UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF FLORIDA
TALLAHASSEE DIVISION

AUGUST DEKKER, et al.,

Plaintiffs,

v. Case No. 4:22-cv-00325-RH-MAF

JASON WEIDA, et al.,

Defendants.
_____/

PLAINTIFFS' MOTION TO EXCLUDE EXPERT TESTIMONY OF SOPHIE SCOTT, PH.D., AND SUPPORTING MEMORANDUM OF LAW

Pursuant to Federal Rules of Civil Procedure 26 and 37, and Federal Rules of Evidence 104, 403 and 702, Plaintiffs respectfully move this Court to exclude the expert report, opinions, and testimony of Defendants' proposed expert Professor Sophie Scott ***in its entirety***. Professor Scott is not a qualified expert on gender dysphoria or its treatment, and her opinions and testimony are neither relevant nor reliable under Federal Rule of Evidence 702 and the standards set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993), and its progeny. Her opinions and testimony are likewise inadmissible because any probative value they may have (and they have none) is substantially outweighed by the danger of unfair prejudice, confusion of the issues, waste of time, undue delay, and needless presentation of cumulative evidence. *See* Fed. R. Evid. 403.

WHEREFORE, Plaintiffs respectfully request an order excluding Professor Scott's report, expert opinions and testimony in their entirety.

MEMORANDUM OF LAW

Professor Scott is not qualified to offer the opinions stated in her report. She opines that puberty delaying medication administered to teenagers "*may have*" unknown, negative effects on brain development. Report, ¶ 15 (**Exhibit A**). She also believes without any scientific support that it is "very possible" that teenagers cannot "fully grasp the implications of puberty blocking treatment." *Id*. ¶ 16. But Professor Scott is not qualified to give these opinions because she has never treated patients with gender dysphoria (at any age) given that she is not a medical provider of any kind, nor has she administered or studied the effects of puberty delaying treatment in any clinical or academic setting. She has never written on these subjects either—except on Twitter.

Aside from her lack of qualifications, Professor Scott's opinions are inadmissible because they are entirely speculative and lack any reliable or testable foundation or methodology. There is no existing data to support her ultimate conclusions, which means her opinions are based on impermissible "leaps of faith." The data that does exist directly contradicts her conclusions, but, strikingly, she never mentions this data in her report. Her opinions moreover are based solely on her unqualified review of other studies, and they are far outside the scientific mainstream. The Court should therefore exercise its gatekeeping function under Rule 702 and exclude Professor Scott's testimony. *See Rink v Cheminova, Inc.*, 400 F.3d 1286, 1291 (11th Cir. 2005).

A. Legal Standard

Rule 702 of the Federal Rules of Evidence governs the admissibility of expert testimony. Pursuant to *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993), and Rule 702, district courts must perform a "gatekeeping" role "to ensure that speculative, unreliable expert testimony does not reach the jury under the mantle of reliability[.]" *Rink*, 400 F.3d at 1291; *Kilpatrick v. Berg, Inc*., 613 F.3d 1329, 1335 (11th Cir. 2010) ("The trial court must make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field.").

To do so, the Court must engage in a rigorous inquiry to determine whether:

> (1) the expert is qualified to testify competently regarding the matters he intends to address; (2) the methodology by which the expert reaches his conclusions is sufficiently reliable as determined by the sort of inquiry mandated in Daubert; and (3) the testimony assists the trier of fact, through the application of scientific, technical, or specialized expertise, to understand the evidence or to determine a fact in issue.

E.g., *United States v. Frazier*, 387 F.3d 1244, 1260 (11th Cir. 2004) (en banc), *cert. denied*, 544 U.S. 1063 (2005). The party offering the expert has the burden of satisfying each of these three elements by a preponderance of the evidence. *Rink*, 400 F.3d at 1292.

B. Professor Scott is Not Qualified To Offer An Expert Opinion on Any Issue in the Case.

A witness may be qualified as an expert by virtue of her "knowledge, skill,

experience, training, or education." Fed. R. Evid. 702. However, "[e]xpertise in one field does not qualify a witness to testify about others." *Lebron v. Secretary of Florida Dept. of Children and Families*, 772 F.3d 1352, 1368 (11th Cir. 2014) (holding that a psychiatrist was properly prevented from opining on rates of drug use because he had never conducted research on the subject, and instead relied on studies to form his opinion).

A scientist, however well credentialed, cannot be "the mouthpiece of a scientist in a different specialty." *Id*. at 1369 (quoting *Dura Automotive Systems of Indiana, Inc. v. CTS Corp.*, 285 F.3d 609, 614 (7th Cir. 2002)); *TB Food USA, LLC v. American Mariculture, Inc*., 2021 WL 4962969, at *4 (M.D. Fla. October 26, 2021) ("[A]n expert must have at least some minimum training, education, experience, knowledge, or skill pertaining to the particular subject matter of his proposed testimony.") (cleaned up). "Merely reading literature in a scientific field does not qualify a witness—even an educated witness—as an expert." *Kadel v. Folwell*, 2022 WL 3226731, at *9, 13 (M.D.N.C. August 10, 2022) (excluding Dr. Lappert's expert opinion about puberty delaying medication because he is a surgeon, not an endocrinologist, and he never treated a patient with hormone therapies). If an expert witness does not intend to testify about matters growing directly out of "research [s]he had conducted independent of the litigation," the expert should be disqualified. *Lebron*, 772 F.3d at 1369 (quoting Fed. R. Evid. 702).

Professor Scott is the Director of University College London's Institute of Cognitive Neuroscience. Report ¶ 6.[1] Her main area of research is "speech, laughter and sound." Tr. 48:25 – 49:4 ("Q. All of these publications are about speech, laughter and sound. Isn't that right? A. There are a few other things. But yeah, that's the majority. That is my main area of research.") (**Exhibit B**). She is proffered as an expert based on her "training and experience as a neuroscientist," her reading and assessment of "the relevant neuroscientific literature on brain development, and the potential effects of [puberty delaying medication] on the developing brain." Report, ¶ 4. However, she has no experience with the provision of puberty delaying medication, gender-affirming medical care or medical treatment of gender dysphoria. She has never published any papers or studies on gender dysphoria, gender-affirming care or puberty delaying medication. Nor has she published any reviews of such studies in her entire career. Tr. 49:5-12 ("Q. Are any of [your publications] about gender-affirming care? A. No. Q. Are any of these publications specific to gender dysphoria? A. No. Q. Any about puberty blockers? No.").

Professor Scott is not a medical doctor, a psychiatrist or a clinical psychologist; she has no medical training. Tr. 34:25 – 35:4; Tr. 35:13-14. She does

[1] According to Professor Scott, cognitive neuroscience is "a scientific field that examines the relationships between human behaviour to the human brain, and how these can be affected by age, disease and individual differences." Report, ¶ 6; Tr. 37:6-10 ("A neuroscientist is somebody who studies brains…[H]e's studying it in a purely basic science position. They're not treating people. They're not prescribing things.").

not treat patients. Tr. 44:22-23. She has never studied gender dysphoria in a clinical setting, nor has she ever administered puberty delaying medication or studied their effects, let alone in humans. Tr. 31:18-24 (Q. So you've never conducted any clinical studies yourself related to gender dysphoria? A. No. Q. What about the effects of gender-affirming care? A. Nope."). Nor has anyone at Professor Scott's place of employment, the Institute for Cognitive Neuroscience ever studied gender dysphoria or the effects of gender-affirming medical care either, meaning Professor Scott has not overseen any such study. Tr. 31:6-8 ("Q. Has anyone at the Institute ever conducted any clinical studies related to gender dysphoria? A. No, not that I'm aware of."; Tr. 31:25-32:2 (Q. Has the Institute ever studied the effects of puberty blockers? A. No.").

Without *any* qualifications, training or experience related to gender dysphoria or puberty delaying medication, Professor Scott is not qualified to give an expert opinion on these subjects. *See Kadel*, 2022 WL 3226731, at *13.[2] Nor is she

[2] *See also, e.g.*, *Fernandez v United States*, 2020 WL 3105925, at *5 (N.D. Fla. June 4, 2020) (excluding an expert because the Plaintiff offered "no information indicating that he has any experience or specialized knowledge regarding medicine generally or any of the branches of medical science which might be relevant to causation); *Doctors Licensure Group, Inc. v. Continental Casualty Company*, 2011 WL 13182969, at *4 (N.D. Fla. September 26, 2011) (excluding a proffered expert on accounting because he was "not an accountant" and had "virtually no experience in accounting"); *Webb v. Carnival Corporation*, 321 F.R.D. 420, 429 (S.D. Fla. 2017) ("Because Mr. Jaques has no experience in toxicology, responsible alcohol vending policies, nor medicine, and has never served onboard the California Dream, he is unqualified to opine on the Decedent's level of intoxication[.]").

qualified to opine on studies related to gender dysphoria or puberty delaying medication conducted by others. *See Dura Automotive*, 285 F.3d at 614 ("[A] scientist, however well credentialed he may be, is not permitted to be the mouthpiece of a scientist in a different specialty.").

This is so particularly here where Professor Scott's opinions and so-called review of literature did not "grow[] naturally and directly out of research [s]he had conducted independent of the litigation." *Lebron*, 772 F.3d at 1369 (cleaned up); *see also* Fed. R. Evid. 702, Advisory Comm. Notes (2000 Amendments). Here, Professor Scott reviewed the literature and developed her opinions in connection with litigation in the UK, namely, *Bell v. Tavistock*, and now seeks to transpose those opinions here without still having done any independent work in the area. Tr. 52:7-18 ("Q. Why did you think that you had an opinion to give in this case? A. *Because I provided an opinion before for the Keira Bell case.* And I discussed that a lot with Paul Conrathe at the time for all the reasons you said. I'm not a clinician. *I haven't worked in this area.* … And I did some reading into the literature, … ."); Tr. 53:6-9 ("Q. So you formed your opinion about puberty blockers in adolescents while you were working on the Bell case? A. Yeah.").

In *Kadel*, a case similar to this one about insurance coverage for gender-affirming medical care, the court excluded a proposed expert (Dr. Lappert) because "[h]e is not a psychiatrist, psychologist, or mental health professional, nor has he

ever diagnosed a patient with gender dysphoria,"[3] and "[h]e is not an endocrinologist, nor has he ever treated a patient with hormone therapies." *Kadel*, 2022 WL 3226731, at *13. Here, Professor Scott, who unlike the excluded expert in *Kadel*, has no medical degree and has never provided medical or mental health care, is likewise "not qualified to render opinions about the diagnosis of gender dysphoria, its possible causes, … the efficacy of puberty blocking medication or hormone treatments, the appropriate standard of informed consent for mental health professionals or endocrinologists, or any opinion on the non-surgical treatments obtained by Plaintiffs." *Id.* Her opinions should be excluded *in toto*.

C. Professor Scott's Opinions are Unreliable.

An expert's reliability concerns whether the reasoning or methodology underlying the testimony is scientifically valid and whether that reasoning or methodology properly can be applied to the facts in issue. *Kilpatrick*, 613 F.3d at 1335. When evaluating whether an expert's methodology is reliable, the Court considers, among other things:

> (1) whether the expert's theory can be and has been tested; (2) whether the theory has been subjected to peer review and publication; (3) the known or potential rate of error of the particular scientific technique; and (4) whether the technique is generally accepted in the scientific community.

[3] While Dr. Scott has an undergraduate degree where she minored in psychology, she is not certified as psychologist, and admits she's "not clinically qualified." Tr. 35:8-17. In her words, she is "a basic scientist." *Id.*

Frazier, 387 F.3d at 1262. The court must undertake an independent analysis of each step in the logic leading to the expert's conclusions, and if any step in the logic is deemed unreliable, the expert's entire opinion must be excluded. *Hendrix v Evenflo Co., Inc.*, 255 F.R.D. 568, 578 (N.D. Fla. 2009) (citing *McClain v. Metabolife Int'l., Inc.*, 401 F.3d 1233, 1245 (11th Cir. 2005)). Likewise, if the expert's opinions are vague or based on "leaps of faith unsupported by good science," then those opinions should be excluded as well. *Id*. at 579; *McDowell v. Brown*, 392 F.3d 1283, 1299, 1301 (11th Cir. 2004) (characterizing the experts' opinions as "too vague" and "more of a guess than a scientific theory."); *Rosen v. Ciba-Geigy Corp*., 78 F.3d 316, 319 (7th Cir. 1996) ([T]he courtroom is not the place for scientific guesswork, even of the inspired sort.").

1. Professor Scott's Opinions Lack Reliability Because They Are Based on Flawed Reasoning or Methodology.

Professor Scott's Report does not provide any basis for her "concern" about puberty delaying medication or her speculations about a teenager's ability to grasp its implications. The reason for this is simple: Professor Scott does not know what the effects of puberty delaying medication are on the brain, and she does not know whether teenagers can fully grasp its implications. She does not know what these implications are herself, and accordingly, all her opinions are hypothetical and unmoored from facts or data.

a. Puberty Delaying Medication

Her report is full of statements about the alleged lack of studies pertinent to the effects of puberty delaying medication. Report, ¶ 7 ("My concern is that we do not yet have enough evidence about the best ways to identify the individuals for whom [puberty delaying medication] are appropriate."); Report, ¶ 15 ("All the papers I can find suggest that we need much more data on the long-term brain effects of [puberty delaying medication] when administered around puberty, [and] the effects this can have on behaviour[.]"). Without any evidence (and with no experience or training in the subject), Professor Scott can only guess the effects of these treatments. Report, ¶ 15 ("As puberty is associated with very marked changes in the structure of the brain…the use of puberty blockers *may* have serious consequences for the development of the human brain.") (emphasis added); Report, ¶ 16 ("We need more research to be able to determine the *potential* for puberty blockers to be effective in alleviating some aspects of gender dysphoria[.]") (emphasis added). Guessing is not permitted under Rule 702. *McDowell*, 392 F.3d at 1301 (noting that while an expert may "draw conclusions from existing data," drawing "conclusions where there was no existing data" amounted to a "mere guess" that "fails the tests for expert opinion"); *Magical Farms, Inc. v. Land O'Lakes, Inc*., 2007 WL 4727225, at *2 (N.D. Ohio March 8, 2007) ("Dr. Ames' report is replete with statements like, 'suggest the possibility,' 'may have,' and 'I would be concerned,' all of which fail to rise to the level of a reasonable degree of certainty required by courts.").

To substantiate her untrained guesswork, Professor Scott briefly discusses—in a single paragraph—just five articles related to puberty delaying medication. *See* Report, ¶ 15. Only one of the articles is an original study pertaining to humans, namely, children with precocious puberty (Mul et al., 2001). *See* Report, ¶ 15; Scott Bibliography. Two other articles are not studies themselves, but rather a single commentary piece (Hayes, 2017) and a review of literature (Wojniusz et al., 2016), both pertaining to the treatment of precocious puberty. *See* Report, ¶ 15; Scott Bibliography. Finally, the other two studies pertain to sheep not people. *See* Report, ¶ 15; Scott Bibliography.[4] None of the studies pertain the use of puberty delaying medications for gender dysphoria in adolescents.

Notwithstanding the above, Professor Scott's discussion is nothing more than a recitation of findings from the above papers. She does not say anything about the methodologies behind those studies, whether they have been peer reviewed, or whether or how they are applicable to the context of using puberty delaying medications as treatment of gender dysphoria in adolescents. In fact, she disclaims them away after discussing them, saying "we cannot say if the results are due to direct effects of [puberty delaying medication] on the brain, heart and behaviour, or if they are secondary to this[.]" Report, ¶ 15. Without any qualifications or training in

[4] *But see Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 147 (1997) (offering animal studies showing one type of cancer in mice to establish causation of another type of cancer in humans is "simply too great an analytical gap between the data and the opinion offered").

these areas, her use of these articles to support her opinions about puberty delaying medication is completely unreliable and the type of hypothetical guesswork prohibited by Rule 702. *Lebron*, 772 F.3d at 1368 ("Expertise in one field does not qualify a witness to testify about others."); *Dura Automotive*, 285 F.3d at 614 ("A scientist, however well credentialed he may be, is not permitted to be the mouthpiece of a scientist in a different specialty.").

Most disturbingly, however, and demonstrative of her extremely flawed methodology, is the fact that she does not discuss any of the original studies that exist pertaining to the use of puberty delaying medications on transgender adolescents. There are at least three original, peer-reviewed studies that have looked specifically into the effects of puberty delaying medications on brain structure and function in transgender adolescents. *See* Corrected Edmiston Rebuttal Report, at ¶¶ 26, 29 (**Exhibit C**) (discussing Heesewijk et al., 2022; Soleman et al., 2016; Staphorsius et al., 2015). Indeed, none of these have found any significant effects of treatment on the brain. *Id.* Plaintiffs do not refer to these studies to argue the merits, but rather to starkly illustrate the flawed nature of Professor Scott's methodology. How can Professor Scott opine of the effects of puberty delaying medications on transgender adolescents' brains when she does not discuss any of the original, peer-reviewed studies looking at that question? The answer is she cannot.

Simply put, Professor Scott's concern over puberty delaying medication as a

treatment for gender dysphoria stems from her own lack of knowledge.[5] Not only does she not cite, let alone discuss, the most relevant studies in this area, but throughout her testimony, she repeatedly used the words "we don't know," when referring to the effects of puberty delaying medication. Tr. 24:11-14 ("[W]hat evidence we do have suggests that there are effects on the brain of delaying puberty. And we don't know what that might mean further down the line. We just don't know."); Tr. 68:20-21 ("Q. But you can't say here that these puberty blockers have any harmful effects on the brain? A. But we know that they change the brain and we don't know that that's not harmful."). Her concern is completely unreliable however because it ignores what we do know about puberty delaying medication. In other

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[5] It could be argued that Professor Scott's opinions really stem not out of just concern or lack of knowledge, but rather from personal feelings and biases about transgender people. Professor Scott is an active Twitter user. She often uses this platform to comment on a wide variety of topics outside her field of expertise, including transgender issues and treatments for gender dysphoria. In one tweet about a children's book for transgender youth and their families—that she did not read—she called the book a "cheap shot" and "reductive" because it "says that girls who like bugs and wear super hero capes and who don't like pink dresses are in fact boys." [**Exhibit E**]; Tr. 163:6-10; Tr. 164:12-16 ("Q. The book is about addressing that issue with your family. You didn't read the book? A. Well, that was – I've just quoted off the bits I saw. This is – you've asked me why I said it and that's why I said it."). Her rash comments about a children's book she did not read suggest a bias against the trans community.

In another tweet, Professor Scott showed disdain for a scholarship application that allowed applicants to "self-identify" as female. She wrote "Of God" in response to a tweet about the scholarship application. **[Exhibit F**] While her explanation speaks for itself, in summary, she believes that the trans community should be sectioned off from the cis community in what she calls "positive discrimination." Tr. 166:11 – 167:10.

words, her opinion ignores the research we have done on these treatments, none of which shows any significant effects on the brain. *See* Corrected Edmiston Rebuttal Report, ¶¶ 26, 29-30. In sum, Professor Scott's overall discussion about these studies is completely unreliable and should be excluded *in toto*.

b. Decision-making

Her concerns about a teenager's ability to grasp the implications of treatment is equally unreliable because the steps in her "analysis" are disconnected. In paragraphs 8-13 of the Report, Professor Scott explains how the brain develops over childhood and adolescence. Then, at paragraph 14, she says this pattern of brain development "*suggests*" that teenagers are prone to risky decision-making more than adults. From there she somehow concludes it is "very possible" that teenagers are unable to "fully grasp the implications of puberty blocking treatment." Report, ¶ 16.

There are several problems with this "analysis." First, her conclusion about teenagers being prone to risky behavior because of brain development is a guess, just like her concerns over puberty delaying medication. She cannot say with any certainty (or authority) that the pattern of brain development during adolescence leads to more risky behavior in teenagers. The same is true for her ultimate opinion about a teenager's ability to grasp the implications of these treatments. She does not cite a single study that supports this opinion. *McDowell*, 392 F.3d at 1301 (drawing "conclusions where there was no existing data" amounted to a "mere guess" that "fails the tests for expert opinion").

Second, there is a disconnect between the two steps in her analysis. Professor Scott never explains how a tendency toward risky behavior effects a teenager's ability to understand the implications of that behavior. In other words, she never explains how her conclusion about risky behavior leads to her concern over whether teenagers can grasp the implications of puberty delaying treatment. She There is thus a large "analytical gap" in her methodology that renders her ultimate conclusion unreliable. *See Joiner*, 522 U.S. at 146.

For her opinions to be reliable, Professor Scott must have "knowledge," which requires "more than subjective belief or unsupported assumptions." *Daubert*, 509 U.S. at 590. Professor Scott does not have the requisite knowledge for either of her opinions. To assume that her opinions are correct (despite a lack of evidence and experience) would be to rely on her *ipse dixit* based on conjecture to judge the reliability of her conclusion. *See Bowers v Norfolk Southern Corp*., 537 F.Supp.2d 1343, 1355 (M.D. Ga. 2007) (" The Court cannot rely on [the expert's] *ipse dixit* to judge the reliability of his conclusion[.]").

2. Professor Scott's Opinions are Vague and Imprecise.

Despite her "concerns" over the "potential effects" of puberty delaying medication, *see* Report, ¶¶ 4, 7, Professor Scott does not believe these treatments should be denied to all teenagers with gender dysphoria. She begins her report by saying it is "entirely possible that the use of puberty blockers is appropriate in some exceptional cases of gender dysphoria in prepubescent and adolescent individuals."

Report, ¶ 7. She repeated that sentiment in her deposition. Tr. 13:10-13 ("I think it's entirely possible that there are people, young people who this is an entirely appropriate course of treatment potentially."). When asked about whether she approves of complete bans pertaining to gender-affirming care, like the Challenged Exclusion in this case, Professor Scott could not give a straight answer. On the one hand, she acknowledged that all-inclusive bans on coverage are a bad idea. Tr. 13:22-23 ("I don't think it's a good idea to ban treatment in a blanket way."; Tr. 14:21-23 ("I think it should be something that's worked out in terms of a scientific and medical approach."). On the other hand, she understood she was offering an opinion in support of one such blanket ban. Tr. 12:21 – 13:8. When asked whether she would vote for or against the Challenged Exclusion in this case, she said she would "abstain like a coward." Tr. 16:17.

Opinions like these are too vague and imprecise to be sufficiently reliable. Professor Scott cannot identify when, or under what circumstances, puberty delaying medication may be appropriate for teenagers. She thus cannot say when the unknown risks of these treatments outweigh their benefits. Where she draws the line is completely unknown, making her opinion vague and imprecise. *See Ward v Carnival Corporation*, 2018 WL 11383459, at *6 (S.D. Fla. July 31, 2018) (excluding expert testimony because it was "unclear precisely what [the expert] was claiming.").

Her opinion about a teenager's decision-making ability is equally imprecise. Professor Scott is not certain whether all teenagers are prone to risky behavior, which

is the sole basis for her opinion. Tr. 141:9-19 (Q. Is [riskiness] common for all adolescents?" A. Well, I mean adolescence is very variable like all humans."). Her opinion is also based on research related to decision-making in a "hot context," Report, ¶ 14, which ignores the body of research and peer-reviewed literature on the contextual nature of decision-making in adolescence. Corrected Edmiston Rebuttal Report at ¶ 18 (discussing eleven (11) peer-reviewed papers on the contextual nature of adolescent decision-making). She also omits all literature on decision-making in the medical context and particularly decision-making about treatment of gender dysphoria occurring over several years. These cavernous omissions render her opinion about decision-making in the "hot" context both imprecise and misleading by leaving out the proper context.

3. Professor Scott's Opinions are Far Outside the Mainstream

General acceptance in the relevant scientific community is an important element to the reliability inquiry. See *Allison v. McGahn Medical*, 184 F.3d 1300, 1313 (11th Cir. 1999). Not only is widespread acceptance an important factor in assessing the reliability of an expert's opinions, but the fact that a known theory "has been able to attract only minimal support within the community may properly be viewed with skepticism." *Daubert*, 509 U.S. at 594. Here, Professor Scott's opinions about the propriety of puberty delaying treatment is far outside the mainstream of medical and scientific opinion. In fact, her views have been explicitly rejected by every relevant scientific and medical community. Professor Scott says she is "slightly

worried" about using puberty delaying medication to treat even precocious puberty, the indication for which it was originally developed and for which it is approved by the FDA. Tr. 78:7-8; *see id*. 78:14-18 (expressing concerns about using puberty delaying treatment for any purpose because it is not "necessarily . . . safe" and "the data is not 100 percent clear that it doesn't have an effect" on cognitive function); Tr. 156:19-21 ("[M]y primary concern is about puberty blockers and giving them in adolescents and the risk associated with that."). Professor Scott claims she "doesn't know" whether her "concerns with puberty blockers for precocious puberty [are] shared by the medical community." Tr. 78:19-22. In fact, they are not shared, and indeed, run counter to the opinions of mainstream scientists and clinicians. See Corrected Edmiston Report, ¶ 38; Shumer Rebuttal Report ¶¶ 7, 54, 64 (**Exhibit D**); Dekker P.I. Hrg. Tr., at 29:16- 36:18 [ECF 62] (noting that the majority of major medical associations support gender-affirming care for adolescents and adults); *see also, e.g.*, *Brandt v. Rutledge*, 551 F.Supp.3d 882, 890 (E.D. Ark. 2021) ("The consensus recommendation of medical organizations is that the only effective treatment for individuals at risk of or suffering from gender dysphoria is to provide gender-affirming care [include puberty delaying treatment]."") (emphasis added), *aff'd*, 47 F.4th at 671. Because Professor Scott's opinions about puberty delaying treatment are "not generally accepted by the scientific community, and [are] unsupported by other studies" her testimony is unreliable. *Allison*, 184 F.3d at 1319.

D. Professor Scott's Opinions Will Not Assist the Trier of Fact.

Expert testimony is helpful to the trier of fact if it explains subjects that are beyond the understanding of the average lay person. *Frazier*, 387 F.3d at 1262. The testimony must offer more than what lawyers can argue in closing arguments. *Id*. Expert testimony is not helpful if it fails to "fit" with the facts of the case. *McDowell*, 392 F.3d at 1299. This happens when a large analytical leap must be made between the facts and the opinion. *See Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997) (offering animal studies showing one type of cancer in mice to establish causation of another type of cancer in humans is considered "too great an analytical gap").

Professor Scott's expert testimony will not assist the trier of fact for several reasons. *First*, her opinion about the ability of teenagers to fully grasp their decision to undergo treatment does not "fit" the facts of the case. She references these decisions as if they were made by the adolescent patient alone, that is, without any advice or assistance from medical professionals or other adults. Report, ¶ 16 ("All the evidence we have suggests that the complex, emotionally charged decisions required to engage with this treatment are not yet acquired as a skill at this age, both in terms of brain maturation and in terms of behaviour."). But the reality is that all decisions about whether to administer gender-affirming care are made by a group of individuals including the patient's family and healthcare providers. And, for individuals under 18, these decisions are ultimately made by the patient's parent or legal guardian. Professor Scott acknowledged this point in her deposition. Tr. 146:5-

10 (Q. But we're not talking about teenagers deciding about gender-affirming care themselves in this case, right? A. No. I understand that this would be something where the consent is not with the teenager."). Accordingly, her opinion on teenager decision-making is irrelevant to the facts of the case. *See Kadel*, 2022 WL 3226731, at *14 (excluding Dr. Lappert's opinion on informed consent in the context of gender dysphoria because the patient's father gave consent).

Second, this same opinion is well within the understanding of the average lay person, and it is certainly something counsel can argue in closing. Professor Scott concedes this point in her report when she describes the following as a "lay understanding of what neuroscience is now confirming." She says: "teenage brains on the whole are structurally and functionally different from adult brains, and this affects both their engaging with risky behaviour, and their understanding of the implications of risky behaviour." Report, ¶ 8. She confirmed the same in her deposition. Tr. 143:7-11 ("Q. Do you need to be an expert in neuroscience to understand that teenagers on the whole engage in risky behavior? A. No. Like I said in my report, it's something that all cultures recognize."). Since there already exists a "lay understanding" of her opinion about teenage behavior that "all cultures recognize," her opinion will not assist the trier of fact in this case. It is well-established that untestable "common sense" does not satisfy Rule 702's requirements. *See Fedor v. Freightliner, Inc.*, 193 F.Supp.2d 820, 832 (E.D. Pa. 2002) ("Generalized common sense does not rise to the level of expert opinion solely

because it is offered by someone with an academic pedigree.").

Third, her opinion about the unknown effects of puberty delaying medication is also within the understanding of the average person. The Court does not need an expert to explain the things we do not know. These can easily be explained in closing argument. *See Frazier*, 387 F.3d at 1262 ("Proffered expert testimony generally will not help the trier of fact when it offers nothing more than what lawyers for the parties can argue in closing arguments.").

Fourth, as noted above, her opinion about puberty delaying medication is based in part on animal studies without any connection to the treatment of gender dysphoria in humans. Report, ¶ 15; Tr. 71:11-15. Professor Scott does not even attempt to link these animal studies to humans, and as a result, such studies do not offer any support for her conclusions about the human brain. Therefore, they do not assist the trier of fact. *Joiner*, 522 U.S. at 146; *Kilpatrick*, 613 F.3d at 1338.

CONCLUSION

WHEREFORE, based on the foregoing, Plaintiffs respectfully request that the Court grant the instant Motion and exclude Professor Scott's expert report, opinions and testimony in their entirety under *Daubert* and the Federal Rules of Evidence.

* * *

Respectfully submitted this 7th day of April, 2023.

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CERTIFICATE OF WORD COUNT

According to Microsoft Word, the word-processing system used to prepare this Motion and Memorandum, there is a combined total of 5,455 words in the Motion and the Memorandum of Law.

/s/ Gary J. Shaw
Gary J. Shaw

CERTIFICATE OF SATISFACTION OF ATTORNEY-CONFERENCE REQUIREMENT

Pursuant to Local Rule 7.1(B), counsel for the Plaintiffs conferred with counsel for the Defendants on April 5, 2023. Counsel for Defendants indicated that same day that Defendants oppose the relief sought.

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the foregoing was served by email on April 7, 2023, on all counsel of record:

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Gary V. Perko (FBN 855898)
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COUNSEL FOR DEFENDANTS

/s/ Gary J. Shaw
ATTORNEY FOR PLAINTIFFS

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF FLORIDA
Tallahassee Division

AUGUST DEKKER, et al.,

Plaintiffs,

v. Case No. 4:22-cv-00325-RH-MAF

JASON WEIDA, et al.,

Defendants.
_____/

DECLARATION OF ATTORNEY GARY J. SHAW IN SUPPORT OF PLAINTIFFS' MOTION TO EXCLUDE EXPERT TESTIMONY OF SOPHIE SCOTT, PH.D.

I, Gary J. Shaw, pursuant to 28 U.S.C. § 1746, declare as follows:

1. I am over the age of eighteen and make this declaration from my own personal knowledge. If called as a witness, I could and would testify competently to the matters stated herein.

2. I am an attorney with Pillsbury Winthrop Shaw Pittman in Washington, D.C., and I have been retained by Plaintiffs as co-counsel in the above-captioned matter.

3. I make this Declaration in support of Plaintiffs' Motion to Exclude Expert Testimony of Sophie Scott, Ph.D.

4. Attached as **Exhibit A** is a true and correct copy of the Expert Report of

Sophie Scott, Ph.D., dated February 16, 2023.

5. Attached as **Exhibit B** is a true and correct copy of the deposition transcript of Professor Sophie Scott dated March 20, 2023.

6. Attached as **Exhibit C** is a true and correct copy of the Corrected Expert Rebuttal Report of E. Kale Edmiston, Ph.D., dated March 22, 2023.

7. Attached as **Exhibit D** is a true and correct copy of the Rebuttal Report of Daniel Shumer, M.D., dated March 10, 2023.

8. Attached as **Exhibit E** is a true and correct screenshot of a tweet dated October 11, 2018.

9. Attached as **Exhibit F** is a true and correct screenshot of a tweet dated September 20, 2016.

Executed on April 7, 2023

By: */s/ Gary J. Shaw*

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EXHIBIT A

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF FLORIDA
TALLAHASSEE DIVISION

AUGUST DEKKER, et al.,

Plaintiffs,

v. Case No. 4:22-cv-00325-RH-MAF

JASON WEIDA, et al.,

Defendants.
_____/

EXPERT REPORT OF SOPHIE SCOTT, PH.D.

Pursuant to 28 U.S.C. 1746, I declare:

1. I have been retained by counsel for Defendants as an expert witness in connection with the above-captioned litigation. I have actual knowledge of the matters stated in this report. My professional background, experience, and publications are detailed in my curriculum vitae. A true and accurate copy of my curriculum vitae, which includes a list of my publications, is attached as Exhibit A to this report.

2. I have testified as an expert witness in the following cases, at trial or in deposition in the last four years: Bell v Mrs A vs Tavistock and Portman Trust, Case No: CO/60/2020, December 2020.

3. I am being compensated at an hourly rate for actual time devoted, at the rate of $400 per hour including report drafting, testimony, and consultation. My compensation does not depend on the outcome of this litigation, the opinions I express, or the testimony I provide.

4. The opinions expressed in this report are based on my training and experience as a neuroscientist, my reading and my assessment of the relevant neuroscientific literature on brain development, and the potential effects of gonadotropin-releasing hormone (GnRH) agonists (the most common form of what are often called puberty blockers) on the developing brain.

5. If called to testify in this matter, I would testify truthfully and based on my expert opinion. The opinions and conclusions I express herein are based on a reasonable degree of scientific certainty.

Introduction

6. I am the Director of University College London's (UCL's) Institute of Cognitive Neuroscience. I have published over 130 peer reviewed scientific papers, including papers in Nature, Science, and the Proceedings of the Academy of Natural Sciences. I am a fellow of the Academy of Medical Sciences, and of the British Academy. Since my PhD was awarded in 1994, I have been working in cognitive neuroscience, a scientific field that examines the relationships between human behaviour to the human brain, and how these can be affected by age, disease and individual differences. *See* Attached Curriculum Vitae.

7. As a neuroscientist I am very familiar with the existence of variations of sexual preference, and the existence of variations in gender identity. I think that the anecdotal evidence we have suggests that transition may, for some younger people, be an effective treatment for gender dysphoria, and that the medical approaches taken to achieve this may therefore be appropriate. Thus is it entirely possible that the use of puberty blockers is appropriate in some exceptional cases of gender dysphoria in prepubescent and adolescent individuals. My concern is that we do not yet have enough evidence about the best ways to identify the individuals for whom they are appropriate: we have not identified any biological markers or other characteristics to identify individuals for whom GnRH antagonists might provide effective; we do not have any reliable studies that show which young gender dysphoric individuals will remain gender dysphoric after adolescence; and we thus do not yet know who might benefit from

this highly medicalised and largely non-reversible treatment. I am also very concerned that the implications of the effects of puberty blockers on the developing brain and body are not well understood. In both of these areas much more research is needed.

8. All cultures recognise the onset of adolescence as the start of the entry into the adult world: it is a journey into that world, and a journey that takes place over several years. In 2005 the US supreme court, influenced partly by this emerging neuroscience research, increased the minimum age for capital punishment to be the same as that for voting and serving on juries. Around the world, many such limitations on the responsibility for teenagers for their own actions are in place – alongside laws which mean that teenagers could not engaging in risky behaviours that could place them or others at risk or having to live with long terms consequences (e.g. ages for driving, drinking alcohol, age of consent, getting a tattoo). Much of this reflects a lay understanding of what neuroscience is now confirming – there is variation from child to child, but teenage brains on the whole are structurally and functionally different from adult brains, and this affects both their engaging with risky behaviour, and their understanding of the implications of risky behaviour.

9. The human brain is formed of approximately 89 billion brain cells, or neurones, most of which are grown during gestation (Bayer et al. 1993; Rakic 1995). Following birth, there is a further period of extended brain development. Directly after birth, the brain grows rapidly, quadrupling in size

between birth and age 6, when it is roughly 90% the size of an adult brain. However the pattern of growth is underpinned by some complex changes that are occurring. These are:

- Synaptic pruning
- Myelination of different brain networks
- Differential growth of specific functional and anatomical areas.

10. Before I go into this in detail it's important to note that brain cells, or neurones, are formed of a cell body, with a long projection (an axon) and branch-like shorter projections (dendrites) from the cell body or from the far end of the axon. The axons can be thought of as ways the neurone can connect to more distant neurones, while the dendrites connect to nearby neurones. These connections are called synapses. Changes in the brain – associated with learning and development - occur largely through the connections between neurones, which can be through the strengthening of existing connections, or through the development of new dendritic connections. The axons are coated in a slim fatty sheath, called myelin: this enables the electrical discharges that enable transfer of information in the brain to be propagated rapidly along the length of the axon. Myelination is a process that increases the speed and efficiently of neural function. Neurones are highly organised in the brain, with the cell bodies forming structure layers on the surface of the brain (the cortex), as well as in sub cortical nuclei of cell bodies: the axons form tracts of connections between cortical areas, to and from sub cortical areas, and between the two hemispheres of the brain.

These tracts look white, due to the fatty myelin sheaths: this leads to the name 'white matter' for these tracts or connective networks. In contrast, the unmyelinated neuronal cell bodies look grey, hence the term 'grey matter'.

11. At birth and in early infancy, many dendritic connections exist and are created between neurones: this is known as *synaptic exuberance*. In the early years of life these are rapidly pruned, at first quickly, then more slowly. During adolescence a more adult profile of synaptic connections starts to appear: this appears most slowly in prefrontal fields compared to sensory and is still not established fully at age 18yrs (Huttenlocher and Dabholkar, 1997). The relationship between synaptic exuberance and pruning and their implications for the developing brain and experience are still being explored, but in terms of brain connectivity, the adult pattern is not yet established at 18: development continues into the early 20s.

12. Myelination in the human brain begins in visual brain areas a couple of months before birth and continues in other sensory brain areas over the first year. This process continues in other cortical and subcortical systems into the middle of the third decade. This has been expressly linked to the development of cognitive skills in children and adolescents, as myelination greatly improves the speed of condition of neurones, and hence their efficiency. Myelination proceeds in a roughly caudal to rostral direction in the brain, which means form back to front. This means that it is frontal and prefrontal fields that are those continuing to be myelinated into the mid 20s: this has been confirmed by more recent studies

looking at fractional anisotopy in the brain (Lebel et al., 2008). At 18yrs old, the connections to the frontal lobes are not myelinated like a mature adult brain, and this is likely to affect frontal lobe functions.

13. Throughout childhood, the brain grows and changes: this involves a non-linear pattern of change in the proportion of white and grey matter, which may partly involve changes in myelination (see above) and also the loss of cells through cell death (Sowell et al. 2004). A recent study looking at this pattern into adolescence found that "First, we found evidence for continued development of both intracranial volume (ICV) and whole brain volume (WBV) through adolescence, albeit following distinct trajectories. Second, our results indicate that CGMV is at its highest in childhood, decreasing steadily through the second decade with deceleration in the third decade, while CWMV increases until mid-to-late adolescence before decelerating" (Mills et al, 2016). This indicates that considerable changes are still happening in the structure of the adolescent brain. In terms of specific brain areas, while the cortex continues to thin through adolescence, the decreases are most marked in the parietal lobes and least marked (or growth is seen) in temporal and prefrontal fields (Tamnes et al, 2017).

Implications

14. The pattern of maturation of the brain in adolescence suggests a particular issue with frontal lobe functions – the frontal and temporal lobes are showing a different pattern of change (in terms of movement towards adult profiles) compared to more caudal fields, and the frontal lobes are the last to be

fully myelinated. The frontal lobes are associated with complex cognitive control processes, so called 'meta-cognitive processes' that enable us to plan our behaviour, control our responses, to be able to adapt our behaviour to different contexts and requirements, and to anticipate the implications and consequences of behaviour. The absence of mature frontal lobe connectivity and functions has been linked to increased impulsivity and risk-taking in adolescence, and to their greater susceptibility to peer opinions and behaviour (Blakemore and Robbins, Nature Neuroscience, 2012). Functional imaging studies – addressing how brains function under different task requirement – have shown that while adults recruit frontal lobe networks during decision making tasks, teenagers are more likely to recruit 'limbic networks" i.e. sub cortical networks linked more to emotional processing and reward processing: the implication is that the differential integrity of frontal lobe connectivity leads to teenagers making different, more risky decisions than adults, and relying on different brain networks to do so. This is backed up by behavioural studies showing that when decision making is 'hot' (i.e. more emotional), under 18yr olds make less rational decisions than when the responses are being made in a colder, less emotional context.

15. Puberty blockers (specifically, gonadotrophin-releasing hormone agonists) work by preventing the release of gonadotrophin-releasing hormone from the hypothalamus. Gonadotrophin releasing hormones have many effects, including stimulating the gonads (testes and ovaries) to produce testosterone and oestrogen. In childhood, the level of Gonadotrophin releasing hormones is very

low, but an increase in this prompts the onset of puberty, with the release of testosterone and oestrogen; these in turn have masculinising or feminising effects on the bodies and the brain. As puberty is associated with very marked changes in the structure of the brain (as outlined above) the use of puberty blockers may have serious consequences for the development of the human brain. We know from studies on sheep (Nuruddin et al, 2013) that treatment around the onset of puberty with gonadotrophin-releasing hormone agonists is associated with significant differences in the size of the amygdala (found to be larger in treated animals) and this was linked to some differences in emotional reactions. The male treated sheep showed greater approach responses and more risk taking behaviours, while the treated female sheep showed higher levels of anxiety and greater avoidance behaviour (Wojniusz et al, 2011). A behavioural study of natal girls who were treated for precocious (early) puberty with Gonadotrophin releasing hormone agonists (Wojniusz 2016) found that they also showed significant greater emotional reactivity on one of the tests used, relative to the control group. The treated girls also showed significantly lower heart rates than the untreated control group. In a commentary on this article (Hayes, 2017) it was pointed out that there were also notably lower scores on IQ measures and sub scales in the group of girls who were treated with Gonadotrophin releasing hormone agonists. He points out that “their reassuring statement in the abstract that girls undergoing GnRHa treatment for CPP and controls “showed very similar scores with regard to cognitive performance” and their conclusion that

"GnRHa treated girls do not differ in their cognitive functioning ... from the same age peers" (Wojniusz et al., 2016) may be overly optimistic. These statements minimize the fairly substantial difference found in IQ scores" (Hayes, 2017). Hayes also points to an older study that found a significant drop in IQ associated with taking triptorelin acetate to treat precocious puberty (Mul et al, 2001). Note that in all of these cases, in humans and other mammals, we cannot say if the results are due to direct effects of the Gonadotrophin releasing hormones on the brain, heart and behaviour, or if they are secondary to this (e.g. due to the altered levels of testosterone or oestrogen, or changes in the heart rate itself). All the papers I can find suggest that we need much more data on the long-term brain effects of Gonadotrophin releasing hormones when administered around puberty, the effects this can have on behaviour, and the extent to which any of this is altered if the treatment with Gonadotrophin releasing hormones is stopped.

16. I am very concerned that the current treatment regime is exposing young people to significant risk of harm. The greater susceptibility to peer pressure in those under 18 may make them especially vulnerable to risk taking, and this may well be enhanced by social media, where actions can be encouraged without any responsibility for outcomes. We need more research to be able to determine the potential for puberty blockers to be effective in alleviating some aspects of gender dysphoria, and to be able to differentiate those who might be helped by this treatment from those who will not. Furthermore, given the risks of puberty blocking treatment, and the fact that these will have irreversible, lifelong

effects, it is very possible for an adolescent to be unable to fully grasp the implications of puberty-blocking treatment, even if the risks are well explained. All the evidence we have suggests that the complex, emotionally charged decisions required to engage with this treatment are not yet acquired as a skill at this age, both in terms of brain maturation and in terms of behaviour.

I declare, pursuant to 28 USC § 1746, under penalty of perjury that the foregoing is true and correct. Executed on February 16th, 2023.

/s/ Sophie Scott
Sophie Scott, Ph.D.

Exhibit "A"

PROF SOPHIE KERTTU SCOTT CBE, FMEDSCI, FBA

Date of Birth: 16-11-1966
Address: Institute of Cognitive Neuroscience, UCL, 17 Queen Square, London, WC1N 3AR
email: sophie.scott@ucl.ac.uk

CURRENT POSITION

2019 – Director, Institute of Cognitive Neuroscience, University College London

EDUCATION/QUALIFICATIONS

1994 University College London, PhD in Cognitive Science
1990 Polytechnic of Central London, BSc (Hons) 2:1, Psychology

PROFESSIONAL HISTORY

1993-1998 MRC Applied Psychology Unit, Cambridge, Senior Scientific Officer
1998-2001 Research Fellow, Institute of Cognitive Neuroscience, UCL.
2001-2005 Wellcome Career Development Fellow, Dept. Psychology, UCL
2004 - Group Leader, Speech Communication Lab
2006- Professor of Cognitive Neuroscience, UCL
2005-2016 Wellcome Trust Senior Fellow, Institute of Cognitive Neuroscience
2013-2019Deputy Director, Institute of Cognitive Neuroscience, UCL
2019 – Director, Institute of Cognitive Neuroscience, University College London
I took maternity leave between June 2006-June 2007.

PRIZES AND RECOGNITION

2022 Awarded an Honorary degree by the University of Westminster
2021 awarded the Michael Faraday prize by the Royal Society
2020 appointed Commander of the Most Excellent Order of the British Empire for services to Neuroscience
2019 Royal Literature Society "Reading Matters" prize, for "The Neuromantics", my podcast with poet and writer Dr Will Eaves
2017 presented the Royal Institution Christmas Lectures
2017 Royal Society Summer Science Exhibition, "What's in a Voice?"
2016 elected as a Fellow of the British Academy
2016-2018 UCL TEDx License holder

2015 spoke at the annual TED conference, Vancouver (talk has been viewed over 4.4 million times on TED.com).
2015 gave Prize Lecture at the Physiology Society meeting, Cardiff.
2014 included in Who's Who
2013 won UCL Provosts' Award for Public Engagement (grade 8 and above category).
2012 Royal Society Summer Science Exhibition, "LOL: Science and Art of Laughter"
2012 Elected as Fellow of the Academy of Medical Sciences
2003 Royal Society Summer Science Exhibition, "Science of Speaking"

SUPERVISION OF GRADUATE STUDENTS

Since 2002, 14 PhD students supervised at UCL, and 35 MSc students at UCL, 2 at City University and one at the University of Reading

EDITORIAL WORK

2015 – associate editor for *The Psychologist* (British Psychological Society monthly journal).
2009 – 2014 Editorial Board of *Cognitive Neuroscience*
2010 – 2013 Section Editor, Language, *Neuropsychologia*
2008 – 2015 Associate Editor of *Brain and Language*
2004 –2009 Associate Editor of the *Quarterly Journal of Experimental Psychology*

MANAGEMENT AND FACILITATION

2020 - PALS Director for EDI
2015 – member, PALS Academic Careers and Diversity Committee
2015-2019– chair of ICN Public Activities Committee
2014 – 2019 deputizing for Prof Neil Burgess (ICN Director) at Faculty of Brain Sciences' Faculty Executive Committee meetings
2004 – representing the Speech Communication Group at the ICN Group Leader's committee

JUDGING AND COMMITTEES

2022 - member of ILCB advisory board
2019- Chair of Board of Trustees, Told By An Idiot theatre company (https://www.toldbyanidiot.org/about/)
2017- 2022 member of the Royal Society Dorothy Hodgkin Fellowship Committee
2015- associate Editor of the Psychologist and Digest Policy Advisory Committee, British Psychological Society

2015 Judge, Comment Awards
2015, 2018 Judge, Philip Leverhulme Prize
2014 Judge, Wellcome science writing prize
2013- Trustee, Jericho House theatre company (registered charity number 1131984)

EXTERNAL EXAMINING

2009-2012 External Examiner, BSc Psychology, University of Sussex
2009-2013 External Examiner, MSc Cognitive Neuroscience, University of York
2015-2019 External Examiner, BSc Psychology, University of Reading

TEACHING

2011-2014 Course convener, "Theories and Paradigms in Cognitive Neuroscience" UCL MSc in Cognitive Neuroscience.
2015- Module Convener, "Science Communication for Cognitive Neuroscientists" UCL MSc/MRes in Cognitive Neuroscience.
2023- Module Convener, "Power, Inclusion, Exclusion and Working with local communities".

GRANTS

Wellcome Trust Hub award development funding, for 'Talking Funny', £13000 over 18 months
Wellcome Trust Public engagement award, for "What's in a Voice" exhibit at the Royal Society Summer Science Exhibition, £20,000 over 12 months.
Wellcome Trust People Award for public engagement activities: for LOL event at the Royal Society, awarded 2012, amount £19,000 over 12 months.
Wellcome Trust Senior Fellowship, awarded April 2010 "Neurobiology of speech communication - cognition, plasticity, and social interactions" total amount awarded £1,184, 506, over 60 months.
Wellcome Trust Senior Fellowship, awarded May 2004 'the Neurobiology of Speech Perception – Cognitive and Clinical Links'. Total amount awarded £800,270, over 60 months.
Wellcome Trust RCDF grant, from April 2001-April 2005, 'the Neurobiology of speech perception'. Total amount awarded £358,376, over 48 months.
Marie Curie Incoming Scientist Fellowship, awarded November 2004, sponsoring Narly Golestani, £100,914 over 24 months.
ESRC grant awarded for new post doctoral researchers, sponsoring Charlotte Jacquemot, awarded May 2004, £30,919 awarded over 12 months.
ESRC grant awarded for new post doctoral researchers, sponsoring Patti Adank, awarded May 2005, £31,591 awarded over 12 months

ESRC +3 studentship award, awarded May 2004 (supervising Carolyn McGettigan)
British Academy meetings award, for the John Morton Festschrift, £2000
Experimental Psychology Society research seminar award, for the John Morton Festschrift, £3000
British Association for the Advancement of Science award for Key events in National Science and Engineering Week, 2008, £1000

ACADEMIC SUPERVISION

2001 -2004 Supervised Charvy Narain, the research assistant on my Wellcome RCDF award. Charvy was awarded a PhD in 2003 and took a job as an editor at Nature Neuroscience: she is now a Scientific Outreach Manager at the University of Oxford.
2002 -2006 supervised Disa Sauter, a research student in the Dept. Psychology at UCL. Disa passed her PhD viva without corrections in December 2006, and currently holds her a lecturer position at the University of Amsterdam..
2004 -2005 Supervised Dr. Charlotte Jacquemot, a post-doctoral fellow. Charlotte has been awarded a permanent CNRS position in France, which she began in 2006
2004 - 2012 supervised Carolyn McGettigan, a research student in the Dept of Human Communication Sciences. Carolyn passed her PhD viva without corrections in March 2008, was employed as a post-doctoral fellow on my Wellcome SRF grant until 2012 when she left to take up a lectureship at RHUL. She is now a Professor at UCL.
2005 -2006 supervised Dr. Patti Adank, a post-doctoral fellow. Patti is now a Professor at UCL.
2005 -2008 Supervised Dr Frank Eisner as a post-doctoral fellow on my Wellcome SRF award. Since January 2009, Frank held post-doc position at the Max-Planck-Institute for Psycholinguistics in Nijmegen, and is now a researcher at the Centre for Cognition of the Donders Institute for Brain, Cognition and Behaviour.
2005 -2007 Supervised Dr. Jonas Obleser as a post-doctoral fellow on my Wellcome SRF award. Since April 2007, Jonas has held a Junior Staff Scientist position at the Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany, where ran his own research group: he is now a Professor at the Department of Psychology, University of Lübeck.
2005-2007 supervised Dr. Narly Golestani, a post-doctoral fellow, who now heads the Brain and Language Lab at the Cognitive Science Hub of the

University of Vienna, Austria, and at the Department of Psychology at the University of Geneva, Switzerland.2008 -2009 – supervised Nicholas Abreu, who has a Fulbright Scholarship to work in the UK for a year. Nicholas started medical school at Harvard in September 2009.
2009 - 2013 Dr Zarinah Agnew appointed as a post-doctoral fellow on my Wellcome SRF grant. Zarinah now works at UCSF as a post-doc in John Houde's lab.
2010 -2015 supervised Pradheep Shanmugalingam as an ESRC funded PhD student. Pradheep is now training in simultaneous translation.
2012 - 2015 supervising Kyle Jasmin as a PhD student on the UCL/NIH program (NIH supervisor Alex Martin). Kyle joined my lab as a post-doctoral fellow and then was awarded a a Leverhulme research fellowship at Birkbeck: he is now a lecturer at Royal Holloway UL.
2012 -2013 Nadine Lavan joined my lab as an RA for 12 months. Nadine left to take up a PhD place at RHUL: she now holds a Wellcome Fellowship at QMUL.
2013 - Supervising Sophie Meekings as an ESRC funded PhD student. Sophie was awarded a BA fellowship at Newcastle University, and was awarded a Dorothy Hodgkin fellowship in 2021, held at University of York.
2013 - Supervising Sinead Chen as a PhD student funded by a grant from the Taiwanese Government. Sinead now works for a policy think tank in Taiwan.
2013 -2015 Samuel Evans joined my lab as a post-doc on my Wellcome SRF grant. Now a lecturer at the University of Westminster
2013 – 2014 Dana Boebinger joined my lab as an RA. Dana left in August 2014 to start a PhD at Harvard, she is now a post do at the University of Rochester.
2015 – 2016 César Lima joined my lab as a senior post-doctoral fellow on my Wellcome SRF grant. César Lima is Assistant Professor of Psychology at Iscte - University Institute of Lisbon since 2017.
2017- Qing Cai joined my lab as a PhD student with funding from the Chinese government from 2018.
2017- Alexis Deighton McIntyre joined my lab as a PhD student with a UCL Graduate School studentship. In October 2021 she joined the MRC CBU as a postdoctoral researcher.
2018-Addison Billings joined my lab as a PhD student
2019 - Efe Caswell Niven joined my lab as a PhD student

SCIENTIFIC PUBLICATIONS-*REFEREED ARTICLES*

1. Scott, SK, Jasmin, K (2022) Rostro-caudal networks for sound processing in the primate brain, Frontiers in Neuroscience, 16, 1076374, 10.3389/fnins.2022.1076374
2. Scott, SK, Cai, CQ, Billing, A (2022) Robert Provine: the critical human importance of laughter, connections and contagion. Philosophical Transactions of the Royal Society B-Biological Sciences, 377(1863) 20210178 10.1098/rstb.2021.0178
3. Scott, SK (2022) Why public engagement is important for neuroscientists. Nature Reviews Neuroscience, 23(8):453-4.
4. MacIntyre, AD, Scott, SK (2022) Listeners are sensitive to the speech breathing time series: Evidence from a gap detection task. Cognition, 225, 105171 10.1016/j.cognition.2022.105171
5. Conde, T, Correia, AI, Roberto, MS, Scott, SK, Lima, CF, Pinheiro, AP (2022) The time course of emotional authenticity detection in nonverbal vocalizations, Cortex, 151:116-132.
6.
7. Billing, ADN, Scott, SK (2022) Possible limitations of perceptual studies for informing production networks-The case of laughter. Cortex, 148: : 218-221.
8. MacIntyre, AD, Cai, CQ, Scott, SK (2022) Pushing the envelope: Evaluating speech rhythm with different envelope extraction techniques, Journal of the Acoustical Society of America. 151(3):2002:2026.
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12. Billing ADN, Cooper RJ, Scott SK (2021) Pre-SMA activation and the perception of contagiousness and authenticity in laughter sounds, Cortex, 143: 57-68.
13. Scott SK (2021) The neural control of volitional vocal production-from speech to identity, from social meaning to song. Philos Trans R Soc Lond B Biol Sci, 377(1841):20200395.

14. Lavan N, Scott SK, McGettigan C (2017) Impaired generalization of speaker identity in the perception of familiar and unfamiliar voices. J Exp Psychol Gen., 145(12):1604-1614
15. Cosme G, Rosa PJ, Lima CF, Tavares V, Scott S, Chen S, Wilcockson TDW, Crawford TJ, Prata D (2021). Pupil dilation reflects the authenticity of received nonverbal vocalizations. Scientific Reports. 11:3733.
16. Meekings S, Scott SK. (in press) Error in the Superior Temporal Gyrus? A Systematic Review and Activation Likelihood Estimation Meta-Analysis of Speech Production Studies. Journal of Cognitive Neuroscience.
17. Cai Q, Chen S, White SJ, Scott SK (2019). Modulation of humor ratings of bad jokes by other people's laughter. Current Biology. 29(14):R677-R678
18. Scott SK (2019) From speech and talkers to the social world: The neural processing of human spoken language. *Science*. Oct 4;366(6461):58-62.
19. Jasmin K, Lima CF, Scott SK (2019) Understanding rostral-caudal auditory cortex contributions to auditory perception. Nature Reviews Neuroscience, 20(7):425-434
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EXHIBIT B

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF FLORIDA

TALLAHASSEE DIVISION

AUGUST DEKKER, et al.,
Plaintiffs,
vs.
JASON WEIDA, et al.,
Defendants.
_____/

REMOTE VIDEOCONFERENCE DEPOSITION
OF
SOPHIE SCOTT, Ph.D.
Taken on behalf of the Plaintiffs

DATE TAKEN: Monday, March 20, 2023

TIME: 11:00 A.M. - 3:00 P.M.

LOCATION: Zoom Videoconference

STENOGRAPHICALLY REPORTED BY:
TRACY LYN FAZIO, FPR
VERITEXT LEGAL SOLUTIONS
JOB NO.: 5823283

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I N D E X

PLAINTIFFS' EXHIBITS MARKED

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BY MR. SHAW:

Q Professor Scott, do you understand what this case is about?

A I understand some of the background in terms of laws that have been changed in the U.S. generally, and this is specifically a case in Florida about the removal of healthcare to people who are transgender.

Q Is that all of your understanding?

A It's the most general sense, yeah. I'm sure there's many, many aspects and details I don't know about.

Q So you understand that we're going to discuss a rule issued by one of the State -- an agency with the State of Florida, correct?

A Uh-huh.

Q And that agency is the Agency for Healthcare Administration or AHCA. Do you understand that?

A I do. Thank you.

Q And you understand that this rule bans all services for treatment of gender dysphoria, including puberty blockers, hormones, sex reassignment surgeries, and any other procedures that alter the primary or secondary sexual

characteristics?

MR. BEATO: Object to form. But you can answer that, Dr. Scott.

A Yes. Yes, I understand that.

BY MR. SHAW:

Q Do you understand that you're giving an opinion in support of that rule?

A I do.

Q Do you support that rule?

A I put this in my report. I think it's entirely possible that there are people, young people who this is an entirely appropriate course of treatment potentially. The problem at the moment is we don't know who those young people are. And probably more importantly, we don't understand and there is no good evidence, and we need to look at more good evidence, on what the influences specifically of puberty blockers are on brain development as well as body development. But I work on the brain.

So I have a complex position in respect to this. I don't think it's a good idea to ban treatment in a blanket way. But I also think that people who are transsexual deserve both the best healthcare and also deserve the best information

about that healthcare and the implications of that healthcare. And at the moment, I don't think that's happening.

Q What do you mean by transsexual?

A So transsexual, transgender. I'm sorry. Error of language. What would have once been called transsexual is now more commonly called transgender. But people who experience gender dysphoria.

Q Okay. I'm going to pause for one moment and just play with my microphone. I'm having a little bit of a hard time hearing you.

(Off the record.)

BY MR. SHAW:

Q I want to ask my question again, because I don't believe I got a clear answer.

A Uh-huh.

Q Do you support the rule issued by Florida Medicaid?

A I don't have a clear answer to give you. I think that it is a mistake to have blanket bans on medical issues generally. I think it should be something that's worked out in terms of a scientific and a medical approach. I also don't think it's a good idea the way that we're currently approaching trans healthcare in that we are not doing the

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mean about being able to give people information about what could happen to them when they embark on this kind of treatment course. There is very little evidence and it's not much of it of a very high quality. And there's almost nothing that we know about humans.

So I think at the moment, you could say that they are making a decision based on the evidence. But it's a lack of evidence and that's not a good position for anybody.

Q So if you had to vote yes or no on this ban, would you vote yes or no?

A I don't think I can give you a good answer to that. I don't -- I can understand why -- the aspect of why the ban has been put in place. I don't think it's a good way of approaching healthcare. I would probably abstain like a coward.

Q You expressed concern with this rule just now. Is that fair to say? Concern.

A I think that it's -- there are going to be people out there who would benefit from therapy around these issues. I mean this in the broadest sense. But I don't think necessarily the way that we're approaching it at the moment, which is often to seeing that there has to be a medicalized ruse is

a particularly good idea. This is what I mean about I think the healthcare options for people who are trans at the moment are not good. They're not evidence-based.

Q Do you think all the treatments that are mentioned in this rule are as you say "not good"?

A Well, I think in that they're all highly medicalized, I think this is a situation where we need a lot more evidence before we blithely put teenagers on a route that might take them to somewhere that might not be something that's consistent with how they feel in a few years' time, and we know that that can happen. Not for everybody. It will help some people, but at the moment we don't know who.

Q Does your opinion change if these decisions are made by not only the patients, but their family and their medical team?

A At the moment, no, it doesn't. There isn't -- there's not high quality evidence about the benefits of this or who would benefit from it. We do know that there is some evidence in favor of watchful waiting. But generally there is just a horrible dearth of good data in this whole area, which means that the clinicians are operating

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This is science in humans.

Q Is your concern -- we talked about your concern with these treatments. Is that limited to adolescence?

A I mean, I think so. The situation with the adolescent brain, it's such a continuing age of importance in terms of brain development that it really should be taken seriously both in what the possible implications of specifically delaying puberty could have on that. We don't have good evidence about this. And I'm -- what evidence we do have suggests that there are effects on the brain of delaying puberty. And we don't know what that might mean further down the line. We just don't know. There aren't follow-up studies. Even the studies that have looked at this in animals haven't then look at the life-long profile for those animals.

So it is a particular area of concern for me, because I'm a brain scientist. I mean, I'm sure people could have legitimate questions about the whole rest of the body. But I care about brains and brains are still developing at that age. I think once somebody's an adult, their body is their own. They should absolutely have autonomy to do whatever they want.

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focused around techniques like functional magnetic resonance imaging and magnetic encephalogram. So looking at electrical distributions in the brain. And we involve both clinical researchers and basic scientists.

Q Has anyone at the Institute ever conducted any clinical studies related to gender dysphoria?

A No, not that I'm aware of. There was up until three years ago, four years ago, there was Sara-Jayne Blakemore was at the ICN. She's now in Cambridge. And she was one of the people who was really investigating the teenage brain in a more general sense, but that's the closest.

Q She was investigating the teenage brain in a gender dysphoria context or just generally?

A In a general sense she was looking at brain development in the teenage years. Yes.

Q So you've never conducted any clinical studies yourself related to gender dysphoria?

A No.

Q What about the affects of gender affirming care?

A Nope. And as I say, I'm not aware of many people actually doing studies on this.

Q Has the Institute ever studied the affects

of puberty blockers?

A No.

Q And just to be clear, you understand what I mean by puberty blockers?

A Yeah.

Q Yeah.

A Suppression of hormones. Yes.

Q You said -- you mentioned Ms. Blakemore was studying the teenage brain.

A Yeah.

Q Does anyone else study teenage brain development at the Institute?

A The only people that are still -- Sara still has some staff working there finishing up grants, that's it.

Q Have you ever studied teenage brain development?

A Yes. I did a study a couple -- probably more than a couple of years, but five years ago with Essi Viding and Eamon McCrory where we were looking at teenage boys, and we were looking at teenage boys at risk for psychopathy. And we were comparing across teenage boys at risk for psychopathy and teenage boys who were neurotypical, and looking at their perception of emotional vocalizations and

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somebody. And we had another group of boys who had conduct disorders, but were low in callous and unemotional traits. And that's an important contrast, because they're not well behaved these boys, but they feel bad if they do something wrong. And it's the boys with this two-fold profile, their conduct disorders and high in callous and unemotional traits, they're the ones at risk of psychopathy. And psychopathy in adulthood is associated with unpleasant and uncaring behavior towards other people.

Q Did any part of that study inform the opinion you gave today?

A Only in the most general sense that the teenage brain is changing. I mean, even in that study, we couldn't conclude if the differences we saw were because of something innately different about boys at risk for psychopathy or because of the experiences they had had as they were growing up. And that's -- and that's a problem you keep coming back to in this literature. You may -- even if you find a difference, how you interpret that difference, what's driven that difference can be very hard to determine. So only very generally.

Q Do you treat patients?

A No. I'm not a medical doctor.
Q Any medical training?
A No. I've worked a lot with patients, but that's been just in basic studies.
Q Are you a psychologist?
A Yes. My training is in biology and psychology, and my Ph.D. is in cognitive science.
Q So are you sort of certified as a psychologist?
A We don't really have that system in the U.K.. If you want to practice, you can join the Healthcare Professionals Association. But I'm not -- I'm not a clinical psychologist. I'm not clinically qualified. I'm a basic scientist. So I'm not affiliated with any professional organization other than the British Psychological Society, which is just most to belong to.
Q Have you ever had a clinical practice in any way?
A No. So I've worked with patients, normally patients with strokes. Some work with psychopathy and dyslexia in children and teenagers. But really all of that, all of it was just basic science. It wasn't -- it wasn't in a clinical study. It wasn't a study of treatment. It

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neurologist?

A Neurologist is somebody who's medically qualified, and then they specialize in diseases of the brain and the nervous system. And they see patients. They work clinically. They prescribe drugs. A neuroscientist is somebody who studies brains. He would probably work with neurologists. But he's studying it in a purely basic science position. They're not treating people. They're not prescribing things.

Q Are neuroscientists qualified to advise on gender affirming care?

A I think in terms of their understanding of how the brain develops, yes. I think we are not at a stage where there's good cognitive neuroscience on the brains of people who are transgender. There are some studies, but it's not -- what they are -- I didn't put this in my report, and I'm answering it because you asked me about it.

What those studies are showing is that what you start to pick up is a difference in the brain that's associated with the dysphoria. I think there was a belief that somehow people with trans -- who are transgender would have the brain of the opposite gender and that's not what you find. So

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MR. BEATO: Object to form. Dr. Scott, you can answer.

A I don't think so yet. Because as I say, the brain scans, that seems to be telling us something about who -- a difference in the brain if somebody is experiencing dysphoria from a neurotypical individual. That's still we're looking at two patterns of activation, but we're not saying two different populations and we could categorize people one way or the other. So it's not telling you about categories and it's also not telling you anything about how that profile might change. If other things were affecting the gender dysphoria, would that resolve in a different way. So it's not going to be good evidence across for predicting for what I suspect.

Q So just sort of to recap a little bit on you. You're the director of -- you're the Director of the Institute on Cognitive Neuroscience. You are a neuroscientist, correct?

A Yeah.

Q You do not treat patients?

A Nope.

Q You have no medical training?

A Nope.

[PAGE BREAK]

Q I just skipped a bunch of pages. Do you recognize what's on the screen?
A Yeah.
Q What is this?
A They are some of my refereed articles.
Q I'm going to scroll down. Are these all the articles that you list on your CV?
A Yeah.
Q I looked -- why is six missing?
A I've got no idea. I think probably because I've copied and pasted.
Q Okay. Just a typo. Is there --
A Almost certainly what I've done is I've copied and pasted the text in from PubMed or something like that to get the reference, and that normally puts in a few extra returns. And they need to be deleted to get the numbers to go right, and I've done that incorrectly. Sorry.
Q Do you recall any publications that you wrote that are missing from this list, publications that you wrote around this time?
A Well, it's difficult to say without having another CV in front of me. But I can't -- I can't see anything obviously. No.
Q Okay. All of these publications are about

speech, laughter and sound. Isn't that right?

A There are a few other things. But yeah, that's the majority. That is my main area of research.

Q Are any of them about gender affirming care?

A No.

Q Are any of these publications specific to gender dysphoria?

A No.

Q Any about puberty blockers?

A No.

Q Any publications on how pharmaceutical drugs interact with the brain?

A If you go back -- I'm trying to remember. We did do some work with methamphetamine and people who had strokes. I'm trying to remember if there was a study on that. If there was, it was a while ago.

Q How long are you -- how long ago? Sorry.

A Twenty years ago. And I now can't remember if there was a published paper from it, so...

Q All right.

A Let me think if there's anything else.

[PAGE BREAK]

Q And what did Mr. Conrathe say?
A He contacted me and some of the other people who -- like I said before, there's a case in Florida, would you be interested in making a similar kind of report to them writing about the same sort of issues.
Q Why did you think that you had an opinion to give in this case?
A Because I provided an opinion before for the Keira Bell case. And I discussed that a lot with Paul Conrathe at the time for all the reasons you said. I'm not a clinician. I haven't worked in this area. And all the people that have -- do work in that area were not prepared to make a comment about issues around consent and issues about puberty blockers. And I did some reading into the literature, and I was concerned enough that I thought somebody needs to, so I did.
Q You mentioned the Keira Bell case.
A Yeah.
Q What was your role in that case?
A I provided very similar testimony about the particular issues of consent for Keira Bell, because that was her particular claim at the judicial review. But also in the course of writing

that, it became very clear to me that, you know, the potential role for gender affirming drugs, puberty blockers or cross-sex hormones during adolescence could really be significant, and that informed my comment.

Q So you formed your opinion about puberty blockers in adolescents while you were working on the Bell case?

A Yeah.

Q Did you submit a written report in that case?

A Yes.

Q Do you recall the date?

A No. I don't recall the date. It would have been -- it was probably over the summer or towards the end of the summer in 2020.

Q Would you be willing to provide us a copy of --

A I'm not certain if I can. Can I chat with Paul Conrathe?

Q Sure. Why would he --

MR. BEATO: I think there may be some confidentiality issues with that in that case, Dr. Scott.

A Yeah. I would need -- the last time

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brain development. But that's not necessarily normalizing anything. That could simply be changing the brain in a way that might be negative.

Q But that's not necessarily harming anything?

MR. BEATO: Object to form. You can answer, Dr. Scott.

A But it's not very good evidence that it's safe, particularly if -- and I remind you of this. The original claim for puberty blockers is that they are just pressing pause, and they're clearly not just pressing pause. They are changing brain structure and they're changing behavior.

Q But you can't say here --

A Sorry. Go on.

Q But you can't say here that these puberty blockers have any harmful effects on the brain?

MR. BEATO: Object to form. But you can answer, Dr. Scott.

A But we know that they change the brain and we don't know that that's not harmful. And that's -- I think that's the critical point. We know that in the -- again, this came up in one of the rebuttals. A different person said, well, wouldn't -- if delayed puberty is problematic for

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blockers are not doing anything bad for the brain, even if you said that, you have to accept that the puberty blockers are going to be changing the effects of testosterone and estrogen in the brain, which at a bare minimum affect the parts of the brain that show sexual differentiation in males and females.

And the evidence and the literature that we have from these papers that I've reviewed more recently indicates that that's already happening before puberty. Even prior to puberty if you give a puberty blocker, something that's cutting off all production of estrogen and testosterone to a female monkey, that will already start to affect her brain structure.

So they're suggesting as they do in the paper there's an effect of estrogen on the whole brain, the whole brain volume before puberty starts.

MR. BEATO: Counsel, I apologize. I just have a quick question. We've been going for about an hour and 30 minutes. Do you think we can work in a five-minute break, or if you have some follow-up questions that relate to what Dr. Scott is saying. We can take a five-minute break a little bit later on.

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the amount of time they've been on the drugs with the things to see if there's an effect of that. But in terms of long-term, I am not certain about that.

Q Is there a study --

A -- looking at cognitive things.

Q So this study does not determine whether there are any long-term effects of puberty blockers for precocious puberty?

A No. Because it's still -- you know, the girls are still young when they're being tested.

Q And yet we still prescribe puberty blockers to treat precocious puberty?

A Yes, as far as I'm aware. Although, in this paper they say it's not completely clear that it's totally not affecting girls, if you see what I mean. Most of the girls are very similar, but there were some points in which they looked different.

Q Why should we be concerned about prescribing puberty blockers to treat gender dysphoria based on a lack of knowledge when we had the same lack of knowledge for precocious puberty?

A I don't think that's necessarily a reason to do it in precocious puberty. I think that the -- I have some concerns about the data in precocious puberty. I don't think that's trouble free. So the

girls -- the normally developing girls, there was a seven point difference in IQ between them and the girls with precocious puberty, which means that some of the girls in precocious puberty had even lower IQs. And that's -- this is not my words. Somebody else wrote a commentary on this saying that's not nothing. There's a small group. It's enough to make you slightly worried.

So I think probably the argument that the medics would make with precocious puberty is that if you're just going to do this for a short amount of time, get them across the right age, you know, a year or two for Tanner Stage 2 to be okay, and then off you go. But I don't think it necessarily means that it is safe.

Q Are your --

A The cognitive function, the data is not 100 percent clear that it doesn't have an effect.

Q Are your concerns with puberty blockers for precocious puberty shared by the medical community?

A I don't know. They are certainly shared by Dr. Hay who wrote that commentary that I cited. But that's, you know -- I suspect that precocious puberty, because their sights on things like height

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aspect of human behavior that is determined by just one factor. That's why things like advertising exists, the marketing, you know, because humans are complicated. You can't just simply manipulate them into doing what you want them to do or find what they like simply. So that's just a general truth about doing psychology. It's always a -- you always end up with complex patterns of things that influence behavior.

Q So you're saying every brain is different?

A Yes. Because the thing that brains change hugely in development over your lifespan, but they also change massively based on your experience. And that means that, you know, if I was to clone you tomorrow, I would still have somebody who even if they had the exact same brain as you would grow up to be different, because they would grow up in a different world. They would grow up in a different environment and their brain would not be exactly the same as yours.

So you got this kind of fascinating, but sort of extremely complex continual interaction between the brain that you have. And remember, you're born with 86 billion braincells. And you have those braincells all your life. You don't grow

[PAGE BREAK]

BY MR. SHAW:

Q I'm going to go back to your report. I want to talk about starting at paragraph nine.

A Yeah.

Q Paragraphs 9 through 14. I can show you all of them if you want. You talk about how the brain develops.

A Yeah.

Q Is it a fair characterization of your report to say that you believe teenager's brains are prone to more risky behavior during that time?

A Yes. There's a recent review. It came out too recently for my report, I think, but in the nature of neuroscience, which described risk-taking behavior in adolescents is a defining feature of the decision-making.

Q Is that common for all adolescents?

A Well, I mean adolescence is very variable like all humans. We're talking about population level here. So yes, there's a reason why in law we try and protect adolescents from the things that could have livelong consequences for them, like what age they get married and what age they have tattoos and what age they get in a car. So that's kind of -- you know, all human cultures recognize to some

degree this difference. And we don't always deal with it well, but it's definitely a brain that's still in development.

Q Would you say that you're an expert in adolescent behavior?

A I'm only somebody that works in brains. I don't -- I have been involved in some studies on this and it's literature I know about. I do not -- this is not in any way my main area of research. But as I say, it's a mistake to assume that because somebody doesn't research something, they don't therefore understand it. As I said, I did write a book on this last year, brains in general.

Q Is it fair to assume -- is it fair to say that -- let me rephrase.

Is it fair to say that because someone does not investigate this area specifically, that they're not an expert in the area?

A Some area of specific expertise. But I think anybody that can -- I can read and understand and interpret and draw across the papers. So my expertise in neuroscience is what I'm speaking to here more generally.

Q Can a nonexpert review the papers?

A I'm absolutely certain they can do.

Q Can they understand them?

A I think some of it gets -- not necessarily understanding is going to be the problem, but interpreting what that could mean and what the bigger implications are might just be easier for somebody who's more familiar with the wider field.

Q Do you need to be an expert in neuroscience to understand that teenagers on the whole engage in risky behavior?

A No. Like I said in my report, it's something that all cultures recognize. It's interesting how long it took science to start asking questions about it. For a long time, developmental psychology meant sort of not to tend. But actually now people think about, as I said, the human brain is a work in progress. I've studied developing brain. I've studied aging brains. Your brain is always changing.

So it's definitely something that's become of more interest. But it's always been I think, you know, as long as can you find history for humans, you find humans complaining about teenagers.

Q How is this increased level of riskiness relevant to your other opinions?

Let me back up. Excuse me. If I may.

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could come back to have implications for you that is more likely for teenagers to engage with. So the hot/cold is important, but it's not capturing all aspects of risk.

Q But we're not talking about teenagers deciding about gender affirming care themselves in this case, right?

A No. I understand that this would be something where the consent is not with the teenager.

Q Say that again. Consent is --

A Consent is being made by somebody else. The informed consent is being made by somebody else. I think one would assume that they're still part of the discussion, and that this isn't being visited on them in an unwilling way. And I think that the point still stands about being able to fully comprehend the implications of factors that could have big influences further down the line.

Q Do you think adults can help teenagers understand fully the implications of gender affirming care?

A I think if those adults had really good information about what the implications are, then yes, I think they could do. But we're back to the

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sometimes larger brain areas. What that translates into we don't know. But smaller brain areas are rarely good.

Q So would you say that your opinion -- would you agree with me that your concern is limited -- let me rephrase.

Would you say that your concern is limited to puberty blockers or all forms of gender affirming care?

A I think the immediate proximal concern has to be puberty blockers given these effects in the published literature. From the brains eye view, this is not a pause button and that concerns me. I have other thoughts about -- that's not true. Anything else I said would be much more of an opinion, and it's harder -- it's not impossible, but because puberty blockers -- no, that's not true. No. I think it's probably fair to say that. I think it's fair to say that my primary concern is about puberty blockers and giving them in adolescents and the risk associated with that.

Q So you're not giving an opinion about adults?

A No.

Q You would agree that a complete

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at the bottom of the screen?

A It's very small. Could you make it bigger, please?

Q Sure can.

A Thanks. Yeah.

Q You said, "The book says that girls who like bugs and wear super hero" -- I think you meant capes there, "and who don't like pink dresses are in fact boys. I think that's your cheap shot right there."

A Yeah.

Q What was your problem with the book?

A Because I was a little girl who dressed like a super hero and collected water beetles, and I was never a boy. So I think it seems reductive to assume that a girl who likes boyish things can't in fact be a girl.

Q Is that really what the book says?

A It's the story of the book, isn't it? She doesn't like girlish things. She wants to be called a boy's name. And then at the end, hooray, she's a boy.

Q Well, the book starts with the premise that it's a trans boy.

A Yes.

Q Right?
A What's a trans boy if that's not a girl?
Q So that it's not about her liking bugs and capes.
A Yeah.
Q It's that she's a trans -- or that he's a trans boy.
A So a girl who likes bugs and capes has to be a trans boy?
Q No. Anyone can like bugs and capes.
A Yeah.
Q The book is about addressing that issue with your family. You didn't read the book?
A Well, that was -- I've just quoted off the bits I saw. This is -- you've asked me why I said it and that's why I said it.
Q Is a trans boy a girl?
A A trans boy is a natal female. If they weren't a natal female, they would just be a boy.
Q That person cannot consider themselves to be a boy?
A Yeah. Absolutely they can consider themselves to be a boy. Totally.
Q So they're a boy, right?
A They're a trans boy. Yeah. Yeah.

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seeing this, what's on the screen?

A What year was this?

Q This is -- I'll bring up your tweet. The reason I'm doing this like this is because the pictures don't show up on the tweet.

A Right.

Q So this is where that picture was.

A Okay.

Q And you tweeted, "Oh, God."

A Yeah.

Q What did you mean by "oh, God"?

A Because I'm guessing -- this is six years ago. There was a shift in people going from asking if you're male or female and for protecting characteristics like male and female being used as terminology to what you identify as. And that is something that has continued and has got no better. And my concern at that time was that if you move something from being specifically for females and make it for anybody who identifies as a female, there is the possibility for -- well, first of all, you're making this less available to do the things you're supposed to do, which is increase applications from females. And also you are at least opening up the possibility for somebody to

self-identify in a way that is trying to take advantage of the situation. I think if you want to make things better and more inclusive for trans people, what you do is you do specific stuff for trans people. That's why I've said that.

Q Doesn't that section them off from everyone?

A Yes. But that's the nature of doing positive discrimination, isn't it? You do specific things for specific groups of people.

Q So you don't think that trans women should be allowed to apply for the same scholarships as cis women?

A If by cis women you mean natal females, then it would depend on what it's for and what they're trying to correct by having the -- if you're in a situation where there aren't enough natal females and you're trying to boost it with scholarships, then probably no. You probably should keep it for natal females. If it's a situation where you are worried about trans representation, you would be entirely appropriate, and certainly the equality is lower in the U.K., would let you have a much more specific group for trans people and to specifically benefit them.

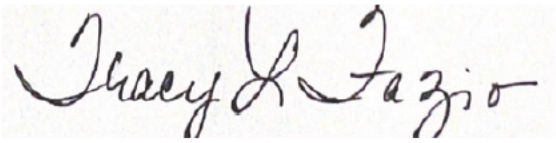
[PAGE BREAK]

C E R T I F I C A T E

THE STATE OF FLORIDA)
COUNTY OF PALM BEACH)

I, the undersigned authority, certify that SOPHIE SCOTT, Ph.D. appeared before me and was duly sworn.

WITNESS my hand and official seal this 23rd day of March, 2023.



Tracy Lyn Fazio, FPR
Notary Public - State of Florida
Commission No. HH 089243
Expires: 02/25/2025

EXHIBIT C

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF FLORIDA
Tallahassee Division

AUGUST DEKKER, et al., *Plaintiffs*, v. JASON WEIDA, et al., *Defendants*.	Case No. 4:22-cv-00325-RH-MAF

CORRECTED EXPERT REBUTTAL REPORT OF E. KALE EDMISTON, PH.D.

I, E. Kale Edmiston, Ph.D., hereby declare and state as follows:

1. I am over the age of eighteen and submit this expert rebuttal report based on my expert opinion.

2. I have been retained by counsel for plaintiffs as an expert in connection with the above referenced litigation. The opinions expressed herein are my own and do not express the views or opinions of my employer.

3. I have actual knowledge of the matters stated herein. If called to testify, I would testify truthfully based on my expert opinion.

Background and Qualifications

4. I am an Associate Professor of Psychiatry at the University of Massachusetts Chan Medical School. Prior to this appointment, I was an Assistant

Professor of Psychiatry at the University of Pittsburgh from 2019 to 2022. I have more than 15 years of experience conducting psychiatric neuroimaging research, with a focus on adolescence and young adulthood, mood and anxiety disorders, and impulsivity and emotional regulation. My methodological expertise lies in neuropsychological assessment, multimodal neuroimaging, psychophysiological measures such as heart rate variability, and measures of neuroendocrine function across adolescent development.

5. I completed a bachelor's degree from Hampshire College in 2007, where I studied cognitive science. I received postbaccalaureate training in psychiatric neuroimaging at the Yale School of Medicine. I earned a PhD in neuroscience from Vanderbilt University in 2015, as well as a graduate certificate in medical humanities, with a focus on bioethics and medical decision-making. I then completed post-doctoral training at China Medical University and the University of Pittsburgh.

6. In 2014, I co-founded the Trans Buddy Program at Vanderbilt University Medical Center, a peer navigator and support program for transgender people seeking healthcare. As a part of this program, my work primarily focused on supporting transgender adolescents experiencing mental health crisis. At this time, I also served as the Co-Director for the Vanderbilt University Program for LGBTI

Health. I later replicated the Trans Buddy Program at the University of Pittsburgh Department of Adolescent Medicine.

7. From 2018-2022, I served as a chapter author for the Assessment chapter of the World Professional Association for Transgender Health's *Standards of Care for the Health of Transgender and Gender Diverse People, Version 8*.

8. I have authored over 100 peer-reviewed manuscripts, book chapters, and conference proceedings in psychiatric neuroscience and transgender health.

9. Further information about my professional background and experience is outlined in my curriculum vitae, a true and accurate copy of which is attached as **Exhibit A** to this report.

Prior Testimony

10. I have not testified as an expert at trial or by deposition within the last four years.

Compensation

11. I am being compensated for my time at a rate of $175/hour. My compensation is in no way contingent on the conclusions reached as a part of my testimony or on the outcome of this case.

Basis for Opinions

12. In preparing this report, I have reviewed: the Complaint in this case; Florida Administrative Code 59G-1.050(7) (the "Challenged Exclusion"); the document titled "Florida Medicaid: Generally Accepted Professional Medical Standards Determination on the Treatment of Gender Dysphoria," published by the Florida Agency for Health Care Administration in June 2022, and its attachments; the expert reports of Drs. Armand Antommaria, Dan Karasic, Johanna Olson-Kennedy, Loren Schechter, and Dr. Daniel Shumer, submitted by plaintiffs; and the expert reports Drs. Michael Biggs, G. Kevin Donovan, Paul Hruz, Kristopher Kaliebe Michael Laidlaw, Patrick Lappert, Stephen Levine, Sophie Scott, and Joseph Zanga, submitted by defendants.

13. My opinions are based on my years of research and academic experience, as well as my professional knowledge, as set out in my curriculum vitae (**Exhibit A**) and the materials listed therein; my knowledge of the peer-reviewed literature relating to neuropsychological assessment and brain development; my knowledge of the clinical practice guidelines for the treatment of gender dysphoria, including my work as a contributing author of WPATH SOC 8; and my review of any of the materials cited herein.

14. I have also reviewed the materials listed in the bibliography attached as **Exhibit B**. I may rely on those documents as additional support for my opinions.

15. The materials I have relied upon in preparing this report are the same types of materials that experts in my field of study regularly rely upon when forming opinions on the subject. 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.

Adolescent Brain Development

16. Dr. Scott's report stating that adolescents are more likely to engage in risky behaviors relative to adults fails to include the specific context in which this is true. That is, the literature indicates that there are *highly specific circumstances* in which adolescents are more likely to engage in risky or impulsive behavior. Indeed, Dr. Scott lists some of these circumstances in her testimony: driving, drinking alcohol, getting a tattoo. However, none of these examples are relevant to the issue at hand: protracted medical decision-making made in the context of adult guidance and consultation with a medical professional.

17. Dr. Scott fails to cite the large body of evidence indicating that adolescents are capable of deliberative decision making in the presence of adults (i.e., healthcare providers and caregivers) and when decision making occurs over a protracted period. This is the exact context in question: decisions about accessing gender-affirming medical care, such as gonadotropin releasing hormone agonists

(GnRHa) and hormone treatment, are made jointly among the adolescent patient, their caregiver(s), and medical professionals. These decisions are also made over time; data show that the typical time between an adolescent realizing they are transgender and coming out to an adult is three years (Bauer et al., 2022). Furthermore, once an adolescent discloses their identity to a supportive adult, they will then have to schedule a healthcare appointment and undergo assessment prior to accessing treatment. This process typically takes months and for some, even years.

18. Dr. Scott misrepresents the literature on adolescent decision making by generalizing findings made in "hot" contexts to those made in "cold" contexts. Indeed, the Blakemore and Robbins review from 2012 that she cites explicitly states that the literature concludes that adolescents demonstrate adult-typical decision-making abilities in cold contexts. It is not that adolescence is associated with a failure to engage cognitive control networks, but rather, that cognitive control networks are engaged with greater variability during this time than during adulthood. Decision-making is a multifactorial process that includes valuation of both risk and reward. While adolescents are more likely to overvalue reward and underestimate risk when peers are present or when decisions must be made quickly, they demonstrate deliberative and appropriate consideration of reward and risk valuation in the absence of peers, in the presence of adults, and when decisions are made over time.

This important difference in the contextual nature of decision-making in adolescence is an established finding that has been replicated across multiple studies (Chein et al., 2011; O'Brien et al., 2011; Simons-Morton et al., 2011; Smith et al., 2014; Weigard et al., 2014; Hartley & Somerville, 2015; Guassi Moreira & Telzer, 2018). Indeed, deliberative decision making in contexts without pressure to decide quickly has been repeatedly shown in adolescents (Byrnes, 2002; Figner et al., 2009; Wolff & Crockett, 2011; Icenogle & Cauffman, 2021).

19. Dr. Scott also states that "at 18yrs old, the connections to the frontal lobes are not myelinated[1] like a mature adult brain, and this is likely to affect frontal lobe functions." This is an oversimplification of an extremely complex literature. A study of over 10,000 participants has shown quite the opposite: that by the age of 18, adult-level cognition is established (Tervo-Clemmens et al., 2022), while other studies have shown mature integration of functional networks by late adolescence (Marek et al., 2015) and fractional anisotropy of prefrontal white matter (Lebel & Beaulieu, 2011, fractional anisotropy is an indirect measure of myelination). Even though, on average, there are developmental differences in prefrontal myelination,

[1] Myelin is a protein sheath that covers the axons of neurons. The axons comprise white matter in the brain, and bundles of these fibers transmit signals from region to region in the brain. When an axon is myelinated, the signal can travel faster down the axon.

there is not strong evidence that these differences are associated with an inability to make deliberative decisions with the support of caregivers and expert clinical guidance.

20. Furthermore, there is a great deal of variation in the timing of development between different prefrontal white matter tracts, as well as a great deal of variation between individuals. Indeed, in Lebel & Beaulieu's longitudinal study of over 100 individuals from childhood to young adulthood, many individuals showed decreases or no changes in fractional anisotropy (FA) during adolescence, and these differences also varied by prefrontal white matter tract (2011). This literature represents differences in group averages and should not be used to predict the behavior or development of an individual adolescent; we cannot draw conclusions about all 18-year-olds from these studies. This is why the WPATH SOC 8 recommends an individualized approach to joint decision-making regarding healthcare.

There is Little Evidence to Support Defendants' Designated Experts' Speculation about Negative Effects of GnRHas on Cognition

21. Dr. Scott cites a 2016 study by Wojniusz and colleagues as evidence of the negative effects of GnRHas on emotional reactivity in a sample of girls with central precocious puberty. This is puzzling because the authors of this paper explicitly state the opposite interpretation: "Overall, our findings do not provide firm

conclusions with regard to differences in emotional processing between the GnRHa treated CPP girls and age-matched controls." (pp13).

22. Perhaps Dr. Scott has misinterpreted the nature of the emotional flanker task. This task asks participants to determine if two simultaneously presented houses are the same or different. The houses are presented at the center of a screen, and emotional or neutral face distractors flank them. The outcome of interest is the reaction time for the determination of whether the houses are the same or different. The idea here is that people with poor emotional regulation will be more distracted during the emotional face condition and therefore take longer to respond. This interpretation can only be made when reaction times are increased in both the emotional face conditions. In this study, the CPP girls showed longer reaction time than controls during the emotional face condition only when the houses were different, but not when the houses were the same. Thus, the findings do not indicate an issue with emotional regulation. More likely, the results are incidental and due to statistical issues regarding false discovery rate correction, an argument that the authors of the paper themselves make.

23. The authors do find reduced heart rate and elevated heart rate variability (HRV) during the emotional task. HRV is distinct from heart rate and is a measure of cardiac vagal tone. HRV is a proxy for parasympathetic system or "rest and

digest" function. Thus, elevated HRV is associated with increased regulatory capacity and is a marker of health. Thus, these findings are a sign of *optimal* emotional regulation. Indeed, the authors state, "…the lower HR and higher HRV could suggest that treated CPP girls have better emotion regulation capacity and higher adaptability to changing contexts than controls" (pp13).

24. Dr. Scott then points out that, in a separate commentary on the article, Dr. Hayes states that there were "notably" lower scores on IQ measures in the CPP group relative to controls. However, Dr. Hayes's comment, and Dr. Scott's reliance on it, is not supported by the findings of the study. Specifically, none of the differences in IQ were statistically significant, and the mean IQ scores for both groups were within the normal range. Furthermore, the mean difference between groups in this study is within the realm of variation that may occur from repeated administration of the WISC-III, i.e., although scores for an individual tend to remain relatively stable over time, there is fluctuation that occurs even within an individual and small differences in IQ (Watkins & Smith, 2013), as reported in this study, are not only not statistically significant, they are not clinically significant. Dr. Scott has, again, offered a misrepresentation of the literature.

25. Dr. Levine cites a single case study as evidence for an effect of GnRHa treatment on IQ. Case studies are the lowest quality of evidence. Case studies can

provide important evidence for future areas of study or to provide an illustrative example of a common clinical phenomenon, but they should not be used to make general conclusions or policy positions. Putting aside the low quality of evidence typical of case studies in general, this case study does not even provide sufficient support for Dr. Levine's opinion as it describes a transgender girl who, prior to initiation of treatment, already had below average IQ. While Dr. Levine highlights the lack of change in fractional anisotropy values over the course of the study in this case, this could be due to developmental delays that are independent of treatment and are instead related to her low IQ. Therefore, the findings of this case study are simply not generalizable to the broader population.

26. Dr. Michael Biggs, a sociologist, also offers speculation regarding cognitive effects of GnRHa treatment as well, describing it as "…stopping normal sexual and cognitive development…" This statement regarding cognitive development appears to be pure speculation as he offers no citation regarding evidence for deleterious effects of GnRHa treatment on cognition. In reviewing the literature, including through specific searches, I have been unable to find compelling evidence of this. I was able to identify two studies that showed no effect of GnRHa treatment on executive function (Soleman et al., 2016; Staphorsius et al., 2015). The

lack of evidence for these effects is itself compelling, given that these medications have been used in adolescents with central precocious puberty for decades.

Evidence for Effects of GnRHa treatment on the Brain

27. Both Dr. Levine and Dr. Laidlaw state that the effects of GnRHa treatment on the brain are both "unknown" and "likely negative." They do not cite any original research that supports this conclusion and thus it is unclear to me how they concluded that the effects are likely negative in the absence of evidence. Dr. Laidlaw even goes so far as to speculate on the individual brain maturation of three specific transgender individuals. Both Levine and Laidlaw admit that there is no evidence from the neuroimaging literature on negative effects of these treatments on brain development, but even if there was, any neuroimaging study that compares group averages would not support an inference about the brains of individual people. There is a great deal of variation between and within individuals in many commonly used neuroimaging measures. For this reason, neuroimaging methods commonly used in research, such as fMRI, cannot be used diagnostically for individual people in the absence of organic brain disease (Schleim & Roise, 2019).

28. Dr. Hruz also speculates in his testimony that there are negative effects of GnRHa treatment on the brain: "A possible effect of blocking normally timed puberty is alteration of normal adolescent brain maturation". Dr. Hruz then cites a

2013 review paper that describes typical adolescent brain maturation but does not mention or describe any effects of blocking or delaying puberty on the brain (Arain et al., 2013). Dr. Hruz therefore has not cited any support for his conclusion, and I have not identified any studies relating to the evidence of negative longitudinal effects on brain development related to GnRHa treatment in central precocious puberty or in transgender adolescents, even after targeted searches for it.

29. There is not a large literature on the effects of GnRHa treatment on the brain in humans, but this does not render such care experimental. GnRHa treatments have been in used for decades, including for the treatment of gender dysphoria. That said, there are a few cross-sectional studies on this issue, and it is significant that none of the experts (nor the GAPMS memo) cited this literature in their testimonies. In a study that compared transgender adolescent boys and girls taking GnRH agonists to cisgender boys and girls, there were differences in brain function in some brain regions that would indicate congruence with gender identity and other differences that would indicate congruence with sex assigned at birth. However, there were no between-group differences in network function on the basis of GnRHa treatment. Furthermore, the authors searched for relationships between duration of GnRHa treatment in the transgender adolescents and brain function and *were unable to find any effects.* In a diffusion tensor imaging study of fractional anisotropy

values, an index of white matter myelination, *again there was no significant association between fractional anisotropy values and GnRHa treatment* (van Heesewijk et al., 2022). Similarly, in an fMRI study comparing cisgender boys and girls to transgender boys and girls, there were no significant differences in brain activity between transgender and cisgender adolescents during a verbal fluency task, and no deficits in verbal ability in transgender youth (Soleman et al., 2013). In a study of transgender individuals receiving GnRHa treatment and cisgender people, there were differences in brain activity between groups, but these differences were not associated with hormone levels, leading the authors to conclude that these differences are associated with group differences that predate GnRHa treatment (Soleman et al., 2016). In summary, to my knowledge, there have been three studies of brain structure and function of transgender adolescents receiving GnRHa treatment, and none of them have found any significant effects of treatment on the brain.

30. A recent primate study provides evidence for some regional neuroprotective effects of GnRHa treatment, although the results are complex (Godfrey et al., 2023). In this study, the authors compared dominant and subordinate adolescent rhesus monkeys. These monkeys form social hierarchies much like human adolescents, and subordinate monkeys are subjected to aggression from the

more dominant monkeys. Both dominant and subordinate monkeys were randomly assigned to a GnRHa treatment or control group and then followed longitudinally. In the primates exposed to chronic social subordination stress, GnRHa treatment rescued the negative effect of stress on regional brain volume over time. These differences were seen in brain regions such as the amygdala that are well-established in the pathophysiology of depression and anxiety. There were also effects of GnRHa treatment in general; treatment in both social groups was associated with smaller hippocampal volume than control animals. Regarding the prefrontal cortex, a critical region during adolescent development, GnRHa treatment was associated with greater prefrontal grey matter volume prepubertally but this difference decreased by adolescence. There was an effect of GnRHa treatment early in puberty on prefrontal white matter volume; however, this difference was no longer present by the end of the study. Importantly, there are species-specific differences in prefrontal volume changes across puberty; the generalizability of the prefrontal findings to humans should be made with caution. Finally, the authors also assessed social behavior in both submissive and dominant primates over time and were able to determine that, at prepuberty, submissive primates were more socially isolated, but that GnRHa-treated subordinate animals had normalized social behavior (reduced time spent alone) and normalized cortisol response to threat (cortisol is a stress hormone

associated with the hypothalamic pituitary adrenal axis). The authors conclude that "…delayed puberty and estrogen suppression may be protective against the impact of social stress" (pp12). This study provides strong evidence that GnRHa treatment normalizes brain structure, physiological stress reactivity, and social behavior in adolescent primates subjected to social subordination, an ecologically valid non-human primate model of the psychosocial environment for transgender youth.

31. There is a small body of literature on the effects of gender affirming hormone care on the brain in transgender adolescents. In a study comparing transgender boys receiving testosterone therapy and those who were not, testosterone treatment was associated with reductions in mood and anxiety symptoms, as well as reductions in body image dissatisfaction. Gender affirming hormone care was associated with an increase of functional coupling between the amygdala and prefrontal cortex while research participants viewed threatening emotional faces, likely indicating improved emotional regulation of the amygdala in the boys who were treated with testosterone. Indeed, in the boys who were treated with testosterone, greater coupling between these two regions was associated with lower anxiety symptom severity (Grannis et al., 2021). Another study of transgender boys receiving testosterone found that testosterone caused a shift in amygdala

activation, such that it became more typical of cisgender boys than cisgender girls (Beking et al., 2020).

32. 17. Both Dr. Scott and Dr. Biggs cite studies from the animal literature regarding the "side effects" of GnRHa treatment on the brain and behavior. However, they misinterpret or misrepresent the meaning of the term "side effect" in this context. Pharmacological agents have effects. The determination of what is a side effect and what is a desired effect is contextual. For example, Scott cites a 2021 rodent study of GnRHa treatment as an example of the "side effects" associated with GnRHa treatment (Anacker, et al., 2021). If one were to read the abstract of the study and not the full text, it may lead some to come to such a conclusion. However, what the study shows is that, prior to GnRHa treatment, there are sex differences in rodent behavior. Following GnRHa treatment, those sex differences are no longer present. This is the expected and desired outcome of GnRHa treatment, not a side effect. For example, female mice show greater locomotion behavior than male mice. Following GnRHa treatment, male mice show greater locomotion behavior than untreated male mice. Similarly, in a test of social interaction, GnRHa-treated males showed differences in the time spent with male versus female mice relative to untreated male mice, but not relative to untreated female mice. In both force-swim tests and a test of feeding behavior, female GnRHa-treated mice differed from control female mice,

but not from male mice. This is a consistent pattern across behavioral assays performed in the study, and this pattern was present in biological assays as well. GnRHa-treated male mice showed greater corticosterone stress response to novelty than control male mice but did not differ from female mice. GnRHa treatment increased neural activity in the hippocampus of female mice, but this activity increase did not differ from male mice. This is not a compelling study of the side effects of GnRHa treatment, but rather, a study that shows us exactly what we would expect: that blocking sex hormones decreases sex differences, the intended outcome for transgender youth.

33. Dr. Scott and Dr. Biggs cite a series of studies of GnRHa effects on sheep from a specific laboratory. One study from this group did show sex-specific changes in feeding behavior and HRV following GnRHa treatment. While Dr. Biggs opts to highlight changes in behavior in the female sheep that could be interpreted as an increase in anxiety-like behavior, he fails to mention that GnRHa treatment was associated with *improvements* in these behaviors in the treated male sheep (Wojniusz et al., 2011). They also fail to mention that other studies from this group show no effects of GnRHa treatment on cognition (Nuruddin et al., 2013; Wojniusz et al., 2013), and, like the Anacker study, brain differences are best explained by an expected reduction of sex differences following treatment (Nuruddin et al., 2013).

This issue of inappropriate reference group is a common problem in the GnRHa animal literature and its extrapolation to transgender youth (Edmiston & Juster, 2022). While the literature regarding the effects of GnRHa treatment on sheep behavior from this research group is complex, it by no means offers compelling evidence of negative effects of GnRHa treatment. Furthermore, Dr. Biggs highlights a negative effect from one study- an increase in anxiety-like behavior in female sheep only. However, we know from studies of transgender youth and young adults that anxiety and depression symptoms decrease with treatment (de Vries et al., 2014; Dhejne et al., 2016; Aldridge et al., 2021; Chen et al., 2023). This is more compelling evidence than a single animal study, as sheep do not have the complex psychosocial identities that humans do.

Evidence for Negative Consequences of Depression and Anxiety on the Developing Brain

34. The brain is more plastic during adolescence than during adulthood. This means that adolescents are particularly vulnerable and at increased risk for the onset of mood and anxiety disorders, and, if untreated, that the onset of mood and anxiety symptoms can permanently alter the developmental trajectory of the brain into adulthood (Holder & Blaustein, 2014). Termed the "kindling effect", the concept here is that, as the efficiency of neural circuits is reinforced over time (i.e., "neurons that fire together wire together"), each depressive episode or

environmental stressor increases the risk for later depressive episodes. This effect may be amplified during adolescence because of the greater plasticity of the brain.

35. There are well-documented disparities in mental health outcomes in transgender youth that are caused by minority stress (for review, see White Hughto et al., 2015). This includes evidence that transgender people who live in areas with more accepting political climates show reduced biological stress markers and fewer mental health symptoms than transgender people who live in less accepting areas (DuBois & Juster, 2022). Others have shown an association between decreased social support and biological markers of stress in transgender adolescents (McQuillan et al., 2021). Given that transgender adolescents report high chronic stress and high rates of depression, anxiety, and suicidality, transgender adolescents are particularly vulnerable to the effects of stress on brain development, stress system regulation, and long-term mental health outcomes (DuBois et al., 2021; Potter et al., 2021; Randall et al., 2022).

36. In Dr. Levine's testimony, he quotes the Hippocratic Oath, "Above All Do No Harm". He makes this argument on the assumption that GnRHa treatment must necessarily cause harm because it is an intervention. This assumes that the psychosocial environment and biology of transgender youth is like that of cisgender youth. There is a great deal of evidence that this is not the case. Instead, in my

opinion not offering an intervention to transgender individuals that require treatment actually does harm.

37. In this case, puberty blockers have demonstrated efficacy in reducing symptoms of depression in transgender adolescents (de Vries et al., 2011), and therefore may in fact be neuroprotective to the cumulative effects of stress caused by gender dysphoria.

Conclusion

38. There is little to support the Defendants' designated experts' speculation about the negative effects of GnRHa treatment on the brain. In contrast, there is a great deal of evidence supporting the mental health benefits of GnRHa treatment for transgender adolescents. Furthermore, it is well-known that transgender adolescents face higher rates of psychosocial stress than their cisgender peers, and there is clear evidence for the negative effects of psychosocial stress and poor mental health on brain development. While the effects of GnRHa treatment on the brain are an important area for future research, this does not render such care experimental. To the contrary, this is treatment that has existed for decades and arguments that a purported lack of evidence is equivalent to known harm are spurious, particularly when there is a large literature indicating benefits of treatment and harm of withholding treatment.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Executed this 22 day of March 2023.

E. Kale Edmiston, Ph.D.

Exhibit A

Curriculum Vitae

E. Kale Edmiston, PhD

Associate Professor
Department of Psychiatry
University of Massachusetts Chan Medical School
kale.edmiston@umassmed.edu

ACADEMIC APPOINTMENTS

Associate Professor of Psychiatry University of Massachusetts Chan Medical School	2022-present Worcester, MA
Assistant Professor of Psychiatry University of Pittsburgh School of Medicine	2019-2022 Pittsburgh, PA
Postdoctoral Scholar University of Pittsburgh Medical Center PI: Mary L. Phillips, MD, MD (CANTAB)	2016-2019 Pittsburgh, PA
Postdoctoral Fellow China Medical University PI: Fei Wang, MD, PhD	2016 Shenyang, China
Research Assistant Yale University School of Medicine PI: Hilary P. Blumberg, MD	2007-2010 New Haven, CT

EDUCATION

PhD, Neuroscience Vanderbilt University	2010-2015 Nashville, TN
Graduate Certificate Medicine, Health and Society Vanderbilt University	2015 Nashville, TN
BA, Cognitive Science Hampshire College	2005-2007 Amherst, MA

RESEARCH

CITATION METRICS (03/23):

Citations: 2087 H-Index: 25 i10 Index: 34

RESEARCH INTERESTS:

social and affective neuroscience, visual processing, anxiety disorders, multimodal MRI, neuromodulation

AWARDED GRANTS:

American Foundation for Suicide Prevention Award 2022

Title: *Real-time study of psychotherapy, suicide risk, and resilience in transgender and non-binary adults*
PI: Sarah Victor
Co-I: **E. Kale Edmiston**
Award amount: $90,000.00

K01 MH117290 Mentored Scientist Career Development Award 2019-2024
Title: *Feed forward visual system function in high trait anxiety*
PI: **E. Kale Edmiston**
Award amount: $868,978.00

Brain and Behavior Research Foundation Early Career Award 2019-2021
Title: *Neuromodulation of visual cortex BOLD in high trait anxiety*
PI: **E. Kale Edmiston**
Award amount: $69,401.00

The Opportunity Fund 2019
Title: *Trans Buddy PGH: Peer healthcare support program*
PI: Gerald Montano
Co-I: **E. Kale Edmiston**
Award amount: $15,000

Center for Interventional Psychiatry 2018
Title: *Neuromodulation of visual cortex and threat sensitivity in high anxiety*
PI: **E. Kale Edmiston**
Award amount: $9,900.00

Campaign for Southern Equality 2017
Title: *The Trans Buddy Program: Mental health advocacy for trans communities*
PI: **E. Kale Edmiston**
Award amount: $1,000.00

University of Pittsburgh Office of Diversity and Inclusion Mini-Grant 2017
Title: *Developing health promotion materials for the transgender community*
PI: **E. Kale Edmiston**
Award amount: $1,000.00

Trans Justice Funding Project 2017
Title: *The Trans Buddy Program: Peer advocacy solutions for mental health care access*
PI: **E. Kale Edmiston**
Award amount: $2,500.00

The Pollination Project 2016
Title: *The Trans Buddy Program: An innovative solution to transgender mental health disparity*
PI: **E. Kale Edmiston**
Award Amount: $1,500.00

Culture, Brain, and Development Grant 2006

Title: *Brain sex differences in mood disorders*
PI: **E. Kale Edmiston**
Award amount: $3,000.00

PEER-REVIEWED PUBLICATIONS (https://orcid.org/0000-0002-3548-6026):
2023:
48. Hoelscher EC, Victor SE, **Edmiston EK**. Gender minority resilience and suicidal ideation: a longitudinal and daily examination of transgender and non-binary adults. Behavior Therapist. (In Press).
47. Schroth-Erickson L, Levin R, Mak K, **Edmiston EK**. A review of the neurobiobehavioral literature of transgender identity. J Gay and Lesbian Mental Health. (In Press).
2022:
46. Coleman E, Radix AE, Bouman WP…**Edmiston EK**…Arcelus J. Standards of care for the health of transgender and gender diverse people, version 8. International Journal of Transgender Health. 2022; 23:1-258.
45. Juster RP, **Edmiston EK**. Refining research and representation of sexual and gender diversity in neuroscience. Biological Psychiatry: CNNI. 2022; 7(21):1251-7.
44. Colic L, Clark A, Sankar A, Rathi D, Goldman D, Kim JA, Villa LM, **Edmiston EK**, Lippard ETC, Mazure CM, Blumberg HP. Gender-related associations among childhood maltreatment on brain circuitry and clinical features of bipolar disorder. European Neuropsychopharmacology. 2022; 63:35-46.
43. **Edmiston EK**, Fournier JC, Chase HW, Aslam H, Lockovich J, Graur S, Bebko G, Bertocci M, Rozovsky R, Mak K, Forbes EE, Stiffler R, Phillips ML. Left ventrolateral prefrontal cortical activity during reward expectancy predicts mania risk up to one year post scan. J Affective Disorders. 2022; 319:325-8.
2021:
42. Bertocci MA, Chase HW, Graur S, Stiffler R, **Edmiston EK**, Coffman B, Greenberg T, Phillips ML. Reward circuitry-targeted cathodal transcranial direct current stimulation impacts reward circuitry and affect in bipolar disorder. Molecular Psychiatry. 2021; 26(8):4137-45.
2020:
41. Feng R, Womer FY, **Edmiston EK**, Chen Y, Wang Y, Chang M, Yin Z, Wei Y, Duan J, Ren S, Li C, Liu Z, Jiang X, Wei S, Li S, Zhang X, Nuo X, Tang Y, Wang F. Antipsychotic effects on cortical morphology in schizophrenia and bipolar disorders. Frontiers Neuroscience. 2020; 14:579139.
40. Wang L, Zhao Y, **Edmiston EK**, Womer FY, Zhang R, Zhao P, Jiang X, Wu F, Kong L, Zhou Y, Tang Y, Wei S, Wang F. Structural and functional abnormalities of amygdala and prefrontal cortex in major depressive disorder with suicide attempts. Frontiers Psychiatry. 2020; 10:923.
39. Wang Y, Wei Y, **Edmiston EK**, Womer FY, Zhang X, Duan J, Zhu Y, Zhang R, Zhang Y, Jiang X, Wei S, Liu Z, Zhang Y, Tang Y, Wang F. Altered structural connectivity and cytokines levels in schizophrenia and genetically high-risk individuals: associations with disease state and vulnerability. Schizophrenia Research. 2020; 223:158-165.
38. **Edmiston EK**, Fournier JC, Chase HW, Bertocci MA, Greenberg T, Aslam HA, Lockovich JC, Graur S, Bebko G, Forbes EE, Stiffler R, Phillips ML. Assessing relationships

among impulsive sensation-seeking, reward circuitry activity, and risk for psychopathology: an fMRI replication and extension study. Biological Psychiatry: CNNI. 2020; 5(7):660-68.

37. Sha Z, Versace A, **Edmiston EK**, Fournier JC, Graur S, Greenberg T, Lima Santos JP, Chase HW, Stiffler R, Bonar L, Hudak R, Yendiki A, Greenberg BD, Rasmussen S, Liu H, Quirk G, Haber S, Phillips ML. Functional disruption in prefrontal-striatal network in obsessive compulsive disorder. Psychiatry Research: Neuroimaging. 2020; 300:111081.

36. **Edmiston EK,** Song Y, Chang M, Yin Z, Zhou Q, Zhou Y, Jiang X, Wei S, Xu K, Tang Y, Wang F. Hippocampal functional connectivity in patients with schizophrenia and unaffected family members. Frontiers in Psychiatry. 2020; 11:278.

35. Wei S, Womer F, **Edmiston EK**, Zhang R, Jiang X, Wu F, Kong L, Zhou Y, Tang Y. Structural alterations associated with suicide attempts in major depressive disorder and bipolar disorder: a diffusion tensor imaging study. Progress in Neuropsychopharmacology & Biological Psychiatry. 2020; 98.

34. Beach L, Eckstrand K, Ehrenfeld J, **Edmiston EK**, Ding J. A model for improving transgender healthcare quality. The Joint Commission Journal on Quality and Patient Safety. 2020; 46:37-43.

2019:

33. Sha Z*, **Edmiston EK***, Versace A, Fournier JC, Graur S, Greenberg T, Lima Santos JP, Chase HW, Stiffler RS, Bonar L, Hudak R, Yendiki A, Greenberg BD, Rasmussen S, Liu H, Buckner R, Quick G, Haber S, Phillips ML. Multimodal disruption of cerbello-thalamo-motor circuit in obsessive compulsive disorder. Biological Psychiatry: CNNI. 2019; 5(4):438-47. *co-first authors

32. Wang L, Zhao Y, **Edmiston EK**, Womer FY, Zhang R, Zhao P, Jiang X, Wu F, Kong L, Zhou Y, Tang Y, Wei S. Structural and functional abnormalities of amygdala and prefrontal cortex in major depressive disorder with suicide attempts. Frontiers Psychiatry. 2019; 10:923.

30. Chang M, **Edmiston EK**, Womer F, Zhou Q, Shengnan W, Jiang X, Zhou Y, Ye Y, Huang H, Zui X, Xu K, Tang Y, Wang F. Spontaneous low-frequency fluctuations in the neural system for emotional perception in major psychiatric disorders: amplitude similarities and differences across frequency bands. Journal of Psychiatry and Neuroscience. 2019; 44:132-41.

29. Xia M, Womer FY, Chang M, Zhu Y, **Edmiston EK**, Jiang X, Wei S, Duan J, Xu K, Tang Y, He Y, Wang F. Shared and distinct functional architecture of brain networks across psychiatric disorders. Schizophr Bulletin. 2019; 47:450-63.

2018:

28. Li J, **Edmiston EK**, Tang Y, Fan G, Xu K, Wang F, Xu J. Shared facial emotion processing in medication-naive major depressive disorder and healthy individuals: detection by sICA. BMC Psychiatry, 2018; 18:96.

27. Chang M, Womer FY, **Edmiston EK**, Bai C, Zhou Q, Jiang X, Wei S, Wei Y, Ye Y, Huang H, He Y, Xu K, Tang Y, Wang F. Neurobiological commonalities among three major psychiatric diagnostic categories: a structural MRI study. Schizophrenia Bulletin. 2018; 44:65-74.

2017:

26. Wang N, **Edmiston EK**, Luo X, Yang H, Chang M, Wang F, Fan G. Comparing amplitude of low-frequency fluctuations in multiple system atrophy and idiopathic Parkinson's disease. Psychiatry Research Neuroimaging, 2017; 269:73-81.

25. Jiang X, **Edmiston EK**, Zhou Q, Xu K, Zhou Y, Wu F, Kong L, Wei S, Zhou Y, Chang M, Geng H, Wang D, Wang Y, Cui W, Tang Y, Wang F. Alteration of a cortico-striatal-limbic neural system in major depressive disorder and bipolar disorder. Journal of Affective Disorders, 2017; 221:297-303.

24. Corbett BA, Blain S, **Edmiston EK**. The role of context in psychosocial stress among adolescents with Autism Spectrum Disorder: piloting a semi-structured, video game-based paradigm. Journal of Intellectual & Developmental Disability. 2017; 43:20-8.

23. **Edmiston EK**, Muscatello RA, Corbett BA. Altered pre-ejection period response to social evaluative threat in adolescents with autism spectrum disorder. Research in Autism Spectrum Disorders. 2017; 36:57-65.

2016:

22. **Edmiston EK**, Donald CA, Sattler AR, Peebles JK, Ehrenfeld JM, Eckstrand KL. Opportunities and gaps in transgender primary healthcare: a systematic review. Transgender Health. 2016; 1(1):216-30.

21. **Edmiston EK**, Jones RM, Corbett BA. Physiological response to social evaluative threat in adolescents with autism spectrum disorder. Journal of Autism Developmental Disorders. 2016; 46(9):2992-3005.

20. **Edmiston EK**, Blain S, Corbett BA. Salivary cortisol and behavioral response to social evaluative threat in adolescents with autism spectrum disorder. Autism Research. 2016; Epub ahead of print.

2015:

19. Tang Y, Chen K, Zhou Y, Wang Y, Driesen N, **Edmiston EK**, Chen X, Jiang X, Kong L, Zhou Q, Li H, Wu F, Xu K, Wang Z, Tang Y, Wang F. Neural activity changes in unaffected children of patients with schizophrenia: a resting-state fMRI study. Schizophrenia Research. 2015; 168(1-2):360-5.

18. **Edmiston EK**, Merkle K, Corbett BA. Neural and cortisol responses during play with human and computer partners in children with autism. Social Cognitive Affective Neuroscience. 2015; 10(8):1074-83.

2014:

17. Corbett BA, Newsom C, Key AP, Qualls L, **Edmiston EK**. Examining the relationship between face processing and social interaction behavior in children with and without autism spectrum disorder. J Neurodevelopmental Disorders, 2014; 6(1):35.

16. Li J*, **Edmiston EK**,* Chen B, Tang Y, Ouyang X, Jiang Y, Fan G, Ren L, Liu J, Zhou Y, Jiang W, Liu Z, Xu K, Wang F. A comparative diffusion tensor imaging study of corpus callosum subregion integrity in bipolar disorder and schizophrenia. Psychiatry Res. 2014; 221(1):58-62.*co-first authors

2013:

15. **Edmiston EK***, McHugo M*, Dukic MS, Smith SD, Abou-Khalil B, Zald DH. Enhanced visual cortical activation for emotional stimuli is preserved in patients with unilateral amygdala resection. J Neuroscience, 2013; 33(27):11023-11031. *co-first authors

14. Liu H, **Edmiston EK**, Fan G, Ku X, Zhao B, Shang X, Wang F. Altered resting-state functional connectivity of the dentate nucleus in Parkinson's disease. Psychiatry Research: Neuroimaging. 2013; 211(1):64-71.

13. **Edmiston EK**, Blackford JU. Childhood maltreatment and response to novel face stimuli presented during functional magnetic resonance imaging in adults. Psychiatry Research: Neuroimaging. 2013; 212(1):36-42.

2012:

12. Fengrong O, Kai L, Qian G, Dan L, Jinghai L, Liwen H, Xian W, **Edmiston EK**; Yang L. An urban neo-poverty population-based quality of life and related social characteristics investigation from northeast china. PLoS One. 2012; 7(6):e38861.

11. Chepenik LG, Wang F, Spencer L, Spann MN, Kalmar JH, Womer F, **Edmiston EK**, Pittman B, Blumberg HP. Structure-function associations in hippocampus in bipolar disorder. Biological Psychiatry. 2012; 90(1):18-22.

2011:

10. Wang F, Kalmar JH, Womer FY, **Edmiston EK**, Chepenik LG, Chen R, Spencer L, Blumberg HP. Olfactocentric paralimbic cortex morphology in adolescents with bipolar disorder. Brain. 2011; 134(7):2005-12.

9. **Edmiston E**, Wang F, Mazure CM, Sinha R, Mayes LC, Blumberg HP. Cortico-striatal limbic gray matter morphology in adolescents reporting exposure to childhood maltreatment. Archives of Pediatric and Adolescent Med. 2011; 165(12):1069-77.

8. **Edmiston E**, Wang F, Kalmar JH, Womer FY, Chepenik LG, Pittman B, Gueorguieva R, Hur E, Spencer L, Staib LH, Constable RT, Fulbright RK, Papademetris X, Blumberg HP. Lateral ventricle volume and psychotic features in adolescents and adults with bipolar disorder. Psychiatry Research. 2011; 194(3):400-2.

2009:

7. Womer FY, Wang F, Chepenik LG, Kalmar JH, Spencer L, **Edmiston E**, Constable RT, Papademetris X, Blumberg HP. Sexually dimorphic features of vermis morphology in bipolar disorder. Bipolar Disord 2009; 11(7):753-8.

6. Jiang Y, **Edmiston E**, Wang F, Blumberg HP, Papademetris X, Staib, LH. Improving the reliability of shape comparison by perturbation. IEEE Biomedical Imaging 2009; 1:686-9.

5. Jiang Y, **Edmiston E**, Wang F, Blumberg HP, Staib LH and Papademetris X. Shape comparison using perturbing shape registration. IEEE Computer Vision Pattern Recognition 2009;683-90.

4. Wang F, Kalmar JH, He Y, Jackowski M, Chepenik LG, **Edmiston E**, Tie K, Gong G, Shah MP, Jones M, Uderman J, Constable RT, Blumberg HP. Functional and structural connectivity between the perigenual anterior cingulate and amygdala in bipolar disorder. Biological Psychiatry 2009; 66(5):516-21.

3. Kalmar JH, Wang F, Spencer L, **Edmiston E**, Lacadie CM, Martin A, Constable RT, Duncan JS, Staib LH, Papademetris X, Blumberg HP. Preliminary evidence for progressive prefrontal abnormalities in adolescents and young adults with bipolar disorder. J Int Neuropsychol Soc. 2009; 15(3):476-81.

2008:

2. Blumberg HP, Wang F, Chepenik LG, Kalmar JH, **Edmiston E**, Duman RS, Gelernter J. Influence of vascular endothelial growth factor variation on human hippocampus morphology. Biological Psychiatry 2008; 64(10):901-3.

1. Wang F, Kalmar JH, **Edmiston E**, Chepenik LG, Bhagwagar Z, Spencer L, Pittman B, Jackowski M, Papademetris X, Constable RT, Blumberg HP. Abnormal corpus callosum

integrity in bipolar disorder: A diffusion tensor imaging study. Biological Psychiatry 2008; 64(8):730-3.

MANUSCRIPTS (IN PROGRESS):

Ravindranath O, Perica MI, Parr AC, Pjha A, McKeon SD, Montano G, Ullendorf N, Luna B, **Edmiston EK**. Adolescent neurocognitive development and decision-making regarding gender affirming care. (Submitted).

Soehner AM, **Edmiston EK**, Wallace M, Chase HW, Lockovich J, Aslam H, Stiffler R, Graur S, Skeba A, Bebko G, Benjamin OE, Wang Y, Phillips ML. Neurobehavioral reward and sleep-circadian phenotypes predict present and next-year mania/hypomania risk. (Submitted).

Sequiera S, Tervo-Clemmens B, Carmel T, **Edmiston EK.** Towards a biopsychosocial model for neurodevelopment in transgender and gender diverse adolescents: understanding risk and resilience for mood disorders. (Submitted).

POSTERS, ABSTRACTS, AND CONFERENCE PROCEEDINGS:

53. Victor SE, **Edmiston EK**. Ecological momentary assessment of gender-relevant versus other interpersonal stressors predicting self-injurious thoughts and behaviors among transgender and non-binary adults. *Association for Behavioral and Cognitive Therapy Annual Convention. Submitted.*

52. **Edmiston EK**, Fournier JC, Chase HW, Phillips ML. Ventral visual stream functional coupling during implicit emotional face perception is associated with internalizing symptoms: a double dissociation by face valence at baseline and six months post-scan. *American College of Neuropsychopharmacology.* 2023.

51. Victor SE, Hoelscher E, Sandel D, Trieu T, **Edmiston EK.** Interpersonal and intrapersonal gender minority stressors as contribution to suicidal ideation among transgender and non-binary adults. *Suicide Research Symposium.* 2022.

50. Aslam MA, Mak K, **Edmiston EK.** Piloting transcranial direct current stimulation to reduce threat sensitivity in high trait anxiety. *University of Pittsburgh Department of Psychology Undergraduate Directed Experiences in Research Poster Day*. 2022.

49. **Edmiston EK** & Strakowski S. Understanding diagnosis and assessment disparities in transgender populations. *Society of Biological Psychiatry Annual Meeting.* 2022. Discussant, Lunchtime “Fireside Chat” Series.

48. Bertocci M, Afriyie-Agyemang Y, Rosovsky R, Aslam H, Graur S, **Edmiston EK**, Chase HW, Bebko G, Stiffler R, Phillips ML. Network interference during emotion regulation in distressed adults consistently predicts depression symptoms. *Society of Biological Psychiatry Annual Meeting.* 2022.

47. Afriyie-Agyemang Y, Bertocci M, Rozovsky R, Aslam H, Graur S, **Edmiston EK**, Chase HW, Bebko G, Stiffler R, Phillips ML. Overcompensation of the central executive network during working memory may be a neural marker for youth at risk for bipolar disorder. *Society of Biological Psychiatry Annual Meeting*. 2022.

46. Schumer MC, Bertocci MA, Bebko G, Stiffler RS, Lockovich JC, Aslam HA, Graur S, **Edmiston EK**, Chase HW, Johnson SL, Phillips ML. Trait urgency mediates associations between neural emotion-processing markers of emotion-triggered impulsivity and mania in young adults at-risk for bipolar disorder. *Society of Biological Psychiatry Annual Meeting*. 2022.

45. Young J, Roepke T, Anacker C, Ehrensaft D, **Edmiston EK**, Guthman EM, Eshel N, Marrocco J. Challenges and opportunities for translational research and clinical strategies within the LGBTQIA2S+ community. *American College of Neuropsychopharmacology Annual Meeting.* 2021. Discussant, Study Group.

44. Phillips ML, Bertocci M, Chase HW, Graur S, Stiffler R, **Edmiston EK**, Coffman BA. Targeted non-invasive neuromodulation impacts reward expectancy-related reward circuitry activity and affect in bipolar disorder and healthy adults. *Society of Biological Psychiatry Annual Meeting.* 2021.

43. **Edmiston EK,** Fournier JC, Rozovsky R, Chase HW, Bertocci MA, Aslam HA, Lockovich J, Graur S, Bebko G, Forbes EE, Stiffler R, Phillips ML. Left ventrolateral prefrontal cortex structure and reward-expectancy related activity predict manic symptom changes one year later. *American College of Neuropsychopharmacology Annual Meeting.* 2021.

42. **Edmiston EK**, Phillips ML, Mak K, Chase HW, Fournier JC. Visual cortex coupling and childhood maltreatment: associations with major depression and a compensatory mechanism. *Society of Biological Psychiatry Annual Meeting.* 2021.

41. Marrocco J, **Edmiston EK**, Anacker C, Bangasser D. The study of sex differences and gender bias, and trans inclusive research practices. *American College of Neuropsychopharmacology Annual Meeting.* 2020. Panelist, Networking Session.

40. Chase HW, Fournier JC, Bertocci MA, **Edmiston EK**, Lockovich JC, Aslam H, Stiffler RS, Graur S, Bebko G, Phillips ML. Decision-making variability in mood disorders: new insights for a replication attempt. *Society of Biological Psychiatry Annual Meeting.* 2020 (Submitted, meeting canceled due to COVID-19).

39. **Edmiston EK,** Fournier J, Greenberg T, Chase HW, Stiffler R, Lockovich J, Aslam H, Graur S, Bebko G, Phillips ML. A double dissociation between anxiety and depression symptom improvement and fusiform coupling and positive and negative emotional face processing. *Society of Biological Psychiatry Annual Meeting.* 2020 (Submitted, meeting canceled due to COVID-19).

38. **Edmiston EK**, Fournier JC, Chase HW, Bertocci MA, Greenberg T, Aslam HA, Lockovich JC, Graur S, Bebko G, Forbes EE, Stiffler R, Phillips ML. Assessing relationships among impulsive sensation-seeking, reward circuitry activity, and predisposition to bipolar disorder: an fMRI replication and extension study. *American College of Neuropsychopharmacology Annual Meeting. 2019.*

37. Paglisotti T, Montano G, Simpson A, **Edmiston EK**. Preliminary implementation of Trans Buddy PGH: establishing trust among transgender patients and healthcare providers. *University of Pittsburgh Medical Center Department of Psychiatry 19th Annual Research Day.* 2019.

36. **Edmiston EK**, Fournier JC, Chase HW, Bertocci MA, Greenberg T, Aslam H, Stiffler R, Lockovich J, Graur S, Bebko G, Phillips ML. Left ventrolateral prefrontal cortical BOLD signal during reward expectancy and impulsive sensation seeking: a replication study. *University of Pittsburgh Medical Center Department of Psychiatry 19th Annual Research Day. 2019.*

35. Chase HW, **Edmiston EK**, Bertocci M, Fournier JC, Greenberg T, Aslam H, Stiffler R, Lockovich J, Graur S, Bebko G, Forbes EE, Phillips ML. Similar neural representation of appetitive and loss avoidance prediction errors across distressed and healthy individuals. *Society of Biological Psychiatry Annual Meeting.* 2019.

34. **Edmiston EK**, Simpson A. Progress report: Quality improvement programming for transgender mental health. Symposium. *TransPride PGH Professional Conference.* 2018.

33. Schroth-Erickson L, Levin R, **Edmiston EK**. Talking to your patients about the biological basis of transgender identity. *Philadelphia Trans Wellness Conference Professional Track. 2018*.

32. **Edmiston EK**, Fournier J, Greenberg T, Chase HW, Stiffler R, Lockovich J, Aslam H, Graur S, Bebko G, Phillips ML. Fusiform gyrus-salience network coupling during emotion processing predicts anxiety and depression symptom change. *University of Pittsburgh Medical Center Department of Psychiatry 18th Annual Research Day*. 2018.

31. **Edmiston EK**, Fournier J, Greenberg T, Chase HW, Stiffler R, Lockovich J, Aslam H, Graur S, Bebko G, Phillips ML. Salience network BOLD response to emotional faces predicts anxiety and depression symptom outcomes. *Society of Biological Psychiatry Annual Meeting.* 2018.

30. Chase HW, Qiu H, Kerestes R, Shah N, Alkhar H, **Edmiston EK**, Soehner A, Greenberg T, Aslam H, Stiffler R, Lockovich J, Graur S, Bebko G, Pan L, Eickhoff SB, Phillips ML. Implication of the visual cortex in resting state fMRI studies of mood and anxiety disorders may relate to the propensity for within-scanner sleep. *Society of Biological Psychiatry Annual Meeting.* 2018.

29. Ding J, Ehrenfeld J, Raynor L, **Edmiston EK**, Eckstrand K, Beach L. A proposed systems level quality improvement model for transgender healthcare delivery. *The National Transgender Health Summit*. 2017.

28. **Edmiston EK.** Setting the agenda for transgender neuroimaging: a critical review and future directions. Symposium. *The National Transgender Health Summit.* 2017.

27. **Edmiston EK,** Fournier J, Greenberg T, Bertocci M, Stiffler R, Aslam H, Lockovich J, Phillips ML. Trait anxiety predicts visual system response to emotional faces. *Developmental Affective Neuroscience Symposium*. 2017.

26. **Edmiston EK.** The Trans Buddy Program: an innovative intervention for increasing health care utilization. Symposium. *TransPride PGH Professional Conference.* 2017.

25. Buchanan K, Richmond M, Sattler AR, **Edmiston EK**. Red state solutions for transgender health care access: provision in low resource areas. Symposium. *Philadelphia Transgender Health Conference.* 2017.

24. **Edmiston EK,** Chase H, Stiffler R, Lockovich J, Aslam H, Graur S, Bebko G, Phillips ML. Predicting quality of life in distressed youth: Cortico-thalamic BOLD signal and reward processing. *University of Pittsburgh Medical Center Department of Psychiatry 17th Annual Research Day*. 2017.

23. **Edmiston EK**, Chase H, Stiffler R, Lockovich J, Aslam H, Graur S, Bebko G, Phillips ML. Cortico-thalamic BOLD signal during reward processing predicts quality of life at follow up in distressed young adults. *Society of Biological Psychiatry Annual Meeting.* 2017.

22. Eckstrand KL, Mitchell L, **Edmiston EK.** The Trans Buddy Program: Transgender Leadership and peer advocacy for reducing health disparities. *University of Pittsburgh Health Sciences Health Disparity Poster Competition.* 2017.

21. **Edmiston EK.** Reframing the search for transgender neuroimaging biomarkers. *New Materialisms Annual Meeting* Warsaw, Poland. 2016.

20. Corbett BA, Muscatello R, **Edmiston EK**, Muse I. Examining the Diurnal Profile of Children and Adolescents with Autism Spectrum Disorder (ASD) and Typical Development between 8 to 17 years of age. *International Society for Psychoneuroendocrinology.* 2016.

19. Corbett BA, Muse I, **Edmiston EK**, Muscatello R. Diurnal and Stress Hormonal Profiles of Testosterone and Cortisol in Adolescents with Autism Spectrum Disorder (ASD) and Typical Development (TD). *International Society for Psychoneuroendocrinology.* 2016.

18. **Edmiston EK**. Psychophysiological characterization of adolescents with Autism Spectrum Disorder. Presentation, *Chinese Psychiatric Association Annual Meeting.* 2016.

17. **Edmiston EK**, Jones RM, Blain S, Corbett BA. Neuroendocrine and physiological responsivity during social stress in adolescents with and without autism spectrum disorder. *Vanderbilt Kennedy Center Science Day.* 2015.

16. **Edmiston EK**, Valencia B, Corbett BA. Autonomic nervous system function in response to social judgment in adolescents with and without autism spectrum disorder. *International Meeting for Autism Research.* 2015.

15. Corbett BA, Newsom C, Key S, Qualls L, **Edmiston EK**. A randomized wait-list control trial of a peer-mediated, theatre-based intervention to improve social ability in children with autism spectrum disorder. *International Meeting for Autism Research.* 2015.

14. Singer B, Eckstrand K, Ehrenfeld J, **Edmiston EK**. Transgender health and advocacy in academic medicine: an empowerment model. Workshop; *Gay and Lesbian Medical Association Annual Meeting.* 2014.

13. **Edmiston EK**, Corbett BA. Behavioral and endocrine alterations in adolescents with autism spectrum disorder. Selected presentation; *Vanderbilt Kennedy Center Science Day*. 2014.

12. **Edmiston EK.** Effects of a neurobiological explanation of sexual orientation on student attitudes towards lesbian, gay and transgender people. *Society for Neuroscience.* 2013.

11. Corbett BA, **Edmiston EK**, Zald DH. Neural and physiological responses during play with human and computer partners in children with autism. *Society for Neuroscience.* 2013.

10. **Edmiston EK,** McHugo M, Dukic MS, Eggers E, Zald DH. Visuocortical BOLD response to emotional stimuli in the absence of a functional amygdala. *Society for Neuroscience.* 2012.

9. **Edmiston EK.** Pelvic and chest exams in transgender men. Workshop; *Philadelphia Trans Health.* 2011.

8. **Edmiston EK**, Blackford JU. Childhood maltreatment affects face processing. *Biology of Prosocial Behavior.* 2011.

7. **Edmiston E,** Wang F, Mazure CM, Sinha R, Mayes LC, Blumberg HP. Cortico-striatal limbic gray matter morphology in adolescents reporting exposure to childhood maltreatment. *Vanderbilt Kennedy Center Science Day.* 2011.

6. Wang F, **Edmiston E**, Hur E, Kalmar JH, Womer FY, Chepenik LG, Blumberg HP. An Altered Developmental Trajectory of Frontotemporal Connectivity in Bipolar Disorder. *Biological Psychiatry* 2010; 67 (Supplement 9): 107.

5. Wang F, Chepenik LG, Shah MP, Kalmar JH**, Edmiston E**, Spencer L, Duman R, Gelernter J, Blumberg HP. Genes Regulating Neurotrophic Factors that Influence the Corticolimbic Connectivity in Mood Disorders: Treatment Implications. *Biological Psychiatry* 2009; 65 (Supplement 1): 174.

4. Kalmar JH, Wang F, Chepenik LG, Shah MP, McDonough A, **Edmiston E**, Blumberg HP. Amygdala functioning during emotional processing in adolescents with bipolar disorder or ADHD. *Biological Psychiatry* 2008; 63 (Supplement 1): 184.

3. Womer F, Wang F, Chepenik LG, Kalmar JH, **Edmiston E**, Spencer L, Constable RT, Papademetris X, Blumberg HP. Structural abnormalities of the cerebellar vermis in bipolar disorder. *Biological Psychiatry* 2008; 63 (Supplement 1): 141.

2. Wang F, Kalmar JK, Womer F, He Y, Chepenik L, **Edmiston E**, Blumberg HP. Abnormal morphological correlations within a cortico-limbic neural system in adolescents with bipolar disorder. *American Academy of Childhood and Adolescent Psychiatry.*

1. Wang F, Kalmar JH, **Edmiston E**, Chepenik LG, Tie K, Spencer L, Jackowski M, Papademetris X, Constable RT & Blumberg HP. Abnormal callosal integrity in bipolar disorder determined from diffusion tensor imaging. *Biological Psychiatry* 2008; 63 (Supplement 1): 43.

BOOK CHAPTERS:

Edmiston EK, Bertocci M, Phillips ML. Neuroimaging and Circuit Mechanisms of Bipolar Disorder. In *Neurobiology of Mental Illness*. 6th Ed. Eds: Eric Nestler & Alexander Charney. Oxford University Press. (In Press).

Tomson A & **Edmiston EK**. Understanding the basis of gender identity development: biological and psychosocial models. In *Trans Bodies, Trans Selves*. 2nd Ed. Ed: Sand Chang. Oxford University Press. 2022.

Edmiston EK. Community-led peer advocacy for transgender health care access in the southeastern United States: The Trans Buddy Program. In *Healthcare in Motion: Mobility forms in health service delivery and accessibility.* Berghahn Books. 2017.

Robles RJ & **Edmiston EK**. Community Responses to Trauma. In *Trauma, Resilience, and Health Promotion for LGBT Patients.* Springer Press. 2017.

Edmiston EK & Mitchell L. Trans Buddy: Innovation Profile. In *The Remedy: Queer and Trans Voices on Health and Health Care.** Arsenal Press. 2016. *Lambda Literary Award Winner, Non-Fiction Anthology

Eckstrand KL, **Edmiston EK**, Potter J. Obstetric and Gynecologic Care to LGBT Individuals. In *Lesbian, Gay, Bisexual, Transgender, and Intersex Healthcare: A Clinical Guide to Preventative, Primary, and Specialist Care*. Springer Press. 2015.

ADDITIONAL SCHOLARSHIP:

Edmiston EK. Letter to the Editor: The legacy of transgender surgery access is complex. *Annals of Plastic Surgery.* 2019.

Edmiston EK. Invited Commentary: Transgender health research must serve transgender people. *BJOG.* 2018.

Edmiston EK. Feminist bioethics and intersex medical interventions: A review of *Making Sense of Intersex*. *Catalyst: Feminism, Theory, Technoscience*. 2016; 2(1).

Jann JT, **Edmiston EK**, Ehrenfeld J. Letter to the Editor: Important considerations for addressing LGBT health care competency. *American J of Public Health* 2015; e1.

HONORS, AWARDS, AND FELLOWSHIPS:

American College of Neuropsychopharmacology Travel Award	2021
Society of Biological Psychiatry Early Career Investigator Travel Award	2019
NYC tDCS Fellowship City University of New York, New York, NY	2018
Trainee, T32 MH018951 Child and Adolescent Mental Health Research University of PIttsburgh, Pittsburgh, PA	2018-2019
Research Day Department of Psychiatry Outstanding Poster Award	2018
PLOS One Travel Award	2017
Fellow, Winter School in the Neuroscience of Consciousness Canadian Institute For Advanced Research	2017
Trainee, T32 MH16804 Transformative Discovery in Psychiatry	2016-2018

University of Pittsburgh, Pittsburgh, PA

WPATH Outstanding Student Award 2015
International honor for contributions to transgender health research

The Trans 100 2015
National honor for excellence in the transgender community

Point Foundation Scholar 2014-2015
One of 20 selected nationally for program that funds education of LGBT students

Vanderbilt Brain Institute Student Leadership and Service Award 2014

Graduate Student Travel Grant, Vanderbilt University 2013

Fellow, Summer Program in Neuroscience Ethics and Success 2013
Marine Biology Laboratory, Woods Hole, MA

Clinical Neuroscience Scholar for Translational Research 2012-2015
Dan Marino Foundation

Neurobiology of Social Behavior Travel Award 2011
Emory University, Atlanta, GA

President's Scholarship 2003-2005
Case Western Reserve University, Cleveland, OH

TEACHING AND MENTORSHIP

SELECTED TALKS:

Invited Speaker: *Neuroscience in Service of Our Community:* 2023
How Research Rooted in Empathy and Humility Makes Us Better Scientists
Neuroscience Diversity Seminar
University of Maryland School of Medicine

Invited Speaker: *Visual Cortex Distinguishes Anxiety and Depression* 2023
Fournier Group Lab Meeting
The Ohio State Medical School

Presenter: *Assessing Visual Perception in Depression and Anxiety* 2023
Department of Psychiatry Faculty Meeting
UMass Chan Medical School

Invited Speaker: *Neuroimaging Studies of Transgender People* 2022
The Friedman Brain Institute and oSTEM
The Icahn School of Medicine at Mount Sinai

Invited Speaker: *Impulsivity and Reward-related Activity:* 2022
A Stable Marker for Bipolar Disorder risk
STEP Seminar
Truman State University

Invited Speaker: *Assessing Relationships Among impulsivity, Reward Circuitry, and Risk for Psychopathology* Magnetic Resonance Research Center Forum Yale School of Medicine	2019
Presenter: *Fusiform Gyrus Alterations During Emotion Processing: Predicting the Future in Anxiety Disorders* Center for the Neural Basis of Cognition Seminar University of Pittsburgh and Carnegie Mellon University	2018
Panelist: *Setting the Research Agenda in Transgender Health* 27th Annual Issues in Medical Ethics Conference The Icahn School of Medicine at Mount Sinai	2017
Panelist: *Neuroimaging in Child and Adolescent Mental Disorders* Chinese Society of Psychiatry 14th Annual Meeting	2016
The Trans Buddy Program: An Innovative Model for Healthcare Access Medicine Health and Society Colloquium Series Vanderbilt University	2015
Panel Organizer: *Intra-community Stigma in LGBT Populations* 615Thrive Conference Tennessee Department of Health	2015
Transgender Health: Provider Considerations Department of Hearing and Speech Sciences Grand Rounds Vanderbilt University	2014
Sexual and Reproductive Health in LGBT Populations Sarah Fogel, PhD Department of Nurse Midwifery Vanderbilt University School of Nursing	2014, 2015
Panelist: *(Im)Possible Politics: Intersectional Trans Organizing* Ben Singer, PhD; Dean Spade, JD; Lisa Guenther PhD Department of Women and Gender Studies Vanderbilt University	2014
Plenary Speaker: *Creating Change for LGBTI Health* Gay and Lesbian Medical Association Annual Meeting	2013
Invited Speaker: *Threat Detection, Visual Cortex, and Anxiety* Department of Radiology Beijing Normal University	2013
Invited Speaker: *Threat Detection, Visual Cortex, and Anxiety* Department of Psychiatry China Medical University	2013

MEDICAL STUDENT TEACHING EXPERIENCE:

Guest Lecturer: *Neuromodulatory Interventions in Mood Disorders* 2022
Neuroscience Area of Concentration Seminar Series
University of Pittsburgh School of Medicine

Guest Lecturer: *Building Trust with your Transgender Patients* 2021,2022
Texas Christian University School of Medicine

Instructor of Record: *Introduction to Scientific Writing* 2016
China Medical University

Guest Lecturer: *Clinical and Biobehavioral Features of Autism* 2016
Clinical Medicine 400
China Medical University

Guest Lecturer: *Building an Inclusive Practice for LGB and T Patients* 2015
First Year Seminar
Meharry Medical College

Guest Lecturer: *Community Models for Improving Trans Healthcare* 2015
Intercession Course
Meharry Medical College

Guest Lecturer: *Providing Excellent Care for LGBT People* 2015
Capstone Series
Meharry Medical College

GRADUATE AND UNDERGRADUATE TEACHING EXPERIENCE:

Guest Lecturer: *Neuromodulation interventions for threat sensitivity* 2022
Biomedical Sciences First Year Seminar
Graduate School of Biomedical Sciences
UMass Chan Medical School

Guest Lecturer: *Impulsivity and reward-related activity: Predicting mania* 2021
Undergraduate Research Methods
Department of Psychology
University of California San Diego

Guest Lecturer: *Transgender people and neuroimaging: a critical review* 2021
Department of Psychology
Mount Holyoke College

Instructor of Record: PSY0205 Psychopathology 2021
Department of Psychology
University of Pittsburgh

Guest Lecturer: *Transgender People and Healthcare Systems* 2015
MHS 2110: American Medicine and the World

Laura Stark, PhD, Vanderbilt University

Guest Lecturer: *Transgender People and Healthcare Systems* 2015
MHS 3890: Documenting the Body
Odie Lindsey, PhD, Vanderbilt University

Guest Lecturer: *Introduction to Social Neuroscience* 2014
PSY3609: Educational Cognitive Neuroscience
Sasha Key, PhD, Vanderbilt University

Guest Lecturer: *Imagining Transgender Bodies in Healthcare* 2013
WGS 290: Theories of the Body
Aimi Hamraie, PhD, Vanderbilt University

Introduction to Cognitive Neuroscience 2013-2014
Vanderbilt Neuroscience Graduate Program Boot Camp

The Center for Teaching, Vanderbilt University 2013
Scholarship of Teaching and Learning Certificate

Teaching Assistant: NSC201 Introduction to Neuroscience 2011
Department of Neuroscience, Vanderbilt University

TRAINEE MENTORSHIP, CERTIFICATION, AND SUPERVISION:

Culturally Aware Mentorship Workshop 2022
University of Wisconsin Madison School of Medicine

Tiffany Nhan (post bac lab assistant) 2022-present

M. Ali Aslam (undergraduate lab assistant) 2022

Paloma Rueda (undergraduate lab assistant) 2020-2021

Shelby Gardner (undergraduate lab assistant) 2020

Kristie Mak (undergraduate lab assistant) 2019-2020

Taylor Pagliosotti, BA (graduate student, Department of Public Health) 2018-2019

Zhiqiang Sha, PhD (post doc, Mood and Brain Laboratory, PI: Phillips) 2019

Alicyn Simpson, BA (research assistant, Adolescent Medicine) 2018-2019

Hana Choi, BA (intern, The Trans Buddy Program) 2016

William Horn, BA (intern, The Trans Buddy Program) 2015

RJ Robles, BA (student worker, Program for LGBTI Health) 2015-2016

Keanan Gottlieb, BA (summer intern, The Trans Buddy Program) 2014

Cameron Donald, BA (summer intern, Program for LGBTI Health) 2014

Jamieson Jann, BA (summer intern, Program for LGBTI Health) 2014

SERVICE

CURRENT MEMBERSHIPS:
Society of Biological Psychiatry

DEPARTMENTAL, INSTITUTIONAL, AND DISCIPLINARY SERVICE:

Editorial Board, *Journal of Mood and Anxiety Disorders* 2023-present

Member, Grand Rounds Committee 2023-present
Department of Psychiatry, UMass Chan Medical School

Interviewer, Graduate School of Biomedical Sciences 2023-present
UMass Chan Medical School

Co-Director, NeuroNexus Institute 2022-present
UMass Chan Medical School

Co-chair, Diversity, Equity and Inclusion Committee 2021-present
Society of Biological Psychiatry

Member, LGBTQIA+ Task Force 2021-present
American College of Neuropsychopharmacology

Editorial Board, *Bulletin of Applied Transgender Studies* 2021-present

Grant Reviewer, Lesbian Health Fund, GLMA 2021

Member, Diversity, Equity, and Inclusion Committee 2019-2021
Department of Psychiatry University of Pittsburgh School of Medicine

Chapter Author, Assessment of Adults with Gender Dysphoria 2018-2022
WPATH Standards of Care 8 Committee

Member, Diversity and Inclusion Committee 2018-2021
Society of Biological Psychiatry

Ad Hoc Member, Diversity and Inclusion Task Force 2020-2021
American College of Neuropsychopharmacology

Member, Cross-Network Transgender Working Group, 2017-2019
NIH Office of HIV/AIDS Network Coordination

Co-Founder, Trans Buddy Pittsburgh 2016-2018

Student Representative, Vanderbilt Brain Institute Diversity Committee 2015-2016

Founding Director, The Trans Buddy Program Nashville 2014-2016

Co-Director, Vanderbilt School of Medicine Program for LGBTI Health 2014-2015

Assoc. Director, Vanderbilt School of Medicine Program for LGBTI Health 2013-2014

Associate Editor, *Vanderbilt Reviews Neuroscience*	2013-2014
President, Vanderbilt Neuroscience Student Organization	2013-2014
Member, Vanderbilt Neuroscience Organization Academic Committee	2012-2013
Board Member, Vanderbilt School of Medicine Program for LGBTI Health	2012-2013
Affiliate, Vanderbilt Kennedy Center	2011-2016

AD HOC PEER REVIEW:
Acta Psychologica; American Journal of Psychiatry; American Journal of Sexuality Education; Annals of Internal Medicine; Biological Psychiatry: Cognitive Neuroscience Neuroimaging; BJOG: An International Journal of Obstetrics and Gynaecology; Bipolar Disorder; Brain and Behavior; Child Abuse & Neglect; Development and Psychopathology; Developmental Cognitive Neuroscience; Frontiers in Neuroscience; Frontiers in Sociology; Human Brain Mapping; Journal of Affective Disorders; Journal of Autism and Developmental Disorders; Journal of Homosexuality; Journal of Medical Systems; Journal of Neuroscience Research; Journal of Psychiatry, Depression, and Anxiety; LGBT Health; Molecular Autism; NeuroImage; Neuropsychologia; Neuropsychopharmacology; Neuroscience Letters; Psychiatry Research: Neuroimaging; PLOS One; Psychological Medicine; Psychology of Violence; Psychoneuroendocrinology; Scientific Reports; Schizophrenia Research; Transgender Health

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Hilary P. Blumberg, MD, John and Hope Furth Professor of Psychiatric Neuroscience, Professor Departments of Psychiatry and Radiology and Biomedical Imaging, Yale School of Medicine. email: hilary.blumberg@yale.edu

Exhibit B

Bibliography

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EXHIBIT D

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF FLORIDA
Tallahassee Division

AUGUST DEKKER, et al., *Plaintiffs*, v. JASON WEIDA, et al., *Defendants*.	Case No. 4:22-cv-00325-RH-MAF

REBUTTAL REPORT OF DANIEL SHUMER, M.D.

I, Daniel Shumer, M.D., hereby declare and state as follows:

1. I have been retained by counsel for Plaintiffs as an expert in connection with the above-captioned litigation.

2. I have actual knowledge of the matters stated herein. If called to testify in this matter, I would testify truthfully and based on my expert opinion.

3. My background and qualifications, review of prior testimony, and compensation have been previously provided in my expert report ("Shumer Rep."). The curriculum vitae attached to my initial expert report remains true, correct and up to date.

4. I hereby provide a rebuttal report to respond to the expert reports provided by the Defendants. This report is provided after my review of reports

submitted by Dr. Michael Laidlaw, Dr. Paul Hruz, Dr. Stephen Levine, Dr. Kristopher Kaliebe, Dr. Sophie Scott, Dr. Michael Biggs, and Dr. Joseph Zanga, as well as my review of plaintiffs' medical records.

5. In preparing this rebuttal report, I have relied on my training and years of research and clinical experience, as set out in my curriculum vitae (attached as **Exhibit A** to my original report) and on the materials listed therein; the materials listed in the bibliography attached as **Exhibit B** to my original report; and the additional materials listed in the supplemental bibliography attached as **Exhibit C** to this rebuttal report. The sources cited in each of these are the same types of materials that experts in my field of study regularly rely upon when forming opinions on the subject, which include authoritative, scientific peer-reviewed publications.

6. I reserve the right to revise and supplement the opinions expressed in this report or the bases for them if any new information becomes available in the future, including as a result of new scientific research or publications or in response to statements and issues that may arise in my area of expertise. I may also further supplement these opinions in response to information produced by Defendants in discovery and in response to additional information from Defendants' designated experts.

EXPERT OPINIONS

7. These expert reports all demonstrate a basic lack of understanding of the nature, evaluation, and treatment of gender dysphoria, the serious consequences of the condition if left untreated, and the strength of the evidence in support of medical management of gender dysphoria, including the efficacy and safety of these treatments. Defendants' experts have limited or no experience with diagnosis and treatment of gender dysphoria. Their opinions are not consistent with current evidence-based standards of care or the general medical consensus – they run counter to recommendations made by leading and well-respected medical bodies.

8. Some of the specific critiques apply in equal measure to more than one expert report.

I. Efficacy of Gender-Affirming Care

9. Dr. Laidlaw and Dr. Hruz are both endocrinologists not involved in the medical treatment of gender dysphoria. Dr. Laidlaw states, "*treatment interventions on behalf of children and adults diagnosed with gender dysphoria must be held to the same scientific standards as other medical treatments. These interventions must be optimal, efficacious, and safe.*" (Laidlaw Rep. ¶ 12). I agree with Dr. Laidlaw's statement; all medical interventions, including treatment for gender dysphoria, require rigorous study and high-quality evidence. The responsibility of all medical

providers is to provide care for patients with a goal of promoting health and wellness while minimizing risk; this can only be done with a thorough knowledge of the patient, their disease process, and the relevant scientific literature.

10. As a pediatric endocrinologist with vast experience in assessing and treating transgender patients, I rely on extremely strong and compelling evidence that hormonal treatments, including pubertal suppression and gender-affirming hormonal care, are efficacious, safe, and promote optimal health outcomes.

11. In my expert report, I referenced several studies which demonstrate the efficacy and safety of gender-affirming care. (Shumer Rep. ¶ 35 (citing de Vries, et al., 2014; de Vries, et al., 2011; Green, et al., 2022; Smith, et al., 2005; Turban, et al., 2022)). These articles represent a small percentage of the full body of literature that was utilized to create evidence-based clinical practice guidelines for the treatment of gender dysphoria in children, adolescents, and adults.

12. The guidelines were published by long-standing and well-respected bodies: the World Professional Association for Transgender Health (WPATH) and the Endocrine Society (Coleman, et al., 2022; Coleman, et al., 2012; Hembree, et al., 2017; Hembree, et al., 2009). Other leading medical bodies including the American Association of Pediatrics, the American Medical Association, the American Psychological Association, the American Psychiatric Association, and American

Academy of Family Physicians all support the tenants of these guidelines due to the rigorous nature of their review of scientific evidence in the field (Rafferty, et al., 2018 (AAP), AMA, 2019; American Psychological Association, 2015; Drescher, et al., 2018 (American Psychiatric Association); Klein, et al., 2018 (AAFP)).

13. Dr. Laidlaw and Dr. Hruz attempt to undermine the WPATH standards of care by characterizing WPATH as an "advocacy organization" (Laidlaw Rep. ¶ 185; Hruz Rep. ¶ 96). WPATH is a longstanding and well-respected standard-setting organization whose mission is "to promote evidence-based care, education, research, public policy, and respect in transgender health" (WAPTH Mission and Vision, 2023). Dr. Laidlaw takes issue with WPATH SOC 7's lack of description of the grading system used. WPATH SOC 8 (the current version) clearly and transparently outlines the grading system used, yet Dr. Laidlaw is still not satisfied, as he would have preferred they presented this information differently (Laidlaw Rep. ¶¶ 196-197). Yet his preference does not undermine the substance of the WPATH standards of care or the evidence on which they rely.

14. Dr. Laidlaw and Dr. Hruz also criticize the Endocrine Society guidelines, pointing to the makeup of the committee and the quality of the data (as existed in 2017). Yet, they fail to provide guidelines published by any well-respected medical body, which, after reviewing the evidence, came to opposite conclusions.

Instead, Dr. Laidlaw references other endocrinologists (one of whom is Dr. Hruz) who form a small group of professionals outside of the mainstream on this topic.

15. Dr. Lappert, who is a retired surgeon – not an endocrinologist – claims that a 2019 article jointly published by the Endocrine Society and other medical bodies on the use of testosterone therapy for women contradicts the Endocrine Society guidelines. But it does not. Adult women sometimes ask endocrinologists for low-dose testosterone because they believe it will help with a variety of concerns including low libido, sexual arousal, wellbeing, mood, or osteoporosis. The consensus report was evaluating those questions. It was ***not*** reviewing evidence related to gender dysphoria or making ***any*** statement for or against gender-affirming care. The words gender, gender dysphoria, and transgender are not contained in the document. The Endocrine Society likely assumed that readers would understand that by use of a title including the word *women* they were talking about *women* and not *transgender men*. Dr. Lappert's confusion here may be related to his refusal to respect transgender men by using any other terminology to refer to them besides "women."

16. Dr. Zanga appears to take issue with the decision of the American Academy of Pediatrics to support the current standards of care related to assessment and management of gender dysphoria (Zanga Rep. ¶¶ 8-18). Dr. Zanga claims that

the AAP ignored potential harms of gender-affirming care (Zanga Rep. ¶ 18). He is wrong. The relevant AAP document very clearly and fairly outlines risks and benefits related to gender-affirming care (Rafferty, et al., 2018). The AAP recommends a "'gender-affirming,' nonjudgemental approach that helps children feel safe in a society that too often marginalizes or stigmatizes those seen as different" (AAP News Release, 2018). This is consistent with the approaches taken by WPATH and the Endocrine Society.

17. Dr. Hruz argues that the outcomes of gender-affirming care are unknown in part because no randomized control trials have been performed (Hruz Rep. ¶ 112). While randomized control trials are an excellent study design in many contexts, management of gender dysphoria is not amenable to this type of study. Due to the current evidence supporting gender-affirming care, it would be unethical to propose a study randomly assigning patients, for example, to GnRHa treatment or placebo. Additionally, the study could not be blinded since patients and families would immediately ascertain which group they were randomized to based on the progression or non-progression of puberty. What is more, patients/families desiring treatment with GnRHa would be unlikely to consent to such a study for fear of being placed in the placebo group. Therefore, researchers in this field must rely on other types of study design, such as longitudinal cohort studies, which monitor change in

symptoms over the course of treatment (de Vries ALC, 2014), or cross-sectional studies comparing treated and untreated persons (Turban, 2022).

18. Dr. Laidlaw chooses to highlight several studies as examples of lack of evidence of effectiveness of gender-affirming treatments (Laidlaw Rep. ¶¶ 201-227). He leads with a review of Dhejne et al.'s study published in 2011. While Dr. Laidlaw is correct that, among other things, this study demonstrates that even after receiving appropriate gender-affirming care, transgender individuals are still at higher risk for negative mental health outcomes than the general population, Dr. Laidlaw ignores that stigma around transgender identity, both 12 years ago in Sweden and in Florida today, makes life more challenging for transgender individuals. What this study did *not* measure, however, is the difference in mental health between transgender individuals who received evidence-based care, and those who were unable to receive this care. In fact, the conclusion of Dhejne is *not* that gender-affirming care is inappropriate, but rather that transgender people require additional support during and after the process of transition.

19. Dr. Laidlaw also describes the efforts undertaken by himself and colleagues to discredit the results of a Swedish study which aimed to investigate rates of mood and anxiety disorder health care visits and antidepressant and anxiolytic prescriptions in patients receiving hormonal or surgical interventions.

Ultimately, while the authors conceded in a letter to the editor that their conclusions were "too strong," they maintained that the study "serves an important purpose and fills an important knowledge gap." The study "lends support for expecting a reduction in mental health treatment as a function of time since completing such treatment" (Branstrom & Pachankis, 2019).

20. Dr. Laidlaw also unfairly discounts the 2015 US Transgender Survey (2015 USTS) and studies based on data from this survey (Laidlaw ¶¶ 210-11). The 2015 USTS serves as the largest survey examining the experiences of transgender people in the United States with 27,715 respondents from all fifty states, DC, American Samoa, Puerto Rico, and U.S. military bases overseas. While extremely large population studies are logistically challenging, the USTS clearly and transparently outlines the recruitment methodology in Chapter 2 of its full report. Its main outreach objective was *to provide opportunities to access the survey for as many transgender individuals as possible in different communities across the U.S. and its territories* (James, 2016). Thus, it was appropriate for the survey to use convenience sampling to achieve its goals. While there are inherent limitations to studies that use this method to reach a large sample, in reviewing any data derived from the 2015 USTS, it is important to consider not only limitations of population-based survey data, but also the significant strengths of being able to capture data

from such a large cohort of individuals. Importantly, Dr. Laidlaw does not point to any studies that contradict the findings of the 2015 USTS.

21. Dr. Hruz takes a similar approach, arguing that studies of gender-affirming care have "major methodological limitations" and attempting to discredit individual studies (Hruz Rep. ¶ 119). What Dr. Hruz ignores is that all scientific studies have limitations. In fact, including a limitation section is required when publishing any manuscript in scientific journals (Lancet, Information for Authors, 2023). That a study has limitations does not mean that the study is dismissed out of hand. To the contrary, each study contributes to the collective knowledge base and health care providers look at the entire body of evidence – as well as their own clinical experience and that of their colleagues – to inform their approach to treatment.

22. For example, Dr. Hruz critiques 2011 and 2014 studies by de Vries et al., which demonstrated that patients with gender dysphoria had improved behavioral and emotional outcomes and depressive symptoms after receiving medical treatment for their gender dysphoria. Dr. Hruz suggests that because the participants were also receiving psychological support, it is not possible to know if it was medical treatment or psychological support which caused the improvement in mental health symptoms (Hruz Rep. ¶ 120). He misunderstands that gender-

affirming care does not mean drugs alone, but rather a constellation of medical treatment and psychosocial support. Separating these aspects of care does not make sense clinically. In my view, the findings in these two studies provide strong evidence in favor of gender-affirming treatment.

23. Dr. Hruz also wrongly claims that the 2020 Turban et al. study is cited as "proof that pubertal blockade prevents suicide in transgender youth" (Hruz Rep. ¶ 120). As I described in my prior report, that study is one of a number of studies supporting the benefits of GnRHa treatment (Shumer ¶ 82). While Dr. Hruz points out that the rate of suicidality is high in both the group treated with GnRHa and the group that did not receive the treatment, that does not mean that the treatment was not helpful. A reduction in suicidality is a significant finding. His critique of the 2022 Tordoff et al. study suffers from the same flawed reasoning.

24. Notably, Dr. Biggs significantly downplays the suffering of transgender adolescents experiencing suicidality by arguing that most suicide attempts are not fatal (Biggs Rep. ¶¶ 15-19). This rhetoric is not only dangerous, but overlooks the fact that reduction in both completed suicide and suicidality are both worthy goals of treatment.

25. While previously faulting studies due to lack of a control group, Dr. Hruz discounts the findings of a study with a control group (van der Miesen, et al.,

2020) on the basis that the group of patients assessed before treatment with GnRHa are younger than the group of patients assessed after treatment. But of course, any pre-GnRHa group will be younger than a post-GnRHa group since GnRHa treatment is started in early puberty and discontinued in later adolescence. Again, Dr. Hruz ignores the inherent limitations of conducting research in clinical medicine.

II. Sex, Gender Identity, and Gender Dysphoria

26. Dr. Biggs's review of the natural history of gender identity differences in children and adolescents is inaccurate (Biggs Rep. ¶ 13). As reviewed in my report (Shumer Rep. ¶ 60), it appears true that the majority of prepubertal *gender diverse* children who are exploring their gender do not develop gender dysphoria and are not expected to become transgender adolescents or adults. But not all *gender diverse* children are *transgender* children. As Dr. Biggs points out, some of these young individuals may have same-sex attraction. They also may simply be gender nonconforming. In contrast, however, children whose gender dysphoria persists into adolescence are highly likely to be transgender (van der Loos, et al., 2022). Dr. Biggs is misinterpreting older studies showing that a large percentage of children diagnosed with gender identity disorder did not grow up to be transgender (e.g., GAPMS Memo at 14; Attachment D (Cantor) to GAPMS Memo at 6-9). Those studies include children who would not fulfill the current diagnostic criteria for

gender dysphoria and, in any case, have no relevance to this case because no medications are prescribed to prepubertal children.

27. Dr. Biggs alludes to the higher rate of autism spectrum disorder (ASD) among children presenting for care at adolescent gender clinics (Shumer et al., (2016); Strang et al., (2018)), apparently suggesting ASD as a cause of gender dysphoria. Dr. Biggs claims that children "on the autistic spectrum are more likely to face difficulties fitting in with same-sex peers, which makes a transgender identity obviously appealing as both an explanation and a solution" (Biggs Rep. ¶14). This is a conjecture by Dr. Biggs, a sociologist, that is not supported by any evidence. It also ignores more plausible explanations. For example, children with ASD may be less aware of social bias or social expectations and therefore be less worried about how others may react to their transition, increasing the likelihood of coming out (Strang, et al., 2016). In any event, there is no research suggesting that treatment for ASD alleviates symptoms of gender dysphoria. Thus, any relationship between the two conditions is irrelevant for the purposes of determining what treatment is effective for gender dysphoria.

28. Similarly, in describing sex and gender, Dr. Hruz completely ignores the role of gender identity (Hruz Rep. ¶¶ 13-21; *see also* Lappert Rep. ¶¶ 31-32). Despite his assertion that sex is not "assigned at birth," it is a fact that the majority

of infants leave the hospital classified as either male or female based on the appearance of their sexual anatomy. Whether or not this assignment or classification will match future gender identity is uncertain. While Dr. Hruz acknowledges that when the sexual anatomy is ambiguous, other elements of sex including chromosomes, hormones, and internal organs can be evaluated to better understand the infant's sex, he fails to recognize that gender identity is another component of sex with biological underpinnings (*see* Shumer Rep. ¶¶ 29-33). Thus, Dr. Hruz correctly explains that in cases of ambiguity, "current practice is to defer sex assignment until the etiology of the disorder is determined and, if possible, a reliable prediction can be made on likely biological and psychologic outcomes" (Hruz Rep. ¶ 18). What does Dr. Hruz mean by this? My interpretation is that when there is discordance is some elements of sex (anatomic, hormonal, chromosomal), the best practice is to delay sex assignment until we feel we can choose a sex for the infant with the highest likelihood of promoting a happy and healthy life, which includes attempting to match sex assignment with future gender identity. Implicit in his statement is that sometimes we are wrong; the sex assigned at birth does not match future gender identity. In children with differences in sex development (DSD), one may then state, "we tried our best to assign sex, but we were wrong; now that the child can express themselves, the other sex assignment would have been correct."

When considering children with gender dysphoria *not* born with a DSD, this same statement would be appropriate. The difference is that there were no clues at birth alerting us to discordant elements of sex. Herein lies the reason that I so thoroughly outlined the biological underpinnings of gender identity (see Shumer Rep. ¶¶ 29-33): while not as obvious as in cases of DSD, a transgender person's sex assigned at birth was equally not correct.

29. Dr. Lappert discounts gender identity on the basis that there is not an "objective, repeatable test, with known error rates, that can be used to detect gender" (Lappert Rep. ¶ 32). There actually is a test which can be used to discover someone's gender identity: simply ask them. It is a human characteristic that is ascertained through a conversation rather than a lab test. Gender identity is a real human characteristic, and it is rooted in biology.

30. Further, I do not agree that providers providing this care (me included) feel compelled to *adopt a patient's self-diagnosis* and feel that his characterization of the evidence-base supporting gender-affirming care is a gross mischaracterization (Hruz Rep. ¶ 91). As a pediatric endocrinologist, when assessing any patient for any condition, my job is to analyze all available information, determine an appropriate diagnosis, and then discuss potential treatment options with patients and parents. This is true regardless of whether I am seeing the patient for gender concerns, slow

growth, thyroid disease, or diabetes. Patients seen in gender clinic who do not have gender dysphoria are not treated with hormonal interventions. Patients who feel that their thyroid is "off" but have normal thyroid function, are not treated with thyroid hormone. Patient "self-diagnosis" has not replaced competent assessment and diagnosis in this field or any other. Rather, providers of gender-affirming care rely upon the well-established and evidence-based standards of care for assessment, diagnosis and management of gender dysphoria.

31. Dr. Laidlaw and Dr. Hruz suggest that because gender dysphoria is in some ways different than other endocrine conditions that they are more comfortable treating, it should not be treated with medication (Laidlaw Rep. ¶¶ 14-27; Hruz Rep. ¶¶ 34, 54). They argue that most endocrine disorders involve hormones made in excess, hormone deficiencies, or structural abnormalities of endocrine glands. Whether or not Dr. Laidlaw and Dr. Hruz would like to classify gender dysphoria as an endocrine condition, several pertinent facts remain clear. First, there is ample scientific evidence that gender identity has a strong biological foundation (Shumer Rep. ¶¶ 29-33). Second, endocrinologists are uniquely suited to treat gender dysphoria due to familiarity with prescribing and monitoring medications such as GnRHa, testosterone, and estrogen. Third, countless medical conditions are diagnosed with clinical observation and questioning rather than with a laboratory

test, an imaging test, or examination of cells under a microscope (e.g., migraines, neuropathic pain, Alzheimer's disease, irritable bowel syndrome, fibromyalgia), but are no less actual medical diagnoses which improve with medical interventions. Fourth, the American Board of Internal Medicine requires knowledge of gender dysphoria and its management in order to become certified as an Endocrinologist (American Board of Internal Medicine, 2023). Ultimately, while I disagree with Dr. Laidlaw's discomfort with classification of gender dysphoria as an endocrine disorder, this debate is mere semantics and not pertinent to the appropriate assessment and management of the condition.

32. Dr. Hruz states that the goal of endocrinology is to restore health (Hruz Rep. ¶ 50). I would offer that this is a goal not only in endocrinology but all of medicine. In my experience and in review of the literature, when I prescribe gender-affirming care, consistent with the Endocrine Society's clinical practice guidelines and WPATH SOC, I am helping to restore health to my patient.

33. Dr. Laidlaw correctly points out that the number of young people being referred to the Gender Identity Development Service in the UK has increased significantly over time (Laidlaw Rep. ¶ 29; *see also* Levine Rep. ¶ 94) but wrongly attributes this increase to "social contagion" and "social media/internet use." I would suggest an alternative explanation that is not only more likely, but also supported by

research. As transgender individuals face less cultural stigma than in previous generations, young people understand that they will be supported and valued by their family and community and are more likely to explore and discuss gender identity openly (Zhang, et al., 2020). Two unrelated examples may make this concept more understandable. First, it should come as no surprise that the rate of openly gay individuals is lower in countries that criminalize homosexuality. Would you suppose that it is more likely that citizens of country X, which criminalizes homosexuality, has very few openly gay citizens because there is naturally a very low rate of homosexuality in that country, or because gay citizens fear retribution for coming out as gay? Second, in many societies left-handed people have been historically encouraged as children to use their right hands for writing and other fine-motor skills. However, in the late 20th century, left-handedness became less stigmatized and the percentage of left-handed people rose from about 4 percent in 1920 to 12 percent in 1980, roughly the same percentage as today (McManus 2009).

34. Dr. Hruz, Dr. Levine, and Dr. Laidlaw also claim that the disproportionate increase in transmasculine adolescents means that gender identity is not biological, but social (Hruz Rep. ¶¶ 116-118; Laidlaw Rep. ¶ 88; Levine Rep. ¶ 95). While they point to research by Dr. Lisa Littman, that research has been heavily criticized, and her conclusions have been called into question (Restar, 2020).

What is more, their logic is flawed. There is no reason why we would necessarily expect the rates of transmasculine people and transfeminine people to be equal. And, it would make sense for those rates to change over time as cultural attitudes towards transmasculine people and transfeminine people change. So long as a cultural bias against transgender people remains, we might not know the true prevalence of transmasculine and transfeminine people. In addition, they ignore that adolescence is a common time for transmasculine people to present to care due to the onset of breast development and menstruation. Transfeminine patients present more commonly younger or older than the mid-adolescent phase which may be in part due to the extreme difficulty for transfeminine adolescents to be accepted and supported by peers (Urquhart, 2017). Furthermore, while they focus on transmasculine adolescents, they ignore that transgender girls and older transgender men are also coming out at higher rates than previously reported (James, 2016; Coleman, 2022).

III. Desistance

35. Dr. Laidlaw and Dr. Hruz assert that rates of "desistance" are very high and therefore treatments as outlined by current standards of care will cause serious and irreversible harm to children and adolescents (Laidlaw Rep. ¶¶ 32-35; Hruz Rep. ¶¶ 63-64). This fallacy, repeated by many opponents of gender-affirming care, misrepresents the data completely. As outlined in my report (Shumer Rep. ¶ 59), it

is true that the majority of prepubertal gender diverse children exploring their gender do not develop gender dysphoria and are not expected to become transgender adolescents or adults, but that is because they are not transgender in the first place. First, as noted above, the studies included gay children and gender nonconforming children who were never transgender. Second, while Dr. Laidlaw cites data from studies of children across wide age groups, age 3-13 in one instance, he does not attempt parse out important clinical information such as the age and pubertal stage of so-called "desisters" in these studies. Lastly, because prepubertal children are not treated with hormonal medications for gender dysphoria, studies that look at prepubertal children, such as those Dr. Laidlaw has cited, have no relevance to the question of how to treat adolescents. Dr. Laidlaw again ignores the fact that children whose gender dysphoria persists into adolescence are highly likely to be transgender (van der Loos, et al., 2022).

36. Dr. Laidlaw also wrongly suggests that the use of pubertal suppression alters the natural course of "desistance," whereby patients prescribed pubertal suppression are very likely to be prescribed gender-affirming hormones later in adolescence (Laidlaw Rep. ¶¶ 111-112). Here Dr. Laidlaw is making a causal theory error – making a claim of causation based on correlational evidence. Children with persisting gender dysphoria into puberty (1) are very likely to have persisting gender

dysphoria into adulthood, and (2) are eligible for treatment with GnRHa. The use of GnRHa is not actually influencing future gender identity. In other words, the fact that patients prescribed pubertal suppression are very likely to later be prescribed gender-affirming hormones simply indicates that clinicians are correctly identifying patients who have gender dysphoria and benefit from medical intervention.

37. Dr. Hruz describes three "approaches" for treating children with gender dysphoria: "reparative therapy," "watchful waiting," and "gender affirming" (Hruz Rep. ¶¶ 60-87). As outlined in my report, gender exploration in childhood is expected and healthy (Shumer Rep. ¶ 60). Of course, parents of a child exploring their gender identity should not push the child to become transgender – this makes no sense and would work just as poorly as parents pushing their children to identify with their sex assigned at birth (referred to as reparative therapy by Dr. Hruz), which is both ineffective and harmful (Shumer Rep. ¶ 28). Unlike the gender-affirming approach, which is well supported by research and the experience of clinicians, including my own, there is no evidence to support the "watchful waiting" approach Dr. Hruz describes, which is not the same as the "watchful waiting" model adopted by the Dutch.

38. Moreover, it is not clear how this approach is supposed to work as a practical matter. When a child asks their parents to use a different name or pronouns,

the parent can either reject this request or accept this request, there is no "neutral" response, as Dr. Hruz suggests. If honoring a child's chosen name and pronouns is gender-affirming and rejecting the request is reparative therapy, what does the watchful waiter do? If asked, I would suggest that parents allow their child to safely explore gender identity, making it clear that whatever the future outcome, the child will receive unconditional love, support, and respect. If using a different name or pronoun would be helpful in the process of gender exploration, parents should consider honoring that request.

39. Furthermore, at the start of puberty, a child with persistent and/or intensifying gender dysphoria is much more likely to be transgender (van der Loos, et al., 2022) and will begin to exhibit secondary sex characteristics. Watchful waiting in this situation is no longer neutral. In these situations, it appears Dr. Hruz would categorize the use of GnRHa as part of the "gender affirming" approach in his three-approach schema, and no medical intervention as "watchful waiting." However, the use of GnRHa in children exploring their gender identity was first described by Delemarre-van de Waal and Cohen-Kettenis (2006) as a reversible intervention allowing for delayed decision-making regarding hormone therapy, a strategy more consistent with the "watchful waiting" concept.

40. I do not agree with Dr. Hruz that providers of gender-affirming care are presuming that development of *natural sex characteristics interfere with the exploration of gender identity* as an impetus to offer GnRHa (Hruz Rep. ¶ 71). Rather, GnRHa can prevent intensification of dysphoria during puberty while enhancing the future ability of the patient to present to the world in a gender congruent manner.

41. I agree with Dr. Hruz that providers should use caution when "interfering with the normal process of maturation" (Hruz Rep. ¶ 72). In my experience, providers in this field *are* using caution when prescribing GnRHa and gender-affirming hormones in adolescence, weighing potential benefits against potential risks with each individual patient, in candid communication with parents, and with the best intentions for the wellbeing of the adolescent in question.

IV. Side Effects of Puberty Suppression and Hormone Treatment

42. The report correctly defines a medical condition, *hypogonadotropic hypogonadism*, as a condition in which the pituitary fails to send signals to the gonads (Laidlaw Rep. ¶ 79). Dr. Laidlaw then describes gender affirmative therapy (specifically, pubertal suppression) as deliberately causing the medical condition *hypogonadotropic hypogonadism* and then, based on a limited review of some of the plaintiffs' medical records, declares that the plaintiffs have developed

hypogonadotropic hypogonadism as a result of their medical care (Laidlaw Rep. ¶¶ 87-91). In his report, Dr. Laidlaw described the use of GnRHa in treatment of prostate cancer and precocious puberty. Interestingly, he did not frame GnRHa as causing the medical condition *hypogonadotropic hypogonadism* in those patients but described the use of GnRHa as effective treatment for these other conditions. He ignores that GnRHa is also an effective treatment for gender dysphoria. Conflating the goal of therapy (suppression of sex hormone production) with causing a medical condition (*hypogonadotropic hypogonadism*) in one instance, but not others, is inappropriate if not disingenuous.

43. Dr. Laidlaw repeats this same wordplay tactic in describing the administration of testosterone as inducing *hyperandrogenism* in transgender men (Laidlaw Rep. ¶¶ 124-148), and administration of estrogen as inducing *hyperestrogenism* in transgender women (Laidlaw Rep. ¶¶ 150-159). He describes the use of testosterone to treat gender dysphoria in transgender male plaintiffs as inducing *hyperandrogenism* and speculates that one of the plaintiffs, a transgender girl, is at risk for *hyperestrogenemia* if she requires estrogen treatment in the future (Laidlaw Rep. ¶¶ 87-91).

44. In reality, when testosterone is prescribed for gender dysphoria as for the transgender male plaintiffs, the goal is to achieve a normal male testosterone

level based on age, meaning a testosterone level that is consistent with the normal testosterone levels for cisgender males of similar age; when estrogen is prescribed for gender dysphoria as it is for transgender females, the goal is to achieve a normal female estrogen level based on age, meaning an estrogen level that is consistent with the normal estrogen levels for cisgender females of similar age. These goals mirror what Dr. Laidlaw or any other endocrinologist would aim for when treating low testosterone or ovarian failure (Laidlaw ¶¶ 115, 149).

45. Dr. Laidlaw frames evidence-based treatments for gender dysphoria as causing medical conditions, rather than acknowledging the similarity in how these medications are used in different contexts. The underlying premise of Dr. Laidlaw's opinions seems to be that gender dysphoria is not a legitimate diagnosis worthy of any medical treatment and that there should not be any transgender people.

46. Dr. Laidlaw also misconstrues the effect of GnRHa on fertility. As outlined in my prior report, GnRHa treatments do not have long-term implications on fertility (Shumer ¶ 79). Dr. Laidlaw correctly explains that giving GnRHa to a four-year-old with precocious puberty will not impair fertility. Likewise, GnRHa will also have no effect on fertility when used in older transgender adolescents.

47. It may seem that Dr. Laidlaw is claiming that GnRHa cause infertility, but he is not; he is merely pointing out that progression through puberty – at some

point – is needed for maturation of sperm and eggs. Dr. Laidlaw posits that gender-affirming hormones could possibly damage immature gonads without providing supportive data. So long as gonads remain in place, there remains fertility potential. To be sure, this would require progression through the puberty associated with the sex assigned at birth.

48. In the context of gender-affirming care, concerns about fertility are discussed with adolescent patients and their families when receiving both GnRHa as treatment and/or gender-affirming hormones. Indeed, SOC 8 recommends that "health care professionals working with transgender and gender diverse adolescents requesting gender-affirming medical or surgical treatments inform them, prior to initiating treatment, of the reproductive effects including the potential loss of fertility and available options to preserve fertility within the context of the youth's stage of pubertal development." (Coleman, et al., 2022).

49. What is more, for transgender adolescents taking GnRHa and for whom hormones appears to be indicated as treatment, it is fairly common for fertility preservation to occur after a brief cessation of GnRHa treatment but before hormones. For example, case reports, including one from Dr. Hruz's own institution, illustrate the success of this approach in fertility preservation. (Martin, et al., 2021; Rothenberg, et al., 2019).

50. Even if gender-affirming hormones were introduced following use of GnRHa, these hormones could be discontinued with a goal of progression through internal puberty and achieving fertility. Withdrawal of hormones in adulthood often is successful in achieving fertility when it is desired (Light, et al., 2014; Knudson, et al., 2017). Dr. Hruz is skeptical that a patient who received GnRHa followed by hormones would have any fertility potential (Hruz ¶ 86). While this would likely require discontinuation of all medication and progression through puberty, there has been a study aiming to investigate this question. Caanen et al demonstrated that transgender men have similar ovarian morphology to cisgender women, even when treated with GnRHa followed by testosterone. These treatments did not cause the ovarian changes which are seen in hyperandrogenic women with polycystic ovarian syndrome and infertility (Caanen, 2017). This lends credence to the expectation that the sequence of GnRHa to testosterone does not cause permanent infertility.

51. Dr. Laidlaw also raises concerns about future sexual function in patients prescribed GnRHa (Laidlaw ¶¶ 98-99). In my experience, it is essential to have open, age-appropriate discussions around sex and sexuality while respecting that all persons, including transgender people, are diverse in terms of sexual orientation and desires. Sexuality and sexual function should be considered and maximized as

transgender patients reach adulthood. However, it should not be underestimated how a positive body image is also associated with better sexual function and satisfaction (Nikkelen, 2018). Additionally, research clearly shows that persons with untreated gender dysphoria may have significant challenges with sexuality and sexual function (Holmberg, 2019).

52. Dr. Laidlaw's concerns about bone density in patients prescribed GnRHa are likewise overblown, if not wholly unfounded (Laidlaw Rep. ¶¶ 100-109; *see also* Hruz Rep. ¶ 87). It is accurate to state that pubertal hormones (either testosterone or estrogen) contribute to bone density accrual. A person who never was exposed to any sex hormones for their entire life would be at high risk of osteoporosis. It is not surprising that the Carmichael study referenced (Laidlaw Rep. ¶ 104) found that there is a reduction in Z-scores in adolescents on GnRHa aged 12-15 during the time of treatment when compared to age-matched controls. What is misleading, however, is that these patients will be transitioned off GnRHa when a decision is made regarding treatment with gender-affirming hormones or to resume puberty consistent with their birth-assigned sex. After exposure to sex hormones, bone density accrual will rise. In practice, risk of lower bone mineral density is mitigated by screening for, and treating, vitamin D deficiency when present, and by

limiting the number of years of treatment based on a patient's clinical course (Rosenthal, 2014).

53. Dr. Scott and Dr. Laidlaw raise a hypothetical concern regarding brain development, suggesting that somehow use of GnRHa has "unknown, but likely negative consequences … with respect to brain development" (Laidlaw Rep. ¶ 110; *see also* Scott Rep. ¶ 15). I have heard this argument from opponents of GnRHa use before but have difficulty understanding its basis. For example, when considering children with naturally occurring delayed puberty, I find no published evidence of negative consequences to brain development compared with children with normally timed puberty. Likewise, Dr. Laidlaw can point to no published evidence in support of this concern in transgender adolescents prescribed GnRHa.

54. As for Dr. Scott, she describes how the brain changes over time, but no description about how pubertal hormones play a role in those changes. Her inclusion of data reviewing GnRHa data in sheep and in girls with precocious puberty have questionable applicability to gender care (Scott Rep. ¶ 15). The other article she cites found, "GnRHa treated girls do not differ in their cognitive functioning … from the same age peers" (Wojniusz et al., 2016). The authors of this article came to this conclusion because there was not a statistically significant difference in IQ, memory, mental rotation, cognitive executive function, processing speed, attention or

executive function in participants treated with GnRHa for precocious puberty. This suggests that Dr. Scott's concerns about GnRHa and brain development are unfounded.

55. Dr. Laidlaw also misrepresents the risks of using the hormone *testosterone* to treat gender dysphoria (Laidlaw Rep. ¶¶ 114-122). He correctly explains that when treating men with testosterone deficiency, the dose of testosterone must be carefully considered and monitored to avoid excess levels (Laidlaw Rep. ¶ 115). This is equally true when using testosterone for treatment of gender dysphoria. He mentions that some individuals abuse testosterone by taking more than prescribed, but it is unclear if he is implying that transgender men would be more likely to do this than others, which I would not expect and find no data to support. All of the adverse effects of excessive testosterone that Dr. Laidlaw avoids by carefully monitoring his patients with low testosterone (e.g., increased libido, headache, erythrocytosis) are similarly avoided by careful monitoring in transgender men.

56. Dr. Laidlaw also appears to argue that transgender men can develop erythrocytosis (elevation in the red blood cell measurement, hematocrit) while being treated with testosterone (Laidlaw Rep. ¶ 148). Dr. Laidlaw is using the female reference range for hematocrit to make this assertion, again considering these

patients as females with *hyperandrogenism* rather than transgender men receiving evidence-based care for gender dysphoria. This is inappropriate; the male reference range for hematocrit should be used for patients on testosterone treatment (Deutsch, 2016).

57. Similarly, Dr. Hruz suggests that testosterone administration to a person assigned male at birth may have different effects than when given to a person assigned female at birth since there are thousands of sex-differentially expressed genes (Hruz Rep. ¶ 82). While this speculation could be potentially true, Dr. Hruz does not provide a clinical example of how this could be of concern, and I am not aware of any research confirming his suggestion.

58. Dr. Laidlaw makes parallel arguments regarding estrogen (Laidlaw ¶¶149-159) by pointing out the elevated estrogen can be associated with health problems, while ignoring that the goal of treatment with estrogen in gender dysphoria is maintenance of estrogen levels in the normal female range. Risk for the health concerns he highlights are avoided by careful monitoring in transgender women.

59. He states that the risk for breast cancer increases when a "male" is treated with "high dose estrogen" (Laidlaw Rep. ¶ 157). This misunderstands the risks. It is of course not surprising that transgender women with breasts are at higher

risk for breast cancer than men without breasts. What Dr. Laidlaw leaves out of his discussion is the complete findings of the Christel article referenced. That article found that despite an increased risk of breast cancer in transgender women compared with cisgender men, there was a lower risk when comparted to cisgender women. The article concluded that "breast cancer screening guidelines for cisgender people are sufficient for transgender people using hormone treatment" (Christel, 2019).

60. Drs. Laidlaw and Hruz argue that risks of gender-affirming care outweigh the benefits. They are incorrect; they have provided a grossly exaggerated and erroneous description of risk while completely discounting the benefits of treatment or the risks of withholding treatment.

V. Informed Consent

61. Dr. Laidlaw argues that it is not possible for parents to make a truly informed consent decision regarding gender-affirming care, and suggests, without reasoning or evidence, that this decision is somehow different than other complex medical decisions that parents and guardians make regarding the health and wellness of their children every day. In my experience as a pediatrician working with children and families every day, Dr. Laidlaw is severely underestimating the capacity of parents and guardians to understand and balance information pertaining to the health of their children. He also ignores that WPATH SOC 8 clearly outlines criteria for

how providers obtain assent and consent for medical intervention (Coleman, et al., 2022).

62. For his part, Dr. Hruz implies that providing care to transgender patients using the standards of care violates principal tenants of medicine; he believes this because he considers these treatments "experimental," and as a result, patients and their parents cannot provide informed consent (Hruz Rep. ¶¶ 98, 105). However, as detailed in my initial report and reiterated above, gender-affirming care is not experimental – it is based on significant scientific research and clinical experience and is supported by every major medical association in the country. As a provider of gender-affirming care, it is my opinion that *withholding* gender-affirming care would violate the basic tenants of medicine. Dr. Levine makes a similar point about the Hippocratic Oath (Levine Rep. ¶ 87). Again, this oath has guided me and my colleagues to provide gender-affirming care when appropriate, weighing the risk of treatment against the harm of not treating.

63. Dr. Hruz further claims that parents cannot provide informed consent because providers often threaten parents that "failure to allow a gender dysphoric child to medically transition will result in suicide" (Hruz Rep. ¶ 106). Dr. Hruz provides no support for this assertion, and I personally have never considered making this kind of statement to patients or their families; this is not common

practice nor is it suggested in the SOC. In contrast, consistent with the SOC, I am always clear with patients and parents that I consider every perspective in the room valid, based on love, and rooted in the intention to make the best decision for the health of the adolescent. Any complete assessment of an adolescent's gender identity includes vital information from parents, who have much more knowledge of their child than their health care providers could ever have. Most often, careful exploration of the desires, fears, questions, and concerns of both patients and parents leads to better understanding and improves collaboration and the ability to make sound medical decisions together.

64. Raising similar concerns to Dr. Hruz, Dr. Scott believes that gender-affirming care may be appropriate in children and adolescents but is concerned about how to identify appropriate candidates for this care (Scott Rep. ¶ 7). Fortunately, assessment by highly competent mental health professionals is a cornerstone of the current standards of care in adolescent gender medicine and helps to identify appropriate candidates for medical treatments.

65. That said, Dr. Hruz's assertion that rates of suicidal ideation and attempt in transgender adolescents are similar to those found in adolescents without gender identity is incorrect and wildly disconnected from the literature.

Unfortunately, the rates among transgender adolescents are significantly elevated (Reisner, et al., 2015).

66. Dr. Hruz then references Dr. Levine (another designated expert for the defendants) in stating that informed consent in this context fails with respect to discussion of the natural history of gender dysphoria in adolescents, the quality of evidence regarding gender-affirming care, and the handling of the question of suicidality (Hruz Rep. ¶108). In my own practice, consistent with the SOC, I am careful to review the evidence as outlined in my report with patients and families and reject the claim that the consent process is limited by "erroneous professional assumptions" or "poor quality of the initial evaluations".

The following section of this rebuttal report (Section VI – Dr. Laidlaw's Opinions Regarding Plaintiffs) is designated as CONFIDENTIAL pursuant to the Protective Order in this matter (ECF No. 77).

VI. Dr. Laidlaw's Opinions Regarding the Plaintiffs

67. Dr. Laidlaw claims that plaintiffs K.F. (Laidlaw Rep. ¶¶ 230-250), Brit Rothstein (Laidlaw Rep. ¶¶ 251-270), S.D. (Laidlaw Rep. ¶¶ 271-293), and August Dekker (Laidlaw Rep. ¶¶294-305) should not be receiving gender-affirming care.

68. I have not spoken with the plaintiffs, the parents of the minor plaintiffs, or their providers. However, as noted above, I have reviewed their medical records, and based on that review, I disagree with Dr. Laidlaw's conclusions about the plaintiffs' treatments. First, his opinions about the plaintiffs rest on his belief that nobody should be prescribed GnRHa or hormones to treat gender dysphoria. Second, his criticisms of the specific care the individual plaintiffs received are unfounded.

69. Based on the available medical records, I do not have any medical concerns regarding the gender-affirming care received by K.F. In fact, I would posit that K.F.'s mental health would deteriorate precipitously if he were unable to continue to receive this care. In review of the clinical course of K.F., he appears to have had clear and consistent male gender identity since at least the age of 6. The decision to make a social transition appears to have been discussed by a mental health professional as is recommended in the Endocrine Society Guidelines

(Hembree, 2017). He happens to have been seen at Boston Children's Hospital in September 2015, just after the completion of my training at this institution. All patients seen at this clinic are assessed by a member of the mental health team. And, regardless of how Dr. Laidlaw may personally feel about nurse practitioners (Laidlaw Rep. ¶¶ 235-236), NPs – including Sara Pilcher, who I personally worked alongside in Boston – are qualified to and provide excellent, thoughtful and evidence-based care under supervision of physicians in pediatric endocrinology and all other fields of medicine.

70. Dr. Laidlaw is looking for signed documentation of discussions regarding risks and benefits of treatment, suggesting that if there is no signed document these conversations must not have occurred. However, it is not my practice, nor to my knowledge a common practice, to have parents sign documents related to medical conversations that take place in gender clinics or other pediatric endocrinology clinics for the treatment of gender dysphoria or other conditions. Dr. Laidlaw attempts to conflate diagnoses of anxiety and ADHD as evidence of deteriorating mental health as a result of gender-affirming therapy. Whether K.F. has or does not have anxiety or ADHD has no bearing on whether the gender-affirming care received is providing benefit for gender dysphoria, a separate medical problem.

71. I disagree with Dr. Laidlaw's review of Brit Rothstein; I do not have concerns about his care and would suggest Mr. Rothstein's mental health would deteriorate if unable to receive gender-affirming care. Dr. Laidlaw's review of Brit Rothstein is notably flawed in a few respects. Similar to his incredulity that nurse practitioners can provide high quality evidence-based care, Dr. Laidlaw seems to believe that the only mental health professionals able to competently work with transgender adolescents are psychologists or psychiatrists (Laidlaw Rep. ¶¶ 254-255). In fact, therapists and social workers not only are trained and licensed to do this work, but in my experience are often more effective due to their ability to see patients regularly and build understanding and rapport. At the Child and Adolescent Gender Clinic at Michigan Medicine, where I serve as Clinical Director, our mental health team consists of two social workers and a child and adolescent psychiatrist. The social workers perform comprehensive biopsychosocial assessments for all new patients, and the child and adolescent psychiatrist sees only a fraction of our patients who have additional psychiatric needs.

72. Dr. Laidlaw points out that Mr. Rothstein is more medically complex than other patients with gender dysphoria (Laidlaw Rep. ¶¶ 259-261). Certainly, a careful review of a patient's medical history is important prior to starting gender-affirming care. But none of Mr. Rothstein's co-occurring conditions contraindicate

the gender-affirming care he has received. Indeed, medically complex patients do present to gender clinics and may benefit from hormonal interventions; comorbid medical problems should not and do not preclude gender-affirming treatment. My review of Mr. Rothstein's records reveals that his providers have been carefully considering his other medical problems and monitoring them in order to help him transition safely.

73. Dr. Laidlaw opines that Mr. Rothstein has developed erythrocytosis (elevation in the red blood cell measurement, hematocrit) while being treated with testosterone (Laidlaw Rep. ¶ 148). But as noted above, Dr. Laidlaw is using the female reference range for hematocrit to make this assertion, which is inappropriate, because the male reference range for hematocrit should be used for patients on testosterone treatment. Indeed, Mr. Rothstein's most recently documented hematocrit was within the appropriate range for a male.

74. I also disagree with Dr. Laidlaw's review of S.D. who has received appropriate care and would likely have deterioration in health if this care were discontinued. In S.D.'s case Dr. Laidlaw again takes note of the fact that Dr. Linda Ouellet and Rebecca Thipsingh are therapists - trained and licensed mental health professionals. As I stated above, this is neither unusual nor inappropriate. He takes particular umbrage with the parents' decision to help S.D. make a social transition,

stating that this decision "had the iatrogenic effect of preventing the natural course of desistance which would occur in the majority of children" (Laidlaw Rep. ¶ 276). This is incorrect, wildly speculative, and unfounded. Social transition does not significantly impact the natural course of a prepubertal child's gender identity. That more children who make a social transition maintain a transgender identity into adolescence can be clearly explained by the fact that children with stronger and more intense identification are both: (1) more likely to make a social transition; and (2) more likely to continue to identify this way as adolescents. Here, Dr. Laidlaw is making the same causal theory error that he made previously when suggesting that GnRHa actually influences future gender identity (see para 5, above).

75. Dr. Laidlaw says he is concerned that S.D. and her mother have unrealistic expectations about gender-affirming care based on the statement that "there is nothing worse in S.D.'s mind than male puberty" (Laidlaw Rep. ¶ 280). But that statement makes complete sense given that S.D. identifies as a girl. What girl wouldn't describe the prospect of going through male puberty as a nightmare? Dr. Laidlaw suggest that "it is common for parents and children influenced by GAT practitioners to believe that a child can go through puberty of the opposite sex. However, they have been misinformed as this is not possible" (Laidlaw Rep. ¶ 280). Dr. Laidlaw insults not only the competence of practitioners to provide complex

information, but more glaringly the intelligence of patients and parents. Patients and parents do not expect testicles to become ovaries. In S.D.'s case, if estrogen is prescribed in the future, she would develop secondary sex characteristics consistent with other girls. Whether or not Dr. Laidlaw refuses to call this "female puberty" is of no practical consequence.

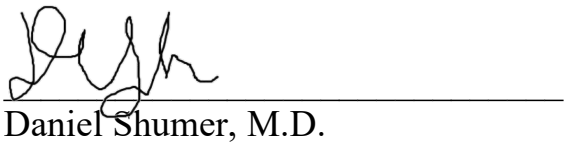
76. I also have no concerns about the care received by August Dekker, disagree with Dr. Laidlaw's review of his care, and feel that he would be at high risk for negative health outcomes if his care were discontinued. In discussing the case of Mr. Dekker (Laidlaw Rep. ¶¶ 294-305), Dr. Laidlaw, an endocrinologist, describes a sort of forensic investigation he performed related to August's mental health professional. While I cannot comment on the status of Abbie Rolf's license, I can state that (1) membership in WPATH is certainly not a reason to reject the assessment of a mental health professional; (2) Dr. Laidlaw's re-assertion that only psychiatrists and psychologists are capable of assessment of gender identity is inappropriate and condescending to mental health professionals; and (3) there is no reason that a mental health professional should require multiple visits with an adult transgender man requiring chest surgery if it becomes clear that he meets criteria for this surgery after a single visit. Dr. Laidlaw is also concerned as to whether Planned Parenthood has an endocrinologist on staff. This is immaterial, as prescribing

testosterone is not restricted to endocrinologists, and it is common and appropriate for practitioners from various disciplines to provide hormone treatment. In my home institution, adult transgender men receive their hormonal care from extremely well-trained and competent providers in a variety of medical disciplines including gynecology, family medicine, internal medicine, urology, and also endocrinology.

This marks the end of CONFIDENTIAL section of this rebuttal report.

I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Executed this 10th day of March 2023.



Daniel Shumer, M.D.

Exhibit C

Supplemental Bibliography

SUPPLEMENTAL BIBLIOGRAPHY

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EXHIBIT E

Debbie Hayton @DebbieHayton · Oct 11, 2018

Meanwhile, vulnerable children are at risk. Books at infants suggest that if a girl likes doing “boy” things then she might be a boy. Present and future female scientists take note!

(tagging in @sophiescott) [12/19]

thepinknews.com

Incredible new children's book tells story of transgender boy

"Jackie doesn't like dresses or her long hair, and she would rather be called Jack."

2 13 55

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@sophiescott

The book says that girls who like bugs and wear super hero cakes and who don’t like pink dresses, are in fact boys. I think that’s your cheap shot, right there.

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Oh god.

> **Bo** @Bozenka_2023 · Sep 20, 2016
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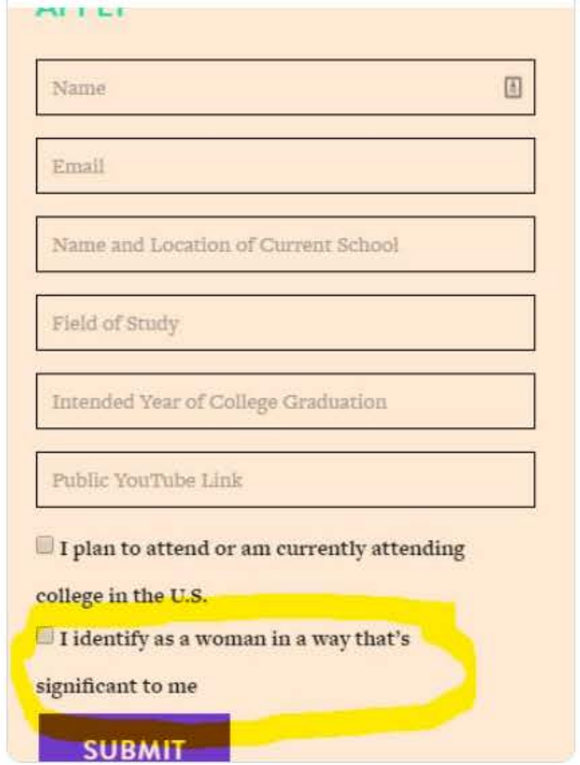

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Replying to @sophiescott
isn't this just an attempt to avoid discriminating against transgender women? (who face v. significant discrimination)

3

Prof Sophie Scott CBE @sophiescott · Sep 20, 2016
Replying to @olijrobinson

Relevant people

Prof Sophie Scott ... @sophiescott — Follow
Director @UCL_ICN. 0.5 of @neuromantics1. RI Xmas Lecturer 2017. Pointless Answer. Dig where you stand. Looking into the dark sea, seeing only brightness.

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