproductive system (98, 99). Genetic males (46,XY) with androgen insensitivity displaying similar features occur through the spontaneous production of inactivating AR mutations in all mammalian species studied, including humans, where they are known as women with CAIS. The converse, genetic females (46,XX) resistant to all androgen action cannot occur naturally in humans or other mammals. This is because fully androgen resistant females must have both X chromosomes carrying an inactivated AR. In turn, this requires acquiring one X chromosome from their father, and hemizygous males bearing a single X chromosome with an inactive AR produce no sperm, as a functional AR is biologically indispensable for making sperm in any mammal. However, androgen resistant females can be bred by genetic engineering using the Cre Lox system (100). An important finding from such studies is that androgen resistant female mice have essentially the same muscle mass and function as wild type androgen sensitive females bearing normal AR, whereas androgen resistant male mice have smaller and weaker muscle mass and function than do wild type males and comparable instead with wild type females (101). This indicates that androgen action, represented by circulating testosterone, is the key determinant of the higher muscle mass and strength characteristic of males compared with females. Furthermore, endogenous circulating testosterone has minimal effects on skeletal muscle mass and strength in female mice because of its low levels. Although these experiments cannot be replicated in humans, their key insight is that the higher circulating testosterone in males is the determinant of the male's greater muscle mass and function compared with females. Nevertheless, there is also evidence that hyperandrogenic women, mostly with PCOS, have increased muscle mass and strength that correlates with mildly increased circulating testosterone in the high normal female range (36, 47).

Observational data

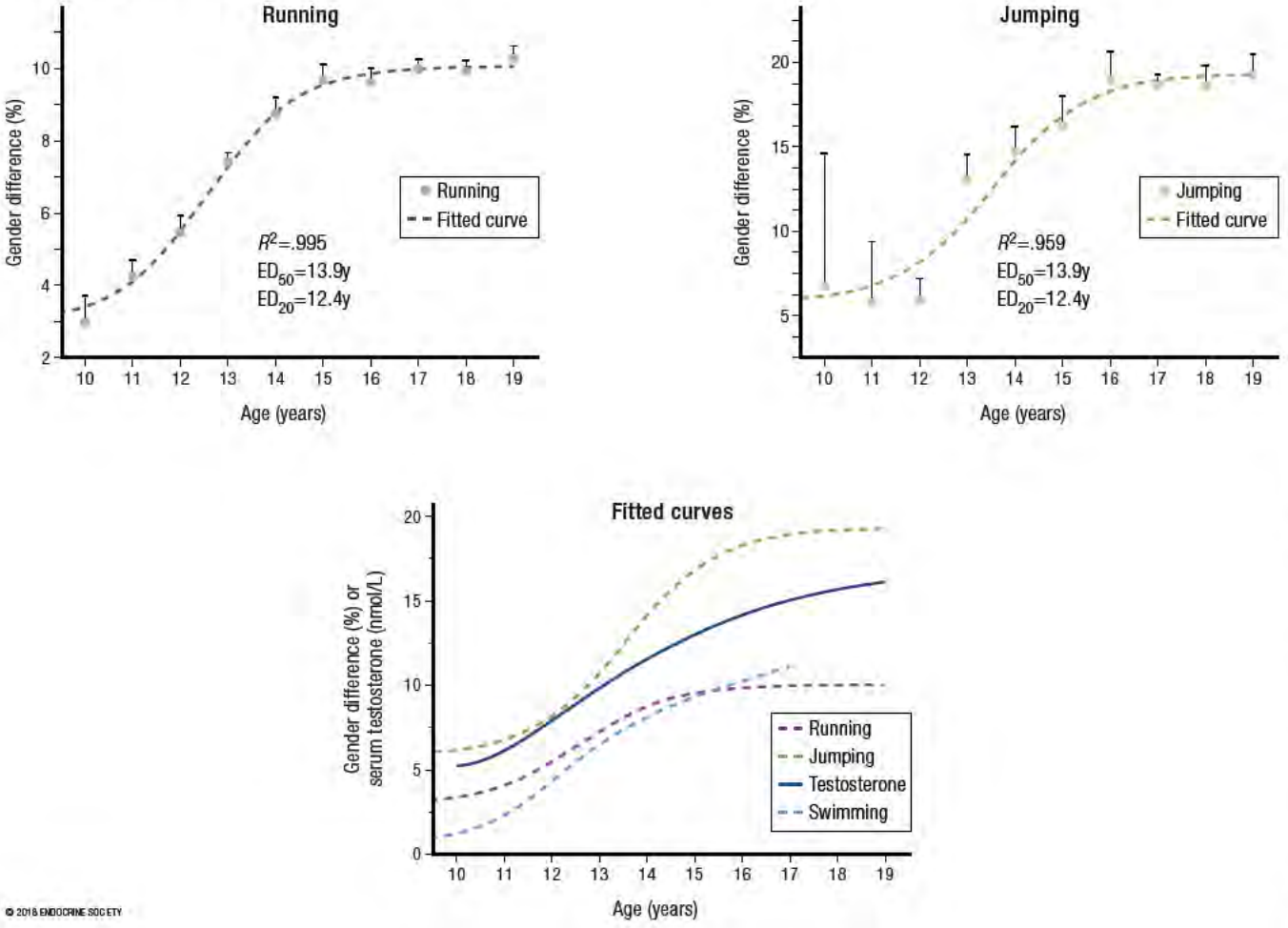
There is a clear sex difference in both muscle mass and strength (102–104) even adjusting for sex differences in height and weight (104, 105). On average, women have 50% to 60% of men's upper arm muscle cross sectional area and 65% to 70% of men's thigh muscle cross sectional area, and women have 50% to 60% of men's upper limb strength and 60% to 80% of men's leg strength (106). Young men have on average a skeletal muscle mass of >12 kg greater than age matched women at any given body weight (104, 105). Whereas numerous genes and environmental factors (including genetics, physical activity, and diet) may contribute to muscle mass, the major cause of the sex difference in muscle mass and strength is the sex difference in circulating testosterone.

Age grade competitive sports records show minimal or no female disadvantage prior to puberty, whereas from the age of male puberty onwards there is a strong and ongoing male advantage. Corresponding to the endogenous circulating testosterone increasing in males after puberty to 15 to 20 nmol/L (sharply diverging from the circulating levels that remain <2 nmol/L in females), male athletic performances go from being equal on average to those of age matched females to 10% to 12% better in running and swimming events, and 20% better in jumping events (8) (Fig. 1). Corroborative findings are provided by a Norwegian study that examined performance of adolescents in certain athletic events but without reference to contemporaneous circulating testosterone concentrations (107). The striking postpubertal increase in male circulating testosterone provides a major, ongoing, cumulative, and durable advantage in sporting contests by creating greater muscle mass and strength. These sex differences render women unable to compete effectively against men, especially (but not only) in power sports.

These findings are supported by studies of non athletic women showing that muscle mass is increased in proportion to circulating testosterone in women with mildly elevated testosterone levels due to PCOS (108, 109), a condition that is more prevalent among elite female athletes who exhibit these features (36, 45, 47), often undiagnosed (46), but that may provide an ergogenic advantage (47), consistent with the graded effects of circulating testosterone on explosive performance in men and women (110).

Studies of elite female athletes further corroborate these findings. One study demonstrates dose response effects of better performance in some (400 m running, 400 m hurdles, 800 m running, hammer throw, pole vault) but not all athletic events correlated with significantly higher endogenous testosterone in female, but not male, athletes. Even within the low circulating testosterone levels prevailing within the normal female range, in these events there was a significant advantage of 1.8% to 4.5% among those in the highest tertile compared with the lowest tertile of endogenous testosterone (35). A further study of elite female athletes corroborates and extends these observations in that endogenous androgens are associated with a more anabolic body composition as well as enhanced muscular performance (36). In this study, 106 Swedish Olympic female athletes were compared with 117 age and weight (body mass index) matched sedentary control women for their muscle and bone mass (by dual energy X ray absorptiometry), their muscular strength (squat and countermovement jumps), and testosterone and DHT, as well as androgen precursors (dehydroepiandrosterone, androstenedione) and urinary androgen glucuronide metabolites (androsterone,

Figure 1. Sex differences in performance (in percentage) according to age (in years) in running events, including 50 m to 2 miles (upper left panel), and in jumping events, including high jump, pole vault, triple jump, long jump, and standing long jump (upper right panel) [for details, see Ref. (8)]. The lower panel is a fitted sigmoidal curve plot of sex differences in performance (in percentage) according to age (in years) in running, jumping, and swimming events, as well as the rising serum testosterone concentrations from a large dataset of serum testosterone of males. Note that in the same dataset, female serum testosterone concentrations did not change over those ages, remaining the same as in prepubertal boys and girls. Data are shown as mean and SEM of the pooled sex differences by age. Reproduced with permission from Handelsman DJ. Sex differences in athletic performance emerge coinciding with the onset of male puberty. *Clin Endocrinol (Oxf)*. 2017;87:68–72.



etiocholanolone, 3 and 17 3α diols) measured by LC MS (36). The athletes displayed higher muscle (and bone) mass than did the sedentary control women, with strength tests correlating strongly with muscle mass whether in total or just in the legs. In turn, muscle mass and strength were correlated with androgens and an drogen precursors. Considering that such studies may be confounded by factors such as menstrual phase and dysfunction, as well as heterogeneous sports disciplines, which weaken the power of the study, these findings can be regarded as quite robust.

Interventional data

Dose response studies show that in men whose en dogenous testosterone is fully suppressed, add back administration of increasing doses of testosterone that produce graded increases in circulating testosterone causes a dose dependent (whether expressed according to testos terone dose or circulating levels) increase in muscle mass (measured as lean body mass) and strength (65, 111). Taken together, these studies prove that testosterone doses leading to circulating concentrations from well below to well above the normal male range have unequivocal dose dependent effects on muscle mass and strength. These data strongly and consistently suggest that the sex difference in lean body mass (muscle) is largely, if not exclusively, due to the dif ferences in circulating testosterone between men and women. These findings have strong implications for power dependent sport performance and largely explain the potent efficacy of androgen doping in sports.

The key findings providing conclusive evidence that testosterone has prominent dose response effects in men are reported in studies by Bhasin and col leagues that proved a monotonic dose response,

extending from subphysiological to supraphysiological range for men for testosterone effects on muscle mass, size, and strength in healthy young men, findings that have been replicated and confirmed by an independent group (65). Both sets of studies used a common design of fully suppressing all endogenous testosterone (to castrate levels) for the full duration of the experiment by administering a GnRH analog. In the Bhasin and colleagues studies, participants were then randomized to five groups and each received weekly injections of 25 mg, 50 mg, 125 mg, 300 mg, or 600 mg of testosterone enanthate for 20 weeks. In effect, this was two subphysiological and two supraphysiological testosterone doses. In these studies, the lowest testosterone dose produced a mean serum testosterone of 253 ng/dL (8.8 nmol/L) in younger men and 176 ng/dL (6.1 nmol/L) in older men. The studies showed a consistent dose response for muscle mass and strength that was clearly related to testosterone dose and consequential blood testosterone concentrations (Fig. 2, upper panel).

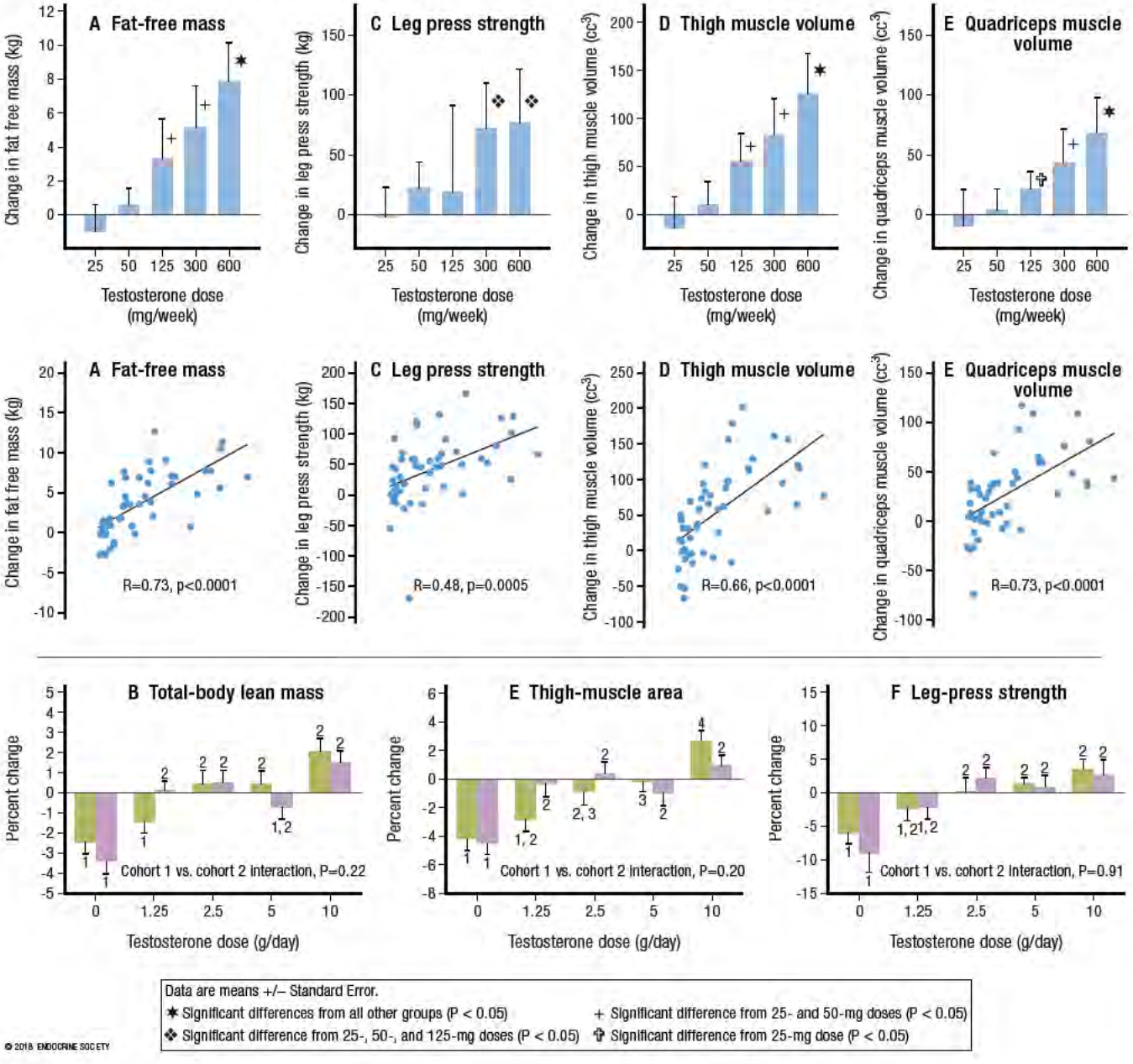
The study of Finkelstein *et al.* (65) involved the same design and involved 400 healthy men aged 20 to 50 years who had complete suppression of endogenous testosterone for the 16 weeks of the study, with testosterone added back using daily doses of 0, 1.25 g, 2.5 g, 5 g, or 10 g of a topical 1% testosterone gel. This again created a graded dose response curve for serum testosterone and for muscle mass and strength. The inclusion of a 0 (placebo) dose allowed differentiation between the 0 and lowest testosterone dose. The placebo (0) dose produced a serum testosterone of 0.7 nmol/L (the typical mean for castrated men, childhood, and women of any age). Meanwhile, the lowest testosterone dose (1.25 g of gel per day) produced a serum testosterone of 6.9 nmol/L, which is equivalent to that of a male in early to middle puberty. A key finding for this review is that, from this study of men, the increase in serum testosterone from mean of normal female concentration (0.9 nmol/L) to supraphysiological female concentrations (6.9 nmol/L) produced significant increases of 2.3% for total body lean (muscle) mass, 3.0% for thigh muscle area, and 5.5% increase in leg press strength (digitized data pooling of both cohorts from lower panel, Fig. 2).

Studies of the ergogenic effects of supraphysiological concentrations of circulating testosterone require studies administering graded doses of exogenous testosterone for months. Owing to ethical concerns regarding risks of unwanted virilization and hormone dependent cancers, however, few studies have administered supraphysiological testosterone doses to healthy women. One well designed, randomized placebo controlled study of postmenopausal women investigated the effects of different testosterone doses on muscle mass and performance and physical function (112). Sixty two women (mean age, 53 years) all had a standard estrogen replacement dose administered during a 12 week run in period (to eliminate any hypothetical confounding effects of estrogen deficiency), after which they were randomized to one of five groups receiving weekly injections of testosterone enanthate (doses: 0, 3 mg, 6.25 mg, 12.5 mg, and 25 mg, respectively) for 24 weeks. The increasing doses of testosterone produced an expected dose response in serum testosterone concentrations (by LC MS), with the highest testosterone dose (25 mg/wk) producing a mean nadir concentration of 7.3 nmol/L. The women whose testosterone concentrations were increased to 7.3 nmol/L achieved significant increases in muscle mass and strength (Table 4), ranging from 4.4% for muscle (lean) mass to between 12% and 26% for measures of muscle strength (chest and leg press, loaded stair climb). As muscle strength measurement is effort dependent, the placebo controlled design of the Huang *et al.* (112) study supports the further interpretation that the highest dose of testosterone also had prominent mental motivational effects in the effort dependent tests of muscle strength. These findings provide salient direct evidence of the ergogenic effects of hyperandrogenism in female athletes confirming that at least up to average circulating testosterone concentrations of 7.3 nmol/L, women display a dose response relationship similar to that of men, with supraphysiological doses of testosterone leading to significant gains in muscle mass and power.

These effects of testosterone administration on circulating testosterone concentrations and muscle mass and strength in females may be compared with the effects in males from the Finkelstein *et al.* (65) and Bhasin and colleagues studies. In men, the lowest testosterone dose (1.25 g/d) increased mean serum testosterone to 6.9 nmol/L (equivalent to levels seen in early to middle male puberty), resulting in significant increases of total body lean (muscle) mass (2.3%), thigh muscle area (3.0%), and leg press strength (5.5%) compared with the placebo dose that resulted in a serum testosterone of 0.7 nmol/L. In the Huang *et al.* (112) study (Fig. 3), muscle mass and strength in postmenopausal women displayed a flat response at the three lower doses, when circulating testosterone concentrations remain <5 nmol/L, and displayed a significant increase only when the mean circulating testosterone concentration produced by the highest testosterone dose first increased circulating testosterone concentrations >5 nmol/L. This pattern, flat at lower doses and rising at the highest dose, represents the lower plateau and the earliest rising portion, respectively, of the sigmoidal dose response curve of testosterone for muscle.

Data corroborating the Huang *et al.* study results comes from another well controlled study in which postmenopausal women who were administered methyl testosterone following a run in period of estrogen replacement displayed a significant increase in lean (muscle) mass as well as upper and lower limb

Figure 2. Strong dose-response relationship between testosterone dose and circulating concentration with muscle mass and strength in men. The upper panels [from Bhasin *et al.* (111)] display the strong dose-response relationships of muscle mass shown as (A) "lean" or "fat-free" mass or volume of (D) thigh and (E) quadriceps muscle and (C) of leg muscle strength with increasing testosterone dose (upper row) or circulating concentration (middle row). Serum testosterone concentrations are in US units (ng/dL; divide by 28.8 to get nmol/L). Adapted with permission from Bhasin S, Woodhouse L, Casaburi R, et al. Testosterone dose-response relationships in healthy young men. *Am J Physiol Endocrinol Metab.* 2001;**281**:E1172 E1181. The lower panels [from Finkelstein *et al.* (65)] show the strong dose-response relationships of (B) whole-body muscle mass, (E) thigh muscle mass, and (F) leg press strength with increasing testosterone dose. Cohorts 1 and 2 were treated with the same increasing doses of testosterone but either without (green fill, cohort 1) or with (purple fill, cohort 2) an aromatase inhibitor (anastrozole), which prevents conversion of testosterone to estradiol. The differences between cohorts (*i.e.*, use of anastrozole) was not significant for muscle mass and strength and can be ignored with results of the two cohorts being pooled. Reproduced with permission from Finkelstein JS, Lee H, Burnett-Bowie SA, Pallais JC, *et al.* Gonadal steroids and body composition, strength, and sexual function in men. N Engl J Med 2013;369:1011 1022.



Data are means +/– Standard Error.
✱ Significant differences from all other groups (P < 0.05)
+ Significant difference from 25- and 50-mg doses (P < 0.05)
❖ Significant difference from 25-, 50-, and 125-mg doses (P < 0.05)
✞ Significant difference from 25-mg dose (P < 0.05)

power during a 16 week double blind, parallel group study (113).

Similarly, two prospective studies of the first 12 months of treatment of transmen [female to male (F2M) transgender] shows a consistent major increase in muscle mass and strength due to testosterone administration. In one study testosterone treatment of 17 transmen achieving adult male circulating testosterone levels

Table 4. Effects of Testosterone on Muscle Mass and Strength in Women

Androgen-Sensitive Variable	Baseline	Increase	% Increase
Lean muscle mass, kg	43 ± 6	1.9 ± 0.5	4.4
Chest press, W	100 ± 26	26 ± 7	26
Leg press, N	744 ± 172	90 ± 30	12
Loaded stair climb power, W	406 ± 77	56 ± 13	14

With data from Huang G, Basaria S, Travison TG, *et al.* Testosterone dose-response relationships in hysterectomized women with or without oophorectomy: effects on sexual function, body composition, muscle performance and physical function in a randomized trial. Menopause 2014;21:612–623. Data are shown as mean and SEM derived from Table 1 and digitized from Figure 4 from Huang *et al.* (112) showing the effects of testosterone (mean circulating concentration, 7.3 nmol/L) on muscle mass and strength in women treated with the highest testosterone dose (n = 11; 25 mg of testosterone enanthate per week).

(mean, 31 nmol/L) increased muscle mass by 19.2% (114). In a second study, 23 transmen administered adult male testosterone doses also produced striking increases in total body muscle size and limb muscle size (by 6.5% to 16.6%) and grip strength (by 18%) compared with age matched untreated control women (115). Conversely, testosterone suppression (using an estrogen based treatment regimen) in 20 transwomen (M2F transgender) that reduced circulating testosterone levels from adult male range to adult female range led to a 9.4% reduction in muscle mass (measured as cross sectional area).

Effects on athletic performance

Muscle growth, as well as the increase in strength and power it brings, has an obvious performance enhancing effect, in particular in sports that depend on strength and (explosive) power, such as track and field events (107, 110). There is convincing evidence that the sex differences in muscle mass and strength are sufficient to account for the increased strength and aerobic performance of men compared with women and is in keeping with the differences in world records between the sexes (116). The basis for the sex difference in muscle mass and strength is the sex difference in circulating testosterone as clearly shown (for example) by (1) the enhanced athletic performance of men compared with prepubertal boys and women (8); (2) the close correspondence of muscle growth (muscle size) with muscle strength in ascending dose studies in men by Bhasin *et al.* (111, 117–119) and Finkelstein *et al.* (65) and in postmenopausal women by Huang *et al.* (112); (3) the effect of male castration in reducing muscle size and strength, effects that are fully rectified by testosterone replacement; and (4) the striking efficacy of androgen doping on the sports performances of German Democratic Republic female athletes (120).

Hemoglobin

Biology

It is well known that levels of circulating hemoglobin are androgen dependent and consequently higher in men than in women by 12% on average; however, the physiological mechanism by which androgens such as testosterone boosts circulating hemoglobin is not fully understood (121). Testosterone increases secretion of and sensitivity to erythropoietin, the main trophic hormone for erythrocyte production and thereby hemoglobin synthesis, as well as suppressing hepcidin (122), a crucial iron regulatory protein that governs the body's iron economy. Hepcidin has to balance the need for iron absorption from foods (the only source of iron required for the body's iron containing proteins) against the fact that the body has no mechanism to shed excess iron, which can be toxic. Adequate iron availability is essential for normal erythropoiesis and synthesis of key heme, iron containing oxygen transporting proteins such as hemoglobin and myoglobin (123) as well as other iron dependent proteins such as cytochromes and DNA synthesis and repair enzymes. Experimental evidence in mice shows that testosterone increases myoglobin content of muscle with potential for augmenting aerobic exercise performance (96), but this has not been evaluated in humans.

Increasing the amount of hemoglobin in the blood has the biological effect of increasing oxygen transport from lungs to tissues, where the increased availability of oxygen enhances aerobic energy expenditure. This is exploited to its greatest effect in endurance sports (1). The experiments of Ekblom *et al.* (124) in 1972 (Fig. 4) demonstrated strong linear relationships between changes in hemoglobin [due to withdrawal or retransfusion of 1, 2 or 3 U (400 mL) of blood] and aerobic capacity, established by repeated testing of maximal exercise induced oxygen consumption before and after each procedure (124). As already noted, circulating hemoglobin levels are on average 12% higher in men than women (125). It may be estimated that as a result the average maximal oxygen transfer will be ~10% greater in men than in women, which has a direct impact on their respective athletic capacities.

Observational data

The proposition that the sex difference in circulating hemoglobin levels is likely to be due to the sex difference in average circulating testosterone concentrations is supported by the fact that male castration (*e.g.*, for advanced prostate cancer) (126) and androgen deficiency due to reproductive system disorders (127) reduce circulating

hemoglobin in men, eliminating the sex difference, whereas testosterone replacement therapy restores circulating hemoglobin to adult male levels (121, 127, 128).

An unusually informative observational study of women with CAH provides unique insight into testosterone effects on circulating hemoglobin in otherwise healthy women (92). Women with CAH require glucocorticoid replacement therapy but exhibit widely varying levels of hormonal control (79). The degree of poor control is associated with increasing levels of circulating testosterone ranging from normal female concentrations up to 36 nmol/L, and these levels correlate closely (r = 0.56) with levels of circulating hemoglobin (Fig. 5). Interpolating from the dose response regression, increases in circulating testosterone measured by LC MS from 0.9 nmol/L to 5 nmol/L, 7 nmol/L, 10 nmol/L, and 19 nmol/L were associated with increases in circulating hemoglobin of 6.5%, 7.8%, 8.9%, and 11%, respectively, establishing a strong dose response relationship. An 11% increase in circulating hemoglobin translates to a 10% difference in maximal oxygen transfer (124), which may account for virtually all the 12% sex difference in male and female circulating hemoglobin (125). To put this into context, any drug that achieved such increases in hemoglobin would be prohibited in sports for blood doping, as this difference is sufficient to have ergogenic effects, even without taking into account any testosterone effects on muscle mass or strength (for which data were not available in that study). Conversely, among elite female athletes with circulating testosterone in the healthy premenopausal female range, circulating hemoglobin does not correlate with athletic performance (35). In women with the mild hyperandrogenism of PCOS, circulating hemoglobin and hematocrit are reported as not (129) or marginally increased (130), findings that may be influenced by the fact that PCOS is associated with reduced or absent menstruation, thereby reducing the iron loss of regular menstruation.

Interventional data

In the Bhasin *et al.* (111) studies, in both young and older men the highest testosterone dose produced a 12% increase in blood hemoglobin compared with the lowest dose, reflecting a strong dose response relationship (Fig. 6) (131). Analogous findings were reported for testosterone treatment effects in postmenopausal women where the highest dose (25 mg weekly) of testosterone, which increased mean serum testosterone to 7.3 nmol/L, had the largest increase (3%) in blood hemoglobin and hematocrit (112).

Corroborative findings are available from studies of transmen (F2M transgender), that is, natal females who subsequently receive testosterone treatment at replacement doses to create adult male circulating testosterone concentrations, who exhibit increases in circulating hemoglobin to male levels [reviewed in (132 134)]. Testosterone treatment in 17 (F2M) transmen that created mean circulating testosterone levels of 31 nmol/L also increased hemoglobin levels by 15% (114). Conversely, one prospective 12 month study of transgender (nonathlete) individuals reported that testosterone suppression (by an estrogen based regimen) to normal female levels in 20 (M2F) transwomen reduced hemoglobin by 14%.

If such an increase in hemoglobin were produced by any chemical substance, it would be considered doping, according to the World Anti Doping Code.

Bone

Biology

There is extensive experimental evidence from genetic mouse models showing that the sex differences in bone

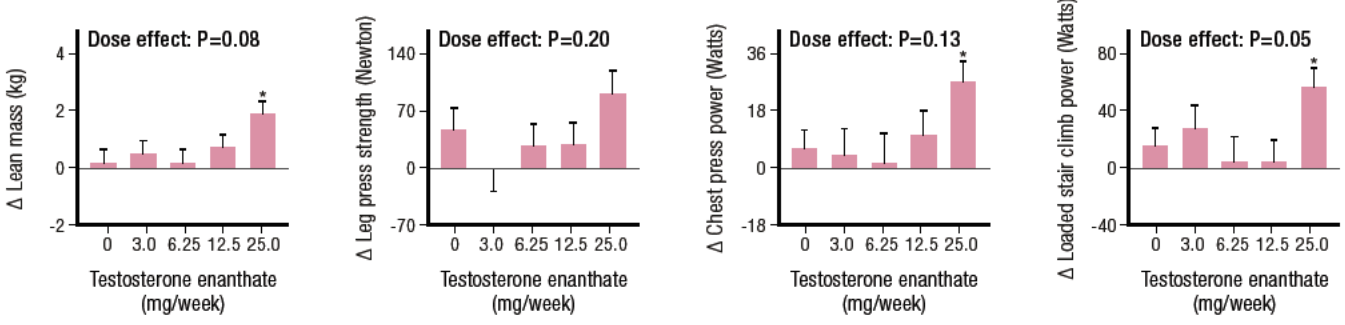
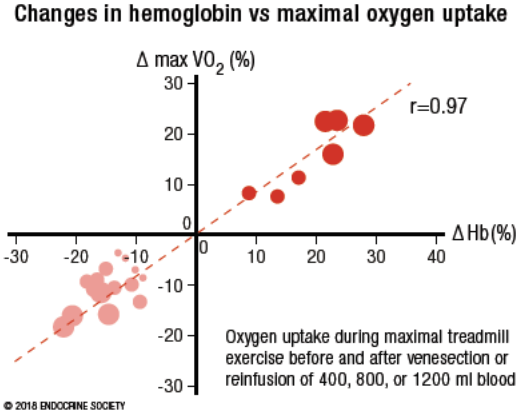


Figure 3. From Huang *et al.* (112): Dose-response effects on lean (muscle) mass and three measures of muscle strength as a result of increasing doses of weekly testosterone enanthate injections in women. Note the effects on all four parameters (three statistically significant) of the highest testosterone dose, the only one that produced circulating testosterone levels exceeding the normal female range. Reproduced with permission from Huang G, Basaria S, Travison TG, *et al.* Testosterone dose-response relationships in hysterectomized women with or without oophorectomy: effects on sexual function, body composition, muscle performance and physical function in a randomized trial. Menopause 2014;21:612 623.

Figure 4. Redrawn results from Ekblom *et al.* (124). Results from the transfusion of additional blood are shown in dark red circles and those after blood withdrawal in light red circles. Adapted with permission from Ekblom B, Goldbarg AN, Gullbring B. Response to exercise after blood loss and reinfusion. J Appl Physiol 1972;33:175 180.



size, mass, and function are due to the sex difference in circulating testosterone. These effects have been reported from studies of global and tissue or cell selective inactivation of ARs or estrogen receptors that show that androgen effects are mediated by both direct effects on the AR as well as indirect effects mediated via aromatization of testosterone to estradiol to act on estrogen receptors [reviewed in (135)]. Bone grows in length due to epiphyseal chondral growth plates that provide cartilage, forming the matrix for lengthening of long bone, which is terminated by an estrogen dependent mechanism that depends on aromatization of testosterone to estradiol. Similarly, bone width and density are increased through appositional growth from periosteal and endosteal expansion that depend on bone loading and androgen exposure together with other factors. An important difference between androgen effects on bone compared with effects on muscle or hemoglobin is that developmental bone effects of androgens are likely to be irreversible.

Observational data

Men have distinctively greater bone size, strength, and density than do women of the same age. As with muscle, sex differences in bone are absent prior to puberty but then accrue progressively from the onset of male puberty due to the sex difference in exposure to adult male circulating testosterone concentrations [reviewed in (135)]. The earlier onset of puberty and the related growth spurt in girls as well as earlier estrogen dependent epiphyseal fusion explains shorter stature of girls than boys. As a result, on average men are 7% to 8% taller with longer, denser, and stronger bones, whereas women have shorter humerus and femur cross sectional areas being 65% to 75% and 85%, respectively, those of men (106). These changes create an advantage of greater bone strength and stronger fulcrum power from longer bones. Additionally, whereas passing through puberty enhances male physical performance, the widening of the female pelvis during puberty, balancing the evolutionary demands of obstetrics and locomotion (136, 137), retards the improvement in female physical performance, possibly driven by ovarian hormones rather than the absence of testosterone (138, 139).

Sex differences in height have been the most thoroughly investigated measure of bone size, as adult height is a stable, easily quantified measure in large population samples. Extensive twin studies show that adult height is highly heritable with predominantly additive genetic effects (140) that diverge in a sex specific manner from the age of puberty onwards (141, 142), the effects of which are likely to be due to sex differences in adult circulating testosterone concentrations.

Bone density (total and medullary cross sectional area) is increased in women with CAH with variably elevated serum testosterone (including into the male range) when it is only partially suppressed by glucocorticoid treatment (143), although more effective glucocorticoid suppression lowers bone density (144).

Interventional data

Well designed, placebo controlled direct interventional studies of supraphysiological androgen effects on bone in females are few, rarely feasible, and unlikely to be performed for ethical and practical reasons. Unlike muscle, which responds relatively rapidly to androgen effects so that muscle studies in humans can be completed within 3 to 4 months (65, 111, 112, 119, 145), comparable bone studies would typically take a year or more to reach plateau effects. Hence, such direct investigational studies in otherwise healthy women would risk side effects of virilization that may be only slowly and partly reversible, if at all, as well as potential promotion of hormone dependent cancers making such studies ethically and practically not feasible.

Effects on athletic performance

The major effects of men's larger and stronger bones would be manifest via their taller stature as well as the larger fulcrum with greater leverage for muscular limb power exerted in jumping, throwing, or other explosive power activities. The greater cortical bone density and thereby resistance to long bone fractures is unlikely to be relevant to the athletic performance of young athletes, in whom fractures during competition are extremely rare and not expected to be linked to sex. Alternatively, stress fractures in athletes, mostly involving the legs, are more frequent in females with the male protection attributable to their larger and thicker bones (146).

Other androgen-sensitive sex dichotomous effects

Biology and observational data

Many if not most other aspects of physiology exhibit sex differences and may therefore enhance the impact of the male advantage in sports performance of the dominant determinants (muscle and hemoglobin). Examples include sex differences in exercise induced cardiac (147, 148) and lung (149) function and mitochondrial biogenesis and energetics (95). However, the limited knowledge of the magnitude and hormonal mechanisms involved, specifically the degree of androgen dependence of these mechanisms, means that it is difficult to estimate their contribution, if any, toward the sex difference in athletic performance. The sex difference in pulmonary function may be largely explained by the androgen sensitive sex difference in height, which is a strong predictor of lung capacity and function (149). Further physiological studies of the androgen dependence of other physiological sex differences are awaited with interest.

Psychological differences between men and women on mental function (*e.g.*, rotational orientation) (150) as well as mood, motivation, and behavioral effects may involve androgen sensitive effects during prenatal and perinatal as well as postpubertal effects (151, 152).

Interventional data

There is some limited direct evidence from well designed, placebo controlled trials that administration of testosterone or other androgens at supraphysiological doses directly affect mood and behavior, notably inducing hypomania (153). In a randomized placebo controlled study of testosterone administration in postmenopausal women (112), in case of those receiving the highest dose (the only one causing circulating testosterone levels to exceed the normal female range), there was not only an increase in muscle mass (4.4%) but a strikingly greater increase in muscle strength (12% to 26%), suggesting an enhanced mental motivational effect of testosterone on the effort dependent tests of muscle strength.

Alternative Mechanisms Proposed to Explain Sex Differences in Athletic Performance

Alternative explanations for the sex difference in athletic performance, other than it being due to the sex difference in postpubertal circulating testosterone, have been proposed, including (1) sex differences in height because height is a predictor of muscle mass (116), (2) genetic sex differences due to the influence of unspecified Y chromosome genes (154), and (3) sex differences in GH secretion (116).

Effects of height

One proposal has been that, as men are taller than women, height differences may explain the sex differences in muscle mass and function, which explains some athletic success (116). Numerous factors contribute to the regulation of adult muscle mass, including genetics, race, adiposity, hormones, physical activity (exercise/training), diet, birth order, and bone size (including height) [reviewed in (155)]. Among the nonhormonal factors, genetics explains a large proportion [~50% to 60% from pooled twin studies (156)] of the variability in muscle mass and strength (157, 158) and may be explained in turn by the equally high genetic contributions to circulating testosterone (37, 38). Some factors influencing muscle mass and strength such as physical activity, adiposity, and bone size are also partly androgen dependent. Prior to puberty there is no sex difference in skeletal features, including height (159, 160). However, with the onset of puberty, girls aged 11 and 12 years are transiently taller than peer aged boys due to their earlier onset of the female pubertal growth spurt, but from the age of 14 years onward the taller stature in males emerges and stabilizes (141). Hence, similar to muscle mass, sex differences in bone size (including length, density, and height) arise after male puberty establishes the marked dichotomy between men and women in adult circulating testosterone concentrations. Taller height is

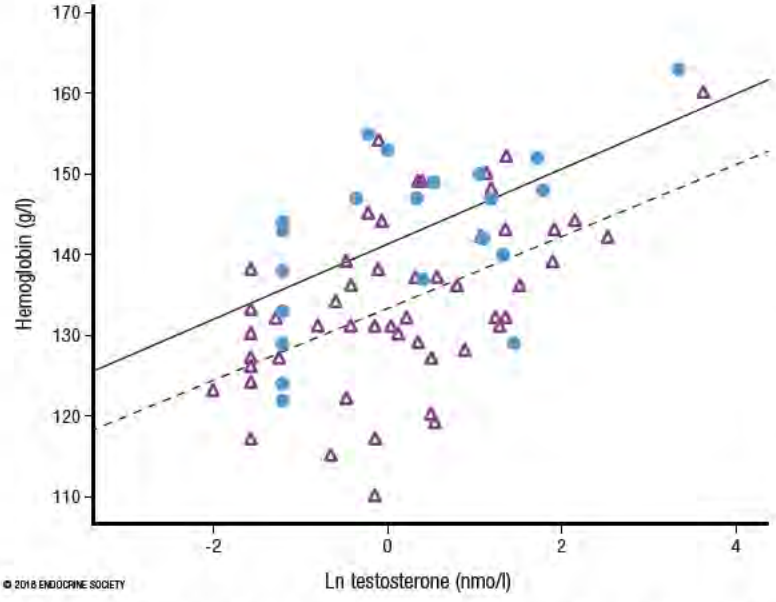


Figure 5. Plot of circulating hemoglobin against the natural logarithm of serum testosterone in women with congenital adrenal hyperplasia [from Karunasena *et al.* (92)]. The filled circles represent a cohort where serum testosterone was measured by immunoassay. The open triangles denote a second cohort, where serum testosterone was measured by LC-MS. Note the systematic overestimation of testosterone by the immunoassay used in cohort 1 vs LC-MS measurement in cohort 2. Despite that overestimation, however, the correlations were similar in both cohorts. Reproduced under a Creative Commons BY-NC-ND 4.0 license from Karunasena N, Han TS, Mallappa A, *et al.* Androgens correlate with increased erythropoiesis in women with congenital adrenal hyperplasia. Clin Endocrinol (Oxf) 2017;86:19–25.

advantageous in some sports (basketball, some football codes, combat sports), but in others (horse racing jockeys, cycling, gymnastics, weightlifting, body building) short stature provides a greater power/strength to weight ratio as well as superior rotational balance, speed, and agility. However, the male advantages in speed, strength, and endurance apply regardless of whether height is advantageous. Hence, the sex differences in height, where they exist, are largely dependent on postpubertal differences in circulating testosterone when sex differences in height are first expressed.

Genetic effects of Y chromosome

It has also been proposed that the sex difference in athletic performance may be due to genetic effects of an unspecified Y chromosome gene that may dictate taller stature (154), as height is correlated with men's greater muscle mass. The small human Y chromosome has few functional genes and none with a known effect on height other than the short stature homeobox (SHOX) gene, located in the pseudoautosomal regions of the tip of the short arms of X and Y chromosomes (161). Adult height displays an apparent dose dependency on SHOX gene copy number that is a major factor contributing to explaining both the short stature of 45,XO females (Turner syndrome), who have a single copy of the SHOX gene, as well as the tall stature of 47,XXY males (Klinefelter syndrome), who have three copies (161). However, when SHOX copy number is the same, men with additional supernumerary Y chromosomes (*e.g.*, 47,XYY) are the same height as 47,XXY men (162). Hence, there is no evidence supporting dosage dependent Y chromosomal gene effects on height independent of SHOX gene copy number, nor does men's possession of a Y chromosome explain the height difference between adult men and women. On the contrary, the tall stature of 47,XXY men is at least partly due to the concomitant androgen deficiency leading to pubertal delay. Pubertal delay prolongs long bone growth due to delayed epiphyseal closure, an estrogen dependent effect that requires adequate production of testosterone as a substrate for aromatization to estradiol, resulting in tall stature. Similar eunuchoidal features and taller stature are evident in 46,XY men with congenital hypogonadotropic hypogonadism (Kallmann syndrome and its variants) with comparable congenital onset of androgen deficiency, also manifest as pubertal delay and long bone overgrowth. Hence, taller height is better explained by impaired testicular function with delayed puberty and epiphyseal closure rather than unspecified Y chromosome dosage effects. In any case, rare aneuploidies in themselves do not explain the sex difference in height in the general population of individuals with normal sex chromosomes.

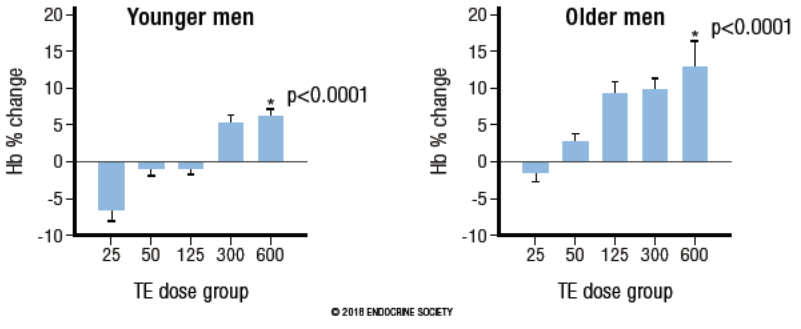


Figure 6. From Coviello *et al.* (131): Depicts the strong dose-response relationship between increasing testosterone dose with resulting change in blood hemoglobin in young and older men. Reproduced with permission from Coviello AD, Kaplan B, Lakshman KM, *et al.* Effects of graded doses of testosterone on erythropoiesis in healthy young and older men. J Clin Endocrinol Metab 2008;93:914–919.

Growth hormone

The proposal that the sex difference in muscle mass and function might be due to sex differences in endogenous GH secretion (116) is refuted by the extensive and conclusive clinical evidence that endogenous GH secretion in young women is consistently higher (typically twice as high) as in young men of similar age (163–170). Those findings cannot explain the male advantage in muscle mass and strength unless GH retards muscle growth/function, for which there is no evidence. Furthermore, estrogens inhibit GH dependent, hepatic IGF-1 production, the major pathway of GH action (171, 172). The weak observational association between low circulating IGF-1 and some, but not other, measures of weak muscle strength and limited mobility among older women may reflect general age associated debility rather than any specific hormonal effects (173). Finally, the evidence that endogenous GH plays no role in sex differences in muscle mass and function is supported by evidence from the most extensive interventional study of GH treatment to non-GH deficient adults, daily GH administration for 8 weeks to healthy recreational athletes produced only marginally significant improvement in exercise performance of men and none in women (174). These findings are consistent with the speculation that GH (or IGF-1) may be an amplifier of testosterone effects and therefore be a consequence of the sex difference in circulating testosterone rather than its cause.

The Impact of Adult Male Circulating Testosterone Concentrations on Sports Performance

Plausible estimates of the magnitude of the ergogenic advantage of adult male circulating testosterone concentrations are feasible from the limited available observational and interventional studies.

Population data on the ontogeny of puberty show that prior to puberty boys and girls have comparable athletic performance, whereas sex differences in athletic performance emerge coinciding with the rise in circulating testosterone from the onset of male puberty. Male puberty results in circulating testosterone concentrations rising from the prepubertal and female postpubertal range (<2 nmol/L) to adult male circulating testosterone concentrations (18). This is associated with a 10% to 12% better performance in running and swimming events and 20% enhancement in jumping events (8).

A minimal estimate of the impact of adult male testosterone concentrations on muscle size and strength in females is provided by the Huang *et al.* (112) study of postmenopausal women. In this study the highest testosterone dose (weekly injections of 25 mg of testosterone enanthate) increased mean circulating testosterone from 0.9 nmol/L to 7.3 nmol/L, which is equivalent to the circulating testosterone of boys in early to middle puberty. After 24 weeks of testosterone treatment, the increase in circulating testosterone concentrations led to significant increases in muscle size of 4.4% and in muscle strength of 12% to 26%. Given the limited testosterone dose (and concentration) as well as study duration, it is likely that these findings underestimate the magnitude of the impact that sex difference in circulating testosterone has on muscle mass and strength, and therefore on athletic performance.

Converse effects of reduced athletic performance in athletes who undergo suppression of circulating testosterone concentrations from those in the male into the female range have been reported. Among recreational (nonelite) athletes, an observational study showed a consistent deterioration in athletic performance of transwomen (M2F transgender) athletes corresponding closely to the suppression of circulating testosterone concentrations (175). Similarly, among elite athletes with circulating testosterone in the male range due to DSDs, comparable findings of athletic performance reduced by an average of 5.7% when circulating testosterone was suppressed from the male range to <10 nmol/L (176). Subsequently, when the IAAF hyperandrogenism rule was suspended in 2015, and so these elite athletes could train and compete with unsuppressed serum testosterone levels, their athletic performances increased by a similar amount. Additionally, circulating hemoglobin levels in these untreated DSD athletes were comparable with male athletes or with female athletes doping with erythropoietin (Fig. 7). However, when circulating testosterone was suppressed to <10 nmol/L the levels of circulating hemoglobin were 12% lower and again comparable with nondoped, non DSD females, corresponding to the 12% magnitude of the sex difference in hemoglobin between men and women (125).

Congruent findings are also known for an elite female athlete whose serial athletic performance based on publicly available best annual times between 2008 and 2016 for the 800 m running event are depicted in relationship to the original 2011 IAAF hyperandrogenism regulation (Fig. 8).

Based on the established dose response relationships, suppression of circulating testosterone to <10 nmol/L would not eliminate all ergogenic benefits of testosterone for athletes competing in female events. For example, according to the Huang *et al.* (112) study, reducing circulating testosterone to a mean of 7.3 nmol/L would still deliver a 4.4% increase in muscle size and a 12% to 26% increase in muscle strength compared with circulating testosterone at the normal female mean value of 0.9 nmol/L. Similarly, according to the Karunasena *et al.* (92) study, reducing circulating testosterone concentration to 7 nmol/L would still deliver 7.8% more circulating hemoglobin than the normal female mean value. Hence, the magnitude of the athletic performance advantage in DSD athletes, which depends on the magnitude of elevated circulating testosterone concentrations, is considerably greater than the 5% to 9% difference observed in reducing levels to <10 nmol/L.

The physiological mechanism underlying these observations is further strengthened by prospective controlled studies of initiation of cross sex hormone treatment in transgender individuals (114, 177). These show that during the first 12 months muscle mass (area) was decreased by 9.4% and hemoglobin levels by 14% in 20 transwomen (M2F transgender) treated with an estrogen based regimen that reduced circulating testosterone concentrations from the male range to the female range. Conversely, in 17 transmen (F2M transgender) treated for the first time with testosterone for 12 months (which increased circulating testosterone levels to a mean of 31 nmol/L), muscle mass increased by 19.2% and hemoglobin by 15% (114). The muscle mass findings remained stable between 1 and 3 years after initiation of treatment, although fat mass continued to change between 1 and 3 years of testosterone treatment (177). These studies did not report muscle strength, but other studies of testosterone dose response relationships for muscle mass and strength show consistently positively correlation (65, 93, 117, 119), although with disproportionately greater effect on muscle strength than on muscle mass. Hence, the muscle mass estimates in these prospective treatment initiation studies in transgender individuals likely underestimate the muscle strength gains from elevated testosterone levels where the circulating testosterone markedly exceeds female range to be within the male range as occurs in severe hyperandrogenism of DSD females, poorly controlled transwomen (M2F transgender), or transmen (F2M transgender). These effects are also the biological

basis of the ergogenic efficacy of androgen doping in women.

Finally, to put these competitive advantages into context, the winning margin (the difference in performance by which a competitor misses a gold medal, any medal, or making the final) in elite athletic or swimming events during the last three Olympics is <1% equally for both male and female events (Table 5).

Gaps in Knowledge and Research Limitations

The major limitations on scientific knowledge of the impact of adult male circulating testosterone concentrations on the sex difference in athletic performance is the lack of well designed studies. Ideally, these would need to replicate adult male circulating testosterone concentrations for sufficient time in women to investigate the effects on muscle, hemoglobin, bone, and other androgen sensitive measures that display consistent sex dichotomy in the population. However, the ethical and safety concerns preventing such studies hitherto are likely to remain formidable obstacles due to the risk of unacceptable and potentially irreversible virilization as well as of promoting hormone dependent cancers in women.

With the exception of one interventional study administering a relatively low testosterone dose (*i.e.*, low for males) to women (112), the available evidence comprises observational studies that can only examine the effects of serum testosterone within physiological female limits or sparse and mostly uncontrolled data from intersex/DSD athletes. Although the available observational findings in healthy females are informative, the key question is the magnitude and dose response of effects at still higher circulating testosterone concentrations on the performances of women. Whereas a testosterone dose response relationship has been established in women at relatively low (for men) testosterone dose and circulating concentrations, it remains unproven (even if clearly plausible) that the testosterone dose response relationships established in men for muscle, hemoglobin, and bone can be extrapolated to women when they are exposed to higher circulating testosterone concentrations (*i.e.*, comparable with male levels). It is theoretically possible there could be differences between men and women in muscle responses to testosterone, as muscle cell populations might express genetic differences in androgen sensitivity (for which there are no data), or alternatively the long term prior pattern of testosterone exposure from conception to adulthood might lead to differences in testosterone dose responsiveness after maturity. Although the dose response relationship in women may be similar to what is seen in men, there is also anecdotal evidence that the dose response curves may be left shifted so that testosterone has greater potency in women than in men at comparable doses and circulating levels. The prediction is supported by the anecdotal evidence from the surreptitious East German national doping program in which the supervising doctors asserted from their experience of illicit cheating that androgens had more potent ergogenic effects in women than in men (120), a speculative opinion shared by many experienced sports medicine physicians.

There is no known means of increasing endogenous testosterone in women to anything like the requisite degree to attempt to answer these questions. In healthy men, circulating testosterone originates almost exclusively from a single source (testicular Leydig cells) and is subject to tight hypothalamic negative feedback control, so that either direct stimulation (by human chorionic gonadotropin) or indirect reflex effects (*e.g.*, from estrogen blockers operating via negative feedback) to enhance Leydig cell testosterone secretion are feasible. However, similar mechanisms do not operate in women, in whom circulating testosterone originates from three different sources (adrenal, ovary, extraglandular conversion of androgen precursors), none of which is subject to tight testosterone negative feedback control. As a result, it is not feasible to produce a sufficient increase in circulating testosterone in women either by direct ovarian stimulation or indirect reflex effects to test this hypothesis even if doing so were deemed ethical and safe. Alternatively, carefully controlled, graded dose studies in F2M transgender individuals might be informative but are largely lacking at this time.

Hence, the only feasible design of such studies would be testosterone (or another androgen) administration to healthy young women. The only well designed, placebo controlled study of testosterone in

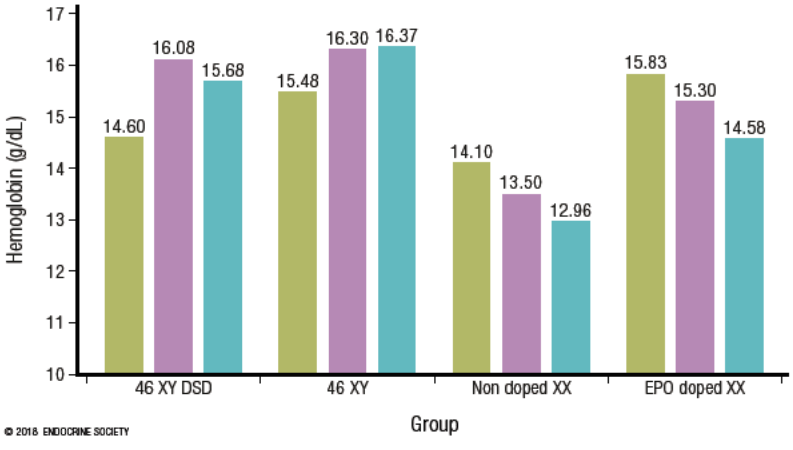


Figure 7. Mean hemoglobin concentrations (g/dL) of 12 elite athletes in 4 groups of 3 XY or XX middle-distance runners. The hemoglobin concentrations were collected as a part of the Athlete Biological Passport and analyzed according to the World Anti-Doping Agency standard methods. Each bar (athlete) is the mean of a minimum of three blood samples. In the 46,XY DSD group, blood was collected in a period when the athlete was not undergoing hormonal suppressive treatment.

otherwise healthy postmenopausal women was restricted to relatively low testosterone doses that, although clearly supraphysiological for women, were only 20% to 25% of male testosterone replacement doses (112). We are currently performing a double blind, randomized, placebo controlled study of the effects of moderately increased testosterone concentration on physical performance and behavior in young healthy women (ClinicalTrials.gov no. NCT03210558). However, obtaining ethical approval to administer supraphysiological testosterone doses that maintain circulating testosterone in the male range for sufficiently prolonged periods, as well as the practical difficulties in recruitment, are likely to remain obstacles to definitive resolution of this question.

In men, analogous ethical concerns over short and long term adverse effects delayed the definitive studies of supraphysiological testosterone doses to healthy young and older men but were eventually overcome. This was despite the fact that, uniquely among hormones, there is no known disease state in men due to pathologically excessive testosterone secretion. In contrast, in women, supraphysiological testosterone effects are known to produce virilization side effects that may be only slowly and partially, if at all, reversible. However, maintaining clearly supraphysiological testosterone concentrations would require treatment of months (muscle) or years (bone) and would replicate not only a known hyperandrogenic disease state (PCOS) but also potentially increasing risk of hormone dependent cancers. In these circumstances, it could only be justifiable to replicate in women the salient testosterone dose response studies available from men if the available evidence of dose response relationship in men was not sufficiently convincing and/or there was reason to think that these dose response characteristics would be substantially different in women. Overall, the unequivocal dose response evidence in men together with the available overlap evidence in women appears sufficiently persuasive, so that it is doubtful that women would respond differently from men if their circulating testosterone levels were raised to the male range. More broadly, there is no more reason to require separate studies in women vs men than there is for every different ethnic subgroup of people. An aesthetic preference for splitting categories is not a sound reason to require the virtually impossible standard of establishing fresh and comprehensive empirical evidence in women of testosterone dose response effects ranging into male circulating testosterone concentrations.

An analogy can be drawn to the World Anti Doping Agency's practice of accepting salient surrogate evidence for banning the plethora of existing and new drugs with potential but individually unproven ergogenic effects where it is not feasible or ethical to require direct proof of the ergogenic effects. In that context, the firmly established ergogenic efficacy of androgens (on muscle mass and strength) and increased hemoglobin (on endurance) [evidence reviewed in (1)] mean that chemical substances or methods that increase endogenous testosterone, erythropoietin, or hemoglobin are also considered ergogenic (178). By parity of reasoning, if a condition causes a female athlete's circulating testosterone levels to be in the male range, well exceeding normal female levels, with consequential increases in muscle, hemoglobin, and bone effects (at least), an ergogenic effect may reasonably be assumed.

Conclusions

The available, albeit incomplete, evidence makes it highly likely that the sex difference in circulating testosterone of adults explains most, if not all, the sex differences in sporting performance. This is based on the dose response effects of circulating testosterone to increase muscle mass and strength, bone size and strength (density), and circulating hemoglobin, each of which alone increases athletic capacity, as well as other possible sex dichotomous, androgen sensitive contributors such as mental effects (mood, motivation, aggression) and muscle myoglobin content. These facts explain the clear sex difference in athletic performance in most sports, on which basis it is commonly accepted that competition has to be divided into male and female categories.

The first IAAF hyperandrogenism regulation specified a hormonal eligibility criterion of a serum testosterone of <10 nmol/L for an androgen sensitive athlete's participation in the protected category of female athletic events. This threshold was based on serum testosterone measurements by immunoassays.

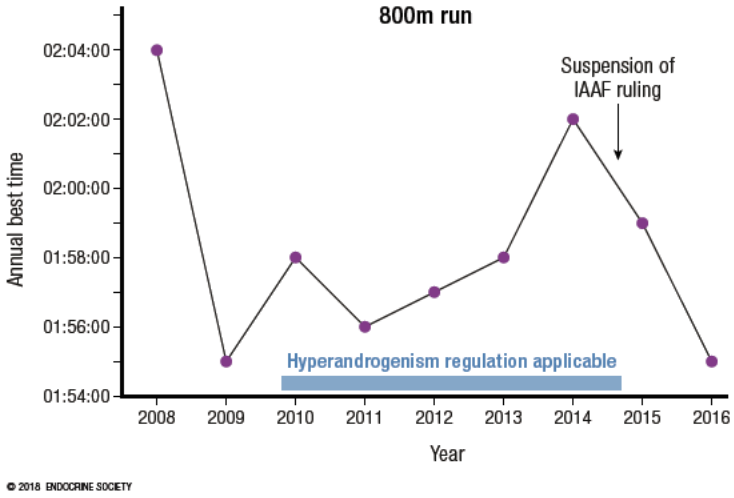


Figure 8. Best annual 800-m times of an elite female athlete between 2008 and 2016. Data provided by Dr. Richard Auchus, University of Michigan, Ann Arbor, Michigan.

Table 5. The Winning Margin in Elite Athletic or Swimming Events During the Last Three Olympics

Median Margin (%)[a]	n	Win Gold	Win Medal	Make Final
Athletics[b]				
Running	81	0.62	0.31	0.22
Jumping	24	0.92	0.42	0.92
Throwing	24	1.93	0.70	0.75
Swimming[c]				
Backstroke	12	0.56	0.28	0.16
Breaststroke	12	0.84	0.14	0.17
Butterfly	12	0.52	0.48	0.12
Freestyle	30	0.49	0.23	0.14
Relay	18	0.37	0.35	0.12

[a]Winning margin is defined as the difference (expressed as a percentage of the faster time) between first and second place (Win Gold), between third and fourth place (Win Medal), and between the last into the final and the first that missed out (Make Final). Years (2008, 2012, 2016) and sexes were combined as there were no significant differences in winning margin between them.
[b]Running includes 100 m, 200 m, 400 m, 800 m, 1500 m, 5000 m, 10,000 m, marathon, and 3000-m steeplechase, 110-m (male)/100-m (female) and 400-m hurdles, 4 × 100-m and 4 × 400-m relays, and 20-km and 50-km walk events. Jumping includes high jump, long jump, triple jump, and pole vault events. Throwing includes javelin, shot put, discus, and hammer events. Heptathlon and decathlon were not included as their final results are in points, not times.
[c]Events comprise 100 m and 200 m for the form strokes and 50 m, 100 m, 200 m, 400 m, 800 m (female)/1500 m (male) and marathon 10 km, with the relays being the 4 × 100-m medley and 4 × 100-m and 4 × 200-m freestyle relays.

However, no reliable method independent consensus threshold could be established using commercial testosterone immunoassays, as these assays differ systematically due to method specific bias arising unavoidably from the specificity of the different proprietary antibodies employed (25). Based on measurements using the more accurate and specific mass spectrometry methods, if the objective is to require female athletes with congenital conditions that cause them to have serum testosterone concentrations in the normal male range to bring those levels down to the same range as other female athletes, then (allowing for PCOS athletes) the threshold used should not be >5.0 nmol/L. This represents a conservative criterion that includes all healthy young (<40 years) women, including those with PCOS. Conversely, this criterion is generous to intersex/DSD females in allowing them to maintain a higher serum testosterone (2 to 5 nmol/L) than most non PCOS competitors in female events even though increases in muscle mass and strength and hemoglobin would be expected in this range. This is so even though the range remains below the circulating testosterone levels of middle male puberty when the major biological effects of men's higher circulating testosterone begin to be fully expressed. Ongoing compliance with the eligibility criterion is also an important variable because the estrogen based suppression of circulating testosterone, typically using daily administered estrogen products, has a rapid onset and offset. Adequate monitoring to prevent gaming of eligibility criteria would require regular random rather than announced blood sampling.

A related matter is how long such a threshold of circulating testosterone should be maintained prior to competition. In both intersex/DSD and transgender individuals, the developmental effects of adult male circulating testosterone concentrations will have established the sex difference in muscle, hemoglobin, and bone, some of which is fixed and irreversible (bone size) and some of which is maintained by the male circulating testosterone concentrations (muscle, hemoglobin). The limited available prospective evidence from initiation of transgender cross sex hormone treatment suggests that the advantageous increases in muscle and hemoglobin due to male circulating testosterone concentrations are induced or reversed during the first 12 months and the androgenic effects may plateau after time. This time course is much faster than the somatic effects of male puberty, which evolve over years and for some variables (*e.g.*, peak bone mass) are not complete for up to a decade after the start of puberty. However, the abrupt hormonal changes induced by medical treatment in intersex/DSD or transgender individuals may be telescoped compared with male puberty where circulating testosterone concentrations increase irregularly and incompletely for some years. Additional data are available from the unique investigative model of men undergoing castration for prostate cancer. Just as androgen sensitivity to testosterone may differ between tissues (65), the time course of offset of

androgen effects following withdrawal of male testosterone concentrations may also differ between the major androgen responsive tissues. For example, circulating hemoglobin shows a progressive fall for 6 months reaching a nadir and plateau at 12 to 16 months in six studies involving 534 men undergoing medical castration for prostate cancer (179 184). Although these studies of older men with prostate cancer must be extrapolated with caution, age, stage of disease, race, and baseline circulating testosterone concentration did not affect the rate or extent of decline in hemoglobin (179, 181). Comparable longitudinal studies of muscle loss, strength, and performance following castration for prostate cancer are well summarized (185), showing progressive loss for 24 months (see Fig. 4). Further clinical studies to define the time course of changes, mainly offset, in testosterone dependent effects, notably on muscle and hemoglobin, are badly needed to determine the optimal duration for cross sex hormone effects in sports.

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Disclosure Summary: D.J.H. is a medical and scientific consultant for the IAAF and to the Australian Sports Anti-Doping Agency. He is a member of the World Anti-Doping Agency's Health, Medicine and Research Committee and of the IOC working group on hyperandrogenic female and transgender athletes. He has received institutional grant support from Besins Healthcare and Lawley for investigator-initiated clinical studies in testosterone pharmacology and has provided expert testimony in testosterone litigation. A.L.H. is a medical and scientific consultant for the Swedish Olympic Committee and a member of the IAAF and IOC working groups on hyperandrogenic female athletes and transgender athletes. She has received grant support from the IAAF for a study on testosterone and physical performance in women. S.B. is a medical and scientific consultant for the IAAF and a member of the IAAF and IOC working groups on hyperandrogenic female athletes and transgender athletes. The authors have no other involvement with any entity having a financial interest in the material discussed in the manuscript. Opinions expressed in this review are the personal views of the authors and do not represent those of the IAAF, IOC, World Anti-Doping Agency, or Swedish Olympic Committee.

Abbreviations

AR, androgen receptor; CAH, congenital adrenal hyperplasia; CAIS, complete androgen insensitivity syndrome; DSD, disorder (or difference) of sex development; F2M, female-to-male; IAAF, International Association of Athletic Federations; IOC, International Olympic Committee; LC-MS, liquid chromatography mass spectrometry; M2F, male-to-female; PAIS, partial androgen insensitivity syndrome; PCOS, polycystic ovary syndrome; SHOX, short stature homeobox.

Exhibit 19

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ORIGINAL ARTICLE

WILEY

Sex differences in athletic performance emerge coinciding with the onset of male puberty

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Summary

Background: Male performance in athletic events begins to exceed that of age-matched females during early adolescence, but the timing of this divergence relative to the onset of male puberty and the rise in circulating testosterone remains poorly defined.

Design: This study is a secondary quantitative analysis of four published sources which aimed to define the timing of the gender divergence in athletic performance and relating it to the rise in circulating testosterone due to male puberty.

Data: Four data sources reflecting elite swimming and running and jumping track and field events as well as hand-grip strength in nonathletes were analysed to define the age-specific gender differences through adolescence and their relationship to the rising circulating testosterone during male puberty.

Results: The onset and tempo of gender divergence were very similar for swimming, running and jumping events as well as the hand-grip strength in nonathletes, and all closely paralleled the rise in circulating testosterone in adolescent boys.

Conclusions: The gender divergence in athletic performance begins at the age of 12-13 years and reaches adult plateau in the late teenage years with the timing and tempo closely parallel to the rise in circulating testosterone in boys during puberty.

KEYWORDS
age group, performance, puberty, swimming, testosterone, track and field

1 | INTRODUCTION

It is well known that men's athletic performance exceeds that of women especially in power sports because of men's greater strength, speed and endurance. This biological physical advantage of mature males forms the basis for gender segregation in many competitive sports to allow females a realistic chance of winning events. This physical advantage in performance arises during early adolescence when male puberty commences after which men acquire larger muscle mass and greater strength, larger and stronger bones, higher circulating haemoglobin as well as mental and/or psychological differences. After completion of male puberty, circulating testosterone levels in men are consistently 10-15 times higher than in children or women at any age.[1] The age at which sex differences emerge is reported as around the age of 12 from a study of individual Norwegian athletes in two running and two jumping events[2] and at 13-14 years in four track and field skills in Polish athletes[3]; however, the relationship to male puberty and circulating testosterone is not clear. This study investigates the age of the gender divergence in performance in elite swimming and a wider range of elite athletic events as well as a community-based study of grip strength among nonathletes to deduce the onset and progression of the gender divergence in performance of athletes and relates this to the timing and tempo of male puberty and the rise in circulating testosterone into adult male levels.

2 | MATERIAL AND METHODS

Four sources of published data were used in this study for which no ethics approval was required. The first was the US Age Group Swimming time standards which lists the prevailing time standard for entry to the top level (AAAA long course criteria) of all boys and girls events for individual years from 1981 to 2016 (accessed Oct 2016).

http://www.usaswimming.org/DesktopDefault.aspx?TabId=2628&Alias=Rainbow&Lang=en

Age groups were classified into five categories 10 and under, 11-12 years, 13-14 year, 15-16 years and 17-18 years. The seven events in common to all age groups were freestyle (50 m, 100 m, 200 m), backstroke, breaststroke and butterfly (all 100 m) and individual medley (200 m).

A second data source was the current world records for boys and girls between the ages of 5 and 19 years available at http://age-records.125mb.com/ (curated by Dominique Eisold, accessed Oct 2016). This included sufficient data to cover the timing of puberty onset with some pre- and postpuberty ages (ages 9-19 years) for a wide range of boys and girls track and field events. For this study, the running events included were 50 m, 60 m, 100 m, 200 m, 300 m, 400 m, 500 m, 600 m, 800 m, 1000 m, 1500 m, 1 mile, 2000 m, 3000 m and 2 miles. Only records recorded by fully automatic timing devices were included whether set indoor or outdoor or at altitude (>1000 m), but wind-assisted records were excluded from this analysis. The jumping events included were high jump, pole vault, long jump, triple jump, standing long jump.

The third data source was from a published study[1] in which serum testosterone was measured in over 100 000 consecutive serum samples processed over 7 years from a single pathology laboratory which was analysed to estimate male and female age-specific reference ranges across the full lifespan.

The fourth was a meta-analysis of secular changes in hand-grip strength in nonathletic children and adolescents from Canada and United States[4] using the data provided on 5676 males and 5489 females in 19 studies conducted between 1966 and 2009.

Data analysis was performed by analysis of variance and nonlinear curve fitting using NCSS 11 Statistical Software (NCSS LLC. Kaysville, Utah, USA). For each event used in this analysis, the age-specific record or age-group time standard was defined for boys (Tb) and girls (Tg) so the difference (expressed as a percentage) between boys and girls for any event was defined as D=(Tg−Tb)*100/Tg. For athletic jumping events, an analogous definition for record length was used (Lb for boys, Lg for girls) with the male advantage defined as D=(Lb−Lg)*100/Lg. For the athletic events where individual year age records were available across the age of puberty, the age-specific difference (as a percentage) for each year of age were pooled into running or jumping categories. For track and field performance, the pooled data were fitted to a four-parameter sigmoidal curve which allowed for asymptotic estimation of the lower (prepubertal) and upper (postpubertal) plateaus from the four parameters. In addition, the timing and tempo of the pubertal increase were defined by the start of puberty, defined as the time when 20% of the ultimate increase due to puberty had occurred (ED_{20}), and mid-puberty as the time when half the ultimate increase had occurred (ED_{50}). For swimming, the pooled gender differences for all strokes and distances were fitted by a smoothed spline curve. For hand-grip strength, the differences were fitted to a piecewise linear-quadratic curve with a single inflexion point.

3 | RESULTS

In swimming performance, the overall gender differences were highly significant with age group ($F_{4,360}$=1481, P<.0001) and stroke ($F_{4,360}$=11.9, P<.0001) as main (between) effects (Figure 1). There was no significant difference according to year (as a within factor, P=.99) so that for further analysis, years were taken as replicates. Using a sigmoidal curve fit for the overall gender differences pooling all strokes and distances, the ED_{20} was 11.4 years and the ED_{50} was 12.8 years.

Within a single stroke (freestyle), in addition to expected age-group effects ($F_{4,525}$=2174, P<.0001), there were also significant effects according to distance ($F_{2,525}$=231.5, P<.0001) whereby the age-group effects was significantly greater the shorter the event distance (Figure 2, 50 m>100 m>200 m, age group x distance interaction, $F_{8,525}$=55.9, P<.0001) (Figure 1). Similarly, for a fixed length of events (100 m) and after taking age-group effects into account, the four form strokes did differ significantly ($F_{3,700}$=12.9, P<.0001) producing significant

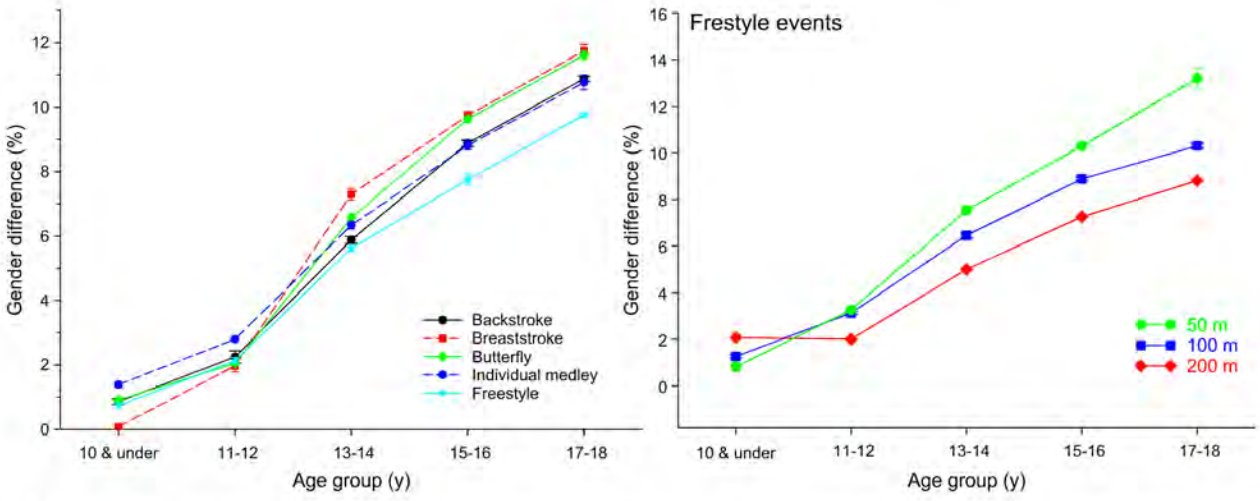

FIGURE 1 Gender differences in performance (in percentage) according to age group and stroke (left panel) or distance in freestyle events (right) in swimming events. Data shown as mean and standard error of the mean. Note greatest increase after the age of 12 years by age in breaststroke and least in freestyle and magnitude of increases are 50 m>100 m>200 m in freestyle events. [Colour figure can be viewed at wileyonlinelibrary.com]

differences between strokes (interaction $F_{12,700}$=23.4, P<.0001), the most prominent being for breaststroke, which displayed the greatest age-group effect, and butterfly followed by backstroke and then freestyle, which showed the least age-group effect (Figure 1).

In track and field athletics, the effects of age on running performance (Figure 2 upper left panel) showed that the prepubertal differences of 3.0% increased to a plateau of 10.1% with an onset (ED_{20}) at 12.4 years and reaching midway (ED_{50}) at 13.9 years. For jumping (Figure 2 upper right panel), the prepubertal difference of 5.8% increased to 19.4% starting at 12.4 years and reaching midway at 13.9 years. The timing of the male advantage in running, jumping and swimming was similar and corresponded to the increases in serum testosterone in males (Figure 2 lower panel).

To examine age of gender divergence in strength in an analogous data set from a nonathletic population (Canadian and US children and adolescents), the age trends in hand-grip strength showed a difference in hand-grip strength commencing from the age of 12.8 years onwards (Figure 3). Prior to the age of 13 years, boys had a marginally significant greater grip strength than girls (n=45, t=2.0, P=.026), but after the age of 13 years, there was a strong significant relationship between age and difference in grip strength (n=18, r=.89, P<.001).

4 | DISCUSSION

The present study shows that the gender divergence in performance for swimming and for running and jumping track and field events is very closely aligned to the timing of the onset of male puberty, which typically has onset at around 12 years of age.[5,6] These findings are consistent with reports on the timing of the gender differences in performance observed among Norwegian athletes in two running and two jumping events[2] and for track and field skills among Polish athletes.[3] This study extends the findings to swimming and a wider range of running and jumping track and field events. This timing is also consistent with the start of the gender divergence in fat-free (muscle) mass[7] and strength increases.[8,9]

In this study, the timing and tempo of male puberty effects on running and jumping performance were virtually identical and very similar

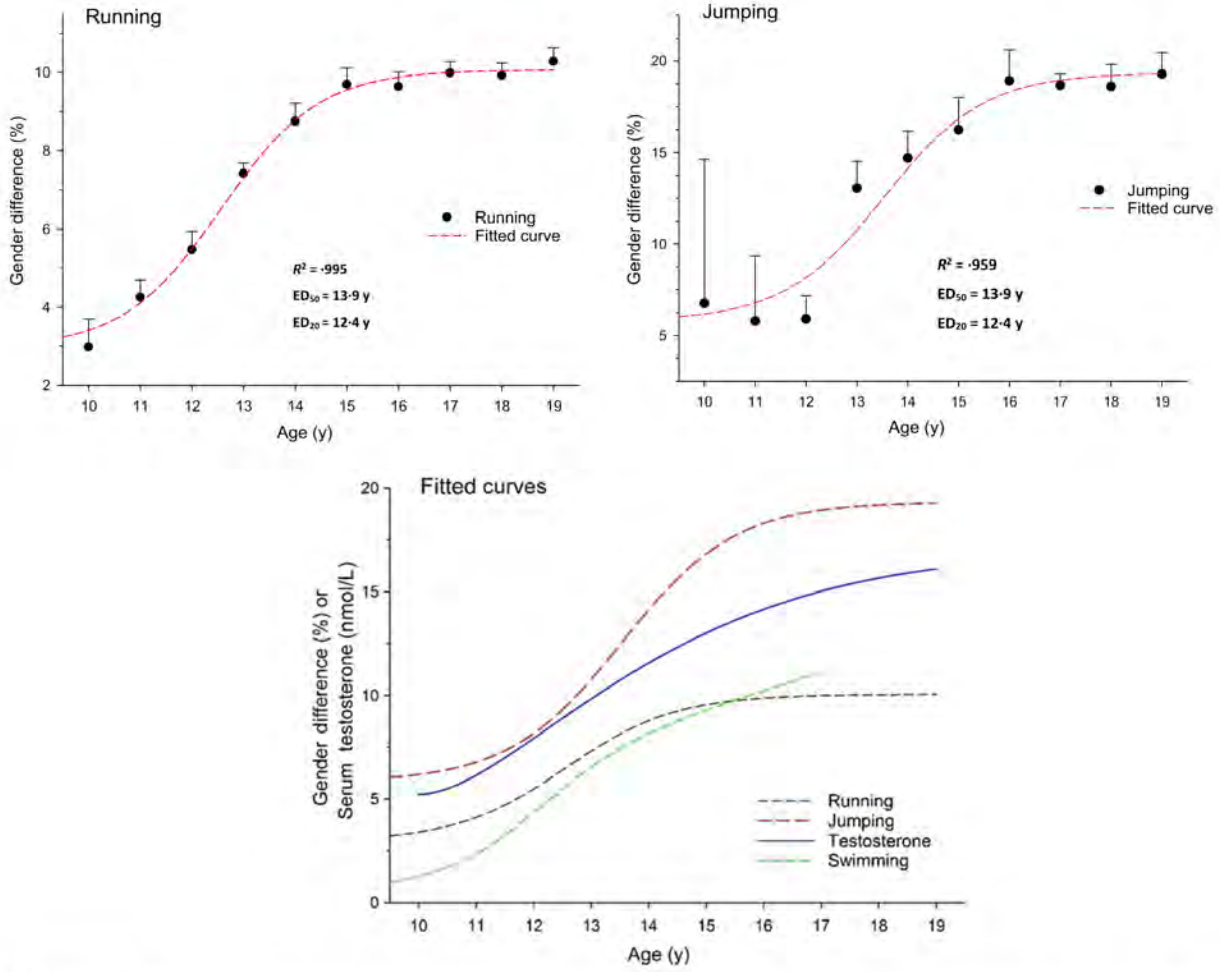

FIGURE 2 Gender differences in performance (in percentage) according to age (in years) in running events including 50 m, 60 m, 100 m, 200 m, 300 m, 400 m, 500 m, 600 m, 800 m, 1000 m, 1500 m, 1 mile, 2000 m, 3000 m and 2 miles (upper left panel) and in jumping events including high jump, pole vault, triple jump, long jump and standing long jump (upper right panel). Fitted sigmoidal curve plot of gender differences in performance (in percentage) according to age (in years) in running, jumping and swimming events as well as serum testosterone (lower panel). Data shown as mean and standard error of the mean of the pooled gender differences by age. [Colour figure can be viewed at wileyonlinelibrary.com]

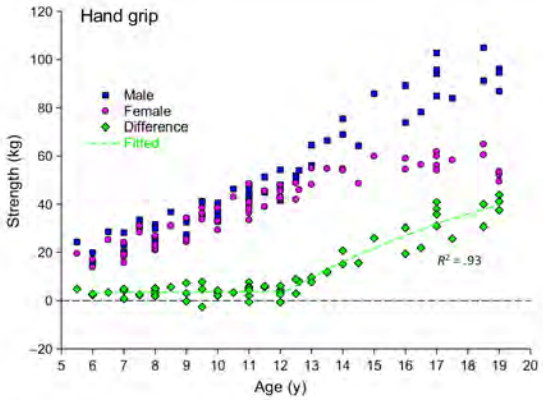

FIGURE 3 Hand-grip strength in children and adolescents from 19 studies including 5676 males (square) and 5489 females (circles) and the differences between male and females (diamonds) conducted between 1966 and 2009. The dotted line represents the fitted curve using a piecewise linear-quadratic curve fit with an automatically defined inflexion point at 12.8 years. [Colour figure can be viewed at wileyonlinelibrary.com]

to those in swimming events. Furthermore, these coincided with the timing of the rise in circulating testosterone due to male puberty. In addition to the strikingly similar timing and tempo, the magnitude of the effects on performance by the end of this study was 10.0% for running and 19.3% for jumping, both consistent with the gender differences in performance of adult athletes previously reported to be 10%-12% for running[10,11,12] and 19% for jumping.[12] The similar magnitude of the plateau effects observed for the oldest (postpubertal) stages in this study with mature adult gender differences suggests there are likely minimal if any further divergences in gender performance among athletes after the age of 20 years.

In the swimming events, despite the continued progressive improvements in individual male and female event records, the stability of the gender difference over 35 years shown in this study suggests that the gender differences in performance are stable and robust. These findings are consistent with a previous report of no narrowing of the gender gap in swimming event performance over more than three decades.[12] These findings contribute to discounting previous suggestions that the gender gap in performance of athletes was narrowing and might even disappear,[13] interpretations which were confounded by the increasing participation of females in elite sports through the 20th century that led to short-term accelerating improvement until women approached closer to contemporary female performance plateau.[12] The greater effect of male puberty on shorter freestyle events is consistent with the greater power demands of short sprint events than for longer freestyle events that involve more endurance. The consistent differences between form strokes over 100-m events, even after accounting for the very dominant age-group effect, suggest that the power demands on performance were most prominent in breaststroke and least in freestyle, presumably due to the different mechanical demands of the different strokes.

The gender divergence in hand-grip strength among nonathletic children and adolescents strengthens the view that these gender divergences are a feature of normal male puberty rather than being a feature that manifests only in elite athletes.

The similar time course of the rise in circulating testosterone with that of the gender divergences in swimming and track and field sports is strongly suggestive that these effects arise from the increase in circulating testosterone from the start of male puberty.[1] Somatic effects of male puberty differ in responsiveness to the postpubertal increase in serum testosterone. Muscle effects of testosterone have been established in well-controlled, interventional clinical experiments in healthy young[14,15] and older[16] men. Testosterone increases muscle mass and strength over weeks to months with a strong dose-response evident from below to above physiological testosterone doses and concentrations. Analogous findings are reported in androgen-deficient (hypogonadal) men administered testosterone replacement therapy[17] and in women receiving appropriately lower testosterone doses,[18] and observational dose-effect relationship between endogenous testosterone and upper or lower body muscle mass is reported in healthy men.[19] Most if not all sex differences in maximal oxygen uptake are explained by differences in muscle mass.[20-22]

Adult male circulating testosterone also has marked effects on bone development leading to longer, stronger and denser bone than in age-matched females.[23] However, testosterone effects on bone are slower in onset and probably less reversible than effects on muscle. For example, men achieve peak bone mass at the end of skeletal maturation only in the early 1920s, about a decade after the start of sustained exposure to adult male testosterone levels. Furthermore, while testosterone deficiency may lead to loss of bone density,[23] the overall structural framework of the skeleton is likely to change slowly if at all. Hence, the extent to which testosterone-induced bone changes contribute to the male advantage in adolescent athletic performance is unclear but is probably at least not maximal until the third decade of life by which time the gender differences are already stabilized.

A further biological advantage of adult male circulating testosterone concentrations is the increased circulating haemoglobin. Men have ~10 g/L greater haemoglobin than women[24] with the gender differences also evident from the age of 13-14 years.[25] Testosterone effects on haemoglobin are replicated by administration of exogenous testosterone in a dose-dependent fashion[26] within 1-3 months.[27] Like the effects on muscle, the erythropoietic effect of testosterone is relatively rapid and reversible in contrast to the slower effects on bone. Although a higher haemoglobin is likely to provide advantages in endurance rather than power events, it is unclear how much the relatively modest magnitude of this gender difference contributes to the male advantage in athletic performance.

Finally, exposure to adult male testosterone concentrations is likely to produce some mental or psychological effects.[28] However, the precise nature of these remains controversial and it is not clear whether, or to what extent, this contributes to the superior elite sporting performance of men in power sports compared with the predominant effects on muscle mass and function.

The strength of the present study is that it includes a wide range of swimming as well as track and field running and jumping events as well as strength for nonathletes for males and females across the ages spanning the onset of male puberty. The similar timing of the gender divergence in each of these settings to that of the rise in circulating

testosterone to adult male levels strongly suggests that they all reflect the increase in muscular size and strength although the impact of other androgen-dependent effects on bone, haemoglobin and psychology may also contribute. Limitations of this study include that it could not extend to all swimming or track and field events due to the restricted participation of younger age groups in more gruelling events. Furthermore, the testosterone measurements were not from the individual athletes included in the analysis of available published data so that the comparisons are cohort-wise rather than based on individuals.

It is concluded that the gender divergence in athletic performance begins at the age of 12-13 years and reaches adult plateau in the late teenage years. Although the magnitude of the divergence varies between athletic skills, the timing and tempo are closely parallel with each other and with the rise in circulating testosterone in boys during puberty to reach adult male levels.

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CONFLICT OF INTERESTS

Nothing to declare.

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Exhibit 20

PLOS ONE

RESEARCH ARTICLE

Sex differences in youth elite swimming

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Abstract

Background

The timing and magnitude of sex differences in athletic performance during early human development, prior to adulthood, is unknown.

Objective

To compare swimming velocity of boys and girls for all Olympic-length freestyle swimming events to determine the age of divergence in swimming performance.

Methods

We collected the all-time top 100 U.S. freestyle swimming performance times of boys and girls age 5 to 18 years for the 50m to 1500m events.

Results

Swimming performance improved with increasing age for boys and girls ($p<0.001$) until reaching a plateau, which initiated at a younger age for girls (15 years) than boys (17 years; sex×age; $p<0.001$). Prior to age 10, the top 5 swimming records for girls were 3% faster than the top boys ($p<0.001$). For the 10^{th}-50^{th} places, however, there were no sex-related differences in swimming performance prior to age 10 ($p = 0.227$). For both the top 5 and 10^{th}-50^{th} places, the sex difference in performance increased from age 10 (top 5, 2.5%; 10^{th}-50^{th} places, 1.0%) until age 17 (top 5, 7.6%; 10^{th}-50^{th} places, 8.0%). For all places, the sex difference in performance at age 18 was larger for sprint events (9.6%; 50-200m) than endurance events (7.1%; 400-1500m; $p<0.001$). Additionally, the sex-related difference in performance increased across age and US ranking from 2.4% for 1^{st} place to 4.3% for 100^{th} place ($p<0.001$), indicating less depth of performance in girls than boys. However, annual participation was ~20% higher in girls than boys for all ages ($p<0.001$).

Conclusion

The top 5 girls demonstrated faster swimming velocities and the 10^{th}-50^{th} place girls demonstrated similar swimming velocities than boys (until ~10 years). After age 10, however, boys demonstrated increasingly faster swimming velocities than girls until 17 years. Collectively,

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these data suggest girls are faster, or at least not slower, than boys prior to the performance-enhancing effects of puberty.

Introduction

Recent high profile cases have raised controversy about whether transgender athletes and XY (intersex) women with differences in sexual development should be allowed to participate in competitions restricted to women, for example Caster v. IAAF [1]. Stemming from the recognized performance-enhancing effects of androgens [2–6], regulation of endogenous androgen levels is now required to be eligible for participation in many women's sports competitions. To better understand the timing and magnitude of sex differences in athletic performance during human development we examined elite swimming performances in youth as a proxy to estimate the expected divergence of athletic performance between girls and boys during periods of low androgen concentrations (pre-puberty) and increasing concentrations of androgens throughout puberty. As reviewed previously [6], human androgens—primarily testosterone and its related metabolite dihydrotestosterone (DHT)—are key factors in the development of muscle, bone and hemoglobin. Androgens largely contribute to bigger and more powerful muscle mass, higher hemoglobin concentrations (and subsequent oxygen carrying capacity), and greater bone strength in men than women [6]. In combination, these androgen-driven and sex-based differences in muscle, bone and hemoglobin contribute to a ~10% higher maximal oxygen consumption capacity ($\dot{V}O_{2max}$) in men compared with women [7, 8].

Androgen levels are not different between the sexes prior to puberty, however, after completion of puberty, circulating testosterone levels are on average ~10–20 times greater in men than children or women at any age [9, 10]. This sex-based difference in circulating testosterone is the basic premise to explain why men have faster performance times than women in many time-based sports including running, cycling, swimming, rowing, etc. [11–17]. Thus, prior to puberty, it would be theorized that sex differences in performance between boys and girls would be negligible—which has been observed previously in athletes ~10–12 years of age [5, 18]. However, the sex-based differences in performance prior to age 10 are unknown and there is no previous data on long distance swimming which likely has the smallest influence from sociocultural biases [13]. Historically, women have had less opportunity to participate in most sports than men, and these differences in opportunity are suspected to contribute to the larger sex differences in performance than would be predicted based on physiological differences between the men and women [14, 19]. Women have been permitted to participate in swimming at the highest levels for many years (since ~1912) and currently more girls typically participate than boys, thus, swimming is an ideal 'experiment of nature' for this question [15]. Elite swimmers are also generally homogenous for high socio-economic status, meaning that sex differences in nutrition or access to medical care are unlikely [20]. There is intensive training from a young age, and practices and competitions are inclusive of both sexes. Additionally, standardized environmental conditions along with state of the art facilities are widely available during championship competitions.

Accordingly, the objective of our study was to determine the age of the divergence of swimming performance between elite boys and girls. To our knowledge, our study is the first to analytically investigate the role of normal human hormonal changes on sex-related differences in sprint and endurance performance in elite youth swimming. We hypothesized that: 1) there would be no sex-differences in swimming performance of girls and boys with similar and low

testosterone concentrations (pre-pubescent years), 2) boys would be faster than girls after the initiation of puberty, and 3) the faster performance of boys would plateau after age 16, as androgen concentrations plateau [21].

Materials and methods

Methods

Finishing times of the top 100 All-Time Freestyle Swimming Records for Long Course Meters for boys and girls between 5 and 18 years of age in one-year age brackets were analyzed for all distances with full datasets (n = 100). Swimming times were downloaded from the USA Swimming Database (https://www.usaswimming.org /Home/times/data-hub) for six freestyle swimming distances from 50 to 1500 meters (50, 100, 200, 400, 800 and 1500 m) on April 4, 2019. Average swimming velocity ($m \cdot min^{-1}$) was calculated from the finishing time as: (race distance) × (finishing time)$^{-1}$. Sex differences in swimming velocity were calculated for each place and event distance as: [(boy's velocity)–(girl's velocity)] × (boy's velocity)$^{-1}$ × 100%. The reduction in swimming velocities of boys and girls across world record place (between 1st and 100th place) was calculated as: (velocity of n^{th} place) × (velocity of 1st place)$^{-1}$ × 100%, for n = 1 to 100. Participation data was accessed via publicly-available membership demographics reports prepared by the USA Swimming Member Services staff for 2015 to 2018 (https://www.usaswimming.org). Additionally, circulating testosterone concentrations of a nationally representative sample of the United States population were downloaded from the National Health and Nutrition Examination Survey (NHANES) coordinated and conducted by the Centers for Disease Control and Prevention (CDC) (https://wwwn.cdc.gov/nchs/nhanes/Search/DataPage.aspx?Compon ent = Laboratory). As described previously [22], testosterone was quantified via isotope dilution liquid chromatography tandem mass spectrometry (ID-LC-MS/MS) based on the National Institute for Standards and Technology's reference method, optimized by the CDC. This analytical quantification method initiated in 2013–2014 testing cycle, and data were analyzed for two consecutive testing cycles (2013–2014 and 2015–2016). These data are representative of the national population in demographic characteristics, and notably, are not specific to an elite-athletic population. All procedures accessed public information and did not require ethical review as determined by the Mayo Clinic Institutional Review Board in accordance with the Code of Federal Regulations, 45 CFR 46.102, and the *Declaration of Helsinki*.

Statistical analysis

Data were reported as means ± SD within the text. Separate full factorial univariate analyses of variance (ANOVAs) were used to compare the dependent variables (swimming velocity and relative performance (%1st place) of boys and girls, and sex differences in swimming velocity) between three independent variables [age (5–18 years), US ranking (1st-100th) and event distance (50 m– 1500 m)]. *Post hoc* analyses (Tukey's HSD multiple comparisons) were used to test for differences between pairs within a data set when significant main effects or interactions were identified for age, US ranking or event distance. Recognizing early puberty may exhibit high statistical leverage on observed sex effects; a sensitivity analysis was conducted by filtering the data to only consist of the top 10th through 50th performance times by age, sex and distance. *Post hoc* Student's t-tests were used to test for differences between boys and girls when a significant interaction of sex was identified. Bonferroni corrected p-values for multiple comparisons ($p < 0.025$) were used for all *post hoc* analyses. Pearson correlation coefficients (r) were used to determine associations between the sex difference in swimming performance and average circulating testosterone concentrations. For all other analyses, significance was

determined at $p < 0.05$. All analyses were performed with IBM Statistical Package for Social Sciences version 25 statistical package (IBM, Armonk, NY, USA) and R version 3.4.2 (Vienna, Austria).

Results

For boys and girls, swimming velocity improved with advancing age according to a quadratic growth curve that was reproducible for each swimming distance (Fig 1). The quadratic growth curve demonstrates rapid improvements in swimming velocity up to 10 years of age after which the age-related improvement in performance slows and approaches a plateau (horizontal asymptote). There were many distinct differences between the age-related performance enhancement curves between girls and boys however. The plateau of swimming velocity was 8.4% lower for girls than boys ($p<0.001$), and the age at which the plateau in performance initiated was younger for girls (15 years) than boys (17 years) for all swimming distances aggregated ($p<0.001$). These data indicate that boys had faster swimming performances than girls particularly at older ages, thus, there was a sex-related difference in swimming performance that increased with age ($p<0.001$).

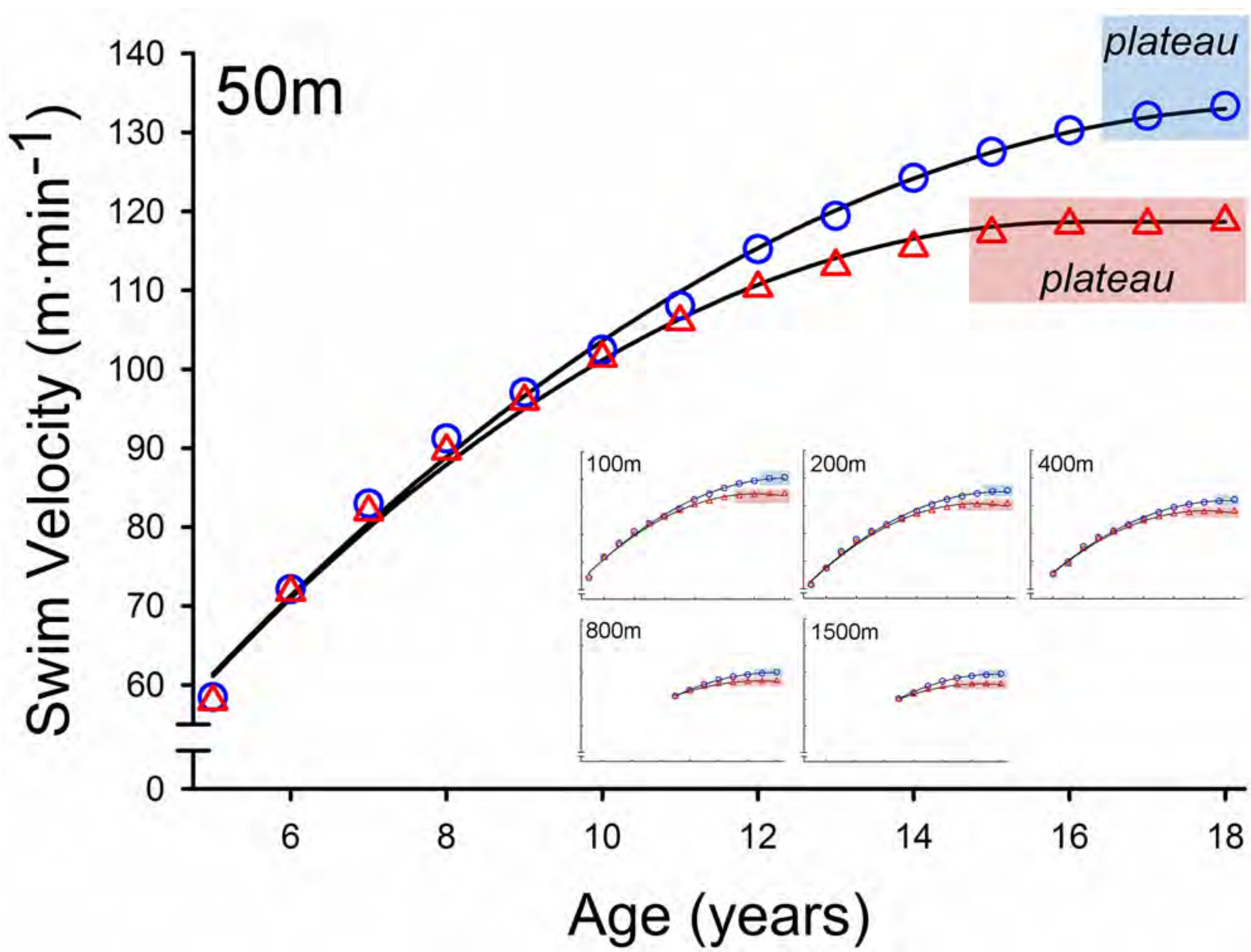

Fig 1. Elite swimming performance. Mean swimming velocity from 5 to 18 years of the top 100 fastest US boys (blue circles) and girls (red triangles) for the 50, 100, 200, 400, 800 and 1500m freestyle swimming distances.

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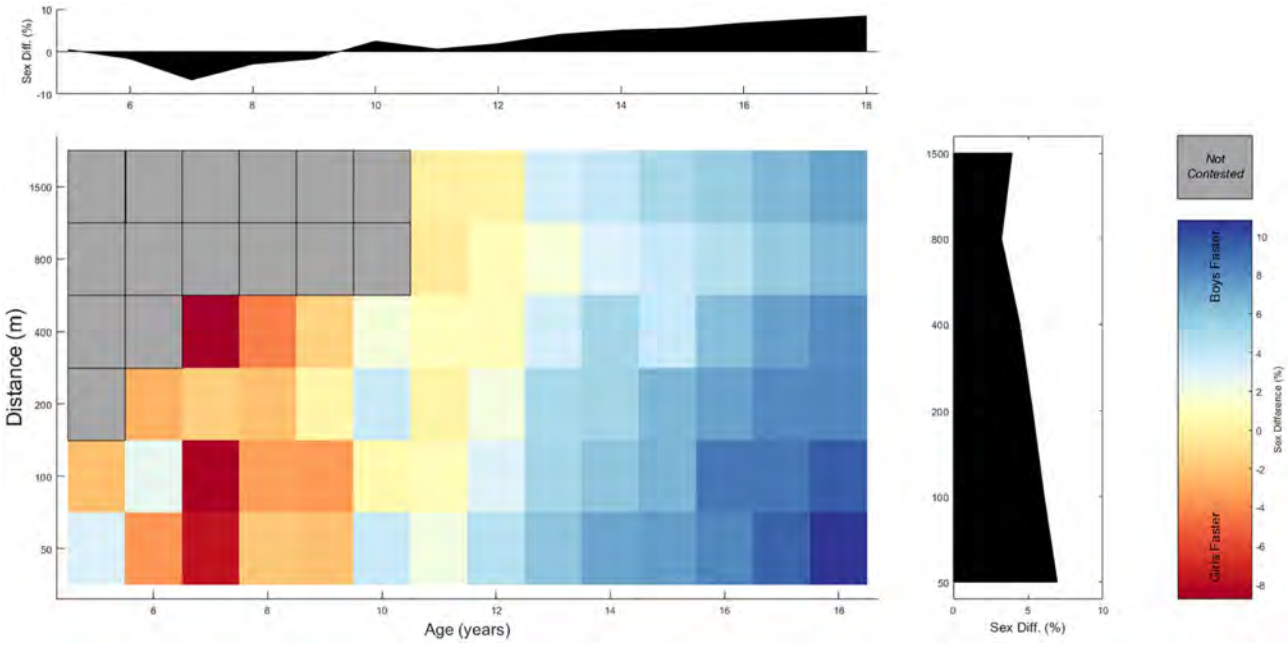

Fig 2. Sex differences in performance of the top 5 places. The primary plot (heat map) displays the sex differences in swimming velocity (% boy's swimming velocity) of the *top 5 US rankings* in each freestyle event distance and age, negative values (red) indicate faster performance of girls. The top displays the mean sex difference across age, and the right plot displays the mean sex difference across swimming event distance.

https://doi.org/10.1371/journal.pone.0225724.g002

Considering the most elite competitors in the top 5 places, girls had 3% faster swimming performance than boys prior to age 10 ($p<0.001$; Fig 2). However, considering the 10th-50th places, there were no sex-related differences in swimming performance between boys and girls prior to age 10 ($p>0.05$; Fig 3D). In the 50m for ages 5–9 years, for example, the average sex-related difference in performance was -2.5% for top 5 (indicating faster performance for girls)

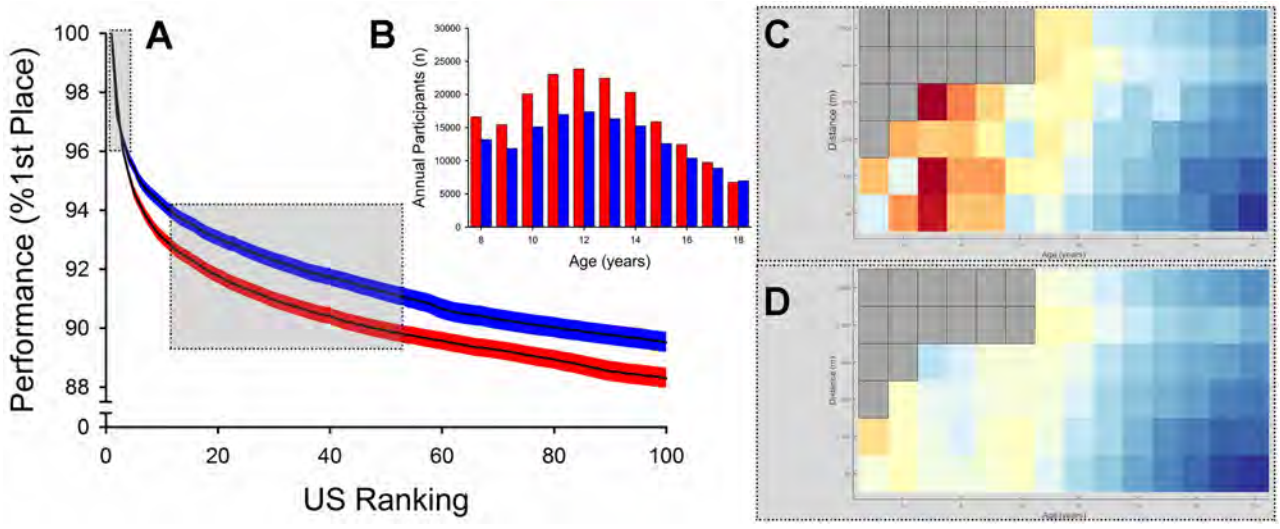

Fig 3. Relative performance decline across US ranking. The decline in swimming performance (% 1st Place) across US ranking for boys (blue) and girls (red; mean ± 95% confidence interval; Panel A). The average annual membership numbers (Panel B) for boys (blue) and girls (red) of USA Swimming. The heat maps (Panels C and D) display the sex differences in swimming velocity of the top 5 US Rankings (Panel C) and the 10th 50th US Rankings (Panel D) using the same color values displayed in Fig 2.

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and 1.2% (indicating faster performance for boys) for the 10th-50th places. Importantly, for both analyses, boys do not exhibit statistically faster swimming velocities than girls at young ages (<10 years). For both the top 5 and the 10th-50th places, pairwise comparisons indicated that the sex difference in performance increased from age 10 (top 5, boys 2.5% faster; 10th-50th places, boys 1.0% faster) incrementally increased for each age until the sex difference plateaued at age 17 (top 5, boys 7.6% faster; 10th-50th places, boys 8.0% faster). Thus, beginning at age 10, boys had faster swimming performance than girls and the sex-difference in performance plateaued at age 17 (Fig 3C & 3D). For top 100 places aggregated, the sex-related difference in performance of 17–18 year olds was larger for the sprint distance events (9.6%; 50-200m) compared with the endurance distance events (7.1%; 400-1500m; $p<0.001$). The larger sex-related differences in performance for sprint distance events were observed for all ages (Fig 2).

Comparison of the relative reductions in velocity between the 1st and 100th place (age groups and event distances aggregated) demonstrated that the girls had greater reductions in relative velocity across place than boys (Fig 3A; sex × place, $p<0.001$). The average 100th place US record holder swam at 90.7 ± 8.8% the velocity of the first place US record holder for the boys and 89.3 ± 9.0% for the girls (age groups and distances pooled). Thus, the sex difference in swimming performance progressively increased with US record place between first place (boys 2.4% faster) to 100th place (boys 4.3% faster) across all ages and distances ($p<0.001$). These data indicate that there was less depth of performance in girls than boys (Fig 3). Despite the lesser depth of performance in girls, annual participation was higher in girls compared to boys ($p<0.001$; Fig 3B).

Circulating testosterone concentration data comprised results from 2,085 measurements. Boys had a greater than 100-fold increase in serum testosterone from ages 6 to 18 (3.6 ± 16.4 to 482.0 ± 232.0 ng·dL^{-1}, $p<0.001$), and this testosterone level began to plateau at 16 years. During the years of low testosterone for boys (<10 ng·dL^{-1})—6 to 10 years—there was no association between testosterone ($p = 0.500$). However, during the years of rapidly increasing testosterone levels for boys—11 to 17 years—mean testosterone was strongly, linearly correlated with the mean sex difference in swimming performance (pooled for all race distances and places; $p<0.001$, $r = 0.990$). See Fig 4.

Discussion

Using 'big data' as a proxy to estimate the ergogenic advantage of androgens in boys compared to girls, we determined the age of the sex-related divergence in elite swimming performance. As expected, boys had faster swimming performance than girls at 18 years in sprint and endurance distances. Participation data provides evidence that there were equal opportunities to participate in swimming between boys and girls (Fig 3B), thus, we propose that the observed mean sex difference in performance across all freestyle events (8.4%) is *solely* due to physiological differences between the sexes. In support of this, the sex difference in world record swimming performances is similar (8.5%) until ~50 years [13]. Between the ages of 11 and 17 years, the sex difference in performance was strongly associated with circulating testosterone concentrations of boys from a nationally-representative sample ($r = 0.990$, $r^2 = 0.980$). These data suggest that endogenous testosterone explains 98% of the variance of the sex difference in performance, and support the previous assertion that the sex difference in circulating testosterone of adults explains most of the sex difference in sporting performance [6].

However, prior to the ergogenic effects of puberty/androgen hormones, there are no sex-related differences in performance for the 10th-50th places and the top 5 girls have faster performances than the top 5 boys. Importantly, the faster performance of the top girls is clearly not due to earlier initiation of puberty because girls are faster at 5 years of age, well before the age

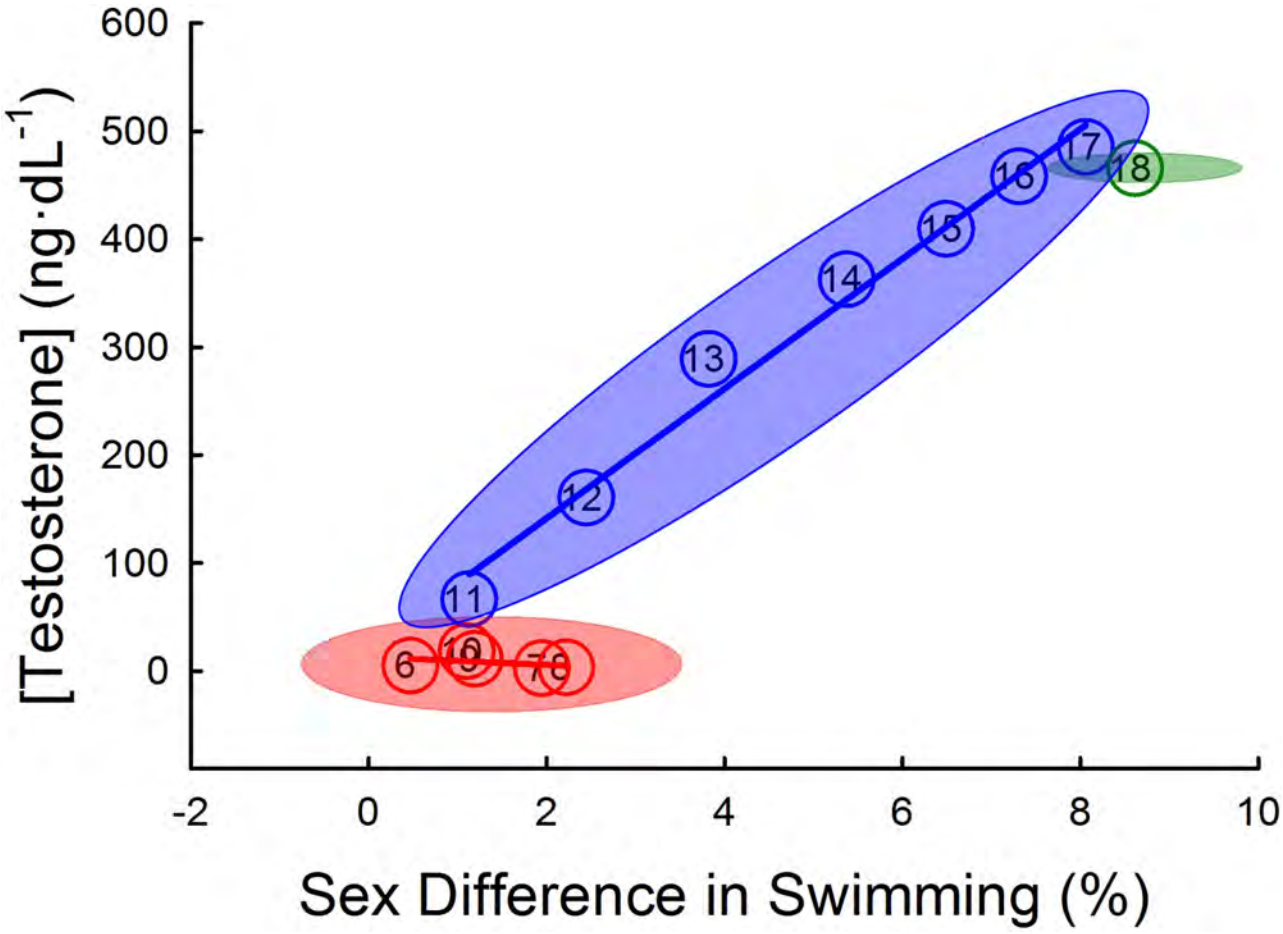

Fig 4. Correlation of boy's serum testosterone and sex differences in swimming performance across age. The mean circulating testosterone concentrations from NHANES database were strongly, linearly correlated with the mean sex difference in swimming performance during the years of rapidly increasing testosterone levels for boys (11 to 17 years; $p<0.001$, $r = 0.990$), but not during the years of low testosterone for boys (6 to 10 years; $p = 0.500$). Each circle represents the mean sex difference in swimming for each age group pooled for all race distances and places (x axis) and mean circulating testosterone level for boys (y axis) with the age group denoted using corresponding Arabic numeral within each circle. The colored ellipses represent the standard error of three separate groupings for the correlation analysis, low testosterone group (6 10 years, red), increasing testosterone group (11 17 years, blue) and plateaued testosterone group (18 years, green).

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of puberty. Although the precise mechanisms are unclear, these data suggest that girls are inherently faster swimmers than boys (or at least not slower) than boys *sans* the performance-enhancing effects of androgens and puberty. As expected before puberty, there are minimal sex differences between boys and girls in stature [23], hand grip strength [5] and hemoglobin content [24]. Thus, if girls are inherently faster than boys prior to puberty, these sex-based differences would likely be due to optimized composition of the genes encoded on the X chromosome (e.g. genes associated with regulation of blood pressure, angiotensin-related enzymes) in girls with two copies of the X chromosome than boys with only 1 copy of the X chromosome [25]. After puberty however, our data and previous data [5, 6] suggest that ~15× greater concentrations of androgens [21] and subsequent physiological changes in boys compared to girls account for the ~8.5% sex-related difference in performance [6].

These data also demonstrate greater participation in swimming for girls than boys, however, this sex-difference in participation narrows with advancing age (Fig 3B). Limited experimental data exist to explain the greater participation among girls, however, the leading

hypothesis is that more girls participate because of longstanding opportunity to participate and compete with (and often outperform) boys. Interestingly, these data also show that despite greater participation for girls than boys, girls have less depth in performance. A prevailing hypothesis (*'sociocultural conditions hypothesis'* [17]) suggests that decreased opportunities and participation contribute to sex differences in sports performance. Indeed, in a previous study examining collegiate rowing, a sport sanctioned by the US National Collegiate Athletic Association (NCAA) for women but not men, greater participation for women was associated with greater depth of performance for women in the heavyweight class [17]. Thus it is unclear why in this study; girls have greater participation and less depth of performance. However it is clear that these data provide one of the only examples of faster (or at least not slower) sports performance for girls than boys.

Conclusion

We conclude that prior to the performance-enhancing effects of puberty; the best girls outperform the best boys at sprint and endurance swimming events. Our findings are in direct opposition to nearly universal findings in elite adult athletes that boys are faster than girls. These data provide evidence that the Y chromosome *per se* does not provide an advantage in sports performance. Rather, our data are consistent with 'doping' ideology and findings that sustained and augmented levels of endogenous androgens induce performance-enhancing adaptations regardless of genotype of the sex chromosomes. This information may be of use to governing bodies of athletic competitions as eligibility regulations for participation in female events are refined.

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Exhibit 21

Race Times for Transgender Athletes

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Race Times for Transgender Athletes

Joanna Harper, Providence Portland Medical Center, USA

Abstract *In recent years, organizations such as the International Olympic Committee have created regulations to allow those athletes who have undergone gender reassignment to compete in their chosen gender. Despite these rules, there is still a widespread belief that transgender female athletes have an inherent advantage over 46,XX female competitors. Until now, there has not been any published data, based on performances of transgender athletes, to either support or refute this belief. There are two main stumbling blocks to creating such a study the first is to determine an appropriate metric to examine and the second is to find participants for the study. This study analyzed race times for eight transgender female runners, who have competed in distance races as both male and female, using a mathematical model called age grading. Collectively, the age graded scores for these eight runners are the same in both genders.*

Keywords *Transgender, Athletes, Distance Running, Gender, Research*

Introduction

Athletes have historically been divided into male and female for the purpose of most sporting competitions. Two components of biological sex, first external genitalia, and later chromosomes were used to make the determination of who was allowed to compete in women's sport. Chromosome testing was initiated for the 1968 Olympics (Elsas et al 2000, 249-254) and thereafter, only those people with XX sex chromosomes among their 23 chromosome pairs, or 46,XX females, were allowed into women's sports. Human biology, however, does not neatly divide into two categories. For instance, some people have neither a 46,XY nor a 46,XX karyotype. Additionally, some people are born with a 46,XY pattern, but with mutations which cause them to be assigned female gender at birth. Chromosome based requirements for participation in female athletics were discontinued in the 1990s (Elsas et al, 249-254), but controversy surrounding athletes with karyotypes other than 46,XX competing in women's sport continues. (Karkazis et al 2012, 3-16).

Transgender people are those whose innate sense of gender, or gender identity, does not match their biological sex. Some transgender people seek gender reassignment. Such people have been termed transsexual, and although the term is descriptive, it is now often viewed unfavorably within the transgender community. While transgender surgery can alter external and internal genitalia, and hormone therapy changes many secondary sex characteristics, neither can alter karyotype; hence it is questionable whether one could claim a change in sex as a result of any intervention. Unambiguous reassignment of gender is, however, possible.

Those who are satisfied with the gender assigned to them at birth can be described as cisgender.

Transgender athletes have sought to compete against other athletes on the basis of their reassigned gender, rather than on their biological sex. While there has been little resistance to the presence of transgender male athletes, sporting organizations were unwilling to allow transgender women to compete against 46,XX women prior to the 21st century. It is notable that in the 1970s, Rene Richards, probably the best-known transgender athlete in history, sued in the United States court system in order to be allowed to play women's tennis (Abrams 2010).

In 2004, the International Olympic Committee (IOC) enacted the Stockholm Consensus (Ljungqvist et al., 2003), that allows transgender women to compete in women's sport once a) gender reassignment surgery had been completed, b) the athlete was legally recognized as female, and c) they had undergone two years of hormone replacement therapy. Transgender men were permitted to compete against cisgender men, although transgender men must file a therapeutic use exception (TUE) form to cover their use of testosterone injections.

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At the time of the Stockholm Consensus, there was no published scientific literature that would justify the inclusion of transgender women. The committee that created the Stockholm consensus relied heavily on information from Dr. Louis Gooren from Amsterdam (Ljungqvist 2104). Dr. Gooren was an expert in transgender studies and would go on to co-author an important paper which studied nineteen transgender women after commencement of hormone therapy (Gooren and Bunck, 2004, 425-429). After one year of testosterone suppression, the subjects had testosterone levels below those of 46,XX women, and hemoglobin levels equal to those of 46,XX women (red blood cell content is very important in endurance sports). Muscle mass differences between the two groups were cut in half. The height of the individuals did not change. There were no additional changes noted at three years. This study was not undertaken on athletes, nor did the researchers directly measure any physical component of athleticism, such as strength, speed, explosiveness, or endurance. The authors concluded that it was reasonable to allow transgender women to compete against cisgender women after appropriate hormone therapy.

It is notable that the Stockholm consensus required two years of hormone therapy, while the published study noted that there were no physical changes in the subjects after one year. This discrepancy was due to conservative estimates given to the committee by Dr Gooren prior to the publication of his study (Ljungqvist 2014).

Many sports followed the lead of the IOC, and in subsequent years there have been transgender women competing in sports such as golf (Mianne Bagger and Lana Lawless), cycling (Natalie Van Gogh, Michelle Dumaresq, and Kristin Worley), martial arts (Nong Toom, and Fallon Fox), and basketball (Gabrielle Ludwig). None of these women has been particularly successful at the highest levels of sport after gender reassignment, and one could argue that this lack of success over ten years would be a strong indication of the fairness of permitting transgender women to compete against cisgender women.

Instead of acceptance, however, there has been a substantial amount of controversy over the presence of transgender women in female athletics. Most people contend that transgender women have an unfair advantage in women's competition (Cavanagh and Sykes, 2006). Opponents of transgender inclusion often point to physical characteristics such as height and hand size, which are not changed by gender reassignment, and suggest that transgender women will always maintain an unfair advantage over cisgender women. These arguments continue today and are not confined to competition at the highest levels. Recently, there were 10,000 emails sent in to protest the decision by the State of Minnesota to allow high school transgender athletes to compete in their chosen gender (Minnesota Star Tribune 2014).

Those in favor of allowing transgender athletic participation inevitably point to the fact that every major sporting organization to look at the issue since 2004 has agreed to allow transgender women to compete against other women. Proponents also will often suggest that science is on their side. However, the only existing published study related to transgender women in sport is the original one by Gooren and Bunk. The science supporting transgender inclusion is very thin indeed.

A thorough literature review of studies applicable to transgender athletes was undertaken for the Canadian Government (Devries, 2008). This review found that "To date no study has conducted any sort of exercise test to assess athletic performance" and concluded that there were no data indicating any sporting advantage or disadvantage for transgender women as compared to over 46,XX women.

The lack of such a study should not come as a surprise. There are two major obstacles involved in compiling any study involving transgender athletes. The first problem is how to formulate a study to create a meaningful measurement of athletic performance, both before and after testosterone suppression. No methodology has been previously devised to make meaningful measurements.

The second problem is to find study participants. There are few transgender athletes, and even fewer who will want to be identified. In order to create a study, a small cohort of competitive transgender athletes must be found in one given sport. Fortunately, there is mass participation in distance running races throughout much of world. All major cities hold road races with many thousands of runners, giving the sport a large base of adult competitors. Thus, the sport of distance running is an obvious choice to try to find suitable candidates.

In 2011, the international governing body for track and field, the IAAF, amended its rules to allow anyone who was legally and hormonally female to compete in the women's category (IAAF, 2011). The portion of the ruling applicable to transgender women lists no requirement for surgical intervention, or specific duration of hormone therapy. It does require an endocrine evaluation prior to any declaration of eligibility. In many parts of the world, legal gender reassignment is not allowed, and this will be a barrier to participation for many transgender athletes.

In 2012, the IOC also adopted a testosterone-based rule for eligibility for women's sport (IOC, 2012); however, the IOC maintained their previous rules pertaining to transgender women. Hence, it would be possible for a transgender woman to compete against other women in the IAAF sponsored 2015 world track championships, but not be eligible to do so in the IOC-sponsored 2016 Olympics.

Methods

Race times from eight transgender women runners were collected over a period of seven years and, when possible, verified. The collection process consisted of seeking out female transgender distance runners, mostly online, and then asking them to submit race times. Even in 2014 few people are open about being transgender, so the submission of race times represented a large leap of faith for the participants. When possible, race times were then verified using online services listing race results. For six of the eight runners, online checking made it possible to verify approximately half of the submitted times. Two of the subjects, runners three and four, would only participate anonymously, creating an ethical dilemma over the use of their times, versus respect their privacy.

Seven of the eight subjects experienced a substantial reduction in running speed upon transition. There are a few methods of comparing men's and women's race times. The simplest involves the well-known approximation that men will, on average, run 10% faster than women (Berman et al. 2013 63–65). There are a couple of other comparison methods as well, but there is only one method that also factors in age. Correcting for age is important because most of the runners in the study were more than 30 years old, and would be faced with declining performance as they grew older, following their gender transition.

Age grading (Grubb, 1998, 509-521) is a method of comparing the performance of athletes of all ages and both sexes. For running events, the athlete's race time (RT) is compared to the fastest time ever run by a person of that age and sex, or the age standard (AS). The resultant age grade (AG) percentage is obtained by the following formula:

$$AG\ (\%) = (AS \times 100) / RT$$

All times are measured in seconds.

In order to understand how age grading works, let's examine two forty-year-old runners who run a 5-kilometer race (5k). The male runner runs 19:30 (1170 seconds). In order to determine his age grade, one compares his time to the fastest time ever run by a forty-year-old male 5k runner, i.e. 13:39 (819 seconds). The equation becomes

$$AG = (819 \text{ seconds} \times 100)/1170 \text{ seconds} = 70$$

and our male runner gets a score of 70.

The female runner has a time of 21:51 (1311 seconds) and her time is compared to the fastest ever time by a forty-year-old woman, i.e. 15:18 (918 seconds). The equation for her AG is

AG = (918 seconds x 100) /1311 seconds = 70

Thus, our male and female runners score the same age grade despite the fact that the male ran more than two minutes faster than the female did. This is fair. Men run faster than women. The two runners are both well above average runners for their age and sex, and deserve to receive equal accolades.

Age grading has become the standard way of comparing performances by older track and field athletes of both sexes. The method has also been rigorously evaluated and improved, specifically with regard to the curve fitting that is needed to connect the age standards associated with different ages. Mathematician Alan Jones (Jones 2010) has made significant improvements to the age-graded tables that Howard Grubb developed in the 1990s.

Results

Collectively, the eight runners had much slower race times in the female gender than as males. Time differences were, in fact, so great, that age graded performances stayed virtually constant for the group. Tables one through four summarize the data from all eight runners over four frequently run race distances varying from 5k to the marathon (42 kilometers). Not all eight women submitted times for all four of these distances.

Table 1: 5k Race Times

	Male	Races		Female	Races	
Runner No.	**Age**	**Time**	**AG**	**Age**	**Time**	**AG**
One	48	18:27	78.7	52	22:43	75.7
Two	30	15:56	81.4	36	17:51	82
Four (a)	30	17:35	73.6	33	21:04	70.6
Five	34	19:39	66.7	35	23:43	63
Six (b)	24	15:07	83.5	53	20:22	85.5
Eight	27	20:29	62.2	30	22:51	64.8

Table 2: 10k Race Times

	Male	Races		Female	Races	
Runner No.	**Age**	**Time**	**AG**	**Age**	**Time**	**AG**
One	49	0:39:05	77.9	56	0:48:45	76.1
Two (b)	22	0:32:37	82.4	36	0:36:58	83.1
Five	34	0:45:33	60.1	36	0:57:40	53.3
Six (a)	46	0:37:10	80	48	0:42:01	80.5

Table 3: Half-marathon Race Times

	Male	Races		Female	Races	
Runner No.	**Age**	**Time**	**AG**	**Age**	**Time**	**AG**
Five	33	1:53:06	52.4	37	2:05:38	53.3
Six (b) (d)	26	1:08:38	86.3	53	1:32:27	83.8
Six (a) (d)	46	1:23:11	77.8	48	1:34:01	77.5
Seven (c)	19	1:48:47	55.7	28	1:48:45	60.5

Table 4: Marathon Race Times

	Male	Races		Female	Races	
Runner No.	**Age**	**Time**	**AG**	**Age**	**Time**	**AG**
Three	49	3:18:58	69.5	54	4:12:31	67.2
Five	34	3:16:59	63.4	35	4:08:33	55.3
Seven (c)	19	3:49:55	55.7	31	2:59:10	75.7
Eight	29	3:08:53	66.1	30	3:44:55	60.2

Notes

(a) These races were run over the same course within three years' time and represent the best comparison points.

(b) Races compared over a long time period have more uncertainty associated with them, but both runner two and runner six reported stable training patterns over this time range. These races also help to confirm the age-grading methodology for tracking progress of a runner over the course of a multi-year time frame.

(c) Runner seven represented the biggest evaluation challenge. She raced as a 19 year-old male recreational runner and then resumed running years later as a female. She got serious about the sport after she resumed, doubled her training load and dropped 10 kg of weight. Not surprisingly, she got faster. This improvement can be seen in the fact that her AG went from 60.5 at age 28 to 75.7 at age 31 (both in female gender). This 15 point change in age grade was much larger than the 5-point change she experienced after transition from male to female.

(d) It is useful to compare times for the same runner over different race courses and at different time periods. The two lower scores occurred on a hilly course at a period of average fitness for runner six. The two higher scores were on flat courses at times of peak fitness.

Table five indicates the average AGs from all eight runners in each gender and the overall averages of all eight.

Table six shows the highest AGs from each runner and the average of these highest AGs. Two tailed t tests were run on both the mean and peak AGs. The p values were p=0.84 for the average AGs and p=0.68 for the highest AG. A p value of less than 0.05 is needed for the values to be considered significantly different, and these p values are very much higher.

Table 5: Average Age Grades

	Average male AG	Average female AG
Runner 1	75.2	77.1
Runner 2	81.8	82.8
Runner 3	69.5	70.8
Runner 4	71.4	64.8
Runner 5	57.7	49.3
Runner 6	83.8	81.9
Runner 7	55.7	61.9 (e)
Runner 8	54.3	59.1
Average	68.7	68.5

Table 6: Highest Age Grade

	Highest male AG	Highest female AG
Runner 1	78.7	79.2
Runner 2	82.9	83.2
Runner 3	69.5	74.3
Runner 4	74.1	74.1
Runner 5	66.7	63.0
Runner 6	87.5	85.6
Runner 7	55.7	63.4 (e)
Runner 8	66.1	64.8
Average	72.7	73.4

(e) The 2:59 marathon time by runner seven was considered an outlier, the result of her substantially altered training and was not used in these tables.

Discussion

The majority of scientists believe that testosterone is primarily responsible for the difference in athletic results between the sexes (Bermon et al. 2014, 4328–4335), although there are dissenters (Healy et al. 2014, 294-303). There have been multiple studies on men's and women's testosterone levels with some variation in results, but a typical set of values would be as follows: Men's range — 10 to 35 nmol/l; female range — 0.35 to 2.0 nmol/l (Haring et al. 2012, 408–415).

Transgender women who have undertaken testosterone suppression change from normal male testosterone levels to normal female levels, in fact, after surgery their testosterone levels are below the mean for 46,XX women (Gooren and Bunck, 425–429). Largely as a result of their vastly reduced testosterone levels, transgender women lose strength, speed, and virtually every other component of athletic ability.

Since this study looks at endurance capabilities of athletes both pre and post testosterone suppression, it is also of significant interest to look at hematocrit or hemoglobin levels of transgender women. One year after testosterone suppression, hemoglobin levels in transgender women fell from 9.3 mmol/l to 8.0 mmol/l. This latter number is statistically identical to the mean hemoglobin level for cisgender women (Gooren and Bunck 425–429).

The reduction of testosterone and hemoglobin levels of transgender women after transition would suggest that endurance capabilities of transgender women athletes should be similar to those of 46,XX women.

The difficulty of finding suitable subjects is underscored by the fact that it took seven years to amass data from eight participants.

The times submitted by the eight runners were self-selected and self-reported. The self-reporting by the subjects certainly affects the strength of the findings. As mentioned previously, almost half of the race times were double checked by the author for accuracy. None of the subjects incorrectly reported any result.

Collectively, the eight runners were much slower in the female gender; slow enough, in fact, that their age graded performances were almost identical to their male AGs. Two of the runners had higher average AGs in male gender than in female gender, while one runner had higher female AGs than male ones. The changes in the age grades of these runners mirrored changes in their training habits.

After transition, runner four began to experience a significant number of injuries which prevented her from training as rigorously as she previously had. It is not surprising that her results got worse as time went on. Runner five experienced both weight gain and a loss

motivation in the years after her transition. In fact her motivation declined to the point that she gave up racing not long after the submission of her results.

On the other hand, runner seven blossomed as a runner after transition. Eventually, she doubled her weekly training distance. She also lost approximately 10 kg of body mass after she started to train harder. It is not surprising that her times and age grade scores showed a subsequent improvement.

The other runners in the study reported relatively stable training loads in both male and female mode, and this is reflected in their more stable age grades in both genders.

Since training loads vary over time for all runners, the author believes that highest age grade might be the best comparison of male versus female athletic potential. But, whether one uses average or highest age grades, the subjects scored statistically identical age grades both as male and as female.

It is significant to note that none of the eight subjects was a truly elite runner. An optimal study would use world-class runners and the results could be used to justify the presence of transgender women in events such as the Olympic Games. Unfortunately, there simply are no world-class transgender distance runners. Three of the eight runners have achieved notable success at the national level, and two of the other runners could be described as sub-elite. Resistance to the presence of transgender women occurs at all levels of sport, and so there is still much merit to the study.

One interesting trend was noted in runners five, six and eight, who age graded higher in shorter events as women than they did in longer events. Runners six and eight scored higher age grades in 5k races than they did as males but lower AGs in longer races – half marathon and up. Runner five scored lower across the board as female than as male but her 5k AGs were much closer to her male ones, than her marathon AGs were. Transgender women carry more muscle mass than 46,XX women (Gooren and Bunck 2004, 425–429). This extra muscle mass might cause increased speed when compared to cisgender women, and hence faster times and higher AGs at shorter distances. Increased muscle mass and heavier bones are not conducive to long distance running, and would actually be a disadvantage when running distances of a half marathon and higher, causing slower times and lower AGs. This effect is small in the three mentioned runners, and none of the other five runners submitted data over a wide enough range of distances to determine whether or not this pattern held true for them; more research would be needed to confirm or refute the hypothesis of distance related variations in age grade scores for transgender women.

It should be noted that these results are only valid for distance running. Transgender women are taller and larger, on average, than 46,XX women (Gooren and Bunck, 2004, 425-429), and these differences probably would result in performance advantages in events in which height and strength are obvious precursors to success - events such as the shot put and the high jump. Conversely, transgender women will probably have a notable disadvantage in sports such as gymnastics, where greater size is an impediment to optimal performance.

The Grubb and Jones age-grading methodology applies only to track-and-field and distance running, but, it should be possible to create a similar analytic method to compare results for other sports, such as swimming, weightlifting, or ski-jumping, which also measure results in times, distances or weights – the so called CGS (centimeter, grams, and seconds) sports. It would be very difficult, however, to devise such a method to analyze performances in most other types of sports.

Conclusions

Despite the fact that transgender women have been allowed to compete against cisgender ones since 2004, there has been no study used to justify this decision beyond the original work of Gooren and Bunck. It bears repeating that this original study was not undertaken on athletes, nor

did it directly measure any aspect of athleticism. In fact, this is the first time a study has been developed to measure the performance of transgender athletes. The author overcame two significant barriers which have prevented any previous study from being performed, i.e. the difficulty in determining an appropriate metric to measure athletic performance both before and after testosterone suppression, and the difficulty in finding enough willing study participants in any given sport.

The author chose to use the standard age-grading methodology which is commonly used in master's (over forty) track meets worldwide, to evaluate the performance of eight distance runners who had undergone gender transition from male to female. As a group, the eight study participants had remarkably similar age grade scores in both male and female gender, making it possible to state that transgender women run distance races at approximately the same level, for their respective gender, both before and after gender transition.

It should be noted that this conclusion only applies to distance running and the author makes no claims as to the equality of performances, pre and post gender transition, in any other sport. As such, the study cannot, unequivocally, state that it is fair to allow to transgender women to compete against 46,XX women in all sports, although the study does make a powerful statement in favor of such a position.

It should also probably be noted that the publication of this study will likely not appreciably change the resistance faced by transgender women who compete against cisgender ones. There will continue to be strong opposition by athletes, parents and fans to the inclusion of transgender women. It will take many more years before the average sports enthusiast understands that transgender women who have undergone testosterone suppression will not dominate women's sports.

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Journal of Sporting Culture and Identities is one of the four thematically focused journals that support the Sport and Society knowledge community—its journal collection, book series, and online community.

The journal focuses on sport's motivations, meanings and purposes. It publishes articles that examine the psychology of sport and the interplay between individual identities and access, equity, and participation in sports.

Journal of Sporting Culture and Identities is a peer-reviewed scholarly journal.

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IN THE UNITED STATES COURT OF APPEALS
FOR THE NINTH CIRCUIT

HELEN DOE, parent and next friend of Jane Doe; et al.,

Plaintiffs-Appellees,

v.

THOMAS C. HORNE, in his official capacity as State Superintendent of Public Instruction; et al.,

Defendants-Appellants,

and

WARREN PETERSEN, Senator, President of the Arizona State Senate; BEN TOMA, Representative, Speaker of the Arizona House of Representatives,

Intervenor-Defendants-Appellants.

On Appeal from the United States District Court
for the District of Arizona

EXHIBITS TO INTERVENOR-DEFENDANTS-APPELLANTS' EMERGENCY MOTION UNDER CIRCUIT RULE 27-3 FOR A STAY PENDING APPEAL

VOLUME 2

Exhibit 22

https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328

Marnee McKay

9 min read

When it comes to sport, boys 'play like a girl'

Primary school-aged boys and girls can play in mixed teams until they reach high school, our research suggests. Clappstar/Flickr, CC BY-SA

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affiliations beyond their academic appointment.

Partners

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Girls in primary school are just as physically capable as their male classmates, according to our research, taking the sting out of the insult “you play like a girl”.

When we compared primary school children’s physical capabilities, differences between girls and boys were not as important as people think. So, they should be happily playing with and competing against each other in the backyard, playground and sporting fields.

Read more:* *It's not just the toy aisles that teach children about gender stereotypes

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As part of wider research to assess people’s physical capabilities across the lifespan, we tested 300 children and adolescents between the ages of 3 and 19.

We tested each child for over two hours, taking more than 100 measurements. These included measuring the strength of 14 muscle groups, the flexibility of 13 joints and 10 different types of balance. We looked at factors including hand dexterity, reaction times, how far kids could walk, how high and how long they could jump, as well as their gait.

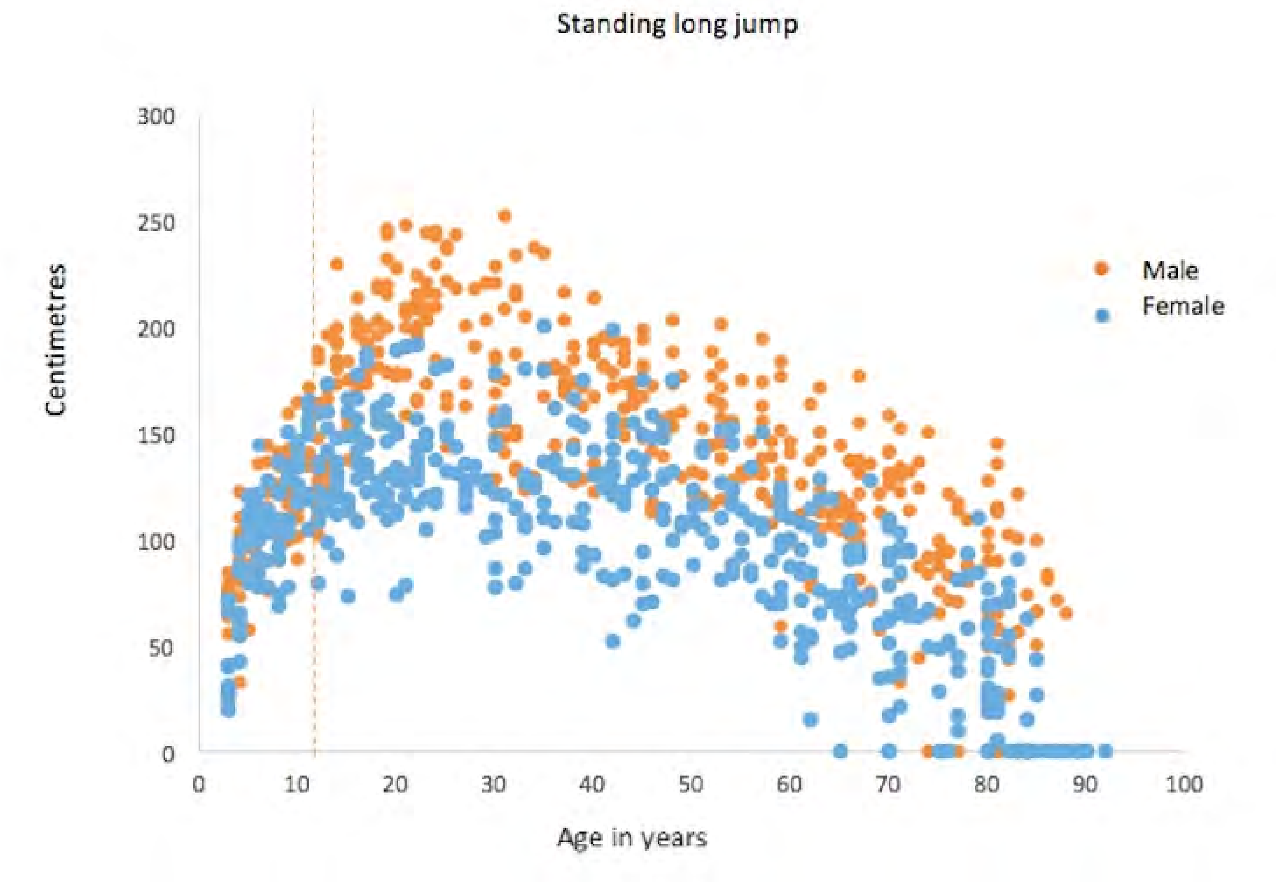
What did we find?

Across all measures of physical performance, there was one consistent finding. There was no statistical difference in the capabilities of girls and boys until high-school age (commonly age 12).

Let's use standing long jump (also known as a broad jump test) as an example. This provides a measure of your legs' explosive power. It needs minimal equipment and the results compare well with the type of information you get from strength testing using expensive equipment. It's also one of the tests would-be American NFL (National Football League) players take to impress talent scouts.

The standing long jump is a test football scouts use to assess explosive power.

We found no difference between boys and girls before they turn 12 (see graph below). Every physical measure followed this pattern.



Before the age of 12, boys and girls do just as well as each other in the standing long jump. Author provided

How do our findings compare?

Other studies have had similar results. These have included ones testing muscle strength, walking, jumping and balancing.

However, it’s difficult to directly compare data from one study to another, as different studies have different sample sizes, include children of different age ranges, and assess different measures. For example, we were the first to use the timed stairs test and stepping reaction time to find what regular children were capable of.

Some studies found differences in physical capabilities between primary school-aged boys and girls using the same types of tests we used. And others reported small differences in the jump height of boys and girls aged 6-17 years but not with the long jump.

These differences can in part be attributed to sampling methods that were limited to specific age ranges or locations and socioeconomic backgrounds, the latter potentially having a significant impact on physical health and activity.

By contrast, the children in our research were generally representative of the Australian population, using data from the Australian Bureau of Statistics about socioeconomic status, ethnicity and body mass index.

What do our findings mean for kids, coaches and parents?

There is no consensus across schools or among different sports about mixed-gender sports for primary school children.

For instance, boys and girls compete separately in most local Little Athletics after age five but field hockey can have mixed gender teams until age 17.

And in tennis, primary school-aged girls and boys play separately in singles matches but can play against each other in mixed doubles.

Our findings support the push for boys and girls to compete in mixed sporting teams until the end of primary school, after which the hormonal changes of puberty mean boys tend to perform better in sports and tasks requiring strength and speed.

Read more:* *Our 'sporting nation' is a myth, so how do we get youngsters back on the field?

There are also some practical advantages to mixed sport in primary school and in weekend competitions:

- fewer scheduling conflicts for councils (allowing school and sport administrations to fit games more conveniently into busy sporting venues)

- fewer clubs or organisations to share already stretched government and private sector funding
- consolidation of coaching and manager talent, and most importantly
- fewer parent-taxi drop offs.

Perhaps perceived differences in physical capability between boys and girls are based on outdated gender stereotypes that appear at birth, when some boys are given their first footy and some girls their first doll.

But whatever the origin of the idea young boys are physically more capable than young girls, the evidence is clear. Boys “play like a girl”, and that’s certainly no insult.

Exhibit 23

Normative reference values for strength and flexibility of 1,000 children and adults

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ABSTRACT

Objective: To establish reference values for isometric strength of 12 muscle groups and flexibility of 13 joint movements in 1,000 children and adults and investigate the influence of demographic and anthropometric factors.

Methods: A standardized reliable protocol of hand-held and fixed dynamometry for isometric strength of ankle, knee, hip, elbow, and shoulder musculature as well as goniometry for flexibility of the ankle, knee, hip, elbow, shoulder, and cervical spine was performed in an observational study investigating 1,000 healthy male and female participants aged 3–101 years. Correlation and multiple regression analyses were performed to identify factors independently associated with strength and flexibility of children, adolescents, adults, and older adults.

Results: Normative reference values of 25 strength and flexibility measures were generated. Strong linear correlations between age and strength were identified in the first 2 decades of life. Muscle strength significantly decreased with age in older adults. Regression modeling identified increasing height as the most significant predictor of strength in children, higher body mass in adolescents, and male sex in adults and older adults. Joint flexibility gradually decreased with age, with little sex difference. Waist circumference was a significant predictor of variability in joint flexibility in adolescents, adults, and older adults.

Conclusions: Reference values and associated age- and sex-stratified *z* scores generated from this study can be used to determine the presence and extent of impairments associated with neuromuscular and other neurologic disorders, monitor disease progression over time in natural history studies, and evaluate the effect of new treatments in clinical trials. **Neurology® 2017;88:36–43**

Meaningful, reliable, and sensitive outcome measures are required to monitor treatment and progression of neuromuscular and other neurologic disorders. While there have been substantial advances in the understanding of the pathogenesis and natural history of many neuromuscular disorders, the identification and development of new outcome measures that best reflect the efficacy of specific treatments have not advanced at the same rate.[1] Establishing valid and responsive outcome measures is a priority for the field.[2] To assist in the development of new outcome measures, normative reference values generated from large populations across the lifespan using standardized methods are required. Normative reference values can be utilized to generate *z* scores, which can be used in multicenter studies to improve outcome measure precision and responsiveness.

Muscle weakness and joint contractures predispose to numerous pathologies requiring intervention. Reference data play an important role in identifying and quantifying these impairments and evaluating the effectiveness of interventions. Currently, few comprehensive datasets detail the normal variation of active range of motion in healthy individuals and are limited by the number of joints assessed,[3] the age range of participants,[4,5] or insufficient sex representation.[6,7]

Supplemental data at Neurology.org

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Coinvestigators are listed at Neurology.org.

Go to Neurology.org for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

Similar limitations exist in strength reference datasets, relevant only to children[8–11] or adult populations,[12–16] or strength measured using equipment not readily available in clinic.[17–19]

The purpose of this study was to generate a reference dataset of normative values across the lifespan for an extensive set of isometric muscle strength and joint flexibility items, stratified for age and sex, and to investigate the influence of demographic and anthropometric factors.

METHODS

Study design and participants. Data were collected as part of the 1,000 Norms Project, an observational study investigating physical function and self-reported health in 1,000 people across the lifespan (see full protocol[20]). One thousand people aged between 3 and 101 years from the Greater Sydney metropolitan area in Australia participated in the project. Participants were recruited from January 2014 to September 2015 using highly structured convenience and snowball sampling techniques, including advertising via social media, e-newsletters, and community flyers. Presentations were held at social and volunteer groups, aged care organizations, playgroups, and schools. Eligible participants were aged ≥3 years, considered themselves healthy for their age, and could participate in age-appropriate activities of daily living. People with significant health conditions affecting physical performance or an inability to follow age-appropriate instructions were excluded. Potential participants with the following conditions were also excluded: diagnosed diabetes mellitus; malignant cancers; demyelinating, inflammatory, or degenerative neurologic conditions; pregnancy; class 3 obesity; severe cardiac or pulmonary disease; joint replacement; infectious or inflammatory arthropathies; or severe mobility impairment necessitating dependence on mobility aids for all ambulation. Equal numbers of male and female participants were recruited and were stratified into 9 age categories. One hundred people per decade were recruited in the age groups of 20–29, 30–39, 40–49, 50–59, 60–69, 70–79, and 80+ years. In order to represent the rapid periods of growth and maturation and to distinguish between young children and adolescents, 20 children per year from 3 to 9 years of age and 16 per year from 10 to 19 years of age were recruited.

Standard protocol approvals, registrations, and patient consents. Ethical approval was granted by the institutional Human Research Ethics Committee (HREC 2013/640) and written informed consent was obtained from all participants or parents/guardians of children.

Procedure. Participants attended the University of Sydney Performance Laboratory once for a 2-hour assessment. Participants had their height, body mass, waist circumference, and lower limb alignment measured. Foot structure was assessed using the Foot Posture Index, a 6-item summed scale from −12 (supinated) to +12 (pronated).[21] Age, sex, current work status, and self-reported ethnicity were collected from all participants or parents/guardians. Work status was classified as working (full-time, part-time, or unpaid) or not currently working (unemployed, student, or retired). Ethnicity was classified into 5 categories: British/European, American, Asian, African, and Aboriginal/Torres Strait Islander.

Two experienced clinical evaluators (physiotherapists) assessed isometric strength and joint flexibility using standardized methodology, including instructions, positioning, and scoring.[20] The dominant limb was assessed and determined as the hand used to write with and the foot used to kick a ball. The strength of 12 muscle groups—hand grip, ankle dorsiflexors and plantarflexors, knee flexors and extensors, hip abductors, internal and external rotators, elbow flexors and extensors, and shoulder internal and external rotators—were assessed by maximal voluntary isometric contraction using a portable hand-held dynamometer (Citec dynamometer CT 3001; CIT Technics, Groningen, Netherlands). The dynamometer was calibrated 0–500 N with certified weights monthly throughout data collection. The strength of knee musculature in participants ≥12 years of age was assessed by fixed dynamometry (CSMi; HUMAC NORM, Stoughton, MA). For unit of measure consistency, knee flexor and extensor strength in children aged 3–11 years were converted to Newton-meters (Nm) using anthropometric tables.[22] Rather than using the fixation device outlined in the protocol, ankle plantarflexion strength was assessed using hand-held dynamometry in long sitting, heel over plinth edge.

Joint flexibility was assessed using a universal goniometer, digital inclinometer, or bubble inclinometer (Baseline; Fabrication Enterprises Inc., White Plains, NY) depending on the joint assessed. Thirteen active joint range movements were assessed: ankle dorsiflexion and plantarflexion, knee flexion and extension, hip flexion, internal and external rotation, elbow flexion and extension, shoulder internal and external rotation, and cervical flexion and extension. Interrater reliability of the clinical evaluators demonstrated satisfactory repeatability of all strength and flexibility measures (intraclass correlation coefficient$_{2,1}$ 0.80–0.99) in a pilot study of 10 participants aged 6–67 years.

Data analysis. Data were collected and managed using REDCap electronic data capture and manually checked for transcription errors. Reference values were generated for each age group and sex in SPSS v22 Statistics for Windows (IBM SPSS; Armonk, NY). Normality of the data was assessed using Kolmogorov-Smirnov test. For analysis, age categories were children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years). To determine if strength and flexibility differed between male and female participants, independent t tests were conducted. A series of multiple regression models was constructed to determine the extent to which muscle strength and joint flexibility were influenced by participant demographic (age and sex) and anthropometric factors (height, body mass, waist circumference, foot posture, and lower limb alignment). First, Pearson product-moment correlation coefficients (r) were generated to explore the bivariate relationships between strength and anthropometric and demographic factors. The same correlations were explored for each joint flexibility measure. Second, factors identified to have an association ($r \geq 0.3$, $p < 0.05$) with strength or joint flexibility were entered simultaneously into a stepwise multiple regression model, which was reduced to a set of factors that best predicted and could be regarded as independent determinants of each strength and joint flexibility measure. To avoid multicollinearity, only one variable from highly correlated ($r \geq 0.7$) variables was included. Standardized β weights were calculated to provide an indication of the relative importance of the contribution of the various factors entered into the model to explain the variance in joint flexibility

Table 1 Isometric strength reference values of children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years)[a]

Muscle group	Entire sample	3–9 years		10–19 years		20–59 years		60+ years	
		Male	Female	Male	Female	Male	Female	Male	Female
Grip, N	187.1 (94.9)	55.8 (29.1)	50.6 (25.3)	195.7 (83.0)[b]	153.8 (46.0)	305.0 (73.0)[b]	190.2 (50.4)	221.6 (49.5)[b]	128.7 (35.4)
Ankle dorsiflexors, N	164.7 (61.2)	87.1 (38.2)	81.6 (29.2)	197.2 (56.6)[b]	166.0 (37.8)	224.6 (48.9)[b]	166.5 (41.6)	173.3 (44.0)[b]	131.5 (38.9)
Ankle plantarflexors, N	257.0 (85.6)	151.7 (52.3)	142.7 (45.9)	309.9 (74.9)[b]	261.2 (52.7)	338.8 (66.8)[b]	243.9 (59.2)	281.4 (62.7)[b]	216.3 (60.3)
Knee flexors, Nm[c]	68.6 (33.8)	27.0 (13.9)	25.2 (11.5)	89.8 (34.6)[b]	65.9 (19.7)	106.3 (28.6)[b]	64.4 (18.9)	76.3 (20.1)[b]	45.8 (13.3)
Knee extensors, Nm[d]	124.0 (66.4)	34.9 (18.1)	34.2 (14.9)	152.8 (71.1)[b]	116.9 (36.6)	202.1 (56.1)[b]	122.6 (33.6)	136.2 (35.6)[b]	81.9 (26.8)
Hip internal rotators, N	146.8 (69.5)	63.3 (31.7)	61.1 (25.8)	178.5 (67.2)[b]	143.2 (45.0)	217.7 (62.4)[b]	136.1 (44.6)	169.7 (55.0)[b]	108.4 (33.8)
Hip external rotators, N	110.4 (52.6)	49.7 (22.7)	43.8 (16.8)	141.7 (53.7)[b]	104.0 (28.7)	169.4 (45.8)[b]	100.7 (29.1)	125.5 (33.9)[b]	76.3 (23.7)
Hip abductors, N	116.1 (50.6)	52.3 (23.1)	52.4 (21.9)	143.4 (47.2)[b]	116.6 (31.9)	170.7 (43.9)[b]	113.1 (32.4)	124.8 (32.8)[b]	83.8 (23.5)
Elbow flexors, N	176.3 (80.0)	71.7 (29.1)	66.0 (26.4)	213.8 (81.1)[b]	148.5 (36.8)	270.2 (59.6)[b]	164.4 (42.3)	209.4 (48.4)[b]	129.7 (33.9)
Elbow extensors, N	135.8 (57.4)	66.8 (24.4)	62.0 (19.7)	159.3 (56.8)[b]	118.3 (30.0)	203.2 (46.1)[b]	121.2 (30.2)	162.1 (36.8)[b]	102.8 (25.3)
Shoulder internal rotators, N	126.7 (64.7)	56.1 (27.1)[e]	47.7 (17.4)	151.5 (63.2)[b]	101.6 (27.7)	202.4 (55.9)[b]	109.7 (33.6)	159.7 (42.9)[b]	86.0 (27.5)
Shoulder external rotators, N	86.4 (41.0)	38.7 (19.5)	34.7 (13.0)	100.6 (38.8)[b]	73.4 (19.1)	134.7 (39.6)[b]	82.2 (20.9)	96.7 (25.3)[b]	63.3 (19.2)

[a] Mean values (SD).
[b] Significant ($p < 0.01$).
[c] Participants aged 3–11 years measured with hand-held dynamometry (mean 94.4, SD 40.6) was converted to Nm.
[d] Participants aged 3–11 years measured with -held dynamometry (mean 126.4, SD 53.5) was converted to Nm.
[e] Significant sex differences ($p < 05$).

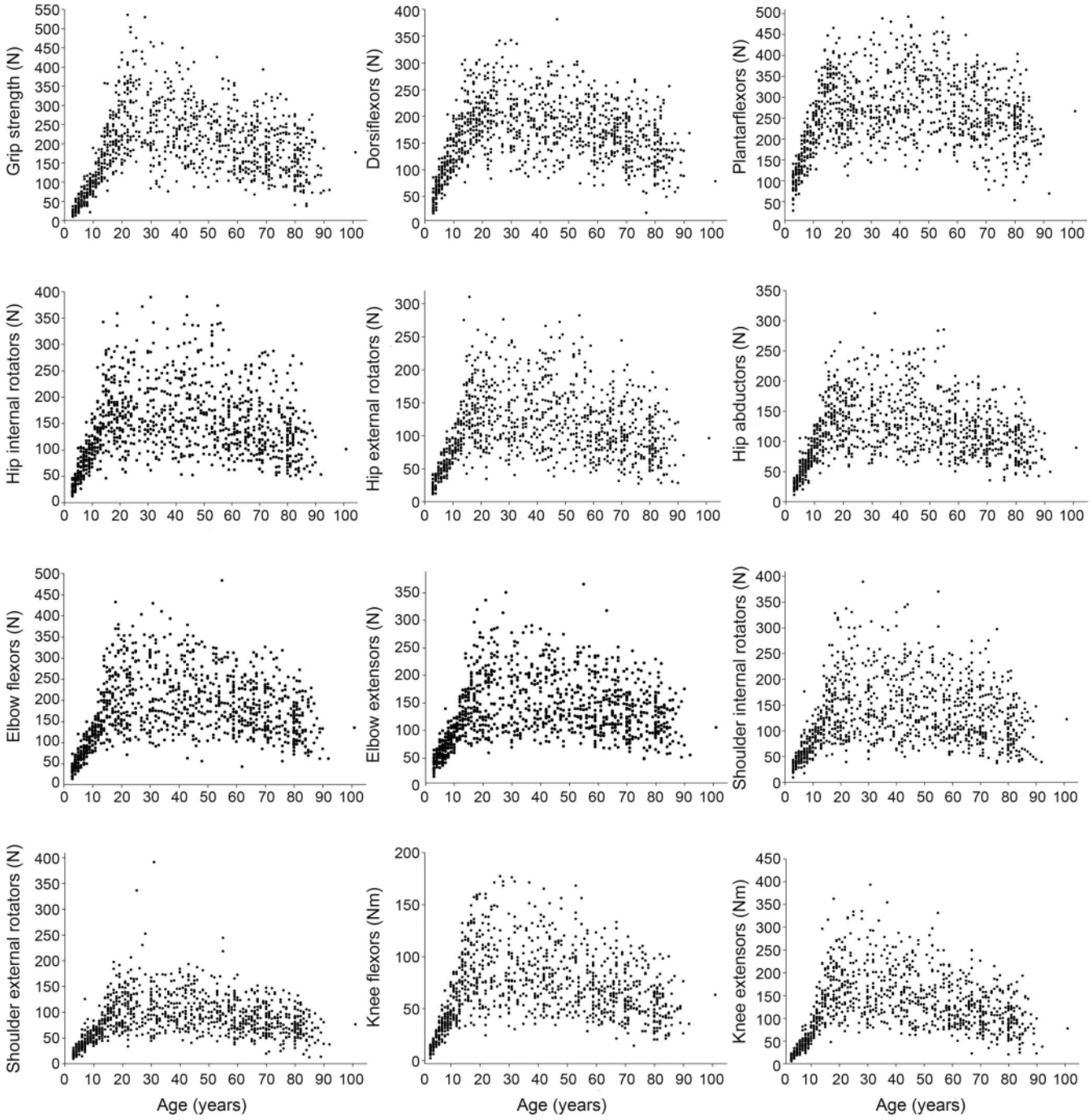
or muscle strength. Variables were retained in the multiple regression model if $p < 0.05$.

RESULTS To recruit 1,000 participants, 2,972 e-mails and 240 phone calls were logged. Ninety-one potential participants were excluded in accordance with the inclusion and exclusion criteria. Among adults aged over 18 years, 56% were currently working and 44% were not (31% retired and 13% students or unemployed). Participants were of diverse geographic ancestry, although the majority of participants were British/European ethnicity (74.4%), followed by Asian (16.6%), North or South American (5.1%), African (2.4%), and Aboriginal or Torres Strait Islander (1.5%). The sample mean (SD) age was 40.9 years (26.1), body mass 62.9 kg (21.1), height 1.61 m (0.02), waist circumference 78.6 cm (15.4), Foot Posture Index 3.5 (2.4), and lower limb alignment 1.8° (2.7). Ninety-three percent were right-footed and 91% were right-handed.

All missing data were accounted for. Four children declined to perform ankle dorsiflexion strength and 12 children were unable to perform cervical flexion and extension joint movements in accordance with the protocol. Ankle plantarflexors for 7 male adults and ankle dorsiflexors for 1 male adult were not assessed with hand-held dynamometry during periods of offsite calibration and servicing; 6 adults and 8 older adults were not assessed using fixed dynamometry due to safety concerns.

Normative reference values for the strength of 13 muscle groups per age category (children, adolescents, adults, and older adults) and sex are presented in table 1 and per decade in table e-1 at Neurology.org. From adolescence, male participants were significantly stronger in all muscle groups across all ages. There were no significant ($p < 0.05$) differences between the strength measures of boys or girls aged 3–9 years, except for shoulder internal rotators ($p = 0.031$), where boys were stronger. Correlations between strength and participant demographics and anthropometrics for children, adolescents, adults, and older adults are presented in table e-2. In children and adolescents, strength and age were highly correlated ($p < 0.05$), confirming that children become significantly stronger as they age from childhood and through adolescence. From 20 years of age, the relationship between strength and age changed. In adults aged 20–59 years, reduced strength with age was evidenced by significant, although weak, correlations with hand grip, ankle dorsiflexors, knee flexors and extensors, and shoulder external rotators. In older adults, decreased strength with increasing age was evidenced in all muscle groups. All muscle groups across all age categories demonstrated that greater height, body mass, and waist circumference were significantly associated with greater strength. The changes in strength measures with advancing age are shown in figure 1. Table e-3 shows the results of the multiple analyses. In children, height, followed

Figure 1 Scatterplots of muscle strength vs age for 1,000 children and adults



by waist circumference, was the most significant predictor of strength. In adolescents, a combination of body mass, sex, and age were shown to be the strongest independent predictors of strength. Sex (male) was the most significant predictor of strength in adults, followed by height and body mass with lower predictive values. In older adults, sex (male) was the most significant predictor, with body mass, height, and age demonstrating lower predictive values.

Normative reference values for active range of motion per age category (children, adolescents, adults, and older adults) and sex are presented in table 2 and per decade in table e-4. There was no significant difference ($p < 0.05$) in joint flexibility between boys and girls aged 3–9 years, except for hip internal rotation ($p = 0.017$), where girls had greater flexibility. Active range of motion was greatest in children compared to older adults. Figure 2 illustrates the inverse relationship between joint flexibility in all joints with age. Pearson correlations (table e-2) demonstrate that a decrease in flexibility with aging occurred in 8 of 13 joints of both adolescents and

Table 2 Joint flexibility reference values of children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years)[a]

		3–9 years		10–19 years		20–59 years		60+ years	
Movement, degrees	Entire sample	Male	Female	Male	Female	Male	Female	Male	Female
Ankle dorsiflexion	30 (6.7)[b]	33 (7.2)	31 (5.7)	32 (5.7)	31 (7.1)	32 (6.1)[b]	29 (6.4)	31 (6.1)[b]	26 (6.3)
Ankle plantarflexion	59 (8.6)[b]	63 (7.3)	63 (9.2)	58 (7.6)[b]	63 (7.3)	56 (7.5)[b]	62 (8.9)	53 (6.8)[b]	57 (7.2)
Knee flexion	137 (7.8)	145 (5.5)	144 (5.7)	140 (6.7)[c]	142 (6.6)	136 (6.1)	137 (6.2)	133 (7.2)	131 (8.1)
Knee extension	1 (2.9)	4 (3.3)	4 (3.9)	2 (2.6)	2 (2.6)	1 (2.3)	2 (2.7)	−1 (2.4)[c]	1 (1.6)
Hip flexion	121 (11.8)[c]	133 (9.1)	133 (9.8)	120 (9.9)[b]	124 (10.2)	120 (8.7)[b]	123 (10.0)	115 (10.7)	114 (12.6)
Hip internal rotation	37 (9.0)[b]	40 (8.4)	43 (9.1)	37 (9.3)	39 (7.7)	36 (7.9)[b]	40 (8.8)	33 (8.0)[c]	35 (8.4)
Hip external rotation	28 (8.5)[b]	32 (8.1)	32 (9.2)	31 (6.4)	31 (9.1)	30 (8.3)[b]	27 (8.3)	26 (7.0)[b]	22 (6.7)
Elbow flexion	148 (5.4)[b]	146 (5.4)	147 (5.8)	148 (5.4)[c]	150 (4.5)	147 (4.9)[b]	149 (5.4)	146 (6.0)[b]	149 (4.7)
Elbow extension	3 (5.9)[b]	7 (4.6)	7 (5.1)	4 (5.4)[b]	7 (5.6)	2 (5.0)[b]	4 (5.1)	−1 (5.0)[c]	0 (5.1)
Shoulder internal rotation	62 (12.9)[b]	67 (14.2)	67 (13.2)	62 (12.2)	66 (12.3)	58 (12.0)[b]	63 (14.0)	57 (11.0)[b]	63 (11.6)
Shoulder external rotation	83 (16.6)	98 (12.2)	99 (12.9)	93 (12.4)	93 (13.2)	83 (13.2)	83 (15.8)	71 (12.2)	72 (13.9)
Cervical flexion	60 (12.6)[b]	72 (13.5)	68 (12.4)	66 (12.3)	64 (10.5)	59 (10.8)[b]	56 (10.2)	55 (12.0)	53 (10.6)
Cervical extension	59 (19.5)[c]	82 (16.0)	80 (19.2)	67 (13.8)[c]	73 (15.3)	58 (13.1)[c]	61 (14.7)	40 (12.3)[c]	43 (13.3)

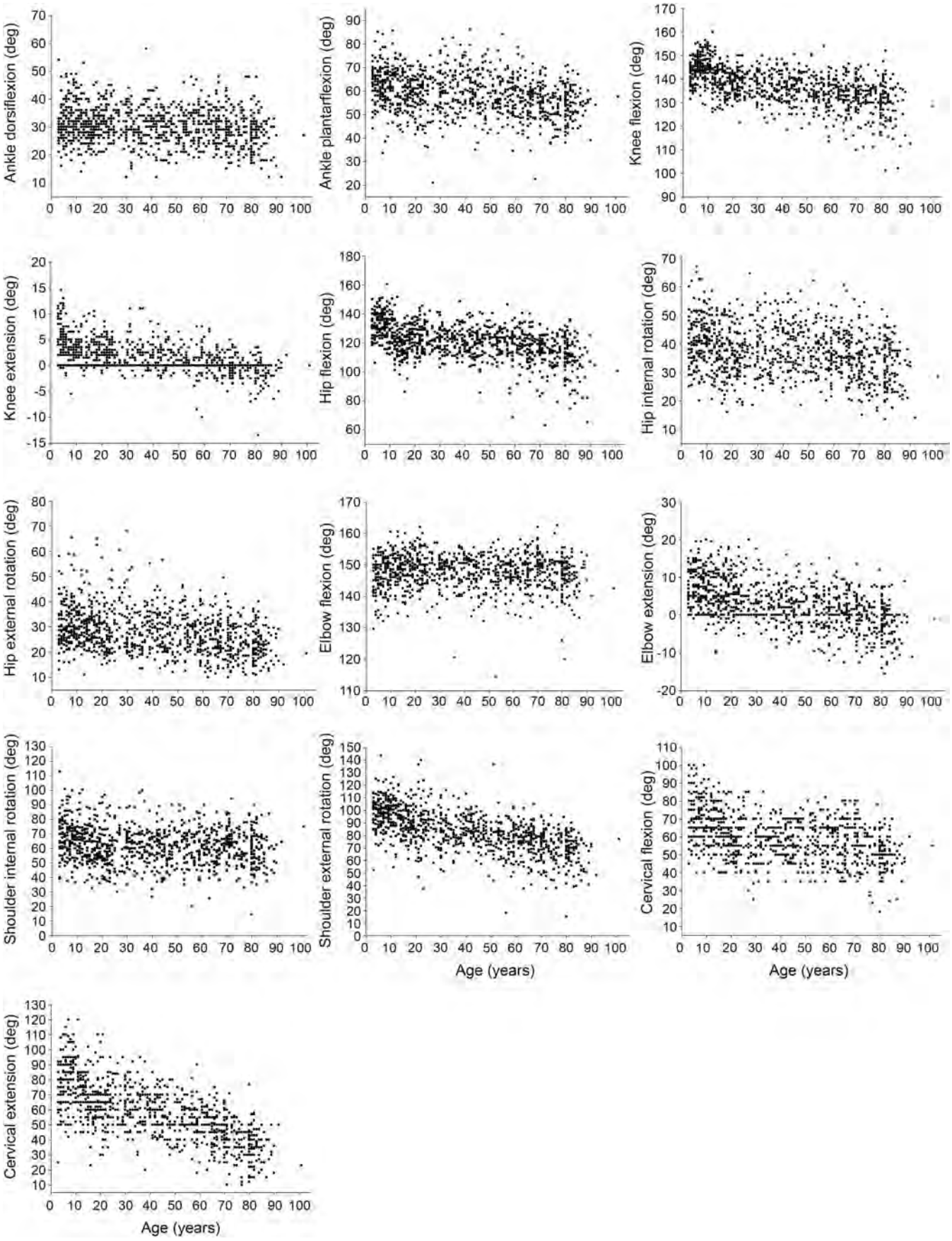
[a] Mean values (SD).
[b] Significant ($p < 0.01$).
[c] Significant sex differences ($p < 0.05$).

adults and in 12 joints of older adults ($p < 0.05$). Greater body mass and waist circumference were associated with a decrease in joint flexibility from 10 years of age. The correlation between height and joint flexibility was strongest in adolescents, where taller individuals demonstrated less joint range of motion. From adolescence to older adulthood, a more pronated foot posture was associated with a greater range of ankle dorsiflexion. Lower limb alignment did not demonstrate any significant correlations, beyond very weak associations, with measures of flexibility in any age category. In children, 2 multiple regression models (see table e-4) reached significance (knee extension and neck extension) and revealed height as the most significant predictor. Age, waist circumference, and height were the strongest independent predictors of flexibility in adolescents. For all adults older than 20 years, age, sex, and waist circumference were the strongest predictors of joint flexibility.

DISCUSSION This study has established a comprehensive reference dataset of isometric muscle strength and joint flexibility in 1,000 healthy people aged 3–101 years. The associations between strength and flexibility measures with demographic and anthropometric variables within different age categories identified some important relationships. As expected, there is a highly significant increase in strength of all muscle groups as children rapidly develop through to early adulthood. From adulthood, this relationship changes and a decrease in muscle strength with aging starts to occur; by older adulthood, all muscle groups demonstrate loss of strength with aging. From 10 years of age, a time of life coinciding with rapid growth and maturation, males are significantly stronger in all measures. In contrast, joint flexibility demonstrates a steady decline with age and no meaningful difference between males and females.

Our results are consistent with previous studies investigating isometric muscle strength in children[8,9] and adults.[12–14] However, direct comparison is limited by differences in age range, sample size, and muscle groups evaluated. Some studies report body mass as the strongest correlate with muscle strength in children,[8–11] while others demonstrate as we did that height showed the strongest relationship and was the most significant predictor of strength.[23,24] In adults, a decline in strength was most strongly associated with aging in 5 muscle groups (hand grip, ankle dorsiflexors, shoulder external rotators, knee flexors, and extensors), while in older adulthood all muscle groups demonstrated a significant decline in strength associated with aging. These results suggest a muscle-specific response to aging during adulthood and that generalized weakness does not occur until older adulthood. This highlights the importance of using age- and sex-matched reference data for specific muscle groups to avoid overrepresentation or underrepresentation of the force capabilities of a particular muscle group. Similar relationships between aging and muscle weakness have been reported in a limited number of adult studies.[12–14]

Figure 2 Scatterplots of joint flexibility vs age for 1,000 children and adults



The association between waist circumference and strength and flexibility has not been reported previously. Waist circumference was identified as a significant predictor of flexibility in adolescents, adults, and older adults and of strength in children. Epidemiologic studies have identified an association between waist circumference and tendon pathology,[25] with preliminary evidence supporting either a mechanical effect (due to increased load) or systemic effect (due to circulating lipids).[26] The influence of adiposity on localized musculo-tendinous tissues in neuromuscular disorders will be an important factor to evaluate with the increasing rates of obesity in society.

Few studies report normative reference values for flexibility in children. Our normative reference values for adults are consistent with the literature.[3,4,7] We identified only one sex difference in the flexibility of children (namely hip internal rotation), and only small differences (2°–6°) between men and women from adolescence through to older adulthood. As such, sex does not seem to have a clinically important effect on the joint flexibility of healthy adolescents and adults. There is no consensus in the literature regarding sex differences and flexibility; some studies report, as we have, that there is no clinically relevant difference,[4] while others report sex differences.[5] We identified a linear decrease in joint flexibility associated with advancing age, consistent with the adult literature.[3] It is likely that in healthy individuals, joint flexibility declines gradually and steadily with age and a substantial or sudden decline should be considered indicative of an underlying pathology.

Studies characterizing the functional decline and rate of progression of neuromuscular disorders such as amyotrophic lateral sclerosis,[27] Duchenne muscular dystrophy,[28] and Charcot-Marie-Tooth disease[29] depend on hand-held dynamometry to capture and track relevant changes in muscle strength. Access to reliable and expansive normative reference values and associated age- and sex-matched *z* scores are necessary to accurately and precisely quantify response to new interventions and to establish minimum clinically important differences.

This study is not without limitation. Participants were recruited through convenience sampling methods and with the exclusion criteria of conditions affecting physical performance may have resulted in a population that were particularly healthy and physically capable for their age. While the mixed ethnicity of our sample is reflective of the Australian population, the ethno-geographic variation in strength and flexibility measures could not be established. The cross-sectional study design was effective in achieving our study aim of generating a reference dataset of strength and flexibility across the lifespan; however, the direction of some of the cause and effect relationships can only be identified in longitudinal studies that track the changes in these measures over time. Ankle plantarflexion strength in healthy adolescents and adults can only ever be estimated with hand-held dynamometry due to the very high force capability (often exceeding 1,000 N).[30] The reported reference values for ankle plantarflexors are likely to underestimate the force capabilities of this muscle group, and values should be used as a lower threshold for weakness in patients with neuromuscular and other neurologic disorders. Finally, the strength and flexibility reference values are specific to the Citec hand-held dynamometer and Baseline goniometer and inclinometer and may not be interchangeable with data obtained from other devices.

The normative reference data generated from this study can be used to determine the presence and extent of impairments associated with neuromuscular disorders and to monitor disease progression over time. The reference values and associated age- and sex-matched *z* scores can be used to develop outcome measures with enhanced precision and responsiveness to be used in clinical trials for neuromuscular and other neurologic disorders.

AUTHOR CONTRIBUTIONS

Marnee J. McKay: study design, data collection, analysis and interpretation, drafting and revising the manuscript. Jennifer N. Baldwin: study design and data collection. Paulo Ferreira: study design and revising the manuscript. Milena Simic: study design and revising the manuscript. Natalie Vanicek: study design and revising the manuscript. Joshua Burns: study conceptualization and design, data interpretation, drafting and revising the manuscript.

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DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

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Exhibit 24

WORLD RUGBY

Transgender Women Guidelines

Can transgender women play rugby?

- Transgender women who transitioned pre-puberty and have not experienced the biological effects of testosterone during puberty and adolescence can play women's rugby (subject to confirmation of medical treatment and the timing thereof)
- Transgender women who transitioned post-puberty and have experienced the biological effects of testosterone during puberty and adolescence cannot currently play women's rugby
- Transgender women can play mixed-gender non-contact rugby
- World rugby are committed to ongoing evaluation of the guidelines and will remain current on all published research that pertains to the biological and physiological implications of testosterone suppression, with a formal review of the Guideline every three years. In support of this, World Rugby will prioritise support for high quality research projects on transgender rugby players, as part of this commitment to evidence-based guidelines.

Why can't transgender women play women's rugby?

Effects of testosterone

re reference is made to "females" and "males" to explain the effects of testosterone, the references are used to diffe

Testosterone is an androgenic-anabolic hormone whose functions include reproductive maturation, along with the genesis of male secondary sex characteristics. From puberty onwards, testosterone levels increase 20-fold in males, but remain low in females, resulting in circulating testosterone concentrations at least 15 times higher in males than in females of any age [1,2]. Among the biological changes initiated by testosterone and its derivatives are:

- Larger and denser lean muscle mass [3,4];
- Greater force-producing capacity of skeletal muscle [5,6];
- Stiffer connective tissue [7];
- Reduced fat mass and different distribution of body fat and lean muscle mass [3];
- Longer, larger and denser skeletal structure [8,9];
- Changes to cardiovascular and respiratory function that include higher haemoglobin concentration, greater cross-sectional area of the trachea and lower oxygen cost of respiration (as described in [1,10-12]).

Collectively, these biological differences account for large sporting performance differences between males and females. These include gaps between 9% and 15% for running, swimming and jumping events [13], between 15% and 35% for functional tasks like kicking, throwing, bowling and weightlifting, and in excess of 50% for tasks that involve upper body force production [10], since the biological effects of testosterone creates disproportionately greater strength on their upper compared to lower body, while females show the inverse [14,15]. In weight-lifting events, for instance, even when matched for mass and stature, males lift approximately 30% more weight than females. Evaluated differently, males are able to lift weights similar to females who weigh 30% to 40% more than them [10]. Functional movements such as explosive jumping are similarly larger in elite males than females, with approximately 30% more power generated during a counter movement jump [10].

The result of these biological differences is that males outperform females in all sporting activities where speed, size, power, strength, cardiorespiratory and anthropometric characteristics are crucial determinants [illegible]formance. This is true for many thousands of boys and men who have undergone a testosterone-

performance differences varies depending on the contributions made by each of the biological variables to performance, and indeed, some may be detrimental to performance in some events (mass during endurance running or cycling events, for example). Generally, however, there is no overlap in performance between males compared to females at all matched levels of competition from high school to the elite level The performance disparity is illustrated by the observation that thousands of teenage boys and adult males are able to outperform the very best biological females every year [13].

Similar performance differences between males and females have been described in non athletically trained individuals. Males have muscle mass 30% to 40% greater than females [4], maximal cardiorespiratory capacities (VO2max) 25% to 50% greater than in females [17], cardiovascular parameters between 11% and 43% greater than in females, lower limb strength approximately 50% higher than in females across the lifespan, and upper body strength 50% to 100% higher than in age matched females [6]. Even when elite females, trained in sports where grip strength is an important component of performance Uudo and handball), do not outperform untrained males in a grip strength task, with the very best female performance corresponding to approximately the 58 th percentile for males, and a 26% advantage for untrained males compared to typical elite females Punching performance, a composite movement reliant on strength, power, co-ordination and mass, has been found to be 162% higher in males than in females [18], and 17-year old boys are able to throw a ball further than 99% of adult females [19].

Biological consideration for rugby union

The implications of biological and performance differences for rugby are two-fold. First, significant differences in strength, size, speed and power have potential consequences for the safety of participants in rugby, where much of the sport involves contacts in the form of tackles, rucks and mauls, as well as numerous periods of high force production during static contests for the ball, such as the scrum and ruck. Given the documented risk of injury in rugby from contact events in particular [20-24], the elevated possibility of all injuries, including serious injury, from large disparities in size, speed, power, and force, is of rn Recent modelling of tackles using validated biomechanical models [25,26] suggests that the

greater risks for smaller and slower players, particularly when size and speed exist in combination.

Given that the typical male player mass is 20% to 40% greater than typical women mass, that males have strength 40% to 80% greater (unadjusted for mass), and that men are 10% to 15% faster than women despite being heavier, the risk of injury created by large imbalances in mass and speed may be considered significant. To explore this, we assessed the range of masses of players at the international level and applied the findings to a biomechanical model to explore possible implications for injury risk should cross-over scenarios occur.

With respect to mass, we documented the range of sizes of elite men's and women's players from the 2011 Rugby World Cup up to the 2019 Rugby World Cup, finding:

- Typical (median) men's players are 41.1% heavier than typical women's players (103 kg vs 73 kg)
- Among forwards, the heaviest 1% of women players are smaller than the typical men's forward (109kg for women vs 112kg for men)
- The heaviest 1% of women's backs are smaller than typical men's backs (89kg vs 92kg)
- The lightest 1% of men's forwards are approximately equal in mass to the heaviest 10% of women's forwards, while the lightest 2% of men's backs are approximately equal to the heaviest 10% of women's backs
- Figure 1 below shows the frequency histograms for men's and women's players in forward and back positions

Figure 1: Frequency histograms of mass of forwards (left panel) and backs (right panel) in elite men's and women's rugby players. Dotted lines indicate the 50th percentile, while dashed lines indicate the 98th percentile for each group.

Implications for injury risk - head injury models

The differences observed between men and women with respects to mass may be combined with differences in speed to create a theoretical framework in which the inertial load and forces faced by smaller and slower player is significantly greater when in contact with a larger, faster player. this model is intended for illustrative purposes and demonstrates the impact of only one variable known to differ between biological males and females - namely mass - on head injury risk, in a basic parametric model, absent force application and complex movements, as a preliminary impact analysis. the principles illustrated by the model would apply to other injuries. The addition of speed, and strength or force exerted during contact would further increase the implications of the findings of this illustrative model, summarized below.

The representative figure below illustrates the concept of mass disparity as a risk of injury for ball carriers. It depicts the linear acceleration (A), angular acceleration (B), neck force (C) and neck moment (D) experienced by ball carriers of different masses when tackled by players with different masses. Using the known masses of international rugby player, the position of the average male (M50) and average female (F50) are plotted on each heat map. F90 shows the scenario where a tackler (T) corresponds to the 90th percentile for women's mass (see Figure 1) tackles a typical female mass ball carrier (BC). X indicates the hypothetical cross-over scenario in which a typical male tackler mass is involved in a tackle against a ball carrier with a typical female mass.

Figure 2. Graphical representations of linear acceleration (A), angular acceleration (B), neck force (C) and neck moment (D) for ball carriers of different masses during tackles by tacklers with different masses. Mso and Fso show the modelled situation when typical/median players tackle one another for men and women, respectively. F90 represents a female ball-carrier with typical mass against a tackler in the heaviest 10% of women's body mass. X denotes the cross-over situation that would hypothetically occur for a tackler at the men's median mass tackling a typical female ball carrier

The modelling shows that a tackle involving players with typical or average mass produces slightly greater accelerations and forces in men (Mso) than in women (Fso). This is a function of the higher mass of men's players. Head and neck kinematic and kinetic variables increase significantly when the heaviest 10% of women's body mass is used for the tackler against a typical ball carrier (F90), but this extreme "within female-bodied" scenario produces smaller kinetic and kinematic outcomes than if the hypothetical cross-over scenario were to occur, where an average male-bodied player is the tackler and the average female-bodied player the ball carrier **(X).** The magnitude of the increase in neck forces, moments and accelerations for the ball carrier is between 20% and 30% for typical cross-over scenario compared to the typical female vs female scenario, and is 10% greater for the male-bodied vs female-bodied crossover scenario than a tackle where the heaviest 10% of women are matched against typical women's mass (F90).

compared to the typical tackle scenario in women's rugby. The magnitude of these extreme head accelerations and neck forces are not seen in women and are created by cross-over of male-bodied players to women's rugby. Similar differences are seen when examining the accelerations and forces for the tackler's head and neck.

The magnitude of the known risk factors for head injury are thus predicted by the size of the disparity in mass between players involved in the tackle. The addition of speed as a biomechanical variable further increases these disparities, which is relevant given that male players weighing 103kg (the median for men) would be expected to run between 10% and 15% faster than typical female players (mass 73kg), and thus considerably faster than female players who are heavier than the median (eg females at the 90 th percentile, Fig 1). This would further compound the disparity created.

Next, it is important to also consider that these models do not account for the ability of players to actively exert force at high rates during tackles. This would be a function of power and strength, which are similarly known to be 30% to 80% greater in biological males than females. When these active applications of force during contact are added to the mass and speed characteristics illustrated and described above, the resultant neck and head forces and accelerations will increase even further, such that the illustrative model shown above depicts the smallest possible risk increase for typical players involved in a tackle as a result of mass alone. The addition of speed and force disparities will increase the magnitude of these risk factors beyond the 20% to 30% we illustrate above.

The implication of these increases is complex to quantify but would result in increased injury risk for the player experiencing the elevated risk outcomes (force and acceleration). This is because head injuries occur when forces and accelerations on the head and neck reach a threshold necessary to cause injury, and which is unique to each tackle situation. A tackle situation that typically produces risk factors within 20% of this threshold would, in the circumstance of a typical male-bodied vs typical female-bodied player illustrated above, be sufficiently increased to cause an injury. The higher risk scenario involving heavier bodied players would further increase injury likelihood, since all tackle situations that normally ce kinetic and kinematic variables within 40% to 50% of an injury threshold would now exceed it, a

causing head injury.

Finally, it must also be considered that the ability to withstand or tolerate forces on the head and neck are required to avoid brain injury. This is the reason neck strength is critical in injury prevention. Since the strength disparities between males and females is so large, including a 50% lower neck isometric strength in females, the reduced ability of female players to tolerate or withstand the forces in tackles is a further risk factor for injury, including head injury as described above, but relevant to all injuries where the rapid application of force or load are responsible for injury.

Implications for injury risk - scrum forces

The implication of greater mass and force-producing ability in males can be seen in forces measured during scrums in both elite and community level rugby. Research on the forces applied during scrums shows that at the elite level, males produce approximately twice the peak force of females in the scrum. Even at the community level, where peak force is 30% lower than in the elite game, males produce approximately 40% greater peak force during scrums than elite females. Given that force producing and receiving ability is likely to be significantly lower in female community players, the implication is that men's community level rugby scrums will be considerably more forceful than women's community level scrums.

The risk of particularly serious and catastrophic injuries during scrums has led to a number of law changes specifically designed to depower the scrum to reduce injury risk. This risk would be amplified by large mismatches in strength between opposing players, since the force applied must be withstood by a direct opponent. This is an illustration of how mismatches in strength and size are directly responsible for forces that result in injury.

It must be noted that the actual testosterone level, measurable in the body, is not a strong predictor of performance within men and within women [27 29] This is because performance is multifactorial, and testosterone's androgenzing effects contribute to, but do not solely influence the biology and resultant performance outcomes within a group who are able to utilize it. The biological basis for male vs female differences is thus the result of testosterone, but it does not necessarily follow that within men and within women, the hormone is a predictor of performance

Further, differences in the sensitivity to testosterone between individuals mean that a given level of testosterone is not a sensitive or specific predictor of performance within each group (males and females) This is in part because most males have elevated levels and some degree of sensitivity, while the level in females is significantly lower and rarely exceeds even the very low end of the male range [1]. Therefore, in two homogenous groups that are matched for either the presence or absence of a given variable (males and females for the presence or absence of testosterone, in this case), the predictive value of that variable within a group is greatly diminished, the same way that V02max is a significant predictor of running or cycling performance across the whole population, but not within a group of elite marathon runners or cyclists, who are already relatively homogenous for that characteristics [30]. Similarly, height is clearly advantageous for professional basketball, but within the National Basketball Association (NBA), where height has already been selected for and participants are in the extreme upper end of the overall population for that characteristic [31], it becomes a poorer predictor of performance.

However, when the same question -does testosterone predict performance across humans of both sexes - is asked of binary categories (males vs females in sport, rather than within males or females), then the predictive power of testosterone is strong, because "high testosterone" during adulthood is a very reliable indicator that the androgenizing effects of testosterone have occurred earlier during life When understood and assessed this way, testosterone is necessary for peak performance (since the top performers within humans are all male), but it is not sufficient to attain it. It is here that the almost perfect sensitivity of biological sex emerges, since in a ranking list of the top thousand performances in port, every year, every single one will be biologically male

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In summary, across all performance levels and ages after puberty, testosterone is primarily (though not exclusively) responsible for very large typical differences in the biology of males and females, and consequently, performances between the sexes. These are summarized in Figure 3 below, which combines the biological differences between males and females with their performance implications, and is reproduced from a recent article currently in review [10].

Figure 3: Summary comparison of biological (left table) and performance (right figure) differences between males and females for a range of biological variables and physical activities/events. Reproduced from Hilton & Lundberg [10]

Given that the women's category exists to ensure protection, safety and equality for those who do not benefit from the biological advantage created by these biological performance attributes, the relevant and **crucial question is whether the suppression of testosterone for a period of 12 months, currently required for transgender women participation in women's sport, is sufficient to remove the biological differences summarized above?**

Current policies regulating the inclusion of transgender women in sport are based on the premise that reducing testosterone to levels found in biological females is sufficient to remove many of the biologically based performance advantages described above. However, peer-reviewed evidence suggests that this is not the case, and particularly that the reduction in total mass, muscle mass, and strength variables of transgender women may not be sufficient in order to remove the differences between males and females, and thus assure other participants of safety or fairness in competition

Based on the available evidence provided by studies where testosterone is reduced, the biological variables that confer sporting performance advantages and create risks as described previously appear to be only minimally affected. Indeed, most studies assessing mass, muscle mass and/or strength suggest that the reductions in these variables range between 5% and 10% (as described by Hilton & Lundberg [10]). Given that the typical male vs female advantage ranges from 30% to 100%, these reductions are small and the biological differences relevant to sport are largely retained

For instance, bone mass is typically maintained in transgender women over the course of at least 24 months of testosterone suppression, with some evidence even indicating small but significant increases in bone mineral density at the lumbar spine [32-34]. Height and other skeletal measurements such as bone length and hip width have also not been shown to change with testosterone suppression, and nor is there any plausible biological mechanism by which this might occur, and so sporting advantages due to skeletal differences between males and females appear unlikely to change with testosterone reduction

With respects to strength, 1 year of testosterone suppression and oestrogen supplementation has been found to reduce thigh muscle area by 9% compared to baseline measurement [35] After 3 years, a further reduction of 3% from baseline measurement occurred [36]. The total loss of 12% over three years of treatment meant that transgender women retained significantly higher thigh muscle size ($p<0.05$) than the baseline measurement of thigh muscle area in transgender men (who are born female and experience female puberty), leading to a conclusion that testosterone suppression in transgender women does not e muscle size to female levels [36].

find that 1 year of testosterone suppression to female typical reference levels results in a comparatively modest loss of lean body mass (LBM) or muscle size, with consistent changes between 3% and 5% reduction in LBM after 1 year of treatment (as summarized from source research studies by Hilton & Lundberg [10]).

Muscle force-producing capability is reduced after testosterone suppression, though as appears to be the case for muscle/lean mass, these reductions are considerably smaller in magnitude that the initial male-vs-female differences in these variables. For instance, hand-grip strength was reduced by 7% and 9% after 1 and 2 years, respectively, of cross hormone treatment in transgender women [39], and by 4% in 249 transwomen after 1 year of gender-affirming treatment, with no variation between different testosterone levels, age or BMI tertiles [45]. Transgender women retained a 17% grip-strength advantage over transgender men at baseline measurement, with a similarly large, retained advantage when compared to normative data from a reference or comparison group of biological females.

Most recently, Wiik et al found that isokinetic knee extension and flexion strength were not significantly reduced in 11 transgender women after 12 months of testosterone suppression, with a retained advantage of 50% compared to a reference group of biological females and the group of transgender men at baseline [41]. This absence of a reduction in strength occurred in conjunction with a 4% to 5% reduction in thigh volume, and no difference in the contractile density of the muscle, which suggests that the reduction of testosterone for a period of a year had no effect on the force-producing capacity per unit of cross sectional area [41], a variable that is known to be higher in males than females.

In conclusion, longitudinal research studies that have documented changes in lean mass, muscle mass/area and strength show consistently that small decreases occur as a result of testosterone suppression, with a resultant relatively large retained advantage in these variables compared to a group of biological females.

Testosterone exerts significant biological effects on biological males during puberty and adolescence. This creates large differences in strength, mass, speed, power, and endurance capacity. In turn, these create player welfare concerns and performance inequality in rugby, given the importance of physical contact and strength in the sport. Longitudinal research studies on the effect of reducing testosterone to female levels for periods of 12 months or more do not support the contention that variables such as mass, lean mass and strength are altered meaningfully in comparison to the original male-female differences in these variables. The lowering of testosterone removes only a small proportion of the documented biological differences, with large, retained advantages in these physiological attributes, with the safety and performance implications described previously. There is currently no basis with which safety and fairness can be assured to biologically female rugby players should they encounter contact situations with players whose biologically male advantages persist to a large degree.

While there is overlap in variables such as mass, strength, speed and the resultant kinetic and kinematic forces we have modelled to explore the risk factors, the situation where a typical player with male characteristics tackles a typical player with female characteristics increases the magnitude of known risk factors for head injuries by between 20% and 30%. In the event of smaller female players being exposed to that risk, or of larger male players acting as opponents, the risk factors increase significantly, and may reach levels twice as large, at the extremes. The basis for regulation is the typical scenario, though risk mitigation must be mindful of the potential for worst-case scenarios that may arise. Both are deemed unacceptably high, because they would result in a number of tackle situations that currently lie beneath a threshold required to cause injury increasing to exceed that threshold.

Thus, it is on the basis of male vs female biological differences, combined with no evidence for removal of their implications for safety and performance, that the guideline is that trans women should not compete in women's rugby.

It is acknowledged that the published studies currently available on testosterone suppression and physiological changes (compiled and described in Hilton and Lundberg, 2020 and reviewed individually in the proposed policy document) have been conducted in untrained transgender women. This invites questions over the validity and generalizability of the studies to a sports-playing population.

This is a valid question, and it is acknowledged that research is required to fully address questions arising out of this limitation. World Rugby is committed to supporting high quality research proposals in this area, should they be submitted as part of World Rugby's Research programme.

However, this limitation can also be assessed within an understanding of the physiological implications of trained compared to untrained individuals undergoing testosterone suppression. The application of insights from complementary studies leads to a conclusion that the available research is in fact sufficient to arrive at firm conclusions about safety, performance and retained advantages, and thus the recognized limitations are not sufficient to refrain from drawing a conclusion on the likely implications of the transgender research for athletes.

In assessing this issue, two primary questions may be asked:

1. How would training undertaken during the process of testosterone suppression affect the changes observed in muscle and lean body mass, and strength variables, compared to studies done in individuals who do not perform training?
2. How would training prior to a period of testosterone suppression influence:
1. The baseline or pre-suppression measures for muscle mass and strength in transgender women, and thus the differences in these variables compared to a reference or control group of biological women (cisgender women)?
2. The likely "end-point" for muscle and lean body mass as well as strength after the testosterone suppression for a period of at least twelve months, once again compared to a reference or comparison of cisgender women?

Training during the intervention to lower testosterone levels can reduce, eliminate, and even reverse any losses in muscle and lean body mass, muscle volume, and muscle strength. This is supported by evidence from various study models in which biological males reduce testosterone to within the female range, and are able to maintain or even increase these physiological variables through training [46-48].

> The implication is that any performance decline as a result of androgen deprivation is minimized or eliminated, and so the studies cited in support of the World Rugby Guideline, while conducted on non-training individuals, establish the minimum possible retained advantage for trans women. That is, they establish that in the absence of training during testosterone suppression, an advantage is retained compared to cisgender women. That advantage is either the same, or very plausibly increased as a result of training.

Training prior to the intervention will cause increased muscle mass and strength variables at baseline. This means that the initial or "pre-suppression" differences in these variables compared to biological females will be greater than in an untrained trans woman. This rebuts the assertion that trans women are weaker, less muscular and thus more similar to biological females at baseline, within a sporting context, since the transgender woman being considered by World Rugby is much more likely to be trained (or will train once transition begins, as described above).

> Further, once the period of testosterone suppression begins, then the degree to which muscle mass and strength decreases may be either the same or relatively greater in the trained trans women as a result of this higher baseline. Even if the relative loss of muscle mass and strength are higher than in untrained trans women, it is inconceivable, and even physiologically impossible, that a pre-trained athletic trans woman is going to lose so much muscle mass and strength that they end at a point where they are less muscular/lean and weaker than a theoretically untrained (and even 'self-starved') transgender woman.

advantage for a pre-trained trans woman. The effect of training can only be to increase this value or to achieve the same value of X percent retained advantage, but it cannot reduce it further, unless one argues that a trained trans woman will lose so much lean mass and strength that they end up weaker and less muscular than a completely non-athletic individual.

Finally, it is relevant that studies comparing untrained biological males and highly trained females, males retain an advantage despite the training status of biological females. For instance, in a study on grip strength, the strongest elite athletically trained females in sports where grip strength is a performance advantage Uudo and handball) are only as strong as untrained biological males at the 58th percentile, with a 26% difference in strength between typical elite females and typical untrained males [49]. Similarly, Morrow & Hosler (1981) found that untrained college-aged males were more than twice as strong as trained female basketball and volleyball players in a bench press task, with the top 5% strongest trained females equal in strength to the weakest 14% of untrained males This establishes that pre-trained biological females can increase strength beyond that of untrained females, but still do not compare to untrained biological males.

The implication is also that since even typical untrained biological males have a large strength advantage compared to elite and trained females, studies that have documented only small reductions in strength and thus persistence of strength advantages with androgen deprivation in untrained biological males (as in Kvorning et al [46], Chen et al [47] and in studies on transgender women cited herein) should be considered relevant for establishing the smallest possible retained advantage that would exist in the absence of training. As described above, and in studies where training is conducted while testosterone is suppressed [46-48], the advantage will only remain this size or increase.

Finally, it is also recognized that not all sports are affected similarly by the variables we have weighted as crucial for rugby (size, strength, speed, power). Indeed, in some sports, excess mass may be disadvantageous, and thus the model for retained advantage and persistent risk may present differently f... ifferent physical activities.

physiological and performance advantages and those to whom it does not are removed sufficiently to enable participation of transgender women in women's rugby. At the present time, however, based on the best published scientific evidence, that position is unsupported.

The referenced research used to support this position can be viewed **here**.

Conclusion - Testosterone, Welfare and Performance

Having considered all of the currently available information, the working group determined that the best evidence **currently** available means that those who experienced the biological effects of testosterone during puberty and adolescence cannot safely or fairly compete in women's rugby. That means that currently, transgender women may not compete in women's rugby.

World Rugby is committed to encouraging transgender people to remain involved with rugby and is currently funding research to continue to review any evidence that may emerge to enable the participation of transgender women in women's rugby. Details of the research currently underway, along with details of how to apply for research funding for those who may be interested, is available **here**.

How do I stay involved in rugby if I can no longer play in the category that I want to?

World Rugby acknowledges that the introduction of this Guideline will mean that some players can no longer play in the category that they want to. It is possible that will change in the future and World Rugby is funding research to try to find out if there are ways to allow that safely and fairly (see **here** for details). In the meantime, there are many other ways to stay involved with rugby;

- Other forms of the game: Many forms of non-contact Rugby exist such as: Tag; Touch; Flag etc all have
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courses for coaches of children, adolescents, and adults. All courses are open to any participant.

- Refereeing: For many people who may not be able to play, refereeing is a viable alternative to stay close to the game. World Rugby and its member Unions offer several introductory courses and a pathway exists in all Unions for fast-tracking talented
- Administration: All clubs rely on volunteer administrators. As individuals enter the latter stages of the long-term participant model, then administration becomes a realistic outlet for many. A number of Unions have dedicated support resources for individuals who wish to pursue this path of staying involved.

World Rugby is currently exploring the possibility of an "open category" of rugby in which any player could play, regardless of gender identity. World Rugby has committed to exploring this option with its Unions, Associations, International Rugby Players, and trans advocate groups including Gendered Intelligence and International Gay Rugby.

What if I have concerns about safety or fairness relating to someone I am playing with or against?

It is important to note that many people do not meet cultural or local norms or stereotypes related to the expression of gender identity. All players and Unions ought to take care to consider this when raising any concerns about another player. In the event that a player or Union has a genuine concern about safety or fairness in relation to another player, the concern should be dealt with as follows:

1. The concerned person should raise their concerns with their Union's Chief Medical Officer (CMO).
2. The Union's CMO should carefully consider the concerns raised, in the context of all of the known facts and if having done so, the CMO determines that the concerns are not frivolous or vexatious, the CMO should contact the World Rugby CMO setting out the basis for the
3. The World Rugby CMO will engage with the CMO of the Union of the player about whom the concerns h[illegible] been raised, ensuring confidentiality for the player and involved team-mates and opponents [illegible]ghout the

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5. In some circumstances, such appropriate actions may include a recommendation that a standardised endocrinological assessment be performed [Appendix].

6. For the avoidance of doubt, no player should or would be forced to undergo any medical or other assessment. It is a player's responsibility to decide on whether he or she wishes to proceed with any assessment. However, it should be noted that deciding not to participate in an assessment, having been requested to do so, may have consequences in terms of the player's eligibility to participate in the category of competition that is consistent with his/her/their gender identity, since it may not be possible to determine whether issues of safety or fairness arise without such assessment.

Exhibit 25

STATE OF ARIZONA
OFFICE OF THE GOVERNOR

DOUGLAS A. DUCEY
GOVERNOR

EXECUTIVE OFFICE

March 30, 2022

The Honorable Katie Hobbs
Secretary of State
1700 W. Washington, 7th Floor
Phoenix, AZ 85007

RE: Senate Bill 1138 irreversible gender reassignment surgery; minors & Senate Bill 1165 interscholastic; intramural athletics; biological sex

Dear Secretary Hobbs,

Today I signed S.B. 1138 and S.B. 1165, legislation to protect participation and fairness for female athletes, and to ensure that individuals undergoing irreversible gender reassignment surgery are of adult age. This legislation is common-sense and narrowly-targeted to address these two specific issues — while ensuring that transgender individuals continue to receive the same dignity, respect and kindness as every individual in our society.

S.B. 1138 delays any irreversible gender reassignment surgery until the age of 18. The reason is simple, and common sense – this is a decision that will dramatically affect the rest of an individual's life, including the ability of that individual to become a biological parent later in life.

Distinguishing between an adult and a child in law, as this bill does, is not unique. Throughout law, children are protected from making irreversible decisions, including buying certain products or participating in activities that can have lifelong health implications. These decisions should be made when an individual reaches adulthood. Further, many doctors who perform these procedures on adults agree it is not within the standards of care to perform these procedures on children.

The irreversible nature of these procedures underscores why such a decision should be made as an adult, not as a child, and further supports the importance of this legislation.

S.B. 1165 creates a statewide policy to ensure that biologically female athletes at Arizona public schools, colleges, and universities have a level playing field to compete. This bill does not deny student-athletes the eligibility to play on teams not designated as "female," and it doesn't impact club sports leagues offered outside of schools. Every young Arizona athlete should have the

opportunity to participate in extracurricular activities that give them a sense of belonging and allow them to grow and thrive.

This legislation simply ensures that the girls and young women who have dedicated themselves to their sport do not miss out on hard-earned opportunities including their titles, standings and scholarships due to unfair competition. This bill strikes the right balance of respecting all students while still acknowledging that there are inherent biological distinctions that merit separate categories to ensure fairness for all.

Sincerely,

Douglas A. Ducey
Governor
State of Arizona

cc: The Honorable Karen Fann
The Honorable Rusty Bowers
The Honorable Warren Petersen
The Honorable Nancy Barto

EXHIBIT 4

WILENCHIK & BARTNESS
A PROFESSIONAL CORPORATION

Jane Doe, by her next friends and parents, Helen Doe and James Doe; and Megan Roe, by her next friends and parents, Kate Roe and Robert Roe

.v.

Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction;
Laura Toenjes, in her official capacity as Superintendent of Kyrene School District;
Kyrene School District;
The Gregory School;
Arizona Interscholastic Association, Inc.

Case 4:23-cv-00185-JGZ

Expert witness statement
Emma Hilton, PhD

1. Qualifications and experience

1.1. I am Emma Hilton. I am a postdoctoral researcher in developmental biology—the study of how embryos grow and how individuals mature—at the University of Manchester, UK, a world top 50 university.[1] My short-form academic curriculum vitae is attached in **Appendix 1**.

1.2. In 1999, I received my Bachelor of Science degree from the University of Warwick, UK, where I studied Biochemistry. My final year dissertation described research to identify a genetic cause of Sotos syndrome, a genetic disorder characterised by, among other features, prenatal and childhood bone overgrowth, leading to unusually-early peak height velocity, increased stature during childhood, and concurrent advanced bone age.[2] In 2004, I received my Doctor of Philosophy degree from the University of Warwick, UK, having identified a gene regulatory mechanism that integrates molecular growth signals to specify the future tissue development of a particular region of the very early "ball-of-cells" stage vertebrate embryo.[3,4]

1.3. Since 2004, I have been employed as a developmental biologist at the University of Manchester, UK. My developmental biology career has focussed on the molecular mechanisms underpinning inherited genetic disorders in humans, including—but not limited to—those that differently affect males and females and those that affect neuromuscular development during embryo development.[5] I am currently employed in a research programme to uncover the molecular development of the skin surface in tadpoles, which is the animal model I have systematically exploited to understand human development and disease.

1.4. I have authored over 20 peer-reviewed publications in developmental biology and genetics journals, and have received over 1300 citations. My h-index is 17.[6] I have contributed a chapter entry to a key medical textbook on genetic disorders.[7] In 2007, I received the honour of being named as an Outstanding Young Investigator by the European Society of Human Genetics for my research on a sex-linked genetic disorder that causes first-trimester death in male fetuses.[8]

1.5. Although not employed in a teaching role, I deliver an annual lecture to undergraduate medical students in genetic disorders, inheritance and the ethics of medical screening. I have previously delivered teaching to ophthalmology Masters students in eye development and genetic disorders of the eye, and to undergraduate dentistry students on craniofacial disorders.

1.6. Developmental biology is not simply the study of specific processes in specific species (for example, as part of my current collaborative research, how a nerve makes a junction with a developing block of muscle to generate a functional movement unit.) The discipline of developmental biology operates on common principles: how regions are zoned; how cells "talk" to each other; how tissues and organs interact in synergistic or exclusive patterns; how such interactions proceed. These common principles apply to

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[1] https://www.manchester.ac.uk/study/experience/reputation/rankings/
[2] https://www.genomicseducation.hee.nhs.uk/genotes/knowledge-hub/sotos-syndrome/
[3] Rex et al., 2002. Multiple interactions between maternally-activated signalling pathways control Xenopus nodal-related genes. Int J Dev Biol 46: 217-226.
[4] Hilton et al., 2003. VegT activation of the early zygotic gene Xnr5 requires lifting of Tcf-mediated repression in the Xenopus blastula. Mech Dev 120(10): 1127-1138.
[5] https://www.research.manchester.ac.uk/portal/emma.hilton.html
[6] https://scholar.google.com/citations?user=A8zl2ggAAAAJ&hl=en
[7] Hilton et al., 2016. "The BCL6 corepressor (BCOR) and oculofaciocardiodental syndrome." In Epstein's Inborn Errors of Development: The Molecular Basis of Clinical Disorders of Morphogenesis. Oxford University Press, Oxford, UK.
[8] https://www.eshg.org/index.php?id=102

multiple events in the global development of all species. A solid understanding of such principles—as I have acquired over my 20-year career—permits any developmental biologist to quickly build a picture of developmental events outside of their specific research programme. The differentiation, development and patterning of the reproductive system and the physical changes induced during maturation are no exception for a trained developmental biologist.

1.7. Over the past six years, I have deepened my academic knowledge of physical sex development in many species, particularly humans. Notably, my active research has always involved extensive sexing and breeding of animals, dissecting reproductive organs like male testes (frogs) and the female uterus (mice), and understanding reproductive issues in my animal colonies (for example, the loss of male sex characteristics with aging in frogs). As part of my previous research in a sex-linked genetic disorder, I have routinely visualised and analysed sex chromosome conformation in mice and humans.[9]

1.8. My expertise in human sex development is increasingly recognised in an academic context. In 2021, I was invited by the editor to publish a letter in the official organ of the Royal Academy of Medicine in Ireland, where I argued that, "*Human sex is an observable, immutable, and important biological classification; it is a fundamental characteristic of our species, foundational to many biology disciplines, and a major differentiator in medical/health outcomes.*"[10] I am the invited lead author of a chapter on human sex development in an academic "primer" textbook to be published in August 2023.[11] Titled "Two sexes", this peer-reviewed chapter describes the evolution trajectory of the two sexes in almost all complex species, the development of sexed anatomy in humans, and common myths regarding the phenomenon of sex. Although not yet published, the chapter text is attached in **Appendix 2**. Since 2022, I have delivered a seminar to undergraduate life sciences students in sex development and the long-term effects of sex hormones on the development of the human body.

1.9. During my school years, I competed in interscholastic and regional competitions in judo, track running, netball, field hockey, cross-country and tennis. As an adult, I have completed two marathons. I currently participate in recreational sports, playing netball in single-sex and mixed-sex leagues, and weightlifting with a personal trainer. I am a sports fan.

1.10. The relevance of developmental biology in sports performance has been typically underestimated, particularly in the context of transgender athletes. A long-standing assumption has been that hormonal intervention is sufficient to secure fairness when transgender women were included in female sports. I and Doctor Tommy Lundberg (Karolinska Institutet, SWE) challenged, for the first time in the academic literature, that assumption. In Hilton and Lundberg (2021),[12] the peer-reviewed academic publication most relevant to this expert statement, we, "*review[ed] how differences in biological characteristics between biological males and females affect sporting performance and assess[ed] whether evidence exists to support the assumption that testosterone suppression in transgender women removes the male performance advantage and thus delivers fair and safe competition.*" We concluded that, *"[T]he muscular advantage*

[9] For example, Hilton et al. 2009. BCOR analysis in patients with OFCD and Lenz microphthalmia syndromes, mental retardation with ocular anomalies, and cardiac laterality defects. Eur J Hum Genet 17: 1325–1335.
[10] Hilton et al., 2021. The reality of sex. Ir J Med Sci 190: 1647.
[11] Hilton and Wright, 2023. "Two sexes." In Sex and Gender: A Contemporary Reader. Routledge, Oxford, UK.
[12] Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51: 199–214.

enjoyed by transgender women is only minimally reduced when testosterone is suppressed."

1.11. In terms of impact (26th June 2023), we published our review in Sports Medicine, an international leader in sports and exercise medicine research, with a five-year impact factor of 13.671.[13] Our Altmetric score is 5471, and our review is ranked 662 out of 23.9 million academic articles published across all fields.[14] It has already been cited 65 times in the academic literature,[14] and also in scientific media including Nature.[15] Hilton and Lundberg (2021) has been cited in the transgender athlete policies of British Triathlon,[16] British Cycling[17] and World Rugby[18] (which was used to formulate the transgender policies of England Rugby, Scottish Rugby and Welsh Rugby), and cited in the scientific reviews underpinning the policies of Union Cycliste Internationale[19] and World Athletics.[20] It was also cited by the UK Sports Council Equality Group in their influential policy document that highlighted the clash between fairness for female athletes and inclusion of transgender women athletes.[21] In 2022, Hilton and Lundberg (2021) was cited in the US Court of Appeals for the 11th Circuit, by Justice Lagoa in her specially concurring opinion in Adams .v. School Board of St. Johns County, Florida.[22] Also in 2022, we were cited in a literature review on transgender athletes, published by the UK Parliamentary Office of Science and Technology, intended to brief UK Members of Parliament on topical issues.[23] Finally, Hilton and Lundberg (2021) is cited in the findings of the Fifty-fifth Legislature of the State of Arizona in Senate Bill 1165 (SB1165; the legislation relevant to this case).

1.12. In 2021, I was invited to author a policy review by the Canadian Macdonald-Laurier Institute.[24] This policy document is a review of the individual authors' peer-reviewed publications and expert knowledge; it was not itself peer-reviewed by the academic community. In this policy document, we review the importance of sex categories in sport, synthesising knowledge across developmental biology, the physiology of transgender women, and sports philosophy. We conclude that a female category that excludes all males, regardless of gender identity, is philosophically coherent in terms of category definition and necessary to ensure everyone can compete fairly and fully. We argue it is reasonable for female athletes to expect that their rights will be upheld by the institutions and procedures of their sports.

1.13. I have been asked to consult with various UK and international sporting bodies seeking advice on policy formation. Many such meetings have been held under conditions of anonymity. In February 2020, I was invited, alongside world experts in transgender endocrinology, sports science and ethics, by World Rugby to give evidence to the

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[13] https://www.springer.com/journal/40279
[14] https://link.altmetric.com/details/95647691
[15] Photopoulos, 2021. The future of sex in elite sport. Nature 592: S12-15.
[16] https://www.britishtriathlon.org/britain/documents/about/edi/transgender-policy-effective-from-01-jan-2023.pdf
[17] https://www.britishcycling.org.uk/zuvvi/media/Transgender_and_Non-Binary_Policy_-_FAQs.pdf
[18] https://www.world.rugby/the-game/player-welfare/guidelines/transgender/faqs
[19] https://assets.ctfassets.net/761l7gh5x5an/4gHOE5EpVItQux9kf39XYC/5c52616af086bdf2c9731679f213c1cd/The_current_knowledge_on_the_effects_of_gender-affirming_treatment_on_the_markers_of_performance_in_transgender_female_cycli.pdf
[20] Not publicly available.
[21] https://www.uksport.gov.uk/news/2021/09/30/transgender-inclusion-in-domestic-sport; Sports Council Equality Group Guidance for Transgender Inclusion in Domestic Sport, 2021.
[22] https://aboutblaw.com/6fe
[23] https://researchbriefings.files.parliament.uk/documents/POST-PN-0683/POST-PN-0683.pdf
[24] Pike, Hilton and Howe, 2021. Fair Game: Biology, Fairness and Transgender Athletes in Women's Sport. Macdonald-Laurier Institute, Canada.

Transgender Working Group, which was tasked with reviewing their regulations for inclusion of transgender women in female categories in elite international competition.[25] After an extensive, 'mock courtroom/adversarial' consultation process, World Rugby determined that female categories can only be safe and fair if males, regardless of gender identity, are excluded from female categories. During 2021, I was consulted as part of a policy project by the UK Sports Council Equality Group.[26] In July 2022, I was invited to present to the Equality, Diversity and Inclusion Commission of World Triathlon, who subsequently tightened restrictions on transgender women athletes in the female category.[27]

1.14. In December 2021, I participated in an online academic seminar hosted by Sports Resolutions, alongside David Grevemberg, the managing director of the Commonwealth Games Federation.[28] In April 2022, I was invited to speak at the Canadian Academy of Sport and Exercise Medicine 2022 Annual Conference, on the topic of transgender athletes, fairness and eligibility.[29] In November 2022, I was invited to speak at the Royal Academy of Medicine (UK), alongside Richard Budgett, the medical director of the International Olympic Committee.[30] In March 2023, I was invited to speak at the 19th World Congress of the International Academy of Human Reproduction, on the topic of transgender athletes in sports.[31]

1.15. Beyond academic activities, I am a vocal advocate for fairness in female sport, and have presented my research findings and arguments in various formats. In January 2021, I was appointed as a board member of Sex Matters, a UK-based human rights group who lobby for clarity on the protected characteristic of sex in law and in institutions.[32] Examples of my outputs for Sex Matters include formal responses to sports policy consultations.[33] I offer advice and input to other resources produced by employees. I vote on board-level decisions regarding strategy, expenditure, employment decisions and other typical administrative duties. My position with Sex Matters is unpaid and my work is voluntary. I receive compensation for travel, food and accommodation at meetings and events.

1.16. Other examples of advocacy include the first presentation of my research findings and arguments in July 2019 at an event organised by two feminist groups, A Woman's Place UK and FairPlay For Women.[34] In this presentation, I mapped the timeline of policy development by the International Olympic Committee (IOC) with the concurrent scientific data. I was—and remain—strongly critical of the IOC policy development trajectory. In April 2022, I was invited to speak at a private meeting at the UK House of Lords (for which I was compensated for travel costs), and wrote a house-wide briefing pack. I have been invited to consult with athlete groups like the US-based Women's Sports Policy

[25] https://www.world.rugby/news/563437/landmark-world-rugby-transgender-workshop-important-step-towards-appropriate-rugby-specific-policy; World Rugby Transgender Guidelines, 2020.
[26] https://www.uksport.gov.uk/news/2021/09/30/transgender-inclusion-in-domestic-sport; Sports Council Equality Group Guidance for Transgender Inclusion in Domestic Sport, 2021.
[27] https://www.triathlon.org/news/article/transgender_policy_process
[28] https://www.youtube.com/watch?v=TbE9aEo8ypA
[29] https://casem-acmse.org/wp-content/uploads/2020/02/ENG_CASEM-AQMSE-Quebec-2022-CASEM-AQMSE-1.pdf
[30] https://www.mededucare.com/_files/ugd/70d91e_b49fb63fc9574bac9ce9c34bfac298a9.pdf
[31] https://hr2023.humanrepacademy.org/scientific-program/
[32] https://sex-matters.org/about/emma-hilton-phd/
[33] For example: https://sex-matters.org/wp-content/uploads/2021/05/Sex-Matters-British-Cycling-policy-response.pdf
[34] https://www.youtube.com/watch?v=pzg9QtQeIR8

Working Group[35] and the Independent Council on Women's Sport (ICONS).[36] For the latter, I presented at their inaugural event in Las Vegas in June 2022, and I am due to present again in Denver in July 2023. I received compensation for travel, food and accommodation at the inaugural ICONS event.

1.17. I have been interviewed in the UK media on several occasions, including on BBC Radio 4 and BBC Radio 5 Live Sport. I have published opinion pieces in the mainstream media, including the Wall Street Journal (on the harms arising from denial of the biological reality of sex).[37] Most recently, I wrote with Professor David Handelsman, an international expert in the pharmacology of androgens and expert witness for World Athletics.[38]

1.18. I have been asked by the legal team for the Arizona Superintendent of Public Instruction to provide my expert scientific opinion on the need for a protected female sports category, and the loss of fairness for female athletes arising from the inclusion of transgender girls and transgender women in competitive school sports. In preparation for this case, I have read Senate Bill 1165 (SB1165). My understanding of SB1165 is that sports teams within public schools (or in schools engaged in competitive sports against public schools) will be designated by sex as male or female, or designated as mixed-sex. Female-designated teams will exclude male athletes. An effect of SB1165 is the exclusion of transgender girls from teams designated as female-only. I understand that transgender girls are free to participate in male-designated and mixed-sex teams.

1.19. I am currently retained to provide expert scientific opinion for the State of Indiana and the State of Utah. There is no conflict of interest to declare.

1.20. The opinions put forward in this statement are my own, grounded in my education and scientific expertise, and do not necessarily reflect those of my employer, the University of Manchester, UK. I will make no personal, social, sporting or academic gains from the opinion I present here.

1.21. I am being compensated for my time researching and preparing this report at a rate of $400 USD per hour. I will be compensated for deposition at a rate of $450 USD per hour. My compensation does not depend on the outcome of this litigation.

[35] https://womenssportspolicy.org/
[36] https://www.iconswomen.com
[37] https://www.wsj.com/articles/the-dangerous-denial-of-sex-11581638089
[38] https://amp.theaustralian.com.au/sport/what-science-tells-us-about-transgender-women-athletes/news-story/cb8b7a30f68745a3fa65442b7ff15694

2. Summary of expert witness statement

2.1. Male development, driven by both genetics and hormones, delivers structural differences (compared with females) from as early as first trimester gestation. Physical differences between males and females that matter for athletic sports are detectable in utero, during childhood, and then cemented during puberty.

2.2. Male athletic advantage over female peers in adolescence and adulthood is undisputed. In childhood, male athletic advantage over female peers is evident across track and field events from 8 years old onwards Males systematically outperform their female peers from 8 years old at a frequency that is vanishingly unlikely to result by chance.

2.3. Protected female sports categories are justified to protect fairness (and, discipline-dependent, safety) for female athletes, who, by virtue of typical female development, do not benefit from male development and thus male athletic advantage. This includes protected categories for young female athletes.

2.4. The suppression of testosterone post-puberty in transgender women does not appear to affect skeletal proportions and reduces acquired muscle mass by only a modest amount. The sparse evidence regarding musculoskeletal metrics in transgender girls who have blocked or partially-blocked puberty reveals metrics like height far exceeding those of typical females.

2.5. It is my professional opinion that the State of Arizona is justified in protecting fairness for female athletes in interscholastic sports competition by restricting from those female categories transgender girls and transgender women, because those individuals will have acquired male athletic advantage by virtue of biological development, and acquisition of male athletic advantage is not entirely removed by either puberty blockers and/or testosterone suppression post-puberty.

3. Sex and gender identity

3.1. Sex is an evolved system function common to almost all complex life on earth. Across the natural world, the words “male” and “female” pertain to the two specific reproductive functions within a system of sexual reproduction that proceeds via two differently-specialised gamete types. They are words used to describe cells, tissues, organs and/or entire individuals that have a physical role in the contribution of small gametes (like sperm) or large gametes (like ova), respectively, to the next generation. “Male” and “female” describe the biology of reproduction and I use these words as neutral descriptions of reproductive biology.

3.2. In humans (and indeed, in almost all animals and many plants), the two reproductive functions are divided between two classes of individual, with each class possessing a distinct and specialised molecular and anatomical pattern corresponding to one of the two reproductive functions. In humans, there are two sexes.

3.3. During embryonic development in utero, males and females develop sex-specific primary sex characteristics that have evolved to facilitate function during future reproduction. In humans, healthy male anatomy comprises gonads in the form of external testes (also called testicles) that will make sperm, internal genital structures like the vas deferens (that carries sperm from the testicles to penis) and external genitalia in the form of a penis and scrotum. In contrast, healthy female anatomy comprises gonads in the form of internal ovaries that will make eggs, internal genital structures like a uterus and vagina, and external genitalia in the form of a vulva, incorporating the clitoris.

3.4. The various parts of the reproductive anatomy of a healthy baby (gonad type, internal genitalia, external genitalia) develop as a system in a regulated and coordinated sequence of events. The sex of a baby is routinely and reliably learned or observed—not “assigned”, which implies an element of choice or arbitrariness—at birth by visual and palpable[39] assessment of external genitalia, which is a highly-sensitive marker for the whole system.

3.5. The above descriptions of primary sex are standard, appearing in dictionaries,[40] key biology textbooks,[41] academic publications[42] and medical consensus statements like that issued by the Endocrine Society in 2021.[43] By these standard descriptions of sex, transgender girls and transgender women are biologically male and not biologically female.

3.6. Transgender girls and transgender women feel deep distress and discomfort with their male sex (“gender dysphoria”) and claim a sense of identification with the female sex (via “gender identity”). The assertion that “*everyone has a gender identity*” (Shumer declaration, 18) is contradicted by the personal testimonies of people, including myself, who do not experience a gender identity and the delineation of the concept of ‘agender’, which describes “*identifying as having no gender*” (quoted from Shumer declaration in Flack et al. .v. Wisconsin Department Of Health Services).[44] It appears incoherent to

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[39] “Palpable” means, roughly, “detect by touching”. This assessment is typically used to confirm the healthy descent of testes in male babies.

[40] Examples include: Oxford English Dictionary; Merriam-Webster Dictionary.

[41] Examples include: Baresi and Gilbert, 2020. Developmental Biology. Oxford University Press, UK; Wolpert, Tickle and Martinez Arias. Principles of Development. Oxford University Press, UK.

[42] Academic publications defining sex, actively researching sex or incidentally dependent on these understandings of sex are too numerous to consider. For example, a search on the scientific publication database PubMed for only “male [AND] sperm” (that is, not an exhaustive search) retrieves over 100,000 results, including multiple results from Nobel Laureates in Physiology or Medicine, and from a huge array of biology and medical disciplines.

[43] Barghava et al., 2021. Considering Sex as a Biological Variable in Basic and Clinical Studies: An Endocrine Society Scientific Statement. Endocrine Reviews, 42(3): 219-258.

[44] http://files.eqcf.org/wp-content/uploads/2019/04/170-Shumer-Expert-Witness-Report.pdf

argue that everyone has a gender identity while recognising the existence of being 'agender'.

3.7. I am scientifically-neutral to the possibility that "*gender identity has a strong biological basis*" (Shumer declaration, 19 and 22). I do not consider gender identity to be a component of sex, which denotes one's physical reproductive development and reproductive role. Even if it is true that gender identity is in some way biological in basis, gender identity is irrelevant to eligibility for sporting categories based on sex. The premise that, in transgender people, sex "*designation turns out to be inaccurate because it does not reflect the person's gender identity*" (Shumer declaration, 27) creates a contradiction where gender identity is asserted as a feature of sex (Shumer declaration, 26) yet is an identity that exists by reference to one's sex (Shumer declaration, 25, decouples gender identity from "*birth sex*").

3.8. Disorders of sex development (DSDs), where the development of reproductive anatomy is atypical or disrupted,[45] are very rare[46] but frequently used to argue that sex in humans cannot be described as simply male and female. While it is true that, rarely even within DSDs, the sex of some individuals is difficult to classify, this is irrelevant when considering the sex of transgender people, who do not typically have DSDs.

[45] For example: Arboleda et al., 2014. DSDs: genetics, underlying pathologies and psychosexual differentiation. Nature Reviews Endocrinology 10(10): 603-615.
[46] Sax, 2002. How common is Intersex? A response to Anne Fausto-Sterling. Journal of Sex Research 39 (3): 174-178.

4. Sex and somatic growth

4.1. Beyond differences in reproductive anatomy, males and females differ in somatic (non-reproductive) physical characteristics. Somatic differences first emerge in utero, are evident at birth, and are further cemented during puberty.

4.2. Small differences in average body length (measured as head-bottom length) can be detected by ultrasound from the first trimester of pregnancy, when males are already slightly longer than females.[47] Larger average skull diameter in male fetuses at twenty weeks has been reported.[48] Gestational growth charts track not just higher male values for skull diameter but also higher abdominal circumference and estimated fetal weight.[49] Analysis of growth charts[50] for male and female infants reveals that, at birth, males are, on average, slightly longer and heavier than females.

4.3. In a large study of male and female fetuses and newborns, Broer-Brown et al (2016) concluded that, "Sex affects both fetal as well as infant growth. Besides body size, also body proportions differ between males and females with different growth patterns."[51] Although the magnitude of size differences in utero and at birth are small, they are consistently-different between males and females; indeed, sex is considered necessary to clinically assess fetal growth with accuracy.[52]

4.4. Males are consistently 1-2 cm taller than females between 0-10 years old. Boys at 10 years old also have a larger vertebral cross-sectional area (larger spinal columns) than girls.[53] Girls enter puberty earlier than boys, typically around 10 years old, and the growth spurt associated with earlier pubertal onset accounts for taller female height between 10-14 years old. Boys catch up and overtake girls in height at around 14 years old.

4.5. At puberty, both sexes undergo rapid somatic changes as they mature in preparation for reproduction, leading to measurably different adult body shapes ('sexual dimorphism').[54] Many male secondary sex characteristics are rooted in our evolutionary history of male fighting ability, displays of strength and competition for mates[55] and become increasingly evident as puberty progresses.

4.6. When—briefly—considering sexually-dimorphic physical characteristics in males compared with females, adolescent and adult males are typically taller with wider shoulders, longer limbs and longer digits. They have larger and denser muscle mass, reduced fat mass, different distributions of muscle and fat, and stiffer connective tissue.

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[47] Pedersen, 1980. Ultrasound evidence of sexual difference in fetal size in first trimester. British Medical Journal 281(6250): 1253.

[48] Persson et al., 1978. Impact of fetal and maternal factors on the normal growth of the biparietal diameter. Scandinavian Association of Obstetricians and Gynaecologists 78: 21-27.

[49] Schwartzler et al., 2004. Sex-specific antenatal reference growth charts for uncomplicated singleton pregnancies at 15–40 weeks of gestation. Ultrasound in Obstetrics and Gynaecology 23(1): 23-29.

[50] For example: World Health Organisation https://www.who.int/tools/child-growth-standards/standards; Centre for Disease Control https://www.cdc.gov/growthcharts/clinical_charts.htm; Royal College of Paediatrics and Child Health https://www.rcpch.ac.uk/resources/growth-charts

[51] Broere-Brown et al, 2016. Sex-specific differences in fetal and infant growth patterns: a prospective population-based cohort study. Biology of Sex Differences 7: 65.

[52] Galjaard et al., 2019. Sex differences in fetal growth and immediate birth outcomes in a low-risk Caucasian population. Biology of Sex Differences 10: 48.

[53] Gilsanz et al., 1997. Differential Effect of Gender on the Sizes of the Bones in the Axial and Appendicular Skeletons. Journal of Clinical Endocrinology and Metabolism 82(5): 1603-1607.

[54] For example: Darwin, C. *The Descent of Man, and Selection in Relation to Sex*. London: Murray, 1871; Well, 2007. Sexual dimorphism of body composition. Best Practice and Research Clinical Endocrinology and Metabolism 21(3): 415-430.

[55] For example: Morris et al., 2020. Sexual dimorphism in human arm power and force: implications for sexual selection on fighting ability. Journal Of Experimental Biology 223(2): 212365; Puts, 2010. Beauty and the beast: mechanisms of sexual selection in humans. Evolution And Human Behaviour 31(3): 157-175.

They have higher amounts of haemoglobin (the molecule that carries oxygen in blood), and larger hearts and lungs.[56]

4.7. The above is a non-exhaustive list of sexually-dimorphic differences between males and females, which could number into the thousands, and include, for example, the fine architecture of muscle tissue like proportions of cell type (fibre type, stem cell populations), cell morphology (numbers of nuclei, amounts of myoglobin) and some 3000 muscle-specific gene expression differences,[57] to the minutiae of different visual perception, hand-eye coordination and tracking capacity.[58]

[56] Reviewed in: Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51, 199–214 (and references therein).
[57] Haizlip et al., 2014. Sex-Based Differences in Skeletal Muscle Kinetics and Fiber-Type Composition. Physiology (30)1: 30-39.
[58] For example: Mathew et al., 2020. Sex differences in visuomotor tracking. Scientific Reports 10: 11863.

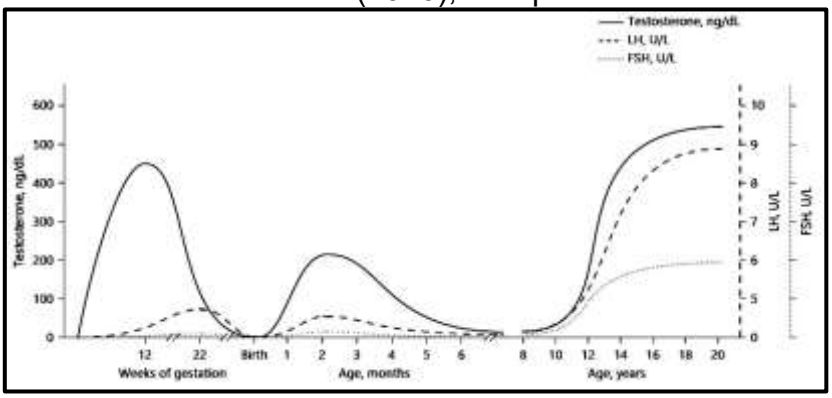
5. Genetics, hormones and development

5.1. Sex differentiation is initiated in utero by the presence or absence of a gene called SRY, typically carried on the Y chromosome, and triggering bipotential gonad development into testes or ovaries in males or females, respectively.[59] The developing gonads, in conjunction with other tissues, establish sex-specific hormonal milieu that, in concert with hormones produced elsewhere, are involved in ongoing male or female physical development.[60]

5.2. It is often assumed that hormones are the driver of all physical sex differences downstream of gonad differentiation.[61] However, analysis of sex-specific genetic architecture in adults reveals some 6500 differences in gene expression, likely to influence development and function outside of hormone effects.[62] Indeed, that "every cell has a sex" dependent on genetics and independent of hormones is recognised and increasingly of scientific interest.[63] REF IOC paper analysis

5.3. A key hormone generating physical differences between males and females is testosterone. Males are exposed to testosterone at three stages of development: 1. in utero; 2. in the post-natal 'minipuberty' period; and, 3. during classic puberty (Figure 1, solid line[64]). Thus, there is an ongoing pattern of differential exposure to testosterone during the development of males and females.

Figure 1. "The three endocrine puberties in boys."
From Becker and Hesse (2020), with permission from S. Karger AG, Basel, CHE



5.4. In utero, testosterone and derived dihydrotestosterone (DHT) are involved in the development of male reproductive anatomy. Testosterone is primarily produced by the male testes.[65] Testosterone promotes the formation of the vas deferens and other male internal genital structures, while DHT is necessary for the development of the penis and prostate gland.[66] The effect of testosterone on somatic development in utero does not appear to be meaningful, and sex differences in fetal size appear unrelated to hormones

[59] Sekido and Lovell-Badge, 2013. Genetic control of testis development. Sexual Development 7:21-32.
[60] Nussey and Whitehead, 2001. Endocrinology: An Integrated Approach. BIOS Scientific Publishers, Oxford, UK.
[61] Lovell-Badge, 1993. Sex determining gene expression during embryogenesis. Philosophical Transactions of The Royal Society (Biological Sciences) 339: 159-164.
[62] Gershoni and Pietrokovski, 2017. The landscape of sex-differential transcriptome and its consequent selection in human adults. BMC Biology 15(1): 7.
[63] For example: Shah et al., 2014. Do you know the sex of your cells? American Journal of Physiology (Cell Physiology) 306(1): C3-C18; Ainsworth, 2017. Sex and the single cell. Nature 550: S6-S8.
[64] Becker and Hesse, 2020. Minipuberty: Why Does it Happen? Hormone Research in Paediatrics 93(2): 76-84.
[65] Richmond and Rogol, 2007. Male pubertal development and the role of androgen therapy. Nature Clinical Practice Endocrinology and Metabolism 3(4): 338-344.
[66] Theakston, 2020. Development of the Reproductive System https://teachmeanatomy.info/the-basics/embryology/reproductive-system

but related rather to the sex-specific genetics of maternal-placental interactions with a male fetus, which affect, for example, nutrient exchange.[67]

5.5 In the post-natal minipuberty period between 1 week to 6 months of age, transient activation of the hypothalamic-pituitary-gonadal axis means males are exposed to a corresponding burst of testosterone.[68] This burst of testosterone supports male penis and testes growth,[69] and is associated with higher growth velocity in the first six months of life,[70] higher weight gain, lower acquisition of body fat and lower body mass index.[71] The transient exposure to testosterone in minipuberty is an excellent candidate to explain the well-established structural differences between males and females in childhood described in **Section 4**.

5.6 At puberty, males experience levels of testosterone up to 20 times greater than in females, driving development during the ensuing teenage years of male secondary sex characteristics.[72] The effects of testosterone on male somatic growth during puberty are well-characterised and hardly require repeating here.[73]

[67] Buckberry et al., 2014. Integrative transcriptome meta-analysis reveals widespread sex-biased gene expression at the human fetal–maternal interface. Molecular Human Reproduction 20(8): 810-819.
[68] Lanciotti et al., 2018. Up-To-Date Review About Minipuberty and Overview on Hypothalamic-Pituitary-Gonadal Axis Activation in Fetal and Neonatal Life. Frontiers in Endocrinology 9: 410.
[69] Boas et al., 2006. Postnatal penile length and growth rate correlate to serum testosterone levels: a longitudinal study of 1962 normal boys. European Journal of Endocrinology 154(1): 125-129.
[70] Kiviranta et al., 2016. Transient Postnatal Gonadal Activation and Growth Velocity in Infancy. Pediatrics 138(1): e20153561.
[71] Becker et al., 2015. Hormonal 'minipuberty' influences the somatic development of boys but not of girls up to the age of 6 years. Clinical Endocrinology 83: 694-701.
[72] Handelsman et al., 2018. Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance. Endocrine Reviews 39(5): 803-829.
[73] Reviewed in, for example: Hiort, 2002. Androgens and puberty. Best Practice and Research Clinical Endocrinology and Metabolism 16(1): 31-41; Richmond and Rogol, 2007. Male pubertal development and the role of androgen therapy. Nature Clinical Practice Endocrinology and Metabolism 3(4): 338-344.

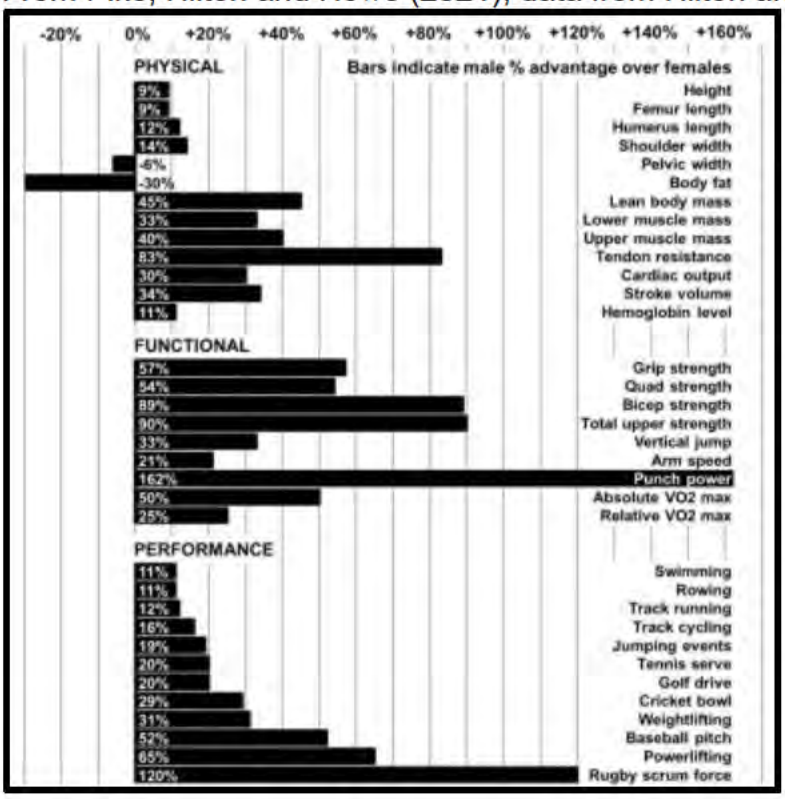
6 Sex and sporting advantage in adolescence and adulthood

6.1 In most athletic sports—those where outcome is affected by speed, stamina, strength and physique—males have a class-level advantage over females. Male advantage is founded in the physical differences, acquired during male development, that underpin functional differences in muscular strength, skeletal levers and proportions, force application, upper to lower body strength, and cardiovascular and respiratory function. In turn, these functional differences confer superior athleticism.[74]

6.2 Examination of sporting records and performances identifies few athletic sporting disciplines where males do not possess performance advantage over females[75], and competitions are typically separated by sex. Volleyball, basketball, soccer and cross-country running are among those where male development provides competitive advantage, and where competitions are therefore separated by sex.

6.3 The physical, functional and performance advantages in adult males are summarised in Figure 2, using reported record performances across multiple sports and sporting actions. Male strength is disproportionately large in the upper body, and sports and sporting movements that require upper body input typically exhibit larger performance gaps than that where lower body strength is key. Performance differences, emerging from the physical and functional differences between adult males and females, have been described as "insurmountable".[76]

Figure 2. Physical, functional and performance differences between males and females.
From Pike, Hilton and Howe (2021); data from Hilton and Lundberg (2021)



[74] For example: Tonnessen et al., 2015. Performance development in adolescent track and field athletes according to age, sex and sport discipline. PLOS One 10(6): e0129014.
[75] For example: Olympic performances https://olympics.com/en/olympic-games/olympic-results; track and field performances https://www.worldathletics.org/stats-zone
[76] Thibault et al., 2010. Women and Men in Sport Performance: The Gender Gap has not Evolved since 1983. Journal of Sports Science and Medicine 9(2): 214-223.

6.4 The significance of male puberty is evidenced by the fact that male performances typically exceed those of elite females in mid-puberty; a comparison of elite female records with male junior records[77] is listed in Table 1. Unsurprisingly, in events like the marathon that are associated with greater strategy and maturity, males are older when they surpass elite female records.

Table 1. Elite female records are surpassed by males in mid-puberty.
Abbreviations: m – metres, km – kilometres, s – seconds, m – minutes, h – hours, yrs – years old

Event	Elite female record	Age at which male records surpass elite female records
100 m	10.49 s	15 yrs (10.20 s)
200 m	21.34 s	14 yrs (20.89 s)
400 m	47.60 s	14 yrs (46.96 s)
800 m	1 m:53.28 s	14 yrs (1 m:51.23 s)
1500 m	3 m:50.07 s	14 yrs (3 m:48.37 s)
5km	14 m:06.62 s	15 yrs (14 m:06.51 s)
10km	29 m:01.03 s	16 yrs (28 m:39.04 s)
Marathon	2 h:17 m:01 s	19 yrs (2 h:11 m:34 s)
High jump	209 cm	14 yrs (217 cm)
Pole vault	506 cm	15 yrs (550 cm)
Long jump	752 cm	15 yrs (785 cm)
Triple jump	1574 cm	15 yrs (1663 cm)
Shot put	2263 cm (4 kg shot)	15 yrs (2386 cm; 5 kg shot)
Discus	7680 cm	15 yrs (7768 cm)
Hammer	8298 cm	14 yrs (8517 cm)
Javelin	7228 cm	14 yrs (7642 cm)

6.5 Importantly, male athletic advantage over females is not limited to those physical and functional differences conferred by male morphology, shape and size. Most obviously, female athletes must typically deal with the effects of the menstrual cycle and the cyclical effects of hormones on training capacity and performance. The menstrual cycle is known to affect cardiovascular, respiratory, brain function, response to ergogenic aids, orthopedics, and metabolic parameters,[78] and represents a barrier to athletic capacity not experienced by males. A third of females report their menstrual flow to be "above average" volume.[79] 37 % of female athletes report heavy menstrual flow, and 90 % report menstrual symptoms, affecting their ability to train and compete.[80]

6.6 Further, injury susceptibility differs between males and females, with subsequent impacts on training time. For example, emerging research shows that compared with males, female rugby players appear more susceptible to concussive injuries, with more severe outcomes. This has been attributed to lower impact resistance in their neck

[77] http://age-records.125mb.com; https://worldathletics.org/records/by-category/world-records
[78] Meignie et al., 2021. The Effects of Menstrual Cycle Phase on Elite Athlete Performance: A Critical and Systematic Review. Frontiers in Physiology 12: 654585.
[79] Bitzer et al., 2013. Women's attitudes towards heavy menstrual bleeding, and their impact on quality of life. Open Access Journal of Contraception 4: 21-8.
[80] Bruinvels et al., 2021. Prevalence and frequency of menstrual cycle symptoms are associated with availability to train and compete: a study of 6812 exercising women recruited using the Strava exercise app. British Journal of Sports Medicine 55: 438-443.

muscles and more delicate brain structures.[81] A study of sex differences in cultured nerve cells has shown that, compared with male neurons, female neurons have a smaller cross-section and contain fewer, less-dense structural “fibres”; female neurons are more easily damaged when subject to stretch trauma, and they exhibit higher injury responses post-trauma.[82] Female athletes have a higher incidence of anterior cruciate ligament injury than males and poorer response to injury-prevention programmes, well-studied in soccer and typically attributed to female lower body anatomy (hip width, muscle ratio, joint flexibility).[83]

[81] www.rugbypass.com/news/long-term-brain-damage-could-be-a-significantly-bigger-issue-in-womens-rugby-than-mens-says-lead-concussion-doctor/
[82] Dollé et al., 2018. Newfound sex differences in axonal structure underlie differential outcomes from in vitro traumatic axonal injury. Exp Neurol 300:121-134.
[83] Crossley et al., 2020. Making football safer for women: a systematic review and meta-analysis of injury prevention programmes in 11 773 female football (soccer) players. British Journal of Sports Medicine 54: 1089-1098.

7 Sex and sporting advantage in childhood

7.1 While few deny the athletic sporting differences between males and females in adolescence and adulthood, sporting performance gaps between the sexes before puberty are less well-characterised.

7.2 In **Section 4**, I outlined known physical differences between males and females in utero and during childhood. At the level of function leading to athletic performance, large cohort studies of fitness data in typical schoolchildren reveals differences evident from as young as 6 years old. In these childhood fitness programs, females consistently outperform males in the sit and reach test, a measure of flexibility. However, males can run 9.8 % faster over short sprints, jump 9.5 % further from a standing start, complete 33 % more push ups in 30 seconds, complete 16.6 % more shuttle runs in a given time and have 13. 8 % higher grip strength.[84] Young males of 6-7 years old have higher absolute (+11 %) and relative (+8 %) VO_{2max} than female peers.[85]

7.3 The Presidential Fitness Test was a US fitness testing program conducted in middle school and high schools until 2013. Awards were given to schoolchildren in the top 15th percentile in their cohort. I calculated the % difference between the top 15th percentile in male and female schoolchildren aged 6-16 years old, listed in Table 2.[86]

Table 2. Male advantage (%) at the top 15th percentile in the US Presidential Fitness Test for schoolchildren.

Abbreviations: yrs – years old, n – number, s – seconds, cm - centimetres

Age	Curl ups n	Shuttle run s	Sit and reach cm	1 mile s	Pull ups n
6 yrs	3.1	2.4	-36.4	9.6	0.0
7 yrs	5.9	5.0	-30.0	11.6	100.0
8 yrs	5.3	5.9	-33.3	12.3	150.0
9 yrs	5.1	1.8	-45.5	10.4	150.0
10 yrs	12.5	4.6	-33.3	14.7	100.0
11 yrs	11.9	4.8	-38.5	16.6	100.0
12 yrs	11.1	5.8	-42.9	14.3	250.0
13 yrs	15.2	6.9	-50.0	16.8	250.0
14 yrs	19.1	9.9	-43.8	19.4	400.0
15 yrs	18.8	10.0	-37.5	22.1	450.0
16 yrs	24.4	13.9	-33.3	26.8	1000.0

7.4 Thus, physical performance differences among schoolchildren are detectable and measurable in school fitness testing programmes. To begin to systematically analyse pre-puberty and early pubertal differences in sports performance between males and females, I interrogated the extensive track and field performance data available in young people. Track and field events comprise the simple "building blocks'—running, jumping and throwing—that are key to athletic performance in many individual and team sports, including volleyball, soccer and basketball. Thus, track and field event performances can be used to understand likely performance differences in more complex sports.

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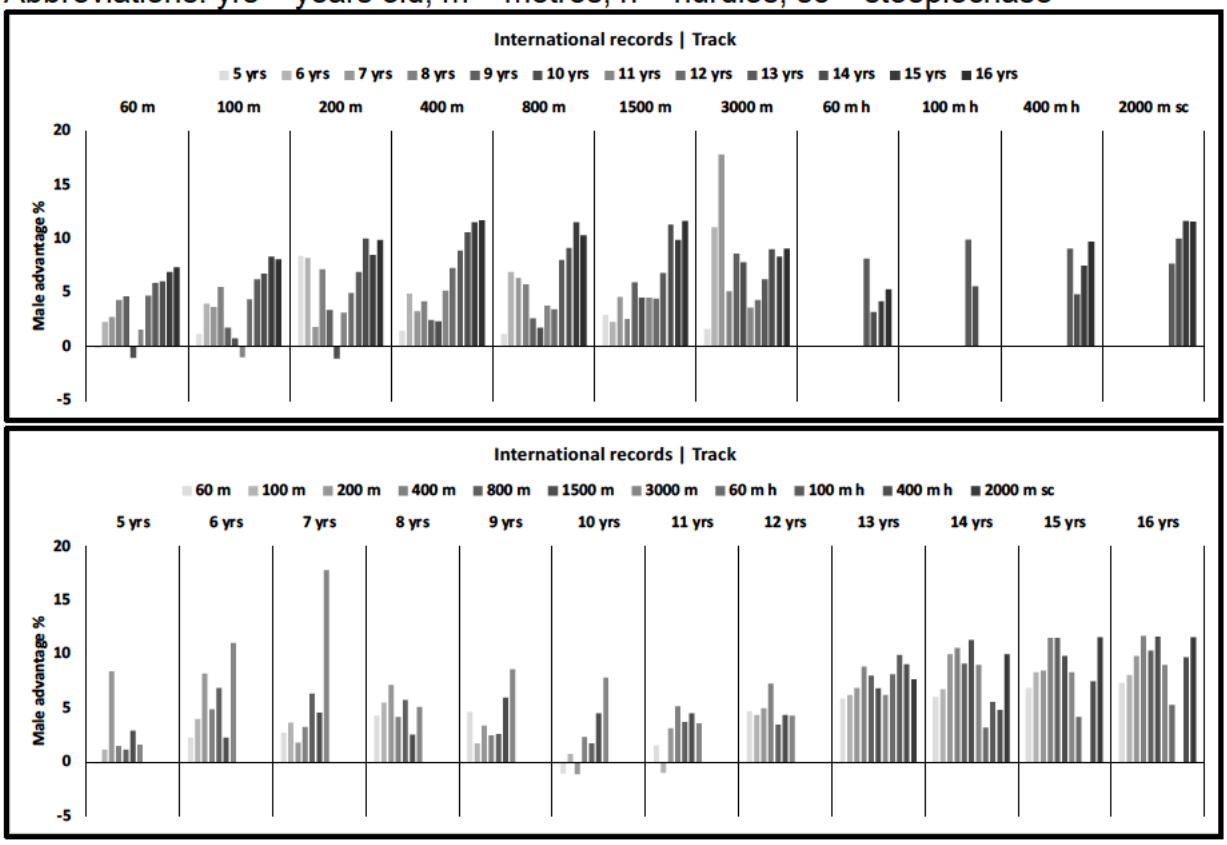
[84] For example: Catley and Tomkinson, 2013. Normative health-related fitness values for children: analysis of 85347 test results on 9–17-year-old Australians since 1985. British Journal of Sports Medicine 47(2): 98–108; Tambalis et al., 2016. Physical fitness normative values for 6–18-year-old Greek boys and girls, using the empirical distribution and the lambda, mu, and sigma statistical method. European Journal of Sport Science 16(6): 736-746.

[85] Eiberg et al., 2005. Maximum oxygen uptake and objectively measured physical activity in Danish children 6–7 years of age: the Copenhagen school child intervention study. British Journal of Sports Medicine 39(10): 725-730.

[86] https://gilmore.gvsd.us/documents/Info/Forms/Teacher%20Forms/Presidentialchallengetest.pdf

7.5 I collected international records in multiple track and field events from both males and females from the ages of 5-16 years old.[87] I then calculated the % difference between the male record and equivalent female record. The male advantages (%) in track, stratified by both event (upper panel) and age (lower panel), are shown in Figure 3. In track events, male advantage is clear in all age groups and for all events.

Figure 3. The male advantage over females in international schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel). Abbreviations: yrs – years old, m – metres, h – hurdles, sc – steeplechase



7.6 There are four track events where female schoolchildren appear to outperform their male peers, listed in Table 3. I examined the age progression of these events to seek to understand this apparent female advantage. These data are shown in Figure 4. For 60 m at 5 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. For 60 m at 10 years old, the male record appears slightly slower than predicted, with no specific explanation for this beyond typical variation. In this same event, the female record is faster than expected, possibly explained by earlier onset of puberty and associated growth spurt that provides transient 'catch up' with male peers. For 100 m at 11 years old and 200 m at 10 years old, again the female records appear faster than expected, again likely underpinned by pubertal growth spurt in these female athletes.

[87] International age records http://age-records.125mb.com

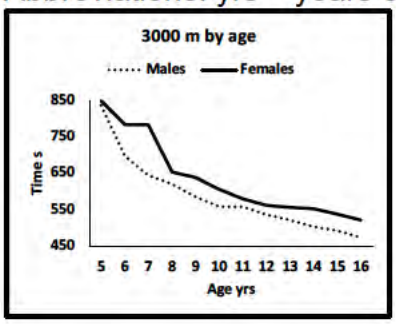
Table 3. Female advantage in international schoolchildren track records.
Abbreviations: yrs – years old, m – metres

Event	Age group	Female advantage %
60 m	5 yrs	0.1 %
	10 yrs	1.0 %
100 m	11 yrs	0.9 %
200 m	10 yrs	1.1 %

Figure 4. Age progression in the 60 m, 100 m and 200 m sprints in international schoolchildren records.
Abbreviations: yrs – years old, m – metres, s – seconds

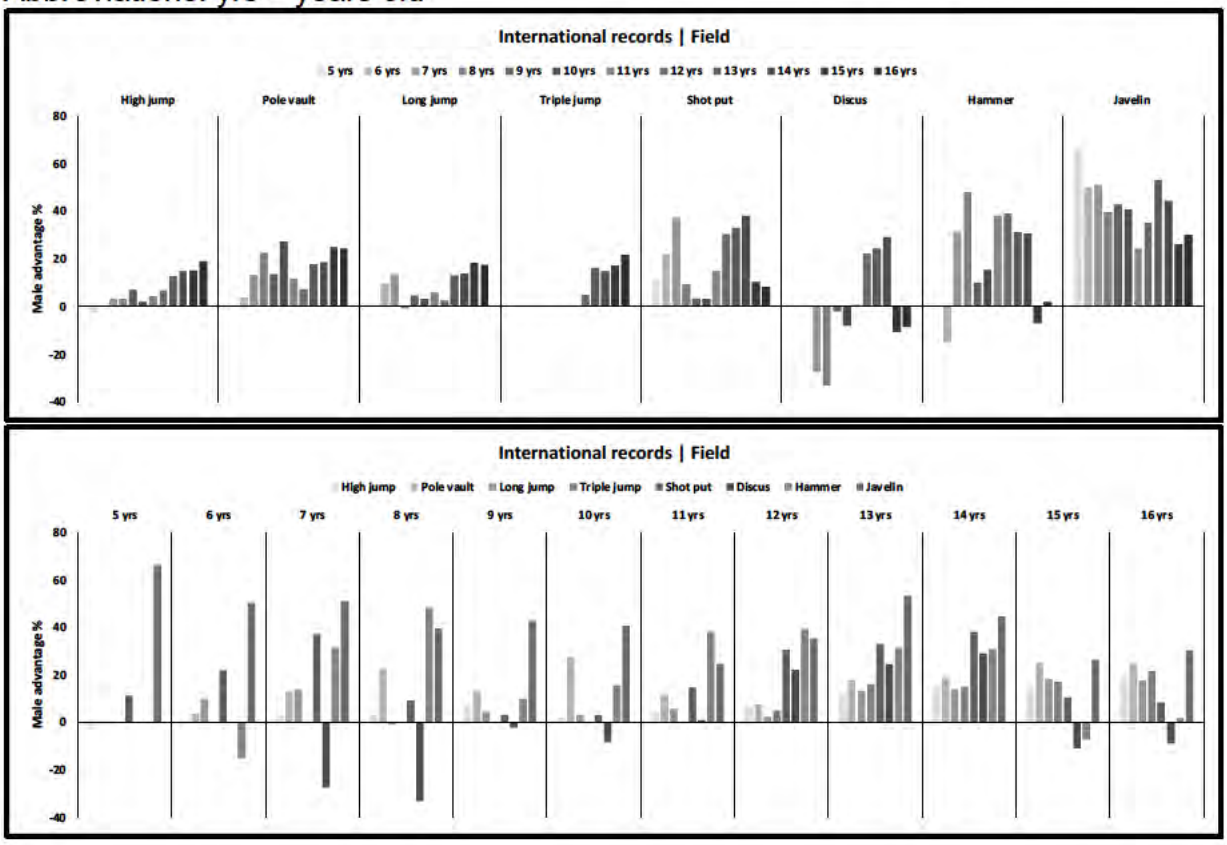
7.7 Also evident in this dataset is an unusually large male advantage for 3000 m at 7 years old. Analysis of the age progression for this event, shown in Figure 5, reveals this is underpinned by an unexpectedly poor female record for 3000 m at 7 years old. Thus, the extent of male advantage here is likely an overestimate of the true performance gap.

Figure 5. Age progression in the 3000 m in international schoolchildren records.
Abbreviations: yrs – years old, s – seconds



7.8 The male advantages (%) in field events, stratified by both event (upper panel) and age (lower panel), are shown in Figure 6. In field events, male advantage is again evident in all age groups and for all events, although this appears less systematic than in track events.

Figure 6. The male advantage over females in international schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old

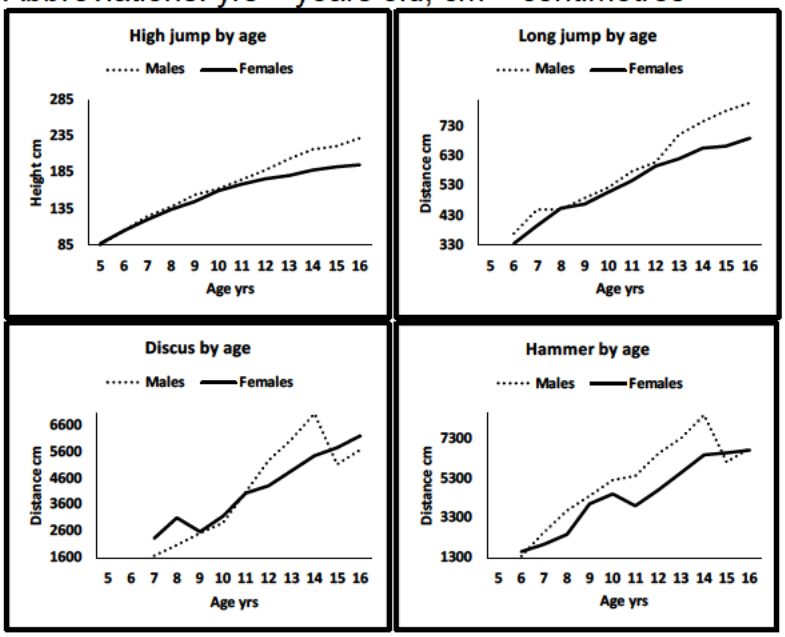


7.9 There are several field events where female schoolchildren appear to outperform their male peers, listed in Table 4. I examined the age progression of these events to seek to understand this apparent female advantage. These data are shown in Figure 7. For the high jump at 5 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. For the long jump at 8 years old, the female advantage appears to be explained by the convergence of an unusually poor male record and unusually good female record in this event.

Table 4. Female advantage in international schoolchildren field records.
Abbreviations: yrs – years old, m – metres

Event	Age group	Female advantage %
High jump	5 yrs	2.3 %
Long jump	8 yrs	0.9 %
Discus	7 yrs	27.4 %
	8 yrs	33.1 %
	9 yrs	2.1 %
	10 yrs	8.1 %
	15 yrs	10.8 %
	16 yrs	8.7 %
Hammer	6 yrs	15.1 %
	15 yrs	7.2 %

Figure 7. Age progression in the high jump, long jump discus and hammer in international schoolchildren records.
Abbreviations: yrs – years old, cm – centimetres



7.10 There are several throw events where female schoolchildren appear to outperform their male peers by a large distance. However, there are important confounding factors in throwing events, given that the weight of throwing implements can differ between male and female athletes at different ages. For the discus, girls at 7-8 years old throw a discus weighing 500 g, compared to boys of the same age using a 750 g discus. I hypothesise that a similar implement weight at 7-8 years old would mitigate or remove the apparent female advantage here. Between 9-14 years old, both sexes use a 1 kg discus. Performance between males and females seems broadly matched until 11 years old, which may be underpinned by earlier female puberty. Males open up the performance gap at 11 years old. At 15 years old, boys switch to a 2 kg discus. I believe it is reasonable, given the increasing male gap to 14 years old with the same implement weight of 1 kg, that a matched implement weight between males and females at 15-16 years old would reverse the apparent female advantage in favour of clear male advantage.

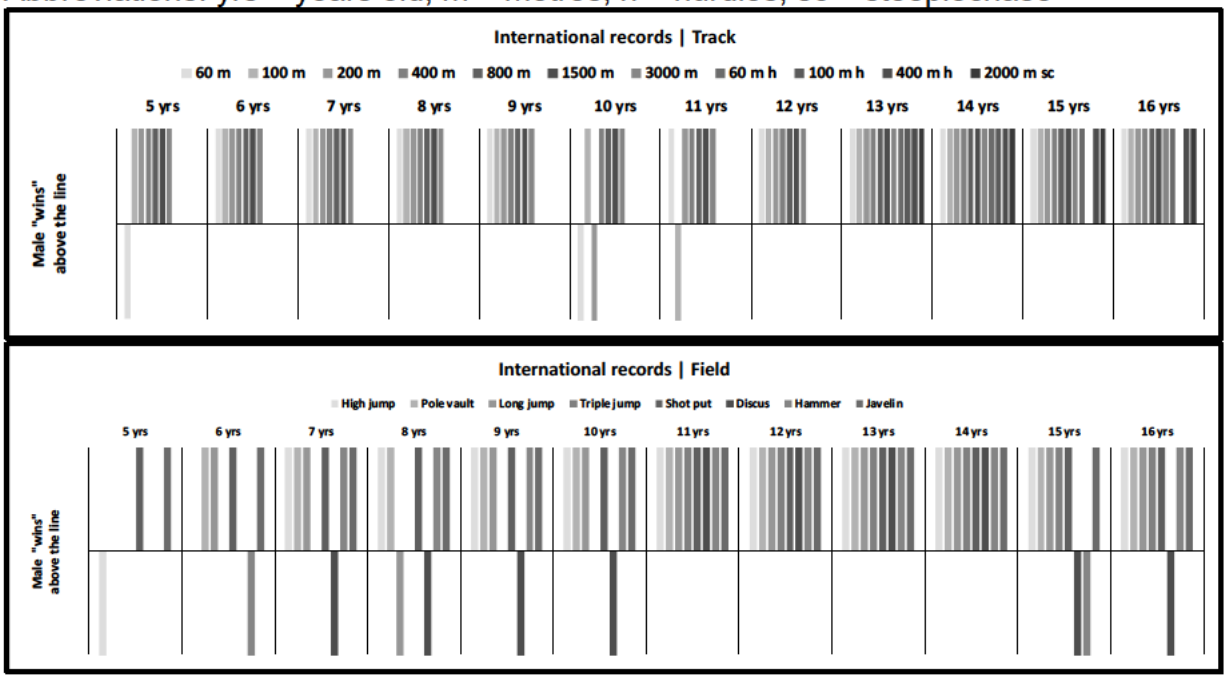
7.11 For the hammer, male and females use a 2 kg implement between the ages of 6-10 years old. At 6 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. Male advantage is evident from 7-10 years old; the 'catch up' with male peers at ages 9-10 years old may be explained by the physical changes of female puberty. Between the ages of 11-14 years old, both males and females use a 4 kg hammer, and male advantage is consistent through these ages. At 15 years old, males switch to a 7.26 kg hammer. I believe it is reasonable, given the male advantage evident throughout the time period where both sexes use a 4 kg hammer, that a matched implement weight between males and females at 15-16 years old would reverse the apparent female advantage in favour of clear male advantage.

7.12 Interestingly, male advantage is evident in all shot put and javelin events at all ages, despite increases in implement weight at 15-16 years old for males.

7.13 I formulated a null hypothesis: if there are no sex differences in athletic performances in schoolchildren, males and females are equally likely to hold the best record in any event. Therefore the frequency of males with the best record should be approximately equal to

the frequency of females with the best record. To interrogate this statistically, I scored all track and field events at all ages as a binary variable of male "wins" versus female "wins" (whichever record was the fastest, longest, etc). I ignored potential confounding explanations in various events; that is, female advantage was scored as a "female win", even if the female advantage is likely an artifact of, for example, earlier puberty or lighter implement weight. Thus, this scoring is deliberately generous to ensure the strength of any findings. Scoring data are visualised in Figure 8, with track events in the upper panel and field events in the lower panel. It is already clear from this analysis that the majority of "wins" go to male schoolchildren.

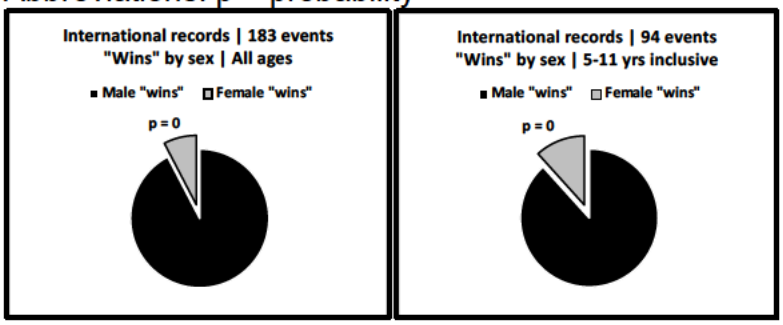
Figure 8. Male versus female "wins" in international schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, sc – steeplechase



7.14 I counted the frequency of male "wins" versus female "wins" at all ages and in a sub-analysis limited to events in pre-puberty (5-11 years old) age groups. I then calculated the probability that the frequency of male "wins" versus female "wins" would occur by chance.[88] These data are shown in Figure 9. The majority of "wins" go to male schoolchildren, whether across all age groups or limited to pre-puberty age groups. The chances of this frequency of male "wins" occurring by chance in either age grouping is calculated at a probability of effectively zero (p = 0).

[88] https://homepage.divms.uiowa.edu/~mbognar/applets/bin.html

Figure 9. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: p – probability



7.15 Following the same process for international records above, I analysed junior records from 8-16 years old from USA Track and Field (USATF)[89] and the US Amateur Athletics Union (AAU).[90] For brevity here, these datasets are compiled in **Appendix 3** (USATF) and **Appendix 4** (AAU). These national datasets confirm the results obtained from international records. **To summarise the data obtained from international and national schoolchildren records in track and field: 1. male advantage over female peers is evident across track and field events from 8 years old onwards; 2. males systematically outperform their female peers from 8 years old at a frequency that is vanishingly unlikely to result by chance.**

7.16 Again, following the same process for international records above, I analysed Arizona middle school records from 8-16 years old (available to 2014).[91] For brevity here, this dataset is compiled in **Appendix 5**. This dataset confirms that male advantage over female peers is predominant across track and field events from 8 years old. In these state level records, more female "wins" are scored in lower age groups than seen in international and national records. However, the frequency of male "wins" between 8-12 yrs old is still statistically unlikely to result from chance ($p = 0.043$, where $p = 0.05$ is the "significance" threshold).

7.17 I analysed the outcomes of two individual middle-school competitions. The first was the Kyrene District Track and Field Championship, held in April 2023.[92] Middle-schoolers participated in 13 events, and I calculated the male advantage for the winners of each matched event. These data are shown in Figure 10. In this school district championship, male advantage was evident in all events. I pooled all events then plotted the frequency of male versus female "wins" in this group of athletes. Again, I calculated the probability that the male "win" frequency would occur by chance. These data are shown in Figure 11. The probability that males would win all these events by chance is vanishingly low.

[89] https://www.usatf.org/resources/statistics/records/championship-meet-records/usatf-national-junior-olympic-track-field-champion
[90] https://aautrackandfield.org/Results
[91] http://www.usatf.com/assoc/az/records.html
[92] https://www.athletic.net/TrackAndField/meet/486419/results/all

Figure 10. The male advantage over females at the Kyrene District Track and Field Championship, held in April 2023.
Abbreviations: m – metres, h – hurdles, SMR – sprint medley relay

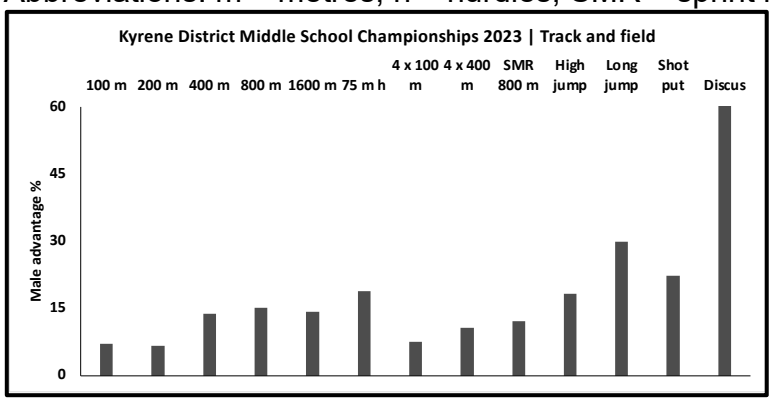
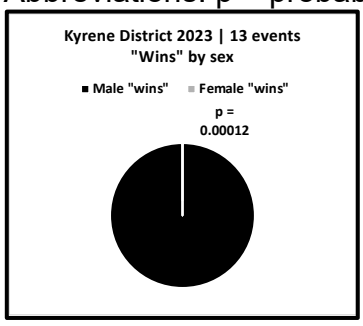
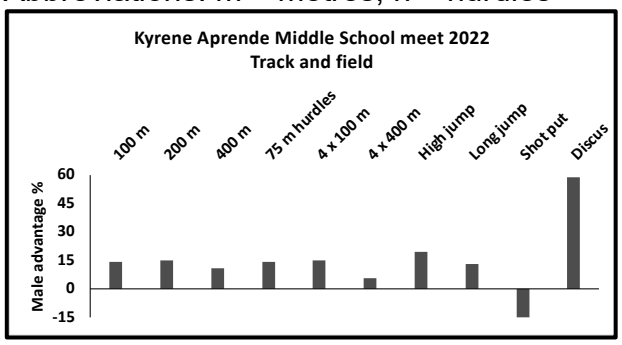


Figure 11. The frequency of male versus female "wins" across the pool of events at the Kyrene District Track and Field Championship, held in April 2023.
Abbreviations: p – probability



7.18 The second middle-school competition I analysed was the Kyrene Aprende Middle School Track and Field meet, held in July 2022.[93] Middle-schoolers participated in 12 events; however, the girls' times for the 800 m and 1600 m were not recorded on the scoresheets so I was unable to include these in my analysis. I calculated the male advantage for the matched winners in the remaining 10 events. These data are shown in Figure 12. In this single school athletics meet, male advantage was evident in all events except the shot put, where the apparent female advantage was an unexpectedly large 14.8 %.

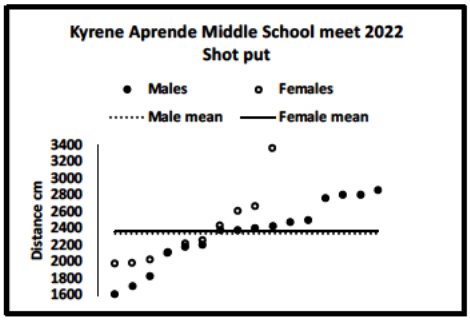
Figure 12. The male advantage over females at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: m – metres, h – hurdles



[93] https://www.kyrene.org/Page/55102

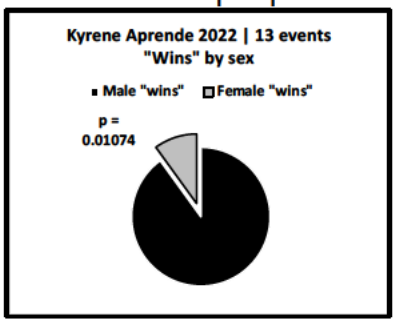
7.19 To understand the source of this female advantage in shot put, I analysed the puts of all the males and females at this middle school meet. These data are shown (with puts in increasing order of distance achieved) in Figure 13. The winner of the female competition putted 3360 cm, well beyond the second placed girl at 2670 cm. This winning female performance is 4.2 standard deviations from the female mean put distance, indicating an extraordinary performance with odds of occurrence of approximately 1 in 15000. A comparison of the mean distance putted by boys and girls shows them to be quite similar; however, the female winner is skewing this mean distance by 110 cm (the male winner only skews the male mean by 35 cm).

Figure 13. Analysis of puts at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: cm - centimetres



7.20 I pooled all events then plotted the frequency of male versus female "wins" in this group of athletes. Again, I calculated the probability that the male "win" frequency would occur by chance. These data are shown in Figure 14. The probability that males would win almost all the events by chance is very low (p = 0.05 is the "significance" threshold).

Figure 14. The frequency of male versus female "wins" across the pool of events at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: p – probability



7.21 Analyses of international, national and state track and field performances in male and female schoolchildren evidence sex differences in athletic performance, even before puberty. Sex differences in athletic performance are evident in middle school track and field meets. Collectively, these data demonstrate that female children require a female category of sport to win.

7.22 Childhood male athletic advantage over females has been proposed as social in origin. That is, higher engagement in sport and exposure to rougher play may represent 'training advantage' over females who are somewhat socialised to engage in less

physical activity.[94] However, despite suggesting that childhood performance gaps are possibly social in origin, Thomas and French (1985) identify an extremely large gap in throwing differences, evident from age 3 years old, that are “unlikely to be completely environmentally caused” and are unlikely, based on biological factors, to be eliminated by training. The performance gap in international and national track and field records, evident even before puberty, somewhat controls for this socialisation effect, given that one might expect engaged, sporty girls to be as well-trained as their male peers.

[94] For example: Thomas and French, 1985. Gender differences across age in motor performance a meta-analysis. Psychol Bull 98(2):260-282.

8 Sports categories and concepts of advantage

8.1 Sports where performance or competitor safety is affected by sex routinely employ a protected female category that excludes males, to secure fairness for (and, discipline-dependent, safety of) female athletes. This separation on the basis of sex in pursuit of fair, safe sports and sporting opportunities for female athletes is permissible under much national equality legislation, including, for example, the UK Equality Act 2010.[95]

8.2 Misunderstandings regarding the nature of categories and advantage are common. Sports categories control for baseline physiological differences in sex, age, and impairment (and occasionally weight) that affect results or outcomes independently of the characteristics sporting competition seeks to reward: talent, strategy, training and dedication. Various initiatives like leagues, which operate alongside categories, exist to permit participation of those with different amounts of talent, strategy, training and dedication.

8.3 Categories are rationalised on biological principles, understanding what effect factors like sex and age have on the human body. They exist to ensure physiological “bonuses” (being male, being young) do not obscure outcomes that should depend on talent, strategy, training, and dedication. It is via categories that fairness is achieved, and we ensure that winning opportunities for the more talented athlete—a fundamental characteristic of sport—are preserved. Protected categories like the female category are a necessary inclusion measure to ensure females have an equal opportunity to compete in sports.

8.4 Advantage exists regardless of magnitude. Indeed, sports bodies have a history of regulating for even very small advantages. For example, inside lane track runners closer to the traditional start gun hear the gun more quickly and more loudly than those in outside lanes, offering them a small kind of advantage unavailable to the whole field. To combat this advantage, worth around 150 milliseconds in a staggered start of a 400m track, runners typically now start races via a loudspeaker at each block.[96] Even if an apparent advantage is small, a category or rule operates to exclude any quantity of it.

8.5 A common argument is to frame ‘advantage’ as simply a property of results (for example, any person who is faster than any other has ‘advantage’, while people who are equally fast are said to be fairly-matched), one undermines the very existence of categories. The logical outcome is sports organised not to reward talent but to reward a combination of talent and talent-independent physical properties that together deliver a winning outcome. In such a framework, almost all sports at every competitive level will be dominated by able-bodied males aged around 20-35 years old.

8.6 What has traditionally been described as a “girl’s/women’s category” is more precisely understood as a category for females that excludes males who have acquired any magnitude of male athletic advantage by virtue of biology, regardless of performance relative to the female field. The ineligibility of those with any male advantage is necessary to maintain the integrity of the female sports category.

8.7 Puberty, where we see a sharp divergence of male and female athletic performance, is typically regarded as the age at which a protected female category becomes necessary. I believe, given the evidence I have presented in **Section 7** that demonstrates male advantage in childhood, that is justified from pre-puberty ages to institute a protected female category that excludes any male advantage, should fairness for young female athletes be a priority for regulators.

[95] UK Equality Act 2010, Part 14, Section 195.
[96] Holmes, 2008. Olympic start gun gives inside runners an edge. New Scientist, 23rd June 2008.

9 Treatment of transgender girls and transgender women

9.1 Transgender girls and transgender women may take social, pharmaceutical and/or surgical steps to be perceived and treated as if they were female. In adulthood, transgender women may opt for testosterone suppression (for example, via gonadotropin-releasing hormone [GnRH] agonists, spironolactone or cyproterone acetate) then/or surgical removal of the testes; both of these interventions have the effect of lowering testosterone levels to those of females[97] and reducing the functional or visual impact of male physical characteristics. Estrogen supplementation typically promotes feminisation of, for example, breast tissue.[98]

9.2 Early pharmaceutical interventions in transgender girls may involve blocking male puberty via GnRH agonists ("puberty blockers"), administered after the onset of puberty (at least Tanner stage 2; in male children, the appearance of pubic hair, increase in testicular volume and reddening of scrotum skin).[99] This is typically followed by a regime of cross-sex hormones from 16 years old.

9.3 Many children reporting gender dysphoria or incongruent gender identity desist; that is, gender identity issues resolve with puberty.[100] For this reason, puberty blockers are not administered until after the onset of puberty and there is observed demonstrable persistence of gender identity issues. Furthermore, the reported effects and side-effects of puberty blockers are serious, including long-term effects on bone growth, brain development, fertility and sexual function, and short-term effects like headaches, hot flashes, mood swings, and depression and anxiety,[101] necessitating caution with their prescription.

9.4 Considering the potential for medical harm while outcomes remain uncertain, many jurisdictions have cautioned against or restricted the use of puberty blockers in children, including the Swedish National Board of Health and Welfare,[102] the Finnish Health Authority,[103] the French National Academy of Medicine[104] and the Norwegian Healthcare Investigation Board.[105] The UK NHS has recently restricted puberty blockers within clnical research.[106] Pioneers of the original protocol for treatment of childhood dysphoria have advocated re-evaluation considering the rapidly-changing cohort demographics.[107]

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[97] Nishiyama, 2014. Serum testosterone levels after medical or surgical androgen deprivation: a comprehensive review of the literature. Urologic Oncology 32(1): 38.e17-28.
[98] Unger, 2016. Hormone therapy for transgender patients. Translational Andrology and Urology. 5(6): 877-884.
[99] Puberty progression is assessed using "Tanner staging", which describes the typical physical changes in boys and girls using landmarks of external genitalia in males (testicular volume, penis length and skin appearance), quantity and coarseness of pubic hair in both sexes, and breast development in girls. In males, Tanner stage 2 indicates the first signs of puberty, around the age of 11 years old, comprising the appearance of downy pubic hair, an increase in testicular volume and reddening of the scrotum skin. At Tanner stage 3, around the age of 13 years old, the penis begins to grow in length. Testicular volume increase and penis growth continues during later stages, and pubic hair becomes course and curly. For more information, see: https://childgrowthfoundation.org/wp-content/uploads/2020/03/Puberty-and-Tanner-Stages_v2.0.pdf
[100] Wallien and Cohen-Kettanis, 2008. Psychosexual outcome of gender-dysphoric children. Journal of the American Academy of Child and Adolescent Psychiatry 47(12): 1413-1423.
[101] Reported by various healthcare providers, for example: Mayo Clinic, NHS, St. Louis Children's Hospital.
[102] https://www.socialstyrelsen.se/globalassets/sharepoint-dokument/artikelkatalog/kunskapsstod/2022-3-7799.pdf
[103] https://palveluvalikoima.fi/documents/1237350/22895838/Summary+transgender.pdf/2cc3f053-2e34-39ce-4e21-becd685b3044/Summary+transgender.pdf?t=1592318543000
[104] https://segm.org/sites/default/files/22.2.25-Communique-PCRA-19-Medecine-et-transidentite-genre.pdf
[105] https://www.bmj.com/content/bmj/380/bmj.p697.full.pdf
[106] https://www.england.nhs.uk/wp-content/uploads/2023/06/Interim-service-specification-for-Specialist-Gender-Incongruence-Services-for-Children-and-Young-People.pdf
[107] de Vries, 2020. Challenges in Timing Puberty Suppression for Gender-Nonconforming Adolescents. Pediatrics 146(4): e2020010611.

9.5 When prescribed as above, puberty blockers do not, by definition, block the entirety of male puberty. They do not block any hormone-derived pre-puberty effects on male development. They are unlikely to interfere with genetic effects on male development.

10 Transgender women in sport

10.1 Given the role of testosterone in the development of the male characteristics that matter for sporting performance, and bearing in mind the typical pharmaceutical and medical treatment sought by transgender girls and transgender women, the International Olympic Committee (IOC) and other sporting federations have historically sought to include transgender women in female sports by regulating levels of testosterone prior to inclusion in female competition.[108] More recently, the IOC have suggested that "*testosterone levels could be investigated as a means to mitigate performance*" in transgender women.[109] It is inferred that the IOC believe testosterone suppression may be sufficient to remove the male performance advantage provided by male-typical secondary sex characteristics.

10.2 In 2020, with the IOC equivocating over a review of their testosterone guidelines, Dr Tommy Lundberg and I tested the existing guidelines' promise to protect fair competition, by reviewing peer-reviewed published longitudinal changes in muscular and skeletal metrics in transgender women suppressing testosterone in adulthood for a minimum of 12 months.[110] Having reviewed measures of bone density, lean body mass, muscle mass and strength tests, we identified a unified consensus in original studies covering approximately 800 transgender women that skeletal metrics like height and bone length were unaffected, bone mass was preserved, and muscle mass and strength was decreased by 4% over 12 months of testosterone suppression. Within this dataset, compared with female control cohorts, higher muscle mass/strength values—between +13-41 %—were maintained for at least three years after testosterone suppression (the limit of current longitudinal studies).

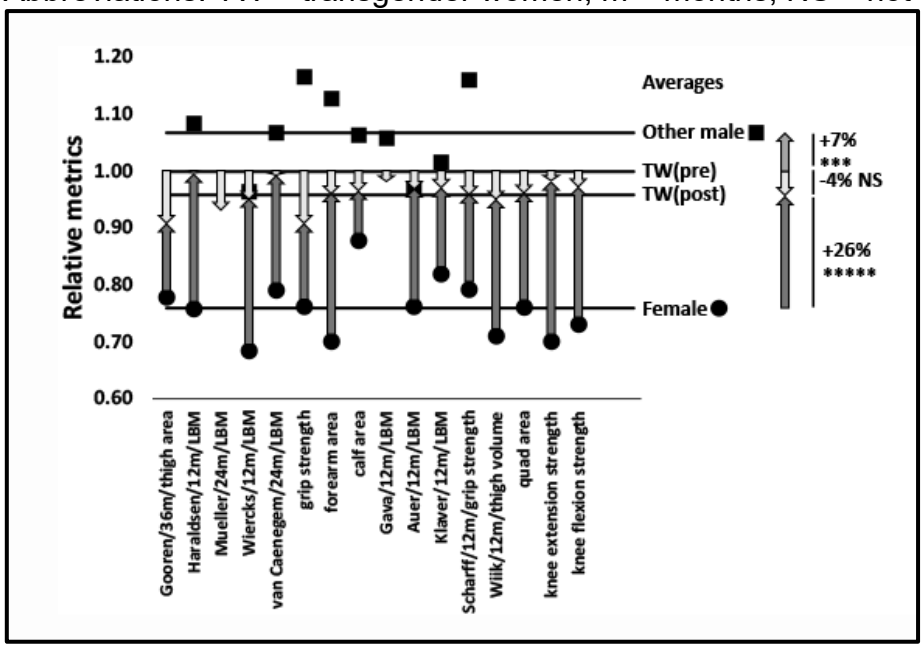
10.3 These observations were subsequently reinforced by a systematic review of the same dataset published by another group later in 2021, which concluded that, in transgender women, "*hormone therapy decreases strength, [lean body mass] and muscle area, yet values remain above that observed in cisgender women, even after 36 months. These findings suggest that strength may be well preserved in transwomen during the first 3 years of hormone therapy.*"[111]

10.4 To gain an overall picture of the baseline metrics and effects on muscle mass and strength in transgender women pre- and post- at least 12 months of testosterone suppression, I compared pre- and post- metrics for transgender women across the Hilton and Lundberg dataset with data from control males and females, shown in Figure 15. Original study metrics were converted to relative percentages, with pre-suppression metrics in transgender women set at 100%. The 4% reduction in muscle mass and strength in transgender women pre- and post- at least 12 months of testosterone suppression was not statistically significant. The difference between transgender women and control males was statistically significant, with transgender women pre- and post- at least 12 months of testosterone suppression deviating from control males by -7% and -11%, respectively. The difference between transgender women and females is also statistically significant; transgender women pre- and post- at least 12 months of

[108] https://stillmed.olympic.org/Documents/Commissions_PDFfiles/Medical_commission/2015-11_ioc_consensus_meeting_on_sex_reassignment_and_hyperandrogenism-en.pdf
[109] Martowicz et al., 2023. Position statement: IOC framework on fairness, inclusion and non-discrimination on the basis of gender identity and sex variations. Br J Sports Med 57:26–32.
[110] Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51, 199–214. Note: the date disparity of the published paper represents the gap between article submission and publication.
[111] Harper et al., 2021. How does hormone transition in transgender women change body composition, muscle strength and haemoglobin? Systematic review with a focus on the implications for sport participation. Br J Sports Med 55: 865-872.

testosterone suppression deviate from control females by +35% and +30%, respectively. It appears that for metrics of muscle mass and strength, transgender women remain within 'male range'.

Figure 15. Relative metrics in transgender women pre- and post- testosterone suppression, compared with control males and females.
Abbreviations: TW – transgender women, m – months, NS – not significant



10.5 In addition to the longitudinal data captured by the above reviews, there are three significant cross-sectional studies of physical metrics in transgender women suppressing testosterone. The first found that transgender women, after an average of 8 years of suppressed testosterone, had a lean body mass in the 90th percentile for females, and grip strength that remained 25 % higher than the female reference value.[112] The second, in transgender women suppressing testosterone for just over 3 years, showed that those transgender women had a mean lean body mass 18 % higher than the mean in control females.[113] The third found that transgender women suppressing testosterone for over 14 years retained higher cardiopulmonary capacity metrics and higher hand grip strength than female controls.[114]

10.6 In 2015, to assess sports performance in transgender women, an observational cohort study of transgender women runners was performed, studying race times before and after testosterone suppression.[115] Participants were club-level middle-distance runners. After applying an age-grading formula typically reserved for Masters athletes, performance in the female category was judged to be maintained at a level equivalent to pre-suppression performance in the male category. This study had a sample size of eight runners self-reporting times that were unverifiable in 50% of cases and spanning a period of decades. The study could not make any controls for ageing, training, diet,

[112] Lapauw et al., 2008. Body composition, volumetric and areal bone parameters in male-to-female transsexual persons. Bone. 43(6):1016–1021.
[113] Bretherton et al., 2021. Insulin resistance in transgender individuals correlates with android fat mass. *Ther Adv* Endocrinol Metab 12:2042018820985681.
[114] Alvares et al., 2022. Cardiopulmonary capacity and muscle strength in transgender women on long-term gender-affirming hormone therapy: A cross-sectional study. Br J Sports Med 56: 1292-1298.
[115] Harper, 2015. Race times for transgender athletes. Journal of Sporting Cultures and Identities 6:1-9.

injury, running course, or course weather conditions. The overall cohort analysis included times from runners who had experienced chronic injury and loss of fitness, resulting in poorer-than-expected performance within the female field. However, excluded from the overall analysis was a runner who had achieved a far higher ranking competing in female running than in male running. This individual improved ranking significantly, and even recorded a marathon that was faster than previous marathon performance in the male category, but was considered an outlier who had seriously intensified her training after transition into female sport. This individual demonstrates, as argued in Hilton and Lundberg, that training during testosterone suppression can mitigate negative performance effects.

10.7 There have been two studies of athletic performance in military personnel using basic fitness testing data.[116] While not athletes, these individuals do represent a trained population of transgender people. Both studies tracked changes in push-up, sit-up and 1.5 mile run performance during annual fitness testing over 3 or 4 years of testosterone suppression. Such tests are 'work to target': recruits are aware of targets that must be achieved to pass the fitness testing process, minimum performances must be achieved for each test, and a cumulative score threshold must be reached to pass the fitness test. Individual officers have the latitude to "choose" how their scores are allocated, such that a particularly strong runner has a lower need to gain points during the push-up test (for example). The performances cannot thus be assessed as maximal performances, but instead may be considered as paced performances with conscious knowledge of a required standard. The authors of the first study acknowledge that, despite being in a controlled environment of the Air Force, the exercise intentions and training habits of the recruits was unknown, and over a period of three years, changes in training with material implications for muscle and cardiovascular performance cannot be known.

10.8 Significantly, the data from the two studies of athletic performance in military personnel make contradictory findings, presented in Table 5. Roberts et al. (2021) finds that both push-up and sit-up performance are statistically equivalent to female performance after 2 years while advantage in running performance is retained to 2 years. However, Chiccarelli et al. (2022) finds that push-up advantage is retained beyond 4 years, sit-up performance is statistically equivalent to female performance at 4 years and running performance is statistically equivalent to female performance at 2 years.

10.9 This set of performance studies suffer from small numbers of participants, lack of controls for performance times, and issues regarding the validity of performance tests. They cannot be used in isolation to inform sports policy, particularly when the overwhelming body of evidence suggests that the effects of testosterone suppression on important metrics like muscle mass and strength are marginal and that male development, and thus male advantage, cannot be reversed.

[116] Roberts et al., 2021. Effect of gender affirming hormones on athletic performance in transwomen and transmen: Implications for sporting organisations and legislators. Br J Sports Med 55:577-583; Chiccarelli et al., 2022 Fit transitioning: When can transgender airmen fitness test in their affirmed gender? Mil Med 2022;usac320.

Table 5. A comparison of the findings of two studies of athletic performance in military personnel.
Abbreviations: NA – not applicable, * – year at which statistical parity with females is reached

	Roberts et al., 2021 Year group % change (% advantage over female controls)			**Chiccarelli et al., 2022** Year group % change (% advantage over female controls)		
	Push-ups	**Sit-ups**	**Running**	**Push-ups**	**Sit-ups**	**Running**
Pre-transition	NA (+45.5 %)	NA (+17.3 %)	NA (+17.2 %)	NA (66.3 %)	NA (+28.3 %)	NA (+17.8 %)
Year 0-1	-5.7 % (+37.2 %)	+1.1 % (+18.6 %)	-7.1 % (+11.3 %)	-13.0 % (+44.7 %)	-6.1 % (+20.5 %)	-10.4 % (+9.2 %)
Year 1-2	-3.1 % (+32.9 %)	-4.3 % (+13.6 %)	-4.4 % (+7.5 %)	-9.4 % (+31.0 %)	-2.6 % (+17.3 %)	-4.5 % (+5.1 %)*
Year 2-3	-19.9 % (+6.5 %)*	-13.5 % (-1.8 %)*	+3.3 % (+10.5 %)	-2.0 % (+28.3 %)	-5.2 % (+11.2 %)	-0.0 % (+5.1 %)*
Year 3-4				-8.3 % (+17.7 %)	-2.6 % (+8.3 %)*	-5.2 % (+0.2 %)*

11 Transgender girls in sport

11.1 Most sporting federations exempt from testosterone regulations those who have blocked puberty before cross-sex hormone treatment. To my knowledge, there is no published data on muscle mass and strength metrics in a cohort of transgender girls who have blocked puberty from Tanner stage 2.

11.2 Recently available is a study by Boogers et al. (2022) called, "Trans girls grow tall: adult height is unaffected by GnRH analogue and estradiol treatment."[117] In this study, transgender girls who had received puberty blockers from around 13 years of age, then cross-sex hormones at 16 years of age, acquired an average adult height of 180.1-185.3 cm, far larger than the population female average (170.7cm) and around the population male average (183.8cm). The authors conclude that the driver of height acquisition is genetic in origin, and not a result of testosterone during puberty.

11.3 In two studies where male puberty was partially-blocked, lean body mass in young adulthood remains higher than in reference females[118] and grip strength remains higher than in a matched cohort of transgender boys.[119]

11.4 Claims that transgender girls who block puberty do not acquire any male athletic advantage in terms of skeletal structure and/or muscle mass are speculative.

[117] Boogers et al., 2022. Trans girls grow tall: adult height is unaffected by GnRH analogue and estradiol treatment. Journal of Clinical Endocrinology and Metabolism. Epub ahead of print, PMID: 35666195.
[118] Klaver et al., 2018. Early Hormonal Treatment Affects Body Composition and Body Shape in Young Transgender Adolescents. Journal of Sexual Medicine 15(2): 251-260.
[119] Tack et al., 2018. Proandrogenic and Antiandrogenic Progestins in Transgender Youth: Differential Effects on Body Composition and Bone Metabolism. Journal of Clinical Endocrinology and Metabolism 103(6): 2147-2156.

I verify under the penalties for perjury that the foregoing representations are true.

Emma Hilton, PhD
27th June 2023

Appendix 1.
Emma Hilton Short-form academic CV

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CURRICULUM VITAE

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ROLES

04/2019 - present	Postdoctoral research associate, Division of Infection, Immunity & Respiratory Medicine, University of Manchester (funding from BBSRC; NC3Rs; Cystic Fibrosis Foundation).
01/2014 - 04/2019	Research Fellow, University of Manchester (funding from MRC, Newlife).
01/2010 - 01/2014	Stepping Stone Research Fellow, Genetic Medicine, University of Manchester (funded internally).
06/2003 - 12/2009	Postdoctoral research associate, Genetic Medicine, University of Manchester (MRC).

ACADEMIC QUALIFICATIONS

2004	Ph.D. Developmental Biology, University of Warwick, UK.
1999	B.Sc. (Honours) Biochemistry, University of Warwick, UK.

PUBLICATIONS (Google Scholar: Citations 1181, h-index 16)

Randles, M., Hamidi, H., Lausecker, F., Humphries, J.D., Byron, A., **Hilton, E.N.**, Clark, S.J., Miner, J.H., Zent, R., Humphries, M.J. and Lennon, R. Integrin-specific signalling pathways determine podocyte morphologies on basement membrane ligands. Submitted, Nat. Commun.

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Hilton E.N., Black, G.C., Manson, F.D., Schorderet, D.F., Munier, F.L. (2007). De novo mutation in the BIGH3/TGFB1 gene causing granular corneal dystrophy. Br J Ophthalmol. 91(8):1083-1084.

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Hilton, E.N., Rex, M., Old, R. (2003). VegT activation of the early zygotic gene Xnr5 requires lifting of Tcf-mediated repression in the Xenopus blastula. Mech Dev. 120(10):1127-1138.

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AUTHORED BOOK CHAPTERS

Hilton, E., Black, G.C.M., Bardwell, V. BCOR and oculofaciocardiodental syndrome. (2008/2013). Epstein's Inborn Errors of Development: The Molecular Basis of Clinical Disorders of Morphogenesis, 2nd/3rd edition, Oxford University Press, Oxford, UK.

GRANT INCOME

2016-2018	Newlife (£115,735). Towards novel therapies for an inherited congenital neuropathy affecting the urinary bladder. Woolf, Newman, Kimber, **Hilton** (Co-app).
2014-2016	MRC (£507,695). Molecular bases of congenital bladder diseases. Woolf, Newman, Gardiner, **Hilton** (Research Co-I).
2010-2013	KRUK (£180,000). Urofacial syndrome (UFS): a novel genetic model to understand human renal tract function and malformation. Newman, Woolf, McKenzie, **Hilton** (Co-app).
2010-2014	University of Manchester (£salary + £40,000 project costs). Xenopus as a model organism for human development and disease. **Hilton** (Stepping Stone Fellowship Award).
2008-2010	Newlife (£100,000). The role of BCL-6 corepressor-modulated TGFβ signalling in MCOPS2 and other microphthalmia syndromes. Black, Manson, **Hilton** (Co-applicant).

Appendix 2.
Hilton and Wright, 2023. “Two sexes”. Sex and Gender: A Contemporary Reader. Routledge, Oxford, UK.

TWO SEXES
Emma Hilton and Colin Wright

‘Why the sexes are, in fact, always two.’
Sir Ronald Fisher, 1930

Sex is an evolved system function common to almost all complex life on earth, a fact that is often forgotten by postmodernist commentators intent on framing sex as a human-centred, human-invented—and thus malleable—construction of scientific understanding. The aim of this essay is to review the biological understandings of the phenomenon that is sex.

In the first section, we will answer the question: why does sex exist? We will explain its evolutionary origins, and the binary gamete system on which ‘female’ and ‘male’ are founded. To finish this section, we explore some of the diversity of sex—female and male—in the natural world, to understand how reproductive bodies in almost all complex life are organised around these functional roles. In the second section, we will focus on developmental biology and how sex manifests in humans: how we make babies and how female and male humans develop. In the final section of this chapter, we will critique emerging ideological misinformation about sex, particularly in humans, addressing arguments that, for example, assert sex as a social construct or seek to deconstruct standard understandings of sex as a binary phenomenon. We will highlight fundamental misinterpretations of sex and its associated characteristics, the unscientific focus on those people with atypical sex development and the dangers of viewing sex as a statistical outcome.

The incursion of ideological misinformation about sex into the academic fields of medicine and biology generates confusion in research and presents potential for harm. ‘Sex matters’ in basic and applied health research (Wizemann and Pardue, 2001) and the US National Institutes of Health, the EU Commission, research funding bodies and academic journals increasingly demand that researchers account for ‘sex as a biological variable’ in their research design, analyses and reporting, whether they include studies of whole organisms or cell lines. However, progress is slow. The UK NHS maintains a confusing system where biological sex cannot be disaggregated (Forstater, 2021), and the World Health Organisation promises to, ‘achieve greater impact on health [using] sex disaggregated data’ (WHO/Health topics/Gender) while simultaneously updating guidance to assert that, ‘sex is not limited to male or female.’ (WHO, 2022). We have publicly argued that, from the wider scientific perspective, ideologically-driven scientists are in danger of sacrificing, ‘empirical fact in the name of social accommodation’ and this is both, ‘an egregious betrayal to the scientific community they represent’ and, ‘undermines public trust in science.’ (Hilton and Wright, 2020). By re-asserting biological knowledge established over the preceding centuries and countering deconstructive discourse, this essay may be considered a reconstruction of sex.

A note on language. Physiologist Ernst Wilhelm von Brucke noted that, ‘Teleology is a lady without whom no biologist can live. Yet he is ashamed to show himself with her in public.’ (Davis and Uhrin, 1991). It is possible in discussions of evolutionary biology to avoid teleological language, but sentence constructions are often overly verbose and clunky. For ease of readability, we sometimes use language that is teleological in tone, but, in the words of zoologist

Simon Maddrell (1998), ‘This should not be taken to imply that evolution proceeds by anything other than from mutations arising by chance, with those that impart an advantage being retained by natural selection.’

WHAT IS SEX?

And why does sex exist? Remarkably, it is not uncommon to find purportedly-scientific articles about sex that neglect to mention its evolved function of reproduction (for example, Ainsworth, 2015; Sun, 2019). Indeed, that science communicators writing about sex often focus only on lists of physical characteristics associated with sex means that, despite the author claims, such articles are not actually addressing the biological phenomenon of sex—what is it? why does it exist? why do humans have sexed bodies? Rather, they are addressing how the sex of a given individual may be identified via some checklist of physical features that—ironically—could only have been created by understanding how those physical features are associated with reproductive function. We return to this conflation of sex (what it is) with the physical characteristics associated with sex (how we recognise the sex of a given individual) in the final section of this essay.

Reproduction The phenomenon of sex is rooted in reproduction, the process by which new individuals are produced from a parent or parents. There are two types of reproduction in the natural world: asexual and sexual. In asexual reproduction, a parent replicates all of its genetic information and generates new individuals by processes such as binary fission—the division of a parent cell into two identical (or, at least, very similar) cells, observed in bacteria—and budding—which produces a new individual from a parental outgrowth, observed in yeast.

In asexual reproduction, offspring receive a full set of genetic information from a single parent; it follows that offspring are genetically-identical clones of that parent. Individual expansion, via asexual reproduction, of a genetically-identical (or genetically-similar) population is a relatively low-cost biological burden, and rapid to enact; consider how quickly mould, which can reproduce asexually via the production of independent spores that populate the local environment, can colonise a loaf of bread, or how quickly bindweed can aggressively invade a garden by sending out roots from which new individuals grow. There are also parental benefits, as each parent passes on all of its genetic information to the next generation.

Yet despite the existence of a low-cost and rather straightforward method of reproduction, the natural world is dominated by species that employ a different reproductive strategy: sexual reproduction.

Unlike asexual reproduction, sexual reproduction involves two parents, almost always from two different classes of individuals called ‘females’ and ‘males’; each contributes half of their genetic material—carried on chromosomes—resulting in the generation of a new and genetically-unique individual. The mixing of genetic material from each parent (and thus, the beginning of a new individual) is achieved, in a process called ‘fertilisation’, by the fusion of two specialised cells called ‘gametes’. Gametes are a unique cell type within sexually-reproducing species and the function of the gamete within any individual is singular—to effect sexual reproduction.

Sexual reproduction is biologically-costly to individuals, not least because mating requires resources (for example, energy expended on locating a mate) and carries health risks (for example, disease transmission and exposure to predators). In most sexually-reproducing populations, half of the offspring will be males who cannot themselves bear offspring; thus, these populations experience lower growth rates than found in asexual populations, where all offspring

can themselves bear offspring (‘the cost of males’; Maynard Smith, 1978). Furthermore, genetic relatedness between parent and offspring is much lower than in asexually-generated clones, and each individual must therefore invest biological resources in producing at least two offspring to have any chance of passing all genetic material to the next generation. Explaining these costs—the ‘queen of problems in evolutionary biology’ (Bell, 1982)—has challenged evolutionary biologists; given the disadvantages, why did sexual reproduction evolve from asexual reproduction to become, by far, the most common method of reproduction in complex species?

The fusion of two gametes means that the new individual possesses a chromosomal makeup different to either parent and, given recombination between the chromosomes in each parent, chromosomes that carry different combinations of genetic material to either parent. The prevalence of sexual reproduction indicates a strong evolutionary advantage for this mechanism of reproduction that mixes genetic material. Such advantage is typically conceptualised as novel combinations of genes and changes in them (mutations) upon which evolutionary selection can act, the foundation of Darwin’s theory of evolution by natural selection (Darwin, 1859), and divided into two broad hypothesis domains: the accumulation of beneficial genetic changes and the removal of detrimental genetic mutations. Accumulation of beneficial genetic traits are advantageous in adaptation to changing environments (the ‘Fisher-Muller model’; Fisher, 1930; Muller, 1932) or co-adaptation, in an arms race, alongside interacting species who are trying to harm you (van Valen, 1973; delightfully called the ‘Red Queen hypothesis’ after Lewis Carroll's character in Through The Looking Glass, who observed, ‘Now, here, you see, it takes all the running you can do, to keep in the same place.’) However, the benefits of bringing together useful genetic traits during sexual reproduction must be balanced by the possibility that already coexisting beneficial traits are separated among offspring (Desai and Fisher, 2007). By contrast, harmful genetic mutations—those that compromise evolutionary fitness—must be weeded out to prevent them from accumulating in a population (see ‘Muller’s ratchet’, from Muller, 1964; also ‘Kondrashov's hatchet’, after Kondrashov, 1988).

The fitness advantages conferred by sexual reproduction explain its near-ubiquity among complex species. Indeed, even plants and simple animal species that typically reproduce asexually in stress-free environmental conditions to which they are comfortably adapted can switch to sexual reproduction during times of stress or environmental change, when genetic mixing may produce a survival advantage among offspring (for example, Becks and Agrawal, 2010). So successful an evolutionary strategy is sexual reproduction that many complex species, including humans, have completely lost the ability to reproduce asexually. No wonder Erasmus Darwin remarked that, ‘Sexual reproduction is the chef d'oeuvre, the masterpiece of nature.’ (Darwin, 1800).

Gametes and sexes While genetic exchange mechanisms exist, well-studied in bacteria and virus-host interactions, where DNA is transferred between different individuals in a non-sexual fashion (Callier, 2019), the evolutionary root of sexual reproduction via specialised gametes lies with the evolution of multicellularity, at least 1.5 billion years ago (Fu et al., 2019). In simple species like yeast (who can reproduce both sexually and asexually), all gametes are structurally similar; this is called ‘isogamy’. However, successful gamete pairing and fusion may be limited by molecular compatibility—mediated by various proteins on the cell surface (for example, Lipke and Kurjan, 1992)—between the cells of the parents. Such compatibility groups are described as ‘mating types’, usually labelled by a system of numbers (for a primer on mating types, see Fraser and Heitman, 2003). The number of mating types within a species can be thousands, and they functionally promote genetic diversity within a population by preventing gamete fusion

between genetically-similar parents. Isogamy is thought to be the ancestral state for sexual reproduction and remains common in simple sexually-reproducing species like yeast.

For an excellent overview of gamete evolution, see Lehtonen and Parker (2014) and references therein. Briefly, modelling of evolutionary scenarios for a variety of gamete characteristics shows that a binary system of gametes is optimal; that is, large gametes and small gametes, with gamete fusion occurring only between one small and one large gamete (not small-small or large-large fusions). We call this binary system of gamete fusion ‘anisogamy’.

In species with two gamete types, the large gamete (and associated biology) is termed ‘female’ and the small gamete (and associated biology) is termed ‘male’. In animals, the female and male gametes take the familiar forms of egg and sperm, respectively. In plants, the female and male gametes are contained in the ovules and pollen, respectively. That two different gametes form the optimal arrangement for sexual reproduction is understood in terms of gamete specialisation. The female gamete, with greater physical volume, single-handedly provides to the developing embryo basic cellular components, many molecules and signals required to direct early growth and energy-creating units called ‘mitochondria’. Strict uniparental—specifically, maternal—inheritance of cellular components—commonplace in anisogamy—is presumed favourable for embryo health by eliminating any biological compatibility between mitochondria (Greiner et al., 2015) and eliminates wasting when both parents invest resources in these components. In contrast, the male gamete sacrifices contribution to offspring beyond the chromosomes contained in its nucleus. Male gametes in many species have typically become specialised for mobility to better access female gametes—consider the tail-like structures of sperm that propel it towards the egg (Lessels et al., 2009) and pollen grains sticking to bee legs (Hu et al., 2008)—and created in large numbers to improve the chances of both an encounter with a female gamete and the outnumbering of small gametes from other males (Parker and Lehtonen, 2014).

Anisogamy is the evolutionary origin of the two sexes—the reproductive roles associated with female or male gamete contributions to offspring. The evolution of gametes into two non-overlapping, morphologically-distinct types necessitates specific cellular and tissue systems to produce either one or the other gamete and favours the subsequent evolution of anatomy that facilitates successful fertilisation events. The evolution of separate sexes is thought to have arisen multiple times in plants and animals, suggesting an evolutionary benefit. Common explanations include higher individual fitness when an individual is specialised for a single reproductive role, rather than trying to balance resources between both male and female functions (Charnov, 1982). In fact, given differential gamete morphology, the subsequent divergence into two separate sexes of individuals has been described as, ‘an almost inevitable consequence of sexual reproduction in complex multicellular organisms.’ (Lehtonen and Parker, 2014). Extending from mere inevitability to essentiality of outcome, Kashimada and Koopman (2010) state that, ‘the development of two sexes is observed in most animals and is essential for their survival and evolution.’

Why this almost inevitable divergence into just two sexes of individuals has occurred—repeatedly in evolutionary history—is the subject of much research. To answer this question, we must review the established knowledge on gamete evolution—the halving of genetic material, uniparental inheritance of intracellular components—and interrogate under what conditions could a third reproductive role—a third sex—evolve and what function could it have? Indeed, an exploration of this question was the prompt for the opening chapter quote. That is, ‘No practical biologist interested in sexual reproduction would be led to work out the detailed consequences experienced by organisms having three or more sexes; yet what else should he do if he wishes

to understand why the sexes are, in fact, always two?' (Fisher, 1930). And from science to science fiction, this question is, wonderfully, puzzled over by Kurt Vonnegut's Billy Pilgrim who, when considering the Tralfamadorians in Slaughterhouse-Five, surmised, 'They said their flying-saucer crews had identified no fewer than seven sexes on Earth, each essential to reproduction. Again: Billy couldn't possibly imagine what five of those seven sexes had to do with the making of a baby, since they were sexually active only in the fourth dimension. [...] It was gibberish to Billy.'

Sexual systems and bodies Across almost all complex life, there are precisely two types of gamete—and thus two and precisely two sexes. But this does not impose restrictions on how sex is allocated in different species. Evolutionary biologist Lukas Scharer illuminates, 'The male and female sexes are not two types of individuals; they actually represent two different reproductive strategies, and in many organisms, these two strategies are distributed among individuals in a population in a variety of ways.' (Scharer, 2017). That is, across the natural world, there is great diversity (and ingenuity, if one can—teleologically, of course—describe characteristics favoured by natural selection as 'ingenious') regarding the allocation of male and female sexes within and between individuals and across populations.

A 'sexual system' describes the physical and functional interactions of the two sexes at the individual and population level. We have learned that the evolution of separate sexes of individuals—a state called 'gonochorism'—is near-ubiquitous in animals; individuals within a gonochoristic species comprise two anatomic classes divided by reproductive role. Typically, male or female sex is fixed early in embryonic development and immutable to change during the lifespan of any individual, even though, of course, the physical characteristics associated with sex may be subject to expected age-related changes or changes acquired via injury or disease (or, at the hands of humans, surgery).

Humans cannot be hermaphrodites—individuals who fulfil both male and female reproductive roles in their lifespan—though hermaphroditism is a natural body plan in many anisogamous species. Many plants—particularly flowering plants—and (few) less complex animals exist as simultaneous hermaphrodites, with both female and male sexes manifested in the same flowers and/or same individual plant or animal at the same time of life. Many aquatic species—most notoriously, clownfish—are sequential hermaphrodites, where changes in reproductive role during the lifespan ('sex change') are evidenced by the switch from male to female (in the case of clownfish) or female to male gamete production, underpinned by anatomical changes in gamete-producing tissues (gonads). In the case of clownfish, this switch of sex (male to female) is driven by the loss of the single breeding female from the colony (Casas et al., 2016). Sequential hermaphroditism appears most common in species where males and females have the same excretory structures for eggs and sperm, and 'sex change' requires no or minimal remodelling of gross anatomy. For example, clownfish fertilisation is external, and male and female clownfish both have a similar ductal system that allows the sperm and eggs, respectively, into the aquatic environment. With highly-specialised and qualitatively-different reproductive anatomies, neither obviously nor easily remodelled post-development, 'sex change' in humans is impossible.

Evolution provides a dazzling array of anatomies and appearances. It is often true that gonochoristic males whose reproductive role is to contribute sperm have evolved appendages for direct introduction of that sperm into females, while the females of many species have evolved internal biology that receives sperm and, in the case of viviparous mammals who give birth to live young, protects the developing offspring from the outside world. But appearances can be

deceptive. For example, male seahorses have a brood pouch in which developing baby seahorses are incubated, a functional role more usually associated in the natural world with female individuals. However, these seahorses are the sex class that contribute sperm to the offspring, and it is that, not their gross anatomy, which defines those individuals as male. Another curious example is that of female spotted hyenas, who have a hyper-enlarged clitoris that resembles a penis, yet they produce eggs that are fertilised by a male hyena and are, by definition, female. Human-centred biological expectations about anatomy, which include, for example, pregnancy in females and penile appendages in males are undoubtedly too narrow to capture the diversity of sexed bodies in the natural world.

Hermaphrodites incorporate both male and female sexes, and gonochorists one or the other. And while gonochorism and simultaneous hermaphroditism represent stable arrangements of the two sexes within a species, there are many that buck these trends in their individual composition. For example, there are species composed of females and hermaphrodites (McCauley and Bailey, 2009), of males and hermaphrodites (Weeks et al., 2009), and of males, females and hermaphrodites (Oyarzun et al., 2020). That is, the two sexes can be differentially-allocated in individuals and between species. Yet, despite the variety of bodies and sexual systems found in the natural world, their organisation around two and only two sexes is a fundamental feature. Reproduction within and between individuals occurs by the meeting of female and male gametes, one of each type, in that precise combination, in a pattern recapitulated across almost all complex life. The binary system of sex is an evolutionary thread stitched through life on earth.

HUMAN SEX

We have established what sex is, that sex describes reproductive role by reference to gamete type, and that there are—and can only be—two sexes. We have also described some of the fascinating manifestations of the two sexes within individuals and within populations. In this section, we turn to developmental biology—the study of how organisms grow and, increasingly, how they age—which is replete with examples of complexity of form built from simple biological principles. The development of the reproductive human is one such instance.

The developmental biology underpinning this section is largely sourced from standard reference textbooks in the field. Readers may also wish to explore Baresi and Gilbert's Developmental Biology (online at the National Centre for Biotechnology Information) and Wolpert's Principles of Development.

Making a baby Humans are mammals and are—like almost all animals—divided into two classes of individuals according to reproductive role. In humans, the act of reproduction itself requires, in the first instance, male sperm to fertilise female eggs, achieved during intercourse between two sexually mature people. Male reproductive anatomy includes testes, contained in a sac of skin called the 'scrotum', that make sperm, delivered to the outside world through the penis. Both testes and penis are external organs, housed outside the male body, while female reproductive anatomy is almost wholly internal. It comprises ovaries that periodically release mature eggs, collected by the nearby oviducts (also called Fallopian tubes) and transported towards the uterus, the hollow muscular organ in which, in the event of a successful fertilisation event, a baby will grow. The uterus connects, via the cervix, to the vagina, which exits the body at the vulva, incorporating the clitoris and the urethral opening, surrounded by folds of skin called labia.

During intercourse, male sperm is mixed with water and lubricants from the seminal vesicles and prostate gland (to create semen), and the penis delivers the resulting semen into the female body via ejaculation. Semen moves through the cervix and uterus to the oviducts, where, should a mature egg be ready, fertilisation occurs. The fertilised egg is transported then implanted into the uterine wall, and commences development proper - at this stage, the female is pregnant. In the absence of a successful fertilisation event, the female body, having already prepared a blood-rich, spongey uterus lining suitable for implantation, breaks down this lining and expels it via the vagina during menstruation. In humans, gestation—the growing of a baby within the pregnant female uterus—lasts around nine months, after which the female gives birth, typically via the vagina (although surgical interventions like caesarean section, where the baby is removed via an incision through the uterus wall, may be necessary in negative medical circumstances or elected as a preference).

Sex determination Reproductive anatomy develops in utero, in a series of complicated yet elegant anatomical steps. The first step in reproductive development, however, is the determination of the future sex of a new embryo: female or male? In humans, sex is genetically-determined at fertilisation via the XY determination system of sex chromosomes. Females possess two X chromosomes, while males possess one X and one Y, with the Y chromosome carrying male-specific genes that activate male development. Given that sex chromosomes, like all other pairs of chromosomes, are divided individually when gametes are made, each human egg contains one X chromosome (and females are called ‘homogametic’) while human sperm contains either an X or Y chromosome (with males termed ‘heterogametic’). Offspring sex is thus dependent on whether an egg receives, at the moment of fertilisation, either an X or Y chromosome from the sperm.

The pattern of chromosomes within an individual is called a ‘karyotype’. Like all chromosomes, sex chromosomes carry genes. In humans, a key sex-determining gene is called SRY (sex-determining region Y) and it is, in genetically-healthy individuals, carried by the Y chromosome (Kashida and Koopman, 2010; Sinclair et al., 1990). The protein product of the SRY gene acts as a ‘master switch’ for male development, initiating a cascade of molecular genetic signals that drives the first anatomical step towards a sexed human body, gonad differentiation.

Embryonic development Gonad differentiation occurs at around six weeks in utero, when a bipotential pair of gonads—small buds of tissue in the abdominal cavity—are triggered to differentiate into ovaries or testes, the gamete-producing tissues in females and males, respectively. XY embryos carrying a functional SRY gene will trigger differentiation of testes via a network of molecular signals; in the absence of SRY activity, XX embryonic gonads begin to differentiate into ovaries, activating distinct molecular signals for that developmental pathway (Lecluze et al., 2020; Mamsen et al., 2017). There is feedback between these differentiation pathways; for example, a signal required for ovarian development—and the later maturation of eggs—also suppresses early testes differentiation (Jaaskelainen et al., 2010).

In embryological terms, gametes do not originate in the growing gonads. Rather, specialised stem cells migrate into the differentiating gonad region where they are embedded as the precursor cells that will ultimately become eggs or sperm, depending on gonad type (Magnusdottir and Surani, 2014). Ongoing gonad development into mature egg- or sperm-producing tissues relies on the differentiation of sex-specific gonad cell types, a process requiring tissue-specific hormone action. However, gonad differentiation into ovaries or testes also directs, via that sex-specific hormone milieu each generates, downstream events in reproductive anatomy development coordinated with future gamete type. That is, ovaries fated

to produce eggs will direct ongoing female development and testes fated to produce sperm will direct ongoing male development. In this sense, gonads can be considered as organiser tissues, coordinating the development of a reproductive system that integrates future gamete type with relevant reproductive anatomy; the absence of future gamete function—infertility—is no barrier to understanding the sex of human.

The first embryonic targets of gonadal organisation, from around eight weeks in utero, are two pairs of ducts running alongside the gonads called the paramesonephric and mesonephric ducts, which will grow into female or male internal genitalia, respectively. Both female and male embryos develop both pairs of ducts in early development; after gonadal differentiation, sex-specific hormonal action promotes growth of one pair over the other. Male testes secrete two major hormones that act upon these pairs of ducts. Testosterone promotes the growth of the mesonephric duct into male internal genitalia, and secreted anti-Mullerian hormone triggers degeneration of the paramesonephric duct, thus eliminating the duct that would develop into female internal genitalia. In females, the presence of ovaries means there is little testosterone to promote growth of mesonephric duct structures, nor anti-Mullerian hormone to trigger degeneration of paramesonephric duct structures. The female hormone environment thus permits growth of female internal genitalia, while the mesonephric duct (and the potential for male internal genitalia) degenerates.

The second embryonic target of gonadal organisation, from around 10 weeks in utero, is the development of external genitalia. The external genitals—vagina, clitoris and labia in females and prostate, penis and scrotum in males—derive from shared precursor tissues called the genital tubercle and urogenital fold. Under the influence of sex-specific gonadal hormones, these tissues are moulded into male or female form. Specifically, a derivative of testosterone (dihydrotestosterone) is produced locally—from testosterone—in the precursor tissues in males, and this derivative is a potent inducer of male external genitalia. In the converse situation, low testosterone and low dihydrotestosterone in females permits this precursor tissue to develop into female external genitalia. Given that male and female external genitalia develop from the same embryonic tissue under differential hormonal influences, analogous structures can be identified: the clitoris and penis share many structural features, while the labia represents an unfused version of the scrotum.

The sex of a newborn baby is routinely and reliably observed at birth by visual and palpable (‘touch’) assessment of external genitalia. Increasingly, the sex of a baby is identified in utero by observation of external genitalia or detection of sex chromosome karyotype/SRY gene presence. This is a matter of observation, woefully mischaracterised by the term ‘assignment’. The language of ‘assignment’ has been co-opted from serious medical decision-making in the case of clinical pathologies of the reproductive system (discussed below).

Puberty and secondary sex characteristics The development of reproductive anatomy in utero is called ‘primary sex development’, and the outcome is a body that has the potential to fulfil the male or female reproductive role. Human sex development undergoes a second phase of development at puberty, between the ages of 10-18 years old. This phase of secondary sex development generates divergence between the body shapes of females and males—a phenomenon called ‘sex dimorphism’—that has evolved under selection pressure to increase one’s likelihood of mating, following two broad strategies: be the most attractive or the most dominant. Both females and males gain height and bone density, experience the onset of libido, and experience typical teenage symptoms like acne and body odour. Under the influence of sex-specific gonadal hormones, female reproductive anatomy matures, ovulation and menstruation

commence, hip width increases, and breast tissue develops, in preparation for future motherhood. As well as experiencing male-typical maturation of reproductive anatomy (increase in testes volume and penile length), males gain greater height than females, grow facial hair, develop deeper voices, broader shoulders, and acquire far larger amounts of skeletal muscle than females.

Atypical sex development We have described the typical reproductive anatomy and sequence of events during development in healthy human beings. However, as a system with multiple biological inputs, steps and components, atypical or pathogenic development of reproductive anatomy can occur; in short, there are many points at which reproductive development can go awry. Collectively, medical conditions resulting from atypical reproductive development are called disorders (or, in patient-facing language, differences) of sex development (DSDs). There are around 40 known DSDs occurring in humans, most a result of mutations in genes required for the healthy development of reproductive anatomy in utero (Arboleda et al., 2014). The category of DSDs is broad, and it spans simple anatomic and hormone differences in otherwise healthy individuals to disorders with acute clinical sequelae that can cause postnatal harm or even death, and that need ongoing management throughout life.

Historically, DSDs have been described by terms such as ‘hermaphroditism’ and—currently falling into disuse—'intersex’. These terms are now deemed clinically-inaccurate and stigmatising to patients. Current nomenclature to categorise DSDs references karyotype and gonad status. Thus, the overarching categories are sex chromosome DSDs, XY DSDs and XX DSDs. For example, sex chromosome DSDs are exemplified by Turner syndrome and Klinefelter syndrome, where patients have irregular numbers of sex chromosomes and develop along typical female and male developmental trajectories, respectively, but experience hormonal issues that compromise sexual maturation and fertility. Other DSDs include conditions where female embryos are exposed to excessive testosterone in utero and develop an enlarged clitoris (an XX DSD called congenital adrenal hyperplasia) or where male embryos fail to produce the dihydrotestosterone required for penis growth (an XY DSD called 5 alpha reductase deficiency). Excellent resources on DSDs and their developmental etiology have been compiled, in collaboration with expert clinicians, by the UK charity DSD Families, and are available at their website.

The frequency of DSDs in the general population is the subject of much misinformation. Fausto-Sterling and her associates have defined as ‘intersexual’ any deviation from ‘ideal, Platonic’ male and female bodies, and arrived at a frequency of 1.7% of the population (Blackless et al., 2000; Fausto-Sterling 2000). Such a loose definition of DSDs captures a large number of people with no biologically-meaningful ambiguity of sex in any aspect of their development (most egregiously, the vast majority of this reported frequency are unambiguous females, often mothers, who have late-onset adrenal hyperplasia and, at some point post-birth, experience elevated testosterone levels as a result of an adrenal problem). This frequency of 1.7% was revised by Hull and Fausto-Sterling (2003) who, after identifying numerous flaws in the original studies, like failing to account for the sex-specific nature of many DSDs, revised the frequency to 0.4% of the population.

When assessing DSD frequency rationally restricted only to those individuals with ambiguous sexual anatomy or who exhibit a disparity between their reproductive (gondal) sex and external genitalia, the original frequency of 1.7% drops dramatically down to approximately 0.018 percent (Sax, 2002). That is, despite atypical sex development, almost all people are identifiable as either female or male. Within modern medicine, workflows to identify internal genitalia, karyotype and

hormonal profiles exist to identify sex in ambiguously-presenting people, and understanding DSDs within the framework of typical developmental trajectories of females and males aids not only diagnosis of these clinical disorders but also informs prognostic decisions regarding management of specific conditions in terms of sexual function and fertility prospects. Nonetheless, the inflated frequency of 1.7% is routinely-cited as definitive (for example, by Amnesty in 2018).

SEX MYTHS

In 2021, in a letter published in the Irish Journal of Medical Science (the official organ of the Royal Academy of Medicine in Ireland), we argued that, 'Public discourse around sex increasingly seeks to deny basic facts of human biology.' (Hilton and Wright et al, 2021). The driver of this anti-science movement is gender identity ideology, which claims that a privately held identity regarding one's sex is the ultimate definer of one's sex. That is, if a person identifies (in some internal, unverifiable sense) as female or male, that person literally is female or male. The overarching aim of gender identity ideologists is to deny that sex—reproductive role and associated characteristics—exists as a natural biological category. The intent behind such a belief appears to be to undermine the common scientific understanding and validity of viewing females and males as discrete biological categories in favour of a wholly subjective and unfalsifiable categorisation scheme based on one's personal and internal sense of self—gender identity. In this section, we will critique emerging misunderstanding, real or contrived, around sex.

Myth: sex is a composite score of body parts Underpinning ideological misrepresentations about sex is the conflation of sex (what is female?) with the physical characteristics associated with sex (how do we recognise female people?). That is, sex is not presented as anatomical patterns that develop in a co-ordinated fashion within the framework of an evolved function but as a checklist of seemingly-independent physical characteristics. This is often explicit; a Nature (2018) editorial asserted sex is, 'a classification based on internal and external bodily characteristics.' in a piece that failed to mention reproduction, the function of sex, or why humans have sexed bodies. And failed to acknowledge the obvious follow-up question: a classification based on internal and external bodily characteristics in which species? Of course, the reference species is assumed human, a peculiarly self-centred view of a biological phenomenon common to almost all complex life. In this sense, the conflation of sex with characteristics associated with sex retrospectively requires the redefinition of sex in every species on earth deploying anisogamy as a means of reproduction, while ignoring the unifying features shared by all.

Writing for The Skeptic in 2021, Hearne accurately defines 'female' as, 'organisms whose gametes are [...] ova or eggs.' yet immediately follows with, 'Unless you are a fertility doctor, it's unlikely you will encounter too many ova, so we must be using other definitions in everyday life.' While it is true that gamete type is not directly assessed in strangers, it does not follow that we use alternative 'definitions' when identifying the sex of a person; more accurately, we use alternative sex characteristics, those that arise from the organisational effects of the gonads (which also dictate gamete type) during primary and secondary development. Hearne claims that features like external genitalia—routinely covered—and breast size—plumped by bras—are insufficient to identify the sex of a stranger, and that we do so by features such as, 'amount and distribution of muscle and fat, the length and distribution of hair, the height and so on.' This is true; in fact, psychiatrist Nirao Shah, who studies behavioral differences between males and females, considers, 'correctly identifying [...] sex [is] a fundamental decision animals make.' (Goldman, 2019). Alongside basic assessments of body shape like shoulder and hip width, humans are expert with faces; sex identification is, 'an automatic and effortless aspect of face

perception' triggering differential brain activity (Kaul et al., 2011). Intriguingly, females are consistently better than males at recognising female faces, even in the absence of (often) gendered cues like hair length (for example, Lewin and Herlitz, 2002). Humans also assess movement like walking gait in sex identification (Pollick et al., 2005). However, none of these data points is, as Hearne's logic would have it, a 'definition' of sex, in the same way that observing the texture and density of a rock allows us to identify it as igneous, where 'igneous' is defined as a rock generated from volcanic lava.

Changing the definition of sex from function to form—explicit in pieces with titles like 'Sex Redefined' (Ainsworth, 2015) and where function is often discarded as irrelevant—is a necessary foundation upon which the deconstruction of sex as a biological category is built. Following the redefinition of sex as a checklist of physical characteristics, claims regarding variability of characteristics can flourish, along two lines of argument. First in line are those people with DSDs who have atypical reproductive development. The description of sex characteristics in people with DSDs sometimes disaggregates a reproductive system into constituent parts like 'genetic sex', 'gonad sex', and so on, to better understand incongruent features, clinical management and prognostic outcomes in people with DSDs (for example, Arboleda et al., 2016). For nearly all people, these constituent parts are aligned—or at least not divergent in any meaningful way—and disaggregation has no utility. If such disaggregation can be considered useful, it is not in the redefinition of female and male sexes, but in the refinement of workflows that generate a complete clinical picture for those people with DSDs. However, since the coining of 'gender identity' by John Money in the 1960s, component parts of sex have occasionally included concepts of 'psychological' and 'social' sex (Moore, 1968), paving the way for 'identity' to be considered a sexed characteristic.

The second line of argument evokes those sex characteristics, like height and hormone levels, that can overlap between the sexes, to attempt to demonstrate that there is no clear boundary between the female and male sexes in humans, and that, 'there is no one parameter that makes a person biologically male or female.' (Elsesser, 2020). The aim here is to destabilise the established categories of female and male. It is, of course, true that, for example, many females are taller than many males, or that some males have low levels of testosterone more typical of the female sex. However, such arguments fail to acknowledge an elephant in the room—we can only know that males are typically taller and have higher testosterone levels than females if we have a means to divide and measure humans by sex, independent of height and testosterone level. And it is centuries of knowledge accrued by the study of sex as a functional property of a species, and the anatomic/molecular organisation of the human species around that evolved function, that serves as that reference point. Put simply, it would be impossible to claim that low and high testosterone levels are correlated with being female and male, respectively, unless the categories female and male already had established meanings that testosterone levels were being correlated with. And the same holds for every other sex correlate.

Myth: sex is not binary Having remapped the definition of sex from function to form, introduced exceptions—arising from clinical disorders—to Fausto-Sterling's 'Platonic ideal', and attempted to blur category boundaries in healthy humans with trivial observations of naturally-overlapping sex characteristics, various commentators have attacked the phenomenon of sex as a binary system, often failing—deliberately or otherwise—to understand what the term 'binary' means when applied to sex. Writing for the Guardian in 2015, Heggie claims 'binary sex' means, 'the idea that there are men and women and they can be clearly distinguished.' (Heggie, 2015). Cade Hildreth (2022) claims that, 'sex is not binary because people cannot be grouped into two separate, non-overlapping groups.' These are straw man arguments.

The functional system of sex is routinely-described as ‘binary’ (including, on many occasions, by us). The use of ‘binary’—meaning, ‘of, pertaining to, characterised by, or compounded of, two’ (Oxford English Dictionary)—in this context intends to indicate, simply, a biological system with two components, and follows the same etymological pattern by which, for example, a system composed of two stellar masses is described as a binary star. Use of the word ‘binary’ operates at a system level across all species employing anisogamy.

However, having constructed a straw man argument that sex in humans is not binary, rejection of the term ‘binary’ is extended into rejection of ‘two’ itself, and the substitution of ideological framings of sex that move the conversation far from biological reality. Many interlocutors posit quantitative descriptions of sex as the necessary alternative to categorical descriptions. The most common quantitative (continuous) data distribution used to frame sex is a bimodal distribution, whereby various quantifiable traits associated with sex, such as adult height and testosterone levels, are conceptualised as multiple, overlapping distributions. These overlapping distributions of individual traits are purported to generate two modes that represent the average or typical female and male (as described by a combination of their average or typical sex characteristics), while shoulders for each mode permit for variation of sex characteristics. Routinely plotted on a horizontal axis crudely labelled ‘sex’, this framework gives rise to the premise that one’s sex is a statistical score generated by measuring multiple quantifiable characteristics. For a widely-circulated conceptualisation of ‘bimodal sex’, Hildreth (2022) describes the modes as, ‘peaks in a graph [that] represent probability clusters.’ Further to claims that sex is bimodal are claims that, ‘The science is clear—sex is a spectrum.’ (Brusman, 2019), an expression of a continuous distribution that replaces modes with, in the words of Brusman, ‘unlimited options.’ The corollary is that the sex of every human is unique to that individual, or, in the words of Fausto-Sterling when considering The Five Sexes, Revisited (2000), ‘Rather than identify a specific number of sexes [...] sex and gender are best conceptualized as points in a multidimensional space.’

The outcome of categorising sex as the sum of continuous descriptions of sex traits is that every person is scored as some percent male or female. The often-denied logical progression of such scoring is that a male with lower than average testosterone, petite stature, or a smaller than average penis, is shifted away from the male mode towards the female mode (typically occupied by people with low testosterone, petite stature and no penis). Such males, by this framework, are scored as ‘more female’ than counterparts with average or high testosterone, great stature and large penises. These damaging judgments equally extend to females with enlarged clitorises, small breasts or increased musculature, who, by the above logic, are scored as ‘more male’ than their larger-breasted and less athletic counterparts.

As sex within a continuous framework becomes a matter of sliding people left or right towards and from typical female and male, the middle of this distribution is cast as the no man’s land where—plus ça change—people with DSDs are placed. For those with little comprehension of DSDs beyond vague imaginings that people with DSDs have ‘both sets’ of genitals, this is intuitive. However, DSDs do not present as random combinations of primary and secondary sex organs, and neither do they simply differ by degree from one another. Rather, DSDs represent dozens of conditions with unique etiologies that manifest in disparate ways. There is no single medical category that is ‘intersex’ nor is there a robust method of ordering them, as would be necessary of a quantitative/continuous distribution of sex. Attempts to order categories of DSD into some continuous distribution are doomed to fail—entirely reasonably—if one cannot order

basic properties like sex chromosome conformation or gonad type within a continuous distribution (for example, Montanez, 2017).

Myth: sex is a social construct The spider's web of arguments touched upon here—and including the occasional reminder that sex development is very complicated (Sun, 2019), as if scientists are not well-trained in dissecting complexity to understand fundamental principles—culminates with the premise that the biological categories of sex are constructed by humans. Butler (1990) writes, 'Perhaps this construct called 'sex' is as culturally constructed as gender.' While it is true that scientists observing the natural world develop language and models to describe the natural world, one cannot credibly argue that the phenomena themselves are constructed by humans. If that were the case, not only have humans invented sex but they have also invented stars, gold, clouds and penguins. We have seen that sex is a fundamental property of almost all complex life, and its evolutionary existence pre-dates the human capacity to describe it.

The argument that sex is socially- or culturally-constructed settles, then, at the boundaries between sex categories, and the asserted arbitrariness straddling a fuzzy boundary (an important 'proof' that sex is not observed but 'assigned' at birth). However, the assertion that the categories of male and female are arbitrary because some rare individuals may present with ambiguous sexual anatomy is like asserting that the two different sides of a coin are arbitrary because there exists a non-zero probability a coin may land on its edge. The fact that sex may be ambiguous for some does not call everyone's sex into question. The categories described in humans by 'female' and 'male' are stable, functional, and the dividing line has emerged from observation of our (and other) species, not a coin toss.

Myth: biologists have alternative understandings about sex Finally we challenge the premise that some new scientific consensus on sex has emerged. Writing for German news site DW, Sterzik (2021) claims, 'Yet the broad scientific consensus now looks different: sex is a spectrum.' The definitions and understandings of sex we present in the first two sections of this chapter are uncontroversial, appearing in dictionaries, key biology textbooks and medical consensus statements like that issued by the Endocrine Society (Barghava et al., 2021). There is a vast literature which depends, explicitly or implicitly, on these understandings of sex. Searches on the scientific publication database PubMed for "male" [AND] "sperm" or "female" [AND] "egg"—that is, not exhaustive searches—retrieve around 100,000 results each, including numerous and recent publications from Nobel Laureates in Physiology or Medicine, and from a huge array of biological and medical disciplines.

Furthermore, searches (performed on 9th July 2022) for phrases like 'bimodal sex', 'sex is bimodal', 'spectrum of sex', 'sex is a spectrum' or 'sex is a social construct' generates no results in the biological or medical literature, although two close matches for 'sex is a spectrum' are returned. The first is a study of how sex—female or male—affects the spectrum of genetic variations acquired in the X chromosome over a lifespan (Agarwal and Przeworski, 2019). The second is a study of fetal sex—female or male—affects the spectrum of placental conditions experienced during pregnancy (Murji et al, 2012). Neither study demonstrates any confusion—quite the opposite—about the nature of sex, and both exemplify the importance of understanding sex in a clinical setting. Although not an exhaustive search, it seems that claims of a new scientific consensus—or, at minimum, an academic divide amongst biologists—regarding sex are rather overblown. Such claims are simple appeals to authority, absent from the scientific literature and apparently manufactured by public commentators.

CONCLUSIONS

In this chapter, we have seen that the most prevalent mechanism of reproduction in complex species has stabilised on a binary system of differential gamete types, and the subsequent evolution of body types around this binary system. The majority of species, including humans, are composed of individual females and males, defined by reproductive role, describing their contribution of large, energy-rich gametes (like eggs) or small gametes (like sperm), respectively, to the next generation.

In humans, notwithstanding atypical reproductive development, there are two evolved anatomical body types, each corresponding to one of the two reproductive functions. In utero, females and males develop sex-specific primary characteristics pertinent to reproduction, in the first instance the differentiation of gonad type that will direct future female or male function. Gonads—ovaries or testes, determined in humans by genetic mechanisms—are responsible for both the development of mature gametes (eggs or sperm) and, via hormones, the coordinated development of the relevant reproductive system. In adults, male anatomy comprises testicles, internal genital structures like the vas deferens, and an external penis and scrotum. Female anatomy comprises internal ovaries, internal genital structures like a uterus and vagina, and an external vulva incorporating the clitoris.

Finally, we have dissected arguments that attempt to challenge these basic understandings of sex. We have revealed that the redefinition of sex from an integrated, anatomical system organised around an evolutionary function to a checklist of human-centred, disaggregated physical characteristics is the foundation on which variability of those physical characteristics (in natural or pathological development) is used to deconstruct sex as a binary system, rendering it a construct of the human mind and, if it suits one's political aims, meaningless. We reject such arguments as purely ideological, with no evidence they are taken seriously in the scientific community, lacking explanatory power, and ultimately spurious. Despite the offered alternative frameworks to describe sex, the foundation that is the binary system shines through, underpinning the bimodal peaks of traits or dictating with which other 'point in multidimensional space' a person can successfully reproduce.

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BIOGRAPHIES

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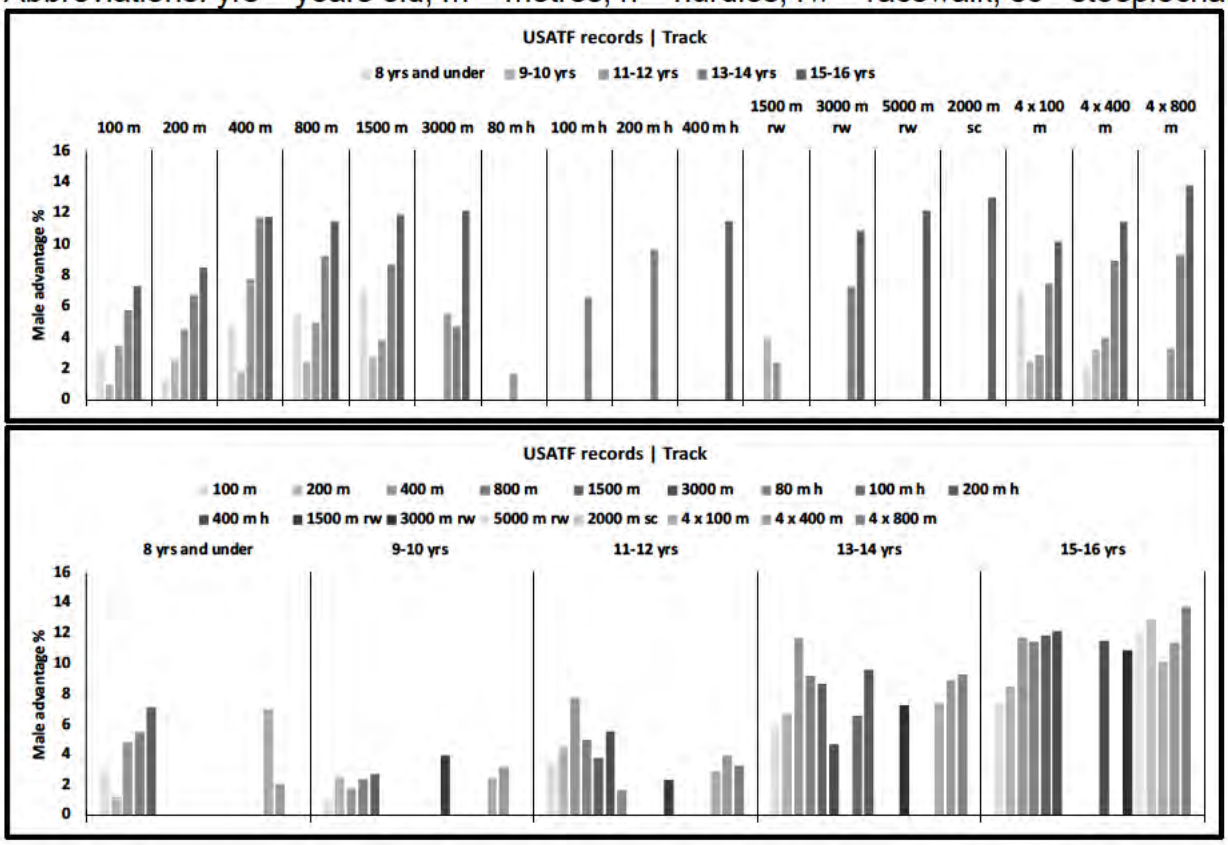
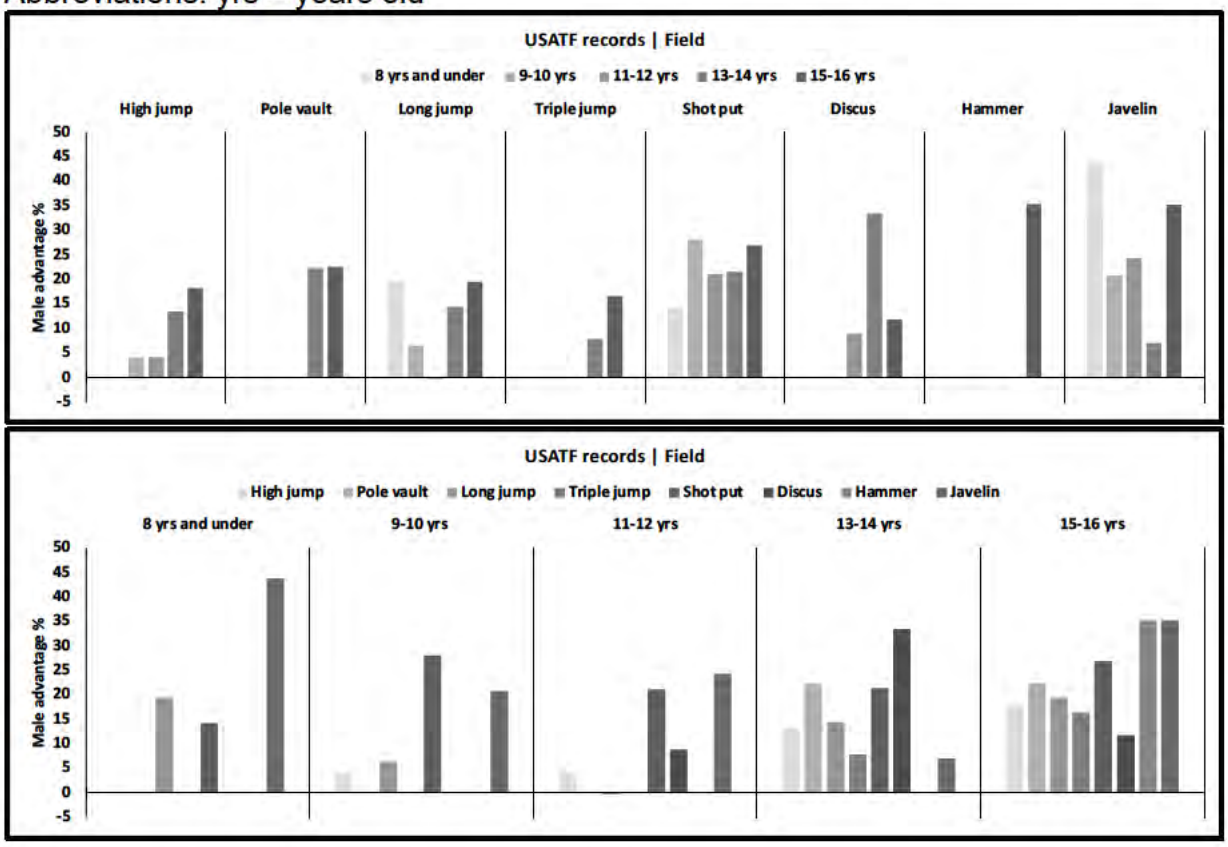
Appendix 3.
USATF junior records from 8-16 years old; analysis of male performance advantage.

Figure A3.1. The male advantage over females in USATF schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase



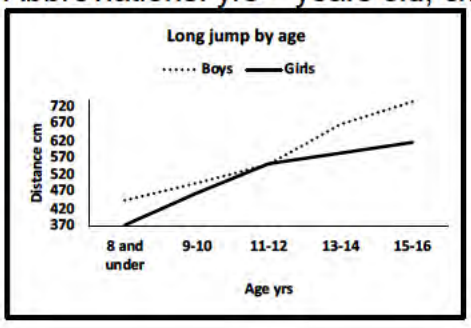
Male advantage is evident in all track events at all ages.

Figure A3.2. The male advantage over females in USATF schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages, except long jump/11-12 years old (female advantage 0.2%).

Figure A3.3. Age progression in the long jump in USATF schoolchildren records.
Abbreviations: yrs – years old, cm - centimetres



For long jump at 11-12 years old, female advantage is explained by the convergence of slightly poor male performance and good female performance; perhaps due to pubertal growth spurt in female athlete.

Figure A3.4. Male versus female "wins" in USATF schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

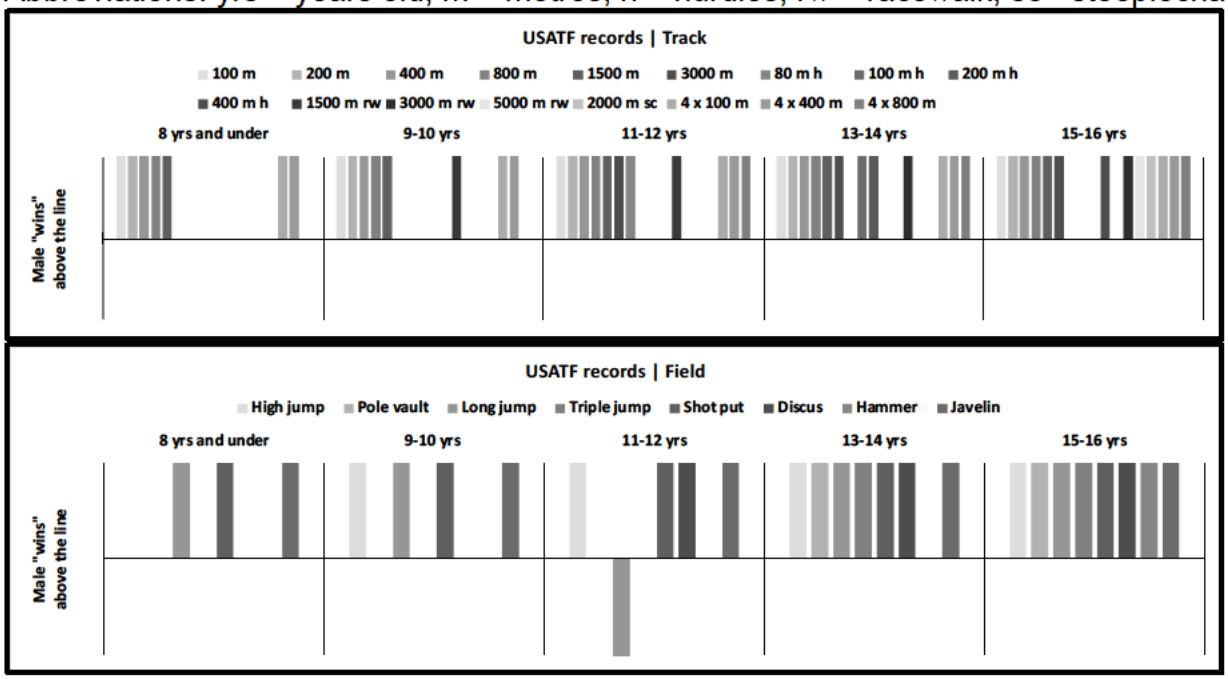
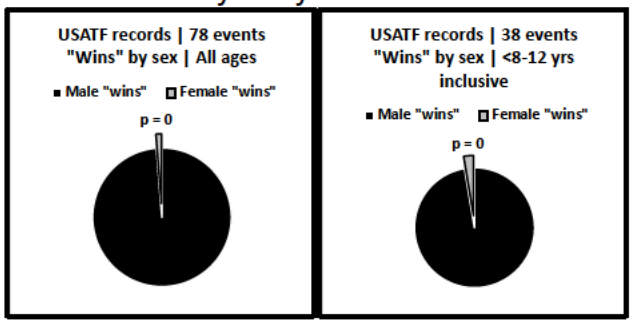


Figure A3.5. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old

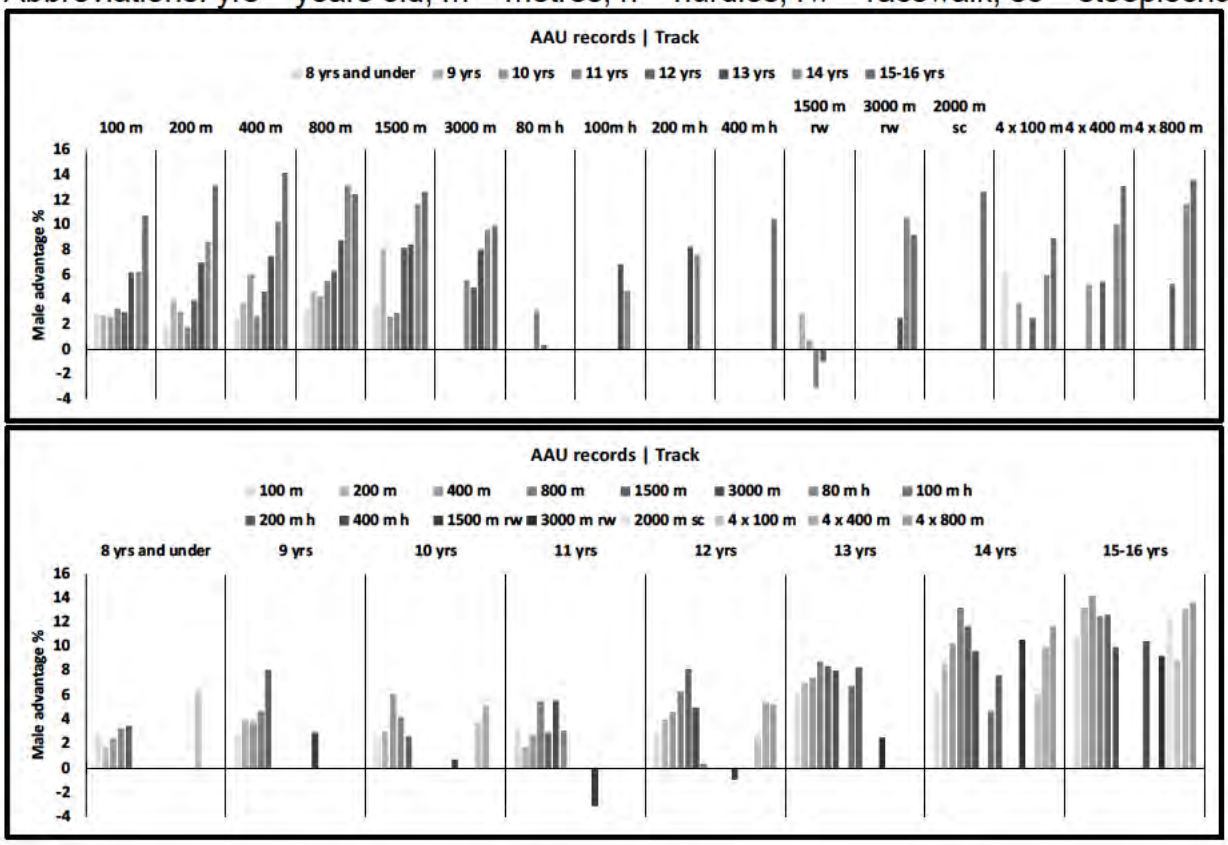


The probability of this frequency of male "wins" occurring by chance, either at all ages or limited to pre-puberty ages, is calculated at as effectively zero (p = 0).

Conclusions from USATF junior record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is vanishingly unlikely to result from chance.

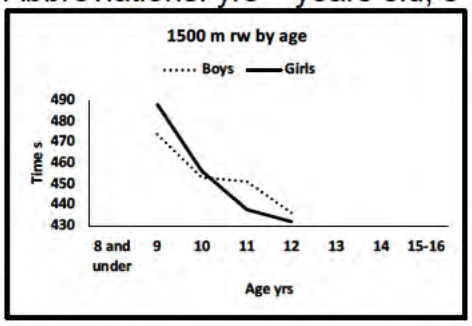
Appendix 4.
AAU junior records from 8-16 years old; analysis of male performance advantage.

Figure A4.1. The male advantage over females in AAU schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc – steeplechase



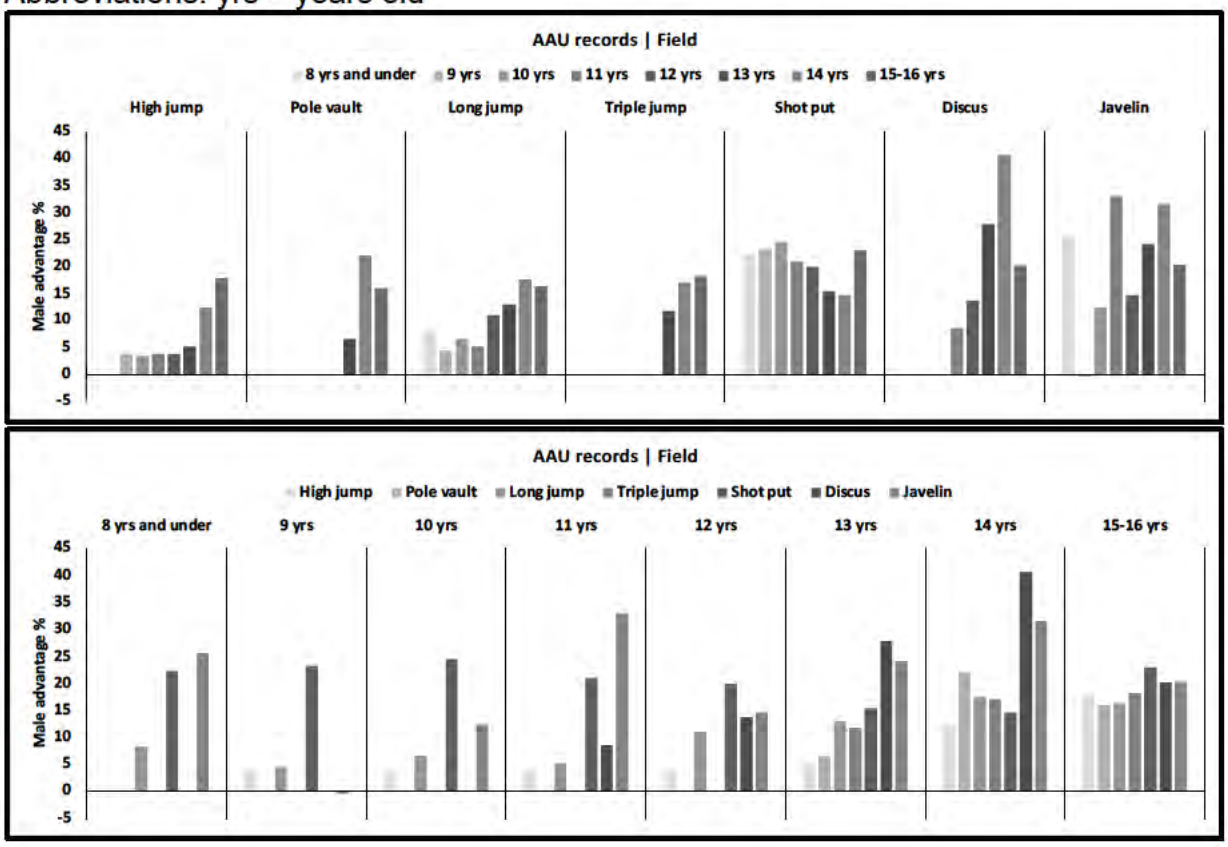
Male advantage is evident in all track events at all ages, except 1500 m rw/11 years old (female advantage 3.1%) and 12 years old (0.9%).

Figure A4.2. Age progression in the 1500 m rw in AAU schoolchildren records.
Abbreviations: yrs – years old, s - seconds



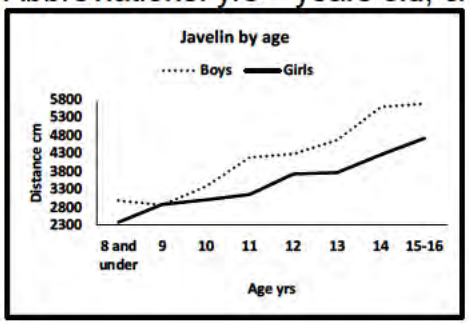
For the 1500 m rw at 11 years old and 12 years old, female advantage is underpinned by good female performances, perhaps explained by pubertal growth spurt synergising with the hip and joint flexibility required for racewalking. This female advantage is transient, and not evident in older age groups in the 3000 m rw event.

Figure A4.3. The male advantage over females in AAU schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages, except javelin/9 years old (female advantage 0.4%).

Figure A4.4. Age progression in the javelin in AAU schoolchildren records.
Abbreviations: yrs – years old, cm - centimetres



For javelin at 9 years old, female advantage may be explained by unexpectedly poor male performance converging with good female performance.

Figure A4.5. Male versus female "wins" in AAU schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

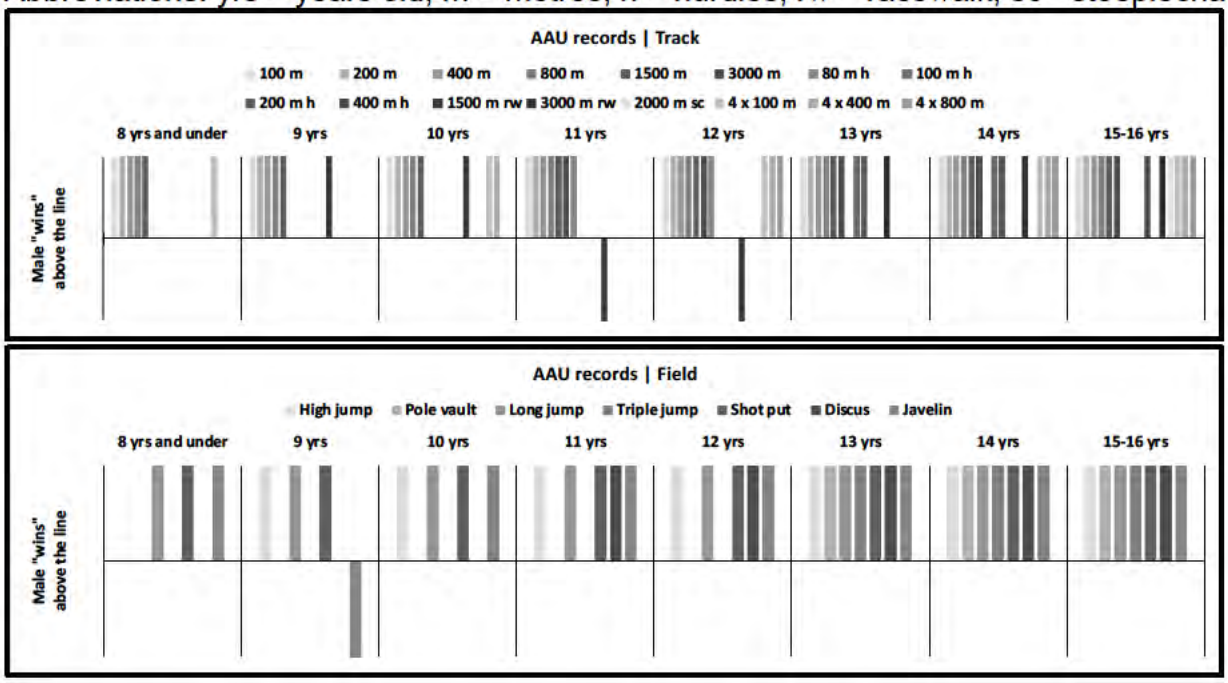
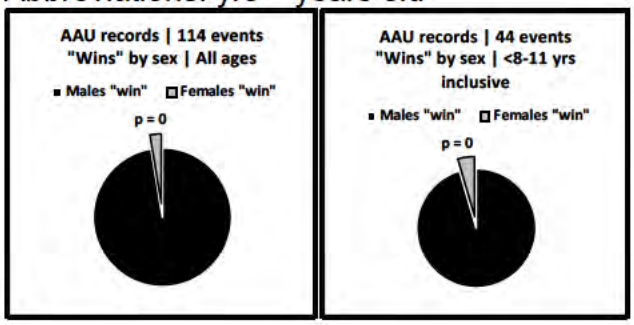


Figure A4.6. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old

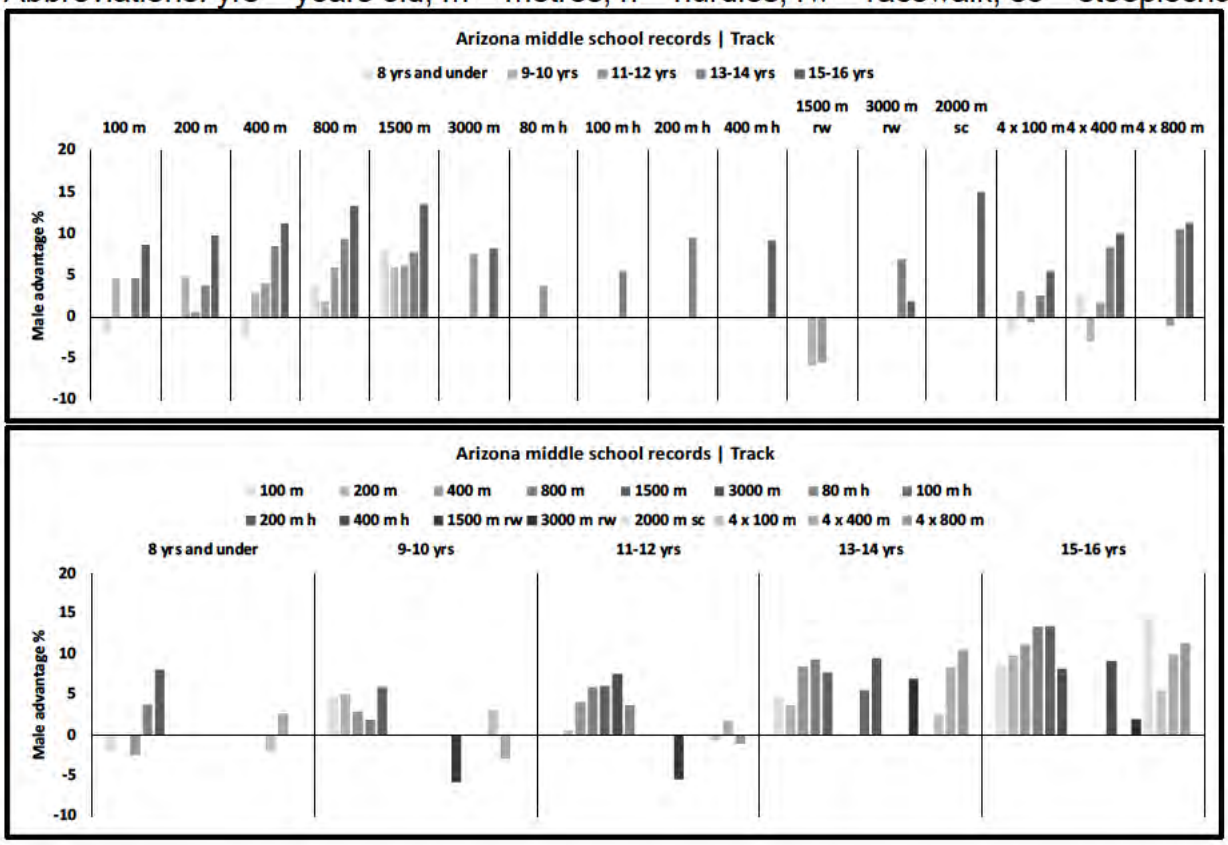


The probability of this frequency of male "wins" occurring by chance, either at all ages or limited to pre-puberty ages, is calculated at as effectively zero (p = 0).

Conclusions from AAU junior record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is vanishingly unlikely to result from chance.

Appendix 5.
AZ middle school records from 8-16 years old; analysis of male performance advantage.

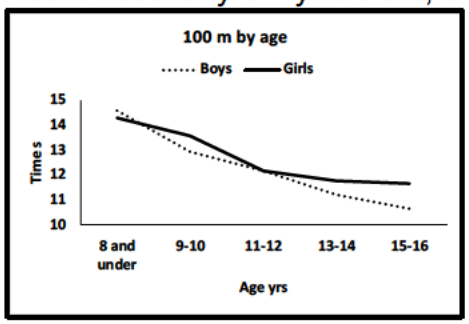
Figure A5.1. The male advantage over females in AZ middle school records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc – steeplechase



Male advantage is evident in most track events at all ages. Female advantage was calculated in 9 events:

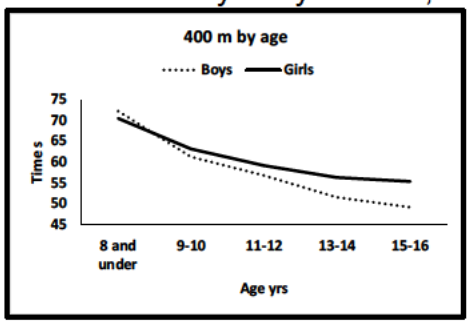
Event	Age group	Female advantage %
100 m	8 yrs and under	2.0 %
	11-12 yrs	0.0 % (same record)
400 m	8 yrs and under	2.4 %
1500 m rw	9-10 yrs	5.8 %
	11-12 yrs	5.4 %
4 x 100 m	8 yrs and under	1.9 %
	11-12 yrs	0.6 %
4 x 400 m	9-10 yrs	2.9 %
4 x 800 m	11-12 yrs	1.1 %

Figure A5.2. Age progression in the 100 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



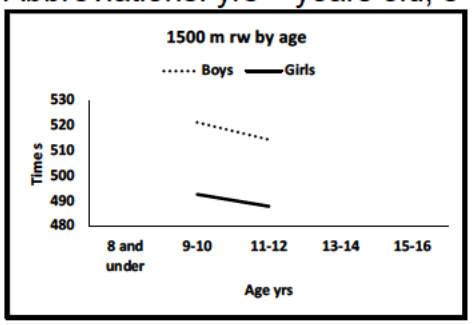
For 100 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. The dead heat at 11-12 years old may be explained by good female performance; perhaps due to pubertal growth spurt in female athlete.

Figure A5.3. Age progression in the 400 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



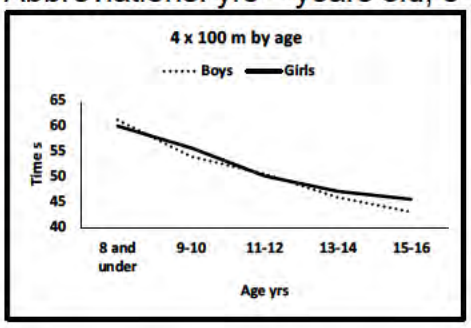
For 400 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here.

Figure A5.4. Age progression in the 1500 m rw in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



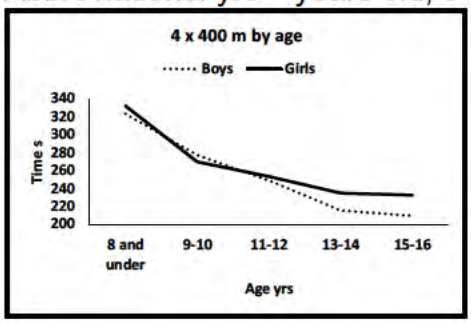
For the 1500 m rw at 9-10 years old and 11-12 years old, female advantage is likely underpinned by good female performances, perhaps explained by pubertal growth spurt synergising with the hip and joint flexibility required for racewalking. This female advantage is transient, and not evident in older age groups in the 3000 m rw event.

Figure A5.5. Age progression in the 4 x 100 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



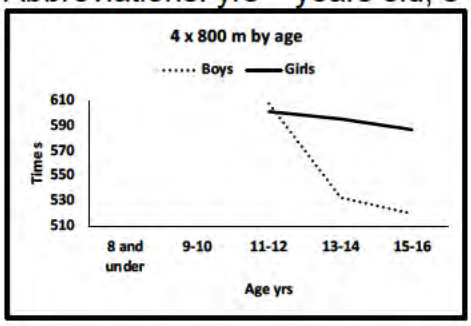
For 4 x 100 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. The female advantage at 4 x 100 m/11-12 years old may be explained by good female performance; perhaps due to pubertal growth spurt in females.

Figure A5.6. Age progression in the 4 x 400 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



The female advantage at 4 x 400 m at 9-10 years old may be explained by good female performance; perhaps due to pubertal onset.

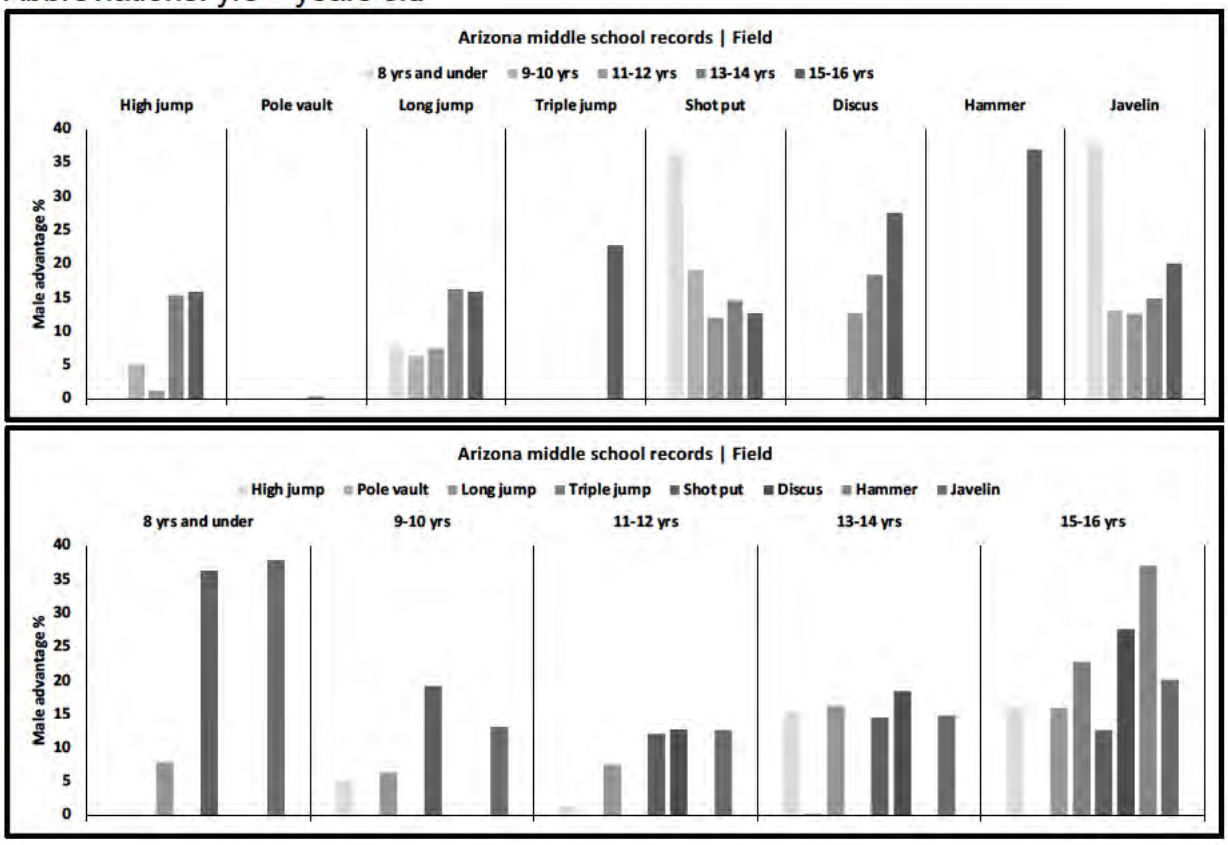
Figure A5.6. Age progression in the 4 x 800 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



For 4 x 800 m/11-12 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. However, the unusually-steep male trajectory to 13-14 years old indicates the female advantage at 11-12 years old is likely underpinned by unexpectedly poor male performance.

Figure A5.7. The male advantage over females in AZ middle school records in field events, stratified by event (upper panel) and age group (lower panel).

Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages

Figure A5.8. Male versus female "wins" in AZ middle school records, scored in track events (upper panel) and field events (lower panel).

Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

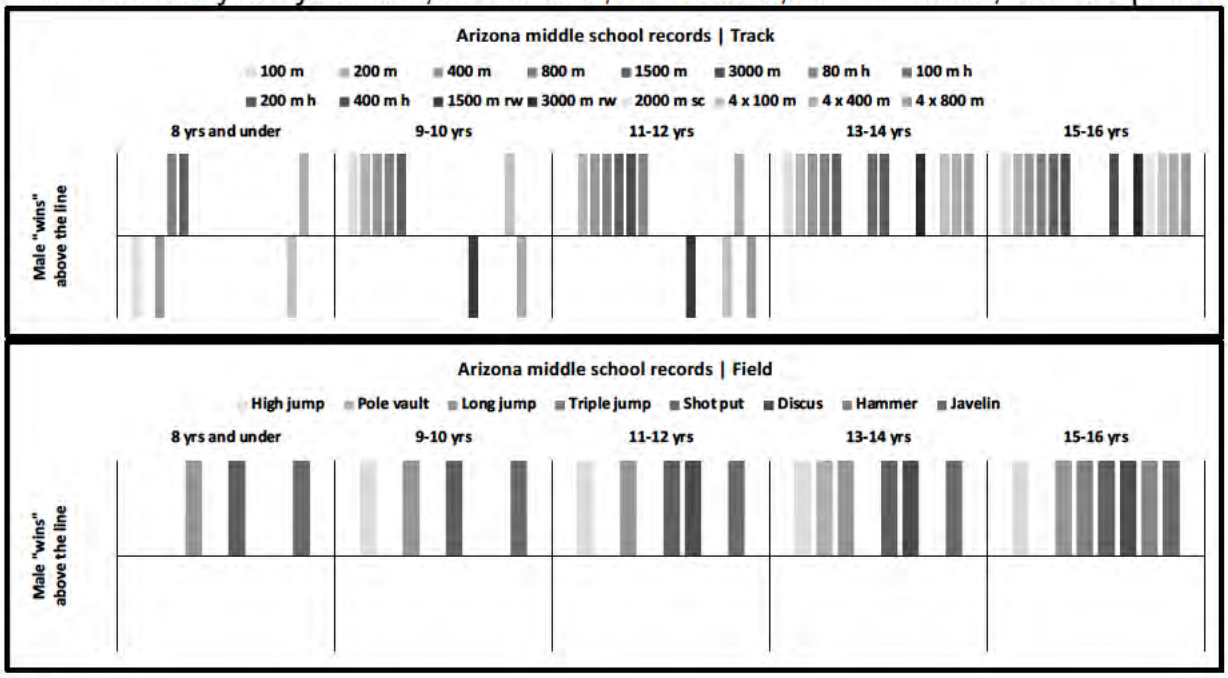
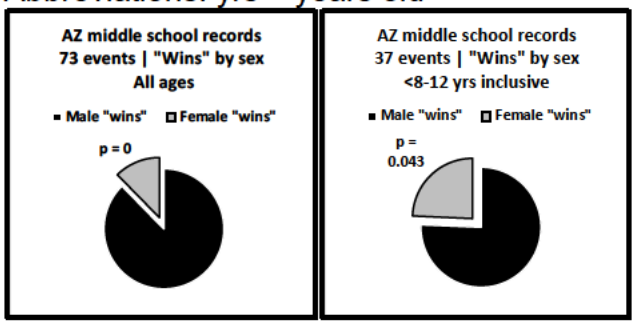


Figure A5.9. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old



The probability of this frequency of male "wins" occurring by chance at all ages is calculated at as effectively zero ($p = 0$). The probability of this frequency of male "wins" occurring by chance at pre-puberty ages is calculated as $p = 0.043$, where $p > 0.05$ represents the threshold of statistical significance in this test. Note: the dead heat in 100 m at 11-12 years old was scored as a female win, to faithfully test the limits of this analysis.

Conclusions from AZ middle school record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is unlikely to result from chance.

EXHIBIT 5

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UNITED STATES DISTRICT COURT

DISTRICT OF ARIZONA

Jane Doe, by her next friends and parents Helen Doe and James Doe; and Megan Roe, by her next friends and parents, Kate Roe and Robert Roe, **Plaintiffs,** **v.** **Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction, *et al.*,** **Defendants.**	**Case No. 4:23-cv-00185-JGZ** **DECLARATION OF DR. LINDA BLADE, Ph.D, IN SUPPORT OF DEFENDANT HORNE'S RESPONSE TO PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION**

I, Linda Blade, declare as follows:

I submit this expert declaration based upon my personal knowledge.

If called to testify in this matter, I would testify truthfully based on my expert opinion.

QUALIFICATIONS

As a former Canadian Champion (1986) and a full-scholarship NCAA All American (1984) in Track & Field (heptathlon) out of the University of Maryland (1982-1985), I worked hard to be a top student. Academic honors included being named Provost Scholar and member of Phi Beta Kappa.

Now licensed as a Chartered Professional Coach by the Coaches of Canada Association with a PhD in Kinesiology (earned in 1994), I have worked for over 30 years as a "Sport Performance Professional" coaching hundreds of athletes from 5 to 70 years of age, beginner to elite, from many different sports: track & field, hockey, soccer, volleyball, basketball, rugby, triathlon, sailboat racing, football, tennis, squash, swimming, diving, gymnastics, figure skating, skiing and bobsledding.

In my profession as a coach, I blend concepts in human biology with practical coaching methods acquired through many years of personal learning and mentorship opportunities as both athlete and coach. The unique way that I integrate theory and practice has proven to be highly effective. Many top athletes have sought my assistance at various times along their pathway to excellence. At the elite level, I have worked with National Hockey League (NHL) professional players (Edmonton Oilers dryland training, 2016-2018), mentored a world-leading female triathlete (Paula Findlay, 2009-2010) and helped train Pairs Figure Skaters, Jamie Salé and David Pelletier, to an Olympic Gold Medal (2002, Salt Lake City).

Truthfully, though, my greatest accomplishment as a coach has been working with beginners; young athletes ages 6 to 12 years.

It started during my first summer vacation after my freshman year in university. Needing a summer job that would be near the track where I had to continue training, I decided to offer a community "Run, Jump, Throw" camp for kids. Over 200 showed up and seemed to enjoy my coaching. Hosting that camp as a private enterprise became my summer job for consecutive years

of college. I learned how to train children and how to help them improve movement skills that would lay a strong athletic foundation for future success in sports.

Almost a decade after those early years of coaching, my life took an interesting turn. I had finished my PhD in Kinesiology with a subspecialty that focused on measurement of physical growth and development of children (anthropometry), and I was stationed in northern Nigeria (West Africa) at the location that is predominantly Islamic. (This is the same region where the Islamic militant group Boco Haram operates.)

The main university in that region is Bayero University, Kano (BUK). I got my first faculty position there in the Department of Physical Education. Admittedly, it was a bit strange to have a Canadian woman (me) teaching courses, including track and field activity courses, to prospective teachers at one of the top centers of Islamic Studies in Africa.

World Athletics got wind of this situation all the way over in Monaco and suddenly I was recruited (1993) by the CEO of World Athletics' global coaching development, Bjorn Wangemann. His plan was to train and send a world-leading female instructor (me) into Islamic countries to teach women how to coach young girls. There was, of course, a need in religiously segregated places to have female instructors deliver the global coaching certification programs.

This is how I came to be teaching the World Athletics Level 1 (for beginners) coaching curriculum in various countries during the 1990s: in Bahrain, Puerto Rico, Guyana, Kenya, and Sri Lanka.

The highlight of that experience was the course I taught in Iran in July of 1995. I was sent into Tehran to deliver the World Athletics certification course to 30 of the top female coaches selected from across that country. I was the first Western woman since Ayatollah Khomeini's 1979 revolution to travel to Iran for the purpose of engaging women and girls in sport.

For me, personally, that trip to Iran was a wakeup call. I witnessed firsthand what life is like when women & girls are not respected nor given the same rights as men and boys in society. Navigating the "opportunity gaps" in search of training spaces where I could teach the women without male interference was unbelievably challenging. It showed me how vulnerable women's rights can be, including the severely limited access that women can have to their own sporting

experiences. I vowed to never again take such things as Title IX and open access to women's opportunities for granted. I could see that what women in the West have achieve in sports is historically unique and politically fragile.

In 1997 a story about my travels as a global coaching instructor appeared in Sports Illustrated.[i]

Once becoming a mother (1998) and I settled down to a life of coaching in Edmonton, Alberta. Almost immediately, I was approached (1999) by a leading authority in Canadian Track & Field with a special request to author a curriculum piece for basic athletics instruction of children ages 5-11. The timing was perfect. I poured every bit of knowledge I had acquired as top athlete, scholar of child growth, academic instructor, and global coaching lecturer into the Athletics Canada "Run, Jump, Throw" (RJT) program (2001).[ii] Eventually, the rights to that RJT program were purchased by the Hershey's Track and Field Youth Program (2007). A video describing the RJT program can be found here: https://www.youtube.com/watch?v=TQMEg2D0TTw.

More recently, I have authored an update to the RJT program for children called the "Mini Legends Program." [iii]

In 2014, after years of developing children's sports programs and coaching hundreds of athletes at all levels of expertise, I became nominated and voted into office as President of the Board at Athletics Alberta - the track and field association for the province of Alberta. It was while attending national meetings as president in 2018 that I became aware of a philosophy that

seeks to allow male athletes to self-identify into female competitions. I could see in an instant that this would be a catastrophe for female athletes.

Throughout my professional career, I have always maintained that it is unfair for males to compete with females at any age. I believe it is a clear example of discrimination on the basis of sex.

My argument as to why female children should have their own category will now be explained.

REASONS WHY PRE-PUBESCENT GIRLS DESERVE FEMALE-ONLY SPORTS

A few items require clarification before I delve into my rationale.

A. Terminology - For the sake of clarity in my usage of language I will use biological terminology to reflect sex, which is the key determinant of physical reality and performance. For a male-born child I use the word "boy" and pronouns "he/him" (irrespective of social identity). Likewise, for the female-born child I must use the word "girl" and pronouns "she/her."

B. Age delimitation - Since puberty onset can happen as early as nine years of age in some children (especially in girls, who mature on average two years earlier than boys) any comparison of boys and girls deemed to be strictly "pre-pubertal" must be delimited to data obtained at eight years of age and earlier. Therefore, any references I make to data collection and results for prepubertal school children will focus on the 6- to 8-year-old range.

C. Data artifact – In the age range of 9-11 years, due to the phenomenon I mention above, some of the top girls can appear to be "catching up" to the boys in measures of fitness and sport performance. Charts often show a narrowing of the sex differences during this age range. This narrowing of differences between boys and girls is a temporary outlier that arises from the early maturation of a few girls. It is important to note that this phenomenon does not happen for *all* girls at this age range. Therefore, as a coach I will never assume that just because

one of the girls (ages 9-11) outperforms her entire class during a drill that it means I should expect the rest of the girls to be able to perform at the same level.

REASON 1 – Physical

The effect of testosterone on human sexual differentiation is an important factor, albeit not the *only* factor in causing boys to have an advantage over girls in sports. "Sexual dimorphism" (male versus female body design differences) arises from the interaction of testosterone and male genetics encoded by the SRY gene (usually found on the "Y" chromosome). The presence of testosterone in the womb triggers a male baby to begin its journey down the pathway to male morphology. There will be thousands of ways (from the cellular level to the overall anatomy level) in which a male baby diverges in form and physiology from a female baby. Height and weight charts at birth are sex specific, of course.[iv] Key differences in brain circuitry and musculoskeletal features develop before birth and will play a role in providing the male child with advantages related to sport performance. These involve the stitching together of subnetworks in the brain that provide a male child with better movement control, coordination, visual and special awareness, and internal proprioception.[v]

The article cited here mentions that there are differences even in the relative bone lengths of the fingers at birth, with boys having a longer 4th digit (ring finger) relative to the 2nd digit (index finger) and girls having a longer index finger (a larger "D2:D4 ratio"). This seemingly insignificant observation hints at sex-based differentiation in skeleton and joints. As a coach I witness with regularity how little boys have so much more strength in their upper body (upper torso, arms, and shoulders) compared to little girls. This manifests most noticeably when children try to climb or do pull-ups. Indeed, when I look at the data charts included in the *President's Council on Physical Fitness and Sports* (1985)[vi], I see that the sex difference is stark when it

comes to such upper-body performance measures as pull-ups and flexed arm hang. Here is a summary of those data:

Average number of pull-ups at ages 6, 7 and 8:

Boys = 1.3, 1.8, 2.3

Girls = 0.7, 0.8, 1.0

Average time (seconds) a child can maintain the flexed arm hang at ages 6, 7 and 8:

Boys = 7.9, 10.6, 12.3

Girls = 7.1, 9.3, 9.7

The task of gripping a bar and pulling up one's own body weight involves a kind of "leveraging" of forces at the shoulder, upper torso, arms, and hands. In my educated opinion, the sex-based differences in this physical test strongly suggest that the bones and muscles of boys develop differently in structure. The shape of the shoulder joint, the angles of pull, the muscular strength, and durability of that entire set of bony and muscular levers, enables the boys to do so much more.

But, of course, there are differences in other measures, too. Data from the same *President's Council* tests include the following items:

- Mile Run (seconds)
- Long Jump (inches)
- 50 Yard Dash (seconds)
- Shuttle Run (seconds)
- 2 Mile Walk (seconds)
- Sit & Reach (inches)
- Sit-ups (number)

Here are the comparisons by age (highlighted scores are the ones where girls are equal or better):

At AGE 6

	BOYS	GIRLS
Mile Run	788.57	829.21
Long Jump	44.59	40.60
50 Yard Dash	10.22	10.68
Shuttle Run	13.47	13.88
2 Mile Walk	2038.02	2114.23
Sit & Reach	.64	2.43
Sit-ups	22.56	22.90

At AGE 7

	BOYS	GIRLS
Mile Run	726.96	789.73
Long Jump	47.36	43.30
50 Yard Dash	9.82	10.19
Shuttle Run	12.96	13.52
2 Mile Walk	2031.31	2146.35
Sit & Reach	.69	2.23
Sit-ups	27.16	25.37

At AGE 8

	BOYS	GIRLS
Mile Run	684.77	763.25
Long Jump	51.83	47.42
50 Yard Dash	9.27	9.71
Shuttle Run	12.39	13.15
2 Mile Walk	1969.93	2078.52
Sit & Reach	.18	2.06
Sit-ups	30.48	28.66

In summary, this testing protocol indicates that boys run faster, have greater endurance, are more agile, jump farther and have greater upper body strength than girls, whereas girls are more flexible (indicated here in the sit and reach test).

This sub-set of results from top finishers at the 2022 AAU National Championship Jr Olympics shows a similar outcome for 8-year-olds[vii]:

	BOYS	GIRLS
100m Dash (sec)	13.87	14.41
200m Dash (sec)	28.56	29.64
1500m Run	5:07.14	5:18.44
Long Jump (m)	4.09	3.86
Shot Put	31 ft 1.00 in	23 ft 4.75 in

This chart (above) provides additional evidence that is prototypical. Once again, boys are faster and throw and jump farther than girls. Measurements of lung function in small children - with boys having a higher lung volume[viii], more air passages and other enhanced capacities throughout the oxygen transport system[ix] - explains why they also do better in endurance tests and the 1500m run as reflected in the charts.

I leave it up to other experts like Dr. Gregory Brown and Dr. Emma Hilton, whose reports I have reviewed in preparing my opinion, to provide more such data. The point I wish to make

here is that consistently across all data bases and amongst the the hundreds of children I have worked with as a coach, boys are better than girls in all fitness parameters except in flexibility and, possibly, balance.

In the realm of physical education and sports we refer to human movement capacities as "biomotor abilities." Some coaches say there are only five, but I recognize ten biomotor abilities (with the main physical factors that influence them in brackets):

- Strength (nervous system, muscles, bone structure & joints)
- Speed (nervous system, muscles, bone structure & joints)
- Stamina (cardiovascular system – heart, lungs, blood & cellular substructures)
- Power (nervous system, bone structure, muscles & joint durability)
- Speed-Endurance (cardiovascular system, bone structure, muscles, nervous system & cellular substructures)
- Muscular-Endurance (cardiovascular system, bone structure, muscles, nervous system & cellular substructures)
- Coordination (proprioception, nervous system, muscles & joints)
- Agility (proprioception, nervous system, muscles & joints)
- Balance (proprioception, location of center of gravity, nervous system & muscles)
- Flexibility (softness of joints; extensibility of muscles and ligaments)

And possibly an 11th one that only top coaches talk about (& professionals like NFL quarterback Tom Brady)[x]:

- Elasticity or Pliability (the ability of the entire body or parts of the body to "whip" – to bend and snap like an elastic band)

Due to the underlying structural differences in the nervous system, musculo-skeletal system, and cardio-vascular system, boys have the advantage in nine out of the eleven biomotor abilities.

Girls do excel in sports where flexibility is a dominant feature. For example, boys typically don't compete in rhythmic gymnastics. It requires body contortions that most males are simply

unable to achieve. On the other hand, having hyper-flexible bodies accompanied by lower muscular strength renders girls are highly prone to impact injury in contact sports.

Since most sports involve a combination of biomotor abilities, the male performance advantage will be amplified. In a sport like volleyball, soccer, and basketball where strength, speed, power, endurance, agility, and coordination all come into play, the performance difference compared to the girls will be more obvious than what might be observed in a singular biomotor skill test.

This concept of "additive advantage" is the reason why changing one variable in a boy (say, testosterone level) will not work to fully diminish his performance advantage over his female counterparts. While hormone therapy might diminish a percentage of his original strength and, possibly, endurance, it will not adequately diminish other factors that add up to giving him an overwhelming advantage. For the sake of argument, if boys are better than girls because they are adding up a set of advantages "A + B + C + D + E + F," they will continue to have an advantage even if factor "D" is removed. The male advantage will then be of the set "A + B + C + E + F." It will *still be* insurmountable for the girls.

In summary, as a coach with extensive education in kinesiology – looking at human form and function - I can confirm without hesitation that prepubescent girls as a class will never be able to overcome the performance edge enjoyed by their male cohorts. While not as overwhelming as the differences encountered post-puberty, the sport performance differences enjoyed by pre-pubescent male children are significant and easily recognized by those of us involved: teachers, coaches, parents, and the children. The important point to be made here is that boys will dominate girls in competition because of prepubescent physical differences.

REASON 2 - Psychosocial

As a coach for almost 30 years observing boys and girls in sports competition, I have regularly observed the psychosocial risks of forcing girls to compete against boys. Most little girls simply do not wish to compete against the boys. Girls recognize the categorical difference in biological sex and, as a coach, I have seen quite often that little girls become intimidated when they are compelled to test themselves relative to boys. On a soccer field, a little girl will often

stand back and let the boy take the ball. In games like dodgeball girls will often shy away from the aggressive play of boys. Conversely, when little girls compete with each other their confidence grows and they become far more engaged in the match.

This is the same phenomenon witnessed in girls-only schools. A disadvantage with having to compete with boys is described thus: "In coeducational classrooms, boys tend to monopolise discussion, and take more domineering roles in group work and in practical exercise." [xi] And: "…teachers [and coaches] tend to ignore the strong correlation between high motivation and high anxiety in many high-achieving girls. In girls-only environments, girls' needs and preferences come to the fore."

Based on my observations and interactions with children and families over the course of my 30 years of coaching, I have repeatedly seen that the moment a boy is mixed in with the girls in a highly competitive environment, much of the focus turns to him and his needs at the expense of the girls, who tend to quietly withdraw their assertiveness. Recently, a father told me that his nine-year-old daughter's soccer team had to play against another team that had a male child who "identifies as a girl." He said that the girls on his daughter's team became less energized than usual and did not even try to take the ball away from the boy. Their team ended up losing by many points and the girls left the field asking why they should even be playing. This is the opposite of female empowerment.

Female empowerment takes another huge hit when male children are allowed to share a locker room with the girls. One needs only to hear the testimony of swimmer Riley Gaines to understand the devastation and humiliation involved in dealing with compelled sharing of an intimate space.[xii] It leads to tears and long-lasting psychological distress.

The essence of positive empowerment is what happened when female-only sports exploded in popularity after the passage of Title IX. The numbers don't lie. While there is no data for

primary schools, we can see what happened with older female students, as summarized in this chart:[xiii]

TIME	**MALE** participation in high school sports (number of boys)	**FEMALE** participation in high school sports (number of girls)
Before Title IX [School year 1971-1972]	3,666,917 (93%)	294,015 (7%)
After Title IX [School year 2018-2019]	4,534,758 (57%)	3,402,733 (43%)

These data show a 1,057% increase in female participation in school sports over a 45-year period. A similar increase is reflected in the NCAA data and the point is that never in the annals of world history has there been such a drastic change (improvement!) in the enthusiastic engagement and physical play of female persons.

The impact upon America has been unprecedented. Twenty years after the passage of Title IX (in the 1990s) along came the phenomenon of the "soccer mom" – mothers across America who piled their kids into the minivan determined to get their children into sports. A generation of both boys and girls now owe it to those moms for engaging them in sports and other physically active past times. Based on my observations, this volunteerism has had a positive impact on many children and on the sports associations.

One significant impact of granting girls the opportunity to engage in fair competition and to experience achievement has been on the American economy and the business environment. In clear contrast to the pre-1980s, there are now thousands of women across the USA who start their own businesses and lead companies.

What does this have to do with sports? Consider these facts revealed in an article by Forbes[xiv] magazine reporting on a study of working women undertaken by Ernst & Young:

"The study found that 90% of the women surveyed had played sports either at primary and secondary school, or during university or other tertiary education, with this proportion rising to 96% among C-suite women."

Almost all top female CEOs have had a sports background.

There can be no doubt that access to sport engendered by TitleIX has promoted the kind of self-confidence in America's little girls that has inspired them to grow into adult women pursuing high achievement. The benefit to society has been priceless.

CONCLUSION:

In conclusion, I must say that I am deeply concerned about the future of sports for young girls. We often hear the phrase, "Trans rights are human rights." This is true, but by the same token, "Female rights are human rights." Everyone has rights. But for an activity to be considered a "sport," the fundamental ingredient must be "fairness."

In 2021 when the UK Sport Council's Equality Group (SCEG) released its thorough review of transgender inclusion, it arrived at the following conclusion:

"As a result of what the review found, the guidance concludes that the inclusion of transgender people into female sport cannot be balanced regarding transgender inclusion, fairness and safety in gender-affected sport where there is meaningful competition."[xv]

According to the SCEG report, authorities in sex-affected sports must make a choice: prioritize transgender inclusion or prioritize fairness and safety for the female athlete.

I disagree in one way. I believe that we already have full inclusion in sports. Every human person has a biological sex, even if one wishes to self-identify or express as something different. Therefore, there can be a place for everyone within our sex-based eligibility systems.

Nobody benefits in the long run by mixing sports categories. It is my view that the Save Women's Sports Act preserves fairness in sports for female participants of all identities on the basis of sex, as intended by Title IX.

I swear or affirm under penalty of perjury that the foregoing is true and correct.

Dated: June 28, 2023 Signed: */s/ Dr. Linda Blade, Ph.D*

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[i]https://vault.si.com/vault/1997/08/25/teach-coaching-see-the-world-traveling-to-third-world-countries-to-train-coaches-is-linda-blades-idea-of-a-perfect-summer-vacation

[ii]chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.northumberlandsportscouncil.ca/wp-content/uploads/2018/08/Run-Jump-Throw-Resource-.pdf

[iii] https://minilegends.ca/

[iv] https://www.cdc.gov/growthcharts/clinical_charts.htm#Set1

[v]chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9331831/pdf/ijerph-19-09103.pdf (page. 3)

[vi] https://eric.ed.gov/?id=ED291714 (Appendix A, pages 56-57)

[vii] http://image2.aausports.org/sports/athletics/results/2022/jogames/jogamescompleteresults.htm

[viii] https://journals.physiology.org/doi/abs/10.1152/jappl.1962.17.4.601

[ix] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5980468/pdf/EDU-0003-2018.pdf

[x] https://www.youtube.com/watch?v=-SSP_qAUtYI

[xi] https://www.gdst.net/publications/why-and-how-girls-thrive-in-girls-only-schools/

[xii] https://www.dailysignal.com/2023/06/21/riley-gaines-describes-sharing-locker-room-lia-thomas/

[xiii] chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.nfhs.org/media/1020205/2017-18_hs_participation_survey.pdf

[xiv] https://www.forbes.com/sites/alanaglass/2013/06/24/ernst-young-studies-the-connection-between-female-executives-and-sports/?sh=7edab51333a2

[xv]https://equalityinsport.org/docs/300921/Guidance%20for%20Transgender%20Inclusion%20in%20Domestic%20Sport%202021.pdf (p. 15)

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UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, by her next friend and parents Helen Doe and James Doe; and Megan Roe, by her next friend and parents, Kate Roe and Robert Roe,

Plaintiffs,

v.

Thomas C. Horne in his official capacity as State Superintendent of Public Instruction; Laura Toenjes, in her official capacity as Superintendent of the Kyrene School District; Kyrene School District; The Gregory School; and Arizona Interscholastic Association Inc.,

Defendants.

Case No. 4:23-cv-00185-JGZ

PLAINTIFFS' UPDATED EXHIBIT LIST FOR PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION

Plaintiffs submit the following list of exhibits, along with copies of exhibits not already filed on the docket, pursuant to the Court's June 14, 2023 Order (ECF No. 80). Plaintiffs respectfully reserve the right to amend this exhibit list in advance of the hearing.

Ex. No.	Description	Location
1	Declaration of Jane Doe	ECF No. 6
2	Declaration of Helen Doe	ECF No. 7
3	Second Declaration of Helen Doe	ECF No. 78
4	Declaration of Megan Roe	ECF No. 8
5	Declaration of Kate Roe	ECF No. 9
6	Declaration of Stephanie Budge, Ph.D.	ECF No. 4
7	Rebuttal Declaration of Stephanie Budge, Ph.D.	ECF No. 65-1
8	Declaration of Daniel Shumer, M.D., MPH	ECF No. 5

9	Rebuttal Declaration of Daniel Shumer, M.D., MPH	ECF No. 65-2
10	AIA's Constitution, Bylaws, Policies, and Procedures 2022-2023, Transgender Policy	ECF No. 51-1
11	Photographs of the Doe Family (*filed under seal*)	ECF No. 108
12	Photographs of the Roe Family (*filed under seal*)	ECF No. 108
13	Jane Doe's Name Change Court Order (*filed under seal*)	ECF No. 108
14	Megan Roe's Name and Gender Change Court Order (*filed under seal*)	ECF No. 108
15	Jane Doe's Passport (*filed under seal*)	ECF No. 108
16	Megan Roe's Passport (*filed under seal*)	ECF No. 108
17	Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2nd Reg. Sess., 00:08:08–01:30:05 (*filed as a non-electronic exhibit*)	ECF No. 88-1
18	David Handelsman, et al., *Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance*, 39 Endocrine Revs. 803 (2018)	ECF No. 88-2
19	David Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clinical Endocrinology 68 (2017)	ECF No. 88-2
20	Jonathon W. Senefeld et al., *Sex Differences in Youth Elite Swimming*, 14 PLOS ONE 1 (2019)	ECF No. 88-2
21	Joanna Harper, *Race Times for Transgender Athletes*, 6 J. Sporting Cultures & Identities 1 (2015)	ECF No. 88-2
22	Marnee McKay & Joshua Burns, *When it Comes to Sport, Boys "Play Like a Girl,"* The Conversation (Aug. 3, 2017), https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328	ECF No. 88-3
23	Marnee McKay, et al., *Normative Reference Values for Strength and Flexibility of 1,000 Children and Adults*, Neurology, 88 (1) (2017)	ECF No. 88-3
24	World Rugby Transgender Women's Guidelines (2020), https://www.world.rugby/the-game/player-welfare/guidelines/transgender/women	ECF No. 88-3
25	Governor Douglas A. Ducey's Letter to Arizona Secretary of State re: Senate Bill 1138 and 1165	ECF No. 88-3
26	Second Declaration of Helen Doe	ECF No. 109
27	Second Rebuttal Declaration of Daniel Shumer, M.D., MPH	Attached

Respectfully submitted this 7th day of July, 2023.

/s/ Colin M. Proksel

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**Admitted pro hac vice.*

Exhibit 27

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UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, by her next friend and parents Helen Doe and James Doe; and Megan Roe, by her next friend and parents, Kate Roe and Robert Roe,

Plaintiffs,

v.

Thomas C. Horne in his official capacity as State Superintendent of Public Instruction; Laura Toenjes, in her official capacity as Superintendent of the Kyrene School District; Kyrene School District; The Gregory School; and Arizona Interscholastic Association Inc.,

Defendants.

Case No. 4:23-cv-00185-JGZ

SECOND REBUTTAL DECLARATION OF DANIEL SHUMER, M.D., IN FURTHER SUPPORT OF MOTION FOR PRELIMINARY INJUNCTION

I, Daniel Shumer, declare as follows:

1. I submit this expert declaration based on my personal knowledge.

2. If called to testify, I would testify truthfully based on my expert opinion.

3. In preparing this declaration, I reviewed the expert declarations submitted by Dr. Emma Hilton ("Hilton Decl.") and Dr. Linda Blade ("Blade Decl.") in support of Defendant Horne's Opposition to Plaintiffs' Motion for Preliminary Injunction. I also reviewed the rebuttal declarations by Dr. Gregory Brown ("Brown Rebuttal Decl."), Dr. Chad Carlson ("Carlson Rebuttal Decl."), and Dr. James Cantor ("Cantor Rebuttal Decl.") that the Intervenors submitted in support of their Opposition to Plaintiffs' Motion for Preliminary Injunction. As with my prior expert declaration, I relied on my scientific education and training, my research experience, and my knowledge of the scientific literature in the pertinent fields. The materials I have relied on in preparing this declaration are the same types of materials that experts in my field of study regularly rely on when forming opinions on these subjects. I may wish to supplement these opinions or the bases for them as a result of new scientific research or publications or in response to statements and issues that may arise in my area of expertise.

<u>Dr. Hilton's Declaration</u>

I. There Is No Evidence Linking In Utero Development or Minipuberty to Athletic Performance and No Credible Medical Reason to Posit Any Such Connection.

4. There is no scientific basis for Dr. Hilton's claim that boys gain an athletic advantage over girls based on exposure to testosterone in utero or during minipuberty. (Hilton Decl. ¶¶ 5.3–5.5.)

5. In a male fetus, testosterone production peaks around 11–14 weeks of gestation (in the first trimester of pregnancy), then declines until it is completely suppressed at birth. Testosterone is necessary during this time for normal development of the genitals. *See, e.g.*, Marianne Becker & Volker Hesse, *Minipuberty: Why Does it*

Happen?, 93 Hormone Rsch. Paediatrics 76 (2020).

6. Male babies also experience an elevation of testosterone after birth, with levels peaking between one to two months old, and returning to prepubertal levels before six months of age. As with the in utero elevation of testosterone, a rise in testosterone during minipuberty correlates positively with growth of the male genitals. *Id.* at 78–79.

7. Contrary to Dr. Hilton's testimony, minipuberty does not result in clinically visible physical changes, other than a possible transient increase in testicular volume.

8. In fact, although Dr. Hilton cites Becker & Hesse's article for the proposition that testosterone levels cause an increase in babies' growth velocity and body weight (Hilton Decl. ¶ 5.5), the article describes the opposite. Becker & Hesse found that testosterone and luteinizing hormone (the hormone that stimulates testosterone production) concentrations "during minipuberty correlate *negatively* with body weight and body mass index [BMI] until the age of 6 years." *Id.* at 80 (emphasis added). A negative correlation between testosterone level and body weight or BMI contradicts Dr. Hilton's assertion that minipuberty in males causes competitive athletic advantage later in life. In addition, the article found that "[d]ata on the influence of minipuberty on growth velocity are conflicting." *Id.*

9. No research has linked this brief exposure to elevated testosterone during minipuberty to any lasting physiological impact, much less to an increase in athletic ability. Nor is there any credible medical basis even to hypothesize such an impact.

II. There Also Is No Evidence Linking Gene Expression to Athletic Performance and No Credible Medical Reason to Posit Any Such Connection.

10. There also is no scientific basis for Dr. Hilton's speculation that boys gain an athletic advantage over girls based on sex-specific genetic architecture that results in approximately 6,500 differences in gene expression. (Hilton Decl. ¶ 5.2.) Dr. Hilton fails to cite any research to connect any differences in gene expression between the sexes to the purported athletic advantage of transgender girls who do not undergo male puberty.

11. Contrary to Dr. Hilton's testimony and as I have previously discussed, there is an overwhelming scientific consensus that the biological cause of average differences in athletic performance between men and women is the rise in circulating levels of testosterone beginning in endogenous male puberty. As Handelsman states, "evidence makes it highly likely that the sex difference in circulating testosterone of adults explains most, if not all, of the sex differences in sporting performance." *See* David J. Handelsman et al., *Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance*, 39 Endocrine Revs. 803, 823 (2018) (summarizing evidence rejecting the hypothesis that physiological characteristics are driven by the Y chromosome).

III. Any Height Differences Among Male and Female Babies Are Negligible and, in Any Event, Largely Disappear Around the Age of Six or Seven.

12. Dr. Hilton's claim that growth charts reveal that "[m]ales are consistently 1-2 cm taller than females between 0-10 years old" (Hilton Decl. ¶ 4.4) is false.

13. Growth charts show that babies' heights are heavily overlapped, with only negligible differences between boys and girls, which differences almost disappear around 6 to 8 years of age, and do not begin diverging again until puberty (see attached full growth charts at **Exhibit A**):

6– 36 months old:

	6 Months		24 Months		36 Months	
Percentile	Boys	Girls	Boys	Girls	Boys	Girls
95th	72 cm	69.5 cm	93 cm	91.5 cm	102.5 cm	101.25 cm
50th	**67 cm**	**65.25 cm**	**87.25 cm**	**86 cm**	**95.75 cm**	**94.75 cm**
5th	63 cm	61 cm	81.5 cm	80 cm	89.75 cm	88.25 cm

7–12 years old:

	7 Years		8 Years		12 Years	
Percentile	Boys	Girls	Boys	Girls	Boys	Girls
95th	130.75 cm	130.75 cm	137.5 cm	137.75 cm	161.5	163 cm
50th	**121.5 cm**	**121.5 cm**	**128 cm**	**128 cm**	**149 cm**	**151 cm**
5th	113 cm	113 cm	118.5 cm	118.25 cm	137 cm	139 cm

14. The numbers begin to diverge again after around 10 years of age, with girls overtaking males in height and weight for a few years because they typically go through the puberty-related growth spurt around two years earlier than males. *See* Charles Brook, *Mechanism of Puberty*, 3 Hormone Rsch. 52, 53 (1999).

15. Moreover, while post-pubertal boys are taller, on average, than post-pubertal girls, the height ranges for boys and girls continue to be overlapping. Ctrs. for Disease Control & Prevention, *Clinical Growth Charts: Children 2 to 20 Years (5th–95th Percentile)*, https://www.cdc.gov/growthcharts/clinical_charts.htm.

IV. There Is No Evidence That Prepubertal Boys Have a Biological Athletic Advantage Over Prepubertal Girls.

16. Contrary to Dr. Hilton's testimony and as I discussed in my prior declarations in this case, there is a well-established scientific consensus that, before puberty, there are no significant differences in athletic performance between boys and girls. *See, e.g.*, Marnee McKay & Joshua Burns, *When it Comes to Sport, Boys "Play Like a Girl"*, The Conversation (Aug. 3, 2017), https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328 (discussing results of research published in American Academy of Neurology Journal).

17. While some studies have found small differences between the performance

of boys and girls with respect to some discrete activities, these studies did not control for other factors, particularly age, location, or athletic experience or exposure. *Id.*

18. When research has controlled for those factors by using representative data, researchers have found that "[a]cross all measures of physical performance, there was one consistent finding. There was no statistical difference in the capabilities of girls and boys until high-school age (commonly age 12)." *Id.* These tests included long jump, muscle strength, walking, jumping, and balancing. *Id*.

19. This finding has been replicated in many other studies, and there is a clear scientific consensus that athletic ability does not diverge significantly until puberty. *See, e.g.*, David J. Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clin. Endocrinol. 68, 70–71 (2017) ("The gender divergence in athletic performance begins at the age of 12-13 years"); Jonathon W. Senefeld et al., *Sex Differences in Youth Elite Swimming*, 14 PLoS ONE 1, 1–2 (2019) (studying child and youth swimmers and concluding that the data suggests "girls are faster, or at least not slower, than boys prior to the performance-enhancing effects of puberty").

20. In support of her contention that boys have at least some biological advantages in athletic performance over girls before puberty, Dr. Hilton relies primarily on data from physical fitness tests or international track and field event records. The data Dr. Hilton relies on in fact shows several areas where pre-pubertal girls outperform pre-pubertal boys. (Hilton Decl. ¶¶ 7.6, 7.9.)

21. Otherwise, the data Dr. Hilton relies on shows that there is a small difference in performance between prepubertal non-transgender boys and prepubertal non-transgender girls.[1] This data merely observes phenomena across a population sample in isolated areas and does not determine a cause for whatever is observed. There is no

[1] Two of the studies cited by Dr. Hilton are also cited in paragraph 6 of the legislative findings of Arizona's statute. *See* S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2022), § 6.

reliable basis for Dr. Hilton to attribute those small differences to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys and girls surveyed. David J. Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clin. Endocrinol. 68 (2017).

22. Dr. Hilton's statement that the "performance gap in international and national track and field records evident before puberty, somewhat controls for this sociali[z]ation effect, given that one might expect engaged sporty girls to be as well-trained as their male peers" (Hilton Decl. ¶ 7.22) is pure conjecture and lacks any reliable factual basis to support it.

23. Dr. Hilton also discusses the outcomes of two individual middle school track and field competitions held at the Kyrene Aprende Middle School in the last year. (Hilton Decl. ¶¶ 7.17–7.20.) It is my understanding from Plaintiffs' counsel that one of the Plaintiffs in this case will begin attending Kyrene Aprende Middle School this month and that she wishes to participate and compete on the girls' cross-country, soccer, and basketball teams, not the track and field team. Moreover, given the age ranges of the children who attend middle school, this data likely includes some males who have undergone male puberty. It is my understanding from Plaintiffs' counsel that the Plaintiff who will be attending Kyrene Aprende Middle School will not undergo male puberty because she will be taking puberty suppressing medication, which I have discussed in more detail in my prior declarations in this case. Therefore, this data is not relevant to this litigation.

24. In any event, as previously discussed, this data does not determine a cause for the observed differences. Even if this data included only prepubertal boys and girls, there is no reliable basis for Dr. Hilton to attribute the differences observed to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys

and girls surveyed.

V. Transgender Girls Who Receive Puberty Suppressing Medication at the Onset of Puberty Have No Athletic Advantage Over Other Girls.

25. Dr. Hilton incorrectly asserts that the administration of puberty suppressing medication (also sometimes referred to as puberty blocking medication) to transgender girls does not eliminate the athletic advantage that men and adolescent boys have over women and adolescent girls.[2] (Hilton Decl. ¶ 9.5.)

26. As I have discussed previously, Tanner staging (also called Sexual Maturity Rating) is used to document and track the development and sequence of secondary sex characteristics of children during puberty. Under current standards of care, transgender adolescents are eligible to receive puberty blockers when they reach Tanner Stage 2, at the first onset of puberty, and long before the development of increased muscle mass and strength associated with later stages of male puberty. *See* Wylie C. Hembree et al., *Endocrine Treatment of Gender-Dysphoric/Gender-Incongruent Persons: An Endocrine Society Clinical Practice Guideline*, 102 J. Clinical Endocrinology & Metabolism 3869–903 (2017).

27. Following the administration of puberty blockers, transgender girls will also receive hormone replacement therapy to allow them to go through puberty consistent with their female gender identity. As a result, these transgender girls will develop many of the same physiological and anatomical characteristics of non-transgender girls, including bone size, skeletal structure, and distinctive aspects of the female pelvis geometry that cut against athletic performance. Thus, a transgender girl who received

[2] Dr. Hilton also briefly discusses the medical treatment of transgender girls and states that many children reporting gender dysphoria desist and that puberty blocking medication is harmful and has uncertain outcomes. (Hilton Decl. ¶¶ 9.3-9.4.) These conclusions are contrary to my experience treating over 600 patients with gender dysphoria. Dr. Hilton is not a medical doctor or mental health professional nor does it appear that she has ever treated a transgender patient. Moreover, Dr. Hilton does not explain how any of her criticisms are relevant to the issue of whether transgender girls should be able to participate on female sports teams. In any event, as discussed in detail in my prior declarations in this case, these criticisms are not well-founded.

puberty suppressing medication followed by hormone replacement therapy does not have the same physiology as a prepubertal non-transgender boy.

28. Because such girls do not undergo male puberty, they do not gain the increased muscle mass or strength that accounts for why post-pubertal boys as a group have an advantage over post-pubertal girls as a group.

29. For that reason, studies on transgender women who have undergone testosterone suppression as adults are almost meaningless when assessing the athletic abilities of transgender girls who have received pubertal suppression beginning at the onset of puberty. The women in those studies did not transition until well after puberty and experienced exposure to testosterone over an extended time, allowing their muscles to keep developing. In sharp contrast, transgender girls who receive Gonadotropin-releasing hormone agonist ("GnRHa") do not go through male puberty and are not exposed to the heightened level of testosterone associated with male puberty.

30. Even so, those studies of adult transgender women show that testosterone suppression resulted in significant mitigation of muscle mass and development in adult transgender women.

31. For example, the only study directly examining the effects of hormone therapy on the athletic performance of transgender female athletes is a small study of eight long-distance runners. The study showed that after undergoing medical interventions, which included lowering their testosterone levels, the athletes' performance had reduced so that relative to non-transgender women their performance was now proportionally the same as it had been relative to non-transgender men prior to any medical treatment. In other words, a transgender woman who performed at about 80% as well as the best performer among men of that age before transition would also perform at about 80% as well as the best performer among women of that age after transition. *See* Joanna Harper, *Race Times for Transgender Athletes*, 6 J. Sporting

Cultures & Identities 1 (2015).[3] Given that adolescent transgender girls who receive puberty suppressing medication do not go through male puberty, there is no medical basis to expect that transgender girls receiving such medications would have an athletic advantage.

32. Dr. Hilton cites two studies that she claims show that transgender girls have an athletic advantage over other girls even when they are receiving puberty blocking medication or hormone therapy; however, neither study supports Dr. Hilton's claim.

33. Dr. Hilton cites to Maartje Klaver et al., *Early Hormonal Treatment Affects Body Composition and Body Shape in Young Transgender Adolescents*, 15 J. Sexual Med. 251 (2018). (Hilton Decl. ¶ 11.3.) Contrary to Dr. Hilton's claim, however, the primary finding of the Klaver study is that receiving puberty blockers and hormone therapy bring the body composition of young transgender women much closer to their non-transgender female peers than their non-transgender male peers. Those results are more pronounced the earlier a transgender girl starts puberty blockers. *Id*. at 255 (finding that "compared with adult transgender persons treated with CHT, larger changes in body shape and body composition are seen in transgender persons who start in adolescence"). It should also be noted that the transgender women participants in the Klaver study started GnRHa at an average age of 14.5 years, and none started prior to age 12. This is because the original Dutch protocol did not provide GnRHa prior to age 12 regardless of whether puberty started at a younger age. The participants in the study by definition had much more testosterone exposure than transgender girls treated with modern protocols,

[3] The legislative findings of the Arizona statute incorrectly state that for transgender women who go through male puberty (unlike the plaintiffs here), the benefit conferred by testosterone "is not diminished through the use of testosterone suppression." *See* S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2022), § 13. While that statement conflicts with available evidence, which shows that hormone therapy significantly reduces muscle mass and strength, it is also irrelevant to the situation of the plaintiffs in this case who have not undergone male puberty and thus are not in the position of having to mitigate the increased muscle mass and strength caused by male puberty. Notably, the legislative findings do not state that transgender girls who receive puberty suppressing medication at the onset of puberty have any conceivable athletic advantage, nor do they cite any evidence that would support that claim.

which initiate GnRHa based on pubertal stage unrelated to age.

34. Dr. Hilton also cites Lloyd J.W. Tack et al., *Proandrogenic and Antiandrogenic Progestins in Transgender Youth: Differential Effects on Body Composition and Bone Metabolism*, 103 J. Clinical Endocrinology & Metabolism 2147 (2018), for the proposition that transgender girls who receive medical treatments purportedly maintain greater grip strength than transgender boys. (Hilton Decl. ¶ 11.3.) But the medication administered in this study is not used in the U.S. and does not have nearly the same impact as puberty blockers and hormone therapy for transgender girls or as testosterone for transgender boys. The medications administered to the study participants did not fully block puberty for either transgender girls or transgender boys. Even with this less effective medication, the study found that transgender girls "showed a significant increase in fat mass and decrease in lean mass, resulting in an increased body fat percentage" and did not experience any increase in grip strength. *Id*. at 2153–54. If anything, this study shows that even with a less effective medication, the physiological impact of medically treating transgender girls in adolescence, rather than when they are adults, is profound.

35. At the beginning of her declaration, Dr. Hilton discusses her involvement with the World Rugby Transgender Guidelines. (Hilton Decl. ¶ 1.13.) However, even these guidelines allow transgender girls and women to participate in women's rugby if they did not experience endogenous puberty, stating: "Transgender women who transitioned pre-puberty and have not experienced the biological effects of testosterone during puberty and adolescence can play women's rugby." World Rugby, *Transgender Women Guidelines* (2019), https://www.world.rugby/the-game/player-welfare/guidelines/transgender/women.

36. In sum, there is no evidence that transgender girls on puberty suppression medication or hormone therapy have an athletic advantage over other girls. There are no studies that have documented any such advantage, and there is no medical reason to posit

that any such advantage would exist.

37. In my clinical practice, I have provided medical care to more than 300 adolescent transgender girls. None of the transgender girls I have treated with the above medical interventions appeared to have any athletic advantage over other girls.

VI. From a Medical Perspective, Menstruation Does Not Provide a Basis to Conclude That Transgender Girls Have an Athletic Advantage Over Other Girls.

38. In her declaration, Dr. Hilton claims that female athletes have an athletic disadvantage because they "must typically deal with the effects of the menstrual cycle," which may affect "training capacity and performance," and that, as a result, transgender girls have an athletic advantage because they do not menstruate. (Hilton Decl. ¶ 6.5.) This conclusion does not have a sound medical or scientific basis because not all adolescent girls menstruate or suffer any athletic disadvantage if they do menstruate.

39. For example, girls with certain medical conditions do not menstruate, and some adolescent girls may take birth control to prevent menstruation or for other medical reasons. In addition, not all adolescent girls who do menstruate suffer any adverse impacts on their training capacity or performance.

VII. Permitting Transgender Girls to Play on Girls' Teams Does Not Pose a Safety Risk to Other Girls.

40. In her declaration, Dr. Hilton testifies that transgender girls who play on girls' teams somehow pose a threat to the safety of other girls because, she asserts, girls have "delicate brain structures" that make them more prone to injury. (Hilton Decl. ¶ 6.6.) While research has found that girls suffer more sports-related concussions than boys, the cause of that differential is unknown, including whether it is cultural or biological or both. *See* William T. Tsushima et al., *Incidence and Risk of Concussions in Youth Athletes: Comparisons of Age, Sex, Concussion History, Sport, and Football Position*, 34 Archives Clinical Neuropsych. 60, 66 (2019). In any event, however, there

is no scientific evidence that girls have more "delicate brain structures" than boys. If a researcher were to view an MRI of a human brain, there would be no way to identify whether it was the brain of a male or a female other than average size. Lise Eliot et al., *Dump the "Dimorphism": Comprehensive Synthesis of Human Brain Studies Reveals Few Male-Female Differences Beyond Size*, 125 Neurosci. & Biobehav. Rev. 667, 668 (2021).

41. Some researchers have theorized that girls may suffer more sports-related concussions because, on average, adolescent girls have weaker neck muscles than post-pubertal adolescent boys. *See* Abigail C. Bretzin et al., *Association of Sex with Adolescent Soccer Concussion Incidence and Characteristics*, 4 JAMA Network Open 4, 6 (2021); Ryan T. Tierney et al., *Gender Differences in Head-Neck Segment Dynamic Stabilization During Head Acceleration*, 37 Med. & Sci. Sports & Exercise 272, 272 (2005). If that accounts for girls' higher rates of concussions (which is unknown), transgender girls on puberty blockers or hormone therapy would be at the same or similar risk for such injury as non-transgender girls. There is no evidence, and no medical reason to believe, that their participation on girls' teams would pose any increased threat of such injuries to other girls.

42. More generally, transgender girls do not present any unique safety risks to other girls. Transgender girls' physical characteristics (in terms of height, weight, and strength) overlap with those of other girls. For example, while some transgender girls may be taller than average, so are some non-transgender girls, and many transgender girls are simply average.

43. There is no more reason to exclude a tall transgender girl for safety reasons than there would be to exclude any other girl for that reason. While some transgender girls may (or may not) have larger skeletons than some non-transgender girls, there is no medical reason to conclude that that physical characteristic poses any elevated safety concerns when not accompanied by high levels of testosterone and corresponding skeletal

muscle. After a transgender adolescent suppresses her level of testosterone, there is no inherent medical reason why her physiological characteristics related to athletic performance should be treated differently from the physiological characteristics of other girls.

Dr. Blade's Declaration

44. Dr. Blade is not a medical doctor, nor does it appear that she has ever treated a transgender patient; in contrast, I have experience treating over 600 hundred patients with gender dysphoria. From a medical perspective, the terms "biological sex," "biological male," and "biological female" are imprecise terms that can cause confusion. A person's sex encompasses several different biological attributes, including sex chromosomes, certain genes, gonads, sex hormone levels, internal and external genitalia, other secondary sex characteristics, and gender identity. Those attributes are not always aligned in the same direction. *See* Joshua D. Safer, *Care of Transgender Persons*, 381 N. Engl. J. Med. 2451 (2019).

45. Contrary to Dr. Blade's testimony and as I have previously discussed, there is an overwhelming scientific consensus that the biological cause of average differences in athletic performance between men and women is the rise in circulating levels of testosterone beginning in endogenous male puberty.

46. Dr. Blade discusses data from physical fitness tests in children to demonstrate that transgender girls have an athletic advantage over other girls before puberty. (Blade Decl. at 7–9.) This data merely observes phenomena across a population sample in isolated areas and does not determine a cause for whatever is observed. As I have discussed previously, there is no reliable basis for Dr. Blade to attribute any small differences between boys and girls to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys and girls surveyed.

47. Dr. Blade also asserts that because prepubertal boys have a greater lung volume and other enhanced capabilities throughout the oxygen transport system, they do better in endurance tests and the 1500m run. (Blade Decl. at 9.) In fact, any such difference between boys and girls is small and has no documented impact on athletic performance. If this small average difference in lung capacity had a significant causal relationship to athletic advantage, we would see significant differences in the athletic performance of prepubescent boys and girls, but we do not.

48. Dr. Blade posits that transgender girls' participation in girls' sports causes psychosocial risks to other girls. (Blade Decl. at 11–12.) Dr. Blade's assertion is based on a misunderstanding regarding transgender girls. As discussed in my prior declarations in this case, a transgender girl is a girl. Moreover, there is no reason to assume a transgender girl's identity would be discernible to other girls, particularly when a transgender girl transitions socially and medically. Lastly, Dr. Blade supports her assertion with mere anecdotes rather than scientific research on the topic. As discussed above, the scientific research demonstrates there is no athletic advantage between transgender girls who have not undergone male puberty and other girls.

Dr. Brown's Rebuttal Declaration

49. Dr. Brown cites a hodge-podge of studies, but none support his view that prepubertal boys have a significant group-based advantage over prepubertal girls, which is contrary to the overwhelming weight of medical evidence and consensus on this issue.

50. For example, although Dr. Brown claims that Handelman's research supports Dr. Brown's position, Handelman himself disagrees, as Dr. Brown concedes.

51. The studies cited by Dr. Brown do not support his thesis for a variety of reasons. First, several of the studies include post-pubertal as well as pubertal children. (*See*, *e.g.*, Brown Rebuttal Decl. ¶¶ 9–10 (citing data that includes children from the ages of 9 to 16).) Second, some of the studies show small physiological differences between prepubertal boys and girls, but do not purport to establish any causal link between those

small differences and athletic ability or establishing only a speculative or hypothetical link. (*See*, *e.g.*, Brown Rebuttal Decl. ¶ 12 (citing data showing that girls have a slightly higher resting heart rate).) And third, even with respect to those small physiological differences between prepubertal boys and girls, unlike the post-pubertal production of testosterone, those differences exist on an overlapping spectrum. For example, while it is true that there is some evidence that prepubertal boys on average may have slightly less body fat than girls,[4] there are some girls who have less body fat than some boys, and some boys who have more body fat than some girls. In contrast, apart from girls with certain intersex conditions or other health conditions, there are no post-pubertal girls with more testosterone than post-pubertal boys; generally speaking, testosterone levels in post-pubertal boys and girls do not overlap.

52. Notably, Dr. Brown agrees that there is no basis for alleging that minipuberty has any impact on athletic ability. (Brown Rebuttal Decl. ¶ 37 (stating "At no point in my declaration are the male athletic advantages differences ascribed to 'minipuberty' (indeed, the term 'minipuberty' is not found within my expert report.")).

Dr. Carlson's Rebuttal Declaration

53. Dr. Carlson acknowledges that the only studies finding small differences in athletic performance between prepubertal boys and girls are cross-sectional studies that, as such, do not "assign causation to any measured differences, such as biology vs. sociological effect." (Carlson Rebuttal Decl. ¶ 6.) In addition, the small differences found by these studies relate to discrete activities, not to strength or athletic performance across the board, and do not rise anywhere close to the level of the broad, clear, and significant group-based differences caused by exposure over time to the elevated levels of testosterone associated with male puberty.

54. Dr. Carlson attempts to rebut the conclusion of McKay's study that there

[4] As noted in my prior declaration, and as Dr. Brown acknowledges (Brown Rebuttal Decl. ¶ 17), this research is not conclusive; some studies have found no differences and have criticized other studies for failing to consider factors such as age, maturational status and obesity status. (Shumer Rebuttal Decl. ¶ 6)

are no significant differences in athletic ability between prepubertal boys and girls, but his analysis is not persuasive. As Dr. Carlson acknowledges, McKay found no significant differences in strength based on sex in children ages 3 through 9—i.e., in prepubertal children, and found such differences only in post-pubertal children. (Carlson Decl. ¶ 9).

55. Dr. Carlson's suggestion that the two girls who are Plaintiffs in this case would have been grouped with the 10 to 19 year olds (Carlson Rebuttal Decl. ¶¶ 10–11) has no logical relevance to the import of McKay's study: significant athletic differences between boys and girls are linked to puberty. The Plaintiffs in this case are receiving puberty suppressing medication, which prevents them from undergoing male puberty and thus from gaining the potential athletic advantage associated with exposure to post pubertal levels of testosterone.

56. Dr. Carlson acknowledges that the studies he cites "carry with them the limitations of cross-sectional comparisons" (Carlson Rebuttal Decl. ¶ 15), and thus cannot establish any causal link between physiology and athletic performance in prepubertal children for the reasons explained above.

57. Dr. Carlson offers no evidence for his assumption that the enactment of Title IX means that prepubertal boys and girls now receive equal coaching and skill training, nor does any such evidence exist. (Carlson Rebuttal Decl. ¶ 19) To the contrary, as discussed below, research shows that girls receive far less opportunities for participation than boys.

58. Relatedly, Dr. Carlson relies heavily on a single article by Lombardo, which in turn rests upon speculative and subjective hypotheses about how boys and girls are treated in various cultures, including, for example, a presumption that Aboriginal boys and girls are equally encouraged to hunt and that German boys "do not throw much and do not have U.S.-like cultural support or encouragement for throwing." (Carlson Rebuttal Decl. ¶ 19(citing Michael P. Lombardo et al., *On the Evolution of the Sex*

Differences in Throwing: Throwing is a Male Adaptation in Humans, 93 Q.Rev. Biology 91 (2018))). Such speculative research based on broad sociological generalizations about other cultures does not provide a valid evidentiary basis to conclude that the small differences in athletic performance found in some cross-sectional studies of prepubertal boys and girls are based on physiology rather than culture, much less that such small differences have any applicability to individual transgender girls or warrant excluding all transgender girls from playing on girls' teams.

59. Research that is more carefully and objectively designed has found that differences in skills training and practice—not innate gender-based differences—account for many specific sex-based differences in athletic performance. For example, a 2019 study of spatiotemporal coordination in throwing found that sex-based differences "only arose from age 20 years onwards and that in individuals with throwing practice, performance disparities leveled out." Dena Crozier et al., *Gender Differences in Throwing Revisited: Sensorimotor Coordination in a Virtual Ball Aiming Task*, 13 Frontiers Hum. Neurosci. 231 (2019).

60. Given the far greater social encouragement and skills training provided to boys than to girls, it is not surprising, as Dr. Carlson notes (Carlson Rebuttal Decl. ¶ 21), that boys have the highest-ranking performances in USA Track & Field. Contrary to Dr. Carlson's suggestion that our society promotes "equal opportunities for boys and girls to participate," the reality is much different. Across the board, girls have far fewer opportunities to play sports and therefore far less coaching and skill training than boys in every age group. *See* U.S. Dep't Health & Hum. Servs., *The National Youth Sports Strategy*, 35–37 (2019), https://health.gov/sites/default/files/2019-10/National_Youth_Sports_Strategy.pdf; Aspen. Inst. Project Play, *Youth Sports Facts: Participation Rates*, https://www.aspenprojectplay.org/youth-sports/facts/participation-rates. For example, during the 2018–2019 year, fifty-seven percent of high school athletics participation opportunities went to boys, with only forty-three percent going to

girls, translating into over one million more opportunities for boys than girls. Ellen J. Staurowsky et al., Women's Sports Found., *50 Years of Title IX: We're Not Done Yet*, 30 (2022), https://www.womenssportsfoundation.org/wp-content/uploads/2022/05/Title-IX-at-50-Report-FINALC-v2-.pdf.

61. Dr. Carlson acknowledges that even the highly restrictive World Rugby policy permits transgender girls who receive puberty suppressing medication to play. (Carlson Rebuttal Decl. ¶¶ 23–24.) Dr. Carlson contends that this exception is not "grounded in scientific review of relevant data," but there is no data showing that such girls have any athletic advantage over other girls, nor is there any medically reasonable basis for assuming that they do. (Carlson Rebuttal Decl. ¶ 24.)

62. Dr. Carlson's suggestion (Carlson Rebuttal Decl. ¶ 25) that puberty suppressing medication fails to suppress the heightened levels of testosterone associated with male puberty in 25 to 49 percent of cases has no medical basis. The article he cites to support that erroneous claim is about the use of testosterone suppressant by adult transgender women who went through male puberty; it has no bearing on the efficacy of puberty suppression for transgender girls, which is highly effective and prevents transgender girls from producing the elevated levels of testosterone associated with male puberty.

63. The Klaver study does not support Dr. Carlson's claim that transgender girls who received puberty suppressing medication have an athletic advantage over other girls (Carlson Rebuttal Decl. ¶¶ 31–32) for the reasons stated in paragraph 33 above. It is not appropriate to use the Klaver article to presume that transgender girls may have more lean body mass on average than other girls because, as noted above, Klaver participants started GnRHa at much older ages than modern protocols would dictate. The findings of the study are not generalizable across decades and not relevant to the question at hand.

64. For the reasons explained in paragraphs 40 through 43 above, Dr. Carlson's claim that transgender girls "are more likely to cause concussions than other competitors"

(Carlson Rebuttal Decl. ¶ 33) has no medical basis. It is particularly unwarranted for transgender girls, like the Plaintiffs in this case, who receive puberty suppressing medication and thus do not go through male puberty.

Dr. Cantor's Supplemental Declaration

65. Dr. Cantor acknowledges that his views place him at odds with the standards of care and practice guidelines developed by the World Professional Association of Transgender Health ("WPATH") and the Endocrine Society, and which have been endorsed by a long list of major medical professional associations, including the American Medical Association, the American Academy of Pediatrics, the American Psychological Association, and many others.

66. Contrary to Dr. Cantor's unsupported claims, which implausibly cast aspersions on the integrity of our nation's leading professional medical organizations, the standards of care and practice guidelines relied upon by medical and mental health professionals who specialize in the treatment of gender dysphoria in adolescents have a sound evidentiary basis. The evidence-based methodology used to generate these guidelines is described in detail in both the WPATH Standards of Care and the Practice Guidelines promulgated by the Endocrine Society and is comparable to that used to generate similar clinical practice guidelines for other medical conditions.

67. Dr. Cantor's views, which seek to cast doubt on the existence of gender identity as a facet of human identity and to advocate the use of therapeutic techniques to discourage or prevent minors from identifying as transgender, do not have a sound scientific foundation and are distinctly at odds with the overwhelming consensus of medical science, experts, and practitioners in this area.

68. Dr. Cantor does not diagnose or treat gender dysphoria in adolescents or adults and has no training or expertise in transgender mental health care or medicine. As such, his strong disagreement with the consensus of medical experts in this area appears to be based more on his personal opinions than on a scientific foundation.

I declare under criminal penalty under the laws of Arizona that the foregoing is true and correct. Signed on the 6th day of July, 2023, in Ann Arbor, Michigan.

Daniel Shumer, M.D.

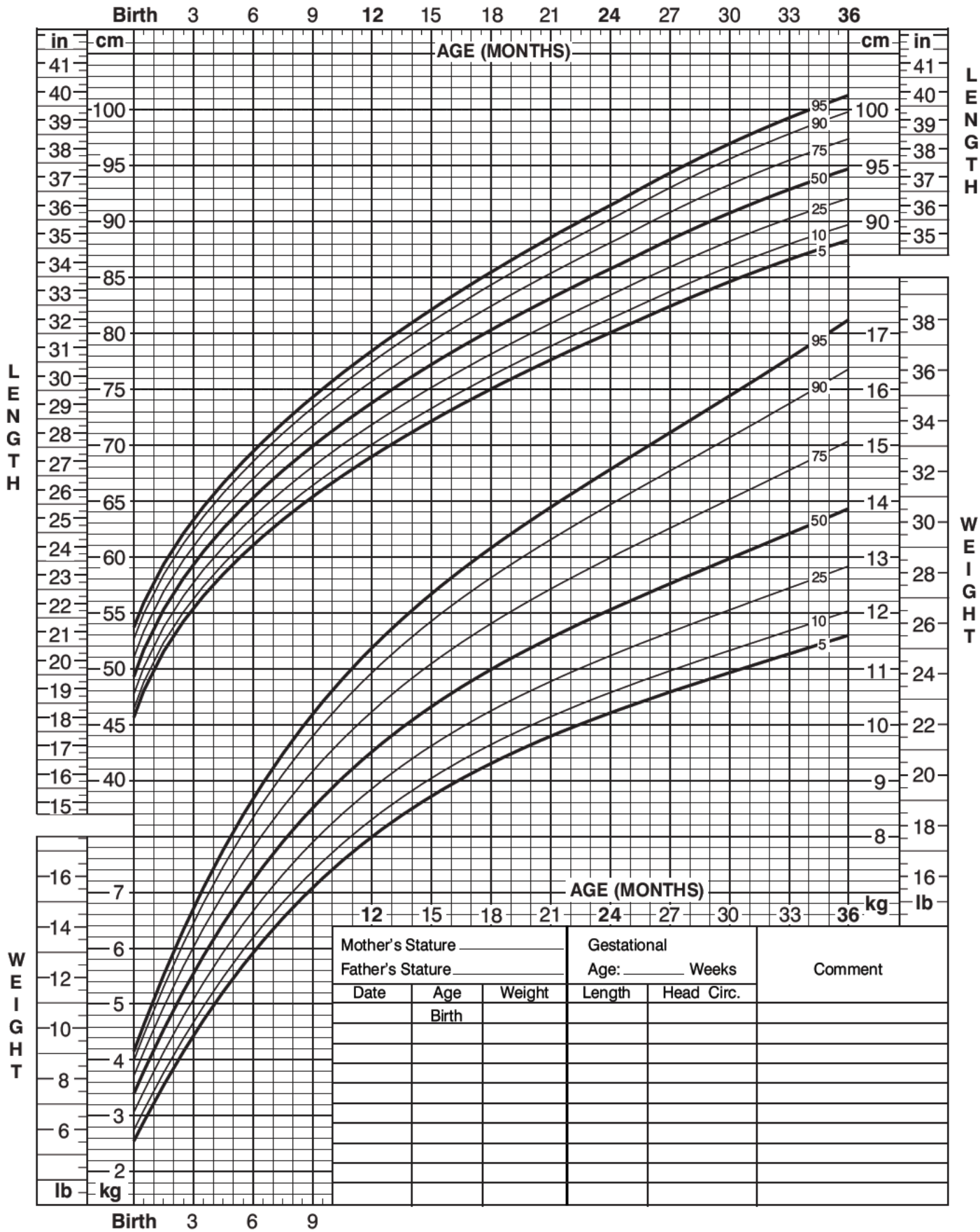
Exhibit A

CDC Growth Charts

Birth to 36 months: Girls
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 4/20/01).

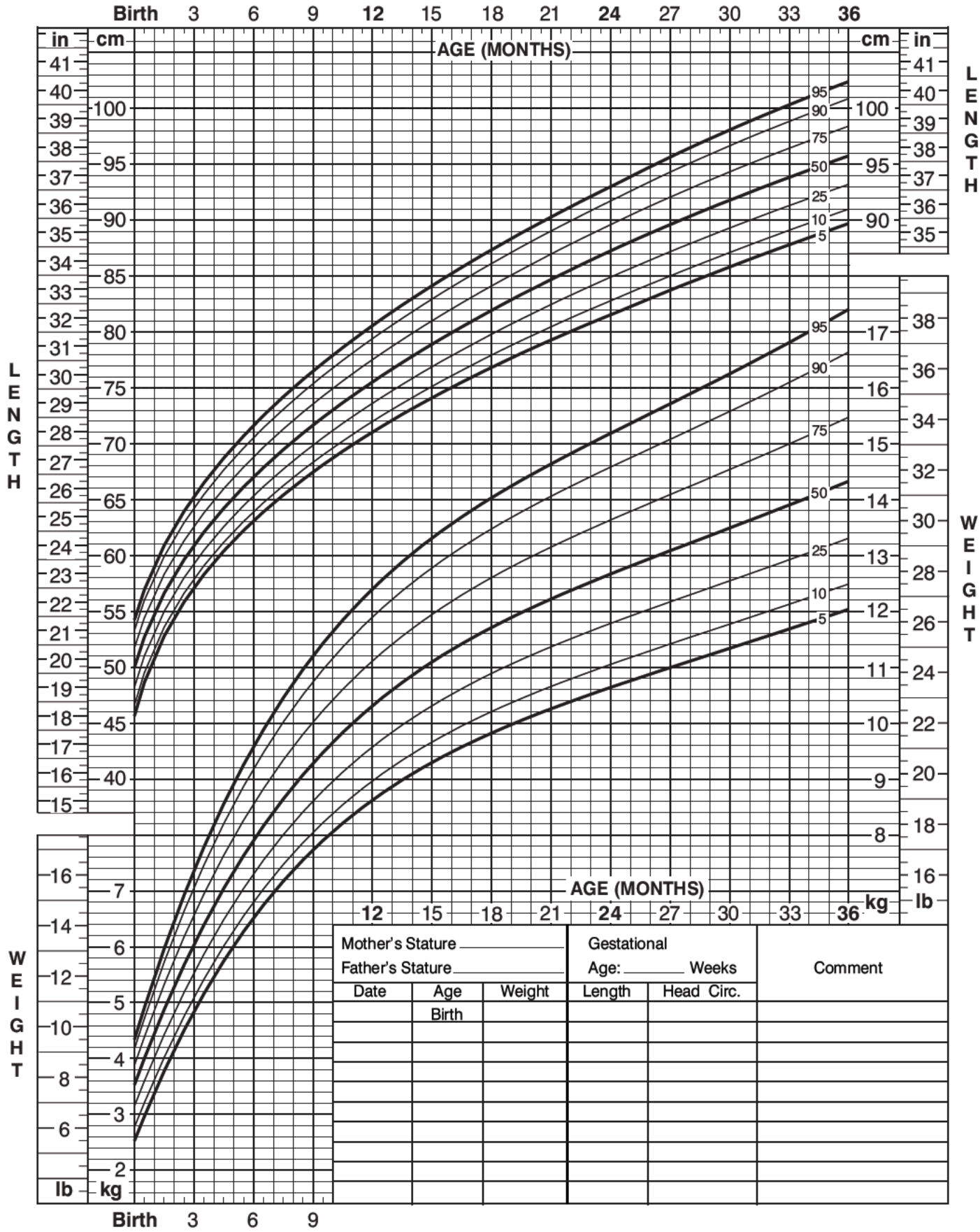
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
SAFER · HEALTHIER · PEOPLE™

Birth to 36 months: Boys
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 4/20/01).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
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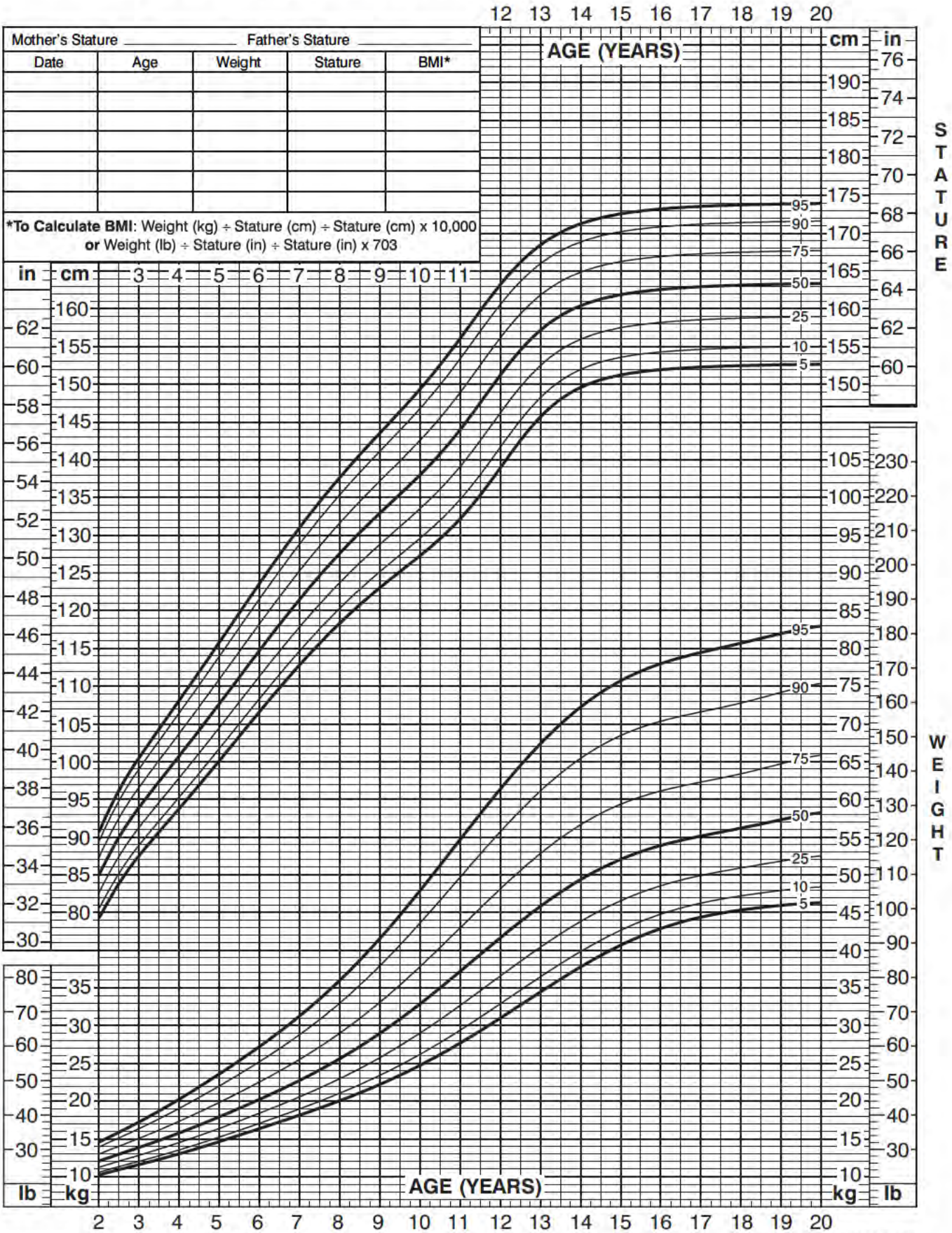
2 to 20 years: Girls
Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____

Mother's Stature _____ Father's Stature _____				
Date	Age	Weight	Stature	BMI*

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).

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2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles

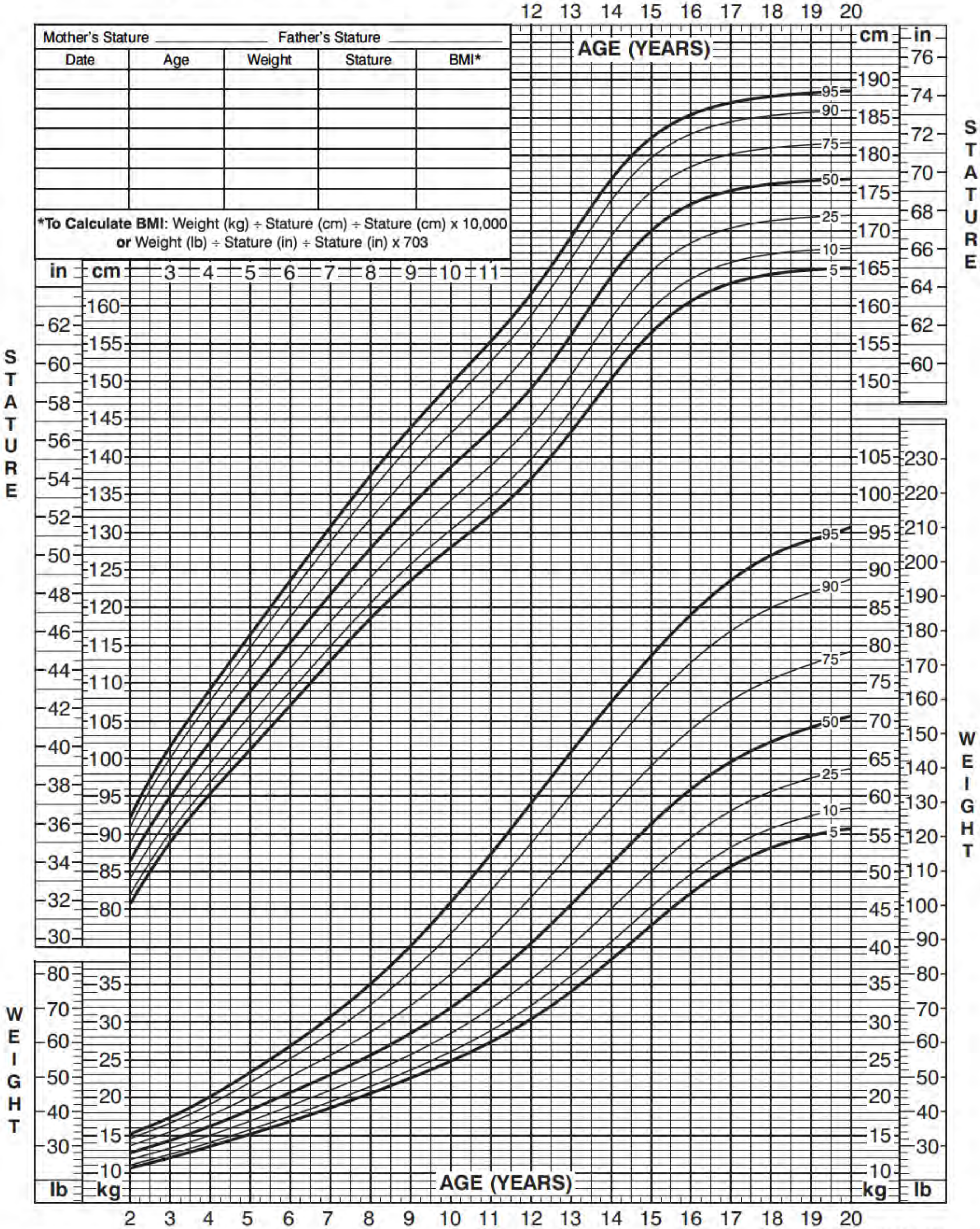
NAME ____________________

RECORD # __________

Mother's Stature __________ Father's Stature __________

Date	Age	Weight	Stature	BMI*

***To Calculate BMI**: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).
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the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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Dated: August 1, 2023

Respectfully Submitted,

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CERTIFICATE OF SERVICE

I hereby certify that, on August 1, 2023, I caused a true and correct copy of the foregoing to be filed by the Court's electronic filing system, to be served by operation of the Court's electronic filing system on counsel for all parties who have entered in the case.

/s/ D. John Sauer