

IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA

B.P.J. by her next friend and mother, HEATHER JACKSON,

Plaintiff,

v.

WEST VIRGINIA STATE BOARD OF EDUCATION, HARRISON COUNTY BOARD OF EDUCATION, WEST VIRGINIA SECONDARY SCHOOL ACTIVITIES COMMISSION, W. CLAYTON BURCH in his official capacity as State Superintendent, DORA STUTLER in her official capacity as Harrison County Superintendent, and THE STATE OF WEST VIRGINIA,

Defendants,

and

LAINIEY ARMISTEAD,

Defendant-Intervenor.

Civil Action No. 2:21-cv-00316

Hon. Joseph R. Goodwin

DECLARATION OF SRUTI SWAMINATHAN

I, Sruti Swaminathan, pursuant to 28 U.S.C. § 1746, declare as follows:

1. I am an attorney at Lambda Legal and counsel of record for Plaintiff B.P.J., with her next friend and mother, Heather Jackson. The following is true of my own personal knowledge, and, if called as a witness, I would and could testify competently thereto.
2. I submit this declaration in support of Plaintiff B.P.J.'s memoranda of law in support of motion to exclude the expert testimony of Stephen B. Levine.
3. Attached to this declaration are true and correct copies of the documents listed in the table below.

Exhibit	Description
A	Decl. and Expert Report of Stephen B. Levine, M.D.
B	Dep. Tr. of Stephen B. Levine, M.D., March 30, 2022
C	Aditi Bhargava et al., <i>Considering Sex as a Biological Variable in Basic and Clinical Studies: An Endocrine Society Scientific Statement</i> . 42(3) Endocr Rev. 219-258 (2021).
D	National Institutes of Health, Office of Research on Women's Health. <i>How Sex and Gender Influence Health and Disease</i> , https://orwh.od.nih.gov/sites/orwh/files/docs/SexGenderInfographic_11x17_508.pdf .
E	National Institutes of Health, <i>Consideration of Sex as a Biological Variable in NIH-Funded Research</i> (2015), https://grants.nih.gov/grants/guide/notice-files/NOT-OD-15-102.html .
F	Cecilia Dhejne et al., <i>Long-Term Follow-Up of Transsexual Persons Undergoing Sex Reassignment Surgery: Cohort Study in Sweden</i> , 6 PLoS ONE e16885 (2011)
G	Rikke Simonsen et al., <i>Long-term Follow-up of Individuals Undergoing Sex Reassignment Surgery: Psychiatric Morbidity and Mortality</i> , 70 Nordic Journal of Psychiatry 241-247 (2015). doi: 10.3109/08039488.2015.1081405.
H	Dep. Tr. of Stephen B. Levine, M.D. in <i>Kadel v. Folwell</i> , September 10, 2021.
I	Dep. Tr. of Stephen B. Levine, M.D. in <i>Claire v. Florida Dep't of Mgmt Servs.</i> , December 21, 2020.

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

Executed on May 12, 2022

/s/ Sruti Swaminathan
Sruti Swaminathan

IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
CHARLESTON DIVISION

B.P.J. by her next friend and mother, HEATHER JACKSON,

Plaintiff,

v.

WEST VIRGINIA STATE BOARD OF
EDUCATION, HARRISON COUNTY BOARD
OF EDUCATION, WEST VIRGINIA
SECONDARY SCHOOL ACTIVITIES
COMMISSION, W. CLAYTON BURCH in his
official capacity as State Superintendent, DORA
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WEST VIRGINIA,

Defendants,

and

LAINY ARMISTEAD,

Defendant-Intervenor.

Civil Action No. 2:21-cv-00316

Hon. Joseph R. Goodwin

CERTIFICATE OF SERVICE

I, Loree Stark do hereby certify that on this 12th day of May, 2022, I electronically filed a true and exact copy of the foregoing document with the Clerk of Court and all parties using the CM/ECF System.

/s/ Loree Stark

Loree Stark

Exhibit A

**IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF WEST VIRGINIA
CHARLESTON DIVISION**

B.P.J., by her next friend and mother, HEATHER JACKSON,

Plaintiff,

vs.

WEST VIRGINIA STATE BOARD OF EDUCATION,
et al.,

Defendants,

and

LAINY ARMISTEAD,

Defendant-Intervenor.

Case No. 2:21-cv-00316

Hon. Joseph R. Goodwin

DECLARATION OF STEPHEN B. LEVINE, MD

I, Dr. Stephen B. Levine, pursuant to 28 U.S. Code § 1746, declare under penalty of perjury under the laws of the United States of America that the facts contained in my Expert Report of Stephen B. Levine, MD., in the Case of B.P.J. v. West Virginia State Board of Education, dated February 23, 2022 and attached hereto, are true and correct to the best of my knowledge and belief, and that the opinions expressed therein represent my own expert opinions.

Executed on February 23, 2022.

A handwritten signature in cursive script that reads "Stephen B. Levine MD". The signature is written in dark ink and is positioned above a horizontal line.

Stephen B. Levine, MD

Expert Report of

Stephen B. Levine, MD

In the case of B.P.J. vs. West Virginia State Board of Education.

February 23, 2022

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I. CREDENTIALS & SUMMARY

1. I am Clinical Professor of Psychiatry at Case Western Reserve University School of Medicine, and maintain an active private clinical practice. I received my MD from Case Western Reserve University in 1967, and completed a psychiatric residency at the University Hospitals of Cleveland in 1973. I became an Assistant Professor of Psychiatry at Case Western in 1973, became a Full Professor in 1985, and in 2021 was honored to be inducted into the Department of Psychiatry's "Hall of Fame."

2. Since July 1973, my specialties have included psychological problems and conditions relating to individuals' sexuality and sexual relations, therapies for sexual problems, and the relationship between love, intimate relationships, and wider mental health. In 2005, I received the Masters and Johnson Lifetime Achievement Award from the Society of Sex Therapy and Research. I am a Distinguished Life Fellow of the American Psychiatric Association.

3. I have served as a book and manuscript reviewer for numerous professional publications. I have been the Senior Editor of the first (2003), second (2010), and third (2016) editions of the *Handbook of Clinical Sexuality for Mental Health Professionals*. In addition to five previously solo-authored books for professionals, I have recently published *Psychotherapeutic Approaches to Sexual Problems* (2020). The book has a chapter titled "The Gender Revolution."

4. In total I have authored or co-authored over 180 journal articles and book chapters, 20 of which deal with the issue of gender dysphoria. I am an invited member of a Cochrane Collaboration subcommittee that is currently preparing a review of the scientific literature on the effectiveness of puberty blocking hormones and of cross-sex hormones for

gender dysphoria for adolescents. Cochrane Reviews are a well-respected cornerstone of evidence-based practice, comprising a systematic review that aims to identify, appraise, and synthesize all the empirical evidence that meets pre-specified eligibility criteria in response to a particular research question.

5. I first encountered a patient suffering what we would now call gender dysphoria in July 1973. In 1974, I founded the Case Western Reserve University Gender Identity Clinic, and have served as Co-Director of that clinic since that time. Across the years, our Clinic treated hundreds of patients who were experiencing a transgender identity. An occasional child was seen during this era. I was the primary psychiatric caregiver for several dozen of our patients and supervisor of the work of other therapists. I was an early member of the Harry Benjamin International Gender Dysphoria Association (later known as WPATH) and served as the Chairman of the committee that developed the 5th version of its Standards of Care. In 1993 the Gender Identity Clinic was renamed, moved to a new location, and became independent of Case Western Reserve University. I continue to serve as Co-Director.

6. In the course of my five decades of practice treating patients who suffered from gender dysphoria, I have at one time or another recommended or prescribed or supported social transition, cross-sex hormones, and surgery for particular patients, but only after extensive diagnostic and psychotherapeutic work.

7. In 2006, Judge Mark Wolf of the Eastern District of Massachusetts asked me to serve as an independent, court-appointed expert in a litigation involving the treatment of a transgender inmate within the Massachusetts prison system. In that litigation, the U.S. Court of Appeals for the First Circuit in a 2014 (En Banc) opinion cited and relied on my expert

testimony. I have been retained by the Massachusetts Department of Corrections as a consultant on the treatment of transgender inmates since 2007.

8. In 2019, I was qualified as an expert and testified concerning the diagnosis, understanding, developmental paths and outcomes, and therapeutic treatment of transgenderism and gender dysphoria, particularly as it relates to children, in the matter of *In the Interest of J.A.D.Y. and J.U.D.Y.*, Case No. DF-15-09887-S, 255th Judicial District, Dallas County, TX (the “*Younger* litigation”). I have provided expert testimony in other litigation as listed in my curriculum vitae. In 2019, I provided written expert testimony in the landmark case in the United Kingdom; *Bell v. The Tavistock and Portman NHS Foundation Trust*.

9. I am regularly requested to speak on the topic of gender dysphoria and have given countless presentations to academic conferences and Departments of Psychiatry around the country. In May of this year, I will be co-presenting a symposium on the management of adolescent-onset transgender identity at American Psychiatric Association’s Annual Meeting.

10. A fuller review of my professional experience, publications, and awards is provided in my curriculum vitae, a copy of which is attached hereto as Exhibit A.

11. I am being compensated for my time spent in connection with this case at a rate of \$400.00 per hour for consultation and \$500.00 per hour for time spent testifying.

12. I have reviewed the “Declaration and Expert Report of Deanna Adkins, MD,” dated January 21, 2022 (“Adkins”). In that declaration Dr. Adkins makes a variety of statements about gender dysphoria, therapies for gender dysphoria, and outcomes of therapies, which I believe to be inaccurate, or unsupported by scientific evidence. Dr. Adkins is a pediatric endocrinologist. I note with some concern that Dr. Adkins makes a number of sweeping and

purportedly scientific assertions but cites almost no peer-reviewed articles or studies that support her opinions.

13. Based on her declaration, Dr. Adkins' practice is focused on children and adolescents; her CV and declaration do not suggest substantial experience in working with adults or older young adults who are living in a transgender identity, or who suffer from gender dysphoria. (This diagnosis requires "clinically significant" distress.) The wider lifecycle view that derives from experience with these adults (and familiarity with the literature concerning them) provides an important cautionary perspective. The psychiatrist or psychologist treating a trans child or adolescent, of course seeks to make the young patient happy, but the overriding consideration is the creation of a happy, highly functional, mentally healthy person for the next 50 to 70 years of life. I refer to treatment that keeps this goal in view as the "life course" perspective.

14. Dr. Adkins' stated belief that the only way to avoid harm is affirmative care is just one of many questionable assumptions that lack firm scientific foundation. Others that frequently ride along with advocates' convictions about affirmative care include:

- a. A trans identity is immutable;
- b. Trans identities are primarily caused by biological forces;
- c. Gender identity and orientation are distinct stable dimensions of identity;
- d. There are no alternative treatments to affirmative care;
- e. Affirmative care lastingly improves mental health and social function;
- f. Affirmative care reduces the rates of suicidal ideation and suicide;
- g. Young teens can give informed consent for hormones because they know best what will make them happy now and in the future;

h. De-transition of affirmed youth is rare;

i. Associated psychopathology during and after affirmative care is primarily due to minority stress.

15. These assertions are inaccurate or unsupported, for reasons that I explain in this Declaration. I will provide citations to published, peer-reviewed articles that inform my judgments.

16. I have also reviewed the “Expert Report and Declaration of Joshua D. Safer, MD,” dated January 21, 2022 (“Safer”). In that declaration Dr. Safer similarly makes a variety of statements about gender dysphoria, therapies for gender dysphoria, and outcomes of therapies, which I believe to be inaccurate, or unsupported by scientific evidence. Dr. Safer also makes a number of sweeping and purportedly scientific assertions that are not substantiated by peer-reviewed articles or studies.

17. It is also my opinion that a number of Dr. Safer’s assertions are inaccurate or unsupported, for reasons that I explain in this Declaration. Similarly, I will provide citations to published, peer-reviewed articles that inform my judgments.

18. A summary of the key points that I explain in this report is as follows:

a. Sex as defined by biology and reproductive function is clear, binary, and cannot be changed. While hormonal and surgical procedures may enable some individuals to “pass” as the opposite gender during some or all of their lives, such procedures carry with them physical, psychological, and social risks, and no procedures can enable an individual to perform the reproductive role of the opposite sex. (Section II.A.)

b. The diagnosis of “gender dysphoria” encompasses a diverse array of conditions, with widely differing pathways and characteristics depending on age of onset, biological sex, mental health, intelligence, motivations for gender transition, socioeconomic status, country of origin, etc. Data from one population (e.g., adults) cannot be assumed to be applicable to others (e.g., children). (Section II.B.)

c. Among practitioners in the field, there are currently widely varying views concerning both the causes of and appropriate therapeutic response to gender dysphoria in children or adolescents. There are no generally accepted “standards of care” and existing studies do not provide a basis for a scientific conclusion as to which therapeutic response results in the best long-term outcomes for affected individuals. (Section III.)

d. Transgender identity is not biologically based. Rather, gender dysphoria is a psychiatric condition that cannot be identified by any biological test or measurement. (Sections IV.A, IV.B.)

e. Disorders of sexual development (“DSDs”) are biological phenomena. It is an error to conflate and/or scientifically link DSDs with incidents of gender dysphoria. (Sections IV.C, IV.D.)

f. The large majority of children who are diagnosed with gender dysphoria “desist”—that is, their gender dysphoria does not persist—by puberty or adulthood. Desistance is also increasingly observed among teens and young adults who have experienced “rapid onset gender dysphoria” — first manifesting gender dysphoria during or shortly after adolescence. (Section V.A., V.B.)

g. “Social transition” —the active affirmation of transgender identity—in young children is a powerful psychotherapeutic intervention that will substantially reduce the

number of children “desisting” from transgender identity. Therefore, the profound implications of “affirmative” treatment—which include taking puberty blockers and cross-sex hormones—must be taken into account where social transition is being considered. (Section VI.A., VI.B.)

h. Administration of puberty blockers is not a benign “pause” of puberty, but rather a powerful medical and psychotherapeutic intervention that almost invariably leads to persistence in a transgender identity and, ultimately, to the administration of cross-sex hormones. (Section VI.C.)

i. The knowledge base concerning the “affirmative” treatment of gender dysphoria available today has very low scientific quality with many long-term implications remaining unknown. (Section VII.A)

j. There are no studies that show that affirmation of transgender identity in young children reduces suicide or suicidal ideation, or improves long-term outcomes, as compared to other therapeutic approaches. Meanwhile, multiple studies show that adult individuals living transgender lives suffer much higher rates of suicidal ideation, completed suicide, and negative physical and mental health conditions than does the general population. This is true before and after transition, hormones, and surgery. (Section VII.B., VII.C.)

k. In light of what is known and not known about the impact of affirmation on the incidence of suicide, suicidal ideation, and other indicators of mental and physical health, it is scientifically baseless, and therefore unethical, to assert that a child or adolescent who express an interest in a transgender identity will kill him- or herself unless adults and peers affirm that child in a transgender identity. (Section VIII.)

l. Hormonal interventions to treat gender dysphoria are experimental in nature and have not been shown to be safe, but rather put an individual at risk of a wide range of long-term and even life-long harms including: physical health risks; sterilization and the associated emotional response; impaired sexual response; surgical complications and life-long after-care; alienation of family and romantic relationships; elevated mental health risks of depression, anxiety, and substance abuse. (Section IX.)

II. BACKGROUND ON THE FIELD

A. The biological baseline of the binary sexes

19. Dr. Adkins asserts that “the terms biological sex and biological male or female are imprecise and should be avoided.” (Adkins at 10.) Dr. Safer further asserts that the term biological sex “can cause confusion,” and moreover that a person’s sex encompasses gender identity. (Safer at 6.) These statements are untrue. Biological sex is very well defined in all biological sciences including medicine. It is pervasively important in human development throughout the lifecycle.

20. Sex is not “assigned at birth” by humans visualizing the genitals of a newborn; it is not imprecise. Rather, it is clear, binary, and determined at conception. The sex of a human individual at its core structures the individual’s biological reproductive capabilities—to produce ova and bear children as a mother, or to produce semen and beget children as a father. As physicians know, sex determination occurs at the instant of conception, depending on whether a sperm’s X or Y chromosome fertilizes the egg. A publication of the federal government’s National Institute of Health accurately summarizes the scientific facts:

“Sex is a biological classification, encoded in our DNA. Males have XY chromosomes, and females have XX chromosomes. Sex makes us male or female. Every cell in your body has a sex—making up tissues and organs, like your skin, brain, heart, and

stomach. Each cell is either male or female depending on whether you are a man or a woman.” (NIH 2022.)

21. The binary of biological sex is so fundamental and wide-ranging in its effects on human (and mammal) development and physiology that since 2014 the NIH has required all funded research on humans or vertebrate animals to include “sex as a biological variable” and give “adequate consideration of both sexes in experiments.” (NIH 2015). In 2021, the Endocrine Society issued a position paper elaborating on the application of the NIH requirement. The Endocrine Society correctly stated that “Sex is a biological concept . . . all mammals have 2 distinct sexes;” that “biological sex is . . . a fundamental source of intraspecific variation in anatomy and physiology;” and that “In mammals, numerous sexual traits (gonads, genitalia, etc.) that typically differ in males and females are tightly linked to each other because one characteristic leads to sex differences in other traits.” (Bhargava et al. 2021 at 221, 229.)

22. The Endocrine Society emphasized that “The terms sex and gender should not be used interchangeably,” and noted that even in the case of those “rare” individuals who suffer from some defect such that they “possess a combination of male- and female-typical characteristics, those clusters of traits are sufficient to classify most individuals as either biologically male or female.” They concluded, “Sex is an essential part of vertebrate biology, but gender is a human phenomenon. Sex often influences gender, but gender cannot influence sex.” (Bhargava et al. 2021 at 220-221, 228.) For purposes of this litigation, Dr. Bhargava’s statement that gender cannot influence sex is of central importance.

23. As these statements and the NIH requirement suggest, biological sex pervasively influences human anatomy, its development and physiology. This includes, of course, the development of the human brain, in which many sexually dimorphic characteristics have now been identified. In particular, the Endocrine Society and countless other researchers have

determined that human brains undergo particular sex-specific developmental stages during puberty. This predictable developmental process is a genetically controlled coordinated endocrine response that begins with pituitary influences leading to increases in circulating sex hormones. (Bhargava et al. 2021 at 225, 229; Blakemore et al. 2010 at 926-927, 929; NIH 2001.).

24. Humans have viewed themselves in terms of binary sexes since the earliest historical records. Recognizing a concept of “gender identity” as something distinct from sex is a rather recent innovation whose earliest manifestations likely began in the late 1940s. Its usage became common in medicine in the 1980s and subsequently in the larger culture. Definitions of gender have been evolving and remain individual-centric and subjective. In a statement on “Gender and Health,” the World Health Organization defines “gender” as “the characteristics of women, men, girls and boys that are socially constructed” and that “var[y] from society to society and can change over time,” and “gender identity” as referring to “a person’s deeply felt, internal and individual experience of gender.” (WHO Gender and Health.) As these definitions indicate, a person’s “felt” “experience of gender” is inextricably bound up with and affected by societal gender roles and stereotypes—or, more precisely, by the affected individual’s *perception* of societal gender roles and stereotypes and their personal idiosyncratic meanings. Typically, gendered persons also have subtly different, often idiosyncratic, reactions to societal gender roles and stereotypes without preoccupation with changing their anatomy.

25. Thus, the self-perceived gender of a child begins to develop along with the early stages of identity formation generally, influenced in part from how others label the infant: “I love you, son (daughter).” This designation occurs thousands of times in the first two years of life when a child begins to show awareness of the two possibilities. As acceptance of the designated

gender corresponding to the child's sex is the outcome in >99% of children everywhere, anomalous gender identity formation begs for understanding. Is it biologically shaped? Is it biologically determined? Is it the product of how the child was privately regarded and treated? Is it a product of the quality of early life caregiver attachments? Does it stem from trauma-based rejection of maleness or femaleness, and if so, flowing from what trauma? Does it derive from a tense, chaotic interpersonal parental relationship without physical or sexual abuse? Is it a symptom of another, as of yet unrevealed, emotional disturbance or neuropsychiatric condition (autism)? The answers to these relevant questions are not scientifically known but are not likely to be the same for every trans-identified child, adolescent, or adult.

26. Under the influence of hormones secreted by the testes or ovaries, numerous additional sex-specific differences between male and female bodies continuously develop postnatally, culminating in the dramatic maturation of the primary and secondary sex characteristics with puberty. These include differences in hormone levels, height, weight, bone mass, shape, musculature, body fat levels and distribution, and hair patterns, as well as physiological differences such as menstruation and ejaculation. These are genetically programmed biological consequences of sex—the actual meaning of sex over time. Among the consequences of sex is the consolidation of gender identity during and after puberty.

27. Despite the increasing ability of hormones and various surgical procedures to reconfigure some male bodies to visually pass as female, or vice versa, the biology of the person remains as defined by his (XY) or her (XX) chromosomes, including cellular, anatomic, and physiologic characteristics and the particular disease vulnerabilities associated with that chromosomally defined sex. For instance, the XX (genetically female) individual who takes testosterone to stimulate certain male secondary sex characteristics will nevertheless remain

unable to produce sperm and father children. It is certainly true, as Dr. Adkins writes, that “[h]ormone therapy and social transition significantly change a person’s physical appearance.” (Adkins at 8.) But in critical respects this change can only be “skin deep.” Contrary to assertions and hopes that medicine and society can fulfill the aspiration of the trans individual to become “a complete man” or “a complete woman,” this is not biologically attainable. (Levine 2018 at 6; Levine 2016 at 238.) It is possible for some adolescents and adults to pass unnoticed—that is, to be perceived by most individuals as a member of the gender that they aspire to be—but with limitations, costs, and risks, as I detail later.

B. Definition and diagnosis of gender dysphoria

28. Specialists have used a variety of terms over time, with somewhat shifting definitions, to identify and speak about a distressing incongruence between an individual’s genetically determined sex and the gender with which they identify or to which they aspire. Today’s American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders* (“DSM-5”) employs the term Gender Dysphoria and defines it with separate sets of criteria for adolescents and adults on the one hand, and children on the other.

29. There are at least five distinct pathways to gender dysphoria: early childhood onset; onset near or after puberty with no prior cross gender patterns; onset after defining oneself as gay for several or more years and participating in a homosexual lifestyle; adult onset after years of heterosexual transvestism; and onset in later adulthood with few or no prior indications of cross-gender tendencies or identity. (Levine 2021.) The early childhood onset pathway and the more recently observed onset around puberty pathway are most relevant to this matter.

30. Gender dysphoria has very different characteristics depending on age and sex at onset. Young children who are living a transgender identity commonly suffer materially fewer symptoms of concurrent mental distress than do older patients. (Zucker 2018 at 10.) The

developmental and mental health patterns for each of these groups are sufficiently different that data developed in connection with one of these populations cannot be assumed to be applicable to another.

31. The criteria used in DSM-5 to identify Gender Dysphoria include a number of signs of discomfort with one's natal sex and vary somewhat depending on the age of the patient, but in all cases require "clinically significant distress or impairment in . . . important areas of functioning" such as social, school, or occupational settings. The symptoms must persist for at least six months.

32. Children who conclude that they are transgender are often unaware of a vast array of adaptive possibilities for how to live life as a man or a woman—possibilities that become increasingly apparent over time to both males and females. A boy or a girl who claims or expresses interest in pursuing a transgender identity often does so based on stereotypical notions of femaleness and maleness that reflect constrictive notions of what men and women can be. (Levine 2017 at 7.) A young child's—or even an adolescent's—understanding of this topic is quite limited. Nor can they grasp what it may mean for their future to be sterile. These children and adolescents consider themselves to be relatively unique; they do not realize that discomfort with the body and perceived social role is neither rare nor new to civilization. What is new is that such discomfort is thought to indicate that they must be a trans person.

C. Impact of gender dysphoria on minority and vulnerable groups

33. Given that, as I discuss later, a diagnosis of gender dysphoria is now frequently putting even young children on a pathway that leads to irreversible physical changes and sterilization by young adulthood, it should be of serious concern to all practitioners that minority and vulnerable groups are receiving this diagnosis at disproportionately high rates. These include: children of color (Rider et al. 2018), children with mental developmental disabilities

(Reisner et al. 2015), children on the autistic spectrum (at a rate more than 7x the general population) (Shumer et al. 2016; van der Miesen et al. 2018), children with ADHD (Becerra-Culqui et al. 2018), children residing in foster care homes, adopted children (at a rate more than 3x the general population) (Shumer et al. 2017), victims of childhood sexual or physical abuse or other “adverse childhood events” (Thoma 2021 et al.; Newcomb et al. 2020; Kozłowska et al. 2021), children with a prior history of psychiatric illness (Edwards-Leeper et al. 2017; Kaltiala-Heino et al. 2015; Littman 2018), and more recently adolescent girls (in a large recent study, at a rate more than 2x that of boys) (Rider et al. 2018 at 4).

D. Three competing conceptual models of gender dysphoria and transgender identity

34. Discussions about appropriate responses by mental health professionals (“MHPs”) to actual or sub-threshold gender dysphoria are complicated by the fact that various speakers and advocates (or a single speaker at different times) view transgenderism through at least three very different paradigms, often without being aware of, or at least without acknowledging, the distinctions.

35. Gender dysphoria is **conceptualized and described by some professionals and laypersons as though it were a serious, physical medical illness that causes suffering,** comparable to diseases that are curable before it spreads, such as melanoma or sepsis. Within this paradigm, whatever is causing distress associated with gender dysphoria—whether secondary sex characteristics such as facial hair, nose and jaw shape, presence or absence of breasts, or the primary anatomical sex organs of testes, ovaries, penis, or vagina—should be removed to alleviate the illness. The promise of these interventions is the cure of the gender dysphoria.

36. Dr. Adkins appears to endorse this perspective, asserting that gender dysphoria is a “medical condition.” (Adkins at 4.) It should be noted, however, that gender dysphoria is a psychiatric, not a medical, diagnosis. Since its inception in DSM-III in 1983, it has always been specified in the psychiatric DSM manuals and has not been specified in medical diagnostic manuals. Notably, gender dysphoria is the only psychiatric condition to be treated by surgery, even though no endocrine or surgical intervention package corrects any identified biological abnormality. (Levine 2016 at 240.)

37. Gender dysphoria is alternatively **conceptualized in developmental terms**, as an adaptation to a psychological problem that may have been first manifested as a failure to establish a comfortable conventional sense of self in early childhood. This paradigm starts from the premise that all human lives are influenced by past processes and events. Trans lives are not exceptions to this axiom. (Levine 2016 at 238.) MHPs who think of gender dysphoria through this paradigm may work both to identify and address causes of the basic problem of the deeply uncomfortable self or a sense of self impaired by later adversity or abuse. The purpose is to ameliorate suffering when the underlying problem cannot be solved. MHPs first work with the patient and (ideally) family to learn about the events and processes that may have led to the trans person repudiating the gender associated with his sex. The developmental paradigm is mindful of temperamental, parental bonding, psychological, sexual, and physical trauma influences, and the fact that young children work out their psychological issues through fantasy and play and adolescents work out their issues by adopting various interests and identity labels.

38. There is evidence among adolescents that peer social influences through “friend groups” (Littman 2018) or through the internet can increase the incidence of gender dysphoria or claims of transgender identity. Responsible MHPs will want to probe these potential influences

to better understand what is truly deeply tied to the psychology of the patient, and what may instead be being “tried on” by the youth as part of the adolescent process of self-exploration and self-definition.

39. In addition, the developmental paradigm recognizes that, with the important exception of genetic sex, essentially all aspects of an individual’s identity evolve—often markedly—across the individual’s lifetime. This includes gender. Some advocates assert that a transgender identity is biologically caused, fixed from early life, and eternally present in an unchanging manner. As I review later, however, this assertion is not supported by science.¹

40. The third paradigm through which gender dysphoria is alternatively conceptualized is from **a sexual minority rights perspective**. Under this paradigm, any response other than medical and societal affirmation and implementation of a patient’s claim to “be” the opposite gender is a violation of the individual’s civil right to self-expression. Any effort to ask “why” questions about the patient’s condition, or to address underlying causes, is viewed as a violation of autonomy and civil rights. In the last few years, this paradigm has been successful in influencing public policy and the education of pediatricians, endocrinologists, and many mental health professionals. Obviously, however, this is not a medical or psychiatric perspective. Unfortunately, it appears to be the most powerful perspective that exists in the public, non-scientific debate.

E. Four competing models of therapy

41. Few would disagree that the human psyche is complex. Few would disagree that children’s and adolescents’ developmental pathways typically have surprising twists and turns. The complexity and unpredictability of childhood and adolescent development equally applies to

¹ Even the advocacy organization The Human Rights Campaign asserts that a person can have “a fluid or unfixed gender identity.” <https://www.hrc.org/resources/glossary-of-terms>.

trans-identifying youth. Because of past difficulties of running placebo-controlled clinical trials in the transgender treatment arena, substantial disagreements among professionals about the causes of trans identities and their ideal treatments exist. These current disagreements might have been minimized if trans treated persons were carefully followed up to determine long term outcomes. They have not been. When we add to this to the very different current paradigms for understanding transgender phenomena, it is not scientifically surprising that disagreements are sharply drawn. It is with this in mind that I summarize below the leading approaches, and offer certain observations and opinions concerning them.

(1) The “watchful waiting” therapy model

42. In Section V.A below I review the uniform finding of eleven follow-up studies that the large majority of children who present with gender dysphoria will desist from desiring a transgender identity by adulthood if left untreated by social transition approaches.

43. When a pre-adolescent child presents with gender dysphoria, a “watchful waiting” approach seeks to allow for the fluid nature of gender identity in children to naturally evolve—that is, take its course from forces within and surrounding the child. Watchful waiting has two versions:

a. Treating any other psychological co-morbidities—that is, other mental illnesses as defined by DSM-5 (separation anxiety disorder, attention deficit hyperactivity disorder, autism spectrum disorder, obsessive compulsive disorder, etc), or subthreshold for diagnosis but behavioral problems that the child may exhibit (school avoidance, bedwetting, inability to make friends, aggression/defiance) without a focus on gender **(model #1)**; and

b. No treatment at all for anything but a regular follow-up appointment. This might be labeled a “hands off” approach **(model #2)**.

(2) The psychotherapy model: Alleviate distress by identifying and addressing causes (model #3)

44. One of the foundational principles of psychotherapy has long been to work with a patient to identify the causes of observed psychological distress and then to address those causes as a means of alleviating the distress. The National Institute of Mental Health has promulgated the idea that 75% of adult psychopathology has its origins in childhood experience.

45. Many experienced practitioners in the field of gender dysphoria, including myself, have believed that it makes sense to employ these long-standing tools of psychotherapy for patients suffering gender dysphoria, asking the question as to what factors in the patient's life are the determinants of the patient's repudiation of his or her natal sex. (Levine 2017 at 8; Levine 2021.) I and others have reported success in alleviating distress in this way for at least some patients, whether the patient's sense of discomfort or incongruence with his or her natal sex entirely disappeared or not. Relieving accompanying psychological co-morbidities leaves the patient freer to consider the pros and cons of transition as he or she matures.

46. Among other things, the psychotherapist who is applying traditional methods of psychotherapy may help—for example—the male patient appreciate the wide range of masculine emotional and behavioral patterns as he grows older. He may discuss with his patient, for example, that one does not have to become a “woman” in order to be kind, compassionate, caring, noncompetitive, to love the arts, and to be devoted to others' feelings and needs. (Levine 2017 at 7.) Many biologically male trans individuals, from childhood to older ages, speak of their perceptions of femaleness as enabling them to discuss their feelings openly, whereas they perceive boys and men to be constrained from emotional expression within the family and larger culture, and to be aggressive. Men, of course, can be emotionally expressive, just as they can

wear pink. Converse examples can be given for girls and women. These types of ideas regularly arise during psychotherapies.

47. As I note above, many gender-nonconforming children and adolescents in recent years derive from minority and vulnerable groups who have reasons to feel isolated and have an uncomfortable sense of self. A trans identity may be a hopeful attempt to redefine the self in a manner that increases their comfort and decreases their anxiety. The clinician who uses traditional methods of psychotherapy may not focus on their gender identity, but instead work to help them to address the actual sources of their discomfort. Success in this effort may remove or reduce the desire for a redefined identity. This often involves a focus on disruptions in their attachment to parents in vulnerable children, for instance, those in the foster care system.

48. Because “watchful waiting” can include treatment of accompanying psychological co-morbidities, and the psychotherapist who hopes to relieve gender dysphoria may focus on potentially causal sources of psychological distress rather than on the gender dysphoria itself, there is no sharp line between “watchful waiting” and the psychotherapy model in the case of prepubescent children.

49. To my knowledge, there is no evidence beyond anecdotal reports that psychotherapy can enable a return to male identification for genetically male boys, adolescents, and men, or return to female identification for genetically female girls, adolescents, and women. On the other hand, anecdotal evidence of such outcomes does exist; I and other clinicians have witnessed reinvestment in the patient’s biological sex in some individual patients who are undergoing psychotherapy. The Internet contains many such reports, and I have published a paper on a patient who sought my therapeutic assistance to reclaim his male gender identity after 30 years living as a woman and is in fact living as a man today. (Levine 2019.) I have seen

children desist even before puberty in response to thoughtful parental interactions and a few meetings of the child with a therapist. There are now a series of articles and at least one major book on the psychological treatment of adolescents. (D’Angelo et al. 2021 at 7-16; Evans & Evans 2021.)

(3) The affirmation therapy model (model #4)

50. While it is widely agreed that the therapist should not directly challenge a claimed transgender identity in a child, some advocates and practitioners go much further, and promote and recommend that any expression of transgender identity should be immediately accepted as decisive, and thoroughly affirmed by means of consistent use of clothing, toys, pronouns, etc., associated with transgender identity. They argue that the child should be comprehensively re-socialized in grade school in their aspired-to gender. As I understand it, this is asserted as a reason why male students who assert a female gender identity must be permitted to compete in girls’ or women’s athletic events. These advocates treat any question about the causes of the child’s transgender identification as inappropriate. They may not recognize the child’s ambivalence. They assume that observed psychological co-morbidities in the children or their families are unrelated or will get better with transition, and need not be addressed by the MHP who is providing supportive guidance concerning the child’s gender identity.

51. Some advocates, indeed, assert that unquestioning affirmation of any claim of transgender identity in children is essential, and that the child will otherwise face a high risk of suicide or severe psychological damage. Dr. Adkins appears to follow this line, asserting that “My clinical experience . . . has been that [patients] suffer and experience worse health outcomes” when they are not permitted to enter all spaces and participate in all activities in a manner “consistent with gender identity.” (Adkins at 9.) This claim is simply not supported by the clinical data we have available to us. Indeed, available long-term data contradicts Dr.

Adkins' claim. I address physical and mental health outcomes in Section VII below, and suicide in Section VIII below.

52. Dr. Adkins also asserts that fully supported social transition is the “only treatment for prepubertal children.” (Adkins at 6.) As I review in the next section, this is not correct. This may be the only treatment that Dr. Adkins considers, but my own conversations and contacts lead me to believe that Dr. James Cantor was correct when he wrote that “almost all clinics and professional associations in the world” do not use “gender affirmation” for prepubescent children and instead “delay any transitions after the onset of puberty.” (Cantor 2019 at 1.)

53. I do not know what proportion of practitioners are using which model. However, in my opinion, in the case of young children, prompt and thorough affirmation of a transgender identity disregards the principles of child development and family dynamics and is not supported by science. Instead of science, this approach is currently being reinforced by an echo-chamber of approval from other like-minded child-oriented professionals who do not sufficiently consider the known negative medical and psychiatric outcomes of trans adults. Rather than recommend social transition in grade school, the MHP must focus attention on the child's underlying internal and familial issues. Ongoing relationships between the MHP and the parents, and the MHP and the child, are vital to help the parents, child, other family members, and the MHP to understand over time the issues that need to be dealt with by each of them.

54. Likewise, since the child's sense of gender develops in interaction with his parents and their own gender roles and relationships, the responsible MHP will almost certainly need to delve into family and marital dynamics.

III. THERE IS NO CONSENSUS OR AGREED “STANDARD OF CARE” CONCERNING THERAPEUTIC APPROACHES TO CHILD OR ADOLESCENT GENDER DYSPHORIA.

55. Dr. Adkins states that “[t]he only treatment to avoid [] serious harm is to recognize the gender identity of patients with gender dysphoria and follow appropriate treatment protocols to affirm gender identity and alleviate distress,” and appears to believe that transition and affirmation of children who suffer from gender dysphoria is a generally accepted “standard of care.” (Adkins at 5.) It is not.

56. As I review in separate sections later, there is far too little firm clinical evidence in this field to permit any evidence-based standard of care. Given the lack of scientific evidence, it is neither surprising nor improper that—as I detailed in Section II—there is a diversity of views among practitioners as to as to the best therapeutic response for the child, adolescent, or young adult who suffers from gender dysphoria. Dr. Adkins is unwittingly confusing therapeutic precedent among those who agree with her views, armed with ideas promulgated by WPATH, with careful scientific documentation of her concepts. She presumes that her views have been scientifically established even though much has been published highlighting the lack of supportive definitive evidence.

57. Reviewing the state of opinion and practice in 2021, the Royal Australian and New Zealand College of Psychiatrists observed that “There are polarised views and mixed evidence regarding treatment options for people presenting with gender identity concerns, especially children and young people.” (RANZCP, 2021.) Similarly, a few years earlier prominent Dutch researchers noted: “[T]here is currently no general consensus about the best approach to dealing with the (uncertain) future development of children with GD, and making decisions that may influence the function and/or development of the child — such as social

transition.” (Ristori & Steensma 2016 at 18.)² In this Section, I comment on some of the more important areas of disagreement within the field.

A. Experts and organizations disagree as to whether “distress” is a necessary element for diagnoses that justifies treatment for gender identity issues.

58. As outlined in Section II.B above, “clinically significant distress” is one of the criteria used in DSM-5 to identify gender dysphoria. This indicates a heightened level of distress that rises beyond a threshold level of social awkwardness or discomfort with the changing body. It is known that many trans-identified youth with incongruence between their sexed bodies and their gender identity choose not to take hormones; their incongruence is quite tolerable as they further clarify their sexual identity elements. This population raises the questions of what distress is being measured when DSM-5 criteria are met and what else might be done about it.

59. I note that there is no “clinically significant distress” requirement in World Health Organization’s International Classification of Diseases (ICD-11) criteria for gender incongruence, which rather indicates “a marked and persistent incongruence between an individual’s experienced gender and the assigned sex.” (World Health Organization 2019.)

60. Therefore, even between these two committee-based authorities, there is a significant disagreement as to what constitutes a gender condition justifying life-changing interventions. To my knowledge, some American gender clinics and practitioners are essentially operating under the ICD-11 criteria rather than the APA’s DSM-5 criteria, prescribing transition for children, hormonal interventions for slightly older children, and different hormones for adolescents who assert a desire for a transgender identity whether or not they are exhibiting “clinically significant distress.” Others adhere to the DSM-5 diagnostic standard.

² See also Zucker 2020 which questions the merit of social transition as a first-line treatment.

61. I will add that even from within one “school of thought,” such as embodied by Dr. Adkins, it is not responsible to make a single, categorical statement about the proper treatment of children or adolescents presenting with gender dysphoria or other gender-related issues. There is no single pathway to the development of a trans identity and no reasonably uniform short- or long-term outcome of medically treating it. As individuals grow physically, mature psychologically, and experience or fail to experience satisfying romantic relationships, their life course depends on their differing psychological, social, familial, and life experiences. There should be no trust in assertions that trans identified youth must be treated in a particular manner to avoid harm for two reasons: first, there is no systematic data on the nature of, and the rate of harms of either affirmative treatment, no treatment, or psychological only treatment. Second, as in other youthful psychiatric and other challenges, outcomes vary.

B. Opinions and practices vary widely about the utilization of social transition for children and adolescents.

62. Dr. Adkins notes that she is a member of the World Professional Association for Transgender Health (WPATH), invokes a guidance document that that organization has chosen to publish under the title of “standards of care,” and asserts that the WPATH Standards of Care are “widely accepted.” (Adkins at 3, 5.) Below, I will provide some explanation of WPATH and its “Standards of Care,” which are not the product of a strictly scientific organization, and are by no means accepted by all or even most practitioners as setting out best practices.

63. Here, however, I will note that WPATH does not take a position concerning whether or when social transition may be appropriate for pre-pubertal children. Instead, the WPATH “Standards of Care” states that the question of social transition for children is a “controversial issue” and calls for mental health professionals to support families in what it describes as “difficult decisions” concerning social transition.

64. Dr. Erica Anderson is a prominent practitioner in this area who identifies as a transgender woman, who was the first transgender president of USPATH, and who is a former board member of WPATH. Dr. Anderson recently resigned from those organizations and has condemned automatic approval of transition upon the request of a child or adolescent, noting that “adolescents . . . are notoriously susceptible to peer influence,” that transition “doesn’t cure depression, doesn’t cure anxiety disorders, doesn’t cure autism-spectrum disorder, doesn’t cure ADHD,” and instead that “a comprehensive biopsychosocial evaluation” should proceed allowing a child to transition. (Davis 2022.) And as I have explained previously, my own view based on 50 years of experience in this area favors strong caution before approving life-altering interventions such as social transition, puberty blockers, or cross-sex hormones.

C. The WPATH “Standards of Care” is not an impartial or evidence-based document.

65. Because WPATH is frequently cited by advocates of social, hormonal, and surgical transition, I provide some context concerning that private organization and its “Standards of Care.”

66. I was a member of the Harry Benjamin International Gender Dysphoria Association from 1974 until 2001. From 1997 through 1998, I served as the Chairman of the eight-person International Standards of Care Committee that issued the fifth version of the Standards of Care. I resigned my membership in 2002 due to my regretful conclusion that the organization and its recommendations had become dominated by politics and ideology, rather than by scientific process, as it was years earlier. In approximately 2007, the Harry Benjamin International Gender Dysphoria Association changed its name to the World Professional Association for Transgender Health.

67. WPATH is a voluntary membership organization. Since at least 2002, attendance at its biennial meetings has been open to trans individuals who are not licensed professionals. While this ensures taking patients' needs into consideration, it limits the ability for honest and scientific debate, and means that WPATH can no longer be considered a purely professional organization.

68. WPATH takes a decided view on issues as to which there is a wide range of opinion among professionals. WPATH explicitly views itself as not merely a scientific organization, but also as an advocacy organization. (Levine 2016 at 240.) WPATH is supportive to those who want sex reassignment surgery ("SRS"). Skepticism as to the benefits of SRS to patients, and strong alternate views, are not well tolerated in discussions within the organization or their educational outreach programs. Such views have been known to be shouted down and effectively silenced by the large numbers of nonprofessional adults who attend the organization's biennial meetings. Two groups of individuals that I regularly work with have attended recent and separate WPATH continuing education sessions. There, questions about alternative approaches were quickly dismissed with "There are none. This is how it is done." Such a response does not accurately reflect what is known, what is unknown, and the diversity of clinical approaches in this complex field.

69. The Standards of Care ("SOC") document is the product of an effort to be balanced, but it is not politically neutral. WPATH aspires to be both a scientific organization and an advocacy group for the transgendered. These aspirations sometimes conflict. The limitations of the Standards of Care, however, are not primarily political. They are caused by the lack of rigorous research in the field, which allows room for passionate convictions on how to care for the transgendered. And, of course, once individuals have socially, medically, and surgically

transitioned, WPATH members and the trans people themselves at the meetings are committed to supporting others in their transitions. Not only have some trans participants been distrustful or hostile to those who question the wisdom of these interventions, their presence makes it difficult for professionals to raise their concerns. Vocal trans rights advocates have a worrisome track record of attacking those who have alternative views. (Dreger 2015.)

70. In recent years, WPATH has fully adopted some mix of the medical and civil rights paradigms. It has downgraded the role of counseling or psychotherapy as a requirement for these life-changing processes. WPATH no longer considers preoperative psychotherapy to be a requirement. It is important to WPATH that the person has gender dysphoria; the pathway to the development of this state is not. (Levine 2016 at 240.) The trans person is assumed to have thoughtfully considered his or her options before seeking hormones, for instance.

71. Most psychiatrists and psychologists who treat patients suffering sufficiently severe distress from gender dysphoria to seek inpatient psychiatric care are not members of WPATH. Many psychiatrists, psychologists, and pediatricians who treat some patients suffering gender dysphoria on an outpatient basis are not members of WPATH. WPATH represents a self-selected subset of the profession along with its many non-professional members; it does not capture the clinical experiences of others. WPATH claims to speak for the medical profession; however, it does not welcome skepticism and therefore, deviates from the philosophical core of medical science. There are pediatricians, psychiatrists, endocrinologists, and surgeons who object strongly, on professional grounds, to transitioning children and providing affirmation in a transgender identity as the first treatment option. WPATH does not speak for all of the medical profession.

72. In 2010 the WPATH Board of Directors issued a statement advocating that incongruence between sex and felt gender identity should cease to be identified in the DSM as a pathology.³ This position was debated but not adopted by the (much larger) American Psychiatric Association, which maintained the definitions and diagnoses of gender dysphoria as a pathology in the DSM-5 manual issued in 2013.

73. In my experience some current members of WPATH have little ongoing experience with the mentally ill, and many trans care facilities are staffed by MHPs who are not deeply experienced with recognizing and treating frequently associated psychiatric co-morbidities. Further, being a mental health professional, per se, does not guarantee experience and skill in recognizing and effectively intervening in serious or subtle patterns. Because the 7th version of the WPATH SOC deleted the requirement for therapy, trans care facilities that consider these Standards sufficient are permitting patients to be counseled to transition by means of social presentation, hormones, and surgery by individuals with masters rather than medical degrees.

D. Opinions and practices differ widely with respect to the proper role of psychological counseling before, as part of, or after a diagnosis of gender dysphoria.

74. In Version 7 of its Standards of Care, released in 2012, WPATH downgraded the role of counseling or psychotherapy, and the organization no longer sees psychotherapy without transition and hormonal interventions as a potential path to eliminate gender dysphoria by enabling a patient to return to or achieve comfort with the gender identity aligned with his or her biology.

³ WPATH *De-Psychopathologisation Statement* (May 26, 2010), available at wpath.org/policies (last accessed January 21, 2020).

75. Around the world, many prominent voices and practitioners disagree. For example, renowned gender therapists Dr. Laura Edwards-Leeper and Dr. Erica Anderson (who, as mentioned above, identifies as a transgender woman) have recently spoken out arguing that children and adolescents are being subjected to puberty blockers and hormonal intervention far too quickly, when careful and extended psychotherapy and investigation for potential causes of feelings of dysphoria (such as prior sexual abuse) should be the first port of call and might resolve the dysphoria. (Edwards-Leeper & Anderson 2021; Davis 2022.)

76. In a recently published position statement on gender dysphoria, the Royal Australian and New Zealand College of Psychiatrists emphasized the critical nature of mental health treatment for gender dysphoric minors, stressing “the importance of the psychiatrist’s role to undertake thorough assessment and evidence-based treatment ideally as part of a multidisciplinary team, especially highlighting co-existing issues which may need addressing and treating.” The Royal College also emphasized the importance of assessing the “psychological state and context in which Gender Dysphoria has arisen,” before any treatment decisions are made. (RANZCP, 2021.)

77. Dr. Paul Hruz of the University of Washington St. Louis Medical School has noted, “The WPATH has rejected psychological counseling as a viable means to address sex–gender discordance with the claim that this approach has been proven to be unsuccessful and is harmful (Coleman et al. 2012). Yet the evidence cited to support this assertion, mostly from case reports published over forty years ago, includes data showing patients who benefited from this approach (Cohen-Kettenis and Kuiper 1984).” (Hruz 2020.)

E. Opinions and practices vary widely with respect to the administration of puberty blockers and cross-sex hormones.

78. There is likewise no broadly accepted standard of care with respect to use of puberty blockers. The WPATH Standards of Care explicitly recognize the lack of any consensus on this important point, stating: “Among adolescents who are referred to gender identity clinics, the number considered eligible for early medical treatment—starting with GnRH analogues to suppress puberty in the first Tanner stages—differs among countries and centers. Not all clinics offer puberty suppression. . . The percentages of treated adolescents are likely influenced by the organization of health care, insurance aspects, cultural differences, opinions of health professionals, and diagnostic procedures offered in different settings.”

79. The use of puberty blockers as a therapeutic intervention for gender dysphoria is often justified by reference to the seminal work of a respected Dutch research team that developed a protocol that administered puberty blockers to children no younger than age 14. However, it is well known that many clinics in North America now administer puberty blockers to children at much younger ages than the “Dutch Protocol” allows. (Zucker 2019.) The Dutch protocol only treated children with these characteristics: a stable cross gender identity from early childhood; dysphoria that worsened with the onset of puberty; were otherwise psychologically healthy; had healthy families; the patient and family agreed to individual and family counselling throughout the protocol. But the experience and results of the Dutch model is being used as a justification for giving puberty blockers to children who differ considerably from these criteria. Its authors have also recently noted this fact. (de Vries 2020.)

80. However, Zucker notes that “it is well known” that clinicians are administering cross-sex hormones, and approving surgery, at ages lower than the minimum age thresholds set by that “Dutch Protocol.” (Zucker 2019 at 5.)

81. Similarly, at least one prominent clinic—that of Dr. Safer at Columbia’s Mt. Sinai Medical Center—is quite openly admitting patients for even *surgical* transition who are not eligible under the criteria set out in WPATH’s Standards of Care. A recent study published by Dr. Safer and colleagues revealed that of a sample of 139 individuals, 45% were eligible for surgery “immediately” under the center’s own criteria, while only 15% were eligible under WPATH’s criteria. That is, *three times* as many patients immediately qualified for surgery under the center’s loose standards than would have qualified under WPATH criteria. (Lichenstein et al. 2020.)

82. Internationally, there has been a recent marked trend *against* use of puberty blockers, as a result of extensive evidence reviews by national medical bodies, which I discuss later. The main gender clinic in Sweden has declared that it will no longer authorize use of puberty blockers for minors below the age of 16. Finland has similarly reversed its course, issuing new guidelines that allow puberty blockers only on a case-by-case basis after an extensive psychiatric assessment. A landmark legal challenge against the UK’s National Health Service in 2020 by “detransitioner” Keira Bell led to the suspension of the use of puberty blockers and new procedures to ensure better psychological care, as well as prompting a thorough evidence review by the National Institute for Health and Care Excellence (NICE 2021a; NICE 2021b).⁴

83. In this country, some voices in the field are now publicly arguing that *no* comprehensive mental health assessment at all should be required before putting teens on puberty blockers or cross-sex hormones (Ghorayshi 2022), while Dr. Anderson and Dr.

⁴ The decision requiring court approval for administration of hormones to any person younger than age 16 was later reversed on procedural grounds by the Court of Appeal and is currently under consideration by the UK Supreme Court.

Edwards-Leeper argue that U.S. practitioners are already moving too quickly to hormonal interventions. (Edwards-Leeper & Anderson 2021; Davis 2022.) It is evident that opinions and practices are all over the map.

84. It is true that a committee of the American Academy of Pediatrics has issued a statement supporting administration of puberty blockers to children diagnosed with gender dysphoria. It is also true that no other American medical association has endorsed the use of puberty blockers, and that pediatricians are neither endocrinologists nor psychiatrists. Dr. James Cantor published a peer-reviewed paper detailing that the Academy's statement is not evidence-based and misdescribed the few scientific sources it did reference. (Cantor 2019.) It has been well noted in the field that the AAP has declined invitations to publish any rebuttal to Dr. Cantor's analysis. But this is all part of ongoing debate, simply highlighting the absence of any generally agreed standard of care.

85. Dr. Adkins asserts that the Society's 2017 Practice Guidelines on Endocrine Treatment of Gender-Dysphoric/Gender-Incongruent Persons (Hembree et al. 2017) amount to "widely accepted standards of care" that were "developed through rigorous scientific processes." (Adkins at 2, 5 and 6.)

86. Contrary to Dr. Adkins' assertion, the 2017 Endocrine Society Guidelines themselves expressly state that they are *not* "standards of care." The document states: "The guidelines cannot guarantee any specific outcome, *nor do they establish a standard of care*. The guidelines are not intended to dictate the treatment of a particular patient." (Hembree et al. 2017 at 3895 (emphasis added).) Nor do the Guidelines claim to be the result of a "rigorous scientific process." Rather, they expressly advise that their recommendations concerning use of puberty blockers are based only on "low quality" evidence.

87. Dr. Adkins notes that the 2017 Guidelines assert that: “patients with gender dysphoria often must be treated with ‘a safe and effective hormone regimen. . .’” (Adkins at 6.) Notably, however, the Guidelines do not make any firm statement that use of puberty blockers for this purpose *is* safe, and the Guidelines go no further than “suggest[ing]” use of puberty blockers—language the Guidelines warn represents only a “weak recommendation.” (Hembree 2017 at 3872.) Several authors have pointed out that not only were the Endocrine Society suggestions regarding use of puberty blockers reached on the basis of “low quality” evidence, but its not-quite claims of ‘safety’ and ‘efficacy’ are starkly contradicted by several in-depth evidence reviews. (Laidlaw et al., 2019; Malone et al. 2021.) I detail these contradictory findings in more detail in Section VII below.

88. While there is too little meaningful clinical data and no consensus concerning best practices or a “standard of care” this area, there are long-standing ethical principles that do or should bind all medical and mental health professionals as they work with, counsel, and prescribe for these individuals.

89. One of the oldest and most fundamental principles guiding medical and psychological care—part of the Hippocratic Oath—is that the physician must “do no harm.” This states an ethical responsibility that cannot be delegated to the patient. Physicians themselves must weigh the risks of treatment against the harm of not treating. If the risks of treatment outweigh the benefits, principles of medical ethics prohibit the treatment.

IV. TRANSGENDER IDENTITY IS NOT BIOLOGICALLY BASED.

90. Dr. Safer asserts that “Although the detailed mechanisms are unknown, there is a medical consensus that there is a significant biologic component underlying gender identity” and

that gender identity is a “largely biological phenomenon.” (Safer at 5, 6.) Many advocates of affirmative care assert this belief.

91. However, it is not true. There is no medical consensus that transgender identity has any biological basis. Furthermore, there is considerable well-documented evidence that is inconsistent with the hypothesis of a biological basis for gender identity—at least in the large majority of currently-presenting patients.

A. No theory of biological basis has been scientifically validated.

92. At the outset, the attempt to identify a single “typically . . . biological” cause for psychiatric conditions (including gender dysphoria) has been strongly criticized as “out of step with the rest of medicine” and as a lingering “ghost” of an understanding of the nature of psychiatric conditions that is now broadly disproven. (Kendler 2019 at 1088-1089.) Gender dysphoria is defined and diagnosed only as a psychiatric, not a medical, condition.

93. Nonetheless, in a published article, Dr. Safer has referred to data that he asserts supports the existence of “a fixed, biologic basis for gender identity.” (Saraswat et al. 2015 at 199.) But on the contrary, this article itself states that studies attempting to find an association between genetics and transgender identification “have been contradictory,” and that “no statistically significant association between particular genes [and transgender identity] has been described.” (Saraswat 2015 at 202.)

94. Similarly, while some have pointed to very small brain scan studies as evidence of a biological basis, no studies of brain structure of individuals identifying as transgender have found any statistically significant correlation between any distinct structure or pattern and transgender identification, after controlling for sexual orientation and exposure to exogenous hormones. (Sarawat et al. 2015 at 202; Frigerio et al. 2021.)

95. Indeed, the Endocrine Society 2017 Guidelines recognizes: “With current knowledge, we cannot predict the psychosexual outcome for any specific child” and “there are currently no criteria to identify the GD/gender-incongruent children to whom this applies. At the present time, clinical experience suggests that persistence of GD/gender incongruence can only be reliably assessed after the first signs of puberty.” (Hembree et al. 2017 at 3876.)

96. In short, no biological test or measurement has been identified that provides any ability to predict which children will exhibit, and which children will persist in, gender dysphoria or a transgender identification. Unless and until such a test is identified, the theory of a biological basis is a hypothesis still searching for support. A hypothesis is not a fact, and responsible scientists will not confuse hypothesis with fact.

B. Large changes across time and geography in the epidemiology of transgender identification are inconsistent with the hypothesis of a biological basis for transgender identity.

97. In fact, there is substantial evidence that the “biological basis” theory is incorrect, at least with respect to the large majority of patients presenting with gender dysphoria today.

98. **Vast changes in incidence:** Historically, there were very low reported rates of gender dysphoria or transgender identification. In 2013, the DSM-5 estimated the incidence of gender dysphoria in adults to be at 2-14 per 100,000, or between 0.002% and 0.014%. (APA 2013 at 454.) Recently however, these numbers have increased dramatically, particularly in adolescent populations. Recent surveys estimate that between 2-9% of high school students self-identify as transgender or “gender non-conforming,” with a significantly large increase in adolescents claiming “nonbinary” gender identity as well. (Johns et al. 2019; Kidd et al. 2021.) Consistent with these surveys, gender clinics around the world have seen numbers of referrals increase rapidly in the last decade, with the Tavistock clinic in London seeing a 30-fold increase in the last decade (GIDS 2019), and similar increases being observed in Finland (Kaltiala-Heino

et al. 2018), the Netherlands (de Vries 2020), and Canada (Zucker 2019). The rapid change in the number of individuals experiencing gender dysphoria points to social and cultural, not biological, causes.

99. **Large change in sex ratio:** In recent years there has been a marked shift in the sex ratio of patients presenting with gender dysphoria or transgender identification. The Tavistock clinic in London saw a ratio of 4 biological females(F):5 biological males(M) shift to essentially 11F:4M in a decade. (GIDS 2019.) One researcher summarizing multiple sources documented a swing of 1F:2M or 1F:1.4M through 2005 to 2F:1M generally (but as high as 7F:1M) in more recent samples. (Zucker 2019 at 2.) This phenomenon has been noted by Dr. Erica Anderson, who said: “The data are very clear that adolescent girls are coming to gender clinics in greater proportion than adolescent boys. And this is a change in the last couple of years. And it’s an open question: What do we make of that? We don’t really know what’s going on. And we should be concerned about it.” (Davis 2022.) Again, this large and rapid change in who is experiencing gender dysphoria points to social, not biological, causes.

100. **Clustering:** Dr. Littman’s recent study documented “clustering” of new presentations of gender dysphoria among natal females in specific schools and among specific friend groups. This again points strongly to social causes for gender dysphoria at least among the adolescent female population. (Littman 2018.)

101. **Desistance:** As I discuss later, there are very high levels of desistance among children diagnosed with gender dysphoria, as well as increasing (or at least increasingly vocal) numbers of individuals who first asserted a transgender identity during or after adolescence, underwent substantial medical interventions to “affirm” that trans-identity, and then “desisted”

and reverted to a gender identity congruent with their sex. (See Section V.B below.) These narratives, too, point to a social and/or psychological cause, rather than a biological one.

102. **“Fluid” gender identification:** Advocates and some practitioners assert that gender identity is not binary, but can span an almost endless range of gender identity self-labels, which a given individual may try on, inhabit, and often discard. (A recent article identifies 72.⁵) I have not heard any theory offered for how there is or could be a biological basis for gender identity as now expansively defined.

103. I frequently read attempts to explain away the points in this Section IV. They include: these problems always existed, but children are now learning that there are effective treatments for their dilemma and are simply seeking them. And; children have hidden their trans identity throughout childhood and now that trans people are recognized and accepted, they are presenting themselves. And; now pediatricians realize that girls can have gender dysphoria and are referring them to gender clinics. But these are all mere hypotheses unsupported by concrete evidence. One set of unproven hypotheses cannot provide support for the unproven hypothesis of biological basis. And none of these hypotheses could even potentially explain the failure of science thus far to identify any predictive biological marker of transgender identification.

104. **Therapies affect gender identity outcomes:** Finally, the evidence shows that therapeutic choices can have a powerful effect on whether and how gender identity does change, or gender dysphoria desists. Social transition of juveniles, for instance, strongly influences gender identity outcomes to such an extent that it has been described a “unique predictor of

⁵ Allarakha, *What Are the 72 Other Genders?*, MedicineNet, available at: https://www.medicinenet.com/what_are_the_72_other_genders/article.htm

persistence.” (See Section V.B below.) Again, this observation cuts against the hypothesis of biological origin.

C. Disorders of sexual development (or DSDs) and gender identity are very different phenomena, and it is an error to conflate the two.

105. Dr. Adkins spends much of her report discussing individuals who suffer from disorders of sexual development (DSDs), apparently as evidence that sex is not binary or clearly defined, or as somehow supporting the idea that transgender identification has a biological basis. (Adkins at 9.) I have extensively detailed that sex is clear, binary, and determined at conception. (Section II.) Here I explain that gender dysphoria is an entirely different phenomenon than DSDs—which unlike transgender identity are indeed biological phenomena. It is an error to conflate the two distinct concepts.

106. Every DSD reflects a genetic enzymatic defect with negative anatomic and physiological consequences. As the Endocrine Society recognized in a 2021 statement: “Given the complexities of the biology of sexual determination and differentiation, it is not surprising that there are dozens of examples of variations or errors in these pathways associated with genetic mutations that are now well known to endocrinologists and geneticists; in medicine, these situations are generally termed *disorders of sexual development* (DSD) or *differences in sexual development*.” Gender Identity on the other hand is uniformly defined as a subjective “sense” of being, a feeling or state of mind. (Section II.C.)

107. The vast majority of those who experience gender dysphoria or a transgender identity do not suffer from any DSD, nor from any genetic enzymatic disorder at all. Conversely, many who suffer from a DSD do not experience a gender identity different from their chromosomal sex (although some may). In short, those who suffer from gender dysphoria are not a subset of those who suffer from a DSD, nor are those who suffer from a DSD a subset of those

who suffer from gender dysphoria. The two are simply different phenomena, one physical, the other mental, defined only as a psychiatric condition. The issue here is not whether biological forces play a role in personality development; it is whether there is strong evidence that it is determinative. Science has come too far to revert to single explanations for gender dysphoria or any psychiatric diagnosis.

108. The importance of this distinction is evident from the scientific literature. For example, in a recent study of clinical outcomes for gender dysphoric patients, Tavistock Clinic researchers *excluded* from their analysis any patients who did not have “normal endocrine function and karyotype consistent with birth registered sex.” (Carmichael et al. 2021 at 4.) In other words, the researchers specifically *excluded* from their study anyone who suffered from genetic-based DSD, or a DSD comprising any serious defect in hormonal use pathways, in order to ensure the study was focused only on individuals experiencing the psychological effects of what we might call “ordinary” gender dysphoria.

D. Studies of individuals born with DSDs suggest that there may be a biological predisposition towards *typical* gender identifications, but provide no support for a biological basis for *transgender* identification.

109. Studies of individuals born with serious DSDs have been pointed to as evidence of a biological basis for transgender identification. They provide no such support.

110. One well-known study by Meyer-Bahlburg reviewed the case histories of a number of XY (i.e. biologically male) individuals born with severe DSDs who were surgically “feminized” in infancy and raised as girls. (Meyer-Bahlburg 2005.) The majority of these individuals nevertheless later adopted male gender identity—suggesting a strong biological predisposition towards identification aligned with genetic sex, even in the face of feminized genitalia from earliest childhood, and parental “affirmation” in a transgender identity. But at the same time, the fact that some of these genetically male individuals did *not* later adopt male

gender identity serves as evidence that medical and social influences can indeed encourage and sustain transgender identification.

111. Importantly, the Meyer-Bahlburg study did *not* include any individuals who were assigned a gender identity congruent with their genetic sex who subsequently adopted a transgender identity. Therefore, the study can provide no evidence of any kind that supports the hypothesis of a biological basis for transgender identity. A second study in this area (Reiner & Gearhart 2004) likewise considered exclusively XY subjects, and similarly provides evidence only for a biological bias towards a gender identity congruent with one’s genetic sex, even in the face of medical and social “transition” interventions. None of this provides any evidence at all of a biological basis for transgender identity.

V. GENDER IDENTITY IS EMPIRICALLY NOT FIXED FOR MANY INDIVIDUALS.

112. Dr. Safer states that gender identity is “durable and cannot be changed by medical intervention.” (Safer at 5.) Dr. Adkins likewise states that gender identity “cannot be voluntarily changed.” (Adkins at 4.) There is extensive evidence that this is not correct. Instead, gender identity changes over time for many individuals.⁶ I summarize their two opinions as: they assert that a trans identity in a child or adolescent is immutable—unchangeable by medical, psychotherapeutic, or developmental processes.

A. Most children who experience gender dysphoria ultimately “desist” and resolve to cisgender identification.

113. A distinctive and critical characteristic of juvenile gender dysphoria is that multiple studies from separate groups and at different times have reported that in the large

⁶ See n1 *supra*.

majority of patients, absent a substantial intervention such as social transition or puberty blocking hormone therapy, it does *not* persist through puberty.

114. A recent article reviewed all existing follow-up studies that the author could identify of children diagnosed with gender dysphoria (11 studies), and reported that “every follow-up study of GD children, without exception, found the same thing: By puberty, the majority of GD children ceased to want to transition.” (Cantor 2019 at 1.) Another author reviewed the existing studies and reported that in “prepubertal boys with gender discordance . . . the cross gender wishes usually fade over time and do not persist into adulthood, with only 2.2% to 11.9% continuing to experience gender discordance.” (Adelson et al. 2012 at 963; see also Cohen-Kettinis 2008 at 1895.) The Endocrine Society recognized this important baseline fact in its 2017 Guidelines. (Hembree 2017 at 3879.) It should be noted that the reason that the Dutch Protocol waited until age 14 to initiate puberty blockers was that it was well known that many children would desist if left free of hormonal intervention until that age.

115. Findings of high levels of desistance among children who experience gender dysphoria or incongruence have been reaffirmed in the face of critiques through thorough reanalysis of the underlying data. (Zucker 2018.)

116. As I explained in detail in Section IV above, it is not yet known how to distinguish those children who will desist from that small minority whose trans identity will persist.

117. It does appear that prevailing circumstances during particularly formative years can have a significant impact on the outcome of a juvenile’s gender dysphoria. A 2016 study reviewing the follow-up literature noted that “the period between 10 and 13 years” was “crucial” in that “both persisters and desisters stated that the changes in their social environment, the

anticipated and actual feminization or masculinization of their bodies, and the first experiences of falling in love and sexual attraction in this period, contributed to an increase (in the persisters) or decrease (in the desisters) of their gender related interests, behaviors, and feelings of gender discomfort.” (Ristori & Steensma 2016 at 16.) As I discuss in Section VI below, there is considerable evidence that early transition and affirmation causes far more children to persist in a transgender identity.

B. Desistance is increasingly observed among teens and young adults who first manifest GD during or after adolescence.

118. Desistance within a relatively short period may also be a common outcome for post-pubertal youths who exhibit recently described “rapid onset gender disorder.” I have observed an increasingly vocal online community of young women who have reclaimed a female identity after claiming a male gender identity at some point during their teen years, and young “detransitioners” (individuals in the process of reidentifying with their birth sex after having undergone a gender transition) are now receiving increasing attention in both clinical literature and social media channels. (It is my understanding that March 12, 2022, is scheduled to be Detransition Awareness Day.)

119. Almost all scientific articles on this topic have appeared within the last few years. Perhaps this historic lack of coverage is not entirely surprising – one academic who undertook an extensive review of the available scientific literature in 2021 noted that the phenomenon was “socially controversial” in that it “poses significant professional and bioethical challenges for those clinicians working in the field of gender dysphoria.” (Expósito Campos 2021 at 270.) This review reported on multifarious reasons for why individuals were motivated to detransition, which included coming to “understand[] how past trauma, internalized sexism, and other psychological difficulties influenced the experience of GD.”

120. In 2021, Lisa Littman of Brown University conducted a ground-breaking study of 100 teenage and young adults who had transitioned and lived in a transgender identity for a number of years, and then “detransitioned” or changed back to a gender identity matching their sex. Littman noted that the “visibility of individuals who have detransitioned is new and may be rapidly growing.” (Littman 2021 at 1.) Of the 100 detransitioners included in Littman’s study, 60% reported that their decision to detransition was motivated (at least in part) by the fact that they had become more comfortable identifying as their natal sex, and 38% had concluded that their gender dysphoria was caused by something specific such as trauma, abuse, or a mental health condition. (Littman 2021 at 9.)

121. A significant majority (76%) did not inform their clinicians of their detransition. (Littman 2021 at 11.)

122. A similar study that recruited a sample of 237 detransitioners (the large majority of whom had initially transitioned in their teens or early twenties) similarly reported that a common reason for detransitioning was the subject’s conclusion that his or her gender dysphoria was related to other issues (70% of the sample). (Vandenbussche 2021.)

123. The existence of increasing numbers of youth or young adult detransitioners has also been recently noted by Dr. Edwards-Leeper and Dr. Anderson. (Edwards-Leeper & Anderson 2021.) Edwards-Leeper and Anderson noted “the rising number of detransitioners that clinicians report seeing (they are forming support groups online)” which are “typically youth who experienced gender dysphoria and other complex mental health issues, rushed to medicalize their bodies and regretted it.” Other clinicians working with detransitioners have also noted the recent phenomenon. (Marchiano 2020.)

124. A growing body of evidence suggests that for many teens and young adults, a post-pubertal onset of transgender identification can be a transient phase of identity exploration, rather than a permanent identity, as evidenced by a growing number of young detransitioners (Entwistle 2020; Littman 2021; Vandebussche 2021). Previously, the rate of detransition and regret was reported to be very low, although these estimates suffered from significant limitations and were likely undercounting true regret (D'Angelo 2018). As gender-affirmative care has become popularized, the rate of detransition appears to be accelerating.

125. A recent study from a UK adult gender clinic observed that 6.9% of those treated with gender-affirmative interventions detransitioned within 16 months, and another 3.4% had a pattern of care suggestive of detransition, yielding a rate of probable detransition in excess of 10%. Another 21.7%, however, disengaged from the clinic without completing their treatment plan. While some of these individuals later re-engaged with the gender service, the authors concluded, “detransitioning might be more frequent than previously reported.” (Hall et al. 2021).

126. Another study from a UK primary care practice found that 12.2% of those who had started hormonal treatments either detransitioned or documented regret, while the total of 20% stopped the treatments for a wider range of reasons. The mean age of their presentation with gender dysphoria was 20, and the patients had been taking gender-affirming hormones for an average 5 years (17 months-10 years) prior to discontinuing. Comparing these much higher rates of treatment discontinuation and detransition to the significantly lower rates reported by the older studies, the researchers noted: “Thus, the detransition rate found in this population is novel and questions may be raised about the phenomenon of overdiagnosis, overtreatment, or iatrogenic harm as found in other medical fields” (Boyd et al. 2022 at 15.) Indeed, given that regret may take up to 8-11 years to materialize (Dhejne et al., 2014; Wiepjes et al., 2018), many more

detransitioners are likely to emerge in the coming years. Detransitioner research is still in its infancy, but the Littman and Vandenbussche studies in 2021 both report that detransitioners from the recently transitioning cohorts feel they were rushed into medical gender-affirmative interventions with irreversible effects, often without the benefit of appropriate, or in some instances any, psychologic exploration.

VI. TRANSITION AND AFFIRMATION IS AN IMPORTANT PSYCHOLOGICAL AND MEDICAL INTERVENTION THAT CHANGES GENDER IDENTITY OUTCOMES.

A. If both a typical gender or a transgender long-term gender identity outcome are possible for a particular patient, the alternatives are not medically neutral.

127. Where a juvenile experiences gender dysphoria, the gender identity that is stabilized will have a significant impact on the course of their life. Living in a transgender identity for a time will make desistance, if it is ever considered, more difficult to accomplish.

128. If the juvenile desists from the gender dysphoria and becomes reasonably comfortable with a gender identity congruent with their sex—the most likely outcome from a statistical perspective absent affirming intervention—the child will not require ongoing pharmaceutical maintenance and will not have their fertility destroyed post-puberty.

129. However, if the juvenile persists in a transgender identity, under current practices, the child is most likely to require regular administration of hormones for the rest of their lives, exposing them to significant physical, mental health, and relational risks (which I detail in Section IX below), as well as being irreversibly sterilized chemically and/or surgically. The child is therefore rendered a “patient for life” with complex medical implications further to a scientifically unproven course of treatment.

B. Social transition of young children is a powerful psychotherapeutic intervention that radically changes outcomes, almost eliminating desistance.

130. Dr. Adkins asserts that social transition is a “a critical part” of the treatment of gender dysphoria. (Adkins at 6, 7). Rather, social transition has a critical *effect* on the persistence of gender dysphoria. It is evident from the scientific literature that engaging in therapy that encourages social transition before or during puberty—which would include participation on athletic teams designated for the opposite sex—is a psychotherapeutic intervention that dramatically changes outcomes. A prominent group of authors has written that “The gender identity affirmed during puberty appears to predict the gender identity that will persist into adulthood.” (Guss et al. 2015 at 421.) Similarly, a comparison of recent and older studies suggests that when an “affirming” methodology is used with children, a substantial proportion of children who would otherwise have desisted by adolescence—that is, achieved comfort identifying with their natal sex—instead persist in a transgender identity. (Zucker 2018 at 7.)

131. Indeed, a review of multiple studies of children treated for gender dysphoria across the last three decades found that early social transition to living as the opposite sex severely reduces the likelihood that the child will revert to identifying with the child’s natal sex, at least in the case of boys. That is, while, as I review above, studies conducted before the widespread use of social transition for young children reported desistance rates in the range of 80-98%, a more recent study reported that fewer than 20% of boys who engaged in a partial or complete social transition before puberty had desisted when surveyed at age 15 or older. (Zucker 2018 at 7⁷; Steensma et al. 2013.)⁸ Another researcher observed that a partial or complete gender

⁷ Zucker found social transition by the child to be strongly correlated with persistence for natal boys, but not for girls. (Zucker 2018 at 5.)

⁸ Only 2 (3.6%) of 56 of the male desisters observed by Steensma et al. had made a complete or partial transition prior to puberty, and of the twelve males who made a complete or

social transition prior to puberty “proved to be a unique predictor of persistence.” (Singh et al. 2021 at 14.)

132. Some vocal practitioners of prompt affirmation and social transition even proudly claim that essentially *no* children who come to their clinics exhibiting gender dysphoria or cross-gender identification desist in that identification and return to a gender identity consistent with their biological sex.⁹ This is a very large change as compared to the desistance rates documented apart from social transition.

133. Even voices generally supportive of prompt affirmation and social transition are acknowledging a causal connection between social transition and this change in outcomes. As the Endocrine Society recognized in its 2017 Guidelines: “If children have completely socially transitioned, they may have great difficulty in returning to the original gender role upon entering puberty. . . [S]ocial transition (in addition to GD/gender incongruence) has been found to contribute to the likelihood of persistence.” (Hembree et al. 2017 at 3879.) The fact is that these unproven interventions with the lives of kids and their families have systematically documented outcomes. Given this observed phenomenon, I agree with Dr. Ken Zucker who has written that social transition in children must be considered “a form of psychosocial treatment.” (Zucker 2020 at 1.)

134. Moreover, as I review below, social transition cannot be considered or decided alone. Studies show that engaging in social transition starts a juvenile on a “conveyor belt” path

partial transition prior to puberty, only two had desisted when surveyed at age 15 or older. Steensma 2013 at 584.

⁹ See, e.g., Ehrensaft 2015 at 34: “In my own clinical practice . . . of those children who are carefully assessed as transgender and who are allowed to transition to their affirmed gender, we have no documentation of a child who has ‘desisted’ and asked to return to his or her assigned gender.”

that almost inevitably leads to the administration of puberty blockers, which in turn almost inevitably leads to the administration of cross-sex hormones. The emergence of this well-documented path means that the implications of taking puberty blockers *and* cross-sex hormones must be taken into account even where “only” social transition is being considered or requested by the child or family. As a result, there are a number of important “known risks” associated with social transition.

C. Administration of puberty blockers is a powerful medical and psychotherapeutic intervention that radically changes outcomes, almost eliminating desistance on the historically observed timeline.

135. Dr. Adkins speaks of the use of puberty blockers as though this major hormonal disruption of some of the most basic aspects of ordinary human development were entirely benign, acting as a “pause.” (Adkins at 7.) This optimistic view is not based on science. In fact, it should be understood that puberty blockers are usually administered to early-stage adolescents as part of a path that includes social transition. Moreover, medicine does not know what the long-term health effects on bone, brain, and other organs are of a “pause” between ages 11-16. Medicine also does not know if the long-term effects of these compounds are different in boys than in girls. The mental health professional establishment likewise does not know the long-term effects on coping skills, interpersonal comfort, and intimate relationships of this “pause” while one’s peers are undergoing their maturational gains in these vital arenas of future mental health. I address medical, social, and mental health risks associated with the use of puberty blockers in Section IX. Here, I note that the data strongly suggests that the administration of puberty blockers, too, must be considered to be a component of a “psychosocial treatment” with complex implications, rather than a “pause.”

136. Multiple studies show that the large majority of children who begin puberty blockers go on to receive cross-sex hormones. (de Vries 2020 at 2.) A recent study by the

Tavistock and Portman NHS Gender Identity Development Service (UK)—the world’s largest gender clinic—found that 98% of adolescents who underwent puberty suppression continued on to cross-sex hormones. (Carmichael et al 2021 at 12.)¹⁰

137. These studies demonstrate that going on puberty blockers virtually eliminates the possibility of desistance in juveniles. Rather than a “pause,” puberty blockers appear to act as a psychosocial “switch,” decisively shifting many children to a persistent transgender identity. Therefore, as a practical and ethical matter the decision to put a child on puberty blockers must be considered as the equivalent of a decision to put that child on cross-sex hormones, with all the considerations and informed consent obligations implicit in that decision.

VII. TRANSITION AND AFFIRMATION ARE EXPERIMENTAL THERAPIES THAT HAVE NOT BEEN SHOWN TO IMPROVE MENTAL OR PHYSICAL HEALTH OUTCOMES BY YOUNG ADULTHOOD.

138. It is undisputed that children and adolescents who present with gender dysphoria exhibit a very high level of mental health comorbidities. (Section II.C.) Whether the gender dysphoria is cause or effect of other diagnosed or undiagnosed mental health conditions, or whether these are merely coincident comorbidities, is hotly disputed, but the basic fact is not.

139. Dr. Adkins asserts that when the “transition, affirmation, and hormones” therapy that she advocates is followed, “gender dysphoria is easily managed” (Adkins at 5), implying that transition and hormone therapy have been proven to be effective in relieving gender dysphoria and the general mental health distress that broadly afflicts these children and adolescents. This is scientifically incorrect. It ignores both what is known and what is unknown.

¹⁰ See also Brik 2020 where Dutch researchers found nearly 97% of adolescents who received puberty blockers proceeded to cross-sex hormones.

A. The knowledge base concerning therapies for gender dysphoria is “very low quality.”

140. At the outset, it is important for all sides to admit that the knowledge base concerning the causes and treatment of gender dysphoria has low scientific quality.

141. In evaluating claims of scientific or medical knowledge, it is axiomatic in science that no knowledge is absolute, and to recognize the widely accepted hierarchy of reliability when it comes to “knowledge” about medical or psychiatric phenomena and treatments. Unfortunately, in this field opinion is too often confused with knowledge, rather than clearly locating what exactly is scientifically known. In order of increasing confidence, such “knowledge” may be based upon data comprising:

- a. Expert opinion—it is perhaps surprising to educated laypersons that expert opinion standing alone is the lowest form of knowledge, the least likely to be proven correct in the future, and therefore does not garner as much respect from professionals as what follows;

- b. A single case or series of cases (what could be called anecdotal evidence) (Levine 2016 at 239.);

- c. A series of cases with a control group;

- d. A cohort study;

- e. A randomized double-blind clinical trial;

- f. A review of multiple trials;

- g. A meta-analysis of multiple trials that maximizes the number of patients treated despite their methodological differences to detect trends from larger data sets.

142. Prominent voices in the field have emphasized the severe lack of scientific knowledge in this field. The American Academy of Child and Adolescent Psychiatry has

recognized that “Different clinical approaches have been advocated for childhood gender discordance. . . . There have been no randomized controlled trials of any treatment. . . . [T]he proposed benefits of treatment to eliminate gender discordance . . . must be carefully weighed against . . . possible deleterious effects.” (Adelson et al. at 968–69.) Similarly, the American Psychological Association has stated, “because no approach to working with [transgender and gender nonconforming] children has been adequately, empirically validated, consensus does not exist regarding best practice with pre-pubertal children.” (APA 2015 at 842.)

143. Critically, “there are no randomized control trials with regard to treatment of children with gender dysphoria.” (Zucker 2018 at 8.) On numerous critical questions relating to cause, developmental path if untreated, and the effect of alternative treatments, the knowledge base remains primarily at the level of the practitioner’s exposure to individual cases, or multiple individual cases. As a result, claims to certainty are not justifiable. (Levine 2016 at 239.)

144. Within the last two years, at least three formal evidence reviews concerning hormonal interventions for gender dysphoria have been conducted. All three found all of the available clinical evidence to be very low quality.

145. The British National Health Service (NHS) commissioned formal “evidence reviews” of all clinical papers concerning the efficacy and safety of puberty blockers and cross-sex hormones as treatments for gender dysphoria. These evidence reviews were performed by the U.K. National Institute for Health and Care Excellence (NICE), applying the respected “GRADE” criteria for evaluating the strength of clinical evidence.

146. Both the review of evidence concerning puberty blockers and the review of evidence concerning cross-sex hormones were published in 2020, and both found that *all* available evidence as to both efficacy and safety was “very low quality” according to the

GRADE criteria. (NICE 2021a; NICE 2021b.) “Very low quality” according to GRADE means there is a high likelihood that the patient *will not experience* the hypothesized benefits of the treatment. (Balshem et al. 2011.)

147. Similarly, the highly respected Cochrane Library—the leading source of independent systematic evidence reviews in health care—commissioned an evidence review concerning the efficacy and safety of hormonal treatments now commonly administered to “transitioning transgender women” (i.e., testosterone suppression and estrogen administration to biological males). That review, also published in 2020, concluded that “We found insufficient evidence to determine the efficacy or safety of hormonal treatment approaches for transgender women in transition.” (Haupt et al. 2020 at 2.) It must be understood that both the NICE and the Cochrane reviews considered *all* published scientific studies concerning these treatments.

148. As to social transition, as I have noted above, considerable evidence suggests that socially transitioning a pre-pubertal child puts him or her on a path from which very few children escape—a path which includes puberty blockers and cross-sex hormones before age 18. As a practical matter, then, a decision about social transition for a child must be made in light of what is known and what is unknown about the effects of those expected hormonal interventions.

149. I discuss safety considerations in Section IX below. Here, I detail what is known about the effectiveness of social and hormonal transition and affirmation to improve the mental health of individuals diagnosed with gender dysphoria.

B. Youth who adopt a transgender identity show no durable improvement in mental health after social, hormonal, or surgical transition and affirmation.

150. As I noted above, the evidence reviews for the efficacy and safety of hormonal interventions published in 2020 concluded that the supporting evidence is so poor that there is “a

high likelihood that the patient will not experience the hypothesized benefits of the treatment.”

There is now some concrete evidence that on average they do not experience those benefits.

151. An important paper published in 2021 by Tavistock clinic clinicians provided the results of the first longitudinal study that measured widely used metrics of general psychological function and suicidality before commencement of puberty blockers, and then at least annually after commencing puberty blockers. After up to three years, they “found no evidence of change in psychological function with GnRHa treatment as indicated by parent report (CBCL) or self-report (YSR) of overall problems, internalizing or externalizing problems or self-harm” as compared to the pre-puberty-blocker baseline evaluations. “Outcomes that were not formally tested also showed little change.” (Carmichael et al. 2021 at 18-19.) Similarly, a study by Branström and Pachankis of the case histories of a set of individuals diagnosed with GD in Sweden found no positive effect on mental health from hormonal treatment. (Landen 2020.)

152. A cohort study by authors from Harvard and Boston Children’s Hospital found that youth and young adults (ages 12-29) who self-identified as transgender had an elevated risk of depression (50.6% vs. 20.6%) and anxiety (26.7% vs. 10.0%); a higher risk of suicidal ideation (31.1% vs. 11.1%), suicide attempts (17.2% vs. 6.1%), and self-harm without lethal intent (16.7% vs. 4.4%) relative to the matched controls; and a significantly greater proportion of transgender youth accessed inpatient mental health care (22.8% vs. 11.1%) and outpatient mental health care (45.6% vs. 16.1%) services. (Reisner et al. 2015 at 6.) Similarly, a recent longitudinal study of transgender and gender diverse youth and young adults in Chicago found rates of alcohol and substance abuse “substantially higher than those reported by large population-based studies of youth and adults.” (Newcomb et al. 2020 at 14.) Members of the clinical and research team at the prominent Dutch VU University gender dysphoria center recently compared mental

health metrics of two groups of subjects before (mean age 14.5) and after (mean age 16.8) puberty blockers. But they acknowledged that the structure of their study meant that it “can . . . not provide evidence about . . . long-term mental health outcomes,” and that based on what continues to be extremely limited scientific data, “Conclusions about the long-term benefits of puberty suppression should . . . be made with extreme caution.” In other words, we just don’t know. (van der Miesen et al. 2020 at 703.)

153. Kiera Bell, who was diagnosed with gender dysphoria at the Tavistock Clinic, given cross-sex hormones, and subjected to a mastectomy, before desisting and reclaiming her female gender identity, and a Swedish teen girl who appeared in a recent documentary after walking that same path, have both stated that they feel that they were treated “like guinea pigs,” experimental subjects. They are not wrong.

C. Long-term mental health outcomes for individuals who persist in a transgender identity are poor.

154. The responsible MHP cannot focus narrowly on the short-term happiness of the young patient, but must instead consider the happiness and health of the patient from a “life course” perspective. When we look at the available studies of individuals who continue to inhabit a transgender identity across adult years, the results are strongly negative.

155. In the United States, the death rates of trans veterans are comparable to those with schizophrenia and bipolar diagnoses—20 years earlier than expected. These crude death rates include significantly elevated rates of substance abuse as well as suicide. (Levine 2017 at 10.) Similarly, researchers in Sweden and Denmark have reported on almost all individuals who underwent sex-reassignment surgery over a 30-year period. (Dhejne et al. 2011; Simonsen et al. 2016.) The Swedish follow-up study similarly found a suicide rate in the post-SRS population

19.1 times greater than that of the controls; both studies demonstrated elevated mortality rates from medical and psychiatric conditions. (Levine 2017 at 10.)

156. A recent study in the American Journal of Psychiatry reported high mental health utilization patterns of adults for ten years after surgery for approximately 35% of patients. (Bränström & Panchankis, 2020.) Indeed, earlier Swedish researchers in a long-term study of all patients provided with SRS over a 30-year period (median time since SRS of > 10 years) concluded that individuals who have SRS exhibit such poor mental health that they should be provided very long-term psychiatric care as the “final” transition step of SRS. (Dhejne et al. 2011, at 6-7.) Unfortunately, across the succeeding decade, in Sweden and elsewhere their suggestion has been ignored.

157. I will note that these studies do not tell us whether the subjects first experienced gender dysphoria as children, adolescents, or adults, so we cannot be certain how their findings apply to each of these subpopulations which represent quite different pathways. But in the absence of knowledge, we should be cautious.

158. Meanwhile, no studies show that affirmation of pre-pubescent children or adolescents leads to more positive outcomes (mental, physical, social, or romantic) by, e.g., age 25 or older than does “watchful waiting” or ordinary therapy.

159. The many studies that I have cited here warn us that as we look ahead to the patient’s life as a young adult and adult, the prognosis for the physical health, mental health, and social well-being of the child or adolescent who transitions to live in a transgender identity is not good. Gender dysphoria is not “easily managed” when one understands the marginalized, vulnerable physical, social, and psychological status of adult trans populations.

VIII. TRANSITION AND AFFIRMATION DO NOT DECREASE, AND MAY INCREASE, THE RISK OF SUICIDE.

A. The risk of suicide among transgender youth is confused and exaggerated in the public mind.

160. While suicide is closely linked to mental health, I comment on it separately because rhetoric relating to suicide figures so prominently in debates about responses to gender dysphoria.

161. At the outset, I will note that any discussion of suicide when considering younger children involves very long-range and very uncertain prediction. Suicide in pre-pubescent children is extremely rare, and the existing studies of gender identity issues in pre-pubescent children do not report significant incidents of suicide. Any suggestion otherwise is misinformed. Our focus for this topic, then, is on adolescents and adults.

162. Some authors have reported rates of suicidal thoughts and behaviors among trans-identifying teens or adults ranging from 25% to as high as 52%, generally through non-longitudinal self-reports obtained from non-representative survey samples. (Toomey et al. 2018.) Dr. Adkins asserted in her declaration submitted in support of Plaintiff's preliminary injunction motion that "Attempted suicide rates in the transgender community are over 40%," and that "[t]he only treatment to avoid this serious harm is to . . . affirm gender identity." (Adkins at 6.) Contrary to these assertions, no studies show that affirmation of children (or anyone else) reduces suicide, prevents suicidal ideation, or improves long-term outcomes, as compared to either a "watchful waiting" or a psychotherapeutic model of response, as I have described above. Rhetorical references to figures such as 40%—and some published studies—confuse suicidal thoughts and actions that represent a cry for help, manipulation, or expression of rage with serious attempts to end life. Such statements or studies ignore a crucial and long-recognized distinction.

163. I have included suicidality in my discussion of mental health above. Here, I focus on actual suicide. Too often, in public comment suicidal thoughts are blurred with suicide. Yet the available data tells us that suicide among children and youth suffering from gender dysphoria is extremely rare.

164. An important new analysis of data covering patients as well as those on the waiting list (and thus untreated) at the UK Tavistock gender clinic—the world’s largest gender clinic—found a total of only four completed suicides across 11 years’ worth of patient data, reflecting an estimated cumulative 30,000 patient-years spent by patients under the clinic’s care or on its waiting list. This corresponded to an annual suicide rate of 0.013%. The proportion of individual patients who died by suicide was 0.03%, which is orders of magnitude smaller than trans adolescents who self-report suicidal behavior or thoughts on surveys. (Biggs 2022b.)

165. Thus, only a minute fraction of trans-identifying adolescents who report thoughts or conduct considered to represent “suicidality” actually commit suicide. I agree with the statement by Dr. Zucker that the assertion by, for example, Karasic and Ehrensaft (2015) that completed suicides among transgender youth are “alarmingly high” “has no formal and systematic empirical basis.” (Zucker 2019 at 3.)

166. Professor Biggs of Oxford, author of the study of incidence of suicide among Tavistock clinic patients, rightly cautions that it is “irresponsible to exaggerate the prevalence of suicide.” (Biggs 2022b at 4.) It is my opinion that telling parents—or even allowing them to believe from their internet reading—that they face a choice between “a live son or a dead daughter” is both factually wrong and unethical. Informed consent requires clinicians to tell the truth and ensure that their patients understand the truth. To be kind, the clinicians who believe

such figures represent high risk of ultimate suicide in adolescence simply do not know the truth; they are ill-informed.

B. Transition of any sort has not been shown to reduce levels of suicide.

167. Every suicide is a tragedy, and steps that reduce suicide should be adopted. I have noted above that suicidality (that is, suicidal thoughts or behaviors, rather than suicide) is common among transgender adolescents and young adults before, during, and after social and medical transition. If a medical or mental health professional believes that an individual he or she is diagnosing or treating for gender dysphoria presents a suicide risk, in my view it is unethical for that professional merely to proceed with treatment for gender dysphoria and hope that “solves the problem.” Rather, that professional has an obligation to provide or refer the patient for evidence-based therapies for addressing depression and suicidal thoughts that are well-known to the profession. (Levine 2016 at 242.)

168. This is all the more true because there is in fact no evidence that social and/or medical transition reduces the risk or incidence of actual suicide. On the contrary, in his analysis of those who were patients of or on the waiting list of the Tavistock clinic, Professor Biggs found that the suicide rate was not higher among those on the clinic’s waiting list (and thus as-yet untreated), than for those who were patients under care. (Biggs 2022b.) And as corrected, Bränström and Pachankis similarly acknowledge that their review of records of GD patients “demonstrated no advantage of surgery in relation to . . . hospitalizations following suicide attempts.” (I assume for this purpose that attempts that result in hospitalization are judged to be so serious as to predict a high rate of future suicide if not successfully addressed.”)¹¹

¹¹ Turban et al. (2020) has been described in press reports as demonstrating that administration of puberty suppressing hormones to transgender adolescents reduces suicide or suicidal ideation. The paper itself does not make that claim, nor permit that conclusion.

C. Long-term life in a transgender identity correlates with very high rates of completed suicide.

169. As with mental health generally, the patient, parent, or clinician fearing the risk of suicide must consider not just the next month or year, but a life course perspective.

170. There are now four long-term studies that analyze completed suicide among those living in transgender identities into adulthood. The results vary significantly, but are uniformly highly negative.

171. Dhejne reported a long-term follow-up study of subjects after sex reassignment surgery. Across the multi-year study, subjects who had undergone SRS committed suicide at 19.1 times the expected rate compared to general population controls matched by age and both sexes. MtF subjects committed suicide at 13.9 times the expected rate, and FtM subjects committed suicide at 40.0 times the expected rate. (Dhejne et al. 2011 Supplemental Table S1.)

172. Asscheman, also writing in 2011, reported results of a long-term follow-up of all transsexual subjects of the Netherlands' leading gender medicine clinic who started cross-sex hormones before July 1, 1997, a total of 1331 patients. Due to the Dutch system of medical and death records, extensive follow-up was achieved. Median follow-up period was 18.5 years. The mortality rate among MtF patients was 51% higher than among the age-matched general population; the rate of completed suicide among MtF patients was six times that of the age-matched general population. (Asscheman et al. 2011.)

173. Importantly, Asscheman et al. found that "No suicides occurred within the first 2 years of hormone treatment, while there were six suicides after 2-5 years, seven after 5-10 years, and four after more than 10 years of CSH treatment at a mean age of 41.5 years." (Asscheman et al. 2011 at 637-638.) This suggests that studies that follow patients for only a year or two after treatment are insufficient. Asscheman et al.'s data suggest that such short-term follow-up is

engaging only with an initial period of optimism, and will simply miss the feelings of disillusion and the increase in completed suicide that follows in later years.

174. A retrospective, long-term study published in 2020 of a very large cohort (8263) of patients referred to the Amsterdam University gender clinic between 1972 and 2017 found that the annual rate of completed suicides among the transgender subjects was “three to four times higher than the general Dutch population.” “[T]he incidence of observed suicide deaths was almost equally distributed over the different stages of treatment.” The authors concluded that “vulnerability for suicide occurs similarly in the different stages of transition.” (Wiepjes et al. 2020.) In other words, neither social nor medical transition reduced the rate of suicide.

175. As with Asscheman et al., Wiepjes et al. found that the median time between start of hormones and suicide (when suicide occurred) was 6.1 years for natal males, and 6.9 years for natal females. Again, short- or even medium-term studies will miss this suicide phenomenon.

176. A 2021 study analyzed the case histories of a cohort of 175 gender dysphoria patients treated at one of the seven UK adult gender clinics who were “discharged” (discontinued as patients) within a selected one-year period. The authors reported the rather shocking result that 7.7% (3/39) of natal males who were diagnosed and admitted for treatment, and who were between 17 and 24 years old, were “discharged” because they committed suicide during treatment. (Hall et al. 2021, Table 2.)

177. None of these studies demonstrates that the hormonal or surgical intervention *caused* suicide. That is possible, but as we have seen, the population that identifies as transgender suffers from a high incidence of comorbidities that correlate with suicide. What these studies demonstrate—at the least—is that this remains a troubled population in need of extensive and careful psychological care that they generally do not receive, and that neither

hormonal nor surgical transition and “affirmation” resolve their underlying problems and put them on the path to a stable and healthy life.

178. In sum, claims that affirmation will reduce the risk of suicide for children and adolescents are not based on science. Instead, transition of any sort must be justified, if at all, as a life-enhancing measure, not a lifesaving measure. (Levine 2016 at 242.) In my opinion, this is an important fact that patients, parents, and even many MHPs fail to understand.

IX. HORMONAL INTERVENTIONS ARE EXPERIMENTAL PROCEDURES THAT HAVE NOT BEEN PROVEN SAFE.

179. Dr. Adkins also appears to assert as a fact—but without citation to peer-reviewed literature—that social transition, puberty blockers, and cross-sex hormones are known to be “safe.” (Adkins at 5-6, 8.) This is not true. And Dr. Adkins, along with a number of voices in the field, also asserts that puberty blockers act merely as a “pause” in the process of puberty-driven maturation, suggesting that this hormonal intervention has been proven to be fully reversible. This is also an unproven belief.

180. On the contrary, no studies have been done that meaningfully demonstrate that either puberty blockers or cross-sex hormones, as prescribed for gender dysphoria, are safe in other than the short run. No studies have attempted to determine whether the effects of puberty blockers, as currently being prescribed for gender dysphoria, are fully reversible. Neither Dr. Adkins nor Dr. Safer cites any such studies, and there are none. There are only pronouncements. In fact, there are substantial reasons for concern that these hormonal interventions are not safe. Multiple researchers have expressed concern that the full range of possible harms have not even been correctly conceptualized.

181. Because, as I have explained in Section VI, recent evidence demonstrates that pre-pubertal social transition almost always leads to progression on to puberty blockers which in turn

almost always leads to the use of cross-sex hormones, physicians bear the ethical responsibility for a thorough informed consent process for parents and patients that includes this fact and its full implications. Informed consent does not mean sharing with the parents and patients what the doctor believes: it means sharing what is known and what is not known about the intervention. So much of what doctors believe is based on mere trust in what they have been taught. Neither they themselves nor their teachers may be aware of the scientific foundation and scientific limitations of what they are recommending.

A. Use of puberty blockers has not been shown to be safe or reversible for gender dysphoria.

182. As I noted above, the recent very thorough literature review performed for the British NHS concluded that *all* available clinical evidence relating to “safety outcomes” from administration of puberty blockers for gender dysphoria is of “very low certainty.” (NHS 2020a at 6.)

183. In its 2017 Guidelines, the Endocrine Society cautioned that “in the future we need more rigorous evaluations of the effectiveness and safety of endocrine and surgical protocols” including “careful assessment of . . . the effects of prolonged delay of puberty in adolescents on bone health, gonadal function, and the brain (including effects on cognitive, emotional, social, and sexual development).” (Hembree et al. 2017 at 3874.) No such “careful” or “rigorous” evaluation of these very serious safety questions has yet been done.

184. Some advocates appear to assume that puberty blockers are “safe” because they have been approved by the Food and Drug Administration (FDA) for use to treat precocious puberty—a rare condition in which the puberty process may start at eight or younger. No such conclusion can be drawn. As the “label” for Lupron (one of the most widely prescribed puberty blockers) explains, the FDA approved the drug only *until* the “age was appropriate for entry into

puberty.” The study provides no information at all as to the safety or reversibility of instead *blocking* healthy, normally-timed puberty’s beginning, and *throughout* the years that body-wide continuing changes normally occur. Given the physical, social, and psychological dangers to the child with precocious puberty, drugs like Lupron are effective in returning the child to a puerile state without a high incidence of significant side effects—that is, they are “safe” to reverse the condition. But use of drugs to suppress normal puberty has multiple organ system effects whose long-term consequences have not been investigated.

185. **Fertility:** The Endocrine Society Guidelines rightly say that research is needed into the effect of puberty blockade on “gonadal function” and “sexual development.” The core purpose and function of puberty blockers is to prevent the maturation of the ovaries or testes, the sources of female hormones and male hormones when stimulated by the pituitary gland. From this predictable process fertility is accomplished within a few years. Despite widespread assertions that puberty blockers are “fully reversible,” there has been no study published on the critical question of whether patients ever develop normal levels of fertility if puberty blockers are terminated after a “prolonged delay of puberty.” The 2017 Endocrine Society Guidelines are correct that there are no data on achievement of fertility “following prolonged gonadotropin suppression” (that is, puberty blockade). (Hembree et al. 2017 at 3880.)

186. **Bone strength:** Multiple studies have documented adverse effects from puberty blockers on bone density. (Klink et al. 2015; Vlot et al. 2016; Joseph et al. 2019.) The most recent found that after two years on puberty blockers, the bone density measurements for a significant minority of the children had declined to clinically concerning levels. Density in the spines of some subjects fell to a level found in only 0.13% of the population. (Biggs 2021.) Some

other studies have found less concerning effects on bone density. While the available evidence remains limited and conflicting, it is not possible to conclude that the treatment is “safe.”

187. **Brain development:** Important neurological growth and development in the brain occurs across puberty. The anatomic and functional effect on brain development of blocking the natural puberty process has not been well studied. A prominent Australian clinical team recently expressed concern that “no data were (or are) available on whether delaying the exposure of the brain to a sex steroid affects psychosexual, cognitive, emotional, or other neuropsychological maturation.” (Kozłowska et al. 2021 at 89.) In my opinion, given the observed correlation between puberty and brain development, the default hypothesis must be that there *would* be a negative impact. For the purpose of protecting patients all over the world, the burden of proof should be on advocates to first demonstrate to a reasonable degree of certainty that brain structure and its measurable cognitive and affect processing are not negatively affected. This recalls the ethical principle: Above All Do No Harm.

188. The Endocrine Society Guidelines acknowledge as much, stating that side effects of pubertal suppression “may include . . . unknown effects on brain development,” that “we need more rigorous evaluations of . . . the effects of prolonged delay of puberty in adolescents on . . . the brain (including effects on cognitive, emotional, social, and sexual development),” and stating that “animal data suggests there may be an effect of GnRH analogs [puberty blockers] on cognitive function.” (Hembree et al. 2017 at 3874, 3882, 3883.) Given this concern, one can only wonder why this relevant question has not been scientifically investigated in a large group of natal males and females.

189. There has been a longitudinal study of one natal male child, assessed before, and again 20 months after, puberty suppression was commenced. It reported a reduction in the

patient's "global IQ," measured an anomalous absence of certain structural brain development expected during normal male puberty, and hypothesized that "a plausible explanation for the G[lobal] IQ decrease should consider a disruption of the synchronic [i.e., appropriately timed] development of brain areas by pubertal suppression." (Schneider et al. 2017 at 7.) This should cause parents and practitioners serious concern.

190. Whether any impairment of brain development is "reversed" upon later termination of puberty blockade has, to my knowledge, not been studied at all. As a result, assertions by medical or mental health professionals that puberty blockade is "fully reversible" are unjustified and based on hope rather than science.

191. Without a number of additional case studies—or preferably statistically significant clinical studies—two questions remain unanswered: Are there brain anatomic or functional impairment from puberty blockers? And are the documented changes reversed over time when puberty blockers are stopped? With these questions unanswered, it is impossible to assert with certainty that the effects of this class of medications are "fully reversible." Such an assertion is another example of ideas based on beliefs rather than on documentation, on hope not science.

192. **Psycho-social harm:** Puberty is a time of stress, anxiety, bodily discomfort during physical development, and identity formation for *all* humans. No careful study has been done of the long-term impact on the young person's coping skills, interpersonal comfort, and intimate relationships from remaining puerile for, e.g., two to five years while one's peers are undergoing pubertal transformations, and of then undergoing an artificial puberty at an older age. However, pediatricians and mental health professionals hear of distress, concern, and social awkwardness in those who naturally have a delayed onset of puberty. In my opinion, individuals

in whom puberty is delayed multiple years are likely to suffer at least subtle negative psychosocial and self-confidence effects as they stand on the sidelines witnessing their peers developing the social relationships (and attendant painful social learning experiences) that come with adolescence. (Levine 2018 at 9.) Social anxiety and social avoidance are common findings in the evaluation of trans-identified children and teens. Are we expected to believe that creating years of being further different than their peers has no lasting internal consequences? Do we ignore Adolescent Psychiatry’s knowledge of the importance of peer groups among adolescents?

193. We simply do not know what all the psychological impacts of NOT grappling with puberty at the ordinary time may be, because it has not been studied. And we have no information as to whether that impact is “fully reversible.”

194. In addition, since the overwhelming proportion of children who begin puberty blockers continue on to cross-sex hormones, it appears that there is an important element of “psychological irreversibility” in play. The question of to what extent the physical and developmental impacts of puberty blockers might be reversible is an academic one, if psycho-social realities mean that very few patients will ever be able to make that choice once they have started down the road of social transition and puberty blockers.

B. Use of cross-sex hormones in adolescents for gender dysphoria has not been shown to be medically safe except in the short term.

195. As with puberty blockers, all evidence concerning the safety of extended use of cross-sex hormones is of “very low quality.” The U.K. NICE evidence review cautioned that “the safety profiles” of cross-sex hormone treatments are “largely unknown,” and that several of the limited studies that do exist reported high numbers of subjects “lost to follow-up,” without explanation—a worrying indicator. (NICE 2020b.)

196. The 2020 Cochrane Review reported that: “We found insufficient evidence to determine the . . . safety of hormonal treatment approaches for transgender women in transition.” (Haupt et al. 2020 at 4.) Even the Endocrine Society tagged all its recommendations for the administration of cross-sex hormones as based on “low quality evidence.” (Hembree et al. 2017 at 3889.)

197. **Sterilization:** It is undisputed, however, that harm to the gonads is an expected effect, to the extent that it must be assumed that cross-sex hormones will sterilize the patient. Thus, the Endocrine Society 2017 Guidelines caution that “[p]rolonged exposure of the testes to estrogen has been associated with testicular damage,” that “[r]estoration of spermatogenesis after prolonged estrogen treatment has not been studied,” and that “[i]n biological females, the effect of prolonged treatment with exogenous testosterone upon ovarian function is uncertain.” (Hembree et al. 2017 at 3880.)¹²

198. The Guidelines go on to recommend that the practitioner counsel the patient about the (problematic and uncertain) options available to collect and preserve fertile sperm or ova before beginning cross-sex hormones. The life-long negative emotional impact of infertility on both men and women has been well studied. While this impact has not been studied specifically within the transgender population, the opportunity to be a parent is likely a human, emotional need, and so should be considered an important risk factor when considering gender transition for any patient.

¹² See also Guss et al. 2015 at 4 (“a side effect [of cross-sex hormones] may be infertility”) and at 5 (“cross-sex hormones . . . may have irreversible effects”); Tishelman et al. 2015 at 8 (Cross-sex hormones are “irreversible interventions” with “significant ramifications for fertility”).

199. **Sexual response:** Puberty blockers prevent maturation of the sexual organs and response. Some, and perhaps many, transgender individuals who did not go through puberty consistent with their sex and are then put on cross-sex hormones face significantly diminished sexual response as they enter adulthood and are unable ever to experience orgasm. In the case of males, the cross-sex administration of estrogen limits penile genital growth and function. In the case of females, prolonged exposure to exogenous testosterone impairs vaginal function. Much has been written about the negative psychological and relational consequences of anorgasmia among non-transgender individuals that is ultimately applicable to the transgendered. (Levine 2018 at 6.) At the same time, prolonged exposure of females to exogenous testosterone often increases sexual drive to a distracting degree. It is likely that parents and physicians are uncomfortable discussing any aspects of genital sexual activity with patients.

200. **Cardiovascular harm:** Several researchers have reported that cross-sex hormones increase the occurrence of various types of cardiovascular disease, including strokes, blood clots, and other acute cardiovascular events. (Getahun et al. 2018; Guss et al. 2015; Asscheman et al. 2011.) With that said, I agree with the conclusion of the Endocrine Society committee (like that of the NICE Evidence Review) that: “A systematic review of the literature found that data were insufficient (due to very low-quality evidence) to allow a meaningful assessment of patient-important outcomes, such as death, stroke, myocardial infarction, or venous thromboembolism in transgender males. Future research is needed to ascertain the potential harm of hormonal therapies.” (Hembree et al. 2017 at 3891.) Future research questions concerning long-term harms need to be far more precisely defined. The question of whether cross-sex hormones are safe for adolescents and young adults cannot be answered by analogies to hormone replacement therapy in menopausal women (which is not a cross-sex usage).

Medicine has answered safety questions for menopausal women in terms of cancer and cardiovascular safety: at what dose, for what duration, and at what age range. The science of endocrine treatment of gender dysphoric youth is being bypassed by short-term clinical impressions of safety even though physicians know that cardiovascular and cancer processes often develop over many years.

201. Further, in contrast to administration for menopausal women, hormones begun in adolescence are likely to be administered for four to six decades. The published evidence of adverse impact, coupled with the lack of data sufficient to reach a firm conclusion, make it irresponsible to assert that cross-sex hormones “are safe.”

202. **Harm to family and friendship relationships:** As a psychiatrist, I recognize that mental health is a critical part of health generally, and that relationships cannot be separated from and profoundly impact mental health. Gender transition routinely leads to isolation from at least a significant portion of one’s family in adulthood. In the case of a juvenile transition, this will be less dramatic while the child is young, but commonly increases over time as siblings who marry and have children of their own do not wish the transgender individual to be in contact with those children. By adulthood, the friendships of transgender individuals tend to be confined to other transgender individuals (often “virtual” friends known only online) and the generally limited set of others who are comfortable interacting with transgender individuals. (Levine 2017 at 5.) My concerns about this are based on decades of observations in my professional work with patients.

203. **Sexual-romantic harms associated with transition:** After adolescence, transgender individuals find the pool of individuals willing to develop a romantic and intimate relationship with them to be greatly diminished. When a trans person who passes well reveals his or her natal sex, many potential mates lose interest. When a trans person does not pass well,

options are likely further diminished. But regardless of a person's appearance, these adults soon learn that many of their dates are looking for exotic sexual experiences rather than genuinely loving relationships. (Levine 2017 at 5, 13; Levine 2013 at 40.)

C. The timing of harms.

204. The multi-year delay between start of hormones and the spike in completed suicide observed by Professor Biggs in the Tavistock data (as discussed in Section VIII above) warns us that the safety and beneficence of these treatments cannot be judged based on short-term studies, or studies that do not continue into adulthood. Similarly, several of the harms that I discuss above would not be expected to manifest until the patients reaches at least middle-age. For example, stroke or other serious cardiovascular event is a complication that is unlikely to manifest during teen years even if its likelihood over the patient's lifetime has been materially increased via obesity, lipid abnormalities, and smoking. Regret over sterilization or over an inability to form a stable romantic relationship may occur sooner. Psychological challenges of being a trans adult may become manifest after the medical profession is only doing routine follow up care—or, in many cases, has lost contact with the patient altogether. Because few, if any, clinics in this country are conducting systematic long-term follow-up with their child and adolescent patients, the doctors who counsel, prescribe, or perform hormonal and surgical therapies are unlikely ever to become aware of the later negative life impacts, however severe. These concerns are compounded by the findings in the recent “detransitioner” research that 76% did not inform their clinicians of their detransition. (Littman 2021.)

205. The possibility that steps along the transition and affirmation pathway, while lessening the pain of gender dysphoria in the short term, could lead to additional sources of crippling emotional and psychological pain, are too often not considered by advocates of social transition and not considered at all by the trans child. (Levine 2016 at 243.) Clinicians must

distinguish the apparent short-term safety of hormones from likely or possible long-term consequences, and help the patient or parents understand these implications as well. The young patient may feel, “I don’t care if I die young, just as long I get to live as a woman.” The mature adult may take a different view. Hopefully, so will the child’s physician.

206. Individual patients often pin excessive hope in transition, believing that transition will solve what are in fact ordinary social stresses associated with maturation, or mental health co-morbidities. In this way, transition can prevent them from mastering personal challenges at the appropriate time or directly addressing conditions that require treatment. When the hoped-for “vanishing” of other mental health or social difficulties does not occur, disappointment, distress, and depression may ensue. It is noteworthy that half of the respondents to the larger “detransitioner” survey reported that their transition had not helped the gender dysphoria, and 70% had concluded that their gender dysphoria was related to other issues. (Vandenbussche 2021.) Without the clinical experience of monitoring the psychosocial outcomes of these young patients as they age into adulthood, many such professionals experience no challenge to their affirmative beliefs. But medical and mental health professionals who deliver trans affirmative care for those with previous and co-existing mental health problems have an ethical obligation to inform themselves, and to inform patients and parents, that these dramatic treatments are not a panacea.

207. In sum, whether we consider physical or mental health, science does not permit us to say that either puberty blockers or cross-sex hormones are “safe,” and the data concerning the mental health of patients before, during, and after such treatments strongly contradict the assertion that gender dysphoria is “easily managed.”

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LEVINE EXPERT REPORT

EXHIBIT A

Stephen B. Levine, M.D.

Curriculum Vita
February, 2022

Brief Introduction

Dr. Levine is Clinical Professor of Psychiatry at Case Western Reserve University School of Medicine. He is the author or coauthor of numerous books on topics relating to human sexuality and related relationship and mental health issues. Dr. Levine has been teaching, providing clinical care, and writing since 1973, and has generated original research, invited papers, commentaries, chapters, and book reviews. He has served as a journal manuscript and book prospectus reviewer for many years. Dr. Levine has been co-director of the Center for Marital and Sexual Health/ Levine, Risen & Associates, Inc. in Beachwood, Ohio from 1992 to the present. He received a lifetime achievement Masters and Johnson's Award from the Society for Sex Therapy and Research in March 2005.

Personal Information

Date of birth 1/14/42

Medical license no. Ohio 35-03-0234-L

Board Certification 6/76 American Board of Neurology and Psychiatry

Education

1963 BA Washington and Jefferson College

1967 MD Case Western Reserve University School of Medicine

1967-68 internship in Internal Medicine University Hospitals of Cleveland

1968-70 Research associate, National Institute of Arthritis and Metabolic Diseases, Epidemiology Field Studies Unit, Phoenix, Arizona, United States Public Health Service

1970-73 Psychiatric Residency, University Hospitals of Cleveland

1974-77 Robert Wood Johnson Foundation Clinical Scholar

Appointments at Case Western Reserve University School of Medicine

1973- Assistant Professor of Psychiatry

1979- Associate Professor

1982- Awarded tenure

1985- Full Professor

1993- Clinical Professor

Honors

Summa Cum Laude, Washington & Jefferson

Teaching Excellence Award-1990 and 2010 (Residency program)

Visiting Professorships

- Stanford University-Pfizer Professorship program (3 days)–1995
- St. Elizabeth’s Hospital, Washington, DC –1998
- St. Elizabeth’s Hospital, Washington, DC--2002

Named to America’s Top Doctors consecutively since 2001

Invitations to present various Grand Rounds at Departments of Psychiatry and Continuing Education Lectures and Workshops

Masters and Johnson Lifetime Achievement Award from the Society of Sex Therapy and Research, April 2005 along with Candace Risen and Stanley Althof

2006 SSTAR Book Award for The Handbook of Clinical Sexuality for Mental Health Professionals: Exceptional Merit

2018—Albert Marquis Lifetime Achievement Award from Marquis Who’s Who. (Exceling in one’s field for at least twenty years)

Professional Societies

1971- American Psychiatric Association; fellow; #19909

2005- American Psychiatric Association, Distinguished Life Fellow

1973- Cleveland Psychiatric Society

1973- Cleveland Medical Library Association

- 1985 - Life Fellow
- 2003 - Distinguished Life Fellow

1974-Society for Sex Therapy and Research

- 1987-89-President

1983- International Academy of Sex Research

1983- Harry Benjamin International Gender Dysphoria Association

- 1997-8 Chairman, Standards of Care Committee

1994- 1999 Society for Scientific Study of Sex

Community Boards

1999-2002 Case Western Reserve University Medical Alumni Association

1996-2001 Bellefaire Jewish Children's Bureau

1999-2001 Physicians' Advisory Committee, The Gathering Place (cancer rehabilitation)

Editorial Boards

1978-80 Book Review Editor Journal Sex and Marital Therapy

Manuscript Reviewer for:

- a. Archives of Sexual Behavior
- b. Annals of Internal Medicine
- c. British Journal of Obstetrics and Gynecology
- d. JAMA
- e. Diabetes Care
- f. American Journal of Psychiatry
- g. Maturitas
- h. Psychosomatic Medicine
- i. Sexuality and Disability
- j. Journal of Nervous and Mental Diseases
- k. Journal of Neuropsychiatry and Clinical Neurosciences
- l. Neurology
- m. Journal Sex and Marital Therapy
- n. Journal Sex Education and Therapy
- o. Social Behavior and Personality: an international journal (New Zealand)
- p. International Journal of Psychoanalysis
- q. International Journal of Transgenderism
- r. Journal of Urology
- s. Journal of Sexual Medicine
- t. Current Psychiatry
- u. International Journal of Impotence Research
- v. Postgraduate medical journal
- w. Academic Psychiatry

Prospectus Reviewer

- a. Guilford
- b. Oxford University Press
- c. Brunner/Routledge
- d. Routledge

Administrative Responsibilities

Principal Investigator of approximately 70 separate studies involving pharmacological interventions for sexual dysfunction since 1989.

Co-leader of case conferences at DELRLLC.com

Expert testimony at trial or by deposition within the last 4 years

Provided expert testimony for Massachusetts Dept. of Corrections in its defense of a lawsuit brought by prisoner Katheena Soneeya, including by deposition in October 2018, and in-court testimony in 2019.

Provided expert testimony by deposition and at trial in *In the Interests of the Younger Children* (Dallas, TX), 2019.

Testified in an administrative hearing in *In the matter of Rhys & Lynn Crawford* (Washington State), March 2021.

Testified multiple times in juvenile court in *In the matter of Asha Kerwin* (Tucson, Arizona), 2021.

Provided expert testimony by deposition in *Kadel et al v. Folwell et al.* (North Carolina), 2021.

Consultancies

Massachusetts Department of Corrections—evaluation of 12 transsexual prisoners and the development of a Gender Identity Disorders Program for the state prison system. Monthly consultation with the GID treatment team since February 2009 and the GID policy committee since February 2010.

California Department of Corrections and Rehabilitation; 2012-2015; education, inmate evaluation, commentary on inmate circumstances, suggestions on future policies.

Virginia Department of Corrections –evaluation of an inmate.

New Jersey Department of Corrections—evaluation of an inmate.

Idaho Department of Corrections—workshop 2016.

Grant Support/Research Studies

TAP—studies of Apomorphine sublingual in treatment of erectile dysfunction.

Pfizer–Sertraline for premature ejaculation.

Pfizer–Viagra and depression; Viagra and female sexual dysfunction; Viagra as a treatment for SSRI-induced erectile dysfunction.

NIH- Systemic lupus erythematosus and sexuality in women.

Sihler Mental Health Foundation

- a. Program for Professionals
- b. Setting up of Center for Marital and Sexual Health
- c. Clomipramine and Premature ejaculation
- d. Follow-up study of clergy accused of sexual impropriety
- e. Establishment of services for women with breast cancer

Alza–controlled study of a novel SSRI for rapid ejaculation.

Pfizer–Viagra and self-esteem.

Pfizer- double-blind placebo control studies of a compound for premature ejaculation.

Johnson & Johnson – controlled studies of Dapoxetine for rapid ejaculation.

Proctor and Gamble: multiple studies to test testosterone patch for post menopausal sexual dysfunction for women on and off estrogen replacement.

Lilly-Icos—study of Cialis for erectile dysfunction.

VIVUS – study for premenopausal women with FSAD.

Palatin Technologies- studies of bremelanotide in female sexual dysfunction—first intranasal then subcutaneous administration.

Medtap – interview validation questionnaire studies.

HRA- quantitative debriefing study for Female partners of men with premature ejaculation, Validation of a New Distress Measure for FSD.

Boehringer-Ingelheim- double blind and open label studies of a prosexual agent for hypoactive female sexual desire disorder.

Biosante- studies of testosterone gel administration for post menopausal women with HSDD.

J&J a single-blind, multi-center, in home use study to evaluate sexual enhancement effects of a product in females.

UBC-Content validity study of an electronic FSEP-R and FSDS-DAO and usability of study PRO measures in premenopausal women with FSAD, HSDD or Mixed FSAD/HSDD.

National registry trial for women with HSDD.

Endoceutics—two studies of DHEA for vaginal atrophy and dryness in post menopausal women.

Palatin—study of SQ Bremelanotide for HSDD and FSAD.

Trimel- a double-blind, placebo controlled study for women with acquired female orgasmic disorder.

S1 Biopharma- a phase 1-B non-blinded study of safety, tolerability and efficacy of Lorexys in premenopausal women with HSDD.

HRA – qualitative and cognitive interview study for men experiencing PE.

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A) Books

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- 2) Sex Is Not Simple, Ohio Psychological Publishing Company, 1988; Reissued in paperback as: Solving Common Sexual Problems: Toward a Problem Free Sexual Life, Jason Aronson, Livingston, NJ. 1997
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- 5) Editor, Clinical Sexuality. Psychiatric Clinics of North America, March, 1995.
- 6) Editor, (Candace Risen and Stanley Althof, associate editors) Handbook of Clinical Sexuality for Mental Health Professionals. Routledge, New York, 2003
 1. 2006 SSTAR Book Award: Exceptional Merit
- 7) Demystifying Love: Plain Talk For The Mental Health Professional. Routledge, New York, 2006
- 8) Senior editor, (Candace B. Risen and Stanley E. Althof, Associate editors), Handbook of Clinical Sexuality for Mental Health Professionals, 2nd edition. Routledge, New York, 2010.
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- 10) Senior editor Candace B. Risen and Stanley E. Althof, Associate editors), Handbook of Clinical Sexuality for Mental Health Professionals. 3rd edition Routledge, New York, 2016

B) Research and Invited Papers

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Exhibit B

IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF WEST VIRGINIA
CHARLESTON DIVISION

B.P.J. by her next friend and)
mother, HEATHER JACKSON,)

Plaintiff,)

vs.)

No. 2:21-cv-00316

WEST VIRGINIA STATE BOARD OF)
EDUCATION, HARRISON COUNTY)
BOARD OF EDUCATION, WEST)
VIRGINIA SECONDARY SCHOOL)
ACTIVITIES COMMISSION, W.)
CLAYTON BURCH in his official)
capacity as State)
Superintendent, DORA STUTLER,)
in her official capacity as)
Harrison County)
Superintendent, and THE STATE)
OF WEST VIRGINIA,)

Defendants,)

LAINIEY ARMISTEAD,)

Defendant-Intervenor.)

VIDEOTAPED DEPOSITION OF
STEPHEN LEVINE
Wednesday, March 30, 2022
Volume I

Reported by:
ALEXIS KAGAY
CSR No. 13795
Job No. 5122884
PAGES 1 - 289

IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF WEST VIRGINIA
CHARLESTON DIVISION

B.P.J. by her next friend and
mother, HEATHER JACKSON,

Plaintiff,

vs.

No. 2:21-cv-00316

WEST VIRGINIA STATE BOARD OF
EDUCATION, HARRISON COUNTY
BOARD OF EDUCATION, WEST
VIRGINIA SECONDARY SCHOOL
ACTIVITIES COMMISSION, W.
CLAYTON BURCH in his official
capacity as State
Superintendent, DORA STUTLER,
in her official capacity as
Harrison County
Superintendent, and THE STATE
OF WEST VIRGINIA,

Defendants,

LAINIEY ARMISTEAD,

Defendant-Intervenor.)

Remote videotaped deposition of
STEPHEN LEVINE, Volume I, taken on behalf of Plaintiff,
with all participants appearing remotely, beginning at
9:09 a.m. and ending at 5:46 p.m. on Wednesday,
March 30, 2022, before ALEXIS KAGAY, Certified
Shorthand Reporter No. 13795.

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18 Videographer:

19 KIMBERLEE DECKER

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WITNESS EXAMINATION

STEPHEN LEVINE

Volume I

BY MS. HARTNETT 14

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Exhibit 104 Video Clip 278

Exhibit 105 Video Clip 279

1 Wednesday, March 30, 2022

2 9:09 a.m. A.M.

3 THE VIDEOGRAPHER: We are on the record at
4 9:09 a.m. on March the 30th of 2022.

5 All participants are attending remotely. 06:09:27

6 Audio and video recording will continue to
7 take place unless all parties agree to go off the
8 record.

9 This is media unit 1 of the recorded
10 deposition of Dr. Stephen Levine, taken by counsel for 06:09:39
11 the plaintiff, in the matter of B.P.J., by her be- --
12 by her next friend and mother, Heather Jackson, versus
13 West Virginia State Board of Education, filed in the
14 U.S. District Court, for the Southern District of
15 West Virginia, Charleston Division, Case 06:09:59
16 Number 2:21-cv-00316.

17 My name is Kimberlee Decker from Veritext
18 Legal Solutions, and I am the videographer. The court
19 reporter is Alexis Kagay.

20 I am not related to any party in this action, 06:10:16
21 nor am I financially interested in the outcome.

22 Counsel and all present will now state your
23 appearances and affiliations for the record. If there
24 are any objections to proceeding, please state them at
25 the time of your appearance, beginning with the 06:10:31

Page 11

1 noticing attorney.

2 MS. HARTNETT: Good morning. I am Kathleen
3 Hartnett from Cooley, LLP, and I represent the
4 plaintiff B.P.J.

5 I will let my co-counsel introduce themselves, 06:10:40
6 starting with my colleagues at Cooley.

7 MR. BARR: Good morning. Andrew Barr from
8 Cooley, LLP, for the plaintiff.

9 MS. VEROFF: Good morning. This is Julie
10 Veroff from Cooley, LLP, for Plaintiff. 06:10:53

11 MS. KANG: Good morning. This is Katelyn Kang
12 from Cooley, LLP, for Plaintiff.

13 MS. PELET DEL TORO: Good morning. This is
14 Valeria Pelet del Toro of Cooley, for Plaintiff.

15 MS. REINHARDT: Good morning. This is 06:11:00
16 Elizabeth Reinhardt at Cooley, for Plaintiff.

17 MS. HELSTROM: Hello. This is Zoe Helstrom
18 from Cooley, LLP, for Plaintiff.

19 COUNSEL SWAMINATHAN: Good morning. This is
20 Sruti Swaminathan from Lambda Legal, for Plaintiff. 06:11:26
21 And I have a paralegal at Lambda, Maia Zelkind, with me
22 as well.

23 MR. BLOCK: Good morning. This is Josh Block
24 from the ACLU, for Plaintiff.

25 MS. DENIKER: Good morning. Susan Deniker 06:11:44

1 from Steptoe & Johnson, PLLC, representing Harrison
2 County Board of Education and Superintendent Dora
3 Stutler.

4 MS. MORGAN: This is Kelly Morgan on behalf of
5 the West Virginia Board of Education and 06:11:58
6 Superintendent Burch.

7 MS. ROGERS: This is Shannon Rogers on behalf
8 of the West Virginia Secondary School Activities
9 Commission.

10 MR. TRYON: This is David Tryon. I'm with the 06:12:12
11 West Virginia attorney general's office, representing
12 the State of West Virginia.

13 MR. BROOKS: This is Roger Brooks with
14 Alliance Defending Freedom, representing the intervenor
15 Lainey Armistead and defending Dr. Levine today in this 06:12:28
16 deposition. With me is my colleague and law clerk,
17 Lawrence Wilkinson.

18 THE VIDEOGRAPHER: Thank you.

19 Will the court reporter please swear in the
20 witness. 06:12:41

21
22 STEPHEN LEVINE,
23 having been administered an oath, was examined and
24 testified as follows:
25

EXAMINATION

BY MS. HARTNETT:

Q Good morning, Dr. Levine.

A Good morning.

MS. HARTNETT: Before we start, I'm just going 06:13:01
to put a housekeeping matter on the record that the
attorneys discussed before we went on the record and
that is that objection to form preserves all objections
other than privilege and that the parties will make an
effort to use "form," "scope" and "terminology" as the 06:13:13
shorthand objections. In addition, an objection by one
defendant is an objection for all defendants.

Could any counsel for the defense let me know
if they have any disagreement with that?

MR. BROOKS: We have agreed, in fact. 06:13:30

MS. HARTNETT: Thank you very much.

BY MS. HARTNETT:

Q So again, my name is Kathleen Hartnett, and
I'm with the law firm called Cooley, LLP.

Can you hear me okay? 06:13:41

A I do. At this point, yes.

Q Okay. Please let me know if that changes.

I use she/her pronouns.

Would you please state and spell your name for
the record. 06:13:53

1 A Stephen Barrett Levine, S-T-E-P-H-E-N

2 B-A-R-R-E-T-T L-E-V-I-N-E.

3 Q And what pronouns do you use?

4 A He/him.

5 Q Thank you. Dr. Levine, you've been deposed 06:14:07
6 many times before; correct?

7 A Yes.

8 Q Was the most recent deposition that you gave
9 in September of last year, 2021?

10 A No. 06:14:21

11 Q What was the most recent deposition that you
12 gave?

13 A In -- within the last month, I was deposed in
14 a Connectica- -- a Connecticut case involving a
15 transgender prisoner. 06:14:41

16 Q Do you know the name of that case?

17 A Probably Clark versus the department of
18 corrections in Connecticut. Connecticut Department of
19 Corrections (sic).

20 Q Okay. And what was your -- the nature of your 06:15:01
21 testimony in that Connecticut case, this recent
22 deposition that you gave?

23 A Well, I provided a psychiatric evaluation of
24 the patient and made recommendations. It -- it was --
25 I'm hesitating because -- I provided a thorough 06:15:28

Page 15

1 psychiatric evaluation of the developmental history and
2 the in prison history of the patient and the -- the
3 psychology of his new transgender identity.

4 Q And you say "new transgender identity."

5 Was the new identity of -- male or female? 06:16:02

6 A The -- the new identity as a transgender
7 woman.

8 MR. BROOKS: And -- and, Counsel, I will
9 caution that obviously any detail about a psychiatric
10 evaluation of an individual prisoner is a matter 06:16:18
11 covered by confidentiality that Dr. Levine is not free
12 to get into detail about.

13 MS. HARTNETT: I hear you. I -- this is not a
14 disclosed matter on his CV and is a recent deposition,
15 so we'll have to just determine whether we need more 09:16:23
16 information, but thank you.

17 BY MS. HARTNETT:

18 Q Could you let me know what -- without giving
19 any personal identifying -- or, I guess, any more
20 detail than you believe appropriate, could you tell me 09:16:33
21 what the nature of any recommendations you made were in
22 that matter?

23 A My recommendations were to provide a pathway
24 towards further evaluation so that eventually a
25 decision could be made about whether sex reassignment 09:16:56

Page 16

1 surgery would be appropriate.

2 The -- the reason I'm hesitating is that that
3 really did not come to be the subject of the
4 deposition. The subject of the deposition really was
5 the contents of my evaluation, which was done two years 09:17:24
6 before, and -- so lots of things had happened in the
7 two years since I saw the patient or interviewed the
8 patient and -- so I was not able to make
9 recommendations based on current knowledge of the
10 patient, and so I did not. 09:17:43

11 Q And was the -- prior to this recent deposition
12 in Clark, was the most recent deposition before that
13 the deposition in September of last year?

14 A Yes.

15 Q Thank you. And I'm asking that by way of 09:18:03
16 introduction just because I want to make sure we're on
17 the same page about the ground rules for the
18 deposition, and it sounds like you've been through this
19 before, but I'll just let you know my basic ground
20 rules and make sure we're on the same page. 09:18:18

21 So I will ask questions, and you must answer
22 the questions unless your counsel instructs you not to
23 answer.

24 Do you understand that?

25 A I do. 09:18:26

Page 17

1 Q And if your counsel objects, you'll still need
2 to answer my question unless you've been instructed not
3 to answer.

4 Do you understand that?

5 A I do. 09:18:35

6 Q If you don't answer (sic) my question, could
7 you please let me know, and I'll be happy to try to
8 rephrase it or make it clear for you?

9 Does that make sense?

10 A I'll try to remember. 09:18:48

11 Q And if you answer, I will assume you
12 understood the question.

13 Do you understand that?

14 A Yes.

15 Q I'm going to try -- try to take a break every 09:19:00
16 hour or so. If you need a break at a different time,
17 please let me know.

18 Do you understand that?

19 A I understand.

20 Q And if I've asked a question, you'll need to 09:19:11
21 provide an answer before we take a break.

22 Do you also understand that?

23 A I do.

24 Q I will do my best not to speak over you -- and
25 please use verbal answers so the court reporter can 09:19:25

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1 transcribe your answers. Nodding or shaking your head
2 can't be captured on the transcript.

3 Do you understand that?

4 A I do, but I can guarantee you you'll have to
5 remind me of that. 09:19:36

6 Q Well, you may have to do the same for me, but
7 we'll try.

8 I also just want to explain what I'm going to
9 mean when I use a couple of terms today.

10 For purposes of this deposition, when I say 09:19:51
11 "cisgender," I will mean someone who's gender identity
12 matches the sex that was recorded for that person at
13 birth.

14 Do you understand that?

15 A Yes. 09:20:02

16 Q And then when I say the word "transgender," I
17 will mean someone whose gender identity does not match
18 the sex for which was recorded at birth.

19 Do you understand that?

20 A Yes. 09:20:13

21 Q And when I say "B.P.J.," I'm referring to the
22 plaintiff in this case.

23 Do you understand that?

24 A Yes.

25 Q Do you understand that you are testifying 09:20:21

1 under oath today just as if you were testifying in
2 court?

3 A Yes.

4 Q Is there anything that would prevent you from
5 testifying truthfully today? 09:20:32

6 A No.

7 Q Are you taking any medication that would
8 affect your ability to give truthful testimony?

9 A Well, I took a sleeping pill last night, but I
10 feel reasonably alert today. 09:20:48

11 Q Okay. So you don't -- you don't have a belief
12 that that medication you took last night will affect
13 your ability to give truthful testimony today?

14 A I -- I don't think it will.

15 Q Do you know what case you're being deposed in 09:21:06
16 today?

17 A Well, I -- yes.

18 Q What case is that?

19 A B.P.J. versus Department of Education.

20 Q And do you know what jurisdiction this case is 09:21:19
21 from?

22 A West Virginia.

23 Q And do you have -- sorry.

24 Do you have an understanding of the issue
25 presented by this case? 09:21:35

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1 A I have an understanding. I'm not sure it is
2 the correct understanding, but I do have an
3 understanding.

4 Q Understood. What is your understanding of
5 this case? 09:21:47

6 A The plaintiff and next friend and mother wish
7 the young person to be able to compete in athletics
8 according to their current gender identity and
9 apparently the State Board of Education is --
10 disagrees. 09:22:13

11 Q Okay. Thank you.
12 So we already touched on that you had been
13 deposed previously. I just want to ask you about a
14 couple of specific depositions you gave to see if you
15 recall those? 09:22:29

16 There was a matter in North Carolina called
17 Kadel that you gave a deposition in September of 2021
18 regarding state employee healthcare.

19 Do you recall giving that deposition?

20 A Would you repeat -- regarding what? I didn't 09:22:41
21 hear that last phrase.

22 Q I'll try to speak more slowly.

23 That was regarding -- so let me just start
24 that one again.

25 So do you recall giving a deposition in a 09:22:51

1 North Carolina matter called Kadel in September of 2021
2 regarding state employee healthcare?

3 A Yes.

4 Q Do you recall giving a deposition in a Florida
5 case in December of 2020 called "Claire"? That was 09:23:07
6 also about state employee healthcare.

7 A Yes.

8 Q There also was a case called Keohane in
9 Florida where you gave a deposition in 2017 and that
10 was a prisoner case. 09:23:21

11 Do you recall that?

12 A Yes.

13 Q Did you give true and correct testimony in
14 those depositions?

15 A Yes. 09:23:31

16 Q Have you always given true and correct
17 testimony in your depositions?

18 A To the best of my knowledge, yes.

19 Q Thank you. And you've had depositions in
20 cases involving prisoners who were seeking care for 09:23:45
21 gender dysphoria; is that correct?

22 A Yes.

23 Q Have you ever testified in favor of a prisoner
24 who was seeking medical care for gender dysphoria?

25 A Yes. 09:23:59

1 Q Can you describe those instances where you've
2 testified in favor of a prisoner seeking medical care
3 for gender dysphoria?

4 A In the last case involving a prisoner by the
5 name of Soneeya, S-O-N-E-E-Y-A, I recommended transfer 09:24:14
6 to a female prisoner and -- sorry -- transfer to a
7 female prison and the opportunity to have sex
8 reassignment surgery if, after a year of adaptation
9 there, there were no significant decompensations or
10 problems. 09:24:44

11 Q And do you remember what year you made that
12 recommendation?

13 A I think it was 2019.

14 Q Okay. And can you -- are you aware of any
15 other examples of you having testified in favor of a 09:25:05
16 prisoner seeking medical care for gender dysphoria?

17 A I'm hesitating because medical care includes
18 many things. And so the answer is yes. It involves
19 accommodations to their current gender identity in
20 terms of canteen items, for example, and it includes 09:25:35
21 the prescription of cross gender -- cross-sex hormones.
22 So I've been involved in the provision of those kind of
23 things repeatedly over the years for prisoners.

24 Q Have you ever, other than in the Soneeya
25 matter, recommended that a prisoner -- sorry -- 09:26:04

1 testified that a prisoner should receive gender
2 confirmation surgery?

3 A I'm hesitating to answer the question because
4 it's about testimony. In my work as consultant, I have
5 repeatedly recommended both surgery and, more -- more 09:26:25
6 commonly, hormone treatment, electrolysis treatment,
7 canteen item treatment. Most of -- the vast majority
8 of these cases never come to trial.

9 Q When is the last time that you recommended
10 that a pres- -- a prisoner should have hormone 09:26:46
11 treatment?

12 A It would have been the third Thursday in
13 March, this year.

14 Q And where is that prisoner located?

15 A Massachusetts. 09:27:06

16 Q Can you estimate how many prisoners you've
17 given a recommendation about through the course of your
18 career?

19 A That would be very difficult. I've been the
20 consultant to the department of corrections gender 09:27:30
21 identity committee since, I think, 2008 and every month
22 since that time, with less than one handful of
23 exceptions, I've been present at discussions, and we've
24 recommended accommodations in prison to people who
25 declare identity as a trans woman. And I would say 09:27:58

1 probably, and I ask you not to hold me to this number,
2 40 times.

3 Q Sorry, 40 times describes what?

4 A That -- that I've joined a group of people who
5 decided to provide electrolysis, canteen item -- 09:28:25
6 special privileges for canteen items, that is, female
7 canteen items, the ability to shower alone, the ability
8 to be tapped down or searched by a female attendant,
9 not a male attendant, a correction officer, hormone --
10 the beginning of hormone treatment and -- and, of 09:28:52
11 course, bilateral mastectomies and -- and on several
12 occasions, male gender confirming surgery for biologic
13 males who are living as trans women. In other words,
14 the whole gamete of services.

15 Q So 40 times you've recommended something -- or 09:29:19
16 joined in a recommendation for something for -- a
17 prisoner to receive medical care, as you've broadly
18 described that term?

19 A Yes.

20 Q And then how many times can you estimate where 09:29:34
21 you had made a recommendation that the prisoner should
22 not receive medal care, as you've broadly defined it?

23 A I don't think I've ever recommended that no
24 treatment be offered to this person. The -- the --
25 because the treatment involves that entire array of 09:30:07

1 matters that I just delineated.

2 And so prisons -- or at least Massachusetts,
3 where I work as a consultant, has been very --
4 eventually, by 2008, has been -- have been very
5 interested in providing individual services to -- to 09:30:26
6 help these people diminish their pain about their
7 incongruence, and I have been one of the people who
8 devised the program.

9 Q The prisoner that you reco- -- you
10 recommended -- sorry -- that you were referring earlier 09:30:49
11 to, the one in the Clark matter, do you recall us
12 discussing that?

13 A I do.

14 Q And that person identifies as female; correct?

15 A Yes. 09:31:00

16 Q Do you view that person as a female?

17 A I view that person as a trans woman.

18 Q You have just testified that you've never
19 recommended that a -- no treatment be offered to a
20 prisoner for gender dysphoria; is that correct? 09:31:22

21 A I'm hesitating because "no treatment"
22 includes -- would include all of the above, of the
23 array I previously listed, and at this moment, I don't
24 recall ever saying no treatment should be given to this
25 individual, no accommodation should be given to this 09:31:47

1 individual.

2 Q Do you recall if you've ever recommended that
3 no surgery be permitted for an individual in prison?

4 A Oh, yes, I have. I have said that I didn't
5 think sex reassignment surgery -- in those days, that's 09:32:06
6 what we called it, but it's now called gender
7 confirming surgery -- I have said I did not think
8 sex -- that kind of surgery was indicated or
9 necessary -- medically necessary.

10 Q And so how many times did you say that surgery 09:32:26
11 was medically necessary?

12 A Would you repeat that, please.

13 Q How many times did you say that surgery was
14 medically necessary for a prisoner?

15 MR. BROOKS: Objection; ambiguous. 09:32:45

16 THE WITNESS: You may or may not know that I
17 do not like the term "medically necessary." I prefer
18 to use the term "would be psychologically beneficial to
19 this person." So that's the reason I'm hesitating
20 answering your question. 09:33:12

21 I generally avoid using the term "medical
22 necessity." Instead, I try to make a determination
23 whether I think, in the -- in the long run, this
24 particular intervention that we're talking about would
25 be psychologically beneficial to the patient. 09:33:29

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1 BY MS. HARTNETT:

2 Q My question is whether you've ever recommended
3 any gender confirming surgery as medically necessary
4 for a prisoner.

5 A Yes, I -- I have signed my name to such 09:33:47
6 documents, such recommendations, because where I work,
7 in Massachusetts, this is the way that the -- most of
8 the staff and -- and -- that -- that is the common term
9 used to -- to justify that kind of intervention.

10 Q How many times have you signed your name to 09:34:10
11 that kind of intervention for a prisoner?

12 A Perhaps five times.

13 Q And you referenced the Soneeya matter;
14 correct?

15 A Correct. 09:34:38

16 Q And years earlier than the 2019 recommendation
17 that you just described, you testified against surgery
18 for that prisoner; correct?

19 A That is not correct.

20 Q What's not correct about that? 09:34:50

21 A That I did not testify -- I did not testify
22 against sex reassignment surgery.

23 Q Did you testify against something earlier in
24 that matter?

25 A I testified the recommendation to -- to have 09:35:05

1 what the judge called a soft landing, like first
2 transferring the person to a female facility, and then,
3 based upon her adaptation there, to have sex
4 reassignment surgery.

5 In fact, that was really -- the issue was not 09:35:29
6 whether the person should eventually have sex
7 reassignment surgery, but -- but whether it should be
8 done before transfer to the female facility or after
9 transfer.

10 Q Did that prisoner seek sex reassignment 09:35:46
11 surgery before transfer?

12 A Please repeat that.

13 Q Did that prisoner seek sex reassignment
14 surgery before transfer?

15 A She did until we presented this idea to her, 09:36:04
16 and she jumped at the idea. She thought it was a very
17 good idea when we interviewed her. And by the time
18 this case got to court, her attorneys were arguing for
19 immediate sex reassignment surgery. But --

20 Q So she -- by the time you were -- oh, pardon 09:36:27
21 me. Please complete your answer.

22 A So we were aware that, because we were in the
23 room when we -- I discussed this with her, she was very
24 happy with the idea of transfer with the -- because she
25 was very positive that she would have a fine adaptation 09:36:41

1 among women prisoners, and she was delighted.

2 And then months later, when this came to
3 trial, the -- her attorney arg- -- was arguing against
4 that.

5 Q So you testified against her wishes as 09:37:05
6 expressed by her attorney at trial; correct?

7 A I never conceived that I was testifying
8 against Soneeya. You may do that, but I -- that's not
9 my concept.

10 Q In the cases where you've given testimony 09:37:24
11 about employee healthcare coverage, you were testifying
12 against the employee healthcare coverage for gender
13 dysphoria; correct?

14 A Incorrect.

15 Q What's incorrect about that? 09:37:38

16 A What I was testifying to is my understanding
17 of the state of science. I was not taking a stand that
18 people should not have healthcare coverage. I was
19 trying to inform the Court about what we knew about
20 this subject and what we don't know about this subject. 09:37:58

21 I didn't take a position that -- that I knew
22 what should be done. I was just here as a -- to offer
23 what I understood about the state of science, about
24 various aspects of surgical and medical and
25 psychological care for the trans population. 09:38:18

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1 Q Are you aware in the Kadel and the Claire
2 matters -- those are the North Carolina and Florida
3 employee healthcare coverage matters -- your testimony
4 was submitted by the defendants in that case against
5 the relief being sought? Are you aware of that? 09:38:37

6 A I was aware that -- who employed me and what
7 their purposes were, but -- but I was not enjoining
8 psychologically with the idea that I was doing anything
9 but offering the Court what I hope to be an objective
10 appraisal of the state of knowledge based upon 09:39:01
11 literature and, you know, participation in trans care
12 over the years.

13 Q So were you, in those two matters, agnostic as
14 to whether the employees received the healthcare
15 coverage or not? 09:39:21

16 A Agnostic?

17 Q That you didn't have a view.

18 A Would you -- would you mind explaining that
19 term? I'm -- I usually understand that in terms of
20 religious notions. 09:39:34

21 Q That you did not have a view -- in those
22 cases, Kadel and Claire, is it fair to say you did not
23 have a view as to whether the healthcare coverage
24 should be extended or not?

25 A I felt insufficient to make a societal 09:39:47

1 decision. I'm not an expert in the insurance industry
2 at all. I -- I am certainly not an expert in the
3 political processes in any particular state. The
4 only -- the only knowledge base that I feel I have
5 comes from the study of the literature and the 09:40:05
6 participation in trans care, both in the community and
7 in prison systems.

8 And so the fact that the State used my
9 testimony does not really equate, in my mind, with my
10 position on whether or not people should have 09:40:31
11 healthcare insurance.

12 I -- again, to repeat, my understanding is I
13 am somewhat knowledgeable about the state of science in
14 this area and that the various people on law -- on the
15 side of -- in -- in -- in judicial issues -- judicial 09:40:48
16 matter want somebody who can articulate the state of --
17 of knowledge. And that's what I do.

18 The state of knowledge should be applied, in
19 my view, to both sides of the issue, not just, you
20 know, the State or the Board of Education. It should 09:41:09
21 be -- it should be established -- it should be relevant
22 to the plaintiff's side.

23 Q Were you paid by the State in the
24 North Carolina and the Florida matters for your
25 testimony? 09:41:27

1 A Ultimately, I think I was paid by the State,
2 but the check did not come from the State. The check
3 came from the lawyer who employed me.

4 Q Understood. Have you ever provided testimony
5 with your -- what you've described as your expertise in 09:41:46
6 favor of -- on the side of extending the healthcare
7 coverage to tran- -- to people seeking care for gender
8 dysphoria?

9 A No attorney representing that side of the
10 issue has ever hired me, but if they would, I would be 09:42:03
11 happy to present my knowledge or -- to, and they can do
12 what they want with that testimony.

13 Q You were deposed in at least one child custody
14 matter in Texas where a child wanted to transition; is
15 that correct? 09:42:26

16 A I was.

17 Q And you testified in trial at that matter,
18 too?

19 A I did.

20 Q And was your testimony in that case in 09:42:37
21 opposition to the desired transition?

22 A The testimony in that case was to present the
23 state of knowledge about this matter. I did not take a
24 position that a child should or should not have a
25 particular treatment. I was just informing the Court, 09:42:56

1 as I previously described to you. I thought I was a
2 witness about the nature of knowledge about trans
3 children.

4 THE WITNESS: Could you get me some water,
5 please.

09:43:16

6 BY MS. HARTNETT:

7 Q Sorry, is your testimony that you, in that
8 case, in the -- this is the Younger matter; is that
9 correct?

10 A Yes. That's what I understand you to be
11 referring to.

09:43:23

12 Q And your testi- -- your testimony today is
13 that you were not testifying in opposition to the
14 transition that the child -- of the child in the
15 Younger matter?

09:43:36

16 A I was hired by the lawyer who was representing
17 the father who did not want his son to be transitioned
18 to a little girl, socially. But I was not testifying
19 that the child should not be transitioned. I was
20 testifying -- I had no knowledge of that -- I wasn't
21 asked for that question. That -- that was never asked
22 of me, Ms. Hartnett. What was asked of me was what we
23 knew about this subject. And, therefore, I felt
24 comfortable sharing the state of knowledge and -- and
25 what is missing from our knowledge.

09:44:02

09:44:23

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1 Again, it -- it has the appearance that I was
2 testifying against the socialization of the child, but
3 I think if you look closely at that, what I was doing
4 was telling the Court what was known and what was not
5 known and what the consequences were, the implications 09:44:45
6 of treating the child one way versus another.

7 Q So you did not testify in that matter that
8 desistance was preferable to affirmation?

9 A I actually don't recall if I made that
10 statement. It's -- I just don't recall. 09:45:09

11 Q Okay. Has your testimony -- oh, sorry.

12 Have you testified in any other matters of --
13 similar to the Younger matter, in which parents were
14 disputing the proper care of their child who sought
15 care for gender dysphoria? 09:45:37

16 A Yes. There was a case that I believe is
17 sealed in the Tucson court. I don't know if I'm
18 allowed to give the name. I presume I can give the
19 name. I don't know.

20 MR. BROOKS: If -- if it's sealed, I would not 09:45:56
21 give any identifying information.

22 THE WITNESS: But the answer to your question
23 is yes.

24 BY MS. HARTNETT:

25 Q And in that matter, did your -- was your 09:46:05

1 testimony used by the party who was opposing the
2 treatment for gender dysphoria for the child?

3 A In that particular matter, it was the parents,
4 who hired me, who objected to losing custody of their
5 child when the child was hospitalized for a suicide 09:46:38
6 gesture and told the people in the hospital that her
7 evil parents were preventing her, at age 13, from
8 transitioning to being a boy. And her parents --

9 MR. BROOKS: I'm just going to interrupt and
10 caution the witness. I'm not part of that case, but 09:47:03
11 I -- nor do I want Dr. Levine to violate any
12 confidentiality obligations.

13 So as you answer, whatever level of generality
14 you think is appropriate, just be very careful not to
15 disclose information that you believe you received in 09:47:18
16 confidence and that remains confidential given the
17 conduct of that case.

18 So I -- I don't want us in our proceedings to
19 violate any obligations of that proceeding.

20 THE WITNESS: Well, given that, I actually 09:47:35
21 think anything I would say about this would violate the
22 confidentiality rule here, and I think I've told you
23 enough about the case.

24 MS. HARTNETT: Well, I don't want to waste our
25 time on the record discussing this, but we have a right 09:47:51

1 to discovery into your testimony, so we will follow up
2 with counsel to figure how to get it.

3 BY MS. HARTNETT:

4 Q When was this testimony given?

5 A In the spring of 2021. And if I'm wrong, it 09:48:13
6 was in the spring of 2020.

7 Q Thank you. And, sorry, what -- was the
8 testimony given in deposition or trial or some other
9 fashion?

10 A In juvenile court. 09:48:32

11 Q In what form did the testimony take?

12 MR. BROOKS: Objection; vague.

13 BY MS. HARTNETT:

14 Q Just, sorry, meaning written or oral.

15 A Oh, in person? I was in -- I was in person by 09:48:50
16 video, and I was cross-examined, you know.

17 I also submitted a report of the psychiatric
18 evaluation.

19 Q Any other testimony that you've given in a
20 case involving parents and the potential care of a 09:49:13
21 child with gender dysphoria?

22 A I submitted a rebuttal to a report in a case
23 in Cincinnati I think the first week of January of this
24 year. The case is called Siefert, S-I-E-F-O-R-D (sic),
25 or E-R-T, something like that. Siefert versus Hamilton 09:49:49

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1 County, which is the Cincinnati county.

2 So that would be the answer to your question.

3 Q And what's the nature of that matter, the
4 Siefert matter?

5 A The -- the child, who was identifying as a 09:50:08
6 trans male, were treated -- the parents were treated
7 during the hospitalization as persona non grata and the
8 hospital refused to discharge the patient even though
9 the patient did not meet criteria for continued
10 hospitalization and -- so the -- the parents were 09:50:46
11 objecting to the loss of parental rights.

12 Subsequently, the child reidentified as a
13 female and -- so I don't know what the outcome has been
14 legally. It's in process.

15 And I just commented on the limitations of 09:51:07
16 the -- another expert who felt that it was justified to
17 keep the child in the hospital against the parents'
18 wishes, for two and a half months.

19 Q In the Tucson matter that you discussed,
20 which, again, we will follow up on, but can you just 09:51:34
21 tell me if that's been resolved? Do you know if that's
22 reached a conclusion?

23 A Yes, that -- the -- the particular judicial
24 issue was -- was resolved. Whether or not the parents
25 are going to continue to sue the -- the child welfare 09:51:56

1 organization, I -- I don't know. I haven't heard -- I
2 haven't had any follow-up on the case since it was
3 adjudi- -- since it was resolved.

4 Q Thank you. Has your testimony ever been
5 excluded by a court? 09:52:18

6 A Yes.

7 Q When?

8 A 2015.

9 Q What matter was that?

10 A It was in the matter of a prisoner named 09:52:33
11 Noseworthy (sic) in California.

12 Q And what is your understanding of how your
13 testimony was excluded?

14 A Well, I didn't actually have testimony. I
15 submitted a psychiatric evaluation and a 09:52:50
16 recommendation, and I was never invited to a -- a
17 courtroom for that.

18 The judge -- I presented, in my written
19 deposition, an account of a female prisoner who had a
20 very extremely negative outcome from genital surgery, 09:53:12
21 and the judge -- the judge thought I was lying about
22 this case, and he also did not think that -- that I
23 followed the Harry -- the WPATH standards of care, and
24 he dismissed my -- without asking me one question,
25 without asking me do I have any evidence to show that I 09:53:39

1 wasn't lying about this case, he -- he dismissed my
2 recommendation.

3 So I'm aware that judges have their -- judges
4 can make mistakes. Because I, in fact, have in my
5 possession the case history, I saved the case history 09:54:01
6 that was presented to me by the California Department
7 of Corrections, and that -- no one seems to know that.
8 Or at least the judge did not inquire about that. I
9 never had a chance to defend myself and -- so that's --
10 that's when my testimony was dismissed. 09:54:24

11 Q Thank you. Is there any other time where your
12 testimony has been excluded by a court?

13 MR. BROOKS: Objection; vague.

14 THE WITNESS: Well, I believe that the impact
15 of that judge in the Noseworthy -- Norsworthy case has 09:54:48
16 influenced two other cases to discredit my position, at
17 least whatever I said on those other cases -- on one
18 other case.

19 One of the cases that -- that my name gets
20 brought up about, I actually never submitted any 09:55:10
21 testimony to, but someone quoted what I had taught in a
22 workshop; and, therefore, the judge dismissed that
23 testimony.

24 You should understand that since that time and
25 even before that time, my testimonies have been 09:55:29

1 accepted by various courts, and -- for example, in the
2 district court of Arizona, in a case involving
3 insurance coverage, the judge quoted my testimony.
4 That -- that was appealed to the Ninth Circuit Court,
5 and the Ninth Circuit Court made -- made a reference 09:55:49
6 to, but did not name my testimony.

7 And so it seems to me that since -- before
8 2015, in that particular case, and subsequent to 2015,
9 my testimony has been accepted by various courts, in
10 various matters involving, you know, trans issues that 09:56:10
11 I am asked to opine about.

12 Q Thank you. Is there any other example you can
13 think of where your testimony has been excluded by a
14 court?

15 MR. BROOKS: Objection, vague. 09:56:37

16 THE WITNESS: Well, I'm aware of the
17 Noseworthy case, the -- the Edmo case, and there's a
18 Hecox case.

19 But again, all these exclusions were
20 objections to my expertise derived from the judge in 09:56:58
21 the Norsworthy case.

22 And the answer to your specific question, I am
23 not aware of any other situation where my testimony was
24 excluded.

25 Q Thank you. For the Noseworthy case, you did 09:57:13

1 submit an expert report; correct?

2 A I -- I -- yes.

3 Q So you understand this case involves sports;
4 correct?

5 A Yes. 09:57:42

6 Q What, if any, prior testimony have you given,
7 whether by declaration or report or oral testimony,
8 about transgender participation in sports?

9 A I believe that both in the Connecticut case
10 and in the Hecox case the expert opinion report that I 09:58:09
11 gave about the state of knowledge in this field has
12 been submitted for the Court's consideration.

13 I am not an expert, as you probably know, in
14 matters of athletics and physiology. I am only
15 providing information that I feel I know about, which 09:58:41
16 is the knowledge and the lack thereof about certain
17 issues related to trans care.

18 So I -- I've never really, as far as I know,
19 as far as I remember, made an opinion about this should
20 happen or this should not happen. I'm just providing 09:59:06
21 information to the courts about what I know and what is
22 not known by society or by science.

23 Q Thank you. So in this case, for example,
24 B.P.J., is it fair to say you do not have an opinion as
25 to whether she should be permitted to play sports? 09:59:25

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1 A I do not have an opinion.

2 Q Have you -- setting aside the context of
3 transgender participation in sports, have you ever
4 given any testimony of any kind in a matter related to
5 sports?

09:59:51

6 A I can't think of any.

7 Q Have you given any prior testimony, whether by
8 declaration, report or oral testimony, about
9 prepubertal trans- -- transgender children?

10 MR. BROOKS: Let -- let me ask you to restate 10:00:16
11 that question. Not to rephrase it, necessarily. I
12 just want to hear it back.

13 MS. HARTNETT: Sure.

14 BY MS. HARTNETT:

15 Q Have you given any prior testimony by 10:00:22
16 declaration, report or oral testimony involving
17 prepubertal transgender children?

18 A I'm hesitating because I have written about
19 informed consent and -- and that my writings about
20 informed consent have covered all trans, beginning with 10:01:02
21 prebu -- prepubertal children. But your question is
22 about giving testimony about that. I would imagine
23 that in the Younger I may have raised the issue of --
24 of what we know -- I mean, I did raise tissue of what
25 was known and what is not known. 10:01:38

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1      So I would imagine the answer to your question
2      must be yes.
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3 And the Arizona case that is sealed is not
4 about a preber -- prepubertal child. But, of course,
5 in taking a history of any child in adolescence, we 10:01:55
6 certainly take histories of their prepubertal period
7 and the behaviors evidenced during that time.

8	So I just find the answer to your -- I'm not	
9	actually sure what the answer to your question should	
10	be.	10:02:13

11 Q Did the Younger case involve a prepubertal
12 child?

13	A	It did.

14 Q And the Arizona case did not involve a
15 preber -- prepubertal child; is that correct? 10:02:26

16 A That's -- that's correct.

17 Q And how about the Cincinnati case you
18 mentioned, was that a prepubertal child?

19	A	No.

20 Q Can you think of any other -- and I'm setting 10:02:34
21 aside your nonjudicial work, but any -- any
22 testimony -- and I -- that was my question. Thank you
23 for focusing on that -- but any testimony you've given
24 other than these examples that you consider to be
25 related to prepubertal transgender children? 10:02:57

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1 A The key word to your question is "testimony."
2 And so I have played -- I have -- I have offered
3 opinions to lawyers that never rose to the point of
4 testimony. So the --

5 Q And let me be clear. 10:03:25

6 A The answer to your question must be no.

7 Q And for this question, I was just trying to be
8 clear when I said "testimony," whether by written
9 declaration, written report or oral testimony.

10 And so I want to -- just using that 10:03:41
11 understanding of "testimony" for this question, other
12 than the Younger case, have you given any prior
13 testimony regarding a prepubertal -- in a case
14 involving a prepubertal transgender child?

15 A I'm trying to be helpful and -- and 10:03:55
16 informative to your question.

17 I think the -- I think the -- the -- to the
18 best of my knowledge, the answer is no, but people use
19 my knowledge, in my previous publications, and call me
20 sometimes and ask me opinions about matters -- the 10:04:24
21 lawyers, I mean, or guardian ad litem persons -- and --
22 but it's not testimony per se. I guess it would be
23 consultation.

24 Q Thank you. And then just again sticking with
25 testimony, which for this question I'm meaning to be 10:04:52

1 written or oral testimony in a judicial proceeding,
2 have you given any testimony about a case involving a
3 transgender adolescent, other than the Arizona case and
4 the Cincinnati case?

5 A At the moment, I can't think of any. 10:05:21

6 Q And have you -- and this is, again, for the
7 purposes of this questions meaning -- "testimony" to
8 mean written or oral testimony in a judicial
9 proceeding. Have you ever given testimony in support
10 of a transgender party? 10:05:40

11 A In support of a transgender what?

12 Q Party.

13 A Party. Please repeat that question.

14 MS. HARTNETT: Could the reporter read that
15 back. I'm not sure I could do it. 10:05:50

16 (Record read.)

17 THE WITNESS: I guess the key word in your
18 question is "support." And I want you to know that
19 when I testify about the state of knowledge, I actually
20 think that because my perspective is a long-term life 10:06:48
21 cycle perspective, I think of that my knowledge base
22 sometimes suggests that I'm actually being quite
23 supportive in -- in trying to have people understand
24 what the consequences of -- of, quote, affirmative or
25 supportive care actually may mean, what the risks are. 10:07:11

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1 So I believe your understanding of the word
2 "support" is different than my understanding of the
3 word "support."

4 But once again, I want to repeat, I
5 conceptualize what I'm doing is accurately stating the 10:07:32
6 state of science, of what is known, what is not known
7 and what we need to do in order to get the answers to
8 the unknown questions. That's what I'm doing.

9 I'm not supporting this or supporting that.
10 I'm not against this. I'm not against that. I'm 10:07:52
11 trying to give an appraisal of what we know, in a
12 scientific sense. Because of the one principles of
13 medical ethics is that science should lead our
14 therapeutics.

15 BY MS. HARTNETT: 10:08:07

16 Q Dr. Levine, you understand that your testimony
17 in this matter has been provided by the State, the
18 defendants, in support of their position; is that
19 correct?

20 A Yes. 10:08:15

21 Q And so when I use the word "in support of," in
22 the context of a judicial proceeding, you understand
23 that your testimony, what has been submitted in these
24 proceedings, is submitted in support of one party or in
25 support of another party; correct? 10:08:36

1 A Yes. But that has to do with legal processes.
2 What -- what I am supporting is to inform the court of
3 what is known and what is not known. If you were to
4 hire me to tell what -- the Court what is known and not
5 known, I think I would be giving the same testimony. 10:08:58

6 Q Let me ask you again, then. Which of -- have
7 you ever previously given written or oral testimony
8 that was submitted in support of the transgender party
9 in a judicial proceeding?

10 MR. BROOKS: Objection. 10:09:16

11 THE WITNESS: You asked that question before,
12 so I'm going to answer it in the same way I answered it
13 before. It depends on your notion or my notion of
14 "support."

15 BY MS. HARTNETT: 10:09:36

16 Q I'm using the notion of "support" that we just
17 discussed, which is -- like, for example, your
18 testimony in this matter is being submitted in support
19 of the defendants. You understand that?

20 A I do. 10:09:44

21 MS. DENIKER: This is Susan Deniker. I just
22 want to place on the record an objection to the form.

23 BY MS. HARTNETT:

24 Q And using that understanding of "support," do
25 you agree with me that you have not previously had your 10:10:00

1 testimony submitted in a judicial proceeding in support
2 of the transgender party; correct?

3 MR. BROOKS: Objection.

4 THE WITNESS: Incorrect. I already told you
5 that I have recommended transfer to a female prison and 10:10:10
6 ultimate sex reassignment surgery and that -- for --
7 for the Soneeya case, and there were -- there was
8 another case -- another prisoner at the same time that
9 we made the same recommendation for.

10 And I've already told you that I have -- I -- 10:10:33
11 I -- I have participated in the support of -- of
12 bilateral mastectomies for female prisoners, but
13 that -- none of those cases have gone to court. So
14 I -- I guess that's not relevant to your question.

15 BY MS. HARTNETT: 10:10:51

16 Q Right. I was asking about whether you've
17 submitted, in a judicial proceeding, an opinion on the
18 side of the transgender party. Have you?

19 MR. BROOKS: Objection.

20 THE WITNESS: I already answered that question 10:11:10
21 three times about Soneeya.

22 BY MS. HARTNETT:

23 Q Can you please answer my question?

24 Have you ever submitted an expert opinion on
25 the side of the transgender party? 10:11:20

1 MR. BROOKS: Objection.

2 THE WITNESS: In your narrative --

3 BY MS. HARTNETT:

4 Q In a --

5 A In your -- 10:11:32

6 Q Sorry, I'm just trying to be really clear
7 since I understand you're disputing the term "support,"
8 which I thought was clear, but I -- I -- I'm listening
9 to you, and now I'm asking you whether, in a judicial
10 proceeding, you've ever submitted testimony on the side 10:11:43
11 of the transgender person, the formal side of the case.

12 MR. BROOKS: Objection. Experts don't
13 themselves submit anything in court.

14 You may answer, if you recall.

15 THE WITNESS: I may answer? 10:12:09

16 MR. BROOKS: If you recall.

17 THE WITNESS: I -- I find myself unable to
18 answer that question.

19 MS. HARTNETT: Okay. I'm going to introduce
20 an exhibit now, so we'll see how this Exhibit Share 10:12:22
21 works for you. Just a moment here.

22 MR. BROOKS: Tell me when you've placed it in
23 the folder, and I will then refresh the folder --

24 MS. HARTNETT: Will do.

25 We're starting with 86. Okay. Just one 10:12:49

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1 moment, please.

2 (Exhibit 86 was marked for identification
3 by the court reporter and is attached hereto.)

4 MR. BROOKS: Are you doing all right, or do
5 you want to take a break? 10:13:02

6 THE WITNESS: Well, she said we would have a
7 break in an hour. It's a little over an hour.

8 MR. BROOKS: If you're -- you're about to
9 introduce a document and you're taking a little time to
10 get that straight, let's take a short break. 10:13:07

11 MS. HARTNETT: That works for me. Thank you.

12 MR. BROOKS: All right.

13 THE VIDEOGRAPHER: We're off the record at
14 10:13 a.m.

15 (Recess.) 10:22:57

16 THE VIDEOGRAPHER: We are on the record at
17 10:23 a.m.

18 BY MS. HARTNETT:

19 Q Now, Dr. Levine, you've been retained as an
20 expert witness in this case, B.P.J.; correct? 10:23:20

21 A Correct.

22 Q Who retained you?

23 A Initially, David Tryon.

24 Q And was there someone who retained you after
25 that? 10:23:37

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1 A I -- I think David Tryon, in the matter and
2 means that I don't understand, created a liaison with
3 Alliance for Defending Freedom, Mr. Brooks, and then
4 they became -- so then I am -- I've been recruited by
5 both Mr. Tryon and Mr. Brooks, their -- their 10:24:10
6 particular institutions.

7 Q And with respect to Mr. Brooks, he's
8 affiliated with the Alliance for Defending Freedom, is
9 that your understanding?

10	A Yes.	10:24:29

11 Q Have you previously worked with the Alliance
12 for Defending Freedom on any matter?

13 A Yes. I -- I think of it as working with
14 Mr. Brooks.

15	Q And I don't want to --	10:24:45

16 A Mr. Brooks is associated with the Alliance for
17 Defending Freedom, so I guess the answer to your
18 question is yes.

19 Q When did you first work with Mr. Brooks?

20	A	In the Young -- Young -- in the Younger case.	10:24:57

21 Q And that was the Texas matter we discussed?

22	A Yes.

23 Q And I think you testified in your deposition
24 in the Claire matter, that's the Florida case, that you
25 worked with a lawyer from the Alliance Defending 10:25:17

1 Freedom to write your report in Younger; is that right?

2 A In -- the question is a little confusing to me
3 because you brought up the Florida case, and I don't --
4 could you repeat the question and ask me just one
5 question? 10:25:36

6 Q Sure. I was trying to orient you that I
7 understand that you gave a deposition in that Florida
8 matter of Claire; correct?

9 A I did.

10 Q And in that case, you were asked some 10:25:44
11 questions about your report. Do you remember that?

12 A You mean my report in the Younger case?

13 Q Correct.

14 A I don't remember that. I'm not denying it,
15 but I just don't remember that. 10:26:00

16 Q Yeah, was just curious about the kind of
17 genesis of your report in this case, and so what -- I
18 guess what I'll ask you is, is it -- is it fair to say
19 that you worked with a lawyer from the Alliance for
20 Defending Freedom to prepare your report in the Younger 10:26:14
21 matter? Correct?

22 A Yes.

23 Q And then your report in the Claire matter in
24 Florida was derivative of the Younger report; correct?

25 A I don't think that's correct. 10:26:27

1 Q What's not correct about it?

2 A I think the Florida case was about three --
3 the plaintiffs, I think, were three adults. The
4 Younger case was about, as we established before, a
5 very young child. 10:26:53

6 Q Okay. So your testimony is that the report
7 you submitted in the Claire case was not a derivative
8 of the report that was submitted in Younger; is that
9 right?

10 MR. BROOKS: Object to the form. 10:27:06

11 THE WITNESS: It's -- it's very difficult for
12 a person like me to know how my clinical activities and
13 my consulting activities interplay and influence one
14 another.

15 I am a very busy person, doing a lot of 10:27:28
16 different things, and I often think about, in a very
17 pleasing way, how my various activities cross-fertilize
18 my -- and stimulate my views, and what I read in one
19 case for one particular matter may stay with me and
20 help me understand yet another matter. 10:27:48

21 So this cross-fertilization is a very
22 intellectually stimulating process, but it makes me
23 very unable to answer the question about what
24 influenced what. You know, sometimes I read a novel
25 and it influences, I think. 10:28:08

1 But it's hard -- I -- I can't really track,
2 with any degree of certainty, what influences what.

3 Perhaps if you had specific -- more specific
4 questions, I may be able to give you an opinion. But
5 based on what you just said, I -- I -- I'm at a loss to 10:28:29
6 answer it definitively.

7 BY MS. HARTNETT:

8 Q So I think my -- just to be clear for the
9 record, then, you cannot answer definitively whether
10 the report you submitted in the Claire case was a 10:28:44
11 derivative of the report that was done in the Younger
12 case; is that fair?

13 MR. BROOKS: Objection; vague.

14 THE WITNESS: Based on how I currently think
15 at the moment, I think it's correct. 10:28:59

16 BY MS. HARTNETT:

17 Q Sorry, correct that you -- you can't take a
18 view on that?

19 A It is correct that I don't know whether the
20 Younger case influenced my -- a specific -- I mean, 10:29:18
21 I -- I probably wrote many, many pages for the Florida
22 case, and so, you know, maybe there's a sentence or a
23 paragraph or two that, in my mind, was conceptualized
24 in part because of -- of my experience in the Younger
25 case. 10:29:38

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1 But at this moment, I cannot tell you
2 definitively this influenced me or this did not
3 influence me.

4 Number one, that was a couple of years ago.
5 Lots of things have happened in my brain in the last 10:29:51
6 couple of years.

7 Q Did any novels affect your expert opinion in
8 this case?

9 A Not that I can think of.

10 Q You mentioned that you first encountered 10:30:03
11 Mr. Brooks on behalf of ADF in the Younger case.

12 Can you tell me how you got connected with him
13 in that matter?

14 A He called me. He had read two papers, I
15 believe, that I had published, and he wanted to talk to 10:30:22
16 me.

17 Q So for this case, B.P.J., what were you asked
18 to do in terms of presenting an expert opinion?

19 A He wanted me to present the state of
20 knowledge, what is known and what is not known, about 10:30:47
21 trans care as a background for this particular case.
22 But he was aware, and -- and I told him very clearly --
23 that he was quite aware. I didn't have to tell him. I
24 just reminded him that I am not an expert in the
25 physiology of estrogen and testosterone blockages for 10:31:11

1 athletic capacities, I'm not an expert in lung volumes
2 and cardiac capacities. And -- and I asked him why --
3 why he would --

4 MR. BROOKS: I'm going to instruct you not to
5 disclose the substance of conversations with your 10:31:28
6 attorneys.

7 THE WITNESS: All right. Thank you.

8 BY MS. HARTNETT:

9 Q Was that a conversation you had before you
10 were retained in this matter, Dr. Levine? 10:31:36

11 A Was that a conversation?

12 MR. BROOKS: Counsel, the -- the witness can
13 answer that question, but any conversations surrounding
14 the retention, I will instruct the witness not to
15 answer. 10:31:53

16 THE WITNESS: I wondered why he needed my
17 testimony in this case. He provided an answer for me.

18 BY MS. HARTNETT:

19 Q Do you view your testimony as relevant to this
20 case? 10:32:07

21 MR. BROOKS: Objection.

22 THE WITNESS: Insofar as you make claims --
23 you -- that your side may make claims that is not --
24 that are not scientifically correct or established, it
25 may very well be relevant. 10:32:29

1 But that is not a question for me to decide.

2 That's a question for lawyers on both sides and for the
3 judge.

4 Again, I'm just -- I'm just -- I just have a
5 certain limited understanding and knowledge which I 10:32:44
6 believe the Court might benefit from having.

7 BY MS. HARTNETT:

8 Q Did you prepare for the deposition today?

9 A Yes.

10 Q What did you do to prepare? 10:33:03

11 And please don't disclose your
12 communications that you had -- the substance of the
13 communications that you had with counsel.

14 A I reread my report Sunday evening. I met with
15 counsel yesterday afternoon. 10:33:17

16 Q How long did you meet for yesterday afternoon?

17 A I'm sorry, how long, did you say?

18 Q Yes, how long did you meet with counsel
19 yesterday afternoon?

20 A Between 1:30 and quarter to 7:00. 10:33:30

21 Q Did you review any documents to prepare for
22 this deposition other than your expert report?

23 MR. BROOKS: And you -- you can answer that
24 question yes or no without identifying specific
25 documents. 10:33:44

1 THE WITNESS: Yes.

2 BY MS. HARTNETT:

3 Q Did you review the rebuttal report of
4 Dr. Safer?

5 MR. BROOKS: I'm going to instruct the witness 10:33:52
6 not to answer questions about what specifically he
7 reviewed with counsel yesterday.

8 MS. HARTNETT: I believe I have a right to
9 know what, if any, additional documents he's reviewed
10 before the deposition other than his report. 10:34:08

11 MR. BROOKS: On the contrary. I believe that
12 selection is my work product. And I stand by my
13 instruction.

14 BY MS. HARTNETT:

15 Q Outside the presence of your counsel, is there 10:34:15
16 anything other than the expert report that you reviewed
17 to -- before your deposition?

18 MR. BROOKS: On your own, outside our session
19 yesterday, did you review anything else in preparation
20 for your deposition? 10:34:32

21 THE WITNESS: No.

22 BY MS. HARTNETT:

23 Q Do any materials other than those cited in
24 your expert report inform your opinion in this matter?

25 MR. BROOKS: Objection. 10:34:49

1 THE WITNESS: As was -- if you have read my
2 curriculum vitae, I have recently published two papers
3 about issues. One is titled the Reflections of a
4 Clinician about the trans -- the care of trans youth
5 that was published in November, in the Archives of 10:35:19
6 Sexual Behavior. And about 16 days ago, a new article
7 appeared online about informed consent, Reconsidering
8 Informed Consent in the Treatment of Trans Children,
9 Adolescents, and Young Adults.

10 And so I can't really separate the processes 10:35:46
11 of writing these papers from, you know, the submission
12 of documents in this particular case.

13 But in a literal answer to your question, did
14 I -- did I review any particular documents in -- in --
15 in preparation for this testimony today, this 10:36:07
16 deposition today? The answer is no. But the process
17 of writing articles is a deep, you know, dive into all
18 kinds of issues and -- so I'm busy with this -- these
19 sub- -- these topic areas.

20 But I guess the answer to your question is no. 10:36:31

21 BY MS. HARTNETT:

22 Q Thank you. And what I need to understand
23 and -- and find a way to get that information from you,
24 notwithstanding your counsel's objection, but he should
25 make any direction he sees fit to make, in -- in your 10:36:46

1 expert report, you refer to certain materials in this
2 case that you had reviewed as a basis for your opinion.

3 Do you recall that?

4 MR. BROOKS: Do you want to direct the
5 witness's attention to what you're referring to? 10:37:03

6 MS. HARTNETT: Yeah, I can do that, I guess.

7 BY MS. HARTNETT:

8 Q You reviewed Dr. Adkins' and Dr. Safer's
9 declarations before you -- as part of your materials
10 that you rely on in your expert report; correct? 10:37:13

11 A Yes.

12 Q And what I'm trying to understand is whether
13 or not you are going to rely on Dr. Adkins' or
14 Dr. Safer's supplemental declarations as part of your
15 expert opinion in this matter. 10:37:29

16 MR. BROOKS: Counsel, let me -- I'll object
17 and, I think, make a suggestion.

18 The -- is your question whether he has
19 considered those rebuttal reports submitted by
20 Dr. Adkins and Safer? Or did you mean something else? 10:37:47

21 MS. HARTNETT: I would like to know if he has
22 reviewed the expert -- supplemental expert report of
23 Dr. Adkins.

24 Will you allow him to answer that question?

25 MR. BROOKS: I will. 10:38:02

1 THE WITNESS: I think at one point I did.

2 BY MS. HARTNETT:

3 Q Do you understand that Dr. Adkins wrote an
4 initial report and then a rebuttal, including to your
5 report?

10:38:13

6 A Yes.

7 Q Have you reviewed Dr. Adkins' rebuttal,
8 including to your report?

9 A Not -- not in preparation for this deposition,
10 no.

10:38:23

11 Q And did you review Dr. Safer's rebuttal
12 declaration in this case, ever?

13 A I think I have. Yes, I --

14 Q And have you --

15 A I --

10:38:36

16 Q Okay.

17 A I have, yeah.

18 Q And have you reviewed the declaration of
19 Aron Janssen in this matter?

20 A Of Aron who?

10:38:43

21 Q Janssen.

22 A I can't recall that. I may have.

23 Q He's a physician at Chicago Children's
24 Hospital. Is that ringing a bell?

25 A No.

10:39:02

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1 Q Okay.

2 A It's ringing a faint bell.

3 Q All right. If you could go into your "Marked
4 Exhibits," there should now be a marked Exhibit 86.

5 MR. BROOKS: I have that on the screen. 10:39:23

6 MS. HARTNETT: Thank you, Roger.

7 And this is a document that starts with the
8 page that says "Exhibit A," and then it goes on to --
9 it's an attached expert declaration.

10 BY MS. HARTNETT: 10:39:38

11 Q Do you see this document, Dr. Levine?

12 A We're scrolling through it here.

13 Expert declaration of Dr. Levine. Robert
14 Ferguson -- Tingley, yeah, okay.

15 Q And so what -- what is this document, if you 10:39:54
16 know?

17 A This is something I submitted several years
18 ago of -- I think it was about an attempt to censor a
19 psychologist who wanted to provide a certain
20 exploration with a patient, and -- and so I was 10:40:19
21 offering an opinion about, I guess, the
22 psychotherapeutic evalua- -- the evaluation of
23 pyscho- -- the psychotherapeutic processes involving
24 patients.

25 Q And just turning to page 2 of this document, 10:40:38

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1 do you see it says -- are -- are you on page 2 of the
2 PDF?

3 A Let's see. How do I know that?

4 Q The page after the page that says "Exhibit A."

5 A I -- I'm on the page that is the title page 10:40:54
6 that says, Expert Declaration of Dr. Stephen Levine.

7 Q And, Dr. Levine, the caption of this page says
8 "Expert Declaration of Dr. Stephen B. Levine in Support
9 of Plaintiff's Motion for Preliminary Injunction";
10 correct? 10:41:14

11 A Correct.

12 Q And I know we had some discussion before the
13 break about what the word "support" means. In this
14 case, did you understand that your declaration was
15 being submitted in support of the plaintiff challenging 10:41:25
16 the practice that you were referring to?

17 A I guess I now understand that, yes.

18 Q Okay. And just flashing back to the end of --
19 this is a declaration that was submitted in a matter in
20 court in Washington State. 10:41:46

21 Do you see that?

22 A Yes.

23 Q And then at the -- it's -- you can page
24 through, but it appears that you signed this
25 declaration on May 10th, 2021; is that correct? 10:41:57

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1 MR. BROOKS: Well, we'll go to the end and see
2 what we see.

3 THE WITNESS: Let's see. May 2021.

4 BY MS. HARTNETT:

5 Q Okay. And what -- what, if any, additional 10:42:17
6 involvement have you had with the Tingley matter other
7 than submitting this declaration?

8 A I think none.

9 Q Okay. Now, just turning back to the first
10 page or any page, frankly, in this document, you can 10:42:41
11 see there's a caption on the top of the page there.

12 Do you see "Case 2:21-cv-00316"? Do you see
13 that?

14 A Yes.

15 Q And that, I would represent, is the caption 10:42:51
16 for the current case, B.P.J.

17 And this was Exhi- -- this -- this
18 declaration, the version that I put before you, is
19 actually the version that was attached in opposition to
20 plaintiff's motion for preliminary injunction in this 10:43:11
21 case.

22 Did you have an understanding that your
23 declaration from the Washington case was going to be
24 submitted as an attachment in support of the defendants
25 in this matter at the preliminary injunction stage? 10:43:25

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1 MR. BROOKS: Objection.

2 THE WITNESS: No.

3 BY MS. HARTNETT:

4 Q Were you asked to -- for permission before the
5 defendants in this case attached your Washington 10:43:44
6 declaration to the opposition to the preliminary
7 injunction motion in this case?

8 A No.

9 Q Do you recall whether you were asked to submit
10 an expert declaration at the preliminary injunction 10:43:58
11 phase of this case?

12 A Would you clarify that question? I'm not
13 exactly sure what you're asking.

14 MS. HARTNETT: Could the reporter read back my
15 question. 10:44:29

16 (Record read.)

17 MR. BROOKS: Objection.

18 THE WITNESS: I don't know what the
19 preliminary injunction phase was. I don't know the --
20 who the implied person who might have asked me. I -- 10:44:38
21 I -- I'm -- I'm a psychiatrist. I am not a -- I'm not
22 very knowledgeable about your -- about the law and the
23 legal processes.

24 So I -- I just can't answer the question
25 because I don't I understand the terms. 10:44:56

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1 Perhaps you can simplify the question for me.

2 BY MS. HARTNETT:

3 Q What I'm trying to understand -- and thank you
4 for -- for that.

5 I'm trying to understand whether you are aware 10:45:09
6 that your declaration from the Tingley matter was
7 submitted in opposition to the plaintiff's motion for
8 preliminary injunction in this case.

9 MR. BROOKS: Objection; asked and answered.

10 THE WITNESS: I thought I already answered 10:45:23
11 that question.

12 By MS. HARTNETT:

13 Q Okay. Right. And you said, I think, that you
14 were not aware. And then what I'm asking you is, were
15 you asked to prepare a declaration specifically for 10:45:30
16 this case at the preliminary injunction phase?

17 MR. BROOKS: Objection; asked and answered.

18 THE WITNESS: Again, I don't know the phases
19 of this case. And the preliminary injunction phase
20 is -- I don't understand specifically what that means 10:45:49
21 in terms of the long process of adjudication in this
22 case.

23 I was asked to submit a report for this case,
24 but I was not told it was for a preliminary injunction
25 or what- -- an injunction that's not preliminary. 10:46:05

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1 I simply don't know the answer to your
2 question.

3 BY MS. HARTNETT:

4 Q Thank you. When were you retained in this
5 case, B.P.J.? 10:46:15

6 MR. BROOKS: Objection.

7 If you recall.

8 THE WITNESS: I presume it was sometime in
9 2021, but I don't recall the specific date. I -- you
10 know, I could find out, but right now, I -- I -- I 10:46:33
11 can't tell you a specific date. I would presume in the
12 last half of 2021.

13 BY MS. HARTNETT:

14 Q Do you have any objection to your declaration
15 from one case being submitted in another case without 10:46:51
16 your approval?

17 MR. BROOKS: Objection.

18 THE WITNESS: Personally do I have an
19 objection for people using my previous testimony? Yes.
20 I don't -- I don't think that's fair to me because 10:47:06
21 every case is somewhat different. And it feels like
22 it's my work product and that -- but the truth is that
23 I'm naive about the -- about the legal processes, and I
24 think when -- the first time I submitted an expert
25 opinion report, I was shocked that people had read it 10:47:30

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1 who weren't involved in the case.

2 So there was this problem with Dr. Levine not
3 being a forensic psychiatrist, just did not understand
4 about what is public and what is not public when it
5 comes to legal documentations. 10:47:51

6 I think I subsequently learned that -- that
7 lots of people read my reports who have nothing to do
8 with the matter at hand because lawyers are looking for
9 experts and precedents and so -- and arguments and so
10 forth. 10:48:12

11 So in a -- in a personal sense, I have some
12 kind of objection to that. It doesn't feel fair to me,
13 but it's also a reflection of my naivety about this --
14 my past naivety about this matter -- about legal
15 matters. 10:48:28

16 BY MS. HARTNETT:

17 Q Thank you. I have added a different --
18 another exhibit that I would like to introduce into the
19 folder, if you could refresh.

20 MR. BROOKS: 87? 10:48:44

21 MS. HARTNETT: That's correct.

22 MR. BROOKS: Shall I open that now?

23 MS. HARTNETT: Yes, if you would.

24 (Exhibit 87 was marked for identification
25 by the court reporter and is attached hereto.) 10:48:48

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1 BY MS. HARTNETT:

2 Q And, Dr. Levine, I've marked as Exhibit 87
3 your expert report and declaration in this matter dated
4 February 23rd, '22.

5 Could you please just take a moment to look 10:49:04
6 through the document.

7 MR. BROOKS: Well, Counsel, the document, I
8 think we'll all agree, is perhaps, what, 70-some pages
9 long, plus bibliography.

10 Would you -- what do you mean by asking the 10:49:17
11 witness to look through the document?

12 MS. HARTNETT: I was just giving him the
13 courtesy of making sure he agrees it's his expert
14 report.

15 THE WITNESS: Well, my -- my signature is on 10:49:28
16 the first page.

17 BY MS. HARTNETT:

18 Q Excellent. So what is this document,
19 Dr. Levine?

20 A Well, I believe it is the report that I 10:49:34
21 submitted at the end of February about -- in this
22 matter.

23 Q Okay. And did you prepare this report?

24 A Yes.

25 Q And do you notice that this one has the 10:49:50

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1 caption for this case on it, on the first page;
2 correct?

3 A It does, yeah.

4 Q How much time did you spend preparing this
5 report? 10:50:06

6 A I could -- I would say approximately 20 to
7 25 hours. I would say closer to 25 hours.

8 Q And were you -- as a basis for this report,
9 did you use a kind of prior report that you had
10 submitted in a different case? 10:50:35

11 A Yes.

12 Q What was the basis -- like, the prior report
13 that you used as a basis for this report?

14 A Well, as I've told you already, I have
15 provided reports about the nature of what is known and 10:50:51
16 what is not known in a scientific sense about this
17 whole matter and -- so that's just part of my thinking.
18 And one report is a sort of modern refinement of a
19 previous report that -- that is selected, added to or
20 deleted from based upon the relevance to the case in 10:51:22
21 point.

22 So every -- every submission that I have made,
23 in a sense, has contributed to the -- to this current
24 report with the understanding that things have been
25 added and things have been deleted every time that I -- 10:51:44

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1 I submit a report for a case.

2 I hope that's an answer to your question.

3 Q Thank you, yes. I guess what I'm trying to
4 get at is was there a particular past report that you
5 used as a template to work from as you made your 10:52:03
6 refinements and edits for this report?

7 A No. That's -- that's -- I think the answer is
8 my -- my -- my knowledge -- my -- I think the answer is
9 to all, all my reports. I guess the answer to your
10 question is no, there's not a particular one, but there 10:52:38
11 are a series of reports, and I sometimes will select
12 from various reports.

13 Well, for example, this -- the -- the simplest
14 thing is if -- in the beginning of the report, when I
15 provide my credentials, for much of that, there is a 10:52:58
16 cut and paste phenomenon and -- and it doesn't much
17 matter which report I cut and paste from, but I only
18 added to it or subtract to it depending on, I think,
19 the relevance.

20 So, for example, if you looked at my report on 10:53:20
21 the North Carolina matter, probably there's much
22 similarity in the beginning of the report.

23 Q Thank you. So this document indicates that
24 the -- at least by my reading of it -- the only
25 documents specific to this case, B.P.J., that you 10:53:39

1 reviewed in preparing your report were the Adkins
2 declaration and the Safer declaration; is that correct?

3 A I think so.

4 Q Are you familiar with the concept of a
5 reasonable degree of scientific certainty? 10:53:57

6 A I hear it as "medical certainty." Is this a
7 reasonable degree -- can you offer this with a
8 reasonable degree of medical certainty, Doctor? And
9 when I've asked what that -- what that meant, I've been
10 told 51 percent certainty. 10:54:17

11 Q Okay. What is your understanding of -- so
12 your understanding of a reasonable degree of medical
13 certainty means 51 percent certainty?

14 A No. I think that's my understanding of the
15 legal definition of medical certainty. My clinical 10:54:35
16 idea and my scientific idea would be very different.

17 I -- I often smile when I think that -- if I'm
18 correct -- that in the legal world, medical certainty
19 refers to 51 percent.

20 Q And what is, in contrast, your clinical 10:54:58
21 standard that you were referring to?

22 A Repeat that, please. What is what?

23 Q The -- I think you were contrasting it with a
24 clinical standard; is that correct?

25 A Right. Oh, clinical or scientific. 10:55:14

1 You know, in -- in science, we have -- in
2 clinician, we have the idea of what is the risk of a
3 false positive and what is the risk of a false
4 negative, and it's a complicated statistical balance
5 between the ability to get it right or to get it wrong. 10:55:31

6 And I am -- I am one who is very humbly
7 impressed by the inability to be certain about things,
8 and I distrust certainty because facts change in
9 medicine.

10 And -- and if I could just tell you a -- an 10:55:52
11 experience that I've had. As a young person, I was
12 interested in becoming a physician, and I went to a
13 premed program at the University of Pittsburgh, and
14 somebody in that program held up Harrison's textbook of
15 medicine, which requires considerable arm strength to 10:56:13
16 lift over your head because it's probably, you know,
17 900 to a thousand pages. And he said, This is what you
18 have to learn when you're in medical school, by the
19 time you graduate medical school. I want to tell you,
20 ladies and gentlemen, that 90 percent of the things in 10:56:33
21 this book are probably not true. They probably will
22 not prove to be true in time. The trouble is I and
23 other people in medicine can't tell which of the
24 10 percent -- which of the facts are correct and which
25 of the facts are not. This is the nature of medical 10:56:48

1 science as it -- and clinical science as it moves
2 forward. We have, at any given time, a set of facts, a
3 set of principles and -- and controversy occurs, people
4 disagree and studies are done, and the facts disappear
5 and new facts take their place. 10:57:12

6 That was my introduction to medical science.

7 And as I've spent most of my -- the majority
8 of my years in this field, I still believe that that
9 little example remains to be -- remains salient and
10 something that all of us need to remember. 10:57:32

11 And so I say to you, 51 percent medical
12 certainty is a joke to me. It -- it -- I always smile.

13 Q Thank you. That -- that's helpful.

14 If we could just go through your CV attached
15 to your report, we can -- I have a few questions on 10:57:52
16 that, and then I'll turn to your report.

17 You'll have to page down a bit. It starts
18 repaginating about page -- after page 81.

19 MR. BROOKS: We are at the beginning of where
20 it says "Brief Introduction," "Curriculum Vita." 10:58:20

21 MS. HARTNETT: Okay. Thank you.

22 BY MS. HARTNETT:

23 Q Dr. Levine, is this your CV?

24 A It is.

25 Q Are you aware of anything, sitting here today, 10:58:27

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1 that needs to be updated or corrected?

2 A Probably if you scroll to the end of the
3 articles, article 151 -- publication 151.

4 MR. BROOKS: We're scrolling. We're
5 scrolling. 10:58:51

6 MS. HARTNETT: I think it might be 147.

7 MR. BROOKS: There's a lot. Pardon me. 86.
8 Here we are at -- just before --

9 THE WITNESS: Oh --

10 MR. BROOKS: -- where it says "Book Chapters." 10:59:00

11 THE WITNESS: I'm sorry, 147. 147 is -- I
12 can -- you know, today -- if I were to give you my CV
13 today, I would give you the exact citation of that
14 article.

15 And if we scroll down to the end of the CV, I 10:59:34
16 will show you something else.

17 MR. BROOKS: I'm not sure there's a further
18 question --

19 THE WITNESS: Oh.

20 MR. BROOKS: -- pending. 10:59:46

21 Or is there a question pending?

22 MS. HARTNETT: Well, yeah, I can -- I can ask
23 one.

24 BY MS. HARTNETT:

25 Q So I take it that 147 has now been published. 10:59:51

1 Is that the difference?

2 A Yes.

3 Q Did you -- is there a -- a more updated
4 version of your CV that goes up to 151?

5 A I think last week, I -- I rearranged the 11:00:03
6 numbers and somehow -- I may be -- I may -- I may not
7 be accurate at 151.

8 Q Okay. And then 146 there is what you were
9 talking about earlier, the November piece about the
10 reflections on a clinician's role? 11:00:26

11 A Yes.

12 Q Thank you. And is there anything further on
13 here you'd like to draw my attention to is in need of
14 updating?

15 A I don't know if -- if this -- this thing has 11:00:40
16 a -- this CV has a -- my -- a podcast I participated
17 in. I never -- unlike many of my colleagues, I never
18 put in my CV the talks I give and the -- you know, and
19 now there's this whole thing about podcasts. I -- I
20 gave a -- I didn't -- I was invited to give a podcast 11:01:04
21 recently and -- so I think it's on my CV, but I'm not
22 sure.

23 Q That was in January of this year?

24 A Was it in January? It was -- it was within
25 several months ago, yeah. 11:01:20

1 Q Have you given any podcasts other than the one
2 you gave in January of this year?

3 A The -- the answer to that question is I don't
4 know. I mean, sometimes people come and talk to me
5 and -- and film me on camera and I never know what 11:01:46
6 happens to -- what hap- -- what -- what -- that
7 happens. I never know what happens to it.

8 Q Are you aware of any other -- sorry.

9 A The answer to your question is I'm not aware
10 that I have been in any other podcast, but, you know, 11:02:04
11 you may dig up some other conversation that is -- that
12 I've had somewhere along the line.

13 Q Thank you. If we could just turn back to the
14 first page of your CV, I would appreciate it.

15 Let me know when you're there. 11:02:26

16 MR. BROOKS: Yeah. We're there.

17 MS. HARTNETT: Okay.

18 BY MS. HARTNETT:

19 Q So on page 1, it notes that you are -- board
20 certified in -- in June of 1976; correct? 11:02:39

21 A Yes.

22 Q In neurology and psychiatry; is that correct?

23 A That's the name of the board that
24 psychiatrists get certified in. It's a little bit of a
25 joke that I'm -- that any psychiatrist is certified as 11:02:59

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1 a neurologist.

2 Q Have you been recertified with that
3 certification?

4 A No. I don't need to be. I'm grandfathered
5 in, as they say. 11:03:13

6 Q Thank you. Do you have any other board
7 certifications?

8 A No.

9 Q So you are not board certified in child and
10 adolescent psychiatry; correct? 11:03:27

11 A No, I'm not board certified.

12 Q Do you have any specialized training in child
13 development?

14 A Yes.

15 Q Can you describe that? 11:03:36

16 A I'm a psychiatrist. All psychiatrists are
17 trained in child development. I, in particular, have
18 been interested in the whole process of adult -- of --
19 of development throughout the life cycle and have -- I
20 think I quoted in my expert opinion report that 11:03:57
21 Tom Insel, who was the head of the NIH, NIMH, said that
22 75 percent of adult psychopathology, that is, suffering
23 as a result of mental disorders, have their origins in
24 childhood.

25 So it's hard for me to conceive that any -- 11:04:16

1 any -- any psychiatrist is not knowledgeable about the
2 processes of growing from birth to death. And I, in
3 particular, am interested in that process. I often say
4 to my -- to other people that I -- development is my
5 field. In fact, when -- when people talk about 11:04:40
6 psychoanalysis and psychodynamic psychiatry, I like to
7 rephrase those terms as developmental psychology.

8 Q Thank you. I just -- my -- my question,
9 though, was whether you have any specialized training
10 in child development. 11:04:57

11 Do you have any specialized training?

12 A Well, of course, I rotated through child
13 psychology when I was a resident. For the purpose- --

14 Q Anything else?

15 A For the purposes of questioning my expertise, 11:05:12
16 I have no specialized credentialed, certificated
17 training in child psychi- -- in -- in child
18 development.

19 However, what I'm saying to you is that my
20 understanding of being a psychiatrist and listening to 11:05:27
21 people's stories about their development all day long,
22 I don't need a special certificate to testify that I am
23 trained in -- in -- in child, adolescent, young adult,
24 middle-aged and older-aged development.

25 Q And would the answer be the same if I asked 11:05:49

1 you whether you had any specialized training in -- in
2 children or adolescents with gender dysphoria?

3 A Specialized training? I was in on the ground
4 floor of these things when there was no specialized
5 training. I was part of the -- I was part of the 11:06:12
6 process that was trying to figure out what this all was
7 about, you see. And --

8 THE WITNESS: Sorry.

9 -- I very much object to that term
10 "specialized training" because I have an understanding 11:06:30
11 of what that really -- the connotation of that term is,
12 and I don't accept that -- the legitimacy of
13 specialized training.

14 I feel what you may mean is indoctrination
15 training. I'm -- I like to distinguish between 11:06:50
16 indoctrination and education.

17 BY MS. HARTNETT:

18 Q Are you an endocrinologist?

19 Are you an endocrinologist?

20 A No. 11:07:17

21 Q And you would not hold yourself out as an
22 expert in endocrinology; correct?

23 A I'm not an expert in endocrinology.

24 Q And are you an expert in sports medicine?

25 A No, I'm not an expert in sports medicine. 11:07:33

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1 Q Are you an expert in athletic performances?

2 A I've already testified to that. The answer is
3 no.

4 Q Yeah, I'm asking because I think your attorney
5 at some point indicated that might be part of your 11:07:44
6 privileged conversation. That's why I'm asking you
7 again.

8 Do you have any -- have you ever had any
9 complaints made against you related to your medical
10 practice? 11:07:56

11 A Yes.

12 Q Could you tell me about those?

13 A Yes. We had a trans adult who wanted
14 hormones, and I was supervising a psychology intern,
15 and the -- we decided the person was mentally unstable 11:08:17
16 and was not in a position to be given hormones just
17 yet, and the patient threatened to murder the
18 psychology intern who told her that -- who told the
19 patient that answer.

20 And I -- when she told me that, I went in and 11:08:36
21 I saw the patient, and I told the -- and I discharged
22 the patient. And I said that patients have obligations
23 and doctors have obligations and you have justified the
24 rule, you have crossed over the line, and I cannot
25 allow you to continue to get care here. 11:08:59

1 The patient then left and then reported me to
2 the State Medical Board, and the State Medical Board
3 investigated and -- and found -- and found that I was
4 perfectly justified in what I did.

5 That is the only awareness that I have of -- 11:09:21
6 of complaints to the State Medical Board about my work.

7 Q Thank you. Just back to the point, we -- we
8 were discussing the notion of specialized training a
9 minute ago.

10 Do you recall that? 11:09:40

11 A I recall.

12 Q So do you -- do you accept the legitimacy of
13 the notion of specialized training in child and
14 adolescent psychiatry?

15 A For people who are interested in having a more 11:09:58
16 extensive experience and plan to spend their lives with
17 young -- young people only or primarily, I think it's a
18 fine thing to -- to -- to -- it's just one of the many
19 houses in the big -- in the mansion of medicine and one
20 of the -- one of the subspecialties in psychiatry. I 11:10:20
21 have no objection to people becoming child and
22 adolescent psychiatrists.

23 Q And just to be clear, that's not a specialty
24 of yours; correct?

25 MR. BROOKS: Objection. 11:10:40

1 THE WITNESS: It's not formally. I -- I don't
2 define myself as a board-certified child and adolescent
3 psychiatrist, but I do define myself as a psychiatrist.

4 And as -- as I've already stated, I believe
5 that psychiatrists, over the -- during the course of 11:10:51
6 their training and -- that is, their initial education
7 and their subsequent life education, practicing
8 psychiatry, comes to understand or should come to
9 understand the influence of childhood positive and
10 negative experiences on their subsequent mental life 11:11:09
11 and behavioral life.

12 BY MS. HARTNETT:

13 Q In your mind, are the concepts of having an
14 understanding of child psychology and actually working
15 with child patients distinct notions? 11:11:25

16 A Well, I think they're -- they are to be
17 separated. One's -- one's theoretical understanding of
18 the processes of development, the stages of development
19 and understanding childhood adversities that -- that we
20 hear about all the time from adolescents and from 11:11:49
21 adults, that's different than actually, you know,
22 seeing five-year-old children or six-year-old children.

23 So I make a distinction between that, sure.

24 Q And how much of your practice throughout your
25 career has involved actually seeing children or 11:12:12

1 adolescent patients?

2 A Well, I -- I spend a lot of time with
3 adolescent patients, and I spend much less time with --
4 with children per se. I spend an enormous amount of
5 time talking about children to their parents. I mean, 11:12:30
6 conversations about childhood are about the -- my -- my
7 older patients, about their childhood, and the parents
8 that I see about their children's processes, that's
9 a -- I would say a daily occurrence in my practice.

10 Q How many child patients have you had in your 11:12:56
11 career?

12 MR. BROOKS: Objection; vague.

13 THE WITNESS: I -- I would have a very hard
14 time answering that question. I've had -- you know,
15 when -- when parents talk to me about their children, 11:13:26
16 for insurance purposes, the patient is the mother or
17 father or both; right? But the subject of our
18 conversation is the child.

19 So I don't know -- you see, and one of the
20 therapeutic activities that I do, I call "parent 11:13:47
21 guidance." And so parent guidance involves the focus
22 on the child's environment and how to improve the
23 child's anxiety problems or whatever, you see.

24 So I don't know if I -- if that constitutes
25 how many children. Can I answer that question in terms 11:14:08

1 of parent guidance?

2 I have a pediatrician, for example, as an
3 adult patient now, and he and I have spent a lot of
4 time talking about his daughter and -- and some of the
5 things I've said to him have really helped his daughter 11:14:25
6 overcome a problem. But he's my patient, you see.

7 I don't -- so I can't answer your question
8 with numerical terms and --

9 BY MS. HARTNETT:

10 Q Children can be patients; correct? 11:14:39

11 A Children can be patients, certainly.

12 Q And so I'm just asking you how many actual
13 children patients you've had over your career, if you
14 could estimate that.

15 MR. BROOKS: Objection; vague as to the term 11:14:51
16 "children."

17 THE WITNESS: Can you clarify whether -- what
18 a child is versus what a teenager is?

19 BY MS. HARTNETT:

20 Q Yeah, I'll ask you for two categories. 11:15:04

21 I'll ask you for prepubertal children.

22 How many prepubertal children have you had as
23 a patient in your career, approximately?

24 A And if I saw that prepubertal child one time,
25 would that -- would that constitute a patient? 11:15:20

1 Q Why don't you give me your estimate of how
2 many prepubertal children you've ever seen as patients,
3 and then we can ask more questions.

4 A I would say a handful. Six.

5 Q And how many of those -- of those 11:15:35
6 approximately six did you see more than one time?

7 A I can't recall one.

8 Q And then I'll ask the same question about
9 adolescents, which I'll mean minors from puberty
10 through being a minor. 11:16:00

11 How many adolescent patients have you had in
12 your career, approximately?

13 A 50.

14 Q And how many of those have you seen more than
15 once? 11:16:14

16 A Most.

17 Q And were most of those, of the adolescent
18 patients you've seen, late adolescence?

19 A No.

20 Q Turning back to your CV, you list yourself -- 11:16:27
21 you're listed as a clinical professor at Case Western
22 Reserve University School of Medicine; correct?

23 A Yes.

24 Q Do you work at Case Western Reserve University
25 School of Medicine full-time? 11:16:51

1 A No. No.

2 Q When did you stop working full-time?

3 A In 19- -- November 1992.

4 Q Are you currently teaching any classes at

5 Case Western?

11:17:09

6 A I've never taught classes per se. That's not

7 how my teaching has ever been. If you think about a

8 college course, I have never -- I don't teach college

9 courses or graduate school courses. I provide seminars

10 sometimes. I've written articles about the sex

11:17:32

11 education of doctors and -- so over the years, I've

12 taught a number of seminars to our residents in

13 psychiatry. I teach -- I give workshops.

14 I recently, for example, gave a

15 four-hour work- -- a four-hour workshop at the Harvard

11:17:59

16 student health service for their mental health

17 professionals where I presented, you know, ideas to

18 them, and we had discussions.

19 So I teach -- I teach sometimes by giving

20 grand rounds. I -- there -- there is a named

11:18:20

21 lectureship in my honor at Case Western Reserve, and

22 once a year, I invite someone to give a talk from

23 another university about some sexual topic.

24 So I have residents who come to spend --

25 for -- I can't -- for probably -- probably -- since

11:18:44

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1 1992, 1993, I've always had a resident with me who
2 comes and sees my patients with me, and they usually
3 spend six months with me, sitting in and seeing my
4 patients together.

5 So my teaching is not in the classic sense 11:19:03
6 that -- that the average layperson would think of
7 teaching classes. It's -- it's much more -- you know,
8 coming in and seeing how an older doctor does work,
9 has, quote, therapy.

10 I also, since 1977, have led two clinical case 11:19:26
11 conferences a week, and residents and medical students,
12 depending on the year, medical students, residents and
13 members of the community come in to those conferences
14 and we discuss cases.

15 So I have multiple avenues, multiple ways of 11:19:45
16 being a teacher, but none of them are through
17 coursework per se.

18 Q Thank you.

19 A I forgot to tell you. I also sometimes am
20 invited to give continuing education lectures. And, 11:20:02
21 for example, at the -- I've given courses, for seven
22 years in a row, at the American Psychiatric Association
23 on sex and love, mostly love I use as -- as the title,
24 and we talk about sexual problems and the barriers to
25 loving. 11:20:25

1 And this year's APA meeting, I -- I am
2 presenting a symposium with three colleagues on whether
3 or not this is time to reexamine the best practices for
4 transgender youth.

5 So all those things are -- in my review, are 11:20:39
6 -- are my teaching.

7 Q I was going to ask you about the May
8 presentation.

9 Who are your copresenters for that?

10 A Sasha Ayad, Lisa Marciano and Ken Zucker. 11:20:55

11 Q Thank you. When is that expected to be
12 presented?

13 A May 24th.

14 Q And do you know if there are other panels or
15 presentations regarding the care of transgender 11:21:15
16 patients at that conference?

17 A There probably are, but I'm -- I haven't seen
18 the entire program. But -- but there are usually --
19 there usually are one or two presentations.

20 Q And you said it was Sasha Ayad, Ken Zucker. 11:21:29
21 And who was the third person?

22 A Lisa Marciano.

23 Q Right. So I just had one -- a couple of
24 follow-up questions about the discussion we were having
25 about seeing prepubertal and adolescent patients. 11:21:46

1 When is the last time you saw a prepubertal
2 child patient?

3 A Physically saw?

4 Q Or -- or virtually. I mean, as your patient.

5 A Maybe two years ago. 11:22:20

6 Q And how about an adolescent, meaning puberty
7 while -- through being a minor?

8 A Three weeks ago.

9 Q And what was the age of that patient?

10 A 17. 11:22:44

11 Q Okay. Let's just turn to page 2 of your CV.

12 I had a couple of questions there.

13 MR. BROOKS: Just checking --

14 MS. HARTNETT: I'm just --

15 MR. BROOKS: Since it's been an hour, I was 11:23:00

16 just checking. The witness says he's fine and doesn't

17 need a break yet.

18 MS. HARTNETT: Okay. Please let me know.

19 This is --

20 MR. BROOKS: We're on -- 11:23:08

21 MS. HARTNETT: So --

22 MR. BROOKS: -- the next page. If you'll
23 direct -- I can't fit the whole page on the screen at a
24 time, so you have to direct me to portions of it.

25 MS. HARTNETT: Okay. It's -- I'm looking 11:23:16

1 at -- under "Professional Societies."

2 MR. BROOKS: All right. I have it up.

3 BY MS. HARTNETT:

4 Q Dr. Levine, on page 2 of your CV, you list
5 professional societies; correct? 11:23:28

6 A Yes.

7 Q Is the Cochrane Collaborative a professional
8 society?

9 A Is the what?

10 Q The Cochrane Collaborative. 11:23:40

11 A I don't know the answer to that question. The
12 Cochrane Library, you're talking about?

13 Q The Cochrane Collaborative.

14 A Cochrane Collaborative.

15 Well, I -- the word "Cochrane" is -- is what 11:23:54
16 comes to mind. It -- the second word changes from
17 whomever is using it.

18 I don't think it's a society. It's an
19 organization that does objective appraisal of -- of
20 scientific questions or controversies. And I -- I 11:24:13
21 don't -- I never thought about that as a society;
22 therefore, it's not listed there.

23 Q Okay. And I apologize. I believe I misstated
24 the name of it. It's on paragraph 4 of your report,
25 which you can look back to, but it then will require 11:24:31

1 flipping forward again.

2 You discussed being an invited member of the
3 Cochrane Collaboration subcommittee, and so I was just
4 trying to understand whether the Cochrane Collaboration
5 is a professional society. 11:24:42

6 A Well, it's an organization, and it's an
7 organization devoted to the objective appraisal of
8 issues that are controversial in medicine, throughout
9 medicine, every branch of medicine, every specialty of
10 medicine. It's an older institution, and it's among 11:25:02
11 the most highly respected institutions about objective
12 scientific appraisal of clinical work, and I -- I am on
13 the -- one of their committee- -- I'm on two of their
14 committees, actually.

15 Q Which committees are you on? 11:25:20

16 A It's the evaluation of puberty blockers and
17 the evaluation of cross-sex hormones for transgender
18 teens.

19 Q Do you know how many committees the
20 Cochrane Collaboration has? 11:25:35

21 A No. I think it's many decades old, and it --
22 that's -- but the answer to your question is I don't
23 know.

24 Q Are you a member of the Cochrane
25 Collaboration? 11:25:53

1 A I'm a member of those subcommittees.

2 Q And can you describe your work on those
3 subcommittees? What does that entail?

4 A I'm hesitating to answer that question because
5 you're going to ask a follow-up question, and it is my 11:26:12
6 understanding that until the publication of our work is
7 finished -- is published, our work is published, that
8 we are not to discuss the processes and the content
9 of -- of that.

10 So I -- I feel constrained to, you know, ask 11:26:35
11 you not to ask me more questions about that.

12 MR. BROOKS: Well, I -- I'm -- I'm not going
13 to instruct the witness either way. I will advise the
14 witness that we can, I'm sure with counsel's agreement,
15 designate a portion of the transcript as confidential 11:26:50
16 and kind of proceed question by question as you are
17 comfort- -- as you are -- as you feel able, given --
18 I -- I don't know the nature of your commitments to the
19 organization.

20 But we can designate a portion of the 11:27:04
21 transcript as confidential, which will make it
22 available to attorneys representing parties in this
23 case but would prevent it from being published
24 generally.

25 So I -- I -- I offer that. I don't -- I don't 11:27:18

1 represent Dr. Levine, and I don't know that in
2 connection with that -- that professional activity, and
3 I don't know the nature of the obligations, but I'd
4 just advise the client of that pos- -- of that --
5 Dr. Levine of that possibility. 11:27:37

6 If you want --

7 BY MS. HARTNETT:

8 Q Does your work with the Cochrane -- does your
9 work with the Cochrane Collabor- -- Collaboration
10 affect your -- sorry. 11:27:46

11 Has your work on the Cochrane Collaboration
12 informed your opinions in this matter?

13 A My work with the Cochrane group, in reading
14 about the evidence on those two -- on that subject of
15 puberty blockers adds to my -- I should say there's -- 11:28:17
16 I'm hesitating because I really don't know whether I
17 should be saying anything about this, even answering
18 your reasonable question.

19 Q I appreciate that, but --

20 A Pardon me? 11:28:51

21 Q -- we do need to know this for your views, and
22 so I would ask if we -- could you -- could -- are you
23 able to answer my questions and we can designate this
24 portion of the transcript as confidential, meaning it
25 would not be publicly disclosed? 11:29:03

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1 A There's nothing that I have -- there's nothing
2 that I have seen in my work with Cochrane that has led
3 me to modify what is in that report.

4	Q Can you please generally describe what the	
5	nature of your work is with Cochrane?	11:29:20

6 A It is to read and respond to summaries of the
7 data, various studies. It has been to help
8 conceptualize what the issue is and what measurements
9 we need -- are needed in order to answer the question
10 in the future about a more -- to provide data in the 11:29:43
11 future if -- based on studies. It's been about trying
12 to limit the number of issues that need to be measured
13 to -- in outcome studies in order to be practical
14 versus comprehensive.

15 So my work has been to participate with other 11:30:06
16 people in Zoom discussions after we read documents and
17 to given our opinions about draft documents.

18 And you may or may not know how Cochrane
19 works, but it's a series of -- like, our subcommittee
20 goes through a number of other committees above them to 11:30:31
21 be consistent and -- with the traditions of Cochrane.

22 And so I'm not, you know, privy to the
23 committees above the subcommittee. I just sometimes
24 hear about, learn about, their -- their responses to
25 druff -- draft reports. 11:30:54

1 So I think that's my answer to your question.

2 Q Okay. Are you a member of the Society for the
3 Scientific Study of Sexuality?

4 A The -- oh, no longer.

5 Q What is the Society for Scientific Study of 11:31:19
6 Sexuality?

7 A It's a bunch of clinicians who are
8 interested in -- it's a bunch of clinicians who are
9 interested in providing services for people's sexual
10 problems. 11:31:36

11 Q And you ended your membership there in 1999?

12 A Yes, apparently so.

13 Q Why?

14 A Apparently so. I -- I -- if I hadn't looked
15 at my CV, I wouldn't have been able to answer your 11:31:56
16 question.

17 Q Okay. I'm sorry, I was asking why you stopped
18 being a member in 1999.

19 A Oh. Because I felt that the majority of the
20 membership thought very differently than me. They 11:32:15
21 weren't -- they were mostly Master's prepared people.
22 They included people who were sexual surrogates. It
23 was a potpourri of people interested in human sexuality
24 that did not have my academic interest in sexuality.

25 I was interested, I guess -- back then, in the 11:32:39

1 '90s, there was the -- there was the Society for Sex
2 Therapy and Research, and there was this society.
3 Quadruple S, it's called. And this was -- and there
4 was another society called AASEC- -- AASECT. And
5 the -- the range of professional degrees, the people 11:32:59
6 who had -- the people in those societies had different
7 ranges of professional degrees, and they had different
8 interest in -- sort of an understanding of sexual
9 disorders and in research, and I thought that the
10 society for scientific study of sex really -- I thought 11:33:23
11 that the activities of the organization did not rise to
12 the level of -- of the title of their organization,
13 that it really wasn't scientific.

14 And, you know, it is amazing to me what --
15 what people call -- who wrap themselves in the mantle 11:33:49
16 of science that really don't have a concept of science.

17 So I -- you know, when I was younger, I wanted
18 to be part of the scene and -- and when I got into part
19 of the scene, I didn't want to be part of the scene.

20 Q Are you aware of the Society for Evidence 11:34:06
21 Based Gender Medicine?

22 A Yes.

23 Q And does that go by an acronym?

24 A Is what?

25 Q Does that go by an acronym? 11:34:15

1 A Yes. SEGM.

2 Q SEGM. Are you a member of SEGM?

3 A I contributed -- when I -- when I learned
4 about SEGM probably a year and a half ago, two years
5 ago, I -- I felt that I -- I wanted to support that 11:34:35
6 because they were interested in evidence, in scientific
7 evidence, so I sent them a check for \$200.

8 So I don't know if I'm a supporter of it or --
9 but I -- they consider me to be an integral and
10 important member of their society. So I guess, based 11:35:02
11 on the fact that I gave them a one-time check of \$200
12 and they hired me to write a -- to -- to develop a
13 paper and they put me on a subcommittee to talk about
14 psychotherapy of adolescents, so I guess I am a member
15 of SEGM. 11:35:21

16 I think I'm a valued member of SEGM.

17 Q Understood. Sorry, you said you were on the
18 psychotherapy -- child psychotherapy subcommittee?

19 A I think we should call it an adolescent -- it
20 doesn't exist anymore. We met -- we met every two 11:35:45
21 weeks for almost a year, but I certainly was an active
22 participant of that.

23 Q And what -- what was the work of that
24 subcommittee?

25 A It was talking about what -- it was talking 11:36:01

1 about how to develop case histories that would teach
2 mental health professionals, in general, on how to
3 approach a -- an -- an approach to transgender children
4 and adolescents.

5 As you probably know, there has been, in the 11:36:33
6 last ten years, a dramatic increase in the number of
7 teenage children who are declaring themselves to be
8 trans people. And so the number of, quote, experts --
9 the epidemiology is such that there is enormous
10 pressure on a -- on the few people who say they're 11:36:53
11 interested in gender, taking care of gender cases.

12 So SEGM was trying to develop concepts that
13 could be taught to people in the community who are not
14 experts. We are trying to interest them in providing
15 psychiatric services, psychological services to 11:37:14
16 families and to the -- the patients themselves.

17 And so we were talking about how to -- how to
18 achieve that, whether we should publish -- whether we
19 should give a conference, whether we should -- they
20 just -- they talked about various ways of -- of 11:37:32
21 informing -- of getting more mental health
22 professionals to -- to stop ignoring this problem and
23 to be interested in -- in how to help these kids and
24 their families.

25 Q Okay. Thank you. 11:37:56

1 So you said that that subcommittee is no
2 longer meeting?

3 A That particular committee is no longer
4 meeting, as far as I know. But that -- but SEGM
5 sponsors many things that I'm totally unaware of. 11:38:07

6 Q Was there a work product that came out of that
7 committee?

8 A Well, in some sense, my paper, my most recent
9 paper, didn't come out of that committee, but it came
10 out of the deliberations of that committee because one 11:38:24
11 of the strategies that SEGM had is that they wanted
12 to -- they wanted to put things in the literature
13 that -- that were based on evidence rather than based
14 on precedent.

15 And so I think that led to the publication of 11:38:45
16 my -- of 147.

17 Q What do you mean, precedent?

18 A Well, as you may or may not know, there's a
19 60-year history of -- of trying to find treatments for
20 transgendered individuals and -- so there has been a 11:39:08
21 precedent of treatment over the years that has preceded
22 the -- the -- the scientific demonstration of the
23 efficacy and the long-term outcomes of that treatment.

24 So I would say that precedent is a -- is a
25 very important influence in how transgender people are 11:39:30

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1 being treated today and -- so that's how I use the term
2 "precedent." That is, we have patterns or fashions of
3 treatment that have gone in -- far in advance of the
4 scientific demonstration of the efficacy and were
5 the -- and the long-term outcomes of those treatments. 11:39:55

6 So that's the term precedent, as I -- as -- as
7 how I use it or how I think about it.

8 Q And was your -- I think your testimony was
9 that you were in the kind of ground floor of starting
10 that precedent; is that correct? 11:40:10

11 A I -- well, if -- well, the ground floor really
12 began in the '70s, and I was --

13 Q I'm sorry, did your counsel say something?

14 MR. BROOKS: No. I looked at him. He looked
15 at me. I didn't say anything. 11:40:28

16 THE WITNESS: Yeah.

17 MS. HARTNETT: Just for the record, the
18 counsel and the witness appeared to be exchanging some
19 sort of a glance, but please continue.

20 THE WITNESS: So the ground floor has to do 11:40:37
21 with the Harry Benjamin International Dysphoria
22 Association, which I think I joined in 1974 or
23 something like that, and I was in that program or in
24 that -- that associ- -- whatever you call that, a
25 society or something. I was in that professional 11:41:02

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1 organization for many, many years. And in 19- -- when
2 the fifth standard of care was being thought about, I
3 was named to be the chairman of the writing group that
4 made what was called the Fifth Edition.

5 So --

11:41:25

6 BY MS. HARTNETT:

7 Q So you were part of creating the precedent;
8 correct?

9 A Yes. The only objection I had, what is ground
10 floor. That's the only word I was responding to. I 11:41:34
11 didn't know what ground floor meant.

12 Q Fair enough. So back to SEGM. Were you part
13 of helping to develop treatment guidelines for the
14 treatment of gender dysphoria with SEGM?

15 A I don't know that SEGM has ever issued 11:41:52
16 treatment guidelines. In a sense, my latest
17 publication is -- is probably in that ballpark.

18 What we're trying to do is to -- I think what
19 we are trying to do is -- is create treatment
20 guidelines. 11:42:19

21 You know, Sweden, Finland, the UK and France
22 have all come out and said that -- let's slow this
23 down, let's be very careful. Even -- even in the
24 United States, there are people who used to be on
25 this -- sort of on a different -- they had a -- they 11:42:46

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1 had a different treatment guidelines.

2 There's been a wave of objectivity --

3 Q I'm sorry to interrupt. I'm sorry to
4 interrupt you, but I -- I really need to ask you to
5 answer my question. And I -- I think we're -- my -- my 11:42:57
6 question was just whether SEGM is developing treatment
7 guidelines.

8 A I think it's the aspiration of SEGM to develop
9 development treatment guidelines in keeping with what
10 is happening scientifically and -- in terms of 11:43:13
11 objective reviews.

12 So I'm not so sure that SEGM has published
13 treatment guidelines yet, but I do think they're
14 interested in -- in providing a different set of
15 guidelines that may have dominating the United States 11:43:34
16 and European countries in the past. And Australian and
17 compani- -- countries in the past --

18 Q Are you part -- are you part of any effort at
19 SEGM to develop treatment guidelines on a going-forward
20 basis? 11:43:55

21 A No, not directly, but I do --

22 Q Are you involved --

23 A I do believe that my recent article will be
24 read by people and considered by people who are
25 going -- if -- if they do develop treatment guidelines. 11:44:12

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1 Q Is -- is -- am I understanding correctly that
2 your article was an effort, in conjunction with SEGM,
3 to affect the practitioner community about how you view
4 treatment should be provided?

5 A To the extent that treatment should be 11:44:35
6 provided based upon a thorough informed consent
7 process, that my article describing informed consent
8 would be affirmative answer to your question that I --
9 I'm hoping that the influence of my article will
10 influence all treatment guidelines in the future, 11:45:01
11 regardless of who issues those guidelines.

12 MR. BROOKS: Counsel, when --

13 BY MS. HARTNETT:

14 Q Are you --

15 MR. BROOKS: When you come to a convenient 11:45:10
16 point, let's take one more break and have one more
17 stint before lunch. I don't mean to disrupt the line
18 of questioning, but when you come to a point, it would
19 be good.

20 MS. HARTNETT: I appreciate that. I have a 11:45:20
21 couple more questions on this, and then we can take a
22 break.

23 BY MS. HARTNETT:

24 Q Are you actively involved in any SEGM work
25 currently? 11:45:29

1 A No.

2 Q Do you know where SEGM receives its funding
3 from?

4 A I believe that -- that the hundred or so
5 people that are, quote, members contribute something, 11:45:55
6 but it's something as modest, perhaps, as I gave, \$200.
7 There must be a large donor or set of donors.

8 And the answer to your question is I don't
9 know the answer.

10 Q Is there someone at SEGM that you think would 11:46:15
11 know that answer?

12 A Yes.

13 Q Who is that?

14 MR. BROOKS: Objection.

15 THE WITNESS: There are several people. 11:46:26
16 May I answer that question?

17 MR. BROOKS: You may answer.

18 THE WITNESS: Stephen Beck, Dr. Stephen Beck,
19 and Ema Zane, E-M-A Z-A-N-E.

20 MR. BROOKS: And, Counsel, we will designate 11:46:47
21 the testimony about finances of SEGM as confidential.

22 MS. HARTNETT: We can -- oh, we can
23 provisionally do that. That's fine.

24 BY MS. HARTNETT:

25 Q You mentioned -- I have just one more. 11:46:59

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1 You -- you mentioned you were a valued member
2 of SEGM. Is that just your -- is there a special group
3 of people that are valued, or do you just kind of view
4 yourself as having a valued role in the organization?

5 A Well, I was asked to develop this paper or a 11:47:12
6 series of papers on informed consent, and to me, I
7 considered that a compliment, and it was based upon my
8 previous publications about this matter.

9 And in the concept -- and in the discussions
10 of the committee on psychotherapy, I just got the sense 11:47:41
11 that -- I offered an opinion and people really -- they
12 often said that was helpful or clarifying or, you know,
13 really good or "Can I use that term?" or whatever.

14 So whatever the subjective appraisal I was
15 making of my role, my status, among these very 11:48:04
16 respected people, I believed that I was a valued
17 member. You know, I could be --

18 Q Do you think you're the most --

19 A -- delusional about that.

20 Q Do you think you're the most -- are you the 11:48:18
21 most highly credentialed professional in SEGM?

22 A No.

23 Q Huh?

24 A No.

25 MS. HARTNETT: Okay. I think this is a good 11:48:34

1 time for a break.

2 MR. BROOKS: All right.

3 THE VIDEOGRAPHER: Off the record at

4 11:49 a.m.

5 (Recess.)

12:00:19

6 THE VIDEOGRAPHER: We are on the record at

7 12:01 p.m.

8 MS. HARTNETT: Thank you.

9 BY MS. HARTNETT:

10 Q Welcome back, Dr. Levine.

12:00:40

11 A Thank you.

12 Q I think I want to turn from your -- we were

13 talking through your CV a bit and now just go to your

14 report. So if you could -- I'm going to be asking a

15 question about paragraph 5, if you want to pull up that 12:00:53

16 page?

17 MR. BROOKS: We now have paragraph 5 on the

18 screen.

19 MS. HARTNETT: Great.

20 BY MS. HARTNETT:

12:01:14

21 Q So you, in the first sentence of paragraph 5,

22 say you first encountered a patient suffering with

23 what -- sorry -- "what we would now call gender

24 dysphoria in July 1973."

25 Do you see that?

12:01:30

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1 A Yes, I do.

2 Q Who was that patient?

3 MR. BROOKS: I will, of course, object to the

4 extent you're asking the doctor to disclose

5 confidential --

12:01:43

6 THE WITNESS: Actually --

7 MR. BROOKS: -- identifying information.

8 THE WITNESS: Actually, the patient and I

9 wrote a paper together and -- and so the patient has

10 used the name, so I feel like I can tell you the name. 12:01:52

11 BY MS. HARTNETT:

12 Q That's why I was asking.

13 A Yeah. So the name was Rutherford Shumaker.

14 Q And did you refer to the patient as

15 "Rutherford" or some other name? 12:02:07

16 A Well, the name of the -- the name of the

17 article was Increasingly Ruth: Towards an understanding

18 of sex reassignment surgery.

19 And so Rutherford, in, I think -- became Ruth.

20 So Ruth and I published that paper, and then I wrote a 12:02:32

21 follow-up to that paper after Ruth committed suicide in

22 her family's home. But that was 1983. I'd have to

23 check the CV.

24 So that was my -- the man coming to me as

25 Rutherford, who eventually became Ruth, came to me in 12:02:56

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1 July of 1973.

2 Q And do you recall how long after you first
3 encountered that patient you encountered your next
4 patient that was suffering from what we would now call
5 gender dysphoria? 12:03:11

6 A Oh, it probably -- it was probably a couple of
7 months.

8 The answer to your question, I don't
9 specifically recall, but --

10 Q Okay. 12:03:26

11 A -- I -- I -- there was enough pressure by
12 patient request for care that we started this -- this
13 clinic.

14 Q Understood. And you note here, on your
15 paragraph 5, you also founded the Case Western Reserve 12:03:37
16 University Gender Identity Clinic; correct?

17 A Correct.

18 Q And you note, later in that paragraph, that in
19 1993, the Gender Identity Clinic was renamed.

20 A In 1993, I left full-time employment at 12:03:52
21 Case Western Reserve, and I continued the program, but
22 we changed the name of the program, but our work
23 evaluating and providing services for trans individuals
24 continued.

25 Q And what did you change the name of the 12:04:15

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1 program to?

2 A Well, I think we just called it the Gender
3 Identity Clinic of Levine, Risen -- Althof, Levine and
4 Risen, which was the name of our clinical practice,
5 Althof, Levine and Risen. So it --

12:04:34

6 Q Okay.

7 A Gender Identity Clinic at ALR.

8 Q And when you -- when the university kind of
9 discontinued -- or you discontinued the affiliation
10 with the university in 1993, did you consider that to 12:04:50
11 be a dark day in the department, in the politics of the
12 department?

13 MR. BROOKS: Objection; compound question.

14 THE WITNESS: Number one, I did not
15 discontinue my affiliation. I changed my affiliation. 12:05:06
16 That is, I was salaried until 1993, and then I left the
17 university and personally, for a while, I did consider
18 it a -- a great disappointment that I left the
19 university.

20 BY MS. HARTNETT: 12:05:30

21 Q Did you consider it a dark day in the
22 department, in the politics of the department, at the
23 university?

24 A That per se wasn't the source of the darkness.

25 That day wasn't it. In my view, it's a very 12:05:43

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1 prejudicial view, the dark day came when a new chairman
2 was selected, who came aboard, who then basically ran
3 the department into a great debt, and then I and
4 several other program- -- my program and several other
5 programs needed to be cut from the department in order 12:06:07
6 to get the department back into solvency.

7 So the fact that one day I left was the
8 by-product of things that had happened over a
9 three-year period.

10 So the dark days began, I think, on day one 12:06:25
11 when the chairman came.

12 Q Thank you. Are you familiar with the
13 University Hospitals?

14 A The department of psychiatry was part of the
15 University Hospitals of Cleveland. 12:06:41

16 Q And you did your psychiatric residency at the
17 University Hospitals of Cleveland?

18 A Yes.

19 Q Do you have an affiliation there now?

20 A I do. I'm a clinical professor. 12:06:52

21 Q And how often do you -- if at all -- do you go
22 to the University Hospitals?

23 A Not very frequently. The -- the resident
24 comes to me, and I -- but I am probably going to be
25 teaching a seminar at University Hospitals in the next 12:07:13

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1 three months because I'm part of a committee to plan
2 the curriculum on sexuality and gender.

3 Speaking of education, the university --
4 other -- other institutions also asked me to teach
5 about this subject. And on August -- on April 7th, I'm 12:07:39
6 going to Akron to teach -- or virtually I'm going to
7 teach a three -- a two-and-a-half-hour seminar.

8 And I forgot to mention to you before, and I'd
9 like you to hear this, that when you were questioning
10 me about my credentials or not having a certificate 12:07:57
11 about -- in child psychiatry, you should know, I forgot
12 to tell you that Cleveland Clinic, department of child
13 psychiatry, and the University Hospitals, the
14 department of child psychiatry, sends residents to be
15 with me as part of their training in child development 12:08:18
16 and child clinical issues, child and adolescent
17 clinical issues.

18 So I think -- I just forgot to mention that.

19 Q Are you familiar with the University
20 Hospitals' LGBTQ and gender care program? 12:08:48

21 A I'm aware that it exists, yes.

22 Q Have you ever talked to any clinicians in that
23 practice?

24 A No one has ever talked to me in that practice.
25 The only time I have interaction with them is when -- 12:09:00

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1 if I present grand rounds, some of those people ask me
2 a question. But they've never consulted me whatsoever
3 in the formation of their clinic and in the ongoing
4 work of their clinic.

5 Although, Cleveland Clinic has a very similar 12:09:20
6 program, and they have called me up and -- for some
7 advice sometimes.

8 But my -- my, quote, own University Hospitals'
9 place I don't really think has any people from child
10 psychiatry in it, but I'm not sure because they have 12:09:38
11 kept me away.

12 Q What do you mean they have kept you away?

13 A Just what I explained. They have never
14 communicated with me. It is -- you know, other people
15 know me as being published in this area. You know, I 12:09:54
16 think I've written 20 articles on this -- you know, I
17 have 20 or so publications in this area. You would
18 think that they would invite me or consult with me or
19 ask me questions, but I think they recognized that they
20 are part of what is called affirmative care and what I 12:10:18
21 would say, rapidly affirmative care, and -- and they
22 sense that I'm not so interested in rapid, that -- that
23 I believe that -- that I have long believed that people
24 who have this kind of dilemma need some patient time in
25 talking about this matter. 12:10:45

1 And while I can't tell you how they feel about
2 me, I can only deduce that they're not interested in my
3 concepts because --

4 Q Have you --

5 A -- they must be different than their concepts. 12:10:57

6 Q Have you offered your -- your services to
7 them?

8 A No.

9 Q You said your understanding is that they
10 provide rapid affirmative care; is that correct? 12:11:10

11 A I presume so. I -- you know, I can't
12 understand why -- why the organizers and the leaders of
13 those -- that team are not interested in anything I
14 have to say because they've never asked me.

15 Q So just because someone hasn't asked you for 12:11:29
16 your view, do you assume that they're not interested in
17 what you have to say?

18 A This -- I wouldn't say as a general principle,
19 but I would say in this case, I have long assumed that,
20 correctly or incorrectly. 12:11:44

21 Q It sounds like you don't agree with rapid
22 affirmative care; is that fair?

23 A Yes. I don't believe that people, after
24 meeting someone for an hour, for example, ought to be
25 given a firm diagnosis and a prescription for hormones. 12:12:00

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1 Q Is that your definition of rapid affirmative
2 care?

3 A That would be one definition, yes.

4 Q Can you give me a more general definition of
5 what rapid affirmative care is? 12:12:17

6 A It would be -- it would be a commitment to be
7 affirmative in -- in being a cheerleader for social
8 transition or taking hormones or having one's breasts
9 removed after what I would consider to be an inadequate
10 evaluation. 12:12:34

11 So it begins with an adequate evaluation.
12 It -- it requires having an understanding of the
13 elements of informed consent. And in dealing with
14 minors, it has to do with working with not only with
15 the patient but with the parents. 12:12:51

16 So rapid affirmative care would be care that
17 does not meet my criteria for thorough evaluation,
18 including a developmental history, a process of
19 informed consent and involvement, over time, with the
20 parents so they consider the weighty -- the weighty 12:13:10
21 implications of -- of what affirmative care represents.

22 So anything short of deliberation in this and
23 careful consideration I would kind of dismiss as rapid.

24 Q If affirmative care is given with deliberation
25 and informed consideration, do you disagree with that? 12:13:33

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1 A No. No. I think parents -- parents have a
2 weighty decision to make, but they ought to be informed
3 about the state of science. The -- the health tour
4 benefits have to be understood in terms of the
5 scientific likelihood of achieving those benefits. And 12:13:51
6 they have to understand the short-term medical but more
7 important the long-term psychosocial risk of what
8 they're doing.

9 And if those competent parents, knowing the
10 child as they know them, decide, after they're 12:14:09
11 informed, they -- they have my blessing to socialize
12 their child in the opposite gender.

13 Whether I think in that particular case it's a
14 wise thing or not, it's not my decision to make. I
15 don't actually believe that people like me ought to be 12:14:29
16 recommending. I think we ought to be educating,
17 evaluating and informing and the parents and the child
18 make the decision with my supportive help, both on the
19 positive side and the negative side.

20 I am to be the trustee, informer of what 12:14:45
21 science knows, and I believe that clinicians who don't
22 know science, who actually think they can evaluate this
23 in a -- in -- in a -- in an hour, I just think that's
24 not good care.

25 Q Is your view that the clinicians at the 12:15:06

1 University Hospitals LGBTQ and gender program don't
2 know science?

3 A I don't know what they know. I don't know
4 what they know. I have no views about that because I
5 have no means of knowing, only that I get to see people 12:15:22
6 brought to me after they've gone to various affirmative
7 care programs and the parents are horrified at the
8 recommendations that are being made. So --

9 Q How many -- sorry. Go ahead.

10 A But in answer to your specific question, since 12:15:44
11 I don't even know the people there and I don't know
12 what they're doing, I'm not -- I would just -- I would
13 just -- I pose these standards, and I don't know
14 whether they meet them or not.

15 I have not been impressed in general that 12:16:04
16 affirmative care programs in various cities that I get
17 to hear about meet those criteria.

18 I'm just trying to help people, you know,
19 realize the importance of trans care and -- and trans
20 care, to me, includes careful evaluation and -- and 12:16:19
21 addressing the comorbidities that are frequently
22 present in these kids.

23 And by "kids," I mean even teenagers.

24 Q Have you had -- sorry, so you -- but your
25 understanding is that the University Hospitals LGBTQ 12:16:39

1 and gender care program does provide the rapid type of
2 affirmative care; is that right?

3 MR. BROOKS: Objection.

4 THE WITNESS: I already --

5 MR. BROOKS: Asked and answered. 12:16:48

6 THE WITNESS: -- answered that question. I'm
7 not -- I'm not aware of what they do. I -- I am --

8 BY MS. HARTNETT:

9 Q Okay. Sorry, I thought you had said you
10 thought that they provided rapid affirmative care, 12:17:01
11 which is why I was asking.

12 A I wouldn't be surprised if their definition of
13 inadequate evaluation is different than my evalua- --
14 my -- my definition of an adequate evaluation.

15 Q Do you know what their definition is of an 12:17:20
16 adequate evaluation?

17 A No. And because I don't know, I don't want to
18 endorse them, nor do I want to condemn them.

19 Q What is the basis for your understanding that
20 there is kind of rapid transition care being provided 12:17:32
21 out there?

22 MR. BROOKS: Objection; vague.

23 BY MS. HARTNETT:

24 Q Sorry, let me just use your term.

25 You said rapid affirmation. 12:17:42

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1 A Well --

2 MR. BROOKS: I was objecting to the outlier as
3 vague. I'm not sure what you -- are you referring to
4 the clinic you've been discussing or something else?

5 BY MS. HARTNETT: 12:17:52

6 Q What is your basis for your view that there
7 are clinicians in the United States performing rapid
8 affirmation care?

9 A Thank you for asking that question.

10 I have been in contact with -- that is, 12:18:03

11 parents -- there -- there are parent groups who cannot
12 find -- there -- there are groups of parents who
13 brought -- were brought together, who came together,
14 bounded -- bound together in organizations who are
15 objecting to what they call rapid affirmation and the 12:18:27
16 inability to find a therapist in their community who is
17 willing to just do psychiatric care like they would do
18 psychiatric care if a child presented simply with
19 anxiety or depression or substance abuse or some other
20 behavioral problem. 12:18:48

21 The -- the basis for -- for my -- the answer
22 to your question is parents, both Cleveland parents,
23 national -- parents from all over the country and
24 parents from the UK. I am aware that parents are
25 particularly perturbed by rapid affirmation and its 12:19:07

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1 treatment, and they -- they have complaints that their
2 child is not understood; that is, their problems have
3 not been understood.

4 Q How many parents have you talked -- how many
5 parents have you talked to about their concern with 12:19:26
6 what you call the rapid affirmation model?

7 A Well, I gave a talk to 35 parents probably a
8 year ago. In 2017, I think I wrote about it in the
9 article that -- the last four or five cases that I was
10 involved with, the parents all said the same thing; 12:19:53
11 that is, they were horrified that after one hour,
12 their -- their child was diagnosed and -- and had
13 recommend- -- and had recommendations that horrified
14 them.

15 Q Sorry, how -- where was the talk that you gave 12:20:10
16 to the 35 parents? What -- what was that?

17 A It was in -- it was in my easy chair in my
18 bedroom.

19 Q What was the convening? What was the venue
20 for that? 12:20:23

21 A It was a group of parents who invited me to
22 give a talk, and what I gave a talk on was -- the
23 aspects of what -- what I knew about human identity,
24 not just --

25 Q What was -- 12:20:38

1 A -- not just gender identity.

2 Q Was this group of parents affiliated with an
3 organization, or how did they -- how did they present
4 themselves? As some sort of an organization?

5 A A woman contacted me and said that she belongs 12:20:50
6 to an organization of -- of concerned parents of trans
7 teenagers or children. I'm not sure which. Mostly
8 teenagers. She actually sent me an analysis of --
9 of -- of -- that she made, a little research that she
10 had done that demonstrated a very high intelligence 12:21:10
11 in -- of their -- all the children in this group and
12 very high incidents of autism and other developmental
13 problems and -- so she sent me that data, and she
14 wanted some advice to -- from me about how to get that
15 published. 12:21:37

16 And -- and then she invited me to give a talk.
17 When we talked, she then said she would get back to me,
18 and she got back to me and invited me to give a talk to
19 the parent group. And so that's what happened.

20 Q Is the parent group called Genspect? 12:21:51

21 A No. I think -- it -- it might -- it -- this
22 was an American group of people and --

23 Q What was the parent's name that did the
24 research?

25 A You know, I -- I would have to look that up. 12:22:15

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1 I don't remember.

2 Q I'm just going to try to -- so I appreciate
3 what you've explained.

4 Could you tell me how many actual parents have
5 described to you, personally, an experience where their 12:22:29
6 child was diagnosed and prescribed treatment in an
7 hour?

8 A Well, if -- some people, it would be two
9 hours, okay?

10 Q Let me just start with one hour. 12:22:46

11 How many parents have told you directly that
12 their child had been prescribed -- diagnosed and
13 prescribed treatment in an hour?

14 A I would say perhaps 50 percent of the people
15 who -- who have consulted me. 12:22:59

16 Q And how many people have consulted you?

17 A I really can't answer. You know, if I told
18 you 11, if I told you 16, if I told you four, I
19 would -- I would have no conviction that I -- that --
20 that that answer is correct. 12:23:19

21 I'm telling you I had the impression that over
22 and over again parents complain about this. They
23 complain about affirmation. They're afraid of
24 affirmation, what that will mean to their child's
25 future. And they complain that they can't get their 12:23:35

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1 point of view to influence their thera- -- the -- the
2 person -- their gender expert that they took their kid
3 to and -- and that they can't find anyone else who
4 has -- who has the courage, they say, to just talk to
5 their kid without saying they believe in affirmation 12:23:55
6 because that's the right thing to do.

7 Q Thank you. I -- I just -- you've talked about
8 the importance of scientific data; correct?

9 A Correct.

10 Q And you've made the representation that there 12:24:09
11 is a practice of rapid affirmation happening in the
12 United States; correct?

13 A As -- as far as I know, yes.

14 Q And what I'm trying to understand is the basis
15 for your understanding that there is a phenomenon of 12:24:22
16 rapid affirmation happening in the United States.

17 And so --

18 A Well --

19 Q -- I guess my question is -- sorry.

20 A -- the basis. And I've tried to answer the 12:24:33
21 basis is -- is that the parents who consult me all
22 tell -- pretty much all tell me the same story. It is
23 multiple patient reports.

24 And when I -- when I was on that committee
25 that we talked about before, of psychotherapy, people 12:24:52

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1 in Australia, people in Ireland, people in London, in
2 various parts of the UK and -- let me think where this
3 is a source of -- and the United States have all
4 reported to me the same thing. Everyone says the same
5 thing, that the parents complained to them about going 12:25:18
6 to specialty care which rapidly confirms the diagnosis
7 and recommends affirmation and tends to make the
8 parents feel like they're -- they're doing a terrible
9 thing by resisting transition.

10 Q You mentioned -- 12:25:39

11 A So the answer to your question is multiple
12 sources, both directly in my clinical practice, both --
13 what I read about sometimes in these legal proceedings,
14 legal documents and in -- and -- and from my
15 colleagues. 12:26:01

16 I -- I just want you to know that if -- that
17 professionals all claim to do thorough evaluations, but
18 I -- I'm not sure that our definition of thorough
19 evaluation is -- is correct.

20 Q Have you talked to any gender-affirming 12:26:18
21 professional to learn what their practice actually is?

22 A Well, I've read Dr. Adkins, for example,
23 reassurance about the thorough evaluations done in her
24 clinic.

25 And -- have I talked to any affirmation -- 12:26:40

1 well, I did talk to the Cleveland Clinic people and --
2 who are -- were sharing with me their angst about what
3 they should do with these borderline personality kids,
4 kids who aren't doing well, who don't want to focus on
5 anything but their transgender state. So they consult 12:26:57
6 me about these -- these case- -- you know, they
7 consulted me about this.

8 So I guess the answer is yes.

9 And if you ask me the number, I would say it's
10 not a large number. I don't -- and I don't -- 12:27:14

11 Q Sorry, other than Dr. -- other than Dr. Adkins
12 and whoever you talked to at the Cleveland Clinic, have
13 you -- are you -- sorry.

14 You've never talked to Dr. Adkins; correct?

15 A I've never personally spoken to her, no. 12:27:25

16 Q So other than the people at the Cleveland
17 Clinic that you referred to, have you spoken to any
18 other gender-affirming professionals about their
19 practices?

20 A Well, in these various legal matters, 12:27:37
21 oftentimes I'm asked to review case material, and I --
22 and I -- I haven't visibly, virtually, talked to -- the
23 answer to your question is no, but I -- I certainly
24 have seen materials that indicate the -- the quality of
25 the interactions that have been between the affirming 12:28:09

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1 and the professional and the patient and sometimes the
2 parents.

3 Q And you mentioned -- you mentioned multiple
4 patient reports, I think, when you were saying what the
5 basis was for your review. 12:28:24

6 Do you recall that?

7 A Yes.

8 Q Are you -- and there, you're talking about the
9 patient would be the -- the parent of the child that's
10 being cared for; right? 12:28:30

11 A Yes. I think if --

12 Q In other words, you were -- you were not
13 getting complaints from the -- the child or adolescent
14 that was being discussed; you were getting the
15 complaint from the patient parent; is that right? 12:28:45

16 A Oh, I've heard -- I -- I've heard patients say
17 that they were a little surprised by the rapidity of
18 things, yes.

19 Q Sorry, one of your child or adolescent --

20 A So it's -- 12:28:58

21 Q -- patients --

22 A It's not entirely parents, but it's largely
23 parents.

24 Q And then I've asked you how many parents
25 you've directly heard reports of -- let's just say 12:29:10

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1 two-hour or less diagnosis and treatment. How many
2 parents have you heard that from directly?

3 MR. BROOKS: Objection; asked and answered.

4 THE WITNESS: I would say 15 sets of parents.

5 And if you allow me to accept the reports of the people 12:29:31
6 on the committee, probably it's over a hundred. But,
7 you know, as I already answered, I can't really -- I'm
8 just giving you numbers because you're asking for
9 numbers.

10 BY MS. HARTNETT: 12:29:54

11 Q Well, isn't it important to have good data?

12 A You're right, it is important to have good
13 information. And data varies in its nature. And
14 parental reports that are consistent over time, to me,
15 is good data. That represents good data. That are 12:30:10
16 good data, rather.

17 Q Have you ever had a parent report to you a
18 positive experience from an affirming practitioner, as
19 you describe them?

20 A Ever had a positive experience. 12:30:35

21 Well, last Sunday morning, I gave a talk at a
22 church, and a grandmother told me that her very
23 disturbed granddaughter has transitioned to a -- living
24 as a boy and she's far less disturbed and much happier
25 and she's beginning to restart her life as a student 12:30:50

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1 now, when she couldn't function as a student before.

2 So if a grandparent -- I mean, it's -- it's --
3 today's Wednesday. So that was Sunday morning.

4 So I think -- that is not the first time I've
5 ever heard from somebody. I've also heard from 12:31:05
6 grandmothers who were deeply concerned about their
7 grandchild.

8 And, actually, come to think of it, I had an
9 interview -- yes, I -- I have heard about a -- another
10 trans male teenager who is doing very well now as -- 12:31:23
11 and much better than they were doing living as a -- as
12 a distressed female.

13 So I do have positive reports of people doing
14 well.

15 And in -- in my years of taking care of -- of 12:31:39
16 adults, I've seen some people, at least who have come
17 back in follow-up after transition, who seem to be
18 doing very well in life.

19 I'm not saying that -- so I -- you know, I get
20 both sides of the coin here. 12:32:01

21 Q You haven't undertaken a scientific sampling,
22 though, to figure out what parents' experiences are
23 with affirming practitioners; correct?

24 A I -- no, I have no follow-up study on this. I
25 am like other people who don't have follow-up studies. 12:32:18

1 Q And it could be that parents that are having
2 negative experiences are the ones that are seeking you
3 out; correct?

4 A Yes. There's always a selection by a -- in --
5 in clinics. When -- when you have data coming from any 12:32:35
6 clinic, one of the methodologic questions is, What is
7 the selection bias?

8 And so I -- I represent a person who has some
9 kind of unknown or known reputation in the community,
10 and so people come to see me because they think I have 12:32:54
11 knowledge or attitude that is consistent with their
12 position.

13 But, you see, in the -- in the fundamentals
14 of -- of the use of statistics and creating scientific
15 methodology, selection bias is a well-known problem, 12:33:12
16 and that's one of the reasons why some studies need
17 to -- that's one of the advantages of having multisite
18 studies and multicultural -- studies from multiple
19 countries, is -- is what we're going to do about
20 selection bias. 12:33:31

21 Q I believe earlier you said that your view is
22 that the doctor's role isn't to recommend the treatment
23 for the minors who may be experiencing gender dysphoria
24 but, rather, to provide information to the parents and
25 the children and the parents and the children should 12:33:47

1 make the decision; is that fair?

2 A Yes. This is the idea that I am trying to
3 educate the world about, that, actually, doctors don't
4 know what the best treatment is for a particular child
5 and that they shouldn't pretend to know because there's 12:34:06
6 no follow-up data that are -- there's no compelling
7 follow-up data. There's just anecdotal reports like
8 you and I were just discussing. Or anecdotal reports.

9 And so given the fact that -- that people
10 believe doctors and they believe that doctors know 12:34:24
11 things and that I know doctors don't know things, you
12 see, what I'm saying, what I'm trying to influence the
13 world to think about is that we should make a -- we --
14 we recommend that you go to surgery for appendicitis
15 because we know the consequences of not having surgery. 12:34:44
16 You're going to die from this condition if you don't
17 have surgery, you see.

18 So we -- based on the consequences, we know
19 what is indicated medically to save life or preserve
20 function. 12:34:59

21 But in this particular area, the long-term
22 follow-up of children or adolescents or even adults who
23 undergo transition are not known. And I -- they're
24 not -- they're simply not known.

25 And because we are -- some doctors make 12:35:15

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1 recommendation to transition a seven-year-old or
2 transition a 14-year-old or remove the breasts of a
3 14-year-old, and I would say that what is the
4 scientific basis of your recommendation to tell
5 parents, who are often trusting of your knowledge base, 12:35:36
6 what is the scientific basis of your recommendation?

7 And I say, given what we know about science,
8 I'm not opposed to transitioning a child or
9 transitioning a teenager or an adult. What I'm saying,
10 that we should be able to educate, objectively, the 12:35:54
11 parents and the child themselves, you see, so that they
12 know the issues here.

13 And it's their child. They are legally
14 responsible and they're morally and ethically
15 responsible for the welfare of their child. And so I 12:36:11
16 think they need to be informed.

17 And -- and what I'm saying is, in the past,
18 doctors have recommended things, and I'm -- so I'm
19 questioning the wisdom of making a strong
20 recommendation because it's based on the allusion that 12:36:25
21 we know what is best for this kid or this adult. And
22 I'm saying, please, doctors, please be humble about
23 what your knowledge is here. Please respect the
24 limitations of your knowledge. That's all I'm saying.

25 So I -- I am objecting. I'm trying to teach 12:36:47

1 the world. If -- I know that sounds rather grandiose,
2 but I'm trying to teach the world that based on our
3 lack of information about the long-term follow-up, we
4 can give options for the treatment of this condition
5 and that option includes what you would call 12:37:03
6 affirmative care.

7 But we should understand the scientific basis
8 of affirmative care, you see, and we should understand
9 the limitations, and we should understand that even the
10 advocates of -- of gender-conforming surgery have 12:37:17
11 published two papers recently saying that the -- the
12 long-term psychosocial outcomes are not clear, that the
13 benefit of -- of -- of genital surgery or breast
14 surgery, in the long run, is not -- they're not clear.

15 And so people have undergone -- undertaken two 12:37:38
16 studies in the last year or two years to prove that
17 there are benefits. So why are we, in 2020 (sic),
18 doing studies to prove there are benefits if -- if we
19 already know the answer.

20 We don't know the answer. And I say because 12:37:56
21 we don't know the answer, there's an ethical
22 responsibility, a professional responsibility, to teach
23 the parents, teach the adult what is known and what is
24 not known.

25 What they decide is their business. It's 12:38:12

1 their prerog- -- it's their prerogative. It's their
2 child. It's their seven-year-old. It's not my
3 seven-year-old. See? It's not your seven-year-old.
4 It's not your 14-year-old. It's theirs. And it's a
5 weighted decision. And the idea that it's not a 12:38:25
6 weighted decision requires you to be an ostrich and
7 bury your head in the sand.

8 Q Do you think that politicians should be making
9 that decision?

10 MR. BROOKS: Objection. 12:38:36

11 THE WITNESS: Well, I -- I do ask myself the
12 question who should be making decisions about the
13 delivery of medical care, you see. And I do realize
14 that in some circumstances, politicians make decisions
15 that influence medical care and medical treatment. 12:38:55

16 I don't know the answer to that question, but
17 I don't know that doctors per se who are not informed
18 about the -- about the state of science really should
19 be making these decisions with the illusion that they
20 know best. I am not sure politicians know what's best. 12:39:16
21 I mean, when it comes to politicians, you know, we --
22 we all have skepticism.

23 But nowadays, what -- who is making decisions
24 are -- are judges, you see. I don't think juries as
25 much as judges and -- and state legislature and 12:39:35

1 governors are making decisions. I don't like that
2 either.

3 I would prefer that an informed medical
4 professional -- I would -- I would prefer that doctors
5 make these decisions based upon accurate scientific 12:39:54
6 information and not political ideology and not mixing
7 up civil rights concerns with medical decision-making.

8 So I realize we're in a -- this is a morass,
9 and I -- all I -- all -- my point to you today is let's
10 look at the science and let -- let the doctors decide 12:40:21
11 or let the politicians decide, let the governors
12 decide, let the judges decide, but on the basis of
13 science.

14 Q And are you aware of any scientific study
15 showing that affirmative care practitioners in the 12:40:40
16 United States are providing rapid affirmation, a
17 scientific study, not just anecdotal reports?

18 A There was a study out of the UK about 20 years
19 ago. I kind of think the author of the study was
20 M-O-L-E. I'm not certain. And they did a follow-up 12:41:10
21 study of people who were given sex reassignment surgery
22 immediately because they asked for it, with -- with
23 very little screening, versus people who were treated
24 as usual, because in that days, people had psychiatric
25 evaluation and psychotherapy, and I think they found in 12:41:33

1 the small numbers of patients that they operated on
2 versus the people who weren't operated on, that there
3 seemed to be -- they seemed to be happier in the short
4 term after surgery than the people who didn't have
5 surgery.

12:41:49

6 But you know what I've been saying to you
7 in -- well, maybe I haven't quite said it yet. What
8 I'm saying is, when we come to evaluate the impact of
9 these treatments, we need to agree upon -- we have to
10 have a consensus, and it should be an international
11 consensus, about what is the ideal way to evaluate the
12 effects of these treatments.

12:42:07

13 Should it be, like, at six months, at
14 twelve months, should it be at six -- two years,
15 five years, ten years. And we should agree upon the
16 mecha- -- the measurements that we're going to use
17 prior to actually doing the study so that we all agree
18 upon both -- both the strengths and the limitations of
19 the methods.

12:42:28

20 So what I'm --

12:42:42

21 Q Yeah, maybe my question --

22 A What I'm trying to do is to refine the
23 requirements to answer your question.

24 Q Thank you. And I think maybe my question may
25 have been unclear.

12:42:55

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1 What I'm trying to figure out is that you've
2 testified about a perception that there's this
3 widespread practice of providing rapid affirmation
4 service in the U.S.; is that fair?

5	A	Yes, I do have that perception.	12:43:05

6 Q And what I'm trying to figure out, is there
7 any kind of scientific or other -- otherwise kind of an
8 analysis of a -- of that healthcare market to determine
9 whether in fact that is actually happening or in fact
10 whether these are just anecdotal occurrences that 12:43:22
11 you've learned of?

12 A There -- your question is one of a series of
13 questions that I would have to answer as far as I know,
14 there are not -- there are not respected scientific
15 methods demonstrating my -- my impression. 12:43:44

16 Q Thank you. If you could turn to page --
17 paragraph 6 of your -- or it's probably on the same
18 page you have there, but I'm going to just ask a
19 question about paragraph 6 of your declaration -- or
20 your report. 12:44:02

21 And you talk about -- you can read the whole
22 thing. I'm not trying to misread it into the record,
23 but I wanted to focus on the sentence that says (as
24 read):

25	I have at one time or another	12:44:13

1 recommended or prescribed or supported
2 social transition, cross-sex hormones,
3 and surgery for particular patients,
4 but only after extensive diagnostic
5 and psychotherapeutic work." 12:44:26

6 Do you see that?

7 A I do.

8 Q Have you ever recommended cross-sex hormones
9 for a minor patient?

10 A No. 12:44:37

11 Q Have you ever prescribed cross-sex hormones
12 for a minor patient?

13 A Is that a different question than you just
14 asked me?

15 Q Well, you have recommended or prescribed or 12:44:53
16 supported, and so I could go into asking you what the
17 difference is, but I just figured I'd ask you -- is
18 there a differences between recommended, prescribed and
19 supported?

20 A Oh, yes. I feel like my view of my role is to 12:45:08
21 write a letter of recommendation describing the patient
22 in detail, the -- the diagnosis, the patient's
23 sensibilities, whether I think this would be beneficial
24 to the patient at this time in his life.

25 The last person that I wrote, I was doing 12:45:26

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1 psychotherapy with a young person, starting at age 16,
2 and saw this person over the course of a year and a
3 half. I promised that if they continued talking to me,
4 at the end of the time, I -- if patient still wanted
5 hormones, I would give hormone- -- I -- I wrote a 12:45:47
6 letter of recommendation.

7 And I did write a letter of recommendation,
8 and the patient did take hormones. He went off to
9 college, failed miserably at college, transferred
10 college, and I sadly I tell you, and I -- I sadly tell 12:46:01
11 you, this person died of a heroin overdose in his dorm
12 room at Ohio State University.

13 And I know from the parents, postmortem, that
14 he acquired a girlfriend, and he then said that it's
15 not so bad -- he's rethinking this matter. It's not so 12:46:23
16 bad being -- being a male and having sex with someone.

17 But I don't know whether -- I -- his heroin
18 overdose, which was his third heroin overdose, was
19 accidental death or suicide.

20 So I have provided hormones. I do have that 12:46:40
21 really negative taste in my mouth from that experience.
22 I don't -- I don't -- I don't have remorse about giving
23 hormones to this person because I promised that if --
24 that it is his decision.

25 His parents weren't happy with that decision, 12:47:02

1 but they also agreed with the decision. And now
2 they're, of course, in perpetual mourning for their
3 deceased 18-year-old child.

4 So, yes, listen, I also have given hormones to
5 someone else who is living okay, who is not made any 12:47:20
6 suicide attempts. But it is, as I described in that
7 paragraph, after I get to know these people. And to
8 tell you, I -- as best as I can tell, they appreciate
9 that.

10 Q Thank you. I'm just -- sorry for the -- for 12:47:35
11 the person that you -- your -- your patient that you
12 mentioned, the -- the 18-year-old, I'm -- I'm sorry to
13 hear about that.

14 Sorry, when was that? What -- what time
15 period? 12:47:47

16 A That was --

17 Q Datewise.

18 A -- March 17th, 2021.

19 Q And did you prescribe the -- or, sorry, write
20 a letter for the hormones before the person was 18 or 12:47:58
21 only once they were 18?

22 A I think the person turned 18 in August or
23 September, and I think I wrote the letter right near
24 the person's birthday. Whether it was before or after,
25 I'm not sure. 12:48:19

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1 Q How about social transition, have you ever
2 recommended or prescribed or supported social
3 transition for a minor?

4 A A minor being someone less than 18?

5 Q Correct. 12:48:34

6 A Have I ever recommended, prescribed -- I have
7 never prescribed. I have met people who already had
8 social transition, and I had supported them even in the
9 face of their parents' objection. But I don't think I
10 have ever prescribed social transition to a person. I 12:49:00
11 cooperate with it. I recognize that -- I recognize
12 that it is the patient's decision. And while I may not
13 have thought it was a wise decision to transition or to
14 surreptitiously take hormones, you know, from China or
15 something, I -- I don't interfere with it. I just talk 12:49:30
16 about it.

17 So -- but if you're really asking have I said,
18 oh, Parents, you should transition your child, I think
19 the answer is no.

20 Q Yeah. So I'm trying to -- that's -- thank you 12:49:43
21 for clarifying that. I -- I'm trying to figure out if
22 you've supported the transition of a -- the social
23 transition of any minor patients.

24 A Yes.

25 MR. BROOKS: Objection; vague. 12:49:53

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1 BY MS. HARTNETT:

2 Q When was the last time you supported the
3 social transition of a minor patient?

4 A Two years ago, I'm guessing.

5 Q Okay. Let me -- do you know who B.P.J. -- 12:50:08
6 B.P.J. is the plaintiff in this case.

7 Do you know if B.P.J. is a girl or a boy?

8 A I know nothing about B.P.J.

9 Q So you've reviewed none of her medical records
10 or anything like that? 12:50:32

11 A Yeah, I would presume that this is a trans
12 boy -- a trans girl who was born a -- a boy, but I
13 wouldn't -- I have no certainty.

14 Q What makes you presume that?

15 A Well, because trans -- trans girls 12:50:47
16 generally -- I mean -- how should I say it? Trans
17 girls -- trans adolescent girls generally don't -- wait
18 a -- I'm getting confused here. Excuse me.

19 I presume that B.P.J. is an -- was born and
20 assigned and is a natal -- was a natal male. 12:51:17

21 But if it's a natal female, I -- I've not
22 heard anything where a natal female becomes a trans boy
23 and wants to compete against boys. If there is a
24 lawsuit like that, that has been raised, I am unaware
25 of it. 12:51:43

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1 When I read these things in the newspaper,
2 it's -- it's -- they're -- they're always about natal
3 boys who live as trans women or girls and want to
4 compete against women. So that's why I presume that
5 B.P.J. must be a natal male. 12:52:04

6 But because my role in this case had nothing
7 to do with the athletic side, it's just to -- to
8 provide some basis of -- some background basis on the
9 science of transgender knowledge and the lack of
10 knowledge, I didn't spend time investigating that. 12:52:23

11 Q Okay. And are you familiar with the law
12 that's being challenged in this case that's called
13 H.B. 3293?

14 A No.

15 Q Could we just turn to page 20 of your 12:52:42
16 declaration, paragraph 50 -- or your -- sorry, I'm
17 saying declaration. I mean report.

18 MR. BROOKS: We're getting there.

19 MS. HARTNETT: No, take your time. Page 20,
20 paragraph 50. 12:53:00

21 MR. BROOKS: Let's see. This is under -- just
22 simply -- since I can't fit it all on the screen at
23 once, it's under the heading that says, "The
24 affirmation therapy model (model #4)." And now, under
25 that, I have paragraph 50 showing on the screen. 12:53:14

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1 MS. HARTNETT: There is a way to, I believe,
2 make that -- I don't know if he needs that to be that
3 large to read it, but there is -- if you hover over the
4 document, you can zoom in or out.

5 MR. BROOKS: Perhaps. But this is, I think, 12:53:31
6 much smaller, and it would be hard to read.

7 THE WITNESS: I have the entire paragraph 50
8 in front of me.

9 BY MS. HARTNETT:

10 Q Okay. Thank you. 12:53:41

11 So I was looking through your report, trying
12 to see if there was a connection to the context here,
13 which is this sport -- whether the plaintiff can play
14 sports, and I'm just looking -- you can look at all of
15 paragraph 50, if you need to, but I'm going to be 12:53:51
16 focused on -- well, feel free to take a look.

17 But you're -- under this part called "the
18 affirmation therapy model." That's the heading that's
19 above paragraph 50.

20 Do you see that? 12:54:04

21 A Yes.

22 Q And you're referring to -- what -- you say
23 that -- you're referring to some advocates and
24 practitioners that go much further. That's in your
25 second line there. And then I'm going to just read one 12:54:14

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1 sentence in the middle of the paragraph. (As read):

2 "They argue that the child should be
3 comprehensively resocialized in grade
4 school to (sic) their aspired-to
5 gender. As I understand it, this is 12:54:27
6 asserted as a reason why male students
7 who assert a female gender identity
8 must be permitted to compete in girls'
9 or women's athletic events."

10 Did I read that correctly? 12:54:37

11 A Yes, you did.

12 MR. BROOKS: And I will -- well, you can ask a
13 question. I'm going to ask the witness to read the
14 entire paragraph so we don't lose the --

15 MS. HARTNETT: He should feel free. I'm 12:54:50
16 not -- this is not a trick.

17 MR. BROOKS: Nope.

18 BY MS. HARTNETT:

19 Q Let me know when you're ready.

20 A I've read the paragraph. 12:55:22

21 Q Do you know whether the law being challenged
22 in this case applies to grade school?

23 A I don't -- I don't know the law being
24 challenged here.

25 Q So you don't know whether the law at issue 12:55:35

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1 requires that transgender youth be comprehensively
2 resocialized; is that fair?

3 MR. BROOKS: Objection.

4 THE WITNESS: When I talk about
5 comprehensively resocialized, it was not in 12:55:51
6 relationship to this law; it was in relationship to the
7 American Academy of Pediatrics' recent study, I think
8 in 2018, by Rafferty, et al., where it was asserting --
9 they were asserting such things that I'm summarizing
10 here. 12:56:18

11 And, see, for them, participation in athletics
12 just follows their fundamental assumption that they
13 know what's best for these children even though they
14 have no long-term -- they don't even have adolescent
15 follow-up, let alone adult follow-up. 12:56:35

16 And so I just think that the case of
17 athletics -- the issue of athletics is a secondary
18 derivative issue about the more fundamental matter of
19 when and how, to what extent, and before -- what
20 requirements are necessary before we socialize a child, 12:56:55
21 you see.

22 So if you think about the -- your issue today
23 about athletics, it's what I would call a downstream
24 issue, downstream from the fundamental thing that we
25 were talking about before the last break about what are 12:57:15

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1 the requirements to ethically enable parents to make
2 this decision without doctors pretending like they know
3 what's best for a seven-year-old or an eight-year-old
4 or a 12-year-old or a 15-year-old, you see.

5 So this is a downstream question about which I 12:57:34
6 feel I have no legitimacy to pretend expertise.

7 So I think every question you ask me about
8 this, I'm going to have to say, listen, this is not
9 my -- this is not my wheelhouse. This is not my
10 knowledge base. My knowledge base is about what we 12:57:54
11 were talking about, you know, about the evaluation of
12 children and teens.

13 BY MS. HARTNETT:

14 Q So here, where you say, "this is asserted as a
15 reason why male students who assert a female gender 12:58:07
16 identity must be permitted to compete in girls' or
17 women's athletic events," when you say -- asserted by
18 whom? Is it the American Academy of Pediatrics? Is
19 that who you're referring to there?

20 A No, I don't think it's entirely that. I think 12:58:23
21 it has to -- you know, this is a -- this is a big
22 cultural issue in many, many states. They made -- the
23 NCAA, you know, the high school athletic associations,
24 whatever the names, the acronyms of those
25 organizations, they have made policies based upon 12:58:48

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1 information that they've gotten from various, quote,
2 expert groups, and -- and there is this -- in education
3 services today, there is this enormous emphasis on
4 diversity and support for all forms of diversity, and
5 so I -- I think the answer is not it's just from the 12:59:12
6 American Academy of Pediatrics. I think the American
7 Academy of Pediatrics is influenced by these larger
8 social trends that have recognized how much harm we've
9 done to various -- to women, for example, or to African
10 Americans or to Asians, and we are trying, as a 12:59:34
11 society, to make things more open and to -- to
12 represent more people in the public discourse in arts,
13 in music, in the theater and so forth.

14 So there's just a broad, broad cultural trend
15 towards being much more inclusive, you see, and -- and 12:59:52
16 I just think the trends -- athletic issue must be
17 viewed in terms of the larger social questions that are
18 being answered in a political sense in our culture.

19 MR. BROOKS: Counsel, when you get to a
20 breaking point, I think it is one o'clock, and it would 01:00:10
21 be a good time to take a lunch break.

22 MS. HARTNETT: We can break now. I have a
23 couple more questions on this paragraph, but we can
24 pick it up after lunch. What would you prefer?

25 MR. BROOKS: You can finish up the paragraph. 01:00:27

1 MS. HARTNETT: Sure.

2 BY MS. HARTNETT:

3 Q So -- so is it your view that allowing a
4 transgender youth to participate on the team of
5 their -- the sex that they present as, is that a 01:00:39
6 psychotherapeutic intervention that would dramatically
7 change the outcome for that child?

8 A I'm not certain.

9 Q What is your concern -- I'm sorry, please.

10 A I think if -- I think if a child, let's say a 01:01:02
11 14-year-old, wants to run track or play a sport as a
12 member of a female -- the female side of the sport and
13 if the school or the -- the State or the -- the
14 organization that -- that organizes high school
15 athletics or junior high school athletics says, no, you 01:01:31
16 can't because you were a natal male and you -- trans is
17 not accepted as -- for athletic purposes, I think that
18 person would be disappointed. I think that would be
19 disappointed. And disappointment may look like
20 depression. It may increase the person's anxiety for a 01:01:52
21 while. But like many, all of us get disappointed in
22 life, and, you know, we deal with it. And sometimes we
23 grow from our disappointment.

24 So I would think they would be disappointed.

25 Whether that is to be considered harm, you see, I don't 01:02:12

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1 think we would -- we should, just on the basis of
2 disappointment, refer to that as harm. Harm is a
3 different concept, you see.

4 And -- so I guess the answer to your question
5 is I'm not sure. 01:02:32

6 Q But do you think that permitting them to play
7 with -- in that example, allowing the 14-year-old
8 person that identifies and is a girl to play with the
9 girl team, do you believe that that would make them
10 more likely to continue to identify as transgender when 01:02:50
11 they otherwise would not?

12 MR. BROOKS: Objection; ambiguous.

13 THE WITNESS: They would otherwise continue --
14 you -- you mean -- if I understand --

15 BY MS. HARTNETT: 01:03:05

16 Q I'm sorry, I'll ask a better questions.

17 I'm just trying to figure out if your opinion
18 is that allowing transgender, let's just say,
19 adolescents to play on sports teams that match their
20 gender identity will cause them to continue to identify 01:03:15
21 as transgender when they otherwise would not.

22 A I have no idea the answer to that question. I
23 would imagine that they would continue to identify as a
24 trans female, but I don't know what would happen to
25 their identity if they didn't. That was the other side 01:03:40

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1 of your question, the last part of your questions.

2 So I guess I can answer part of the question.

3 It would be my opinion, if we allowed a child
4 who currently identifies as a trans girl to participate
5 in a girl's athletic -- organized athletics, that that 01:03:57
6 would do nothing -- that would -- that would reinforce
7 the idea that she continues -- that she is a trans
8 girl. Not that she is a girl, but that she's a trans
9 girl. That's -- I think that would be my opinion.

10 About the other aspect to your question, I 01:04:20
11 don't know the answer.

12 Q But is your opinion that there's a -- is that
13 a -- in your opinion, is there something wrong with
14 reinforcing the girl being on -- sorry -- the girl's
15 gender identity of being on the team? 01:04:33

16 Like, do you have a problem with that, or are
17 you okay with the 14-year-old girl playing on the --
18 transgender girl playing on the girls' team if the
19 rules allow it?

20 MR. BROOKS: Objection; vague, compound. 01:04:42

21 THE WITNESS: If you -- if you look narrowly
22 at the individual girl, we get one set of
23 considerations.

24 If we look at fairness, if we look at the
25 perspective of the other girls, the natal girls who are 01:05:07

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1 participating, we get another perspective.

2 If we look at the parents' perspective of the
3 very talented athletes who are natal girls who may be
4 defeated by these trans girls, we get yet a third or
5 fourth perspective. 01:05:31

6 BY MS. HARTNETT:

7 Q Well, that's not your area of expertise;
8 correct?

9 A But you -- you just anticipated what I was
10 going to say. I mean, you're asking me opinions that I 01:05:39
11 have no legitimate expertise to answer. I -- I'm
12 just -- I'm separating the perspectives for you. And I
13 say your -- your question is not as simple as it
14 sounded because there are these other perspectives to
15 be considered which people other than me are going to 01:05:57
16 consider.

17 There is -- shall I repeat?

18 There is the child --

19 Q No, I don't think so. I don't think you
20 should repeat. But what I do -- would like would be 01:06:08
21 before we have lunch, just an answer, which is do you
22 object --

23 MS. HARTNETT: Can you -- can the reporter
24 read back my last question, please.

25 THE REPORTER: Yes. 01:06:15

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1 (Record read.)

2 MR. BROOKS: Objection; compound, form of the
3 question, vague.

4 You can answer, if you are able and know what
5 the question is. 01:07:02

6 MS. HARTNETT: That's -- enough coaching.

7 THE WITNESS: Pardon me? I didn't hear what
8 you just said.

9 BY MS. HARTNETT:

10 Q I was telling your counsel to please stop 01:07:07
11 coaching you. And I can ask a better question.

12 A Oh.

13 Q Is it your perspective that allowing a
14 transgender girl to participate on a girl team,
15 consistent with her gender identity, is harmful to the 01:07:18
16 transgender girl?

17 A No, I don't think it's harmful in the short
18 run to the transgender girl. In the long run, if the
19 transgender girl detransitions, say, in five years, I
20 wonder what he will now think about what happened five 01:07:36
21 years before when she was competing against girls as a
22 girl.

23 But in the -- I presume your question is in
24 the short term, you see? And I guess in the short
25 term, I don't think it would harm the child to the 01:07:58

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1 extent that it reinforces their current identity.

2 But as you may or may not know, gender
3 identity can evolve over time. And so when people
4 detransition and return to presenting themselves as a
5 boy and thinking of themselves as a boy, they then have 01:08:20
6 to -- they then have to consider what happened when
7 they were -- when they were presenting themselves as a
8 girl and believing that they were a girl. They no
9 longer believe that they're a girl, but they did back
10 then, you see? 01:08:39

11 So I don't know, I don't think anybody knows,
12 what implications, what harm, might come from their --
13 what retrospective view of the harm that -- that they
14 cause themselves by presenting -- by competing against
15 girls. So -- 01:08:58

16 Q Does anybody know the implications of the
17 disappointment that the transgender girl might
18 experience from exclusion, or is it similarly
19 indeterminant?

20 MR. BROOKS: Objection. 01:09:09

21 THE WITNESS: Well, I -- I think I've already
22 answered the question, that disappointment -- I would
23 expect it if a -- if the girl -- the trans girl wanted
24 to participate and was prohibited by some larger force
25 from participating, they would be disappointed, and it 01:09:24

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1 may have -- it may have -- it -- and I couldn't predict
2 the outcome of the disappointment, whether it would
3 precipitate depression or whether it would precipitate
4 giving up their trans identity, as being unrealistic,
5 that other people are saying I am very unrealistic 01:09:47
6 and -- and this is unfair and I'm asking for an unfair
7 advantage.

8 So, you know, I can't -- I don't -- these are
9 not areas that I -- that anyone has had any experience
10 with, you see. And -- and I -- it's hard for me to 01:10:01
11 give you a simple answer.

12 It feels to me, Ms. Hartnett, that you are
13 trying to get me to answer a question in a certain way,
14 and I'm just trying to say I think it's more
15 complicated. And I think you're asking me to give an 01:10:16
16 opinion about which I don't have adequate knowledge,
17 and I don't -- that's all. Period.

18 Lunch.

19 MS. HARTNETT: Let's go to lunch.

20 THE VIDEOGRAPHER: We are off the record at 01:10:35
21 1:11 p.m.

22 (Lunch recess.)

23 THE VIDEOGRAPHER: We are on the record at
24 2:11 p.m.

25 MS. HARTNETT: Thank you. 02:11:22

1 BY MS. HARTNETT:

2 Q Welcome back, Dr. Levine.

3 I think before the break, we had -- I'm not
4 sure what page you have up, but I -- I'm at
5 paragraph 50 of the declaration. 02:11:31

6 A So are -- so am I.

7 Q Okay. Let's -- I was trying to -- and the
8 reason why we were talking about that is there was a
9 mention of athletic events there, and the other mention
10 of athletic events in your declaration is at 02:11:43
11 paragraph 130. So if you could go to 130, I'll have a
12 question about that.

13 Let me know when you get to 130, please.

14 MR BROOKS: We are at 130, which fits on the
15 screen. 02:12:14

16 BY MS. HARTNETT:

17 Q Great. So here in this paragraph, you say, in
18 the third sentence, the following (as read):

19 "It is evident from the scientific
20 literature that engaging in therapy 02:12:26
21 that encourages social transition
22 before or during puberty—which would
23 include participation on athletic
24 teams designated for the opposite
25 sex—is a psychotherapeutic 02:12:37

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1 intervention that dramatically changes
2 outcomes."

3 Do you see that?

4 A I do.

5 Q And you don't know if H.B. 3293 applies to 02:12:46
6 prepubertal kids; right?

7 A I'm sorry, would you repeat that question.

8 Q You don't know if H.B. 3293 applies to
9 prepubertal kids?

10 A I already testified that I don't know the 02:13:03
11 content of the deal.

12 Q So is it your opinion that allowing
13 transgender children and adolescents to play on sports
14 teams will continue -- will cause them to continue to
15 identify as transgender? 02:13:21

16 A I think it -- well -- well, you know, my
17 hesitance is because you used the word "cause."

18 Q I'm just trying to --

19 A A child --

20 (Simultaneous speaking.) 02:14:10

21 BY MS. HARTNETT:

22 Q Oh, sorry, go ahead.

23 A That's why I have taken so long. I'm -- I'm
24 thinking about the word "cause" and its implications in
25 my mind. I -- I do think that various aspects of 02:14:20

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1 social transition tend to continue the child on a life
2 course consistent with trans life, whether or not
3 they're aware of the risk that they're entailing or
4 not.

5 I think that's as close to an answer I can 02:14:45
6 give you.

7 Q Are you aware of any research indicating that
8 by preventing children from playing on sports teams
9 consistent with their gender identity that will prevent
10 them from continuing to identify as transgender going 02:14:59
11 forward?

12 A I'm not aware of research literature about
13 athletic teams and its impact, positive or negative, at
14 all. I'm totally unaware.

15 Q Okay. Do you think that by excluding 02:15:14
16 transgender girls from playing on the girls' team the
17 law that's being challenged in this case stigmatizes
18 transgender girls?

19 MR. BROOKS: Objection.

20 THE WITNESS: I think it may disappoint 02:15:48
21 transgender girls. Stigma has another concept. You
22 know, it has to do with social things.

23 I -- I think a reasonable mental health
24 professional could assume that if a child wanted
25 something and was prohibited from it, they would be 02:16:03

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1 disappointed, at least initially.

2 Other than that, I -- I don't care to comment.

3 BY MS. HARTNETT:

4 Q Well, say a child wants a cookie and they
5 aren't allowed to have it. That's disappointing; 02:16:23
6 right?

7 A Yes.

8 Q Is the disappointment that a transgender child
9 would have from being excluded from a sports team
10 consistent with their gender identity essentially that, 02:16:31
11 equivalent of the cookie denial?

12 MR. BROOKS: Objection; calls for speculation.

13 THE WITNESS: I don't know if you even put my
14 smile into the text.

15 Obviously, you know, there -- there are 02:16:57
16 degrees of disappointment in the universe. And to
17 equate that with a cookie, I don't know. I prefer not
18 to even answer that question.

19 BY MS. HARTNETT:

20 Q Well, your -- your point of view is that 02:17:10
21 people that experience being transgender also generally
22 experience a wide range of other distressing feelings
23 and conditions; correct?

24 A My point of view is what?

25 Q That people who are transgender also 02:17:27

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1 experience a wide range of other concerns and -- and
2 issues; correct?

3 A Yes, I think -- yes.

4 Q That they're subject to serious mental health
5 issues, that's your point of view; correct? 02:17:47

6 A I think they're apt to encounter a number of
7 frustrations in their future lives that could add to
8 their social anxiety, their sense of pervasive sadness
9 and it lead to solving the problem in ineffective ways,
10 like substance abuse. 02:18:13

11 So, yes, I do think that being transgender,
12 for -- for many, many people, poses adaptive challenges
13 in the present and in the future.

14 Q How do you know that that's based on being
15 transgender as opposed to how the transgender people 02:18:34
16 are being treated, or do you not distinguish between
17 the two?

18 A Because -- because some of the -- in children,
19 some of the psychiatric problems that they have are --
20 occur well before there's any awareness of the society. 02:18:54

21 And in every cross-sectional study of adults
22 in the transgender community have shown that the --
23 that they're a vulnerable population and they're
24 vulnerable to many psychiatric difficulties, and the
25 common explanation for that, among trans advocates, is 02:19:19

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1 that it's entirely due to social discrimination whereas
2 I think if you look at the premorbid and the
3 accompanying psychiatric difficulties of many trans
4 people, these -- these -- the social discrimination has
5 only added to the -- the internalized conflicts about 02:19:37
6 what they're doing.

7 So I think it's far more complicated than it's
8 merely a result of stigma, so to speak.
9 "Discrimination" would be a better word, I guess.

10 Q Yeah, I'm -- thank you. And I'm trying to 02:19:54
11 reconcile that view with the notion that excluding a
12 transgender youth who, in your view, might be subject
13 to these various preexisting psychological problems,
14 why -- where you're having -- where -- what is the
15 basis for you believing it would just be a simple 02:20:09
16 source of disappointment for the trans youth to be
17 excluded from a team, consistent with their gender
18 identity, as opposed to a more severe harm?

19 MR. BROOKS: Objection.

20 THE WITNESS: Number one, I don't think 02:20:22
21 there's any research in this area. So whatever --
22 whatever you would like to conclude, I think there's no
23 basis for it.

24 I'm just trying to understand, based on my
25 knowledge of human beings, that for one person, it 02:20:37

1 would be a major disappointment and it might lead to
2 harm for that person, and for another person, it might
3 be a major disappointment that leads to no harm, and
4 for another person, it might be, oh, well, so what, and
5 it's not a big -- not a big deal. 02:20:52

6 Every study of human beings shows the variety
7 of human beings. And we can't predict that if you
8 exclude a child from anything on the basis of their
9 gender identity, that it's going to cause --
10 automatically, you can guarantee it will cause harm. 02:21:12
11 There's just no reason to think that.

12 It doesn't mean there isn't a child who might
13 not be harmed, but it doesn't mean that all the
14 children will be harmed, and it doesn't mean that the
15 harm will follow in the same manifestation. 02:21:27

16 Human beings have a variety of responses to
17 everything.

18 BY MS. HARTNETT:

19 Q So is your view for the trans girls that would
20 be excluded under a policy of not allowing them to play 02:21:43
21 on the team consistent with their gender identity, that
22 they should just toughen up and stomach the
23 disappointment?

24 MR. BROOKS: Objection.

25 THE WITNESS: You're putting words in my 02:21:55

1 mouth. That's not my view. That's not how I was --
2 that's not how I have spoken about it. You're
3 summarizing it in a very negative way for me. I don't
4 accept your language. It's not me.

5 BY MS. HARTNETT:

02:22:09

6 Q Okay. You don't have to.

7 How would you put it?

8 A I already put it.

9 MR. BROOKS: Objection.

10 BY MS. HARTNETT:

02:22:15

11 Q You mentioned before the break that you also,
12 in your view, had to look at the potential harms or the
13 effects on the other people at issue, and I think you
14 mentioned the other girls on the team; is -- did I hear
15 you right?

02:22:26

16 A I think I did mention that.

17 Q Are you giving an expert opinion in this case
18 about the harm to girls on a team where they would have
19 to include a transgender girl?

20 A I don't know how many times, Ms. Hartnett, I
21 have to tell you that I don't consider myself having an
22 expert opinion on this subject. I have stated what I
23 stated, but I don't -- I don't -- I don't feel like I
24 represent an expert.

02:22:41

25 And so the answer to your question is, no, I

02:22:59

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1 don't have an expert opinion on that.

2 Q Thank you. I have a few questions about your
3 expert report. I'm just going to go back to the
4 beginning and go through sequentially, and I'll --
5 please feel free to read the paragraphs I cite to you 02:23:16
6 while I'm asking you questions.

7 My first one is going to be back on
8 paragraph 5, page 2.

9 MR. BROOKS: Getting there.

10 Paragraph 5 is on the screen. 02:23:36

11 MS. HARTNETT: Yeah, we were there before.

12 BY MS. HARTNETT:

13 Q I just had a question about -- so I was
14 comparing this report to the declaration that was
15 submitted at the beginning of the case. That was the 02:23:47
16 one from the Washington State declaration that had been
17 attached to an earlier motion in the case. And that's
18 something I introduced as Exhibit 86. So if you need
19 to refer to it, feel free.

20 But I will just represent to you that in the 02:24:02
21 version of paragraph 5 that was in your earlier
22 declaration, you had certain language that's no longer
23 in this report. I'll read it to you and then -- just
24 curious as to why you removed it.

25 You -- this is the declaration that you signed 02:24:15

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1 in May of 2021. (As read):

2 "As the incidence of gender dysphoria
3 has increased among children and youth
4 in recent years, larger numbers of
5 minors presenting with actual or
6 potential gender dysphoria have
7 presented to our clinic.

02:24:29

8 I currently am providing psychotherapy
9 for several minors in this area. I
10 also counsel distressed parents of
11 these teens."

02:24:41

12 Do you know why you removed that language from
13 your -- this report?

14 MR. BROOKS: And, counsel, are -- asking that
15 question, are you representing that that or similar
16 language doesn't appear somewhere else in the report?

02:24:54

17 MS. HARTNETT: I was unable to find that
18 language in this report. It was in paragraph 4 of the
19 PI declaration, which is now paragraph 5 of this
20 report, and I was not able to find that language.

02:25:09

21 THE WITNESS: I would imagine the answer to
22 the question is I didn't think it was relevant to this
23 particular document.

24 Please understand, in preparing this document,
25 I did not read the -- Exhibit 86.

02:25:29

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1 BY MS. HARTNETT:

2 Q Is it true that larger numbers of minors have
3 been presenting with actual or potential gender
4 dysphoria to your clinic?

5 A No. It's true that across the world larger 02:25:46
6 numbers of minors are requesting services for gender.
7 That's an epidemiologic phenomenon that exists on four
8 continents.

9 Q Is it true that you are currently providing
10 psychotherapy for several minors in this area? 02:26:07

11 A Yes.

12 Q How many?

13 A It depends on what era you're -- what month,
14 what week, what -- what year you're talking about. If
15 you're talking about within the last year, I would say 02:26:22
16 probably four or five kids.

17 Q Can you give me the ages of those kids?

18 A Probably from 14 to 17.

19 Q And how many of those have you seen more than
20 one time? 02:26:41

21 A Each of them.

22 You should -- well, okay.

23 Oh, one of them I've seen once, I'm sorry.

24 I -- let me correct that.

25 Q For the other four, do you see them on a 02:27:01

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1 monthly basis?

2 A No. I -- I tend to see them more often.

3 Q Are there any of those patients that you have
4 seen on a monthly or less basis, other than the one you
5 only saw once? 02:27:21

6 A Well, I hear from patients I see in the past
7 periodically, sometimes. I hear from their parents. I
8 sometimes hear from them. But it's -- it's not
9 anything regular.

10 Q Yeah, I'm -- thank you. I'm just trying to 02:27:45
11 understand. There was a statement made in your
12 May 2021 declaration that you were currently providing
13 psychotherapy for several minors in this area, and I'm
14 just trying to figure out, is that actually true today?

15 A No, it's not true today to the same extent 02:27:59
16 that it was when I wrote the original -- the Tingley
17 declaration.

18 Q Thank you. Moving down in here, you have on
19 page -- paragraph 7 and paragraph 8, you identify a
20 couple of cases where you previously provided 02:28:15
21 testimony.

22 A Yes.

23 Q There's the -- the case in the Eastern
24 District of Massachusetts, in the First Circuit, that
25 you refer to in paragraph 7. 02:28:29

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1 Do you see that?

2 A Yes.

3 Q And then there's the Younger litigation in
4 paragraph 8.

5 Do you see that? 02:28:37

6 A Yes.

7 Q And you do cross-reference your CV list and
8 then the Tavistock case.

9 Do you see that?

10 A Yes. 02:28:47

11 Q Why did you choose to highlight the
12 Massachusetts and the Younger case here?

13 A Well, the Massachusetts case, under
14 Judge Wolf, Judge Wolf asked me to be a judge's
15 witness. That was the beginning of my legal 02:29:10
16 involvement in that whole area of transgenderism. So I
17 think that that's noteworthy. It's also noteworthy
18 because that became -- among the DOC attorneys across
19 the nation, that's a very landmark case, and it's often
20 quoted in various other legal matters. 02:29:29

21 So it seemed to me that you ought to know that
22 I began in that area in 2006 with Dr. -- with
23 Judge Avery.

24 And what was the second part of your question?

25 Q Oh, the Younger case and why you included that 02:29:49

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1 here.

2 A I included that because that was my entry case
3 into transgender children and the -- when parents don't
4 agree on the treatment of their trans child and -- and
5 courts are involved and -- I mean, that is not just 02:30:10
6 happening in the Younger case. That's happening in
7 other jurisdictions as well. And so I --

8 Q In the Younger -- oh, sorry.

9 A That that's the kind of thing you wanted to
10 know. That is a credential, in a sense. Or I thought 02:30:26
11 that you would like to read that case, if you could.

12 Q Are you aware the jury rejected the father's
13 claim in the Younger case and awarded the
14 decision-making to the mother?

15 MR. BROOKS: Objection; mischaracterizes the 02:30:43
16 record.

17 THE WITNESS: One of my complaints about my
18 participation is I -- I often am not informed about the
19 outcome and the progress of the cases that I've
20 testified in. 02:30:55

21 I did -- I did hear something like you --
22 what -- what you said, but it seems to me that it was a
23 more complicated decision than you summarized.

24 BY MS. HARTNETT:

25 Q Are you aware that -- of the more recent 02:31:14

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1 litigation in Texas regarding a directive from the
2 attorney general about the investigation of the --
3 sorry -- by the directive of state officials to
4 investigate those providing transgender care for child
5 abuse? Does that ring a bell? 02:31:30

6 MR. TRYON: Objection.

7 THE WITNESS: I only know about that because I
8 read it in the papers. I have not --

9 BY MS. HARTNETT:

10 Q Okay. That's what I was going to ask you. 02:31:40

11 Were you involved in that? Were you asked to
12 provide an expert opinion in that case?

13 A Never.

14 Q Is there a reason why you didn't include the
15 Nosewor- -- Norsworthy case when you were summarizing 02:31:50
16 your background here in paragraph 7 and 8?

17 A The Noseworthy case is one of, I don't know,
18 seven or eight cases. I -- if you look at my CV, I'm
19 sure it's listed in my CV.

20 This is a prisoner case. I didn't think it 02:32:22
21 had to do with -- it just didn't seem it had to do with
22 athletics and -- and teenagers.

23 Q Are you aware that your testimony was
24 partially excluded in a case called Claire in Florida
25 that was about the -- it was precluded with respect to 02:32:40

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1 the testimony about the motivations that plaintiffs had
2 for seeking gender confirmation surgery.

3 A I was not --

4 MR. BROOKS: Objection.

5 THE WITNESS: I was not aware. 02:32:51

6 BY MS. HARTNETT:

7 Q Just flashing forward to paragraph 13 here.
8 This is a paragraph where you're discussing, in part,
9 Dr. Adkins' declaration. And my first question is, at
10 the end of this paragraph, you talk about a life course 02:33:15
11 perspective?

12 A Yes.

13 Q I'm just curious if that's a term that you
14 coined or that's from somewhere else in the literature.

15 A If I took credit for coining that term, I 02:33:36
16 think it would be -- I didn't -- I didn't coin the term
17 "life perspective."

18 I'm a -- I'm a psychiatrist, and I see people
19 throughout the life cycle, and so I am constantly
20 confronted with the consequences of early life 02:33:54
21 decisions and of behavioral patterns.

22 I have a natural life perspective on matters.
23 I certainly didn't -- I don't believe I coined the
24 term.

25 Q Well, I ask because it's in quotes, and so I'm 02:34:10

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1 just wondering if it's something that you refer to your
2 method as the life course perspective or if that's a
3 method I could look to in the literature somewhere.

4 A I think it's in quotes -- I think it's in
5 quotes because I wanted to emphasize the perspective 02:34:25
6 that this whole question about how to take care of
7 trans youth needs to be understood, not does it make
8 them happy in the current life, but what will it do to
9 the whole course of their life.

10 And so by putting it into italics (sic), I -- 02:34:46
11 I -- perhaps -- perhaps I shouldn't have done that, but
12 I was just trying to bring the reader's attention to
13 the perspective here that the decisions that are made
14 in teenage years, for example, or in their 20s or in
15 their 30s have implications, serious implications, for 02:35:08
16 10 years, 20 years, 30 years down the pike.

17 And as an adult psychiatrist who deals with
18 people, you know, from 96 down, I certainly see the
19 impact of previous life decisions on their current
20 suffering. 02:35:32

21 And so that's all it refers to, that -- and I
22 do believe that if you spend your time in pediatrics,
23 you probably don't have as -- as sharp a focus on the
24 life perspective that an adult person -- adult -- a
25 per- -- specializes in adults or who has a lot of 02:35:50

1 experience with adults have. That's all I'm trying to
2 say.

3 Q Is it your view that Dr. Adkins' approach is
4 to make the young person happy as opposed to creating a
5 happy, high-functional, mentally healthy person for the 02:36:06
6 next 50 to 70 years of life?

7 A I believe that Dr. Adkins has hope that she is
8 going to create a happy, functional human being for the
9 next 70 years of life, but I do believe she's
10 influenced, primarily, on making her child -- her 02:36:20
11 current patients happy.

12 The question is does Dr. Adkins have any
13 evidence whatsoever that the decisions that she has
14 been making with teenagers and younger children,
15 does -- does she know that creates happiness in ten 02:36:38
16 years or in five years. And certainly, I don't think
17 she knows what happens in 30 years.

18 But I think as a society, you and I as
19 representatives of society, can recog- -- recognize the
20 relevance of the question. 02:36:56

21 We want to separate, at all times, physicians'
22 beliefs from the evidence that supports those beliefs.

23 Q What's the basis for your notion that
24 Dr. Adkins lacks an understanding of how to create a
25 happy, highly functional, mentally healthy person for 02:37:15

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1 the next 50 to 70 years of life?

2 A Because she's a pediatric endocrinologist.
3 Because she's a busy person dealing with young people.
4 Because she doesn't follow-up her patients, I'm sure,
5 for 30 years. 02:37:31

6 Q Do you follow-up your patients for 30 years?

7 A Some of them, yes. You know I published a
8 paper about a 30-year follow-up of a trans person.
9 Maybe you don't know. I published a paper about
10 returning to the male gender role after 30 years. 02:37:48

11 Now, I can't say that I have, you know, 20
12 patients I've followed for 30 years, but I -- I have
13 certainly written about that case, and in -- in writing
14 about that case, I have raised certain issues that are
15 germane to your questioning right now. That is, a life 02:38:05
16 perspective, a life course perspective is something
17 that's reasonable and that an educa- -- a physician
18 needs to be thinking about the long-term outcome of
19 what is being done today.

20 Q What is the basis for you -- but you're -- 02:38:24
21 sorry, I think you've already stated it, but I -- is
22 there any other reason you have to believe that
23 Dr. Adkins is not informing herself about the
24 consequences of her actions on her patients 30 --
25 30 years from today? 02:38:39

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1 A Only that she could not know what happens.
2 She hasn't been practicing 30 years, I don't believe.
3 And I don't believe she is in a position, considering
4 the work that she does, to have systematic follow-up,
5 even for shorter periods of times, on her patients. 02:38:54

6 If, for example, she has systematic follow-up
7 on 80 percent of the patients she's ever given a
8 hormone treatment for, that should be in the
9 literature. And she knows, she should know, given
10 the -- the -- what's absent from the literature, how 02:39:15
11 welcome such a study would be, such a report would be.
12 But as far as I know, she hasn't published that
13 information.

14 Q So your testimony is that you're basing your
15 assumption that Dr. Adkins doesn't conduct systematic 02:39:28
16 follow-up on her failure to publish a study showing her
17 systematic follow-up?

18 A I'm sorry, you'll have to repeat that. Too
19 many similar phrases.

20 MS. HARTNETT: Can the -- well, I'll try. 02:39:42
21 BY MS. HARTNETT:

22 Q Is the basis for your assumption that
23 Dr. Adkins doesn't engage in systematic follow-up of
24 her patients her failure to publish research indicating
25 her systematic follow-up? 02:39:52

1 A No. I am sure Dr. Adkins follows her
2 patients, but she's a pediatrician, basically, and
3 usually, and I can't be certain about this, that at 18,
4 pediatrics people turn the kids over to adult
5 endocrinologists. 02:40:23

6 And so I think just in the nature of being a
7 pediatric endocrinologist, although she may see some
8 kids into their 20s, I would imagine that the usual
9 trend in pediatrics is to hand kids off, when they're
10 18, to other practitioners; and, therefore, she 02:40:37
11 probably has limited systematic follow-up after 18.

12 And if you extend that by years, like five
13 years and ten years and so forth, I would imagine that
14 she may have a case or two that she follows or knows
15 about, but it would not be anything like systematic. 02:40:55

16 So the answer to your question is the basis --
17 did she not publish, and that's the basis. I'm giving
18 you an additional basis.

19 Q Thank you. You mentioned one patient you had
20 followed up over the course of 30 years, and I think 02:41:10
21 said something like maybe 20 or -- how many patients,
22 overall, do you feel like -- do -- do you believe that
23 you followed up with over a period of decades in your
24 practice?

25 A Very -- very few. Because I exist in America, 02:41:26

1 and in America, we have no means of guaran- -- of -- of
2 insisting on follow-up.

3 And on -- in -- another reason why is that
4 when people transition, they -- they want to get rid of
5 their professionals who dealt with them, and they don't 02:41:47
6 naturally come back.

7 In fact, all attempts at follow-up, not just
8 in my clinic, but elsewhere, we -- we reach -- we reach
9 very few people.

10 For example, in a 2002 study of everyone who 02:42:02
11 had sex reassignment surgery by one surgeon, only
12 30 percent of the people who ever had surgery by this
13 one surgeon actually were available for follow-up.

14 And all follow-up studies -- very few
15 follow-up studies can have a hundred percent of the 02:42:22
16 data of all the patients.

17 Follow-up is a problem. It's a much better
18 problem -- it's solved much better in Scandinavia than
19 it is in the United States. The United States have 50
20 states. They have different rules. Nobody -- I don't 02:42:39
21 think we -- we don't publish follow-up studies in the
22 United States very often.

23 Q What do you do to try to follow up with your
24 patients?

25 MR. TRYON: I think we have a connection 02:43:08

1 problem.

2 MS. HARTNETT: Is that me? It could be me.

3 THE VIDEOGRAPHER: We're just going to pause
4 and see if he -- there he is. He's back.

5 MR. TRYON: There -- he came back. 02:43:15

6 BY MS. HARTNETT:

7 Q Sorry, I think you froze.

8 Did you hear my question?

9 MR. BROOKS: No, I think we don't -- we did
10 not hear a pending question in this room. 02:43:32

11 Can you hear us now?

12 MS. HARTNETT: Okay. Sorry. The video froze
13 from your end.

14 MR. BROOKS: We -- we see --

15 BY MS. HARTNETT: 02:43:40

16 Q My question was, what do you do to follow up
17 with your patients?

18 A I ask them to follow up with me after their
19 surgery, for example, or after their consultation with
20 another person, another professional, and they actually 02:43:54
21 rarely do.

22 Q Do you try to find them if they don't come
23 back to you --

24 A Yes.

25 Q -- afterwards? 02:44:07

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1 A Yes.

2 Q How?

3 A I write them notes. I write them a letter.

4 Sometimes I write them a cute little postcard reminding

5 them of who I am. But they know what I mean.

02:44:15

6 Q If you have such limited follow-up with your

7 own patients, how do you know your method has -- what

8 the effect of your method is on people 30 years later?

9 A I don't know. And I -- I am like other people

10 in this field. I don't know the 30-year implication of

02:44:47

11 what we're doing. I don't know the 20-year implication

12 of what we're doing. I'm just raising the question,

13 shouldn't we be concerned about a life course

14 perspective.

15 I don't know and the people who are advocates

02:45:05

16 don't know, you see. I don't know how they can be so

17 sure that they're going to create a happy life.

18 Q So for all you know, your method could

19 actually be harming your patients more than the other

20 methods; is that fair?

02:45:24

21 A You mean in the long run I may be harming them

22 by talking with them, say, for six months about their

23 decision, what -- what they should go -- what -- what

24 they want to do?

25 I can't imagine that -- that my

02:45:48

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1 psychotherapeutic -- my relationship with them that is
2 helping them to consider their thoughts, their feelings
3 and their futures is -- is harming them and in 30 years
4 they're going to have some terrible result of my
5 intervention, you see. 02:46:07

6 What you're trying to contrast is talking to a
7 person, say, for six months, every -- twice, three
8 times a month for six months with socializing them in a
9 new gender or supporting, giving them hormones and --
10 and saying yes to genital surgery or mastectomy or 02:46:24
11 sterilizing procedures, you see.

12 You're comparing Dr. Levine or
13 psychotherapeutic talking, conversation, extended
14 evaluation, with major biologically sterilizing,
15 sexually dysfunction in causing interventions. 02:46:44

16 I really think -- we're not talking about
17 apples and oranges here. I think we're talking about
18 apples and zebras.

19 Q Your report discusses four competing models of
20 therapy; correct? 02:47:13

21 A Correct.

22 Q So you have the apple, the zebra and two other
23 things in that; correct?

24 MR. BROOKS: Objection.

25 THE WITNESS: No. 02:47:20

1 BY MS. HARTNETT:

2 Q The four competing models are watchful
3 waiting, sub 1; sub 2, psychotherapy; and the
4 affirmation model.

5 That's what you've set forth; correct? 02:47:30

6 A That's right.

7 Q And I'm asking you whether, for all you know,
8 the psychotherapy model may be creating more harm for
9 people than the affirmation theory model. You just
10 don't know? 02:47:46

11 A I think I've already testified that it's hard
12 for me to even conceptualize that I'm causing harm.
13 Sometimes I'm causing frustration because "I want
14 hormones now" and you're 14, and I'm sorry, we have --
15 I want to talk about this. 02:48:14

16 But I don't really think that's harm in the
17 way that when I look at the cross-sectional data on
18 adults who have transitioned and -- and the
19 comorbidities that they have, I consider those to be
20 manifestations of harm, you see. 02:48:32

21 I don't really think that talking briefly
22 and -- and honestly and examining things is -- is a
23 source of harm.

24 It is --

25 Q But your -- your practice isn't to talk 02:48:46

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1 briefly to someone. You're talking -- right?

2 The -- the -- the model that you're setting
3 forth is to talk with them at length and get to know
4 them; correct?

5 A Yes, this used to be the model -- before 2011, 02:48:55
6 this was the endorsed model by the World -- by WPATH,
7 you see. I'm not talking -- I'm not inventing a new
8 model here. This was the model we had in the '60s, the
9 '70s, the '80s and the '90s and in the 2010s and --

10 Q And it's your view that the psychotherapy -- 02:49:14

11 A The view model changed.

12 Q It's your view that the psychotherapy model
13 cannot, by its nature, harm anyone?

14 A I know some people think that it harms people.
15 I don't believe that, actually. 02:49:28

16 Q Well, let me give you an example.

17 Say you're meeting with a patient and they
18 want to talk you about their need or their perceived
19 need for cross-sex hormones and you don't agree or
20 choose not to support them with a letter. 02:49:45

21 Do you -- is that a fair -- just assume that,
22 okay?

23 And that person then goes on to stop seeing
24 you, has been taken off course from getting the
25 cross-sex hormones, ends up becoming distraught at 02:49:55

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1 their condition and commits suicide.

2 Is that a situation where the psychotherapy
3 model might be responsible for causing harm?

4 MR. BROOKS: Objection; calls for speculation.

5 MR. TRYON: Objection. 02:50:08

6 THE WITNESS: If that -- such a patient goes
7 to me -- comes to me and after -- in the first session
8 wants a letter and I refuse to provide it, I will help
9 that person -- if the person doesn't know, I will refer
10 them to clinics -- to other resources. 02:50:26

11 The idea that my refusal would cause them to
12 suicide is enormous and deep that leaves out so many
13 intervening factors as to make me say I can't possibly
14 agree with what you said.

15 BY MS. HARTNETT: 02:50:43

16 Q But it's possible that your patients, for
17 example, have higher rates of suicide than other
18 patients that have gone through a different model;
19 correct? You just don't know?

20 MR. TRYON: Objection. 02:50:52

21 THE WITNESS: It's equally possible that the
22 patients have a lower rate of suicide that have gone
23 through Dr. Levine's care.

24 BY MS. HARTNETT:

25 Q But it's also possible that they have had a 02:51:04

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1 higher rate of suicide going through Dr. Levine's care;
2 correct?

3 MR. BROOKS: Objection --

4 MR. TRYON: Objection.

5 MR. BROOKS: -- calls for speculation. 02:51:13

6 BY MS. HARTNETT:

7 Q You said it's possible that they have a lower
8 rate. It seems that the flip side of that is it's
9 possible that they had a higher rate; is that correct?

10 A You're -- 02:51:23

11 MR. BROOKS: Same -- same objection.

12 THE WITNESS: You're asking me to speculate
13 about something you know I don't have the answer to, so
14 why should I give you an answer that I don't have? Why
15 are you asking -- 02:51:32

16 BY MS. HARTNETT:

17 Q You testified that it's possible that --

18 MS. HARTNETT: I'm going to ask for an answer
19 to my question without coaching, please.

20 BY MS. HARTNETT: 02:51:37

21 Q My -- I asked if it's possible that the
22 patients of Dr. Levine have a higher rate of suicide
23 than patients going through another method, and then
24 you responded it's possible that they have a lower --
25 lower rate. That's an answer. 02:51:49

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1 I'm asking you, is it possible that they also
2 have a higher rate?

3 MR. BROOKS: And I have objected to the
4 question as calling for speculation.

5 BY MS. HARTNETT: 02:52:01

6 Q Please answer.

7 A In order to -- in order to have an answer to a
8 rate question, one has to have a denominator and
9 numerator. I have neither a denominator or numerator;
10 and, therefore, I can't really ask -- in any expert 02:52:23
11 way, I cannot answer a question about the rate.

12 You're asking me theoretical possibilities,
13 and there probably are at least three theoretical
14 possibilities, and I could probably think of more,
15 but -- 02:52:40

16 Q What are the three?

17 A There would be no difference in the rates,
18 right? The rates could not be ascertained because the
19 denominator -- the numerator and the denominator
20 couldn't be determined. And then the fifth one would 02:52:52
21 be because the numerator can't be determined.

22 So if you ask me a question about rate, it's a
23 mathematical question. It's a scientific question.
24 But you're not asking it in a scientific way at all.
25 And I can't answer it. 02:53:07

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1 To the extent that I have any expertise, it's
2 on the science. It's not on the speculation side of
3 things.

4 Q Your expert opinion is that the affirmative
5 model is more harmful than the psychotherapy model; 02:53:18
6 correct?

7 A My -- my expert opinion is that the
8 affirmative model does not have the scientific
9 justification to declaim -- to -- to declare it to be
10 the best practice. That's my expert opinion that -- 02:53:35

11 Q Does the psychotherapy model have any more
12 justification than the affirmative model?

13 A Only the tradition that if any other
14 psychiatric problem presented in a 14- or 15-year-old,
15 no one, no one would object to an extended evaluation, 02:53:53
16 a psychotherapeutic exploration and the use of a
17 medication to a drug -- to address some comorbidity.

18 It's just that when a -- when the child
19 declares themselves trans, we want to create a whole
20 different approach to this situation. That's my point. 02:54:12

21 Q And just to make sure that we close the loop
22 on the other point, because I'm not quite sure what the
23 answer was there, is it your testimony that it's
24 possible that your -- that Dr. Levine's patients could
25 have lower rates of suicide than other methods? 02:54:29

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1 MR. BROOKS: Objection; calls for speculation.

2 THE WITNESS: I'm afraid -- although you don't
3 understand my answer to the question, I feel like I've
4 answered the question repeatedly already.

5 BY MS. HARTNETT: 02:54:46

6 Q Well, you've said that it could be -- I
7 thought you -- I thought I understood you to say you
8 could have lower rates, you could have a missing
9 numerator or denominator or equivalent, but I didn't
10 hear whether or not you think another possibility is in 02:54:54
11 fact that the rates of suicide could be higher from
12 your patients.

13 A Well, perhaps you missed the implication of
14 what I said, that it could be higher, it could be
15 lower, it could be the same, it could be indeterminant 02:55:06
16 because of the denominator issues, and it could be
17 indeterminant because of the numerator issues.

18 Q I appreciate that. Thank you.

19 We've talked about Dr. Adkins a bit here. I
20 just wanted to ask you -- this is flashing back to -- I 02:55:22
21 think we're in paragraph 13.

22 You then go on, in paragraph 16, to talk about
23 Dr. Safer. Let me know when you're there.

24 A Got it.

25 Q Other than reviewing Dr. Safer's expert 02:55:43

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1 report, do you have any other familiarity with
2 Dr. Safer's practices?

3 A I believe he's the head of a New York gender
4 team, clinic.

5 Q Have you ever met him before? 02:55:58

6 A Not that I am aware of.

7 Q Have you ever been to his clinic?

8 A No.

9 Q Have you ever spoken to any of his patients?

10 A Not that I'm aware of. 02:56:11

11 Q How about Dr. Adkins, have you been to her
12 clinic?

13 A No.

14 Q Have you spoken to any of her patients?

15 A Not that I'm aware of. 02:56:23

16 Q So do you know whether or not Dr. Safer's
17 approach is focused on creating a happy, healthy --
18 sorry -- happy, highly functional, mentally healthy
19 person for the next 50 to 70 years?

20 A Ms. Hartnett, I think everyone in this field 02:56:42
21 is hoping that what they're doing is creating that
22 outcome. I would presume that Dr. Safer believes that
23 and Dr. Adkins believes that. I just go back to the
24 fact that we don't know the answer in what they're
25 doing and what they're doing is a rather dramatic 02:57:04

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1 interventions in a person's biology, their physiology,
2 their anatomy and their social roles, and it seems to
3 me that if we're making such a very, very
4 life-changing -- or cooperating with such a life
5 change, a profound life change, that's going to effect 02:57:21
6 every aspect of their lives, or most aspect of their
7 lives, we ought to at least acknowledge that we don't
8 have the follow-up data to match our belief systems.

9 And as I wrote about in the most recent
10 publication, I do think that ethically we have a 02:57:40
11 responsibility to inform people of what science knows
12 and what we as professionals believe, but it's not
13 supported by science.

14 So in answer -- to summarize my answer, I
15 believe that your experts believe that they are 02:57:58
16 creating a happy, healthy, functional life, even in the
17 face of the fact that they -- cross-sectional studies
18 of adults who are transgender and those who have had
19 complete medical surgeries have significant problems.

20 And so what I have been saying, in summary, is 02:58:18
21 that we -- we should separate our beliefs from what
22 science knows.

23 Q You said "cross-sectional studies." You're
24 just saying that those are lacking to -- to -- to -- to
25 substantiate their approach. Is that what you're 02:58:37

1 saying?

2 A Please repeat that. You sort of -- I couldn't
3 understand.

4 Q Sorry. You had -- yeah, fair -- fair enough.

5 I think you said something about 02:58:44
6 cross-sectional studies being lacking to support their
7 approach. Is that what you were saying?

8 A Yes. Not only cross-sectional studies failed
9 to support the idea that everyone is living happily
10 ever after or the majority are living happily ever 02:59:04
11 after, the -- the Swedish study that was published in
12 2011 that had outcome data on everyone who had sex
13 reassignment surgery over a 30-year period. You may
14 know that as the D-H-E-N-J-A (sic) study, et al. They
15 demonstrated -- the -- the recommendation of that study 02:59:26
16 is that everyone after sex reassignment surgery should
17 have lifelong psychiatric care because the suicide rate
18 was 19 times higher after this than the general
19 population. The death rates were higher of cancer and
20 of heart disease, the criminal rates were higher, and 02:59:45
21 the admission rates to psychiatric hospitals were
22 higher, after, then general population.

23 So that group in Sweden, in 2011, said, wow,
24 these people are not necessarily doing so well as a
25 group; that is, everyone that was -- everyone who had 03:00:01

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1 sex reassignment surgery was in that. So
2 we wouldn't -- we wouldn't call that a cross-sectional
3 study. We would have a life perspective study, you
4 see. You are aware --

5 Q Was that -- was that comparing it to the 03:00:14
6 general population, though? Not transgender people
7 that had gone untreated, right?

8 A That study did not include people who were not
9 treated with surgery, that's right.

10 Q Right. So to figure out if surgery makes a 03:00:26
11 difference, wouldn't you study a population that had
12 had surgery versus the population that had not had
13 surgery, all of transgender people?

14 A Yes, I often wondered why the authors of that
15 study did not study those people that they had records 03:00:39
16 on who didn't have surgery. It's one of the missing
17 issues about that. It doesn't take away from the fact
18 that relative to non-transgender people of either sex,
19 these people don't do nearly as well in life. But it
20 doesn't answer the question that you're raising, and 03:00:59
21 that's been amazing -- that's an amazing absence. One
22 wonders why that is absent. I don't know why.

23 Q So just to be clear, the -- the thing that's
24 absent is testing whether or not it's actually the
25 medical interventions with the transgender people that 03:01:16

1 are accounting for the difference in suicide from
2 the -- is that what you were saying?

3 MR. BROOKS: Objection; vague.

4 THE WITNESS: I'm saying that it would have
5 been nice to have four control groups. And they only 03:01:35
6 had three control groups. And I don't --

7 BY MS. HARTNETT:

8 Q Right.

9 A I don't understand why there wasn't the fourth
10 control group that you are raising because it does -- 03:01:43
11 you know, I already testified that nothing is certain,
12 but this would have increased our conviction about
13 whether or not people are dying of cancer and heart
14 disease and HIV and suicide and so forth at a higher
15 rate compared to those who are transgender but who 03:02:08
16 weren't getting the surgery.

17 So I don't know the answer.

18 Q Could I go to -- paragraph 18 has several
19 subparagraphs. I just have a couple of questions on
20 this. The first is on paragraph 18A. 03:02:28

21 I just had a -- it was a minor reference, but
22 I'm just curious about your own use of terminology.
23 You had, here in the second sentence of 18A (as read):

24 "While hormonal and surgical
25 procedures may enable some individuals 03:02:45

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1 to 'pass' as the opposite gender
2 during some or all of their lives..."

3 And the sentence continues.

4 In the declaration you had -- that had been
5 filed, your declaration that was filed at the PI stage, 03:02:55
6 the words "female identifying male" were used instead
7 of "some individuals."

8 Is -- is there a reason why that would have
9 been changed?

10 A In the original -- what was in the original 03:03:15
11 draft that you looked at?

12 Q It said "a female identifying male" as opposed
13 to "some individuals."

14 MR. BROOKS: I'll object to the question as
15 characterizing that as original. 03:03:24

16 BY MS. HARTNETT:

17 Q Well, it was the declaration -- I compared the
18 declaration that was apparently submitted without your
19 knowledge on your -- in -- in the PI stage of this case
20 with the report, thinking that you had done both of 03:03:36
21 them, and I'm -- what I'm just observing was that the
22 words "female identifying male" had been used in this
23 paragraph and then now has been replaced by "some
24 individuals," and I'm just curious as to why that
25 change was made, if you know. 03:03:47

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1 A I don't know. I don't remember that phrase.
2 That seems like -- that seems like a rather awkward
3 phrase, you know, that you quoted.

4 Q Yeah, why -- is that a phrase you use --
5 "female identifying male," is that a phrase that you 03:04:00
6 use?

7 A I -- I may have at one time or another used
8 that phrase.

9 Obviously, for everyone concerned, the
10 language -- the vocabulary -- the -- the -- the 03:04:12
11 socially acceptable vocabulary in this field changes so
12 often.

13 So, you know, as I told you, I spent probably
14 25 hours developing this, and there are numerous
15 changes here and there which I could not possibly 03:04:33
16 recall.

17 And I can't answer your question. I really
18 don't know the answer.

19 Q Okay. Well, I'll ask one more in that vein,
20 and then we'll move on. 03:04:42

21 For paragraph 18L, which is at the top of
22 page 8 -- and this a paragraph where you're
23 describing -- you say that (as read):

24 "Hormonal interventions to treat
25 gender dysphoria are experimental in 03:05:01

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1 nature and have not been shown to be
2 safe, but rather put an individual at
3 risk of a wide range of long-term and
4 even life-long harms..."

5 And then you go on to list all that. 03:05:10

6 A Yes.

7 Q The prior version of this -- in the same place
8 had -- had language that said -- I'm going to just read
9 it to you. (As read):

10 "Putting a child or adolescent on a 03:05:21
11 pathway towards life as a transgender
12 person."

13 And that has been removed. I'm just curious
14 as to why that was removed.

15 MR. BROOKS: Late objection. 03:05:28

16 THE WITNESS: I actually -- I can't give you a
17 specific answer to the question. I have no memory
18 of -- of -- of making that editorial change.

19 I -- I -- I am sensitive to and actually have
20 a preference to not using the same phrase endlessly in 03:06:01
21 any document. And one of my concerns about previous
22 documents has been the redundancy of phrases, and so
23 I -- I try not to repeat certain powerful phrases.

24 I -- I think they actually have more impact on the
25 reader if they read them once or twice and not 15 03:06:26

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1 times. So that may have been an example of that.

2 As a writer, I'm very sensitive to redundancy,
3 and I prefer to have things done short -- in shorter
4 versions than in longer versions, but that is not
5 always in keeping with legal requirements. 03:06:46

6 Q Turning to paragraph 19, this is -- I'm not
7 going to -- there's a couple of questions I had
8 about -- or, sorry, not -- 20. You're talking about
9 biological sex.

10 Do you see that? 03:07:01

11 A Yes.

12 MR. BROOKS: Sorry, you want 19 or 20?

13 MS. HARTNETT: I'll move to 20.

14 BY MS. HARTNETT:

15 Q You say that (as read): 03:07:08

16 "Sex is not 'assigned at birth' by
17 humans visualizing the genitals of a
18 newborn; it is not imprecise.

19 Do you see that?

20 A Yes. 03:07:17

21 Q Do you have any experience with the process of
22 assigning sex to newborns at birth?

23 MR. BROOKS: Objection.

24 THE WITNESS: You know, I -- probably for a
25 week in my medical school pediatrics rotation I was

03:07:32

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1 part of the newborn nursery and delivery -- and in
2 obstetrics. The newborn delivery room phenomenon of
3 saying, Mother, your -- you have a daughter. Or,
4 Mother, you have a son. So I guess that's part of my
5 experience. I'm a parent, so I've had that experience. 03:07:52
6 What I -- period. I think that's an answer.

7 BY MS. HARTNETT:

8 Q Thank you. You also say in this paragraph,
9 among other things, that sex is determined at
10 conception; correct? 03:08:06

11 A Yes, when -- yes, I do -- that's when sex is
12 determined, yes.

13 Q You say that at the end of the first
14 sentence of -- sorry -- the second sentence of
15 paragraph 20. And the source that you cite in this 03:08:22
16 paragraph for everything in this paragraph is a
17 document that says "NIH 2022."

18 Do you see that?

19 That's at the top of page 9.

20 A Yes. 03:08:34

21 Q What is NIH 2022?

22 A I think the first author's name is Aditi
23 B-H-R-A-R- -- Bhar- -- Bhargara or something like that,
24 but it has probably 15 authors, the paper.

25 Q So that's a paper that you were citing? 03:08:55

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1 A Yes.

2 Q Okay. Let me move down to section D. So that
3 starts on page 14 of your report.

4 MR. BROOKS: We have it.

5 BY MS. HARTNETT: 03:09:26

6 Q And you -- this is your section about "Three
7 competing conceptual models of gender dysphoria and
8 transgender identity."

9 Do you see that?

10 A Yes. 03:09:35

11 Q Is this your construct, these three models?

12 A Yes.

13 Q Paragraph 37, you describe the developmental
14 paradigm, I guess; is that fair?

15 A Yes. 03:09:50

16 Q I was comparing the declaration submitted at
17 the earlier stage of the case with the report here, and
18 I noticed that some language was deleted, and I will
19 double-check to represent to you that it is not still
20 here. 03:10:09

21 But the language that was deleted from
22 paragraph 37 is as follows (as read):

23 The developmental paradigm does not
24 preclude a biological temperamental
25 contribution to some patients'

03:10:22

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1 life (sic); it merely objects to
2 assuming these problems are biological
3 in origin. All sexual behaviors and
4 experiences involve the brain and the
5 body." 03:10:31

6 Is there some reason that you removed this
7 language from this report?

8 A Well, I think I said it in a different way. I
9 said (as read):

10 "The developmental paradigm is mindful 03:10:42
11 of temperamental, parental bonding,
12 psychological, sexual, and physical
13 trauma influence (sic), and the fact
14 that young children work out their
15 psychological issues through fantasy 03:10:53
16 and play and adolescents work out
17 their issues by adapting various
18 interests and identity labels."

19 This is -- this is the material that I
20 prepared as the expert witness report for this 03:11:07
21 particular case.

22 Over time, you see, I have a different -- I --
23 I say things more efficiently, I believe.

24 I could elaborate that, but I don't think it's
25 relevant. 03:11:27

1 Q No. Thank you. I appreciate it.

2 But you agree, sitting here today, that all
3 sexual behaviors and experiences involve the brain and
4 the body?

5 A I agree that all behaviors involve -- well, 03:11:38
6 the brain and the body is really one thing, you know.
7 They're just part of the biology of a -- of the
8 human -- of human beings, and that -- those biology --
9 multiple biologic factors interact with other
10 psychosocial factors throughout life to shape our 03:12:03
11 feelings and our behaviors and so forth.

12 Q In paragraph 38, you refer to a Littman 2018
13 study.

14 Do you see that?

15 A Paragraph 38, yeah. 03:12:17
16 Yeah.

17 Q Are you aware that that article was -- had to
18 be withdrawn and corrected and republished?

19 MR. BROOKS: Objection.

20 THE WITNESS: I am aware that there was a lot 03:12:32
21 of political brouhaha about that and that various trans
22 advocates accused that author of bad things or whatever
23 but that the restatement of the study really did not --
24 did not amount to a great change.

25 But -- but, in fact, there was a brouhaha by 03:13:01

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1 the publication objecting to her methods so to speak,
2 but really were -- they were objecting to her
3 conclusions.

4 BY MS. HARTNETT:

5 Q Was her method an anonymous survey of parents? 03:13:16

6 A Her -- it was a survey of parents, right.

7 Q Do you know if they were anonymous or not?

8 A At this moment, I don't know.

9 Q You go on in section E here, starting on
10 page 16, to talk about four competing models of care. 03:13:32

11 MR. BROOKS: Sorry.

12 BY MS. HARTNETT:

13 Q I also was wondering --

14 MS. HARTNETT: Oh, sorry.

15 MR. BROOKS: I hit the wrong thing, and the 03:13:38
16 document disappeared off the screen. Let me -- I'm not
17 sure what's going on here.

18 Okay. Sorry, I -- it accidentally closed as I
19 tried to get rid of some pop-up on the screen, and we
20 will get us back. 03:14:04

21 And, I'm sorry, what paragraph were you at?

22 MS. HARTNETT: It's section header E, page 16.

23 MR. BROOKS: Page 16.

24 BY MS. HARTNETT:

25 Q I'm just asking whether the four competing 03:14:25

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1 models of care is your schema.

2 A I think it borrows from other things in the
3 literature. I wouldn't want to claim, you know,
4 authorship for that per se. It's really hard for me to
5 know where all my ideas come from because I read so 03:14:54
6 much and go to meetings and so forth, and I hear
7 things, and it influences me.

8 I -- I -- it's my summary of -- when we think
9 about what are the options that we can offer to people,
10 this is all I think of. Maybe tomorrow -- 03:15:11

11 Q Okay.

12 A -- I'll think of a fifth option.

13 Q Can you go down to paragraph 53?

14 And this is after you walk through the
15 watchful waiting model, A and B, a psychotherapy model 03:15:25
16 and then the affirmation model and then coming to
17 paragraph 53.

18 MR. BROOKS: Let me just find the heading
19 above it.

20 So we're under the affirmation therapy model 03:15:38
21 number 4, if I'm scanning the --

22 MS. HARTNETT: Yeah.

23 MR. BROOKS: Okay.

24 MS. HARTNETT: That's correct.

25 And then paragraph 53. 03:15:46

1 MR. BROOKS: Okay.

2 BY MS. HARTNETT:

3 Q Out of these four models, you do not know what
4 proportion of practitioners are using which model; is
5 that correct? 03:15:57

6 A Yes.

7 Q Okay. Oh, sorry, I had one question about 49,
8 which was within the psychotherapy model area, if you
9 could flip up to there.

10 MR. BROOKS: Yes, let me just find the heading 03:16:11
11 again so we understand how much material --the
12 psychotherapy model begins at the top of page 18, and
13 you now want to direct us to paragraph 49? Was that
14 the paragraph you mentioned?

15 MS. HARTNETT: Correct. 03:16:29

16 MR. BROOKS: All right.

17 BY MS. HARTNETT:

18 Q And is the psychotherapy model the model you
19 follow, Dr. Levine?

20 A It's the model that I approach new patients 03:16:43
21 with, and depending on the situation of the patient in
22 the family's life, I then go from there. So individual
23 patients, I may counsel the support of the -- I may
24 counsel parents to support the transgender
25 identifications of their child. 03:17:09

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1 But it begins with trying to figure out what's
2 going on here and going on here with the child and the
3 child's history and the parents and their history and
4 the interactions between the -- the parents and the
5 child. 03:17:25

6 So it's not my model for all therapy. As I've
7 said, I think earlier, that I have supported trans care
8 for individuals, affirmative care for individuals. But
9 if you ask me how I begin, I don't not -- I do not
10 begin with the affirmative model. I begin with let's 03:17:44
11 investigate this situation thoroughly so we can
12 eventually make a prudent decision.

13 Q You say in paragraph 49 (as read):
14 "To my knowledge, there is no evidence
15 beyond anecdotal reports that 03:18:01
16 psychotherapy can enable a return to
17 male identification for genetically
18 male boys, adolescents, and men, or
19 return to female identification for
20 genetically female girls, adolescents, 03:18:13
21 and women."

22 Do you see that?

23 A I do.

24 Q And you stand by that statement?

25 A Yes. 03:18:24

1 Q Paragraph 50, this is at the beginning of the
2 affirmative therapy model, on the next page. I think
3 we've already covered this, so we don't need to belabor
4 it, but here, you -- among other things, you say that,
5 under the affirmation therapy model, practitioners -- 03:18:44
6 and I'm going to read from the first sentence. And I'm
7 not reading the whole sentence, but you can obviously
8 read whatever you want. I'm reading from the middle of
9 it. (As read):

10 "...promote and recommend that any 03:18:58
11 expression of transgender identity
12 should be immediately accepted as
13 decisive..."

14 I'm just going to stick on that part, the
15 "immediately accepted as decisive." 03:19:08

16 What is your basis for believing that the
17 affirmation model proceeds with an immediate acceptance
18 as decisive?

19 A Because --

20 MR. TRYON: Objection. 03:19:19

21 Go ahead.

22 MR. BROOKS: Mr. Tryon is objecting.

23 You have to give him time.

24 THE WITNESS: In a previous -- in a -- in a
25 previous portion of this informed consent, I said that 03:19:29

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1 it is my impression that many people in the affirmative
2 model have a number of beliefs that I don't think are
3 scientifically accepted or acceptable or correct and
4 including the fact that this is biologically dictated,
5 that anytime a person, any stage in life, declares a 03:19:52
6 transgender identity, it's because prenatally that was
7 determined and it merely unfolded at a different rate
8 at different times.

9 So the -- the justification for immediate
10 affirmation is based upon this idea, one, that it's 03:20:13
11 biologically dictated; and, two, that it's
12 unchangeable.

13 BY MS. HARTNETT:

14 Q Yeah, I'm sorry, I think -- just given that
15 we're -- have only so much time and I -- I think my 03:20:25
16 question, though, was what was your basis for
17 understanding that the practitioners engage in this
18 practice.

19 MR. BROOKS: Objection; vague as to "this
20 practice. 03:20:36

21 BY MS. HARTNETT:

22 Q Well, the practice of immediate acceptance as
23 decisive.

24 A I think I've already testified how many
25 parents have told me these things and how many patients 03:20:43

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1 have told me these things and -- and -- well, I won't
2 repeat what I began to tell you about.

3 Q No. Thank you. That -- that just helps me
4 connect that that -- that basis of evidence is the same
5 that's at issue here. 03:20:59

6 Paragraph 56, I had a question there.

7 MR. BROOKS: And, Counsel, we should take an
8 hourly break soon.

9 MS. HARTNETT: Now is fine.

10 MR. BROOKS: All right. Now is it -- now it 03:21:14
11 is.

12 THE VIDEOGRAPHER: We are off the --

13 MS. HARTNETT: Come back at --

14 THE VIDEOGRAPHER: Off the record at 3:21 p.m.

15 (Recess.) 03:35:28

16 THE VIDEOGRAPHER: We are on the record at
17 3:36 p.m.

18 MR. BROOKS: And -- and --

19 MS. HARTNETT: Thank you.

20 MR. BROOKS: -- Josh, if you would turn off 03:35:34
21 your camera, you will -- will be able to see the
22 questioner better.

23 There we go. Thank you.

24 MS. HARTNETT: Okay. Great.

25 ///

1 BY MS. HARTNETT:

2 Q Before the break, we were talking, at least a
3 bit, about the four models that you had in the
4 psychotherapy model, and I was asking you if you follow
5 that, and we were having a discussion. And I want to 03:35:54
6 make sure I don't misconstrue your approach.

7 Is it fair to say that you kind of follow the
8 psychotherapy model, but also not to the exclusion of
9 providing medical care or recommending medical care, if
10 it's appropriate, after some course of psychotherapy? 03:36:07

11 A Yes, I -- to summarize, the initial approach
12 to a patient, I believe my model, what I endorse, is an
13 extended evaluation, an opportunity to talk over time
14 in what I call psychotherapy. Other people may call it
15 extended evaluation. And then depending on what I 03:36:34
16 understand about the patient and his or her life and
17 their aspirations and their capacities to understand
18 the present and the future and the past, then I may in
19 fact say, you know, Fine. You know, do what you -- do
20 what you -- use your best judgment. And I will write a 03:36:55
21 letter for you, you know, telling your -- the surgeon
22 or telling the endocrinologist about you.

23 And I do that.

24 Q And was that general approach extended to
25 minors as well? 03:37:17

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1 A Well, if -- if minors are children, I actually
2 have never recommended socialization of a child in
3 that -- that is, in a new gender. I have seen -- I
4 have never recommended that.

5 When it comes to teenagers, the closer they 03:37:36
6 get to 18, the more I'm willing to talk to them about
7 the possibility of hormones and being supportive of it
8 after a certain period of time.

9 When it comes to older people, it's -- it's
10 not as broad a question. 03:37:57

11 Q And how long is your -- when you discuss an
12 extended evaluation, how -- how long is that?

13 A It doesn't have a definable length.

14 Q Is there -- and I'm just trying to really
15 understand. Is it a matter of hours, days or longer? 03:38:13

16 A It's certainly -- a -- a psychotherapeutic
17 hour is typically one; right? But when people come to
18 Cleveland for an evaluation, I often spend two days.
19 And so I may spend, you know, four hours over two days
20 or maybe even more with a patient and then separately 03:38:37
21 with their parents and sometimes together with their
22 parents.

23 But when I'm talking about an extended
24 evaluation, I mean that in two terms. One is for
25 people who want to come for an intense evaluation that 03:38:53

1 at the end of two days will give some -- give some
2 feedback to them and -- but the usual sense for people
3 who live in Cleveland, where I reside, that is over
4 weeks and months of talking over time, considering
5 various -- the things I've already articulated. 03:39:14

6 Q Have there been situations where after the
7 sort of intense extended evaluation, the two days
8 and -- four hours over two days period, where you've
9 supported or recommended any medical treatment after
10 that period? 03:39:33

11 A Well, about -- about a year ago, a -- a --
12 a -- a college student who wasn't doing very well, who
13 got actually hormones on a one-hour visit, to the
14 student health service, the -- we recommended that the
15 patient could decide whether to continue hormones or 03:39:53
16 not. The parents did not want the person to continue
17 hormones, and the patient continued hormones. And we
18 just made a recommendation. We thought there was an
19 advantage to stopping and reconsidering life, but it
20 was the patient's choice, you see. It wasn't the 03:40:12
21 parents' choice. It wasn't my choice, you see. But
22 it's the respect for the patient's autonomy.

23 Q Did you write a letter there or some sort of
24 authorization for him to get the hormones?

25 A No. He already had the hormones. As I said, 03:40:29

1 he got the hormones after one hour with a person who
2 knew nothing about his background, really, that -- what
3 I would say, relatively nothing.

4 Q Where was that treatment?

5 A That was at the University of Rochester. 03:40:40

6 Q Okay. So -- and then my question is just for
7 kind of -- I guess, what's the shortest period of
8 extended evaluation that you've performed after which
9 you've written a letter for someone to get transgender
10 medical care? 03:41:00

11 A I'm going to elaborate your question into me
12 or my staff because in some --

13 Q Thank you.

14 A It's a whole -- it's a committee of work, a
15 group of people. 03:41:13

16 I would say four hours.

17 Q Thank you. You had mentioned your -- the
18 recently published article about the -- the
19 reconsidering informed consent piece; correct?

20 A Yes. 03:41:32

21 Q And in there, you note that -- kind of --
22 you're talking about the affirmation -- what you
23 characterize as the affirmation approach; right?

24 A Correct. There's a section on that, yeah.

25 Q And then you note that the "research about 03:41:46

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1 alternative approaches, such as psychotherapy or
2 watchful waiting, shares the scientific limitations of
3 the research of more invasive interventions; there are
4 no control groups, nor is there systematic follow-up at
5 predetermined intervals with predetermined means of 03:42:03
6 measurement."

7 Does that --

8 A Yes.

9 Q Is that something you have in the article?

10 A I think I made the same point in -- in this 03:42:10
11 document that I gave to you.

12 Q Right. I was just trying to connect the two.

13 So that's basically the same point you've been
14 making, that -- the kind of lack of evidence, from your
15 perspective, as to which approach is kind of 03:42:26
16 scientifically based; is that right?

17 A Yes.

18 Q Okay. If we could flip forward, I -- sorry,
19 going backward for a minute and then we'll go forward
20 again, in your declaration, but I had a question about 03:42:36
21 paragraph 18, little L. Sorry, that's not right. It
22 is 18, little -- sorry, one second.

23 I'll try again.

24 Can I direct your attention to paragraph 18H,
25 on page 7? 03:43:03

1 MR. BROOKS: And let me just first start on
2 the top of 18 so we know what the major proposition
3 here -- a summary of key points. All right.

4 And, I'm sorry, you said H?

5 MS. HARTNETT: Correct. 03:43:18

6 BY MS. HARTNETT:

7 Q So I'm going to direct your attention to
8 paragraph H, on page 7, which you talk about
9 administration of puberty blockers not being a benign,
10 quote, pause of puberty. 03:43:31

11 Do you see that?

12 A I do.

13 Q And this, I noticed, was something newly added
14 to this declarations from the one that you had
15 submitted at the preliminary injunction stage. 03:43:42

16 My question for you is what the basis is for
17 your qualification, in your perspective, to talk about
18 the effects of puberty blockers.

19 MR. BROOKS: Object to the form of the
20 question. 03:43:57

21 THE WITNESS: What is the basis of my
22 objection to the use of puberty blockers?

23 BY MS. HARTNETT:

24 Q Sorry, the basis for your understanding of
25 whether -- how they function on the body and whether 03:44:04

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1 they're a benign pause of puberty or not.

2 A The initial justification for puberty blockers
3 being a benign thing is that it merely was a pause and
4 that if it was fully reversible, puberty would -- would
5 return when puberty blockers were removed, if they were 03:44:32
6 chosen to be removed.

7 I often reacted to that word "pause" because I
8 was aware that I was unaware of the rich biological
9 details that puberty changes every organ in the body.
10 Puberty not only causes growth of bones, but puberty 03:44:53
11 causes growth of the liver, of the lungs, of the heart,
12 of the brain. You name the organ, and the pubertal
13 changes are occurring, and they occur in a sequence.
14 And one of the developmental aspects of development is
15 that there are windows of opportunity for development, 03:45:15
16 and when the window closes, we're not sure whether
17 things can be totally reversed.

18 And I noticed that there was a benign
19 connotation to the word "pause" which did not strike me
20 as true or possibly true or certifiably true. 03:45:35

21 And so I began looking at various statements
22 from various authors about saying this.

23 And in the early years, people talked about
24 complete reversibility and it's only a pause, but I
25 realized, in reading their subsequent sentences, that 03:45:56

1 they didn't consider -- they were talking about bone.
2 They were talking about the onset of puberty. They
3 weren't talking about the subtle changes of -- of, say,
4 for two or three years of interfering with the
5 processes that were naturally happening in your and my 03:46:11
6 children and the children of society.

7 So -- and then I looked closer at it, and I
8 said, what about the impact, the psychological, social,
9 sexual impact of having one's peers have these major
10 changes in every aspect of their body while the person 03:46:31
11 was paused in a puerile state, has anyone considered
12 that when they said it's completely reversible.

13 Nowadays, I think people are not certain it's
14 completely reversible, and they're beginning to
15 articulate the possibility that I just articulated. 03:46:53

16 They're beginning to say we don't know what
17 the psychosocial impact of being puerile while your
18 peers are pubertal.

19 And while your peers are pubertal, you're
20 getting -- you're starting to deal with your sexual 03:47:10
21 feelings and your sexual conflicts, and you're getting
22 to operationalize your -- what the early orientation
23 aspects of early puberty are, you see. And the puerile
24 child is not.

25 And so I thought the word pause was a kind of 03:47:23

1 rhetoric that -- that justified doing something that
2 was much more complicated and had not been articulated
3 well by the people who began using it.

4 I'm not sure that today's people are talking
5 in the same way that they did when -- 20 -- ten years 03:47:41
6 ago.

7 Q When did you come to --

8 A I think they're more sophisticated today.

9 Q When did you come to this understanding or
10 view about the -- your kind of concern with using the 03:47:53
11 term "pause"?

12 A I think it's been evolving in my mind over the
13 last two or three years.

14 Q Do you know whether the pubertal response
15 would be the same -- basically, if the puberty blockers 03:48:06
16 were used and then a child were to go off the puberty
17 blockers, do you know whether it would be the same
18 pubertal response that would have been had without the
19 blockers?

20 A Well, I think endocrinologists have said that 03:48:21
21 it's same, but I don't know if they have even the -- I
22 don't know that -- I don't know that I trust that
23 they're right about that. I don't know that they're
24 wrong. I just don't know that they're right. Because
25 in concepts of development -- for example, if you 03:48:43

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1 don't -- if you don't hear at a certain stage in life,
2 say the first two years of life, and even if we do a
3 cochlear implant, and we put -- we -- you can hear
4 starting at age three or age four or age five, you
5 can't speak as clearly as you and I can speak. 03:49:01

6 So, you see, there's a window of opportunity
7 when the brain is changing and we -- it's -- that --
8 that other -- other aspects of life develop. And I
9 think this is probably true throughout life as a
10 principle. 03:49:18

11 So the idea that, oh, we can give a kid for
12 three years or four years and keep them paused while
13 they decide what they want to do, whether they want to
14 go cross-sex hormones or not, and then if they decide
15 not to go the cross-sex hormone route, that they will 03:49:33
16 just go into puberty and everything be normal, I just
17 think that's a naive idea. But I was proposing that,
18 you see. I can't prove it and either can -- either can
19 the endocrinologist prove it. That's my point.

20 Q Thank you. 03:49:47

21 MS. HARTNETT: I've put in the "Marked
22 Exhibits" folder Exhibit 88. If you -- your counsel
23 could look at that.

24 Let me know if you see that.

25 (Exhibit 88 was marked for identification 03:50:05

1 by the court reporter and is attached hereto.)

2 MR. BROOKS: I do see it now.

3 BY MS. HARTNETT:

4 Q This is -- Dr. Levine, do you see -- this is
5 testimony that you gave to the Pennsylvania legislature 03:50:11
6 in March of 2020.

7 A Okay.

8 Q Do you recall giving this testimony?

9 A I recall testifying, yes.

10 Q Okay. I'm -- I have a question that -- you 03:50:19
11 had your kind of prepared remarks, and then you got
12 some questions from the legislators, and what I would
13 like to do is ask you about something on page 61, which
14 was your response to a question about puberty blockers,
15 if you could page forward to 61. 03:50:33

16 MR. BROOKS: Will you direct us to the
17 question?

18 Let me see here. I -- I --

19 MS. HARTNETT: Okay. If -- yeah. It's a
20 question from Representative Zimmerman, and it's asking 03:50:47
21 about the reversibility of puberty blockers, on
22 page 61.

23 MR. BROOKS: Oh, the question on 61 is
24 fragmentary; right?

25 "If puberty blockers are started," is that the 03:51:06

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1 question you're referring to?

2 MS. HARTNETT: You can feel free to look
3 above, but I'd like to ask about the passage on 61.

4 He asked a two-part question, and he had then
5 asked to be reminded about the second part of the 03:51:21
6 question.

7 And Representative Zimmerman said, "Yes. If
8 puberty blockers are started."

9 And then Dr. Levine said, "Oh, reversible,
10 yes, sorry." 03:51:30

11 And what I'd like to ask him is to read this
12 passage -- hear his testimony and just whether he
13 continues to believe what he's testified to.

14 THE WITNESS: I've read the paragraph.

15 MR. BROOKS: The -- 03:52:06

16 BY MS. HARTNETT:

17 Q I guess, just --

18 MR. BROOKS: Just continue --

19 THE WITNESS: Oh, you want me to continue?

20 MR. BROOKS: I want you to read to the end of 03:52:11
21 that answer.

22 MS. HARTNETT: Correct. Thank you.

23 THE WITNESS: Okay.

24 BY MS. HARTNETT:

25 Q Do you stand by the testimony that you gave in 03:52:32

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1 these two paragraphs?

2 A I don't see a -- a major difference between
3 what I just said to you except -- than what I said
4 here. Here, I was talking about one year. And -- and
5 it depends on -- you know, if you give a puber- -- an 03:52:51
6 eight-year-old child a puberty blocker versus a
7 nine-year-old child versus a 14-year-old child. I
8 think we're talking about different phenomenon, you
9 see. The -- not only biologic phenomenon, but
10 psychosocial phenomenon. Because if you give it to an 03:53:09
11 eight-year-old, their peers are still puerile, you see.
12 And -- and when -- if you give it to 14-year-old or a
13 12-year-old, their peers are rapidly growing and
14 changing and being involved in all kinds of
15 psychosocial and -- processes that -- that a 03:53:23
16 nine-year-old is not, the eight-year-old is not.

17 So I think today's testimony elaborates upon
18 what I was saying in a less sophisticated way to
19 Mr. Zimmerman.

20 Q Thank you. You talk about desistance at 03:53:37
21 length in your report; correct?

22 A I hope so, yes.

23 MR. BROOKS: Counsel, do you want me to take
24 down 88 or leave it up?

25 MS. HARTNETT: You can take down 88. 03:53:50

1 BY MS. HARTNETT:

2 Q Do you believe that desistance should be the
3 goal of treating patients with gender dysphoria?

4 A I think I previously stated that the goal of
5 treating gender dysphoria is to have an informed 03:54:05
6 consent process in a brain -- for a person whose brain
7 is old enough to consider the possibilities about the
8 risks, and the goal of -- of their gender expression
9 has to rely primarily on them and their process of
10 coming to grips with what it needs, not just in 03:54:24
11 fantasy, but in reality, for them to portray themselves
12 as a trans person.

13 So I don't -- your question has previously
14 been answered by me. Parents would very much like me
15 to be able to return their child efficiently and 03:54:44
16 quickly to a tran- -- to a cis state, but I can't
17 promise that as a goal. I can't even hold that out as
18 a goal. What I hold out is what I just said to you.

19 Q If you could -- you -- so you don't believe
20 it's possible to talk somebody out of being 03:55:08
21 transgender; is that fair?

22 MR. BROOKS: Objection.

23 THE WITNESS: It's not the language that I
24 would ever use. I don't talk people out of things. I
25 don't talk people out of getting married to a person. 03:55:22

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1 I don't talk people out of going to this college versus
2 that college.

3 I -- I -- I sort of elicit their feelings. I
4 help them see where there is conflict. I help them
5 articulate the pluses and minuses, as we can predict 03:55:38
6 the future. I look at trends.

7 I don't talk people out. It's not what a --
8 what Dr. Levine, the psychiatrist, does, talk people
9 out of X, Y or Z. And Z may be transgender identity.

10 Q If you could treat everyone to have them cease 03:55:58
11 being transgender who -- sorry.

12 For the transgender patients you have, if you
13 were able to treat them such that they would no longer
14 be transgender, would that be your preferred outcome?

15 MR. TRYON: Objection. 03:56:19

16 THE WITNESS: It depends what cost it would
17 have to be -- to return to living as a cisgender
18 person. It would not be my goal if it would cost them
19 their sanity, for example, if it would cost them
20 continued anguish. My goal is -- is stated to -- I've 03:56:38
21 already stated my goal.

22 The -- there is a belief that life is hard
23 enough as a cisgender person, you see. But these
24 things -- you see, I -- I -- I'm interested in what it
25 is about being a cisgender person that is so hard for 03:57:08

1 you, you see. Why is it that this is so difficult for
2 you. What is it about femaleness or maleness or
3 your -- your -- your sex, your original sex, you know,
4 your sex, what it is about it that is so offensive and
5 offending to you. Why is there such incompatibility. 03:57:30
6 Tell me. Teach me.

7 Q But using the language from your -- at least
8 your declaration earlier in the case where you had
9 described, you know, the -- the risks and harms that
10 would come from, quote, putting a child or adolescent 03:57:44
11 on the pathway towards life as a transgender person --
12 I'm just trying to understand if -- if you, Dr. Levine,
13 could put all the young people that were experiencing
14 gender dysphoria on a pathway toward being
15 non-transgender, would you do that? 03:57:59

16 A What I would say about that, if I could put
17 them on a pathway of being non-transgender, I would
18 expect that the vast majority of them would end up to
19 be homosexual in their orientation. And the
20 cisgender with -- you know, if they were males, they 03:58:16
21 would probably be cisgender with a little feminine
22 aspects to them, but they would be homosexual. And if
23 they were biologic females, they would be cisgender
24 lesbians with a little touch of masculine patterns and
25 so forth. 03:58:35

1 So that would be cisgender to me, but I
2 wouldn't be cisgender heterosexual. I think we already
3 know scientifically the outcome of gender atypicality.
4 Cross-gender atypicality in boys and girls is
5 homosexual orientation. 03:58:52

6 Q Is it your opinion that it's better to be a
7 cisgender homosexual than a transgender heterosexual?

8 MR. BROOKS: Objection to the form of the
9 question.

10 THE WITNESS: Well, you do no harm to your 03:59:09
11 stability. You do no harm to your anatomy. You do no
12 harm to your physiology. In that sense, I think -- you
13 don't -- you don't risk any of the complications of
14 cross-sex hormones, and you don't risk any of the
15 complications of surgery. And I think it's probably -- 03:59:24
16 although I can't tell you the facts, but I do believe
17 it's probably easier to be a gay person in society than
18 to be a trans person. And I don't mean it's easy to be
19 any sexual minority in our society.

20 BY MS. HARTNETT: 03:59:43

21 Q Do you know what autogynephilia is?

22 A I -- I didn't understand what you just said.

23 Q Apologies. Do you know what autogynephilia
24 is?

25 A Yes. 03:59:56

1 Q What is autogynephilia?

2 A Well, "autogynephilia" is a word that means
3 love of the self as a woman. It's a characteristic of
4 internal life that was popular in the trans literature,
5 beginning in about 1988. It was a concept suggested by 04:00:11
6 Ray Blanchard of Toronto. It was a supposition that --
7 that autogynephilic trans people had a form of
8 paraphilia and that it -- I think it was a concept that
9 replaced pretty much the concept of fetishistic
10 transvestism that had existed since the 1900s, early 04:00:44
11 1900s.

12 So at about -- the trans community objected to
13 the idea of autogynephilia, very profoundly objected to
14 the idea.

15 Anne Lawrence, who is a transsexual 04:01:06
16 researcher, wrote a book on men who are trapped in
17 men's bodies, and it was all about gyne- --
18 autogynephilia, men who -- who recognized that they
19 were autogynephilic.

20 I recently had a patient who came to see me 04:01:15
21 because he couldn't find anyone who knew anything about
22 autogynephilia.

23 But I think you don't find that word used in
24 the literature -- in the modern literature anymore.
25 Because I think with 2011 standards of care, there was 04:01:29

1 much less interest in the pathways to transgenderism
2 and more interest in the treatment of transgenderism,
3 and so it became too many advocates, politically
4 irrelevant and obnoxious to -- to even use the term
5 "autogynephilia." 04:01:55

6 Q Do you find autogynephilia to be a helpful
7 concept?

8 A For some people.

9 Q Have you ever heard it said that transgender
10 people are either gay, mistaken or have autogynephilia? 04:02:06

11 MR. BROOKS: Objection.

12 THE WITNESS: I don't recall hearing that
13 sentence before.

14 BY MS. HARTNETT:

15 Q Do you think that that -- is that something 04:02:19
16 that you would agree with, that being transgender --
17 people think that transgender are either gay, mistaken
18 or have another malady, like autogynephilia?

19 MR. BROOKS: Objection.

20 THE WITNESS: It's not something that I would 04:02:32
21 summarize by saying. Those three options seem
22 pejorative and unscientific.

23 BY MS. HARTNETT:

24 Q Do you think the term --

25 A I'm sorry, I -- I object to the idea of 04:02:50

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1 mistaken.

2 Q Do you think the term or that use of
3 autogynephilia is obnoxious?

4 A No.

5 Q Do you think that being transgender is a 04:03:07
6 paraphilia?

7 MR. BROOKS: Objection.

8 THE WITNESS: To the extent that -- to the
9 extent that autogynephilia is a paraphilia and that
10 some men develop a transgender identity as a 04:03:18
11 consequence of autogynephilic behaviors, that was --
12 that may be one pathway towards transgender identity.

13 But I wouldn't certainly -- I -- I certainly
14 would not say that at all transgenders or most
15 transgendered people are autogynephilic. 04:03:38

16 BY MS. HARTNETT:

17 Q I mentioned the -- one possible formulation
18 that people that are identifying as trans are just gay,
19 mistaken or have a malady like autogynephilia, and I
20 think you said that you took issue with the notion of, 04:03:55
21 among other things, the idea of it being a mistake; is
22 that fair?

23 A I -- yeah, I take -- I take issue with that,
24 yeah.

25 Q Why? 04:04:05

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1 A A mistake is something that a patient decides
2 after they've trans- -- detransitioned and they say it
3 was a mistake to do that.

4 It's not something I would say. I would say
5 that they -- they have a current gender identity, and 04:04:21
6 I'm not sure they're -- I'm not sure anyone's gender
7 identity is not going to evolve in some way in the
8 future. Especially I would like to say that about
9 young adolescents.

10 But please don't -- please don't quote me 04:04:38
11 because I have never authored that sentence.

12 Q Thank you. Do you think that transgender
13 identity is something that can be cured?

14 A Can be cured?

15 Q Yeah. 04:04:54

16 A Is that what you said?

17 MR. BROOKS: Objection.

18 BY MS. HARTNETT:

19 Q Cured.

20 A If you read the end of my paper on the patient 04:05:02
21 who trans- -- detransitioned 30 years ago, I think I
22 said something like even though medical psychiatric
23 knowledge does not know how to transform a person from
24 a trans state to a cis state or a previous state, it
25 doesn't mean that life doesn't transform people into 04:05:25

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1 detransitioned people.

2 We need to understand the modesty and the
3 differences between what we know how to do to create
4 behavioral change, which is quite modest throughout
5 psychiatry and what happens to people over time if we 04:05:44
6 take a life course perspective.

7 So my case illustration in that case was
8 Dr. Levine did not change his -- did not cause his
9 detransition at all; right? Life processes, which he
10 described in great detail in the that paper, changed, 04:06:02
11 and it took him years to make that change, years of
12 anguish, years of the sense of inauthenticity as a
13 woman, which at first he tried to deny.

14 So I would -- I would refer you to the last
15 paragraph in that paper if you wanted to find out how I 04:06:22
16 said it. I can't -- I can't quote it. I'm just
17 paraphrasing it if for you.

18 Q But is that an example of someone that you
19 think was cured?

20 MR. BROOKS: Objection. 04:06:41

21 THE WITNESS: It was an example of a person
22 who changed their presentation and now is terribly
23 embarrassed about what he had -- I can call him "he"
24 now -- what he had done, or what she had done; right?

25 And now -- and it is now a person who -- I think I'm 04:06:57

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1 quoting -- hates all the advocates of the -- in the
2 trans world for, he believes, misleading people that
3 they can have a happy life.

4 But that's just one person's opinion, you
5 know. 04:07:13

6 But if you read the paper, I think, you know,
7 there's lots to think about in the paper.

8 Q Is it embarrassing to be transgender?

9 A In -- in some settings, it probably is, yes.

10 Q Do you think that transitioning, for a 04:07:28
11 transgender person, is something that you find to be an
12 embarrassing concept?

13 A No.

14 Q Well, you said that your -- I'm just -- I'm
15 not putting your patient's words in your mouth, but you 04:07:38
16 were describing him as having been embarrassed by the
17 whole thing. I -- I took that to mean he was
18 embarrassed by having transitioned; is that right?

19 A Yes, he's now angry at himself and angry at
20 those who facilitated his original transition. 04:07:52

21 But that's one person, you know.

22 Q But do you feel embarrassment for your
23 patients that have to go through transition?

24 MR. BROOKS: Objection.

25 THE WITNESS: Do I feel embarrassment? No. I 04:08:09

1 feel --

2 BY MS. HARTNETT:

3 Q I'm just --

4 A No. That's -- that would not describe a

5 dominant feeling I have. I have concern for my 04:08:20

6 patient. I have worry about this, but I'm not

7 embarrassed by it.

8 Q Is shame one of the feelings?

9 MR. BROOKS: Objection.

10 Of whom?

04:08:35

11 BY MS. HARTNETT:

12 Q Do you (technical difficulty) shame for them?

13 MR. BROOKS: Objection.

14 THE WITNESS: I'm a little hard of hearing,

15 and I actually could not discern what you said. 04:08:43

16 BY MS. HARTNETT:

17 Q Sorry, I'll speak up.

18 I was asking if you felt shame for your

19 patients experiencing transition.

20 A No, I'm not -- am I ashamed? 04:08:52

21 Q Yes.

22 A No.

23 Q Do you think that people can change their

24 sexual orientation?

25 MR. BROOKS: Objection; outside the scope of 04:09:10

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1 this witness's testimony.

2 THE WITNESS: I think the work of Lisa Diamond
3 has demonstrated that among women who are -- who assert
4 a lesbian identity, that that lesbi- -- there is a lot
5 of two-way traffic between a heterosexual identity and 04:09:43
6 a homosexual identity, or orientation, we would say,
7 and -- so I don't know how to change a person's sexual
8 orientation, but I do think, especially among natal
9 women, sexual orientation is -- people experiment with
10 different ways of life and that there are -- there's 04:10:06
11 more two-way traffic between lesbian and a heterosexual
12 life among women. There's much more bisexual behavior
13 and bisexual eroticism among natal born females than
14 there is among natal born males.

15 So that would be my answer to your question, 04:10:29
16 without a yes-or-no answer.

17 Q Do you agree that gay people, on average, have
18 a harder time than straight people, on average, just
19 navigating life?

20 A Yes. 04:10:40

21 MR. BROOKS: Objection.

22 BY MS. HARTNETT:

23 Q Do you have similar views to those you've
24 expressed about caution before encouraging youth to be
25 transgender -- or to inhabit their transgender gender 04:10:51

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1 identity? Do you have similar views about youth
2 expressing homosexuality?

3 A No.

4 Q Why not?

5 A Well, again, I think I'm going to make a 04:11:03
6 distinction between homosexuality as it occurs in men,
7 as it occurs in women, and the eroticism of a person is
8 a bunch of fantasies and thoughts and attractions that
9 makes sex comfortable or anxious and makes romance easy
10 or hard to -- to participate in, and given the power of 04:11:37
11 orientation, I believe that people have to come to
12 grips with -- with who they are attracted to and -- and
13 what is easy for them and what is difficult for them.

14 And so I just think that that's part of the
15 human landscape and that people can -- can -- they 04:12:03
16 know -- they know their orientation, and then they have
17 to choose how -- how to act or not act on their
18 orientation, and it's a very personal, private and
19 often difficult decision, and I respect that, and I'm
20 happy to hear about it when it comes up in my gay 04:12:23
21 patients.

22 And, you know, I see a lot of people who have
23 orientations that are not heterosexual.

24 Q I'm just curious why the same principle
25 doesn't hold for people that have a gender identity of 04:12:37

1 transgender, if they have an innate sense that that's
2 their identity, why would you not approach that the
3 same way you approach homosexuality.

4 MR. BROOKS: Objection.

5 THE WITNESS: Because homosexuality does not 04:12:51
6 involve the -- it's not against the first principle of
7 medical ethics; above all, do no harm.

8 It doesn't involve changing the body's
9 reproductive capacity. It doesn't change the body's
10 sexual physiology, you see. It doesn't change the 04:13:08
11 ability to find a love partner, a stable mate. It --
12 it -- it doesn't -- trans- -- we're talking about here
13 changing the anatomy, changing the physiology, creating
14 the inability to have a child, interfering with the
15 ability to have sexual pleasure as we understand it in 04:13:32
16 the general population as, you know, orgasm.

17 So -- so we understand -- transsexuality is
18 exposing yourself to surgical complications. And
19 surgical transformation of a teenager, before a child
20 has lived long enough to -- to come to grips with the 04:13:51
21 multiple dimensions of being an older person, that is,
22 a 20-year-old or a 19-year-old, and romance and so
23 forth, that's why it's different. It's not the same.

24 You're trying to take a principle and -- and
25 apply it to a group of people that -- that you're 04:14:10

1 talking about the possibility of harming them. Not
2 just their -- their -- their reproductive capacity, but
3 harming them in numerous ways. And they have to take
4 responsibility for that choice, and they -- I just have
5 been saying all morning and all afternoon, I just want 04:14:29
6 them to be informed.

7 And, you know, 13-year-old passionate kids
8 cannot be informed easily.

9 Q I'm glad you brought that up.

10 Could you turn to paragraph 202 of your 04:14:49
11 declaration, page 69.

12 MR. BROOKS: Yeah. And it was long. I didn't
13 think it was that long.

14 Page 69. Let's see here.

15 You said 202. Yes, we have that on the 04:15:13
16 screen.

17 BY MS. HARTNETT:

18 Q Yeah, I wanted to ask you, these are within a
19 larger section, well, about various harms that come
20 from, I guess, treating or -- or validating a 04:15:26
21 transgender person's identity. But this paragraph 202
22 talks about harm to family and friendships, and then
23 203 talks about sexual-romantic harms.

24 Do you see that?

25 A Yes. 04:15:41

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1 Q And my question is, the harms you set forth in
2 these paragraphs -- first of all, you cite your -- only
3 your own publications for these two paragraphs; is that
4 correct?

5 A Yes, it's my only citation. 04:15:49

6 Q Is there any other basis for these assertions?

7 A Well, there's an article in the Archives of
8 Sexual Behavior about being the fetish object, when --
9 a transsexual adult talking about -- a survey of
10 transsexual adults, that they get really upset that 04:16:10
11 people want to have sex with them because they're what
12 they call a fetish object, that they're -- they -- they
13 have attractions to transsexuals and they want to have
14 an experience.

15 And so it's really about the frustration of 04:16:25
16 adult tran- -- sexually active transsexual, I think --
17 transsexuals who are complaining about difficulties in
18 romantic relationships because they feel they're being
19 used by people with perverse adventures, some
20 curiosities, as opposed to genuine romantic 04:16:47
21 relationships.

22 So I was happy to read that article because it
23 had confirmed one of the stories that I had been
24 hearing from many patients over the years by --

25 Q Can you direct me -- 04:17:00

1 A Sorry.

2 Q What article is that? Can you direct me --

3 A I -- I certainly can get you the reference.

4 It's in the Archives of Sexual Behavior. It's probably

5 within the last two years. And I think the first 04:17:13

6 author's name is either -- starts with an A, B or C.

7 Anyway, I -- you -- it's about tran- -- in the

8 title, there's something like "transgender and fetish

9 objects." So I --

10 Q Okay. 04:17:38

11 A I can -- if you want, I will eventually give

12 you the exact reference, yeah, but --

13 Q That's --

14 A -- you're -- you're not interested in wasting

15 time, I'm sure. 04:17:48

16 Q No, no, I -- I -- I just want to know the

17 basis for these -- these paragraphs, so I appreciate

18 you telling me that.

19 My question is -- you know, I read 202 and

20 203, and you say -- you list various perceived harms 04:17:58

21 and challenges from being transgender; is that fair?

22 A Yes.

23 Q What I'm confused about is, is this premised

24 on the notion that there's a way to dissuade someone

25 from being transgender so that they don't have these 04:18:14

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1 outcomes?

2 A Exactly. I -- this is what I'm trying to do.

3 This is why I say to parents, you know, we have to

4 support and love this child regardless of what --

5 what -- what they pass through because mental health is 04:18:35

6 determined, in part, by the ability to -- to be valued

7 by your family before you can be valued by other

8 people.

9 And I think the outcomes -- I mean, so many of

10 my patients have in fact been alienated from their 04:18:53

11 families. And -- sorry -- you've heard about runaway

12 kids and throwaway kids and -- and I --

13 Q Well, why isn't -- sorry, why isn't that

14 the family's --

15 MR. BROOKS: Counsel -- Counsel, the witness 04:19:08

16 is busy talking, in the middle of his --

17 MS. HARTNETT: Yeah, I'm aware of that, but

18 he's also taking a long time to respond to

19 straightforward question.

20 BY MS. HARTNETT: 04:19:18

21 Q My question is whether or not --

22 MR. BROOKS: Counsel, the witness is entitled

23 to finish his answer.

24 MS. HARTNETT: He's not entitled to

25 filibuster. 04:19:23

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1 MR. BROOKS: He's not filibustering; he's
2 answering your question.

3 MS. HARTNETT: I've been very permissible all
4 day with his answers, but I'm happy to have him finish
5 his answer. 04:19:35

6 MR. BROOKS: Thank you.
7 If you have -- if you feel that you haven't
8 finished, you may finish.

9 THE WITNESS: I have heard considerable
10 stories over the years about family relationships, 04:19:43
11 about alienations, about isolation. And in answer to
12 your question, in -- in hearing those stories, it has
13 led me to counsel both the patient and the parents to
14 do whatever they can to maintain their relationships,
15 despite what the child or the grownup, the adult, has 04:20:02
16 decided because I know the suffering of mothers and
17 fathers and grandmothers and grandfathers and of
18 patients.

19 And so it's an adverse outcome to have family
20 alienation. And from the very beginning, I say the 04:20:19
21 first principle evaluation is to preserve family
22 relationships, and I think you can read that in my 2021
23 paper.

24 BY MS. HARTNETT:

25 Q My question is -- so in the example of the 04:20:31

1 child who's -- or the adolescent who's experiencing
2 gender dysphoria and would like to be affirmed and the
3 parents that are horrified, why isn't the answer to try
4 to work with the parents to be more tolerant and
5 understanding rather than to try to change the child? 04:20:48

6 A I think I do work with the parents. I do.
7 But it's not an either-or thing. It's not an either-or
8 phenomenon.

9 And just because --

10 Q Is your -- 04:21:07

11 A Just because we work with a parent doesn't
12 mean I'm capable of changing the parent's behavior,
13 changing the parent's values, changing the parent's
14 knowledge of the child and changing the parent's fear
15 for their future. 04:21:22

16 Q I'm just puzzled by these paragraphs because
17 it strikes me that the person is going to be
18 transgender regardless if they get transgender
19 healthcare and, therefore -- I don't understand the
20 point that giving them healthcare is going to harm them 04:21:37
21 more than they would have otherwise been harmed if they
22 were transgender, but just without healthcare.

23 MR. BROOKS: Objection; assumes facts not in
24 evidence, argumentative.

25 THE WITNESS: I accept the fact that you don't 04:21:47

1 understand.

2 BY MS. HARTNETT:

3 Q Can you explain to me why -- so, I guess --
4 let me ask you this: Do you disagree that these people
5 are transgender even if they don't get the healthcare? 04:21:56

6 MR. BROOKS: Objection.

7 THE WITNESS: I agree that the patient who
8 says that "I'm transgender" is currently transgender.
9 That's what I believe. They're currently transgender.

10 Do I believe they will always be transgender? 04:22:14

11 No.

12 Can I predict which ones will be transitioned
13 and not? Not -- not with any certainty, no.

14 But, you see, I believe that many of the
15 assumptions behind your questions is that 04:22:28
16 transgenderism is a fixed phenomenon, it never changes,
17 and I -- if I am correct that that is your assumption,
18 then you and I disagree.

19 BY MS. HARTNETT:

20 Q And do you agree that there's no evidence 04:22:44
21 to -- assuming those are different assumptions, that
22 there's not evidence out there that would prove either
23 of us correct on that one?

24 MR. BROOKS: Objection.

25 THE WITNESS: No, I don't agree with that at 04:22:53

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1 all. Not at all.

2 BY MS. HARTNETT:

3 Q Do you believe that --

4 A I -- and -- and I give you evidence of

5 detransition. 04:22:59

6 Q Is there anything other than anecdotal
7 evidence to say whether or not gender identity is fixed
8 versus not labeled?

9 MR. TRYON: Objection.

10 THE WITNESS: You know, you and I have 04:23:13
11 different ideas of what is anecdotal.

12 Is Lisa Diamond's work anecdotal, about
13 homosexuality? Is that anecdotal?

14 And -- and, you know, there is something
15 called a proof of concept study that if you can 04:23:29
16 demonstrate that it is possible, for example, to cure a
17 particular cancer with a new drug that has never been
18 tried before, that proof of concept then leads to more
19 definitive studies.

20 And we're in -- we're -- we already have proof 04:23:47
21 of concept that -- that there are many people who
22 detransition.

23 In fact, if you look at the UK studies, the
24 two UK studies that have been done in the last, I
25 think, six months, we all now have a rate of 04:24:07

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1 detransition. We now, for the first time, have a rate
2 of detransition data.

3 And so I would say it's not anecdotal.
4 It's -- it's an emerging new branch of transgender
5 science, so to speak, or knowledge that the error rate 04:24:24
6 in trans -- in -- in -- in affirmative care is now
7 becoming more clear than it ever was.

8 Q You are aware that some transgender -- many
9 transgender people have fulfilling romantic
10 relationships and family relationships; correct? 04:24:37

11 MR. BROOKS: Objection.

12 THE WITNESS: I am aware.

13 BY MS. HARTNETT:

14 Q In paragraph 203, you say (as read):
15 After adolescence, transgender 04:24:47
16 individuals find the pool of
17 individuals willing to develop a
18 romantic and intimate relationship
19 with them to be greatly diminished."

20 A Yes. 04:24:57

21 Q Do you have any basis for making that
22 statement other than your own anecdotal experience?

23 A Well, if you look at -- if you look at
24 cross-sectional data about the percentage of people who
25 are married and cohabitating among trans people versus 04:25:09

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1 cis people, there are -- there are far less marriages,
2 and there are far less stable relationships.

3 If you look at a series of psychosocial
4 histories of -- of patients, many of them do not come
5 to us with stable functional relationships. I don't -- 04:25:31

6 Q You --

7 A I actually -- I actually don't think this
8 is -- this is anecdotal, but it is perhaps
9 impressionistic based upon 50 years of taking care of
10 these people. 04:25:50

11 Q Is it possibly also dated?

12 MR. BROOKS: I'm -- I'm sorry, I couldn't hear
13 the question.

14 BY MS. HARTNETT:

15 Q Is the notion also possibly dated? 04:25:57

16 A Well, the big hope in the trans advocate
17 community has been as society improves, the lives --
18 society recognizes and accepts transgender people,
19 there will be less suffering and less isolation in
20 trans people. That -- that is -- you can find that in 04:26:15
21 many, many studies that -- that articulate the -- the
22 frequency of psychiatric problems. And there's the
23 hope that as -- the whole idea of the minority stress
24 theory is that if we improve society, fewer people will
25 suffer. 04:26:40

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1 I don't know whether that -- I hope it's true
2 that as society has improved its defense of -- of
3 gender diverse people, that more gender diverse people
4 will be able to have satisfying, intimate, stable
5 relationships. I hope that is true. And I hope it 04:26:56
6 will be worked through in ten years.

7 Q Thank you. In the paragraph 202, you say, in
8 the middle of that paragraph (as read):

9 "By adulthood, the friendships of
10 transgender individuals tend to be 04:27:11
11 confined to other transgender
12 individuals (often 'virtual' friends
13 known only online) and the generally
14 limited set of others who are
15 comfortable interacting with 04:27:24
16 transgender individuals."

17 Do you see that?

18 A Yes.

19 Q Is there a basis for that beyond your own --
20 you cite yourself for that, but are you aware of 04:27:39
21 whether or not that actually represents the lived
22 experience of transgender individuals in 2022?

23 A Well, I think in that sentence, if I could
24 edit it, I would emphasize rather than "by adulthood,"
25 I would say "during adolescence." And the basis is not 04:28:00

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1 just my clinical experience. The basis is the clinical
2 experience of the people in the psychosocial therapy
3 group that I mentioned earlier this morning. That
4 seems to be a broad consensus, that many of their trans
5 people are -- have social isolation problems in their 04:28:19
6 friendships and their romances, and I've seen this in
7 my practice. They really are occurring through --
8 through the Internet.

9 And when they're not occurring through the
10 Internet, they're occurring with people in the sexual 04:28:34
11 minority community, other people who may not be trans
12 themselves, but who are excited by their trans and
13 supportive of their trans status.

14 So that's the basis of it.

15 Q You've referred to the trans community, at 04:28:53
16 times, in our conversation today; correct?

17 A I'm sure I've said that, yes.

18 Q Are you aware that the trans community, as a
19 general matter, takes issue with your viewpoint?

20 MR. BROOKS: Objection. 04:29:08

21 THE WITNESS: Yeah, I am aware that there are
22 members in the trans community who find me a hateful
23 person and who believe that I'm against medical,
24 surgical and social care and against the civil rights
25 of transgender people. 04:29:28

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1 I can't control what they believe about me,
2 you see. But I am aware that some people are very
3 appreciative of me and other people think I'm an enemy.

4 BY MS. HARTNETT:

5 Q If 95 percent of trans people opposed your 04:29:47
6 methods, do you think that they would make sense to
7 continue suggesting them for trans people?

8 MR. BROOKS: Objection --

9 THE WITNESS: What was the --

10 MR. BROOKS: -- lack of foundation, calls for 04:29:56
11 speculation.

12 THE WITNESS: What was the last part of your
13 sentence?

14 BY MS. HARTNETT:

15 Q I'm just trying to ask you if -- like, say, 04:30:04
16 assuming 95 percent of trans people opposed your
17 methods, would you have concern for continuing to
18 promote them?

19 MR. BROOKS: Objection.

20 THE WITNESS: To promote my methods? 04:30:13

21 BY MS. HARTNETT:

22 Q Towards --

23 MR. BROOKS: Objection.

24 BY MS. HARTNETT:

25 Q -- trans people. 04:30:17

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1 A My method of -- of informed consent and my
2 method of -- of being thoughtful and considerate
3 about -- about -- about the sources and the
4 consequences?

5 I don't believe that -- that a person 04:30:32
6 thinks -- misunderstands my position would make me give
7 up my position. If you show me that -- that my
8 position is not tenable in a -- in a -- in a -- in a
9 strong scientific basis, I'm certainly able to change.

10 The fact that public opinion, in some 04:30:53
11 commun- -- some sectors of the community, you know,
12 think -- misunderstand me and -- and don't really know
13 what I'm saying, you see, that -- that wouldn't make me
14 give it up.

15 And I don't know how you could assume that 04:31:09
16 95 percent of people, you see. I don't know -- you're
17 just presuming things.

18 Q Are you opposed to civil rights for
19 transgender people?

20 A Absolutely not. I am not -- 04:31:20

21 Q Do you understand --

22 A I am not --

23 Q Sorry?

24 A -- opposed to civil rights for transsexual
25 people. 04:31:26

1 Q Do you know that your opinion in this case is
2 being used to support excluding an 11-year-old
3 transgender girl from a middle school track team that
4 wants her to play on it?

5 MR. BROOKS: Objection. 04:31:36

6 MR. TRYON: Objection.

7 MR. BROOKS: Foundation.

8 THE WITNESS: I already told you I don't know
9 the details of this particular case, the B.P.J.

10 BY MS. HARTNETT: 04:31:50

11 Q I know. And I'm going to tell you that your
12 opinion is being used by some of the defendants in this
13 case to seek to deny an 11-year-old transgender girl
14 from playing on a girls' cross-country and track team
15 where her school otherwise would be willing to have her 04:31:57
16 play, with the support of her parents and family.

17 MR. BROOKS: Objection.

18 There's no question pending, so far as I
19 understand.

20 BY MS. HARTNETT: 04:32:12

21 Q Do you know that that's what your opinion is
22 being used for in this case?

23 MR. BROOKS: Objection.

24 THE WITNESS: I am not aware.

25 ///

1 BY MS. HARTNETT:

2 Q Do you object to your opinion being used to
3 deny an 11-year-old girl the ability to run on a track
4 team at her middle school in West Virginia when she's
5 already otherwise socially transitioning and is 04:32:26
6 supported by her family and her school?

7 MR. BROOKS: Objection; mischaracterizes the
8 witness's opinions.

9 THE WITNESS: I've heard the objection that
10 you're -- you're mischaracterizing my opinion. 04:32:41

11 I -- I don't understand.

12 My opinion has to do with the things I've
13 testified to. I did not testify to anything about an
14 11-year-old girl.

15 And what you are telling me about, I trust 04:32:54
16 you're telling me the truth.

17 I actually don't think about -- when I think
18 about civil rights, I am thinking much more about, I
19 think, older people, you know, housing, educational
20 discrimination in colleges and things like that, 04:33:18
21 vocation, right to vote.

22 You will have to -- it's a -- it's a new thing
23 for me to even think about the civil rights of a
24 six-year-old or a seven-year-old or an eight-year-old.

25 ///

1 BY MS. HARTNETT:

2 Q Well, your -- I'll help you.

3 Your opinion was also submitted in the case of
4 Lindsay Hecox, a college student who was seeking to run
5 consistent with her identity, gender identity, on her 04:33:39
6 college cross-country and track team.

7 A Yes.

8 Q You're aware that your -- your testimony was
9 submitted in support of prohibiting her from running on
10 the team? 04:33:51

11 MR. BROOKS: Objection; mischaracterizes that
12 case.

13 THE WITNESS: Again, my testimony --

14 MS. HARTNETT: I'm counsel of record in that
15 case, and I can tell you that I'm accurately 04:34:03
16 characterizing the case, which is that Dr. Levine's
17 declaration was submitted in support of a motion to
18 ban -- to -- to uphold a statute that would not permit
19 Lindsay Hecox to run, consistent with her gender
20 identity, on a college sports team. 04:34:15

21 And I'm asking him, in light of his statement
22 that he does not oppose transgender civil rights, how
23 he can reconcile that with having his testimony used in
24 this manner.

25 MR. BROOKS: Objection; argumentative. 04:34:26

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1 The witness has explained that his opinions
2 are about science.

3 MS. HARTNETT: Please stop testifying.

4 MR. BROOKS: Please stop arguing.

5 BY MS. HARTNETT: 04:34:35

6 Q Dr. Levine, how can you reconcile --

7 (Simultaneous speaking.)

8 MR. BROOKS: This is not a debate. This is a
9 deposition.

10 MS. HARTNETT: And this -- you're not the 04:34:45
11 witness, either. I'd like to ask Dr. Levine and get an
12 answer as to how he can reconcile having his testimony
13 be filed to oppose the participation of a college
14 student on her college team consistent with her gender
15 identity. 04:34:59

16 THE WITNESS: I don't find it easy to
17 reconcile -- this is just part of some of the great
18 conflict embedded in -- in -- my -- my knowledge is
19 about science. And I do recognize that people
20 interpret what I say in various ways and -- but I don't 04:35:25
21 think I'm responsible for how that is interpreted. I'm
22 just making statements based on my knowledge, based on
23 my clinical experience. And I am uncomfortable, at
24 times, with various aspects of what people make of --
25 of what I have said. 04:35:46

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1 I -- I am uncomfortable, to some extent, by
2 how the lawyers have used some of my -- you know, at
3 times. And I am certainly uncomfortable at how the
4 trans community has used some of what they think I
5 stand for.

04:36:04

6 I'm trying to be clear what I -- what I think
7 and what I stand for. And I am somewhat uncomfortable,
8 at times, about many things, including this, but --

9 BY MS. HARTNETT:

10 Q Do you understand that you're being paid as an 04:36:16
11 expert witness in both the Hecox case and in this case
12 by the defendants in order to submit testimony that
13 will be used against the participation of the
14 transgender students?

15 MR. TRYON: Objection. 04:36:31

16 THE WITNESS: I don't think I fully understand
17 that. I don't think -- I don't think that's -- I -- I
18 guess the answer to the question is I don't fully
19 understand it.

20 BY MS. HARTNETT: 04:36:48

21 Q Okay. Because I -- I'm -- I'm genuinely
22 perplexed because you've said that you're supporting
23 transgender civil rights and you wish for a time where
24 there's less discrimination and that -- yet your
25 submission is not being submitted in a neutral manner 04:36:59

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1 in this case; it's being submitted in support of the
2 side of the case that's seeking to defend the exclusion
3 of the transgender student.

4 And so we don't need to belabor the point, but
5 I'm just trying to -- I'm happy to tell you that. And 04:37:11
6 if you have something you would like to say on the
7 record as to how you can reconcile the use of your
8 testimony for that, with the views you've expressed in
9 this deposition about seeking to make the world better
10 for transgender people, I would appreciate your chance 04:37:24
11 to respond to that.

12 MR. BROOKS: Objection; mischaracterizes --

13 MR. TRYON: Objection.

14 MR. BROOKS: -- testimony and is outside the
15 scope of this witness's expert opinions. 04:37:30

16 THE WITNESS: Well, I thank you for pointing
17 that out. I will think about it more.

18 MS. HARTNETT: Thank you.

19 I think we can take a break now.

20 THE VIDEOGRAPHER: We are off the record at 04:37:46
21 4:38 p.m.

22 (Recess.)

23 THE VIDEOGRAPHER: We are on the record at
24 4:55 p.m.

25 MS. HARTNETT: Thank you. 04:55:19

1 BY MS. HARTNETT:

2 Q Hi, Dr. Levine. We discussed the SEGM
3 organization earlier.

4 Do you recall that?

5 A I do. 04:55:25

6 Q And you described it as an evidence-based
7 organization; correct?

8 A Yes. That's the title, yes.

9 Q And you view them as an organization that
10 strictly adheres to the facts; correct? 04:55:35

11 A Well, facts are interpreted, but, yes, they
12 have a basis in facts.

13 Q In January, you earlier, in the deposition,
14 mentioned that you did a podcast; correct?

15 A I did. 04:55:53

16 Q And that podcast was with two of the lead
17 advisors of SEGM; is that right?

18 A I don't think they're the lead advisors.
19 They're -- they were members of the psychotherapy
20 group. I don't -- I don't -- I wouldn't describe them 04:56:10
21 as lead advisors to SEGM, no.

22 Q Okay. They're -- are they affiliated with
23 SEGM in some way?

24 A They're members of SEGM, yeah.

25 Q And that would be Sasha Ayad and Stella 04:56:21

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1 O'Malley; is that right?

2 A Yes.

3 Q Were the thoughts that you shared with them
4 during that podcast all truthful?

5 A I hope so. 04:56:32

6 Q Okay. I'm just going to -- and I referenced,
7 before we went on the record, uploading a few audio
8 files. I've excerpted some excerpts from the talk you
9 gave, which was, for the record, available at
10 [https://gender-a-wider-lens.captivate.fm/episode/60-](https://gender-a-wider-lens.captivate.fm/episode/60-pioneers-series-we-contain-multitudes-with-Stephen--S-T-E-P-H-E-N) 04:56:53
11 [pioneers-series-we-contain-multitudes-with-Stephen --](https://gender-a-wider-lens.captivate.fm/episode/60-pioneers-series-we-contain-multitudes-with-Stephen--S-T-E-P-H-E-N)
12 [S-T-E-P-H-E-N](https://gender-a-wider-lens.captivate.fm/episode/60-pioneers-series-we-contain-multitudes-with-Stephen--S-T-E-P-H-E-N) -- Levine, dated January 28th, 2022.

13 Dr. Levine, do you recall whether the podcast
14 was -- the conversation you had with Ms. O'Malley and
15 Ms. Ayad actually took place on January 28th? 04:57:38

16 A I think it did, yes.

17 Q Okay. So I'm going to just play for you an
18 excerpt, and I'll ask you a question about it.

19 MS. HARTNETT: Could you please play
20 Exhibit 89. 04:57:56

21 (Exhibit 89 was marked for identification
22 by the court reporter and is attached hereto.)

23 THE WITNESS: I'm not hearing anything.

24 THE VIDEOGRAPHER: Just -- just a moment. I
25 believe he's working on it. 04:58:22

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1 MR. REISBORD: Were you unable to hear that?

2 THE VIDEOGRAPHER: Correct.

3 MS. HARTNETT: We did not hear that.

4 MR. REISBORD: Let my try one more time.

5 (Video Clip Played.) 04:58:40

6 "In 1973" --

7 MR. REISBORD: Are you able to hear that?

8 MS. HARTNETT: Yes.

9 THE WITNESS: Yes.

10 MR. REISBORD: Okay. 04:58:45

11 (Video Clip Played.)

12 "In 1973, after 30 days in -- in practice, I

13 was at a department of psychiatry and had a halftime

14 private practice. I got a man who told me he was

15 sitting in the backyard with a gun in his mouth, under 04:59:00

16 his oak tree, and he decided either to kill himself" --

17 MS. HARTNETT: We can't hear it anymore.

18 (Video Clip Played.)

19 -- "see a psychiatrist who used to be my

20 supervisor a month ago, and my supervisor said, Well, 04:59:17

21 there was an expert in human sexuality down at the

22 university. Why don't you go see him?

23 "And that was the beginning of my career

24 working with people who wanted to change their sex.

25 "You know, he almost killed himself at that 04:59:33

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1 point in 1973."

2 BY MS. HARTNETT:

3 Q Dr. Levine, was that the patient that you were
4 referring earlier to in the deposition?

5 A Yes. 04:59:48

6 Q Rutherford or Ruth; correct?

7 A Yes.

8 MS. HARTNETT: Could you play tab 40, please.

9 MR. REISBORD: Tab 40 would be Exhibit 90.

10 MS. HARTNETT: Oh, sorry, thanks. 05:00:07

11 (Exhibit 90 was marked for identification

12 by the court reporter and is attached hereto.)

13 (Video Clip Played.)

14 "And -- and nine years later, he in fact did
15 kill himself after he changed his gender and left his 05:00:11
16 family and left his country and then returned back to
17 live in America and just decided to end his life. So
18 that was my introduction, my nine-year introduction, to
19 adults who wanted to change their sex.

20 "This was a highly accomplished man. He was 05:00:30
21 the head of our county library system. He had a degree
22 in divinity. And he was a joy to talk to. And he --
23 one day, about four years before he actually killed
24 himself, he slashed his -- at his neck, and when he was
25 admitted to the hospital, he -- he told me that I was 05:00:55

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1 deficient as a therapist because I failed to
2 investigate how angry he has been all of his life at
3 his parents."

4 BY MS. HARTNETT:

5 Q Dr. Levine, is what was just played an 05:01:10
6 accurate account of -- I'm sorry, is -- is what -- do
7 you stand by the account that you provided to SEGM, as
8 just played in that sequence?

9 MR. BROOKS: Objection to the description.

10 THE WITNESS: Are you asking if -- if -- if I 05:01:28
11 said these things that you're recording --

12 BY MS. HARTNETT:

13 Q Yeah, thank you, I'll ask a better question.

14 Is that what you said on the SEGM podcast
15 earlier this year? 05:01:40

16 A I don't call this "the SEGM podcast." This is
17 a --

18 Q I'm sorry.

19 A -- podcast of these two women who have a
20 business in providing information to others who are 05:01:47
21 interested.

22 So I --

23 Q Okay.

24 A -- did say these things, as you -- as is
25 obvious, I said these things. 05:01:56

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1 Q And they were truthful; correct?

2 A Was I telling the truth? Yes --

3 Q Yes.

4 A -- I was -- I tell --

5 Q Okay. 05:02:06

6 A -- the truth.

7 Q Sorry, it's partially a formality of -- I'm
8 just trying to confirm that what you were saying to
9 them is also true today, and so that's why I'm asking
10 you the question, but I won't refer to it as "the SEGM 05:02:17
11 podcast."

12 MS. HARTNETT: Could you please play tab 41,
13 Exhibit 91.

14 (Exhibit 91 was marked for identification
15 by the court reporter and is attached hereto.) 05:02:24

16 (Video Clip Played.)

17 "It was quite an educational experience for
18 me, both as a he and as a she, and -- and she and I
19 wrote a paper in the Archives of Sexual Behavior in
20 19-, I think, -83 called Increasingly Ruth: Towards an 05:02:37
21 understanding of sex reassignment surgery.

22 And then in 1984, when he died, I wrote a
23 letter to the editor about Ruth's suicide.

24 Q Dr. Levine, was that a recording of you
25 speaking to the podcast earlier this year? 05:03:03

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1 A Yes.

2 Q You mentioned that you wrote a letter to the
3 editor after Ruth's death, and in that letter, you said
4 that Ruth's unfortunate legacy to those who invested in
5 her is psychologic injury due to her abandonment of 05:03:18
6 them; is that correct?

7 A Would you repeat that? I don't recognize
8 those words.

9 Would you repeat them slowly?

10 Q I'm sorry. Ruth's unfortunate legacy to those 05:03:30
11 who invested in her is psychologic injury due to her
12 abandonment of them.

13 A Yes, that was --

14 Q Did you write that?

15 A Yes. I don't want to give you more 05:03:39
16 information than you're asking for, but -- the answer
17 to your question is yes.

18 Q Thank you.

19 MS. HARTNETT: Could you play tab --

20 Exhibit 92, please. 05:04:01

21 (Exhibit 92 was marked for identification
22 by the court reporter and is attached hereto.)

23 (Video Clip Played.)

24 "So I've been accused of being very
25 conservative on this issue and biased by -- by that 05:04:06

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1 experience, and, in fact, I plead guilty. I am -- I --
2 I -- that was my introduction."

3 Female: "Yeah."

4 "And it -- and, unfortunately, it's not the
5 only case of -- of people who have aspirations who 05:04:21
6 think that their troubles as a person will disappear
7 if -- if they change their gender presentation and
8 change their bodies and -- and only to discover that
9 life is not as easy as they imagined, and they didn't
10 escape much. 05:04:44

11 "So I plead guilty to being biased, and I
12 think all of us have a kind of bias, and we ought to
13 own it."

14 BY MS. HARTNETT:

15 Q Dr. Levine, were those your statements on the 05:04:55
16 podcast earlier this year?

17 A Yes.

18 Q And were they your truthful statements?

19 A Yes.

20 MS. HARTNETT: Could you please play 05:05:10
21 Exhibit 93.

22 MR. TRYON: This is Dave Tryon. I'm going to
23 object to --

24 (Video Clip Played.)

25 "I have a Mas-" -- 05:05:16

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1 MR. TRYON: I'm going to object to to playing
2 these excerpts without the full context.

3 MS. HARTNETT: And I will just say for the
4 record that there is -- I think the -- the person that
5 gave the podcast knows the context, and I've given the 05:05:26
6 web URL for anyone to look at the full context.
7 There's not a written transcript online.

8 MR. TRYON: My objection stands.

9 MS. HARTNETT: Of course. Thank you.

10 Could you play Exhibit 93, please. 05:05:43

11 (Exhibit 93 was marked for identification
12 by the court reporter and is attached hereto.)

13 (Video Clip Played.)

14 "I have a Master's prepared person, just got
15 out of her -- her internship, who told me how you're 05:05:48
16 supposed to treat transgender people, and I was just
17 astounded.

18 "I gave a seminar two years ago to residents
19 who told me -- residents in psychiatry -- who told me
20 how trans people ought to be treated. 05:06:05

21 "See, they had a chain in trust. Somebody
22 taught them, and they believe it, the passion, they
23 believe it. They have the zeal of the new -- of the
24 convert to being a psychiatrist or being a counselor,
25 whatever it is. And -- and -- and when I give them 05:06:21

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1 facts, they think I'm an outlier or they think I'm an
2 old fuddy-duddy, there's something wrong with me. They
3 don't believe me.

4 "Because the truth is that trans is normal,
5 you see, and -- and that they can have highly 05:06:33
6 successful lives, just like anybody else.

7 "And it's not based on experience. It's
8 certainly not based on any scientific scrutiny, you
9 see.

10 "And so what I'm really saying is that so many 05:06:46
11 of the doctors just practice how they've been taught to
12 practice. They -- they -- we -- we -- none of us have
13 the brain power -- we take care of so many different
14 things, we can't be experts in -- in -- in the original
15 train of -- that chain of trust at all, you see. 05:07:05

16 "So of course we oversimplify everything.

17 "And, you know, there -- we rely on -- on a
18 few skeptics like -- like the three of us."

19 BY MS. HARTNETT:

20 Q Dr. Levine, was that clip of you speaking on 05:07:22
21 the podcast earlier this year?

22 A It is.

23 Q Was that your truthful statements?

24 MR. TRYON: Objection.

25 ///

1 BY MS. HARTNETT:

2 Q Sorry?

3 A I said --

4 Q I --

5 A -- those things that you heard on the podcast, 05:07:44
6 yes.

7 Q And were they your truthful statements?

8 A Yes.

9 MS. HARTNETT: Okay. Could you play
10 Exhibit 94, please. 05:07:53

11 (Exhibit 94 was marked for identification
12 by the court reporter and is attached hereto.)

13 (Video Clip Played.)

14 "And then three years later, there was the six
15 standards of care that was almost word for word for 05:07:59
16 what our group did except for one letter was necessary.
17 That is, he wanted to make it easier to get
18 transgender."

19 BY MS. HARTNETT:

20 Q Dr. Levine, was that you speaking on the 05:08:15
21 podcast earlier this year?

22 A Yes. And it's my truthful statement.

23 Q Thank you. You used the term "get
24 transgender" on that clip. I was just wondering what
25 you mean by that. 05:08:27

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1 A I think that was referring to hormones, access
2 to hormones.

3 We used to have a standard that two
4 independent individuals or one group committee were
5 required to write a recommendation for hormones, and 05:08:44
6 Dr. Richard Green, who was the head of the organization
7 at the time, didn't like that at all. He was a strong
8 advocate of immediate care. And he told me so, he
9 didn't like it. And -- and he reconstituted --
10 accepted the fifth standards of care, and he formed a 05:09:05
11 new committee with the -- you know, with the charge to
12 get rid of that criteria for hormones.

13 Q Do you typically use the term "get
14 transgender"?

15 A No. This was a spontaneous conversation. I 05:09:24
16 don't -- it's a funny phrase. I don't know. It came
17 out of my mouth. I don't know why. That's --

18 Q Okay.

19 A -- not my usual language.

20 But again, this was not a paper I was 05:09:33
21 delivering that I, you know, worked on. This is
22 something that happened rather spontaneously.

23 Q I understand.

24 MS. HARTNETT: Could you please play
25 Exhibit 95. 05:09:49

1 (Exhibit 95 was marked for identification
2 by the court reporter and is attached hereto.)
3 (Video Clip Played.)
4 "I think it's time for a re-examination of the
5 wisdom of affirmative care. I'm not saying affirmative 05:09:55
6 care doesn't help some people, but I'm not so sure how
7 many people it harms."

8 BY MS. HARTNETT:

9 Q Dr. Levine, was that your truthful statement
10 on the podcast earlier this year? 05:10:09

11 A It --

12 MR. TRYON: Same objection as before.

13 Thank you.

14 You may answer.

15 THE WITNESS: I -- it is my true statement. 05:10:18

16 I'm still not sure what percentage of people
17 are ultimately harmed and how to measure those harms
18 and when to measure those harms.

19 MS. HARTNETT: Thank you.

20 Could you play tab -- sorry -- Exhibit 96, 05:10:33
21 please.

22 (Exhibit 96 was marked for identification
23 by the court reporter and is attached hereto.)

24 (Video Clip Played.)

25 "The problem is that we do not have rigorous 05:10:38

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1 follow-up studies of people who made the transition."

2 BY MS. HARTNETT:

3 Q Dr. Levine, is that your truthful statement
4 made earlier this year?

5 MR. TRYON: Objection. 05:11:00

6 THE WITNESS: Yes.

7 MR. TRYON: I just want to place on the record
8 evidence rule 106. Thank you.

9 Go ahead and answer.

10 BY MS. HARTNETT: 05:11:05

11 Q Dr. Levine, do you agree that there is not
12 rigorous follow-up studies of people who have made the
13 transition?

14 A Yes. I believe I testimony -- I testified to
15 that earlier today. 05:11:24

16 Q And for all of these statements that I've
17 asked you about, do you stand by those statements,
18 sitting here today?

19 A Number one, I have said those things, and I
20 believe them to be essentially correct today, yes. 05:11:36

21 Q And, thank you, I'm asking only to -- in light
22 of the objection, not to repeat my questions to you.

23 MS. HARTNETT: Could you please play
24 Exhibit 97.

25 (Exhibit 97 was marked for identification 05:11:48

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1 by the court reporter and is attached hereto.)

2 (Video Clip Played.)

3 "The people who come to me who are depressed,
4 you know, those -- those -- after transition, those are

5 just anecdotal reports. I have no idea what the -- 05:12:00
6 what the denominator is, you see."

7 BY MS. HARTNETT:

8 Q Dr. Levine, do you agree with the statement
9 that was just played?

10 A Yes. 05:12:10

11 MS. HARTNETT: Could you please play
12 Exhibit 98.

13 (Exhibit 98 was marked for identification
14 by the court reporter and is attached hereto.)

15 MR. TRYON: Counsel, before you play it -- 05:12:19

16 MS. HARTNETT: Yes.

17 MR. TRYON: Counsel, will you just agree to
18 give me a standing objection to these excerpts?

19 MS. HARTNETT: Yes.

20 MR. TRYON: Thank you. 05:12:28

21 (Video Clip Played.)

22 "And -- and because we don't know, because we
23 don't know, I think we have to say why do we have all
24 this enthusiasm, why do we have all this chain of trust
25 passion that this is the best treatment. We don't know 05:12:46

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1 is the best treatment, you see."

2 BY MS. HARTNETT:

3 Q Dr. Levine, do you agree with that statement
4 that you made earlier this year?

5 A I do. 05:12:58

6 MS. HARTNETT: Could you please play
7 Exhibit 99.

8 (Exhibit 99 was marked for identification
9 by the court reporter and is attached hereto.)

10 (Video Clip Played.) 05:13:05

11 "Now, I want to quickly say that while I'm an
12 advocate of someone who thinks or wants to be or
13 considers themselves a transgendered person, I think
14 they ought to have a psychotherapeutic approach before
15 they make any -- any life-changing decisions, but I 05:13:22

16 admit that I have no follow-up. This is not on the
17 basis of randomized control study. I am in the same
18 difficult position that the affirmative care doctors
19 are in, only I have more faith based upon a hundred
20 years of doing psychotherapy as a tradition, you see, 05:13:42
21 and they only have a few years, with no follow-up."

22 BY MS. HARTNETT:

23 Q Dr. Levine, is that your truthful statement?

24 A Yes.

25 MS. HARTNETT: Could you please play 05:14:02

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1 Exhibit 100.

2 (Exhibit 100 was marked for identification
3 by the court reporter and is attached hereto.)

4 (Video Clip Played.)

5 "So -- so what I'm saying is that in the early 05:14:05
6 studies, the death rates from cancer and cardiovascular
7 disease and -- and accidents were -- were elevated and
8 what -- and what that really means is that the
9 lifestyle things predispose them to physical diseases.

10 "So, you know, if you're a parent, you -- 05:14:27
11 you -- you want to die -- you want to die before your
12 children, you see.

13 "So for many -- for many of these kids,
14 they're going to be sick.

15 "And I just saw a slide of the famous -- 05:14:41
16 Jazz Jennings. Do you know that name?

17 Female: Yeah.

18 "Apparently Jazz Jennings was a very thin,
19 very attractive person when she had surgery, and in the
20 postoperative time, she's now grossly obese. She is -- 05:14:58
21 I saw a picture of her. She is grossly obese.

22 "So, you know, this is one of the -- this is
23 one of the things that never gets talked about, what
24 are the physical manifestations, what are the
25 psychological manifestations, what are the outcomes." 05:15:13

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1 BY MS. HARTNETT:

2 Q Dr. Levine, is that your truthful statement?

3 A Yes.

4 Q Is it your contention that Jazz Jennings is
5 grossly obese because she had gender confirmation 05:15:29
6 surgery?

7 A No. She became grossly obese after gender
8 confirmation surgery. In addition, she had -- she had
9 other problems as well, I think.

10 I only know that because Jazz Jennings is a 05:15:50
11 public, you know, celebrity, so to speak, and people
12 talk about her and people showed me pictures of her.

13 So I've never -- that's -- that's what I know.

14 Q But you've never met Jazz Jennings; correct?

15 A I have never met Jazz Jennings. 05:16:09

16 MS. HARTNETT: Could you play Exhibit 101,
17 please.

18 (Exhibit 101 was marked for identification
19 by the court reporter and is attached hereto.)

20 (Video Clip Played.) 05:16:19

21 "And the -- the affirmative care doctors like
22 to blame all these comorbidities and the shortened
23 lifespan on minority stress, and you would -- I
24 think -- I think we recognize that it is stressful to
25 be -- to belong to a sexual minority, but -- but 05:16:32

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1 children who are cross-gender identified, who have
2 separation anxiety and depression and so forth, they're
3 not -- they're not having minority stress.

4 "And -- and the kids who -- you know, if
5 you -- if you walk in -- if you walk in and see your 05:16:50
6 postpartum depressed mom hanging from the rafters and
7 then you decide three weeks later that you're going to
8 change your gender, this is not minority stress."

9 BY MS. HARTNETT:

10 Q Dr. Levine, is that your truthful statement? 05:17:07

11 A Yes.

12 Q Are you aware of any example of an actual kid
13 who walked in and saw their postpartum depressed mom
14 hanging from the rafters and three weeks later decided
15 to change gender? 05:17:22

16 A Absolutely.

17 Q Can you tell me what -- where is that example?

18 A I think that case was presented to me.

19 Q By whom?

20 A One of my staff. Or it was presented to me, 05:17:33
21 you know, by somebody else.

22 Occasionally, I supervise other people.

23 But that came -- that -- that came from a
24 recent -- a recent January 20th case history that I
25 heard. 05:17:53

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1 It -- it has to do, you see, with not taking a
2 history, giving people, very quickly, affirmative care
3 and not appreciating the forces that might have shaped
4 this -- that -- that may be very -- that may play out
5 and may -- very difficult to have a happy, successful 05:18:15
6 life as a trans person.

7 So I -- I can't give you the -- I can't tell
8 you at the moment who told me that, but I can tell you
9 I am not telling -- I am telling the truth. This is
10 what I recently heard prior -- 05:18:34

11 Q Was that as a -- sorry.

12 A Pardon me.

13 Q Was that -- was that an anecdote that came to
14 you from somebody in your clinic?

15 A As I said before, it might have been someone 05:18:43
16 in my clinic; it might have been some other
17 professional who talked to me about that.

18 Q Do you know if the person at issue, the --
19 the -- that was seeking a transition, whether they had
20 any signs of gender dysphoria prior to the mom hanging 05:18:58
21 from the rafters?

22 A I think the implication was that they hadn't,
23 but I don't remember enough details to -- I couldn't
24 tell you the case history. That's the aspect of the
25 case history that I recall. 05:19:18

1 Q Thank you.

2 MS. HARTNETT: Can you play Exhibit 102,
3 please.

4 (Exhibit 102 was marked for identification
5 by the court reporter and is attached hereto.) 05:19:26
6 (Video Clip Played.)

7 "Lots of girls have temporary eating
8 disorders, and some of them end up overcoming it, but
9 they overcome it sometimes by becoming vegetarians or
10 vegans. So it's okay, and it's much better. It's much 05:19:42
11 better than having an eating disorder."

12 BY MS. HARTNETT:

13 Q Dr. Levine, was that your truthful statement?

14 A Yes.

15 Q What point were you trying to make by drawing 05:19:59
16 an analogy to eating disorders and vegetarians and
17 vegans?

18 A I think you would have to play for me what
19 preceded that, but off the top of my head today, two
20 months after I made that statement, more than two 05:20:14
21 months after I made that statement, I was probably
22 making reference to the fact that among adolescent
23 girls who declare themselves to be trans boys, a large
24 percentage of them have a pre- -- a predeclaration
25 eating disorder, that this is part of the -- the 05:20:35

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1 psycho- -- the -- if we can agree that an eating
2 disorder is a true problem and not just a dietary of
3 something or other, the -- this evidence of the
4 psychopathology that precedes transgender
5 identification, the crystallization of a trans 05:20:59
6 identification, eating disorder is just another way of
7 self-harm where -- where one cannot live comfortably in
8 the self as it is developing.

9 So that's probably what I was making reference
10 to, the pre-crystallization of a transgender, the 05:21:18
11 problems that are some- -- that are often seen in girls
12 prior to their coming out as a trans boy.

13 Q Is it your view that you could correct the
14 eating disorder and the person may stop identifying as
15 transgender? 05:21:38

16 A Well, I think most eating dis- -- what I was
17 saying -- I think you misunderstood -- is the -- the
18 prelude to the eating disorder was transgender. I will
19 say if you could help the person understand the
20 motivation for the eating disorder and help her to come 05:22:00
21 to grips with what she's doing is harmful to herself in
22 the short and in the long run, then it wouldn't -- it
23 may prevent -- it may help her to find another
24 solution, for example, becoming a vegan or -- that
25 would be a benign -- a less -- less problematic 05:22:25

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1 solution than having to become transgender, forget her
2 eating disorder and focus on something else in a way
3 that dominates her life.

4 So you -- you dominate your life by thinking
5 that you're too fat when you're 93 pounds, and now 05:22:43
6 you're domi- -- you give that up, and then you dominate
7 your life because you're really a boy trapped in a
8 girl's body and --

9 So I'm telling you, as a psychiatrist, life is
10 complicated and histories are complicated and our 05:22:57
11 ability to predict things is not very good, and I just
12 want us to rely on science, as -- whatever the
13 limitations of sciences are, I want to rely on science
14 and not something shorter than science, you know,
15 fervent, passionate beliefs, whatever. 05:23:19

16 Q So in that instance -- I'm just trying to make
17 sure I understand -- your -- the idea would be that
18 it's better to end up being vegan than transgender?

19 A If -- if you put it in that way, if you reduce
20 everything to that simplicity, I guess the answer is it 05:23:35
21 would be better to have a -- that would be a better
22 supplementation of your original concerns about
23 yourself and your body and the sexual meaning of your
24 body than it is to repudiate your femininity entirely
25 and try to remove your breasts surgically and take 05:23:56

1 hormones and so forth, yes.

2 MS. HARTNETT: Could you play Exhibit 103,
3 please.

4 (Exhibit 103 was marked for identification
5 by the court reporter and is attached hereto.) 05:24:04
6 (Video Clip Played.)

7 "It's your current sexual identity --

8 Female: Yeah.

9 -- "you see. I mean, I'm sure I've had
10 identities -- I used to be a stamp collector, you know. 05:24:15
11 I had an identity as a stamp collector. And I don't
12 collect stamps anymore."

13 BY MS. HARTNETT:

14 Q Dr. Levine, are those your truthful
15 statements? 05:24:28

16 A I was a stamp collector.

17 Q I was a baseball card collector.

18 Is being transgender like being a stamp
19 collector?

20 A No. 05:24:38

21 MS. HARTNETT: Could you play tab --
22 Exhibit 104, please.

23 (Exhibit 104 was marked for identification
24 by the court reporter and is attached hereto.)
25 (Video Clip Played.) 05:24:55

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1 "I think the doctor's responsibility is to
2 diagnose this, understand the factors that is pushing
3 the child in that direction and the family in that
4 direction and to inform what -- the parents and the
5 child of what is known and what is not known and what 05:25:10
6 the alternative treatments are, and the parents and the
7 child make the decision, not the doctor. The doctor
8 does not have the data to make the decision."

9 BY MS. HARTNETT:

10 Q Dr. Levine, is that your truthful statements? 05:25:28

11 A That is, although I'm embarrassed, but I used
12 the wrong -- I should have said "are" and not "is" in
13 the first sentence.

14 Q I think I just did the same thing.

15 I have one more excerpt to play. 05:25:42

16 MS. HARTNETT: Could you play Exhibit 105,
17 please.

18 (Exhibit 105 was marked for identification
19 by the court reporter and is attached hereto.)

20 (Video Clip Played.) 05:25:48

21 "So if I'm an expert in something, it's a very
22 narrow topic I'm an expert in. Even though I'm a
23 doctor and you -- somebody may think, well, he's a
24 doctor; right? But the doctor doesn't know much about
25 most things. 05:26:01

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1 "And -- and there is the wisdom, I think, is
2 the difference between demagoguery, which I think many
3 affirmative care doctors are demagogues, and experts,
4 many of whom are just uneasy about what is not known."

5 BY MS. HARTNETT:

05:26:23

6 Q Dr. Levine, were these your truthful
7 statements from earlier this year?

8 A Yes.

9 Q Do you consider yourself to be a demagogue or
10 an expert?

05:26:36

11 A I consider myself, on this issue of the
12 scientific basis of -- of trans delivery -- care
13 delivery, to be an expert in this very narrow field
14 because my definition of an expert, knows the
15 difference between what is known and what is not known, 05:26:53
16 you see.

17 On many subjects that I have to work on every
18 day as a psychiatrist, I -- I have -- I -- I'm not sure
19 what -- the difference between what I know and what is
20 known by more expert people in the field. 05:27:10

21 I seem to have enough to have credentials as a
22 practicing doctor, but I'm not an expert in most things
23 I take care of.

24 When it comes to the data about this matter of
25 trans care, I feel I'm a relative expert, and I think I 05:27:28

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1 have more perspective and more basis for that
2 perspective than many people who have been taught how
3 to take care of transgender people.

4 Q Do you believe Dr. Adkins is a demagogue?

5 A I don't know Dr. Adkins well enough to -- to 05:27:49
6 make that decision. I don't want to be insulting at
7 all to my colleagues, but if -- if Dr. Adkins believes
8 this is genetically determined and if she believes that
9 it's fixed and if she believes she's helping and she
10 has evidence that she's helping people live happy lives 05:28:11
11 for the next 40 years, I believe she is much more
12 closer to my definition of a demagogue than, say, a
13 person who can't distinguish between what she knows and
14 what is known versus an expert.

15 But I don't want to pass judgment on her 05:28:27
16 because, you know, I've just read her report, that's
17 all.

18 Q How about Dr. Safer, would you have the same
19 view there, that -- do you believe he's a demagogue, or
20 you wouldn't want to pass judgment? 05:28:39

21 A You know, one of the ethical principles of
22 being a doctor is to speak respectfully of one's
23 colleagues.

24 I -- I would say, I just want to repeat, that
25 most practicing doctors have a belief system that 05:28:58

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1 they're working on the side of angels, and that's a
2 different set of ideas than what science has already
3 demonstrated.

4 So to the extent that people believe,
5 passionately believe, that what they are doing is 05:29:10
6 ensuring a -- a -- a productive, successful,
7 asymptomatic, fulfilling life and there's no evidence
8 for it, well, I think they're not -- they shouldn't be
9 certain about that.

10 And they're closer to an ordinary physician or 05:29:30
11 a demagogue than they are to an expert.

12 Q Thank you. Could you just -- I have a --
13 hopefully, a couple of final questions about your
14 expert report.

15 Could you pull that back up? That was 05:29:44
16 Exhibit 87.

17 MR. BROOKS: Coming, coming.

18 BY MS. HARTNETT:

19 Q And I'm going to be just going to
20 paragraph 81. 05:29:54

21 MR. BROOKS: Which is on.

22 MS. HARTNETT: It's on -- take your time, but
23 page 31, paragraph 81.

24 MR. BROOKS: What heading are we under here?

25 MS. HARTNETT: You are under -- 05:30:10

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1 MR. BROOKS: I see it. I see the heading at
2 the top of page 30.

3 Is that the right heading? Am I missing
4 anything --

5 MS. HARTNETT: Correct. 05:30:23

6 MR. BROOKS: -- or is that --

7 Under "Opinions and practices vary widely..."
8 Okay.

9 And then you said paragraph 81?

10 MS. HARTNETT: Right. And this is a paragraph 05:30:29
11 about -- Dr. Levine is describing a Lichenstein
12 article; is that correct?

13 MR. BROOKS: Let me just say, Dr. Levine, if
14 you want to look at any paragraphs between the heading
15 and this one, for context, you should feel free to, or 05:30:46
16 if not -- if you don't feel the need, then you don't
17 need to.

18 THE WITNESS: Okay.

19 BY MS. HARTNETT:

20 Q So this paragraph is talking about, in your 05:31:09
21 words, the "loose standards" at Dr. Safer's clinics at
22 Mount Sinai in Columbia; correct?

23 A Yes.

24 Q And do you say that he's -- I'm just reading
25 from the first sentence, but you a say at least one 05:31:22

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1 prominent clinic, quote, is quite openly admitting
2 patients for even surgical transition who are not
3 eligible under the criteria set out in WPATH's
4 Standards of Care.

5 Do you see that? 05:31:36

6 A Yes. The last sentence, right.

7 Q Is it your understanding that patients were
8 receiving care there without meeting the WPATH
9 standards?

10 A WPATH standards are just one set of standards, 05:31:53
11 and I guess Dr. Safer has a different set of standards.

12 I don't think that WPATH needs to be followed,
13 you know. I don't think they're -- they are in fact
14 the standards of care. They are just an organization
15 that is providing some guidelines, which they call 05:32:19
16 standards of care, but aren't true standards of care.
17 They're just guidelines from a professional
18 organization that is -- that is an advocacy
19 organization for -- for the treatment -- for
20 affirmative treatment. 05:32:36

21 Q But are you aware that Mount Sinai went
22 through the process of having those people satisfy the
23 WPATH standards before they had surgery notwithstanding
24 that they would have also met the other standards set
25 forth by Sinai? 05:32:47

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1 MR. BROOKS: Objection.

2 THE WITNESS: I'm -- I'm not deeply involved

3 in the process of how Dr. Safer has done his work.

4 This would be not an area of my expertise about --

5 about his criteria.

05:33:04

6 BY MS. HARTNETT:

7 Q I guess my question for you is whether you

8 know, sitting here today, whether in fact Dr. Safer's

9 center allowed patients to have surgery under what you

10 call the "loose standards" without satisfying WPATH.

05:33:17

11 A Well, it was my understanding from the quoted

12 study that -- that he was providing -- or giving

13 permission for surgical care for people who may not

14 have met the few criteria that -- that we have -- had

15 organized in 2000- -- in, you know, the seventh

05:33:44

16 edition.

17 Q Did you read the Lichtenstein article before

18 citing it here?

19 A I must have read it, but it's probably one of

20 hundreds of articles, and right now, I can't recall the

05:33:54

21 details.

22 Q Thank you.

23 MS. HARTNETT: Could I take a -- go off -- I

24 think I'm almost -- or -- done, if not done.

25 But could we go off the record briefly for me

05:34:06

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1 to collect my nets and then hopefully we'll be done?

2 THE VIDEOGRAPHER: We are off the record at

3 5:34 p.m.

4 (Recess.)

5 THE VIDEOGRAPHER: We are on the record at 05:44:53

6 5:45 p.m.

7 MS. HARTNETT: Thank you, Dr. Levine. I have

8 no further questions, but reserve the right to any

9 recross if there's further questioning of you.

10 THE WITNESS: You're welcome. 05:45:12

11 MS. HARTNETT: Thank you.

12 MR. BROOKS: Speaking for the -- Roger Brooks,

13 speaking for the intervenor, I have no questions for

14 the witness.

15 MR. TRYON: This is Dave Tryon on behalf of 05:45:20

16 the State of West Virginia.

17 Dr. Levine, thank you for your time.

18 I have no questions.

19 MS. MORGAN: This is Kelly Morgan on behalf of

20 the West Virginia Board of Education and Superintendent 05:45:29

21 Burch. I have no questions. Thank you.

22 MS. DENIKER: Dr. Levine, this is Susan

23 Deniker, counsel for defendants Harrison County Board

24 of Education and Superintendent Stutler, and I have no

25 questions for you.

1 Thank you for your time.

2 THE WITNESS: You're welcome.

3 MS. ROGERS: Dr. Levine, this is Shannon
4 Rogers on behalf of the West Virginia Secondary School
5 Activities Commission. I have no questions either. 05:45:53

6 Thank you.

7 THE WITNESS: You're welcome.

8 MS. HARTNETT: Dr. Levine, thank you for your
9 time.

10 THE VIDEOGRAPHER: Thank you. 05:46:00

11 We are off the record at 5:46 p.m., and this
12 concludes today's testimony given by Stephen Levine,
13 Dr. -- Dr. Stephen Levine.

14 The total number of media units was seven and
15 will be retained by Veritext Legal Solutions. 05:46:16

16 Thank you.

17 (TIME NOTED: 5:46 p.m.)

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3
4 I, STEPHEN LEVINE, do hereby declare under
5 penalty of perjury that I have read the foregoing
6 transcript; that I have made any corrections as appear
7 noted, in ink, initialed by me, or attached hereto;
8 that my testimony as contained herein, as corrected, is
9 true and correct.

10 EXECUTED this ____ day of _____,
11 20____, at _____, _____.
(City) (State)

12
13
14
15 _____
STEPHEN LEVINE

16 VOLUME I
17
18
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25

1 I, the undersigned, a Certified Shorthand
2 Reporter of the State of California, do hereby certify:

3 That the foregoing proceedings were taken
4 before me at the time and place herein set forth; that
5 any witnesses in the foregoing proceedings, prior to
6 testifying, were placed under oath; that a record of
7 the proceedings was made by me using machine shorthand
8 which was thereafter transcribed under my direction;
9 further, that the foregoing is an accurate
10 transcription thereof.

11 I further certify that I am neither financially
12 interested in the action nor a relative or employee of
13 any attorney of any of the parties.

14 IN WITNESS WHEREOF, I have this date subscribed
15 my name.

16
17 Dated: April 15, 2022

18
19 
20

ALEXIS KAGAY

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

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COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

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Exhibit C

Scientific Statement

Considering Sex as a Biological Variable in Basic and Clinical Studies: An Endocrine Society Scientific Statement

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Abbreviations: ACTH, adrenocorticotrophic hormone; AT₂R, angiotensin type 2 receptor; BMI, body mass index; cAMP, cyclic adenosine monophosphate; CKD, chronic kidney disease; CRF, corticotropin-releasing factor; CVD, cardiovascular disease; dMRI, diffusion magnetic resonance imaging; fMRI, functional magnetic resonance imaging; FCG, Four Core Genotypes (model); GMV, gray matter volume; GPCR, G-protein coupled receptor; HPA, hypothalamic-pituitary-adrenal; KYN, kynurenine; LC, locus coeruleus; MIH, Müllerian inhibitory hormone; PAR, pseudoautosomal region; PKA, protein kinase A; PTSD, posttraumatic stress disorder; RAAS, renin-angiotensin-aldosterone system; rs-fMRI, resting state functional magnetic resonance imaging; sMRI, structural magnetic resonance imaging; UCN, urocortin.

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Abstract

In May 2014, the National Institutes of Health (NIH) stated its intent to “require applicants to consider sex as a biological variable (SABV) in the design and analysis of NIH-funded research involving animals and cells.” Since then, proposed research plans that include animals routinely state that both sexes/genders will be used; however, in many instances, researchers and reviewers are at a loss about the issue of sex differences. Moreover, the terms *sex* and *gender* are used interchangeably by many researchers,

further complicating the issue. In addition, the sex or gender of the researcher might influence study outcomes, especially those concerning behavioral studies, in both animals and humans. The act of observation may change the outcome (the “observer effect”) and any experimental manipulation, no matter how well-controlled, is subject to it. This is nowhere more applicable than in physiology and behavior. The sex of established cultured cell lines is another issue, in addition to aneuploidy; chromosomal numbers can change as cells are passaged. Additionally, culture medium contains steroids, growth hormone, and insulin that might influence expression of various genes. These issues often are not taken into account, determined, or even considered. Issues pertaining to the “sex” of cultured cells are beyond the scope of this Statement. However, we will discuss the factors that influence sex and gender in both basic research (that using animal models) and clinical research (that involving human subjects), as well as in some areas of science where sex differences are routinely studied. Sex differences in baseline physiology and associated mechanisms form the foundation for understanding sex differences in diseases pathology, treatments, and outcomes. The purpose of this Statement is to highlight lessons learned, caveats, and what to consider when evaluating data pertaining to sex differences, using 3 areas of research as examples; it is not intended to serve as a guideline for research design.

Key Words: brain-gut, cardiovascular disease, chromosome complement, gender, sex differences, steroid hormones

Sex is an important biological variable that must be considered in the design and analysis of human and animal research. The terms *sex* and *gender* should not be used interchangeably. Sex is dichotomous, with sex determination in the fertilized zygote stemming from unequal expression of sex chromosomal genes. By contrast, gender includes perception of the individual as male, female, or other, both by the individual and by society; both humans and animals have sex, but only humans have gender. Both sexes produce estrogens, androgens, and progestins; there are no male- or female-specific sex hormones, *per se*, although these steroids are present in substantially different levels in males and females. Sex differences are caused by 3 major factors—sex hormones, genes, and environment. To understand disease mechanisms and exploit sex differences in protection or exacerbation of diseases, one needs to determine the relative contribution of factors, including observer effect (1), causing sex differences. Here—using 3 broad research areas as examples—the roles of sex differences in brain anatomy, brain-gut axis, and cardiovascular disease are discussed. Contemporary brain imaging methods show age- and sex-related differences in brain size, global and regional gray matter volume, white matter connectivity, and neuroanatomic regulation of appetite and satiety; while these differences are seen in large population-based studies, there is tremendous individual overlap, but such group-level findings do not inform findings, physiology, or pathology at the individual level. Sex differences in disorders of the brain-gut axis, obesity, type 2 diabetes,

and metabolic syndrome are caused by differential actions of brain-gut peptide and steroid hormones. The activation, signaling, and pharmacotherapy responses of the components of the hypothalamic-pituitary-adrenal (HPA) axis differ between the sexes. Heart and kidney functions are linked. Age, hormones, and sex biases seen in cardiovascular and chronic kidney diseases also differentially influence pharmacologic responses in men and women. Thus, sex differences pervade biology and medicine, and while not discussed in this Statement, must be considered in virtually all areas of biomedical research.

Section I

Sex Versus Gender

Much of the American public is surprisingly prudish about the word *sex*; it has now become commonplace to use the seemingly more genteel term *gender* when one really means *sex*. In *Moritz v Commissioner of Internal Revenue* (469 F. 2d 466 [1972]), Ruth Bader Ginsburg (subsequently, The Honorable Ruth Bader Ginsburg) argued against discrimination “on the basis of sex” not “on the basis of gender,” thus clearly, knowledgeably, and presciently understanding that “sex” does not equal “gender.” In a decision 48 years later (*Bostock v Clayton County*, 590 US, decided June 15, 2020), the United States Supreme Court separately ruled against discrimination on the basis of gender. *Gender* is often misused as a synonym for *sex*—for example, when filling out forms for various activities, we are routinely

asked to check a box labeled “gender,” but the only available options are boxes labeled “M” and “F.” But *sex* is not the same thing as *gender* and using these terms as equivalents obfuscates differences that are real and important in society in general and biomedical research in particular.

Biological Sex: The Definition of Male and Female

Sex is a biological concept. Asexual reproduction (cloning) is routine in microorganisms and some plants, but most vertebrates and all mammals have 2 distinct sexes. Even single-cell organisms have “mating types” to facilitate sexual reproduction. Only cells belonging to different mating types can fuse together to reproduce sexually (2, 3). Sexual reproduction allows for exchange of genetic information and promotes genetic diversity. The classical biological definition of the 2 sexes is that females have ovaries and make larger female gametes (eggs), whereas males have testes and make smaller male gametes (sperm); the 2 gametes fertilize to form the zygote, which has the potential to become a new individual. The advantage of this simple definition is first that it can be applied universally to any species of sexually reproducing organism. Second, it is a bedrock concept of evolution, because selection of traits may differ in the 2 sexes. Thirdly, the definition can be extended to the ovaries and testes, and in this way the categories—female and male—can be applied also to individuals who have gonads but do not make gametes.

In mammals, numerous sexual traits (gonads, genitalia, etc) that typically differ in males and females are tightly linked to each other because one characteristic leads to sex differences in other traits. The type of gonads is controlled by the presence of XX or XY chromosomes, and gonadal secretions in turn regulate formation of female or male reproductive tissues, and characteristics that differ in typical males or females. These characteristics include external genitalia, uterus and oviducts, sperm ducts, and secondary sexual characteristics such as facial hair and pitch of voice. However, many people cannot make either eggs or sperm, yet are recognized as female or male based on other physical characteristics; people who do not have either ovaries or testes are rare. For individuals that possess a combination of male- and female-typical characteristics, these clusters of traits are sufficient to classify most individuals as either biologically male or female. For example, a person with testes and a penis, who cannot make sperm, is usually classified as a biological male, as long as the person does not possess female features such as a vagina, ovaries, or uterus. Based on evidence presented, to define male and female individuals in general society, we expand the defining characteristics of sex to include nongonadal traits, as well as classical gonadal traits.

A simple biological definition of male and female, satisfactory to all people, is elusive. In human societies, the terms *female* and *male* can have several meanings, as they refer both to a person’s biological sex and to their social roles. Most people learn to discriminate males and females from an early age, but often not based on biological traits (4). For example, behaviors such as pair-bonding, sexual activity, offspring defense and care, and mate/partner selection (5) involve complex interplay between sex steroid hormones and peptide hormones (oxytocin and arginine vasopressin); these behaviors are encouraged differently in women and men, which influences their role in the society and culture in which they live to behave as “females” or “males.” While these factors have little impact on their biological sex, they can have profoundly different outcomes in the behavior and health of an individual. Biological sex is dichotomous because of the different roles of each sex in reproduction. For scientific research, it is important to define biological sex and distinguish it from other meanings.

Sex Chromosomes and Biological Sex Determination

Among mammals and many other taxa, males are characterized as the heterogametic sex (6), having 2 different sex chromosomes, X and Y, whereas females are homogametic (XX). By contrast birds, many reptiles, and some other organisms have Z and W chromosomes (7). In these organisms, the female is the heterogametic sex (ZW) and males are homogametic (ZZ). Some adult fish and reptiles can also change sex in response to environmental factors (8, 9), and even the adult mouse gonad can undergo partial sex reversal when specific genes are deleted (10, 11). Human biological sex is often assessed by examining the individual’s complement of sex chromosomes as determined by karyotypic analysis: males are XY and females are XX. Karyotypic sex is actually a surrogate for genetic sex, determined by the presence of the *SRY* gene on the Y chromosome (12, 13). However, karyotypic analysis may be misleading, as there are well-described 46,XX males (with testes). Most of these individuals carry a short segment of the Y chromosome that includes *SRY* transferred to an X chromosome, but up to 10% lack an *SRY* gene (14, 15). Similarly, there are 46,XY females, who have *SRY* but also have a duplication of *DAX1* (dosage-sensitive sex reversal, adrenal hypoplasia critical region, on chromosome X, gene 1) (16).

Sex Determination and Sex Differentiation

In mammals, sex determination begins with the inheritance of XX or XY chromosomes, which are the only factors that are different in XX and XY zygotes. Thus, all phenotypic sex differences, including gonadal development, stem originally from the unequal effects of XX and XY

sex chromosomes. Phenotypic sex differences develop in XX and XY embryos as soon as transcription begins. The categories of X and Y genes that are unequally represented or expressed in male and female mammalian zygotes, which could cause phenotypic sex differences, fall into 3 main categories (17).

1. *Y genes causing male-specific effects.* These Y-linked genes do not have homologous genes on the X chromosome. The most important Y-linked gene is *SRY*, the testis-determining gene, which encodes the *SRY* transcription factor expressed during embryonic life in the bipotential gonadal ridge; *SRY* activates downstream autosomal genes such as *SOX9* to cause formation of a testis (18). In the absence of *SRY*, autosomal and X chromosome genes (*WNT-4*, *DAX-1*, *FOXL2*, *COUP-TFII*, and *RSPO1*) are activated to cause formation of an ovary (19–22). Both testicular and ovarian development are subject to active genetic regulation (12, 13, 16). Pathways downstream of *SRY* inhibit ovary-determining pathways, and ovary-determining pathways also inhibit pathways for testis development. Once the testes form, they secrete sex hormones that act widely throughout the body to cause male differentiation of nongonadal tissues. Other Y genes also have male-specific effects (for example, those required for spermatogenesis) (23, 24).
2. *X gene dosage or parental imprint.* Because XX nongermline cells inactivate one X chromosome (25, 26), it was long thought that both XX and XY cells have only one active X chromosome, with little inherent difference in expression related to the number of X chromosomes. The inactivated regions of the X chromosome are “coated” with large noncoding RNA transcribed from the X-inactive specific transcript (*XIST*) gene, part of the XIC (X inactivation center) located on Xq13 (27, 28). But some genes escape X inactivation (termed as *X escapees*), and therefore are expressed more in XX than XY cells, resulting in imbalance or incomplete dosage compensation (29). About 23% of human X-linked genes are more abundantly expressed in XX cells than XY cells in many tissues (30, 31). Recent evidence from mouse studies suggests that the inherent male-female difference in expression of X genes leads to significant sex differences in disease phenotypes. For example, sex differences in placental *Ogt* expression are associated with sex differences in prenatal vulnerability to stress (32). X escapee *Kdm6a*, a histone demethylase, contributes to sex differences in mouse models of bladder cancer (33), autoimmune disease (34), and Alzheimer disease (35). Similarly, variations in human *KDM6A* are associated with prognosis of bladder cancer or cognitive decline in female patients (33). The dose of another X escapee histone demethylase, *Kdm5c*,

contributes to sex differences in adiposity and body weight in mice, and variations in *KDM5C* in humans are associated with body mass (36).

Sex differences may also arise from genes in the pseudoautosomal regions (PARs) of the sex chromosomes, small regions of sequence similarity on the X and Y chromosomes that allow for X and Y chromosome pairing during meiosis. Both XX and XY cells have 2 PARs, implying equivalent effects of XX and XY PARs. Paradoxically, the process of X inactivation appears to spill over into the PAR and reduce expression on one X chromosome only in XX cells, leading to greater expression of PAR genes in XY cells compared to XX cells in the human transcriptome (30). A third potential source of X-linked imbalance stems from parentally imprinted genes in XX cells, which have one X chromosome from each parent and thus are influenced by any imprint on X genes from either parent. XY cells only receive imprints from the mother, and thus differ phenotypically from XX cells (37).

3. *XX mosaicism.* Female mammals are a mosaic of cells of 2 types: those expressing the X chromosome from the father (Xp), or from the mother (Xm) because of X inactivation (25). In contrast, XY individuals will lack this diversity within cell types in each organ because only one X (Xm) chromosome and only the maternal imprint of X genes will be expressed in each cell. The mosaicism in females means that in genetically diverse populations, the effects of disease-promoting X-linked alleles, inherited from one parent, will be muted in XX cells because half of the cells will have a different allele (38), and genomic imprints from each parent will only be expressed in half of the cells. In general, XX tissues are thought to have less extreme phenotypes than XY tissues, because the effects of extremely deleterious or beneficial alleles or imprints are buffered by the diversity of X alleles and imprints. For example, hemophilia A and hemophilia B (clotting factor VIII and IX deficiencies, respectively), are X-linked diseases that affect men, whereas most women are asymptomatic carriers.

Sexual Differentiation Caused by Gonadal and Nongonadal Hormones

In mammals, the process of reproductive system development requires the action of hormones (peptide/gonadotropins and steroids) from the pituitary gland, the adrenal cortex, and the gonads. Testicular development leads to secretion of Müllerian inhibitory hormone (MIH, also termed anti-Müllerian hormone, AMH), a glycopeptide, and testosterone, which affects many sex differences in nongonadal tissues (39). In contrast to the fetal testis, the fetal ovary makes minimal steroid hormones

(40), and ovarian function is not needed for development of the female reproductive system, as evidenced by the normal female anatomy of individuals with Turner syndrome, who have 45,X gonadal dysgenesis. The pioneering work of Alfred Jost suggested that 2 classes of testicular hormones are involved in sexual differentiation. First, testicular androgens drive the differentiation of the fetal external genitalia from female morphology to that of the male and are required for the differentiation of embryonic Wolffian ducts into male internal reproductive structures (41, 42). Androgens, secreted by Leydig cells, are required for the differentiation of embryonic Wolffian ducts into male internal reproductive structures (epididymis, vas deferens, ejaculatory ducts, prostate, and seminal vesicles), and drive the differentiation of the undifferentiated external genitalia toward male morphology. Second, the testis produces locally acting MIH that causes involution of the Müllerian ducts, which would otherwise develop into the fallopian tubes, uterus, and cervix (43, 44).

It was long thought that only the involution of the Müllerian ducts was an active process, with the Wolffian ducts simply involuting in the absence of androgens. Recent evidence from mice indicates that Wolffian involution is also an active process controlled by the transcription factor COUP-TFII (22, 45), but the nature of any factors stimulating COUP-TFII remains unknown (22). Some aspects of gonadal differentiation are active throughout life,

preventing ovarian follicle cells from transdifferentiating into “testis-like” cells (11). MIH is secreted by Sertoli cells and androgenic steroid hormones, usually testosterone, are secreted by Leydig cells. Testosterone and its more potent derivative dihydrotestosterone are responsible for the development of the male external genitalia (46). Androgens from adrenal glands and alternative pathway androgen biosynthesis in the human placenta can influence virilization of the developing fetus (47, 48). The adrenals of adult primates also produce abundant androgens, profoundly influencing phenotypes, so that not all sex steroids are gonadal (see Boxes 1 and 2). Although the term *sexual differentiation* is usually applied to the development of sex differences in genitalia and other organs such as the brain in the growing fetus; sex differences also occur later in life during the minipuberty of infancy (49), puberty, the female menstrual cycle, menopause in women, and andropause in men. The actions of gonadal and nongonadal hormones as well as sex and autosomal chromosome gene products in adult people causes many sex differences in health and disease.

Influence of Gonadal Steroid Hormones and Nongonadal Hormones in Brain Development

Differentiation of the brain by gonadal hormones is implemented during a restricted critical window, which is operationally defined by the onset of copious androgen

Box 1. Steroidogenesis in gonadal and nongonadal tissues

All biologically active sex steroids, whether gonadal or nongonadal in origin, are derived from cholesterol by the process of steroidogenesis. Two steroidogenic steps must be considered (for details see (50)). First, the cholesterol side-chain cleavage enzyme, P450_{scc} (CYP11A1) initiates steroidogenesis by converting cholesterol to pregnenolone; expression of P450_{scc} renders a tissue “steroidogenic,” that is, able to make steroids *de novo* (51). The gonads, adrenals, and placenta express abundant P450_{scc} and produce the familiar circulating endocrine steroids, but the brain, skin, and some other organs also express low levels of P450_{scc} and produce steroids involved in paracrine actions. Brain steroidogenesis has been studied mainly in fetal rodents, with little information in other systems (52). Many nonsteroidogenic tissues (liver, kidney, fat, breast, heart) do not express P450_{scc} but express other steroidogenic enzymes that modify steroids taken up from the circulation. Fat and breast express CYP19A1 (aromatase), permitting local production of estradiol from circulating 19-carbon (C19) steroids; this estradiol is important in breast cancer but is not a gonadal steroid. Similarly, prostate and genital skin express several enzymes leading to dihydrotestosterone, accounting for the failure of “androgen deprivation therapy” by gonadectomy in prostate cancer. Not all gonadal steroids are sex steroids, as both the ovary and testis secrete some “upstream” steroids that are precursors of the classic sex steroids. For example, dehydroepiandrosterone (DHEA) does not bind to sex steroid receptors, but it can be converted into testosterone and estrone. Second, synthesis of all sex steroids requires P450_{c17} (CYP17A1), which catalyzes 17 α -hydroxylation and the 17,20 lyase activity that changes 21-carbon steroids to C19 precursors of androgens and estrogens. P450_{c17} is abundantly expressed in the gonads of all vertebrates and in the adrenals of most vertebrates other than rodents, but the rodent *Cyp17A1* gene is silenced by tissue-specific methylation (53). Consequently, rodents make only miniscule amounts of adrenal C19 steroids and also use corticosterone instead of cortisol as their glucocorticoid. In most mammals, P450_{c17} has low 17,20 lyase activity, so that their adrenals produce rather small amounts of C19 steroids, but primate P450_{c17} has abundant 17,20 lyase activity, generating abundant C19 androgen precursors (DHEA, DHEA-sulfate, androstenedione) (47, 48). Furthermore, production of these C19 steroids proceeds by different pathways in rodents and primates: primates favor the “ Δ 5 pathway,” through DHEA, whereas rodents favor the “ Δ 4 pathway” through 17OH-progesterone (17OHP) (50). Primate adrenals also produce a true androgen, 11-keto-testosterone (54), profoundly influencing phenotypes (apocrine odor; female sexual hair). Thus, not all sex steroids are gonadal: ~ 50% of the circulating androgens in adult women are of adrenal origin.

Box 2. Gonadectomy and sex steroids

Many animal studies employ gonadectomy to eliminate the actions of sex steroids (estrogens, androgens, progestins). If using this approach, the investigator must consider whether nongonadal tissues will produce sufficient sex steroids to influence the study. The gonads produce most but not all circulating sex steroids; furthermore, some tissues produce steroids that act locally and do not enter the circulation, hence absence of a measurable steroids in blood does not ensure absence of its action in the target tissue. Both sexes produce all steroids and their metabolites, hence there are no male- or female-specific sex hormones, *per se*. In male mammals, testosterone release is highly pulsatile in nature (49, 55) and in laboratory mice, strain-dependent variations in androgen levels are reported (56). In female rodents, circulating levels of estradiol, testosterone, and DHT are highest in proestrus phase; a comprehensive analyses of sex steroids in intact and gonadectomized rodents can be found elsewhere (57). Circulating concentrations of testosterone in adult women are similar to those of boys in early puberty, and estradiol concentrations in men are similar to those in mid-cycle women, but the tenfold higher concentrations of testosterone obscure its effects. Rodents are widely used in research, but they differ from primates in several important aspects of steroidogenesis (see Box 1), and hence must be used with caution in studies seeking to model aspects of human physiology that might be influenced by steroids. These differences include: (i) In humans, substantial amounts of circulating sex steroids are bound to sex hormone-binding globulin (SHBG), whereas this carrier protein is not present in rodent circulation (58). (ii) Dehydroepiandrosterone (DHEA) and androstenedione, 19-carbon (C19) precursors for testosterone and estrone, that do not bind to sex steroid receptors, are secreted from the adrenal glands, the ovary and testis in humans, but not rodents (59). Thus, not all gonadal steroids are sex steroids. (iii) The rodent ovarian corpus luteum produces progesterone throughout pregnancy but in human pregnancy the corpus luteum involutes early in the second trimester, after which the placenta produces the progesterone needed to suppress uterine contractility, permitting term pregnancy. (iv) Adrenal-specific methylation of rodent *Cyp17A1* prohibits their adrenal synthesis of C19 precursors of sex steroids; however, changes in methylation status can occur under conditions of pathology. (v) As a further consequence of adrenal *Cyp17A1* methylation, rodents utilize corticosterone as their glucocorticoid, whereas almost all other vertebrates use cortisol. (vi) Rodent adrenals use high-density lipoproteins (HDL) taken up via scavenger receptor B1 (SRB1), as their principal source of cholesterol for steroidogenesis, whereas primates use low-density lipoproteins (LDL) taken up by receptor-mediated endocytosis. (vii) Several genes encoding steroidogenic enzymes are duplicated; rodents and primates differ in which copy(ies) of these genes are expressed: *CYP21*; *HSD3B*, *HSD17B*, *AKR1-3*. Such differences may affect laboratory results in unanticipated fashions. (viii) In rodents, nonsteroidogenic tissues such as the gut, liver, kidney, fat, breast, heart, thymus, skin, and the placenta have all been shown to make steroids. Thus, gonadectomy may eliminate most, but not all, circulating sex steroids, depending on the species being studied and may not reveal much about the paracrine effects of sex steroids present in the tissue(s) under investigation. Nonetheless, gonadectomy is an invaluable research tool that helps unequivocally confirm the influence of gonadal hormones in sex differences.

production from the fetal testis. Human fetal androgen production begins at 8 to 10 weeks postconception and in rodents is closer to parturition, at embryonic days 16 to 18, with birth following 2 to 4 days later. An important effect of this androgen surge is to masculinize the rodent brain. Steady but pulsatile release of the gonadotropins luteinizing hormone and follicle stimulating hormone from the pituitary gland support continuous steroidogenesis and production of sperm (60). In female rodents, the feminization of the brain proceeds in the absence of exposure to high levels of androgens or their aromatized byproducts, estrogens, a developmental strategy highly analogous to that used for masculinization of the gonads, reproductive tract, and secondary sexual characteristics, with the exception that estrogens are actively downregulated in male rodents. In human females, gonadotropins from the pituitary gland regulate ova development, induction of ovulation, and stimulation of estradiol and progesterone from the ovaries (49). An important feature of this developmental strategy is the existence of a sensitive period in female rodents (61). Male rodents must be exposed to high levels of

androgens during the critical period; if exposure occurs too early or too late it will be ineffective at inducing masculinization. However, females are also sensitive to androgens during a restricted period of development, hence a sensitive period in rodents. In males, the critical period closes shortly after androgen exposure because the cellular and molecular processes of masculinization have been initiated and cannot be reversed; the train has left the station. In both primates and rodents this process is largely prenatal, but female rodents remain sensitive to androgen exposure into the first postnatal week. Injecting a newborn female rodent with androgens will initiate the process of masculinization, thus she is still sensitive. After the first week, the feminization process cannot be overridden by androgens and thus the sensitive period has closed. The existence of the sensitive period in females is useful as a research tool—it is important in understanding the potential impact of exposure to endocrine-disrupting compounds or other cellular agents of masculinization that act in an analogous manner to androgen exposure in modulating female brain development. There is evidence for a later sensitive

period for brain feminization mediated by small increases in estrogens (62); this topic warrants further investigation. The closing of the sensitive period in primates, especially humans, remains poorly understood, but it appears to end prenatally, similar to the critical period in rodents. The sources of androgens that females can be exposed to during the sensitive period include from: (i) experimental interventions; (ii) male littermates in animals; (iii) or human adrenals carrying genetic mutations in the steroidogenic pathway (as in congenital adrenal hyperplasia).

Given that the critical and sensitive periods for sexual differentiation are defined by the production and response to gonadal steroids, it is not surprising that steroids are the primary drivers of developmental origins of sex differences in brain (and probably other tissues) and behavior. But how do steroids achieve this? The first step in any investigation is often is to identify the active steroid metabolite(s). In rodents, circulating fetal testicular testosterone enters the fetal brain where it can serve as a direct precursor for estradiol synthesis via aromatase (*Cyp19A1*) (see Box 1). Fetal and adult neurons can aromatize testosterone to estradiol in a nonrandom distribution: neurons of the hypothalamus, preoptic area, and amygdala are particularly active for local estradiol synthesis, whereas the hippocampus and parts of the cortex, midbrain, and spinal cord are also active at a lower level (63). For most reproductive endpoints, it is the local actions of estradiol that drive neural phenotype toward masculinization, which to some seems counterintuitive, given that estradiol is so often referred to as a “female” hormone (64), and further highlights that it is impossible to completely eliminate the effects of sex steroids, especially in the brain, by simple gonadectomy (see Box 2). Developing rodent embryos sequester maternal estrogens by binding to circulating alpha-fetoprotein, which is present only during the critical/sensitive period; when it is genetically deleted, all the offspring are masculinized (65). However, in humans, sex hormone-binding protein, not alpha-fetoprotein, is the major serum glycoprotein that binds androgens and estrogens with an undetermined role in fetal sexual development (66, 67).

In rodents, there is abundant evidence that gonadal androgens are metabolized to estrogens in the brain and mediate “masculinizing” effects on the brain; similar evidence in primates is limited. In primates, the principal masculinizing agents are androgens, not estrogens, and although there is alpha-fetoprotein present in fetal circulation, it has a weak binding affinity for estradiol (68), and instead it plays a much broader role in brain and body development (69). The conclusion of no strong role for estrogens in humans is based on individuals with dysfunctional aromatase or androgen receptors. Males lacking aromatase still identify as men,

while XY individuals with complete androgen insensitivity identify as women (70). The disparity between the principal differentiating hormones in primates versus rodents suggests that findings may not be easily extrapolated, and it is important to specify both the hormone and species under investigation. To discern whether the biological basis of sexual differentiation of brain and behavior differs between primates and rodents, one needs to identify mechanisms by which steroids transduce signals to modify the trajectory of the nervous system. While those mechanisms are incompletely understood, a few general principles are clear. First, there is no unified mechanism that applies broadly across the brain, with the exception that androgens and estrogens are the primary drivers of masculinization during a restricted developmental window. Similar masculinizing effects of testicular androgens may also occur during puberty (71). Second, all aspects of neural development are capable of being “organized” or programmed by sex steroids. This includes cell genesis, migration, myelination, dendritic and axonal growth and branching, synapse formation, synapse elimination, and neurochemical differentiation. Effects are not limited to neurons, with both astrocytes and microglia also exhibiting morphological sex differences. Third, each discrete brain region, nucleus, or subnucleus appears to have unique mechanisms of cellular masculinization. In some brain regions, such as the preoptic area, there are multiple separate mechanisms at play simultaneously. Sex steroids act in both paracrine and endocrine manners to influence structural development and function (72, 73).

Biological Basis of Diversity in Sexual/Gender Development and Orientation

Given the complexities of the biology of sexual determination and differentiation, it is not surprising that there are dozens of examples of variations or errors in these pathways associated with genetic mutations that are now well known to endocrinologists and geneticists (74); in medicine, these situations are generally termed *disorders of sexual development* (DSD) or *differences in sexual development* (75). DSD includes genetic disorders in the sexual determination pathway (76), disorders of steroidogenesis (50, 77), disorders of steroid hormone action, especially androgen insensitivity syndrome (78), and less well-defined “developmental field defects” (79), such as Mayer-Rokitansky-Küster-Hauser syndrome (80). The study of genes and factors underlying DSD and the diagnosis and management of the various forms of DSD is a complex and rapidly evolving area of endocrinology: clinical management is complex (81) and requires both contemporary molecular genetics (82) and well-integrated interdisciplinary care (83).

Gender includes perception of the individual as male, female, or other, both by the individual and by society. *Gender identity* is a psychological concept that refers to an individual's self-perception; while associations between gender identity, neuroanatomic, genetic, and hormone levels exist, a clear causative biological underpinning of gender identity remains to be demonstrated. Both animals and human beings have biological sex, but only humans have evident self-awareness that allows them to express gender; self-awareness in animals has not been investigated in this context. Gender also includes differences that males and females experience in their social and physical environments, which can have differentiating effects on the sexes. Human social environments are poorly modeled in laboratory animals and thus animal studies are usually limited to addressing sex differences. For centuries, the concept of male and female did not distinguish between biological sex differences and those caused by consistent differences in the environments. Thus *sex differences* are those caused by biological factors, whereas *gender differences* reflect a complex interplay of psychological, environmental, cultural, and biological factors (Fig. 1).

At birth, individuals are assigned a sex or gender ("natal gender"), almost always based on the appearance of the

external genitalia. In most individuals, the various biological determinants of sex are consistent with one another, and this biological sex is also consistent with the individual's self-perception—the sex and gender are concordant. However, a substantial minority of people who do not have DSD have some degree of variation in their self-perception of their gender, which may differ from their biological sex; this is usually termed *gender incongruence* (84). The term *gender disorder* has been replaced with the term *gender dysphoria* which describes the distress that an individual might feel as a consequence of having gender incongruence. *Transgender* (often called *trans*) refers to individuals who do not identify themselves as being of their natal gender, whereas *cisgender* (*cis*) people do not experience gender incongruence (85). Readers are also referred to Endocrine Society's 2017 Clinical Practice Guideline and Transgender Health Fact Sheet (84). Estimates of the prevalence of male-to-female transgender individuals among general populations range from 0.5% to 1.3% and estimates for female-to-male transgender individuals range from 0.4% to 1.2% (85). State level population-based surveys indicate that 0.6 % of US adults (25-64 years of age) and 0.7% of adolescents and young adults (13-24 years of age) identify as transgender. Other studies of US high school

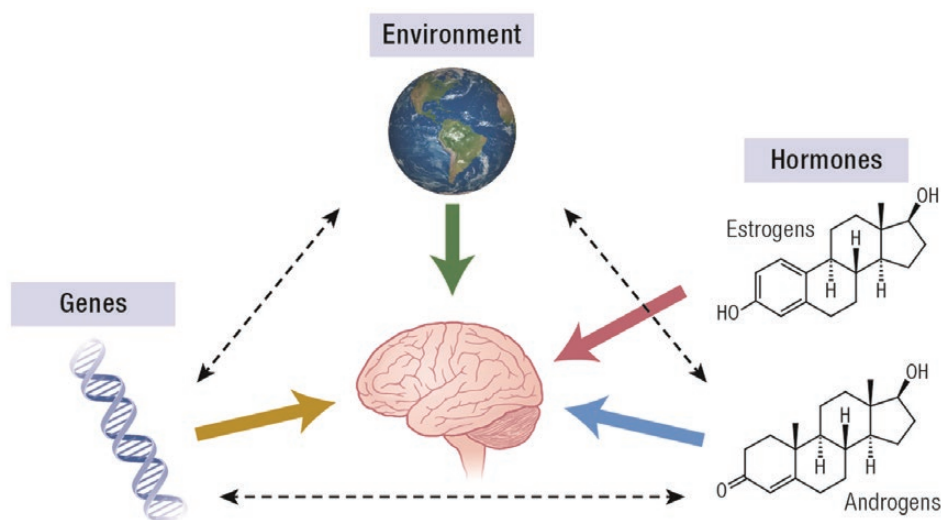


Figure 1. Simplified view of the factors influencing sex differences in the brain. Three broad groups of factors influence the sexually dimorphic brain, as indicated by the broad, colored arrows. 1) Genes and genetic factors that influence the brain include both those on sex chromosomes and autosomes, and include both the DNA itself (represented by the classic double helix) but also chemical modification of DNA (eg, methylation) and modifications of proteins associated with DNA to form chromatin, including histones, and also changes in proteins that bind to DNA. 2) Hormones clearly influence sexual dimorphism in the brain; these are represented by the principal sex steroids, estradiol and testosterone, but also include other steroid and protein hormones (progestins, MIH, oxytocin, prolactin, etc). 3) The environment includes a wide spectrum of influences, including perinatal nutrition and familial support, socioeconomic and demographic factors, intrinsic factors of brain development, age, and gender, and larger environmental factors, such as education, profession, and societal expectations (the "gendered environment"). In addition to each class of factor influencing the brain (bold arrows), the human brain also reciprocally influences each of these groups of factors. Furthermore, each group of factors influences the other, as represented by the dotted arrows. Some examples include: the environment influences genes via epigenomics and genes influence the environment by population sizes and domains; the environment influences hormones by seasonal variations and the actions of xenobiotics, and hormones influence the environment by promoting reproduction and consumption of foodstuffs; genes directly influence hormones by regulating their production and action, and many hormones, including all steroid hormones, regulate gene transcription.

students suggest a prevalence of 1.8% to 2.7% of being gender nonconforming or transgender (86–88). However, several factors may influence reported prevalence of gender dysphoria: (i) small sample sizes; (ii) differences in assessment techniques leading to incomplete ascertainment of gender dysphoric individuals; (iii) unwillingness of some individuals to respond fully and honestly, especially in older studies or studies deriving from locales where gender incongruence is a social taboo; (iv) differences in the subjects ages. *Sexual orientation*, not to be confused with gender identity, refers to the group of persons to whom an individual is sexually attracted; both cisgender and transgender individuals may be hetero-, homo-, or bi-sexual (89).

Although gender is strongly influenced by environmental and cultural forces, it is unknown if the choice to function in society in male, female, or other role(s) is also affected by biological factors (89–91). A general issue is that the association of sex, gender, or sexual orientation with specific brain structures, or with other biological variables, does not establish whether the biological variables are causes or consequences or noncausal correlates of the behavioral characteristics or function of the individuals studied. Three areas of biological difference have been studied fairly extensively: neuroanatomy, genetics, and hormones. Studies have reported differences in the hypothalamic INAH3 nucleus in men vs women and in homosexual vs heterosexual men (92, 93). Although initially controversial, others have confirmed sex differences in INAH3 numbers, not in size or densities, whereas no evidence for sexual dimorphism of any other INAH structures are reported (94). Studies in people with gender dysphoria found that the phenotypes of specific brain structures, such as the bed nucleus of the stria terminalis, of transgender women and transgender men differ from cisgender men and women, with partial, but incomplete sex reversal of sexually dimorphic structures (95). Brain networks involved in one's body perception, (pregenual anterior cingulate cortex, temporo-parietal junction, and fusiform body area) differ in individuals with gender dysphoria compared with cisgender individuals (96–98). Neuroimaging shows that testosterone treatment resulted in functional and structural changes in brain areas associated with self-referential and own body perception (99). Transgender men have thicker medial prefrontal cortex than cis men. Testosterone treatment does not change prefrontal cortex thickness in transgender men, but it has other effects on cortical thickness, connectivity, and fractional anisotropy (99).

Genetics may play a role in gender identity (100): monozygotic twins have 39% concordance for gender dysphoria (101). Attempts to identify specific genes governing gender identity have been plagued by small numbers of subjects and low statistical significance; no

specific gene has been reproducibly identified. However, such studies have suggested associations with genes encoding steroidogenic enzymes and sex steroid receptors, and it is generally agreed that androgens play an important but not determinative role. For example, many 46,XX individuals with severe virilizing congenital adrenal hyperplasia (steroid 21-hydroxylase deficiency) are exposed to intrauterine testosterone concentrations typical of those in normal male fetuses and consequently have severely virilized external genitalia; nevertheless, most have a female gender identity, but about 5% to 10% of such individuals have gender dysphoria, an atypical gender identity (89, 102, 103), or atypical sexual orientation and gender behavior (104, 105). Similarly, about half of 46,XY individuals with defects in androgen synthesis who were raised as females revert to a male gender role (106). The biological underpinnings of sexual orientation and gender identity are apparently related but are not the same (107). Thus, there is ample but incomplete evidence for biological substrates—neuroanatomic, genetic, and hormonal—for gender orientation, making this an important area of ongoing research.

Hormonal Versus Sex Chromosome Effects

Sex differences are caused by 3 major factors—sex hormones, genes on sex chromosomes/autosomes, and environment (Fig. 1). To understand disease mechanisms in both sexes and exploit sex differences in protection or exacerbation of diseases, it is important to determine the relative contribution of each of these factors in causing sex differences (17). Many sex differences caused by gonadal hormones have been discovered by measurements of sex steroids and gonadotropins during human development, and in animals by similar measurements or by interventional methods, such as gonadectomy, hormone administration, or the expression of synthetic enzymes or receptors in transgenic mice. Sex steroids play an integral part in many physiological processes (Box 1). Whereas the gonads are the major site of sex steroid synthesis, the adrenals, placenta, brain, and skin can also initiate steroidogenesis, and steroid-modifying enzymes are found elsewhere, especially in liver and fat, permitting synthesis of sex steroid hormones in multiple other sites (50). Thus, animal gonadectomy may provide information about endocrine effects of gonadal steroid hormones but cannot address tissue-specific paracrine effects (Box 2). Moreover, gonadectomy cannot mimic low pre-pubertal levels or physiological conditions in which hormone levels decrease, such as aging or menopause. Manipulations of human gonadal hormones are routinely used in contraception and in the management of sex steroid-dependent cancers (eg, breast, prostate). When

a sex difference is discovered in human disease, and modeled in animals, the investigation of possible hormonal causation of the sex difference is usually the first option considered.

To detect effects of sex chromosomes that cause sex differences, one can compare people who have differences in their sex chromosomes, revealing effects of X or Y chromosome number (108-110). These results strongly suggest direct sex chromosomal contributions to sex differences in cell function. Comparison of brains of XY patients with complete androgen insensitivity (who are phenotypically female), with brains of control XY males and XX females, suggests that cortical thickness and functional connectivity between the limbic regions and the cortex are influenced not only by testosterone actions, but by sex chromosome factors as well (111). However, changes in the sex chromosome ploidy also alter gonadal hormones, so it can be difficult to isolate sex chromosome effects not mediated by gonadal hormone effects. Circulating human embryonic/fetal sex steroid concentrations are poorly characterized, and the tissue concentrations are almost totally unknown. Another approach is to use mice to identify genes on the X or Y chromosome that act outside of the gonads to cause sex differences, and then seek evidence that the orthologous human genes cause human sex differences. Controlled experiments are possible in which XX or XY mice with comparable gonadal hormones can be compared. A frequently used model is the Four Core Genotypes (FCG) model, in which the testis-determining mouse *Sry* gene is deleted from the Y chromosome (creating the Y⁻ or “Y minus” chromosome) and inserted as a transgene on chromosome 3 (*Sry*+) (Fig. 2 and Box 3) (112). The utility and limitations of these models have been extensively discussed (113, 114).

Considering Sex and/or Gender as Variables in Health and Disease

Women and men differ in many physiological and psychological variables. It is important to establish the mechanisms causing such differences in health and disease, and to consider sex-related variables in studies of human health and disease. These variables include, but are not limited to, sex- and gender-related factors. The inability to control all variables in human studies means that it may be impossible to determine the relative roles of environment and biology in causing a difference between women and men, when both types of variable can influence the trait. Furthermore, while “gender expression/behavior” can be observed, “gender identity” can only be known by what an individual states. Thus, gender identity, *per se*, cannot be studied in animals. In human studies, it is unethical to selectively manipulate specific biological and environmental variables, and most currently available data derive

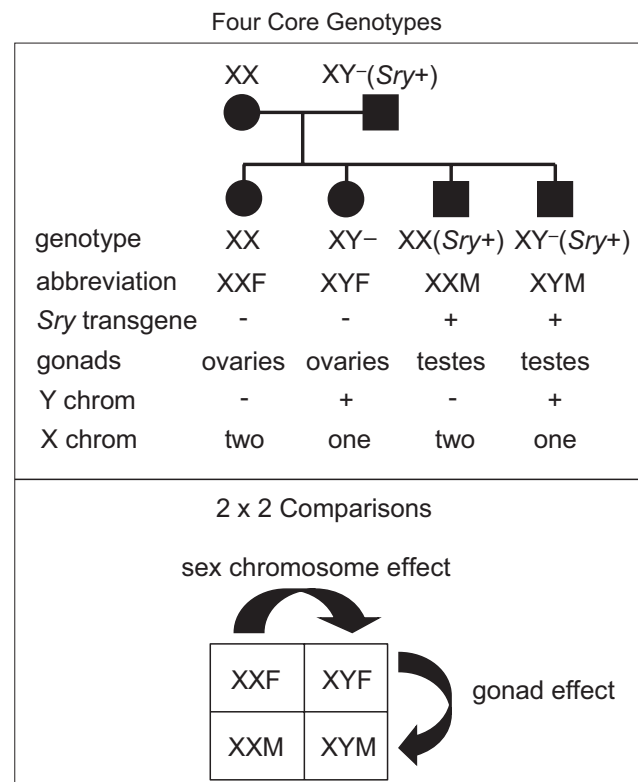


Figure 2. Schematic diagram of the Four Core Genotypes mouse model. The testis-determining gene *Sry* is deleted from the Y chromosome, producing the Y⁻ chromosome. An *Sry* transgene is inserted onto chromosome 3. Thus, the type of gonad is no longer linked to the sex chromosomes. The model produces XX and XY mice with *Sry* and testes, and XX and XY mice without *Sry*, with ovaries. Sex differences in phenotype can be attributed to an effect of gonadal hormones, comparing mice with ovaries and testes, or to an effect of sex chromosomes, comparing XX and XY mice with the same type of gonad. [Modified with permission from Arnold AP & Chen X. *Front Neuroendocrinol*, 2009; 30(1) © Elsevier Inc. (112)].

from studies comparing groups of men with groups of women. It is therefore difficult to disentangle the specific contribution of sex-related genes, hormones, gender-related variables, and other variables that contribute to being female or male. Because sex has long been defined by gonadal type, the list of sex-influencing factors has been primarily associated with gonadal hormones, especially estrogens, progestins, and androgens (121). However, some phenotypic sex differences develop before the gonads differentiate as testes or ovaries (122), so other factors also contribute to sex differences (123) but are seldom considered.

Sex is an essential part of vertebrate biology, but gender is a human phenomenon; sex often influences gender, but gender cannot influence sex. Studies of animal physiology must consider sex as a variable (124), with sex steroids (of both gonadal and nongonadal origins), sex chromosomes, and other factors contributing to sex differences in many physiologic processes. Similarly, studies of human physiology and disease must also consider sex for the same reason (125) and its disorders must

Box 3. Investigating sex chromosome complement versus gonadal hormones in health and disease: the four core genotypes (FCG) model

The FCG model allows for discriminating hormonal vs sex chromosome effects in animals. Gonadal males ($XY^{-}(Sry^{+})$), bred to XX gonadal females, produce 4 types of offspring: XY^{-} and XX mice with the *Sry* transgene and testes, and XY^{-} and XX gonadal females lacking the *Sry* gene (Fig. 2). Thus, it is possible to compare XX and XY mice with the same type of gonad, in 2 separate comparisons. Differences between XX and XY are attributed to effects of sex chromosome genes acting on nongonadal tissues. To determine if this sex chromosome effect is caused by X or Y genes, a second model is studied, the XY^{*} model (113, 114). This model produces genotypes that are similar to XO, XX, XY, and XXY. An effect of number of X chromosomes is discovered by comparing XO and XX, or XY and XXY. An effect of the Y chromosome genes is discovered by comparing XO and XY, or XX and XXY. These mouse models have been used to demonstrate sex chromosome effects causing sex differences in a wide variety of phenotypes and disease models, including brain and behavioral phenotypes, metabolism, autoimmune, cardiovascular and pulmonary diseases, Alzheimer disease, aging, and cancer (35, 113, 115). These models have facilitated discovery of several disease phenotypes in which the number of X chromosomes contributes to sex differences (116), and a smaller number of sex-biasing effects of Y genes (117). Sex chromosome effects occur in the same disease systems alongside sex-biasing effects of gonadal hormones, such that the 2 effects can synergize to increase the amount of sex difference, or counterbalance each other to reduce a sex difference. Moreover, genes encoded on the Y chromosome can have gene-specific effects, and/or effects that overlap with those of X genes (118). In the cardiovascular system and associated physiological/disease states, sex chromosomes and gonadal hormones can have opposing effects. Estrogens generally protect from cardiac ischemia/reperfusion injury and other cardiovascular diseases, reducing disease in female relative to male mice. However, studies of ischemia/reperfusion injury in gonadectomized FCG mice reveal that the XX sex chromosome complement is associated with worse outcomes, relative to XY (119). In another study, sex chromosome effects in angiotensin II-induced hypertension showed that arterial pressure was greater in gonadectomized XX mice than in gonadectomized XY mice (120). Sex chromosome complement also influences the development of abdominal aortic aneurysms, fat metabolism and adiposity, plasma lipids and lipoprotein levels (particularly HDL-C) (115).

also consider gender. However, human gender is a spectrum from feminine to gender-neutral to masculine, and also likely includes individuals who do not fit readily on a simple linear continuum (84). Studies addressing the endocrine care of transgender youth during the time of their potential gender transition (84, 89) find that they have a higher prevalence of stress-associated mental health disorders such as depression and anxiety, which can be ameliorated by gender-affirming endocrine treatment (126). It is essential to recognize these sex and gender differences as our health care systems endeavor to develop “individualized medicine.”

Despite the fact that biological sex is such a fundamental source of intraspecific variation in anatomy and physiology, much basic and clinical science has tended to focus studies on one sex (typically male). Few studies have done side-by-side testing for sex differences at baseline and in experimental models of human diseases (127-129). Studies in laboratory animals that manipulate biological (eg, genes and hormones) and environmental variables (eg, housing conditions, diet, physical activity, etc) demonstrate that many variables can affect sex-related aspects of an animal’s physiology. However, laboratory rodents may show male-female differences caused by different housing conditions, which could be misinterpreted as being caused directly by biological differences without environmental mediation. In studies concerning animal behavior, the sex and gender of the researcher conducting behavioral measures may also influence outcomes (130). Thus, for reproducibility and proper interpretation of the data, at the minimum, it is important to state the precise housing

conditions, anesthetics, analgesics (different effects in sexes), doses, surgical manipulations, diet, sex, strain, species, and age of animals used, as well as sex/gender of the researcher(s) performing experiments.

Having laid the foundation for several factors that contribute to sex versus gender, this Statement will use 3 areas of research as examples (not as a literature review) where human and animal sex differences are well known. First, sex differences in specific brain regions of healthy men and women are increasingly being documented along with differences in brain connectomes; these will be discussed in detail in Section II. Second, stress-related pathophysiologies are known to affect twice as many women as men. However, few studies systematically include study designs to ascertain function or mechanisms that may be similar or different between males and females. Hormones and signaling pathways that contribute to sex-specific differences in stress-based pathophysiologies will be discussed in Section III. Similarly, sex differences in manifestation of cardiovascular and renal diseases are well recognized and will be discussed in Section IV.

Section II

Developmental Origins of Sex Differences in Brain Anatomy, Function, and Behavior

Sex differences in the human brain are a topic of intense popular and scientific interest. Several scientific observations motivate the search for sex differences in brain structure

and function. First, the act of sexual reproduction requires that the male and female animals show qualitatively different reproductive behaviors. The stereotyped emergence of these reproductively critical and sexually differentiated behavior reflects biologically programmed (or “innate”) sex differences in the organization of those brain circuits that support the motivational and consummatory phases of copulatory behavior (131). Second, the fact that males and females make different biological investments in reproduction—eg, the risks of pregnancy in mammals are borne entirely by the female—sets up sex differences in the behavioral strategies that optimize reproductive fitness (132). Sexual selection based on sex-biased behavioral strategies is predicted to drive the evolution of sex differences in those brain circuits that are responsible for sexually selected behaviors. Third, males and females can show consistent sex biases in broader behavioral domains beyond those that directly relate to reproductive strategies. In our own species for example, there are highly consistent sex differences in the prevalence of physical aggression and violence (both male-biased) (133), as well as extensively documented sex differences in risk for different mental disorders (134).

In this section, we will first describe the main neuroimaging techniques commonly used in comparisons of brain anatomy, connectivity, function, and subnetwork organizations. We then review the key aspects of sex-biased brain anatomy and connectivity that have been revealed by these techniques; sex differences in stimulus-based or task-based functional magnetic resonance imaging (fMRI) studies are not addressed here. Next, we discuss specific disease states that appear to have different outcomes in the 2 sexes due to baseline differences in the “connectome” and animal models used in neuroimaging. Finally, we will address some important caveats and controversies in the field of brain imaging.

Brain Imaging Techniques

Modern neuroimaging methods make it possible to characterize diverse aspects of brain structure, function, and connectivity in vivo. This large toolbox of methods has been used to examine sex differences in brain organization at several levels of analysis. These techniques aim to analyze, map, and visualize regional and inter-regional (connectomic) features of the brain at macroscopic (systems-level) and mesoscopic (neural circuit architecture) levels in order to illuminate brain organization in health and disease (135). Of note, cellular-level details are beyond the resolution of most in vivo brain imaging techniques.

Sex differences in global and regional brain anatomy can be measured in vivo using structural magnetic resonance imaging (sMRI). Several considerations have made

sMRI an especially popular technique in the study of brain sex differences in humans. First, sMRI allows a quick and spatially comprehensive screen of the entire brain that can quantify thousands of morphometric properties simultaneously in vivo across a large number of individuals. These characteristics not only facilitate testing for sex differences outside defined regions of interest, but also allow longitudinal measurements that can track the emergence of brain sex differences over development (136, 137). Second, because sMRI considers structure rather than function, it can leverage evolutionary conservation of the basic mammalian brain plan (138), and it is therefore particularly well-suited for cross-species investigation of sex differences in humans and animals. Thus, a critical role for sMRI research in the study of brain sex differences is to screen for brain regions that can then be prioritized for closer analysis using more resource-intensive assays that are typically applied in a regionally selective manner.

Complimenting sMRI, other in vivo neuroimaging techniques such as diffusion MRI (dMRI), resting state functional MRI (rs-fMRI), and fMRI provide unprecedented insights into tissue microstructure and brain connectivity. fMRI maps brain circuitry based on stimulus- or task-based brain functional responses. In contrast, rs-fMRI, by measuring changes in blood flow in the brain generated by signals dependent on blood-oxygen-levels, helps explore the brain’s functional organization by providing insights into intrinsic brain activity without requiring participants to be trained in specific tasks, thereby eliminating task performance as a confounder (139, 140). dMRI measures the differential patterns of water diffusivity in biological tissue revealing details of tissue microstructure, especially in white matter (141). Fiber tractography on dMRI enables mapping the fiber architecture of the brain, and subsequently, the network organization of the brain through structural connectomes (142–144). A brain connectome is an extensive map of the white matter structural or functional connections of the brain, created using dMRI or rs-fMRI (145). Modeling efforts, such as the Human Connectome Project, and the use of connectome-based predictive modeling, have provided an integrative, in-depth, and multilevel understanding of the structural and functional connectivity (regions that get coactivated) of the neuronal networks (146, 147).

Sex Differences in Global and Regional Brain Anatomy

It is well established that men have an average total brain volume that is approximately 10% greater than that of women (148, 149). A similar sex difference in average

human brain volume (~8%) appears to be present at birth (150) and is sustained throughout childhood and adolescence (151). The sex differences for total brain volume also hold for the 2 main subdivisions of brain tissue—gray matter and white matter—despite these 2 brain compartments following very different developmental trajectories (151, 152) (Fig. 3).

The robust sex difference in brain volume identified through human sMRI research cannot be fully explained by the fact that brain volume is positively correlated with height (average height is greater in men than in women). Statistical control for body size diminishes, but does not remove, sex differences in total brain volume (149), and boys also show greater average brain volume than girls during early adolescent development, at a time when girls are taller than boys (153). Thus, available literature supports a consistent picture in which there is overlap between the distribution of brain size in men and women, but the mean of this distribution is significantly greater in men than women. The medium effect size of sex on brain volume exists above and beyond sex differences in stature. However, it is important to note that no known functional sex differences associate with the sex difference in overall brain size. Sex differences in overall brain size, and their developmental timing, are both theoretically and methodologically important when considering: (i) whether neuroanatomical sex differences are conserved across species; (ii) whether there are sex differences in regional brain anatomy above and beyond sex differences in overall brain size; and (iii) whether

there is concordance between sex differences in brain size and any observed associations between brain size and putative biological causes of sex differences, such as gonadal or sex chromosome status (see below).

The patterning of sex differences in behavior and mental illness risk across the lifespan suggest that sex differences in human brain organization are likely to vary across different brain sub-systems or regions, and potentially also across different developmental periods. Structures in human gray matter compartments mediate neural computation and information processing—in contrast to axon-rich white matter compartments that are primarily involved in connectivity between different brain regions (see “Sex Differences in Brain Network Organization: The Brain Connectome,” below). Here, we focus on sMRI studies that have tested for sex differences in regional gray matter volume (regional GMV) after controlling for sex differences in overall brain size. Regional GMV sex differences that survive statistical correction for total brain volume variation are of special interest because they exist beyond global sex differences in brain size. We emphasize GMV rather than other morphometric properties of the brain such as cortical thickness, sulcation, or the shape of subcortical structures (144, 154), because GMV provides a common metric that can be examined across cortical and subcortical structures, with equal applicability to humans and mice. Independent large-scale human sMRI studies in biobanks have identified a reproducible pattern of sex differences in regional GMV using sample sizes that are

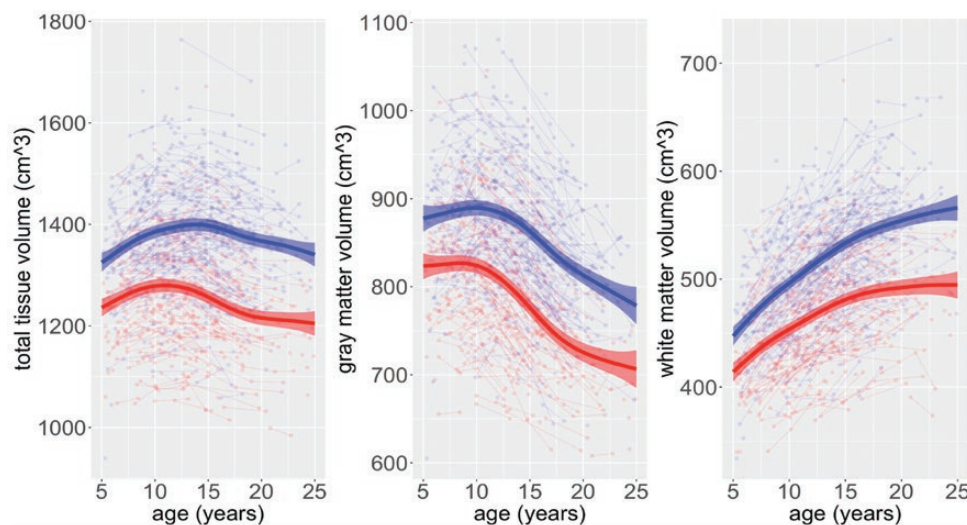


Figure 3. Developmental trajectories for total brain tissue volume, gray matter volume, and white matter volume in men and women over Development. Person-level data are shown for women (red) and men (blue) as points, with lines linking measures from the same person over time. Note the large interindividual variation in volumes within each sex, and the overlap of these distributions, between the sexes. Superimposed on these person-level data are group-level best fit volume trajectories (bold lines with shaded 95% confidence intervals). The developmental window covered is 5 to 25 years of age. For all plots, there are statistically significant sex differences in both trajectory shape (ie, sex differences in the tempo of volume change, $P < 0.00001$), and trajectory “height” (ie, sex differences in absolute volume across ages, $P < 0.00001$). [Adapted with permission from Giedd JN et al. *Neuropsychopharmacology*, 2015; 40 © Springer Nature (153)].

significantly larger than those used in earlier work (148, 149, 155). A structural neuroimaging study involving >2000 individuals demonstrated that higher regional expression of sex-linked genes was coupled with greater GMV in men relative to women (155). These studies, by different laboratories, using different datasets and different techniques for sMRI analysis, find a largely overlapping regional pattern of GMV sex differences after correction for sex differences in total brain volume. These independent replications of regional sex differences in GMV are also in agreement with meta-analytic studies (156). Together, these studies show that, in adulthood, regional GMV is (on average): (i) greater in women than men within superior parietal, dorsolateral frontal, and anterior cingulate cortices; and (ii) greater in men than women within occipital, fusiform, and parahippocampal cortices as well as the amygdala and putamen. Furthermore, while these studies lack temporally resolved developmental maps of male-female differences in regional GMV throughout the brain, there is extensive evidence from focused studies of particular structures that neuroanatomical sex differences can vary dynamically over development, such as observed with amygdala volume and shape (156).

The rapidly expanding body of sMRI research on regional GMV sex differences in the murine brain shows important overlaps and differences with findings from human studies (137, 157). These murine sMRI studies—which are most commonly conducted *ex vivo* at a spatial resolution of <100 μm throughout the whole brain—have been able to confirm the identification of all classically sexually dimorphic nuclei of male-biased volume from prior histological research, including the bed nucleus of the stria terminalis and medial amygdala (137, 157). These brain regions play a predominant role in modulating social and goal-directed behaviors, pain, and cardiovascular control, all of which are conserved among mammalian species and subject to sexually dimorphic outcomes. By allowing a full-brain screen, murine sMRI has also newly identified a reproducible set of regions with greater GMV in females, including the cerebellar cortex, ventral thalamus, and somatosensory cortex (137, 157). Furthermore, a longitudinal sMRI study in mice found that the set of regions with male-biased GMV can be detected by early postnatal life (with some accentuating over puberty), whereas regions of female-biased GMV in murine adulthood appear to emerge in adolescence (137). To date, there are no studies that formally seek to compare the spatiotemporal patterning of regional GMV sex differences in humans and mice, although existing work already suggests some potential homologies, including foci of greater cerebellar cortex GMV in females vs males by adulthood (137, 148) and the adolescent accentuation of male-biased amygdala volume (158, 159).

An important technical challenge in assessing the degree of anatomical homology between regions of sex-biased brain anatomy in humans and mice is that most of the best-established and histologically validated foci of sex-biased brain volume in mice (eg, bed nucleus stria terminalis, medial preoptic nucleus of the hypothalamus) are hard to image in humans due to their small size and intrinsic tissue contrast properties.

Sex Differences in Brain Network Organization: The Brain Connectome

The structural or functional brain network is represented by a “connectome,” wherein the structural or functional connectivity between coactivated regions is encoded either through fiber tracts or functional co-activations (160). These connectomes can be studied at the level of subnetworks like visuospatial, auditory, cognitive control, or macro-scale level through global measures of network segregation, integration, and efficiency, to obtain functional associations (161).

A study of 949 individuals (aged 8–22 years; 428 males and 521 females) showed that on average, there are significant differences between the sexes in their structural connectomes (Fig. 4) (162). On average, men had greater within-hemispheric connectivity, as well as enhanced network segregation, whereas between-hemispheric connectivity and network integration predominated in women (Fig. 4A), but these differences were most prominent during adolescence (Fig. 4B–4D). However, an opposite trend was seen for cerebellar connections, which developed differently between human males and females in adolescence and adulthood. The structural connectivity findings were consistent with a behavioral study conducted on the parent cohort (the above-mentioned imaging study was performed on a subset of participants), with women outperforming men on attention, word and face memory, and social cognition tasks, and men performing better on spatial processing and motor and sensorimotor speed tasks (163). An analysis of the Human Connectome Project rs-fMRI data identified age and sex as independent variables that contributed to differences in functional connectivity (164). In brains of men, functional connectivity was more clustered locally in all lobes, except in the cerebellum, whereas the brains of women showed a higher clustering coefficient at the whole-brain level. Thus, brains of men were classified as more segregated and brains of women as more integrated, which agrees with the structural connectivity findings (162). In connectomes, the identification of subnetwork properties (165) can reveal how the complex functional and behavioral repertoire emerges from the simultaneous processes of segregated neuronal clusters and their

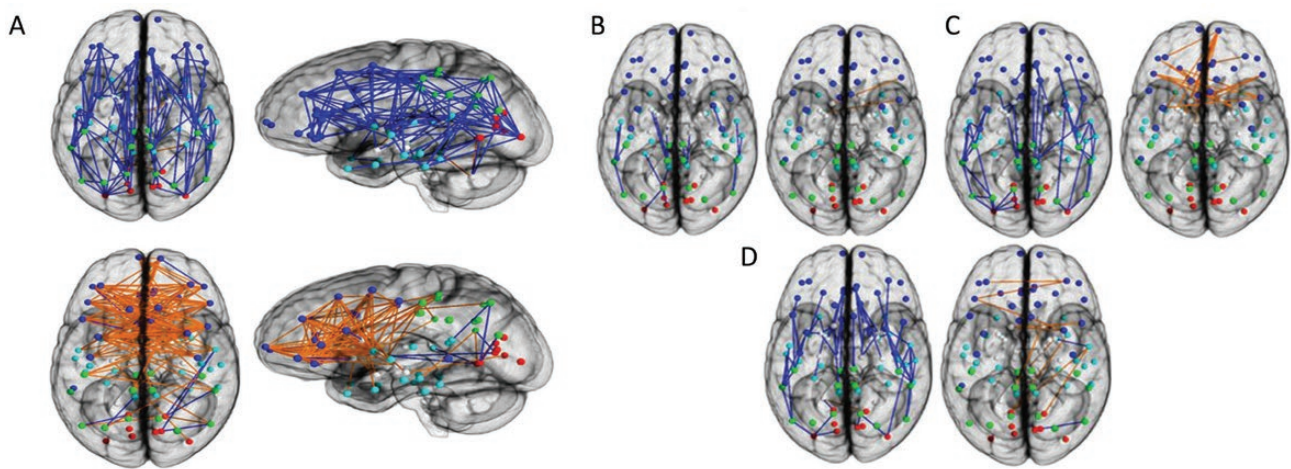


Figure 4. Sex differences in structural connectomes across development. Connectomes representing the white matter structural connectivity in the brain, with nodes indicating the brain regions and edges between the nodes representing the structural connectivity between the nodes. Node colors representing respective brain regions are as follows: dark blue, frontal; cyan, temporal; green, parietal; red, occipital; white, subcortical. The depicted edges shown are those that survived permutation testing at $P = 0.05$. **A**, shows increased intrahemispheric connectivity in men (Upper, in blue) and increased inter-hemispheric connectivity in women (Lower, in orange) on average. **B-D**: Connectivity differences shown in **A** separated by age groups are shown: **B**, under 13 years, **C**, adolescent (13-18 years), and **D**, young adults (18-22 years). Left image: Men/Boys; Right image: Women/Girls. [Adapted with permission from Ingahlhalikar M et al. *Proc Natl Acad Sci U S A*, 2014; 111(2) © National Academy of Sciences (163)].

integration during complicated cognitive tasks (166, 167). Consistent with the behavioral findings on sex differences, men had increased connectivity between motor and sensory (auditory) systems, along with increased connectivity in the fronto-parietal and cingulo-opercular systems that are traditionally associated with complex reasoning and control, whereas women had higher connectivity between reward, memory, and sensory (auditory) systems (163, 168). Better spatial skills in men and improved memory and social cognition skills in women have been reported in behavioral literature (169, 170).

It is important to point out that observed group-level differences in brain structure, function, or connectivity in men and women may reflect the influence of several extraneous factors. For example, in a set of elegant studies, brains of men were imaged to ascertain the contribution of performing complex spatial navigation tasks as part of their daily work on gray matter volume. These studies found that posterior hippocampi of London taxi drivers were significantly larger compared with controls (171), although the work did not address sex differences. Driving a taxi in London before the era of digital maps/navigation systems required extensive training and learning to navigate complex routes before being given a license to operate. In a subsequent study, comparison between London taxi drivers and bus drivers matched and controlled for age, education, intellectual, and stress levels, as well as years of driving experience, showed that taxi drivers had greater GMV in the posterior and less volume in the anterior hippocampi compared with bus drivers (172). Interestingly, years of

navigation experience associated with hippocampal volume in taxi drivers alone, but they were significantly worse at acquiring or retrieving novel visuo-spatial information than bus drivers. Importantly, no differences in other GMV, including the caudate nucleus, were found between the taxi and bus drivers; the caudate nucleus is associated with a myriad of cognitive and emotional functions. These studies illustrate brain plasticity and that professional work and years of performing certain tasks can result in brain structural, volume, and connectivity differences that may have little to do with sex or gender per se, but more with training, social environments, and behaviors. In other studies, GMV changes were greater in professional musicians, or after induced training (juggling for 3 months), and in early bilinguals, and white matter volume changes were found in adults learning a second language, irrespective of sex, when reported (173-176). These findings suggest that brain structure retains its plasticity and controlling for factors other than sex or gender are key in interpreting data on structural volumes and associated functions.

The above-mentioned existing datasets did not collect the requisite information on self-report of gender, thereby precluding retrospective analysis of gender in these cases. As identifying correspondence between behavioral scores and the regions that are involved in the manifestation of that behavior remains challenging, analyses of subnetworks pertaining to functional and behavioral domains can help elucidate a brain-behavior correspondence. The detailed description of sex differences in brain organization at the group level, and concerted efforts to specify

the role of sex-biased biological factors in shaping such sex differences, is of fundamental importance (177) and also provides a crucial adjunct for indispensable studies on environmental and wider societal contributions to sex-biased brain development. Such studies should be undertaken jointly using structural and functional connectivity. These studies elucidate the various ways in which sex differences in brain microstructure and connectivity can be investigated.

Sex Differences in Structural and Functional Brain Regions in Obesity

The hypothalamus has long been known as the “center” where peripheral and neural signals converge in the regulation of food intake and energy homeostasis in both sexes. Advances in neuroimaging studies have helped identify activation of several distinct brain regions comprising brain networks in response to eating in men and women. Behavioral and sociocultural factors may play a role in the observed sex differences in ingestive behaviors, appetite, and cravings related to obesity (178). Women report higher prevalence of maladaptive ingestive behaviors such as binge eating, food cravings, and “food addiction,” and the lifetime prevalence of disordered eating behaviors are about 3 times higher in women than in men (179, 180). Women also experience episodes of food cravings of greater intensity (181, 182), and greater frequency (183-185), and are less able to suppress food cravings than men (184, 186). Despite the wealth of data indicating that women experience disproportionately higher rates of food cravings, stress eating, and eating disorders than men, the reasons for these differences are incompletely understood (184, 187).

Regulation of food intake entails both homeostatic and nonhomeostatic factors (188). Homeostatic regulation balances energy needs with energy consumption, whereas nonhomeostatic regulation—in particular hedonic regulation and food addiction—involves reward-seeking behaviors that drive humans and animals to consume food beyond their metabolic needs, leading to the development of obesity (189-191). These findings have directed attention toward the extended reward system in obesity-related research, which consists mainly of basal ganglia regions and is involved in dopamine signaling and addiction-like behaviors (192). The extended reward system is composed of 6 interconnected brain networks—salience, central autonomic, basal ganglia, somatosensory, executive control, and emotional regulation (192).

Functional MRI studies have found that, in response to food images, obese individuals show greater activation than normal-weight individuals in regions associated with

reward anticipation, dopamine signaling, and addiction-like behaviors (193-196). Greater activity in brain regions of the extended reward network may drive obesity-related behaviors, such as greater responses to food odors and food consumption (197-199). Recent meta-analyses have further supported the role of the brain in disrupting the balance between energy consumption and expenditure. This combination of increased activity in regions associated with reward-driven behaviors and decreased activity in regions moderating top-down control of appetite may lead to consumption of excess calories (188).

Furthermore, sex-specific activations in response to food intake have been observed in cognitive, emotional, and reward-related regions (200-202). For example, obese men had greater activation than obese women in the supplementary motor area, precentral gyrus, fusiform gyrus, and inferior parietal lobule, which are associated with motor control, visuospatial attention, and responding to salient new or alerting stimuli (203). In this same study, obese women showed greater activation than obese men in the caudate and parahippocampal gyrus, regions implicated in reward processing and memory (203). Using graph theory to define the underlying architecture of brain structural connectivity obtained from diffusion tensor imaging, sex differences were observed in the topological measures of centrality (which determine the degree of information flow in specific brain regions) in regions of reward and salience networks in women, and in reward and sensorimotor networks in men (204). Resting state fMRI studies have found sex differences and commonalities in body mass index (BMI)-related connectivity associated with specific defined regions of interest in the reward network (205). For example, women had increased associations between BMI and increased connectivity in the in right globus pallidus and bilateral putamen. In men, BMI was associated with increased connectivity in the medial frontal cortex. A study of sex differences in response to visual and auditory food cues found that women experience greater activation in lateral and dorsolateral prefrontal and parietal cortical regions involved in cognitive planning and executive guidance and evaluation of behavior, compared with men (202). When viewed together, these studies highlight the importance of investigating sex differences in obesity-related alterations in the core and extended reward networks.

Although many single-sex studies of fMRI and obesity have been published, with the majority having all-female subjects, few studies have specifically investigated sex differences in brain function and structure in obesity. Despite the literature supporting sex differences in the brain, including in regions implicated in reward behaviors and energy homeostasis, few comprehensive reviews of sexually dimorphic brain signatures related to obesity have

been performed. A recent meta-analysis using an activation likelihood estimation approach to evaluate comparisons in functional responses to stimuli by obesity and by sex revealed differential sex- and BMI-related activations in reward anticipation and response, in shaping food-related memories, and in generating top-down control of appetitive processes. Together, these findings have important implications for sex-specific obesity treatments.

Models to Study Sex Differences in Normal Brain Structure and During Pathophysiology

Studies of sex differences offer important considerations for personalized medicine. The prevalence, clinical presentation, and symptomatic progression of many neurological and psychiatric disorders are remarkably different between the sexes. In addition to common X-lined mental retardation syndromes, men have a greater prevalence of neuropsychiatric disorders such as autism, attention-deficit/hyperactivity disorder (ADHD), and Tourette syndrome (206), whereas women have a greater prevalence of mood and eating disorders (207, 208). From the perspective of developmental disorders, the differences in the developmental trajectories of the sexes perhaps represent different vulnerabilities of maturing brain circuitry, leading to differences in symptoms, onset, and severity of neurological disorders. There are also sex differences in the risk factors, average age of onset, and prevalence of late-life dementias, as well as cerebrovascular disease (209). Additionally, in traumatic brain injuries, where the network organization of the brain is affected by the injury, such as the corpus callosum region, sex differences in inter-hemispheric connectivity and brain subnetworks may influence the impact of injury, and hence subsequent recovery. Thus, sex differences in brain connections are crucial to identify, as they may elucidate mechanisms in disease risk and potential treatment and recovery (210).

Most models of sex-biased mammalian brain development are based on experimental data from rodents (now largely from mice, but previously also from guinea pigs and rats). One of the most systematic dissociations of gonadal and chromosomal contributions to sex-biased anatomical brain organization in mammals is provided by a recent sMRI study of adult mice from the FCG model (112, 211). By combining sMRI with behavioral assays, these studies determined the contribution of sex chromosomes and gonads to adult mouse brain structure and function (211). This study revealed: (i) an effect of sex chromosomes on regional GMV in the cerebellar cortex and olfactory bulb; and (ii) an effect of gonads on regional GMV in the parietotemporal cortex and the bed nucleus of the stria terminalis. Some of these effects overlapped

with regions of normal sex differences in murine GMV (eg, cerebellar cortex and bed nucleus of the stria terminalis), and some brain regions were anatomically sensitive to both effects (basal forebrain and periaqueductal gray matter). Sex-chromosome effects on regional gray matter anatomy have also been reported by complementary sets of sMRI studies in both mice and humans that compare groups of euploid individuals with groups carrying X-chromosome aneuploidy (157, 212). Finally, in both mice (137) and humans (155), the spatial patterning of sex differences in regional GMV in adulthood appears to be preferentially aligned with the spatial patterning of sex-chromosome gene expression—which points toward a potential role of sex-linked genes in the establishment of maintenance of regional GMV sex differences. These studies emphasize the need for integrative models that view biological contribution to sex-biased brain development as a developmental dance of coordinated influences from both gonads and sex chromosomes.

Caveats and Critiques Relating to Neuroimaging of Brain Sex Differences

While several sMRI studies apparently establish that there are highly reproducible male-female differences in regional gray matter volume after controlling for variation in total brain size in humans, this conclusion should be considered in the light of several important caveats and critiques to avoid misinterpretation. First, all sMRI phenotypes that show reproducible and statistically significant sex differences also show a considerable overlap between men and women. This overlap is illustrated by total brain volume: total brain volume averages 10% greater in men than women, but many women have a total brain volume above the 30th centile for male brain volume, and many men have a total brain volume below the 30th centile for female brain volume (149). Sex differences in brain structure and organization are present across the lifespan and vary based on age, so inferences should be drawn cautiously. Thus, while total brain size shows a robust mean difference between men and women, an individual's total brain volume is a weak predictor of biological sex. These 2 facts arise because biological sex is only one source of variation in brain size (149), and other factors/variables that influence total brain size are unknown and/or hard to model statistically (Fig. 1). By extension, because sources of anatomical variation can differ between brain regions—the same individual can have GMV values that appear to be “sex-typical” in one region, but “sex-atypical” in another (when typical and atypical are defined by an individual's percentile position relative to the distribution of population-level trait variation in each sex) (213). This interpretation offers one

potential explanation for the observation that an individual brain can show varying degrees of GMV “sex-typicality” in different brain regions (relative to the population distribution). Alternative explanations have been proposed, including regional variations in programs of sex-biased development such that one individual’s brain may be considered a “mosaic” of male and female parts regardless of their chromosomal and/or gonadal sex (213).

Second, although sex differences in regional GMV are highly reproducible in humans and mice, these meso-anatomical sex differences *cannot* be assumed to correlate with behavioral sex differences. The functional relevance of neuroanatomical sex differences is hard to establish experimentally in humans, but correlations between anatomical and behavioral sex differences could be modeled in humans using several feasible study designs. To date, however, very few studies have directly tested for such structure-function correlations in humans (161), and this is an important priority area for future research. Several other challenges will need to be addressed in future work for any given sex-biased sMRI phenotype, including which aspects of behavior to measure and how to consider properly all possible configurations of brain-behavior association in 2 groups (eg, varying intercepts and/or regression slopes across groups). Moreover, some sex-biased sMRI phenotypes, such as trajectories of anatomical change, can only be estimated from group-level data, which complicates comparisons with interindividual variation in behavior. More fundamentally, however, regional GMV sex differences may be useful for understanding the brain basis for sex-biased behavior without GMV variation itself being the behaviorally relevant marker. For example, sex differences in mean regional GMV may help to define brain circuits that subserve sex-biased behaviors through their molecular, cellular, or connectivity features rather than through their volume *per se*. It is also important to entertain the possibility that sex differences in the anatomical organization of a given brain system may actually serve to equilibrate function between the sexes despite each sex having a categorically different genetic starting point.

Third, in addition to the functional considerations above, full understanding of a given sex bias in regional brain anatomy requires a mechanistic account that can link observed anatomical sex differences back to specific genetic and/or environmental factors that differ between men and women. It is usually impossible to disentangle biological sex differences from those which could be the result of environmental influences during development, differences in gender, and in sexual orientation

(Fig. 1). Strict causal tests for mechanistic models of sex-biased brain development are very hard to achieve in humans, although several informative approaches have been pursued including: (i) modeling sMRI data using normative variation in hypothalamic-pituitary-gonadal axis maturation or function (214); (ii) applying sMRI methods to cohorts undergoing gender-reassignment (215); and (iii) studying how sMRI features differ between typically developing groups and those affected by medical disorders involving the sex chromosomes (eg, sex chromosome aneuploidies) or sex steroids (eg, androgen insensitivity, congenital adrenal hyperplasia) (215, 216). However, the opportunistic and correlational nature of these approaches places considerable limits on the inferential power of mechanistic studies of human sex-biased brain development. Moreover, as challenging as it is to study chromosomal or gonadal factors in humans, it is even harder to address empirically the many plausible hypotheses about the potential for experiential and societal influences to differentially shape brain development in both sexes (121) or genders.

Section III

Sex Differences in Molecular Mechanisms Underlying Brain-Gut Disorders

The brain and the gut communicate with each other in a bidirectional way through parallel and interacting channels, involving immune, endocrine, and neural signaling mechanisms (217). The brain is able to modulate gut permeability, motility, intestinal transit, and microbial function via the autonomic nervous system (217), and the gut in turn sends signals to the brain to modulate behavior, in rodents (218). This brain-gut communication is especially critical in mediating stress responses and in stress-based disorders. In psychiatric and other neurological diseases, there are notable sex differences that point to different underlying neurobiological mechanisms in men vs women (219–221). Despite their clear documentation, these sex differences have largely been ignored, in order to develop broadly applicable pharmacotherapies that come at a considerable cost, especially for women’s health (222, 223). Sex biases in psychiatric risk are particularly instructive as they are developmentally patterned in a manner that is highly reproducible across different cultural settings and historical epochs: early-onset neurodevelopmental and gut disorders are more prevalent in boys than girls, while the opposite sex-bias is seen for adolescent-emergent mood disorders (134, 224). Brain-gut disorders are more prevalent in women than men, but this may be due to underreporting by men due to social stigma associated with several of these

disorders. The etiologies and risk factors for several brain-gut disorders differ between the sexes, yet study designs include predominantly male sex. In this section, we discuss the possibilities that shared and distinct mechanisms operate in males and females resulting in similar as well as distinct manifestation of symptoms for a given disease/disorder.

Sex-Related Differences in Obesity

Although prevalence rates for obesity are at unprecedented levels in all ages (225) and are almost equal in men and women (except when stratified by race or ethnicity) (226), recent surveys indicate an increase in the incidence of obesity in adults and sex differences in the associations between weight, physical health, and psychosocial functions (227, 228). Sex differences in body fat distribution have also been observed (178, 229), with women showing an increased propensity to gain total body fat, especially subcutaneous abdominal fat, whereas men tend to have more visceral adipose fat (230), which is associated with higher risks of type 2 diabetes, hypertension, dyslipidemia, and cardiovascular disease (231). Most clinical trials do not report sex differences related to health outcomes or treatment responses, but a few existing reports suggest women are less likely to complete treatment, tend to lose less weight than men, have a greater number of unsuccessful attempts to maintain weight loss resulting in the well-known “yoyo” diet phenomenon, and have limited responses to pharmacological treatments (225). Obesity-related studies in humans and rodents have expanded in scope to not only focus on structural and functional brain differences between obese and lean male and females, but also include investigations into the bidirectional signaling associated with the brain-gut microbiome axis (232, 233). In obese individuals, changes in the relative abundance and gut microbial diversity have been linked to changes in metabolism, insulin resistance, inflammation, and fat deposition (234). The importance of the intestinal microbiome to human health has been of interest over the past few decades, with multiple studies now linking the microbiome to energy homeostasis, immune function, and development of obesity and metabolic syndrome (235–237), even though few studies have addressed causality.

Not only does the brain-gut axis demonstrate changes in obese individuals, but evidence also highlights differences in the microbiota based on sex hormones (238). More recently, the effect of sex hormones on the composition of the gut microbiota has been explored, with differences seen in the microbiota between men and women during various stages of human development and maturation (238). These

sexually dimorphic microbiome signatures are likely to contribute to differences in susceptibility to autoimmune and metabolic diseases between the sexes. Studies performed in immunocompromised mouse models have shown delayed onset and lessened severity of type 1 diabetes in female mice who receive male microbiota transplants; testosterone activity and androgen receptor signaling was essential for this protection (239, 240).

These sex-specific differences in the microbial communities persist throughout adult development, with murine models demonstrating the role of testosterone in orchestrating these divergences in host selection of microbial communities (240). In rodents, males exhibit lower microbiome variability relative to females, likely due to the pulsatile nature of estrogens (240). Human studies comparing the microbiome of twins also revealed more divergences in microbial composition in opposite-sex versus same-sex twins (241). When the cecal contents from adult male mice is transferred into female mice, metabolomic profile changes and masculinization of the hormonal profile results, suggesting the gut microbiota’s influence on sex-specific metabolic and behavioral phenotypes (239, 242).

Circulating estrogens in the body are metabolized by the liver and undergo methylation, hydroxylation, and conjugation reactions to produce metabolites that affect host metabolism (243). Certain metabolites are excreted through the bile and are further processed by microbial enzymes in the distal small and large intestine. Certain microbial species secrete beta-glucuronidase, an enzyme that deconjugates biliary estrogen metabolites and allows for its reabsorption into the bloodstream to act on distal sites through binding of estrogen receptors (244). Dysbiosis and decreased microbial diversity result in decreased production of absorbable estrogen metabolites. This mechanism has been implicated in pathologies associated with low circulating estrogens, such as obesity, metabolic syndrome, cardiovascular disease, and cognitive decline in women (245, 246); however, estrogen replacement therapy does not reverse these conditions (247). Growth hormone similarly contributes to sexually dimorphic responses in the above-mentioned diseases (248). In addition, estrogens modulate inflammatory pathways driving disease processes such as nonalcoholic fatty liver disease (NAFLD) and type 2 diabetes (249, 250). More specifically, estrogens regulate adipokines and lipopolysaccharides, which respectively are adipocyte-derived hormones and endotoxins that have been associated with type 2 diabetes (251). Adipokines play a role in metabolic homeostasis as well as in mediating the beneficial and detrimental effects of inflammation (252). The androgen- and estrogen-dependent regulation of adipokines, including leptin, resistin, adiponectin, and visfatin, provides a possible mechanistic link between metabolic disorders (obesity,

atherosclerosis, insulin resistance) and autoimmune dysfunction. The estrogen-microbiome axis can provide a potential avenue for a sex-specific approach to combating metabolic disorders and highlights the bidirectional interaction of estrogens and microbial communities in the pathogenesis of disease processes.

Although the exact signaling mechanisms underlying the communication within the brain-gut-microbiome axis remain incompletely understood, tryptophan metabolites have been implicated as important signaling molecules (253). The most extensively studied tryptophan metabolite is serotonin (5-HT), a molecule with diverse roles in both the gastrointestinal tract (ie, peristalsis, secretion, and absorption) and the central nervous system (ie, mood, pain modulation, behavior, sleep, and ingestive and cognitive functions) (254). Tryptophan also acts as a precursor to the kynurenine (KYN) family of molecules (255). In obesity, the KYN pathway is preferentially activated and may contribute to immune-mediated inflammation, which may drive inflammation-associated changes to the extended reward network described in previous brain studies, particularly changes involving the amygdala and lateral orbitofrontal cortex (256-259). KYN may also modulate signaling within the brain-gut-microbiome axis through downstream neuroactive metabolites, such as kynurenic acid and quinolinic acid, functioning as N-methyl-D-aspartate (NMDA) antagonists and NMDA excitotoxins, respectively (260). Sex differences have been reported in these metabolite products in obese individuals, with lower tryptophan levels but elevated KYN and KYN/tryptophan ratios in women with high BMI compared to men with high BMI (256, 261, 262).

Sex Differences in Stress-Based (Patho) Physiologies

Epidemiological data reveal that the majority of psychiatric disorders occur at different rates in men and women. For example, men are more likely to suffer from attention-deficit/hyperactivity disorder (ADHD), whereas women are more likely to suffer from major depression and posttraumatic stress disorder (PTSD) (219, 263-265). Even when the rates of disorders are similar, their presentations can differ. Schizophrenia, for example, is only slightly more common in men than women, but men develop schizophrenia at an earlier age and present with more negative symptoms, such as social withdrawal and lack of motivation. (224). In the case of bipolar disorder, rates are similar between the sexes, but women more often have more rapid cycling and mixed episodes and they report higher comorbidity with eating disorders and PTSD, whereas men report higher comorbidity with alcoholism (266). Not only does the risk

and presentation of psychiatric disorders vary between men and women, but there are differences in treatment responses. For example, the efficacy of antidepressants differs between the sexes: men respond better to tricyclic antidepressants, whereas women respond better to selective serotonin reuptake inhibitors (267, 268). These findings implicate neurobiological sex differences in contributing to disease. In support of this idea, recent studies using animal models are beginning to uncover molecular processes that can bias males and females toward different pathology. Findings from some of these basic research studies will be highlighted here as examples of how including sex as a biological variable can inform our understanding of the etiology of stress-based disorders, as well as guide the development of better treatments.

While there are sex differences in rodent studies in the structure and the size of certain brain regions that can contribute to sex differences in behavior (211), imaging studies that focused on sex differences in cortical thickness and gyration suggest a role for these brain regions in humans as well. In adolescent girls, cortical thinning in the right temporal regions, the left temporoparietal junction and the left orbitofrontal cortex is faster than in boys (154). In contrast, changes in cortical folding were only found in one cluster of the right prefrontal region, suggesting that the mechanisms underlying changes in cortical thickness and gyrification in adolescents are distinct. Sexual dimorphism in the developmental course of the cortical maturation, which coincides with the onset of puberty, might explain sex differences in the age of onset and clinical presentation of many psychiatric disorders (154). Recent evidence has revealed that molecular sex differences in the brain are more widespread than initially thought and such seemingly small-scale differences can have a large impact on physiology and behavior (269). Neurons typically communicate with each other via neurotransmitters and neuropeptides, which are released from a presynaptic neuron and travel across a synapse to bind to receptors on the postsynaptic neuron to exert downstream cellular effects. There are sex differences in production and release of many neurotransmitters and neuropeptides that can result in behavioral changes. In other instances, sex differences in these systems are compensatory, leading to similar behavior endpoints via different mechanisms. For example, both male and female juvenile rats play, but the release of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) into the lateral septum mediates juvenile play only in female rats (270). There are also sex differences in receptors that can influence how these neurochemicals affect their downstream targets. For instance, dopamine 1 (D1) receptors, which belong to the family of G protein-coupled receptors (GPCRs), in the nucleus accumbens, are necessary for social

withdrawal in female but not male California mice (271). The function of GPCRs is often complex and they can induce different downstream effects depending on their conformation and location. Sex differences can occur at each level of receptor function, in some cases altering physiology differently in male vs female rodents. Sex differences in GPCR signaling are particularly important to consider, especially given that GPCRs are the most studied drug target family for a myriad of indications; in fact, 34% of all US Food and Drug Administration (FDA)-approved drugs are targets of GPCRs (272). As an example of the myriad of sex differences that can be mediated by receptors, we will use the corticotropin-releasing factor 1 and 2 (CRF₁ and CRF₂, respectively) receptors that facilitate responses to stress, exhibit sexually dimorphic expression pattern, are modulated by both estrogens and androgens, and have been relatively well characterized in both sexes (273, 274).

Upon perception of stress or perturbation of homeostasis, CRF is synthesized in the paraventricular nucleus and released from the median eminence of the hypothalamus into the pituitary portal circulation, which in turn stimulates the synthesis and secretion of adrenocorticotrophic hormone (ACTH) from the anterior pituitary into the general circulation. ACTH acts on the adrenal cortex to stimulate the synthesis and release of glucocorticoids and other steroids. This activation of the HPA axis in the classic “flight or fight” response by the CRF system is present in all mammals. The mammalian CRF family comprises 4 agonists, CRF and 3 urocortins (UCN1-3); and 2 known class B GPCRs, CRF₁ and CRF₂. While CRF₁ and CRF₂ share ~68% identity at the amino acid level (275), they perform distinct functions; CRF binding to CRF₁ initiates stress responses by activating the HPA axis, whereas UCN1-3 binding to CRF₂ brings systems back to homeostasis (274). Not surprisingly, perturbations in the components of the CRF family impact several organs and lead to brain-gut disorders, type 2 diabetes, metabolic syndrome, cardiovascular, and reproductive diseases, among others (274). There are sex differences in CRF’s endocrine effects. In female rats, higher levels of CRF mRNA in the paraventricular nucleus are reported that associate with the estrous cycle (276, 277) and are reviewed elsewhere (274). Perhaps as a compensatory response, CRF binding protein, an endogenous protein that sequesters CRF thus preventing its bioavailability, is expressed at higher levels in the pituitary of female compared with male mice (278). In humans, there is evidence for increased CRF receptor sensitivity at the level of the pituitary of women relative to men, because peripherally administered CRF, which acts at the pituitary, increases ACTH to a greater degree in women (279).

During stress, CRF is also released centrally into many brain regions, where its neuromodulatory effects coordinate cognitive and behavioral changes to promote stress coping (280). There are sex differences in the way these brain regions respond to CRF that are largely due to sex differences in CRF receptor signaling (274). For example, there is greater CRF₁ receptor binding in the basolateral amygdala in female rats (281). In contrast, binding of the CRF₂ receptor subtype, which is involved in stress recovery, is greater in the central nucleus of the amygdala in male rats (281). It is unknown precisely how these sex differences affect behavior, but given that the amygdala is critically involved in fear, it is likely that these receptor sex differences differently alter fear processing in males and females. In the brain, CRF₂ is most abundant in the bed nucleus of the stria terminalis, a region that regulates sexual behavior and stress-related functions (282, 283). Promoters in genes for CRF₁ and CRF₂ receptors harbor estrogen and androgen responsive elements and show tissue-specific modulation by sex hormones (284, 285). The sexually dimorphic expression pattern of these receptors at normal physiological states and during stress or disease pathology are summarized in a recent review (274).

Sex differences in CRF₁ receptor signaling have been identified in the noradrenergic-containing nucleus of the locus coeruleus (LC) and these differences have important implications for understanding disease vulnerability (273). The LC-noradrenergic system regulates levels of arousal such that higher levels of norepinephrin are associated with greater levels of arousal (286-289). Stressor exposure causes CRF to be released into the LC, which speeds up LC neuronal firing, increasing norepinephrin release (290, 291). Activation of this system during an acute or moderate stressor is thought to be adaptive, because it is important to be alert during a stressful event. However, if this system is activated inappropriately or persistently it can lead to hyperarousal that contributes to agitation, restlessness, impaired concentration, and sleep disturbance. Hyperarousal is a key feature of PTSD and reported in a subset of depressed patients (292, 293). Similar sex differences in spatiotemporal expression of CRF₂ and its ligands are found in humans with gut disorders, where they could contribute to differences between males and females in vulnerability to brain-gut disorders (127, 294).

There are sex differences in CRF₁ receptor signaling in the LC that increase female sensitivity to CRF. In the LC, CRF receptors primarily couple to Gs to initiate signaling through the cyclic adenosine monophosphate (cAMP)-protein kinase A (PKA) signaling pathway (295-297). Sex differences in CRF₁-induced cAMP-PKA signaling are linked to greater coupling of the CRF₁ receptor to Gs in females compared to males (298). This sex difference in

coupling of Gs may indicate that the CRF₁ receptor has a different conformation or binding partner in females vs. males, permitting different proteins to preferentially bind in each sex. Further support for this idea comes from studies demonstrating that, in male rats, acute swim stress increases the binding of a different protein, β -arrestin2, to the CRF₁ receptor, and this effect is not observed in female rats (298). The increased β -arrestin2 in male rats likely contributes to the greater CRF₁ receptor internalization in stressed males (298). When taken together, these findings suggest that CRF₁ receptors preferentially signal through different pathways in males (small GTPases) and females (cAMP-PKA) (299). This difference in signaling could alter physiology and disease risk. In fact, sex differences in CRF₁ receptor signaling in cortex were linked to increased Alzheimer-related pathology, including increased tau phosphorylation and amyloid β signaling in female compared with male mice (300). Few studies investigate sex differences in GPCR signaling, but it is likely that sex differences in GPCRs are also found in receptors other than CRF and that these differences could confer vulnerability and resilience to many diseases.

In human studies, single nucleotide polymorphisms in the CRF receptor gene (*CRHR2*) are associated with negative emotions in patients with irritable bowel syndrome (IBS) (301). Immune cells secrete CRF₂ in extracellular vesicles that circulate in the plasma and associate negatively with disease severity scores in IBS-diarrhea patients (294). Single nucleotide polymorphisms in *CRHR2* are also associated with lifetime PTSD in women (302) and with type 2 diabetes (303). The prevalence of type 2 diabetes and insulin resistance is greater in men (304). Epidemiological studies have shown that men with high levels of self-reported perceived stress have a 1.4 higher odds ratio of developing type 2 diabetes during a 10-year follow-up period and are at 2-fold higher risk of developing diabetes than women with similar levels of reported stress (305). In agreement with human data, male mice lacking functional stress receptors (*Crhr2*^{-/-}) and haploinsufficient (*Crhr2*^{+/-}) mice have worse glucose and insulin tolerance, microvesicular hepatic steatosis, and dyslipidemia than female *Crhr2*^{-/-} or C57BL/6 male and female mice in a high-fat diet-induced model of diabetes (129). Female *Crhr2*^{-/-} mice had significantly greater brown adipose fat mass on high-fat diet than C57BL/6 female or male mice of either genotype, suggesting greater thermogenic responses that might be protective. However, the mouse study did not address whether steroid hormones contributed to changes in adipose mass or function. Thermogenesis in brown adipose tissue in humans in response to a meal or cold stress suggests that women have greater thermogenic responses

than men and that these responses correlate positively with progesterone levels, but negatively with cortisol levels (306). Thus, analyzing data from both sexes provides insights into sex-specific mechanisms that regulate physiological processes in both sexes.

In colonic tissues of pediatric patients with Crohn's disease, subcellular localization of CRF₂ differs between boys and girls (127). Furthermore, lack of CRF₂ revealed several sex-specific signaling pathways and differential degree of inflammatory responses in male and female mice (127). Treatment with UCN1, a high-affinity agonist for both CRF receptors, rescued *Crhr2*^{-/-} male mice from colitis-induced mortality, whereas UCN1 treatment increased mortality in *Crhr2*^{-/-} female mice (127). Both diabetes and Crohn's disease show sex differences in disease prevalence and outcomes, yet most animal studies use male sex to delineate mechanisms. Analysis of the data by segregating the 2 sexes can reveal significant insights into distinct and shared mechanisms and factors that exist at baseline and during disease. For example, sex differences exist in the etiology of pancreatitis: alcohol and tobacco predominate in men, whereas idiopathic and obstructive etiologies predominate in women (307), yet to date only a few studies have used both sexes to study mechanisms involved in pancreatitis. While both males and females develop pancreatitis in animal models, when administered identical doses of the pancreatic stressor caerulein, C57BL/6 female mice show less severe pancreatitis and histological damage than male mice (128). Lack of CRF₂ rendered female mice more susceptible to caerulein-induced pancreatitis compared with male *Crhr2*^{-/-} mice (128), with both male and female *Crhr2*^{-/-} mice exhibiting similar levels of total histological damage (128). Detailed analysis of components contributing to histopathological damage showed that female C57BL/6J mice have less necrosis, zymogen granules, and vacuolization than male mice with pancreatitis, but they have similar levels of edema and neutrophil infiltration as male mice (128). This data segregation allowed isolation of factors that differentially contribute to histological damage, which otherwise would be lost, if grouped together in this analysis. Taken together, these data support a role for the CRF receptors, product of an autosomal gene and regulated by steroid hormones to bring about sex-specific cellular signaling and function.

Sex Differences in Pharmacotherapy of Stress-Based Diseases

Sex differences in GPCR signaling are also relevant for pharmacology. Biased ligands can shift signaling toward

β -arrestin pathways and away from G-protein-mediated pathways based on how they bind to the GPCR (308). These biased ligands are being designed with the hope of providing more targeted therapies with fewer side effects (308, 309). Understanding sex differences in signaling and how such differences contribute to changes in physiology can inform the development of these biased ligands. For example, a CRF₁ receptor ligand that biases signaling through β -arrestin pathways may be useful for treating hyperarousal symptoms or reducing the progression of Alzheimer disease, especially in women. An idea for such a compound would never have come about if women were excluded from preclinical and clinical studies on CRF₁ receptor function.

The idea of using CRF₁ antagonists to treat depression, PTSD, and irritable bowel syndrome has been around for decades, but these compounds were ineffective in several clinical trials (222, 310). Sex differences in CRF₁ and CRF₂ receptor signaling may also explain the failure of different selective CRF₁ antagonists as treatments for these disorders. While there are likely many reasons for their failure, critical ones could be sex differences in their target, association of CRF receptors with different binding partners in female versus male cells, or heteromerization of CRF receptors (311-313), all of which can result in altered signaling. The consistent efficacy of CRF₁ antagonists in reducing anxiety-like and depressive-like behavior in rodents and nonhuman primates was established in studies primarily conducted in male animals (222, 314-317). In a study in which females were included, local blockade of CRF₁ receptors in the dorsal raphe with an antagonist reduced anxiety in male but not female mice, highlighting sex differences in efficacy (318). Yet these compounds developed primarily in male rodents were tested in clinical trials with participants of both sexes or only in women. Notably the only CRF₁ antagonist study that had success in reducing depressive symptoms, NBI-34041, was conducted only in men (222, 319). The approach of developing compounds in male animal models is not unique to CRF₁ antagonists and has been common practice (222). Collectively, these studies suggest that a failure of certain therapeutics may result from ignoring sex differences in their targets. Sex differences in targets are not well known because most preclinical studies use only male rodents (320, 321). Excluding females in the drug development stage particularly impacts women's health. Indeed, it is likely that some compounds deemed ineffective in male rodents would work in females, yet such compounds never would have a chance to make it to market, because of testing exclusively in male subjects. Moreover, the fact that most

drugs are designed using males also likely contributes to the higher rates of adverse drug reactions in women compared to men (322).

Including both sexes in mechanistic studies is critical for developing drugs that work efficaciously in both sexes (see Box 4). Latent sex differences can also impact drug development: a compound targeting a mechanism in men may not work in women. As the field moves forward, we may find that sex-specific therapeutics based on understanding latent sex differences are required to truly improve patient outcomes. In sum, there are observable sex differences in behavior that extend beyond reproductive function. Molecular sex differences in several organs, such as the gut and the central nervous system, play a key role in driving these functional and behavioral differences. Moreover, even when function and behavior are consistent between the sexes, the underlying processes can differ. Thus, including both sexes in preclinical molecular studies guiding drug development is key for improving the health of men and women.

Section IV

Sex Differences in the Cardiovascular-Renal System

Cardiovascular disease (CVD) is the major cause of premature death in both sexes worldwide, although women generally develop CVD 10 years later than men (328). In 2016, ~18 million people died from CVD, representing ~30% of all deaths worldwide (329). There are marked sex differences in CVD and renal disease. For example, women are protected from heart disease during the reproductive years but are more likely to die in the first year following a cardiovascular event than males (330). Most heart conditions, including myocardial infarction, Takotsubo syndrome, and cardiac arrhythmia, exhibit sex differences in symptoms and severity (331). Chronic kidney disease (CKD) is more prevalent in women but, once established, progresses more rapidly in men (332). However, this female advantage is lost after menopause. These sex differences in cardiovascular and renal disease have long been overlooked and underappreciated. The clinical presentation, the response to pharmacotherapies, standard care practices, and the underlying pathophysiological mechanisms differ in women compared to men. Furthermore, lack of understanding of sex differences in mechanisms underpinning cardiovascular and renal disease has led to poorer outcomes in women than in men. A major problem is that mechanistic preclinical studies in animal models have largely been conducted in males (333). Yet, it has become increasingly clear that sex differences

Box 4. Sex differences in pharmacokinetics and pharmacodynamics of drugs

Thalidomide, a sedative that was prescribed to many pregnant women to relieve pregnancy-associated nausea, was first sold in Germany (without a prescription) in 1957; it had been tested in animals and in men, but not in women. It was soon noted to cause multiple birth defects, most notably phocomelia (arrested limb development) and postnatal deaths. Fortunately, it was never approved in the United States, but thousands of children were affected around the world. In 1962, the US Congress passed the Kefauver-Harris Drug Amendments Act requiring manufacturers to prove a drug is both safe and effective (323). Consequently, the US Food and Drug Administration (FDA) recommended against drug testing on women, particularly those of child-bearing age, until the early 1990s. To date, most treatment guidelines are based on results from clinical trials conducted on middle-aged men. Dosage, pharmacokinetics, and pharmacodynamics data for women (and children) are lacking for most drugs. Activities of cytochrome P450 (CYP) enzymes show significant sex differences in drug metabolism in Phase I clinical trials (324). Gastric enzymes involved in oxidative degradation such as alcohol and aldehyde dehydrogenases are significantly more active in men than in women resulting in higher bioavailability of ethanol in women versus men. In Phase II trials, glucuronidating enzymes and some efflux transporters have been shown to be more active in men than in women. Together with estrogens and androgen that alter transmembrane transporters, these processes contribute to efficacy of metabolism in both Phase I and II. Drugs used for treatment of cardiovascular disease, such as angiotensin-converting enzyme inhibitors (ACE inhibitors), angiotensin II receptor blockers, diuretics, the aldosterone blocker eplerenone, antiplatelet agents, and oral antithrombotic medications, all show sex differences in efficacy and safety (325, 326). Over-the-counter nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen and naproxen are more effective in men than women; there is more liver toxicity with acetaminophen use in women, whereas opioids and benzodiazepine work better in women. While some sex differences in metabolic clearance for statins and beta-blockers are known for these frequently prescribed drugs, dosing and adverse event monitoring in routine clinical practice is inadequate. Alosetron, a serotonin receptor 3 antagonist, is approved for treatment of severe irritable bowel syndrome–diarrhea symptoms in women, as it is largely ineffective in men (327). These findings emphasize that women and men take divergent routes (molecular mechanisms and signaling pathways) to reach the same destination (normal function or diseased state), with paths often intersecting. In the era of personalized medicine, there is no one-size-fits-all therapy, and considering sex-specific outcomes in pharmacokinetics and pharmacodynamics of drugs as well as clinical guidelines is warranted to ensure efficacy and safety of medications.

are apparent in all endocrine systems, which are modified by sex chromosomes and sex hormones, with temporal actions across the lifespan.

Blood Pressure Links Cardiovascular and Renal Diseases

Cardiovascular and renal diseases are linked by the relationship of each to arterial pressure (Fig. 5). The cardiovascular system determines arterial pressure, with the heart generating cardiac output and the blood vessels determining total peripheral resistance. The kidneys contribute by regulating extracellular and intravascular fluid volume, and hence blood volume, and venous return. It is established that CVD leads to chronic kidney disease (CKD) and that CKD leads to the development of CVD. For example, following a myocardial infarct, cardiac output declines and arterial pressure falls causing the kidney to vasoconstrict and retain extracellular fluid, with the effect to increase venous return and normalize cardiac output. However, this has the unwanted effect of placing further stress on the failing heart. Conversely, kidney failure causes fluid retention and hypertension (334). Thus, cardiovascular and kidney function are intertwined, as are the endocrine systems that regulate organ function; including the renin-angiotensin-aldosterone system

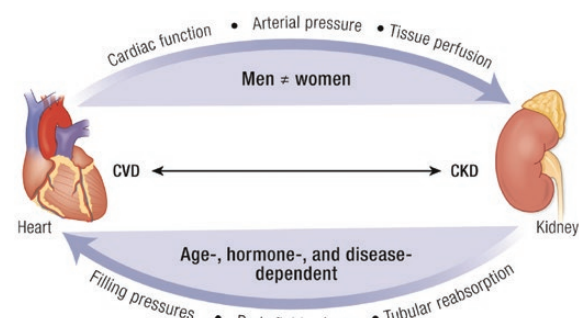


Figure 5. Heart and kidney functions are linked. Sex differences exist in many aspects of heart and kidney function at baseline and in CVD and CKD, as shown. Both organs feed-forward and influence each other's function. Genes, hormones, and age are some known factors that modulate this relationship in a sex-specific manner. Abbreviations: CKD, chronic kidney disease; CVD, cardiovascular disease.

(RAAS), the endothelin system, atrial natriuretic peptides, vasopressin, and glucocorticoid and mineralocorticoid hormones. There is an increasing recognition that there are fundamental sex differences in each of these systems. For example, aldosterone contributes to obesity-induced CVD with a greater impact in females than males (335). However, further research is required to fully elucidate the sex differences present in each endocrine system and how these impact disease development and progression.

Sex Differences in Arterial Pressure and Hypertension

Hypertension is a major risk factor for cardiovascular and renal disease. Over the lifespan there are age- and sex-related differences in arterial pressure. The majority of the data are derived from cross-sectional studies, but a few powerful studies have tracked arterial pressure over decades within a population (332, 336-339). Arterial pressure increases in both men and women with age, although the slope of the relationship is different between men and women. Sex differences in arterial pressure emerge during adolescence and are maintained throughout adulthood until women reach menopause (336, 337, 339). Arterial pressure is ~5 to 10 mmHg greater in men than age-matched women during the reproductive years (340-342). Postmenopause arterial pressure rises steeply in women regardless of race, ethnicity, or country of origin (340-342). One of the most striking characteristics of hypertension is that the prevalence and severity is lower in premenopausal women than in age-matched men. The prevalence of hypertension is ~10% in young premenopausal women, ~50% in postmenopausal women and by the age of 75 years almost ~80% of women are hypertensive (342-344).

Nonhuman mammalian species also display sex differences in arterial pressure. Arterial pressure in adult females is lower in normotensive dogs, sheep, rabbits, rats, and mice as compared with adult males (338, 345). Furthermore, in rodents, rabbits, and sheep, females of reproductive age are protected against the development of hypertension, such that arterial pressure increases significantly less in females than in males, in settings of disease (338). Thus, sex differences are present in the pathophysiology of cardiovascular and renal diseases. Yet, the mechanisms underlying the sexual dimorphism of arterial pressure in men and women as they age are poorly understood. However, extensive evidence indicates that sex hormones likely contribute to the regulation of arterial pressure through their actions on endocrine systems.

Sex Differences in Endocrine Control of Arterial Pressure and Kidney Function

There are subtle differences in most endocrine actions between men and women. It is not the maximal response of each system but rather the slope of the response that is altered. In this manner, a system responds maximally in a hemodynamic crisis (eg, hemorrhage) but in a sex-specific manner to lesser challenges. For example, a greater dose of the vasoconstrictor angiotensin II is required to increase arterial pressure in female than male mice (346). Consistent with this finding, the same dose of angiotensin II caused a

greater reduction in renal blood flow in men than women, with the suggestion that this was an angiotensin type 2 receptor (AT_2R) mediated effect (347). In rodents, females of reproductive age have a greater AT_2R to angiotensin type 1 receptor (AT_1R) ratio than males, which contributes to the reduced pressor response to angiotensin II (348). This has been indirectly demonstrated in women, in studies examining forearm vascular resistance responses to AT_2R blockade (349). The AT_2R also mediates a leftward shift in the pressure natriuresis-diuresis relationship, an effect that is greater in female than male mice (350). In women, indirect evidence also indicates a more pronounced role for the AT_2R in the regulation of renal blood flow responses to angiotensin II (347). This is linked to differential expression of components of the RAAS in males and females, which have been demonstrated in most mammalian species, including humans (351). In the context of the above example, estrogen interacts with the glucocorticoid response element on the X-linked *AGTR2* gene, to increase AT_2R expression in females (352). In addition, there are sex differences in human aminopeptidase A, aminopeptidase N, and angiotensin-converting enzyme 2 levels, responsible for generation of the angiotensin peptide fragments, angiotensin III, and angiotensin-(1-7), which have a high affinity for the vasodilatory AT_2R and Mas receptors, respectively (353-356). Lastly, there are marked and important sex differences in the production and function of aldosterone, although this has only recently been started to be examined (335). Thus, in females the RAAS is balanced toward the protective depressor RAAS arm, which at the lower physiological range may prevent arterial pressure increasing to the same extent as in males. However, this delicate balance may be lost in women after menopause and in the situation of metabolic syndrome.

Other vasoconstrictor systems also have sexually dimorphic actions. Endothelin-1 causes vasoconstriction via the endothelin type A receptor (ET_AR), and vasodilation and sodium excretion via the ET_BR . Testosterone increases ET_AR and estrogen increases ET_BR expression, which contributes to the differential control of arterial blood pressure and renal function between the sexes (357). Vasopressin, with important roles in circulatory and water homeostasis, is affected by age and sex. Urinary concentrating ability declines with age, but more steeply in women. Young men produce more concentrated urine than women, in part due to higher plasma arginine vasopressin levels and greater vasopressin type 2 receptor expression in the collecting ducts of the kidney in males (358, 359). Renal vasopressin type 2 receptor expression declines with age in association with a reduction in maximal urine concentrating ability (358, 359). Interestingly, aldosterone signaling via mineralocorticoid receptors is associated with increased CVD risk and is

enhanced in obese women (another example of how the RAAS is differentially modulated in females), which has been linked to leptin-induced endothelial dysfunction (360, 361). Moreover, evidence in rodents indicates that sodium reabsorption along the length of the renal tubule is sexually dimorphic, with reabsorption shifted to the later segments in females compared to males. This was associated with greater sodium epithelial channel expression, under the control of aldosterone, in the collecting duct, which could also contribute to the increased cardiovascular and renal risk associated with aldosterone in females (362). Finally, oxytocin, relaxin, and prolactin, which are traditionally known for their roles in pregnancy, have differential cardiovascular and renal actions in nonpregnant female and male rodents (348, 363, 364). Thus, evidence points to sex differences in endocrine control of extracellular fluid homeostasis and vascular function, which likely contribute to age- and sex-related disparities in renal and cardiovascular disease risk. Further studies are warranted to understand this complex issue more fully. In particular, it is important to take into account the subtle effects within the physiological range that counterbalance function of each hormonal system, rather than examine the impact of pharmacological doses which can mask sex differences in responses.

Cardioprotective Mechanisms in Women Sustain a Healthy Pregnancy

The cardioprotective mechanisms that predominate in women during the reproductive years enable the extensive hemodynamic adaptations required to meet the metabolic demands of the developing fetus and a successful pregnancy. During a normotensive pregnancy, blood volume increases and cardiac output increase by ~30% to 50%, but arterial pressure declines due to marked peripheral vasodilatation (365, 366). The associated renal vasodilatation accommodates an increase in glomerular filtration rate to process the additional blood volume, but an increase in vasopressin type 2 receptor expression enables increased tubule reabsorption of sodium and water. However, in women with preeclampsia, a pregnancy-induced form of hypertension, these cardiovascular adaptations are perturbed. Accumulating evidence now indicates that women with a history of pregnancy-associated hypertension have a 2- to 5-fold increased risk of CVD in later life (367). Understanding the mechanisms underpinning this dysregulation of vascular function in pregnancy-related hypertension may lead to the identification of new therapeutic targets for the treatment of cardiovascular disease in both sexes. For example, relaxin, which is known best for its role in pregnancy but is also produced in males, plays

roles in the regulation of renal function, blood pressure, and tissue fibrosis (363). Thus, it is a mistake to assign hormonal systems a specific role as most have wide-ranging tissue-specific pleiotropic effects.

Sex Hormones and Sex Chromosome Complement in CVD

Sex hormones contribute to sexual dimorphism in endocrine control of the cardiovascular system, with evidence suggesting that there is a “sweet spot” for both testosterone and estradiol, as unusually high or low levels of either promote disease (368-370). This has been the cause of apparent discrepancies in the literature. In particular, this remains a problem in animal studies in which the dose of estrogen used to study the impact of estrogen replacement in aged or gonadectomized models varies widely (~1000-fold), as does the route or length of administration; none of which accurately reflect the cyclic pattern of *in vivo* production. This lack of rigor into investigation of the effects of sex hormones in preclinical models likely contributes to the controversy that surrounds hormone replacement therapy for the prevention of CVD risk. Despite extensive evidence that hormone replacement therapy is cardioprotective, the negative results of the Women’s Health Initiative Trial effectively halted the use of hormone replacement therapy (371). Certainly, high-dose estrogen can increase blood pressure and cardiovascular risk in women (372). However, continued investigation supports the use of hormone replacement therapy in subsets of women, and further work in this area is required (373). In contrast, in men with low testosterone, beneficial cardiovascular effects are seen with testosterone replacement (374). In women with polycystic ovary syndrome, high testosterone levels are associated with elevated blood pressure (374). Dose-ranging studies are required to delineate these effects.

The sex chromosomes may have a direct impact on sex differences in the physiology and pathophysiology of the cardiovascular system and cardiovascular risk, independent of sex hormones. Human sex chromosome aneuploidies, such as Turner and Klinefelter syndromes, suggest that sex chromosome abnormalities can carry an increased risk of CVD. Women with Turner syndrome have around a 3-fold greater mortality and reduced life expectancy relative to the general population (375-377). CVD is a leading cause of increased mortality in Turner syndrome (375-377). Congenital cardiac anomalies, hypertension, coarctation of the aorta, diabetes, ischemic heart disease, and stroke are commonly associated with this condition (378). Similarly, men with Klinefelter syndrome have a high cardiovascular risk profile (379, 380), and an increased risk of

mortality from cardiovascular disease (381, 382). However, observations from studies in individuals with sex chromosome aneuploidies are complicated by confounding factors, including abnormal gonadal sex hormone levels associated with gonadal failure. Thus, it is very difficult to distinguish between hormonal versus genetic mechanisms and cardiovascular risk in these human conditions.

Experimental approaches, such as the FCG mouse model discussed in “Section I,” and Box 3 can discriminate between hormonal and sex chromosome effects in cardiovascular disease (115). Beyond genes on the sex chromosomes, there are sex differences in autosomal gene expression, which can be both organ or cell specific (383). In the kidney and the heart, hundreds of rat and human genes are regulated differently between the sexes (384–386). This disparate expression is triggered by sex hormones in ~30% of cases, with the other 70% linked to sex chromosome and microRNA dimorphisms (384, 385). For example, sex differences have been reported in the expression of nitric oxide synthase, tyrosine hydroxylase, and sodium channels in the rodent heart and kidney (332). However, few studies to date have compared gene expression and the effect on the proteome between the human sexes, and further studies are required.

Sex Differences in Pharmacotherapy for Cardiovascular and Renal Disease

Men and women respond to disease differently: kidney diseases progress faster in men than women, kidney transplants from women to men tend to fail more frequently than the reverse, and the effects of diabetes on the kidney differ between the sexes (387–392). Furthermore, symptoms and mechanisms of heart failure differ between the sexes (393). This suggests that sex-specific treatments for CKD and CVD could be required. There is currently little evidence to suggest that men and women respond differently to current treatments for hypertension (394). In large part, this is because clinical trials have lacked statistical power to take this into account. It will be difficult to achieve such an outcome for drugs that have already received FDA approval. However, some treatments are more frequently prescribed, without any basis in evidence (395). There are also marked differences in pharmacokinetics and pharmacodynamics (see Box 4), leading to more frequent adverse drug reactions in women, related to differences in drug clearance and breakdown (396). Therefore, sex should be taken in account for new treatments seeking approval in the future. When women are considered, important and unexpected sex differences are observed in almost every aspect of cardiovascular and renal function in health and

disease. Further research is required to fully understand these differences, and in turn to guide the development of sex-specific treatment guidelines for CVD and CKD.

Section V

Challenges for the Future of Sex Differences Research—Areas Requiring Special Attention

Sex differences exist in anatomy, behavior, and physiology across the animal taxa. By extension, because of these innate differences, sex differences exist at molecular and cellular levels in mechanisms that underlie these processes. Despite concerted efforts by the Office of Research on Women's Health and the Organization for the Study of Sex Differences in educating researchers about the distinction between sex versus gender, the indiscriminate use of the word “gender” continues to pervade scientific literature. The sex of established cultured cell lines is another issue; in addition to aneuploidy, chromosomal numbers change as cells are passaged and are dependent upon the tissue of origin (397, 398), but this aspect is beyond the scope of this Statement. Not surprisingly, sex differences are seen in etiology, prevalence, and outcomes in a myriad of human diseases that range from psychological and autoimmune to gastrointestinal, cardiovascular, renal, and reproductive; SARS-CoV-2 causes more severe COVID-19 disease in men than in women despite similar infection rates (399–401). Besides genetic makeup (predisposition), extraneous factors, such as the socioeconomics, demographics, education level, profession, age, and the environment, greatly influence an individual's health; COVID-19 disease outcomes especially highlight the contribution of these extraneous factors in health disparities. Factors such as the endocrine-disruptive chemicals can disproportionately affect one sex over the other; regardless, whether favorable or adverse effects are present in one or both sexes, the effects would impact trans and cisgender persons, and hence these sex-specific effects should not be overlooked or underestimated (402). Some human studies addressing sex differences take these factors into account, whereas others are more selective. Many studies of disease pathways are sensitive to levels of gonadal steroid hormones, which contribute to sex differences. In human studies, unless gender information is explicitly collected or available, the study deals with biological sex, not gender. Use of sex and gender interchangeably deemphasizes the importance of studying gender as an independent variable.

In animals or experimental models of human diseases, effects of estrogens have been investigated more often than effects of progestins and androgens, which should

be corrected. Paradoxically, female sex is often excluded from experimental design on the basis that: (i) the estrus cycle will interfere with data interpretation; (ii) mechanisms that operate in the male sex will operate in the female sex and thus only need to be confirmed in females; (iii) metabolic demands are similar between the sexes; (iv) the X chromosome in males and females is subject to similar regulation; and (v) autosomal genes will be subject to equal variance between the sexes. The same studies often ignore the diurnal cycling nature of testosterone in males; testosterone levels in male rodents can show more day-to-day variability than estrogen and progesterone levels in females. Other steroid hormones, such as glucocorticoids, that show circadian rhythm and whose levels differ between the sexes also influence gene expression and function. In rodents but not primates, sex differences in secretion of growth hormone result in sexually dimorphic hepatic metabolism of drugs and xenobiotics (403). In rodents, endocrine disruption can have transgenerational effects on male and female reproductive systems (404). Since changes in hormone levels and gene expression are dynamic, can be localized, and are spatiotemporally distinct, no one study design or condition can be used as a gold standard. Animal housing and handling conditions can also create sex differences, and thus any experimental design and data interpretation should take these variables into account. If sex-segregated data does not differ for the aspects under study, then data can be pooled from the 2 sexes and reported accordingly.

Studies in animal models have just begun to uncover unequal effects of the sex chromosomes in XX vs XY cells, so we expect further discoveries about such effects in the future. Once genes that cause sex differences are discovered in animals, the findings generate new hypotheses and rationalize human studies to determine whether the same gene also creates sex differences in humans. That question can be studied by the methods of human genetics, relating genetic variation to disease incidence and outcome. Without the animal studies, however, it is difficult to understand detailed molecular mechanisms. It is also important to remember that no single rodent or animal model can capture the complexity of any human disease, but each model provides valuable insights into one or another major aspect of disease. If different etiologies of a given disease share mechanisms, then mimicking the precise conditions that initiate human disease may not be critical.

The study of sex chromosome effects is in its infancy and has focused on proving that sex chromosomes play a role and finding the genes responsible for the effects. So far there has been little effort to understand how these factors interact with steroid hormones to cause sex differences. If

both types of factors cause differences in disease incidence, are they affecting the same or different downstream pathways? Do their effects converge, or do they independently affect different mechanisms that each influence a complex disease? Do male-biased factors (hormones, Y-chromosome genes) act synergistically to induce a male-specific state, or do they counteract each other to reduce the difference between males and females (123, 405)? Are the diverse sex-biasing factors changing in their effects across the lifespan, leading to changes in the type or amount of sex difference at different ages?

When studying sex differences in animal models of human diseases, it is important to first understand and elucidate differences at baseline in gonadally intact animals. As pointed out earlier, steroidogenic enzymes are also present in nongonadal tissues, especially the brain, thus it is not entirely possible to eliminate effects of sex steroids from all tissues. Moreover, tamoxifen-inducible *Cre* recombinase used to routinely perform lineage tracing and gene inactivation studies in mice has its own problems (406, 407) that are largely ignored and can further confound sex-specific data analysis; tamoxifen antagonizes actions of estrogen receptor- β and inhibits expression of over 70 genes (408), but the contribution of these tamoxifen-regulated genes on study results and outcomes is never accounted for and requires careful consideration. Before mechanisms behind sex differences in physiology and disease can be elucidated, a fundamental understanding of sex differences that exist at baseline, is needed.

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Exhibit D

How SEX and GENDER Influence Health and Disease

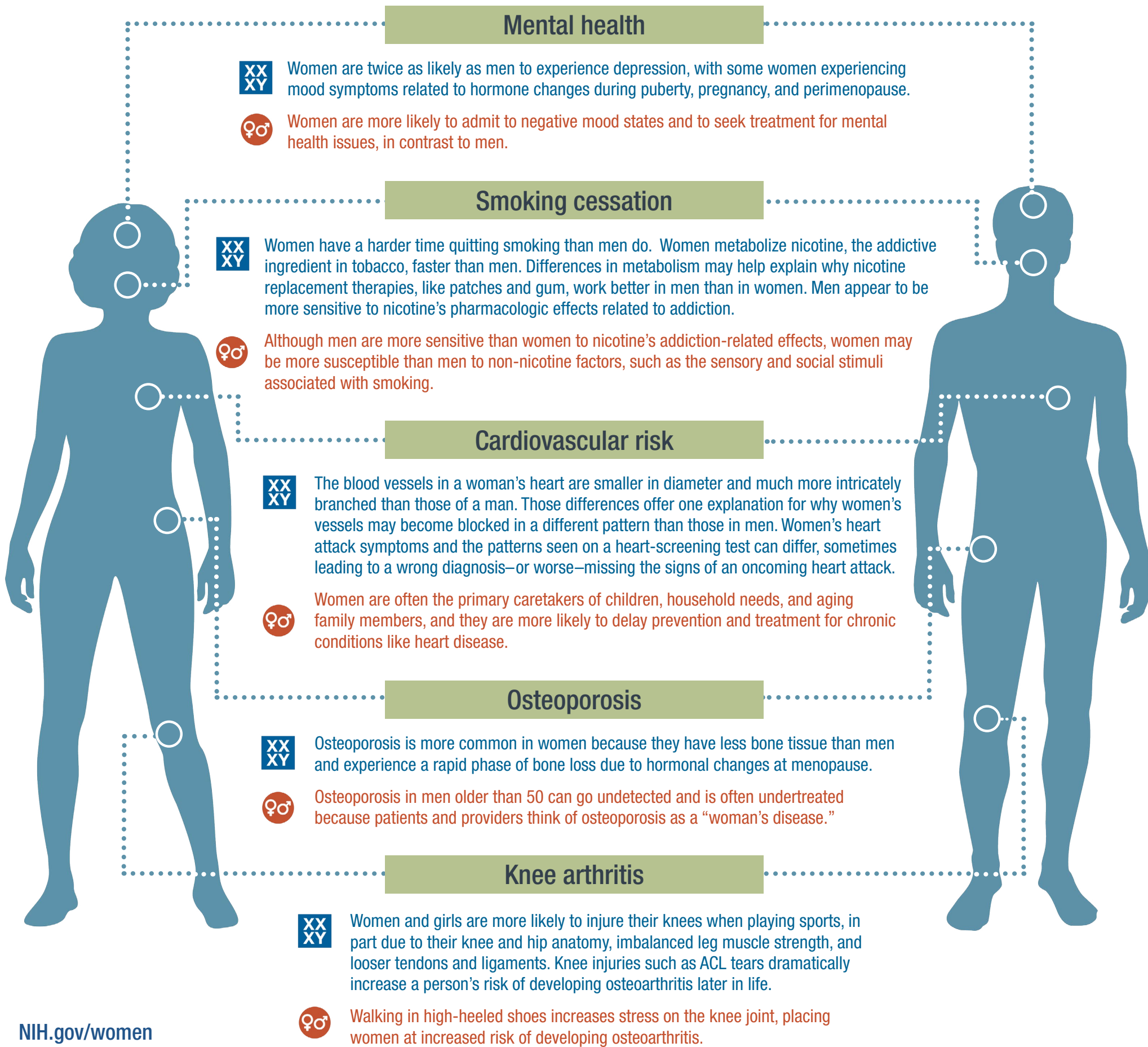
Sex and gender can influence health in important ways. While sex and gender are distinct concepts, their influence is often inextricably linked. The scientific studies that generate the most complete data consider sex and/or gender influences in study design, data collection and analysis, and reporting of findings.

Sex is a biological classification, encoded in our DNA. Males have XY chromosomes, and females have XX chromosomes. Sex makes us male or female. Every cell in your body has a sex—making up tissues and organs, like your skin, brain, heart, and stomach. Each cell is either male or female depending on whether you are a man or a woman.

Gender refers to the socially constructed roles, behaviors, expressions, and identities of girls, women, boys, men, and gender diverse people. It influences how people perceive themselves and each other, and how they act and interact. Gender is usually conceptualized as binary (girl/woman and boy/man), yet there is considerable diversity in how individuals and groups understand, experience, and express it.

Visit **NIH.gov/women** to learn how studying sex and gender strengthens science.

Examples of SEX and GENDER influences



NIH.gov/women
 @NIH_ORWH
#SexinScience

Sources: Institute of Medicine. Canadian Institutes of Health Research. World Health Organization. National Institute on Drug Abuse. NIH Osteoporosis and Related Bone Diseases National Resource Center. National Institute of Arthritis and Musculoskeletal and Skin Diseases. Kerrigan, D. C., Johansson, J. L., Bryant, M. G., Boxer, J. A., Della Croce, U., & Riley, P.O. (2005). Moderate-heeled shoes and knee joint torques relevant to the development and progression of knee osteoarthritis. Archives of Physical Medicine and Rehabilitation, 86(5), 871-875.

Exhibit E

Consideration of Sex as a Biological Variable in NIH-funded Research

Notice Number: NOT-OD-15-102

Key Dates

Release Date: June 9, 2015

Related Announcements

[NOT-OD-16-034](#)

[NOT-OD-16-031](#)

[NOT-OD-16-012](#)

[NOT-OD-16-011](#)

[NOT-OD-15-103](#)

Issued by

National Institutes of Health ([NIH](#))

Purpose

The National Institutes of Health (NIH) is committed to improving the health outcomes of men and women through support of rigorous science that advances fundamental knowledge about the nature and behavior of living systems. Sex and gender play a role in how health and disease processes differ across individuals¹, and consideration of these factors in research studies informs the development and testing of preventive and therapeutic interventions in both sexes. This notice focuses on NIH's expectation that scientists will account for the possible role of sex as a biological variable in vertebrate animal and human studies. Clarification of these expectations is reflected in plans by NIH's Office of Extramural Research (OER) to update application instructions and review questions; once approved by the Office of Management and Budget (OMB), these updates will take effect for applications submitted for the January 25, 2016, due date and thereafter. Please refer to [NOT-OD-15-103](#) for further consideration of NIH expectations about enhancing reproducibility through rigor and transparency.

Background

Women now account for roughly half of all participants in NIH-supported clinical research, which is subject to NIH's Policy on the Inclusion of Women in Clinical Research.² However, more often than not, basic and preclinical biomedical research has focused on male animals and cells.³ An over-reliance on male animals and cells may obscure understanding of key sex influences on health processes and outcomes.

Accounting for sex as a biological variable begins with the development of research questions and study design. It also includes data collection and analysis of results, as well as reporting of findings. Consideration of sex may be critical to the interpretation, validation, and generalizability of research findings. Adequate consideration of both sexes in experiments and disaggregation of data by sex allows for sex-based comparisons and may inform clinical interventions. Appropriate analysis and transparent reporting of data by sex may therefore enhance the rigor and applicability of preclinical biomedical research.⁴

NIH expects that sex as a biological variable will be factored into research designs, analyses, and reporting in vertebrate animal and human studies. Strong justification from the scientific literature, preliminary data, or other relevant considerations must be provided for applications proposing to study only one sex. Investigators are strongly encouraged to discuss these issues with NIH program staff prior to submission of applications. Further information regarding NIH expectations for the consideration of sex as a biological variable is provided at the following website:

http://orwh.od.nih.gov/sexinscience/overview/pdf/NOT-OD-15-102_Guidance.pdf

- 1 Institute of Medicine. 2001. Exploring the Biological Contributions to Human Health: Does Sex Matter?
<https://www.iom.edu/Reports/2001/Exploring-the-Biological-Contributions-to-Human-Health-Does-Sex-Matter.aspx>
- 2 https://grants.nih.gov/grants/funding/women_min/guidelines_amended_10_2001.htm
- 3 <http://www.nature.com/news/policy-nih-to-balance-sex-in-cell-and-animal-studies-1.15195>
- 4 <http://www.nature.com/news/policy-nih-plans-to-enhance-reproducibility-1.14586>

Inquiries

Please direct all inquiries to:

Email: NIHReproducibilityEfforts@nih.gov

[Weekly TOC for this Announcement](#)
[NIH Funding Opportunities and Notices](#)

Exhibit F

SEXUAL MEDICINE

BASIC SCIENCE

Long-Term Follow-Up of Individuals Undergoing Sex-Reassignment Surgery: Somatic Morbidity and Cause of Death



Rikke Kildevæld Simonsen, MA,¹ Gert Martin Hald, PhD,² Ellids Kristensen, MD, FECSM,³ and Annamaria Giralddi, PhD, MD, FECSM³

ABSTRACT

Introduction: Studies of mortality and somatic well-being after sex-reassignment surgery (SRS) of transsexual individuals are equivocal. Accordingly, the present study investigated mortality and somatic morbidity using a sample of transsexual individuals who comprised 98% (n = 104) of all surgically reassigned transsexual individuals in Denmark.

Aims: To investigate somatic morbidity before and after SRS and cause of death and its relation to somatic morbidity after SRS in Danish individuals who underwent SRS from 1978 through 2010.

Methods: Somatic morbidity and mortality in 104 sex-reassigned individuals were identified retrospectively by data from the Danish National Health Register and the Cause of Death Register.

Main Outcome Measures: Somatic morbidity and cause of death.

Results: Overall, 19.2% of the sample were registered with somatic morbidity before SRS and 23.1% after SRS ($P =$ not significant). In total, 8.6% had somatic morbidity before and after SRS. The most common diagnostic category was cardiovascular disease, affecting 18 individuals, 9 before and 14 after SRS, and 5 of those 14 who were affected after SRS had cardiovascular disease before and after SRS. Ten individuals died after SRS at an average age of 53.5 ± 7.9 years (male to female) and 53.5 ± 7.3 years (female to male).

Conclusion: Of 98% of all Danish transsexuals who officially underwent SRS from 1978 through 2010, one in three had somatic morbidity and approximately 1 in 10 had died. No significant differences in somatic morbidity or mortality were found between male-to-female and female-to-male individuals. Despite the young average age at death and the relatively larger number of individuals with somatic morbidity, the present study design does not allow for determination of casual relations between, for example, specific types of hormonal or surgical treatment received and somatic morbidity and mortality.

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Key Words: Follow-Up; Gender Identity Disorder; Somatic Morbidity; Sex-Reassignment Surgery; Transsexualism

INTRODUCTION

Transsexualism refers to a condition in which the core characteristic is an individual's experience of profound incongruence between assigned sex at birth and the experienced gender.¹ According to the *International Statistical Classification of*

Diseases and Related Health Problems, 10th Edition (ICD-10),² the diagnostic criteria of transsexualism are (i) the desire to live and be accepted as the opposite sex, (ii) usually a sense of discomfort with or inappropriateness of one's anatomic sex, and (iii) a wish to have surgery and/or hormonal treatment (HT) to make the body as congruent as possible with the preferred sex. To develop characteristics of the opposite sex, treatment with cross-sex hormones (HT), castration, and genital reconstructive surgery (sex-reassignment surgery [SRS]) might be conducted.

The parent category of transsexualism in the *ICD-10* is gender identity disorder (GID).² In Denmark, individuals with GID are referred to the Gender Identity Unit, University of Copenhagen (GIUUC) under *ICD-8*³ code 302.39 and 1993 *ICD-10*² codes DF64.0 to DF64.9 by a general practitioner or psychiatrist. Assessment, in accordance with Danish Health Authority

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<http://dx.doi.org/10.1016/j.esxm.2016.01.001>

guidelines,⁴ includes blood sample analyses for chromosomal and hormonal abnormalities, screening for psychiatric and somatic morbidities, psychological testing, and sessions with a psychologist or psychiatrist.

If SRS is desired by the individual diagnosed with transsexualism, an observational period of at least 1 year 6 months (in the study period, 2 years), including 1 year of HT and living in the gender role as the opposite sex, is obligatory before applying for SRS to the Danish Health Authority. The Danish legal criteria for SRS and castration are an *ICD-10* diagnosis of transsexualism (F64.0), persistent wish for and understanding of the consequences of castration, and a minimum age of 18 years (during the study period, ie, 1978–2010, the minimum age was 21 years).⁵ All treatment is paid for by the public Danish medical system. Treatment with cross-sex hormones and genital reconstructive surgery has existed for more than 60 years, but findings on mortality and somatic well-being after SRS in long-term follow-up studies are equivocal.⁶ For possible somatic consequences of HT, the following outcomes have been studied the most: cardiovascular disease (CVD), bone growth, and hormone-sensitive cancer malignancies.

A review and meta-analysis of 16 studies, including 1,471 male-to-female (MtF) and 651 female-to-male (FtM) individuals, found no overall significant effect of HT on CVD.⁷ However, the type of HT (ethinyl estradiol) and the manner in which HT (oral estrogens) was administered in MtF patients were significantly associated with CVD.^{8,9} Further, in a Swedish study, increased CVD mortality in FtM and MtF individuals at least 10 years after HT was found,⁶ indicating a possible delay of adverse somatic consequences from HT on cardiovascular pathology.¹⁰

Studies of muscle and musculoskeletal diseases, bone growth, and bone deficiencies overall did not show an increased risk of osteoporosis in FtM individuals.^{11–16} However, in MtF individuals, lower bone mass density, possibly from androgen deprivation, was found after treatment compared with before treatment with HT.^{17–19} However, because of increased bone density before treatment and no loss of bone density from menopause, MtF individuals maintain a lower risk of osteoporosis than assigned women.²⁰

In cancer studies involving transsexuals receptive of SRS and/or HT, the focus has been on breast cancer, although the overall number of studies in relation to this issue is limited. The conclusions emerging from these studies suggest that for MtF individuals^{20–23} the risk of breast cancer is lower than the expected risk of breast cancer in assigned women but similar to that expected in assigned men. For FtM individuals, male sex hormones might have an antiproliferative effect on breast cancer cell lines.^{24,25} Thus, few cases of breast cancer in FtM individuals have been reported,^{26,27} indicating FtM individuals have similar risk as expected for male breast cancer.

Concerning cancer malignancies, a Belgian study, in which the average time of HT was 6 years (FtM) or 7 years (MtF), found

no increase in cancer malignancies among included transsexuals compared with controls randomly selected from the population.²⁸ In contrast, a Swedish study found borderline significant risk of death from neoplasms compared with controls.⁶ Lifestyle habits such as smoking and avoidance of the health care system were suggested as possible mediating mechanisms.

When studying increased and decreased risks of cancer in transsexuals receiving HT, it is important to note that HT has been used for 60 years in some transsexual individuals. Accordingly, the duration of exposure to HT might not be long enough for tumors to manifest and the number of individuals exposed is small.²⁹ Further, it has been suggested that inconsistency in reporting cancer incidents among transsexuals might lead to an underreporting of cancer in this cohort,^{21,30} likely affecting prevalence and incidence rates.

Studies of mortality in transsexuals have suggested an increased mortality risk compared with controls.^{6,10} For example, a Swedish study of 324 MtF and FtM individuals after SRS (follow-up = 11.4 years) found that the all-cause mortality rate was three times higher in this cohort compared with controls.⁶ Similarly, in a Dutch long-term follow-up study of 966 MtF and 365 FtM individuals (follow-up = 18.5 years), a 51% higher mortality rate was found in MtF subjects compared with the general population.¹⁰ For FtM subjects, no increased mortality was found compared with the general population. A Dutch study of 1,109 individuals receiving HT found no increased mortality overall, but in MtF subjects 25 to 39 years old, mortality was significantly increased because of suicide, acquired immune deficiency syndrome, CVD, drug abuse, and unknown causes.³¹ The only Danish study on transsexualism conducted thus far, which included 37 individuals, reported three deaths of 29 reassigned MtF individuals and no deaths of 8 FtM individuals studied from 1956 through 1978.³²

Somatic morbidity after alcohol abuse has not been investigated previously, although studies of substance abuse in individuals with transsexualism have been conducted. A Belgian study (N = 35) conducted at the University Hospital of Gent found alcohol and drug abuse in 50% of MtF and 61.5% of FtM individuals.³³ A Spanish study (N = 230) of individuals with complaints of GID seen at the Hospital Clinic (Barcelona, Spain) found current alcohol- and substance-related disorders in 11% MtF and 1.4% of FtM subjects.³⁴ A Swiss study found that 45% of 31 GID individuals diagnosed by the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*³⁵ had lifetime substance abuse (MtF = 50%, FtM = 36.4%).³⁶ A Swedish study of 233 individuals found substance abuse in 18.2% of FtM and 11.9% MtF individuals.³⁷ However, in a different Swedish study of 324 MtF and FtM transsexual individuals, no significant risk of being hospitalized for substance abuse was found compared with the general Swedish population.⁶ Lung diseases related to or caused by smoking have not been investigated previously in persons with transsexualism, although lesbian, gay, bisexual, and transgender persons have a higher incidence of smoking.^{38,39} Accordingly, this was included as an outcome in the present study.

For many of the studies that have focused on somatic morbidity and mortality, including those reviewed earlier, the following methodologic shortcomings apply: small sample, recruitment and diagnostic biases and inconsistencies (eg, place of participant recruitment and differences in diagnostic criteria), heterogeneity of treatment regimens, and varied duration of follow-up periods.⁶

The aim of the present study was to (re)investigate somatic morbidity and mortality using registry data in a cohort including 98% of all Danish individuals referred to a public GID clinic in Denmark who underwent SRS from 1978 through 2010 after a diagnosis of transsexualism.

AIMS

The specific aims of the study were to investigate (i) somatic morbidity before and after SRS and (ii) cause of death and its relation to somatic morbidity.

METHODS

Procedure

The study was approved by the Danish Data Protection Agency and the Danish Health Authority. Permission was obtained from the Civil Law Board to identify names and social security numbers of individuals who underwent SRS from 1978 through 2010 and who were treated at the GIUUC.

National Registers

The Danish National Health Register (LPR) was used to draw data on somatic morbidity. The LPR contains diagnoses and dates of onset and end of treatment of all somatic episodes at hospitals from 1977 (inpatients) and from 1995 (outpatients). In the LPR, diagnoses are coded according to the *ICD-8* (1969–1993) or *ICD-10* (1994–).^{2,3} Data from the LPR from 1977 to January 2013 were included in the study.

The Cause of Death Register has recorded all deaths and causes of death in Denmark since 1970. Death events occurring up to April 2014 were included in the study.

Study Population

Included in the study were 104 individuals (56 MtF and 48 FtM) diagnosed with transsexualism according to the *ICD-8*³ or *ICD-10*² at the GIUUC. All participants underwent castration with permission from the Danish Health Authority from 1978 through 2010. Verification that an individual had undergone SRS was accomplished using social security numbers (ie, Danish Cause of Death Register numbers); numbers ending in even numbers indicate female-assigned sex and those ending with odd numbers indicate male-assigned sex. Accordingly, changes in this number from even to odd or vice versa indicate the official change of assigned sex (ie, successful completion of SRS). Baseline data (Table 1) were obtained from medical records.

Table 1. Baseline Data

Variables	Male to female (n = 56)	Female to male (n = 48)
Mean age at referral (y), mean (SD)	30.3 (9.8)	27.0 (8.7)
Mean age at permission for SRS (y), mean (SD)	37.1 (9.7)	32.6 (8.0)
Mean age at initiating cross-sex hormones (y), mean (SD)	32.0 (9.9)	29.8 (8.4)
Mean length of follow-up (y), mean (SD)	16.38 (7.1)	10.21 (6.1)

SRS = sex-reassignment surgery.

Sociodemographic data (Table 2) were obtained from medical records and are further described by Simonsen et al.⁴⁰

Because of the lack of a specific code for SRS, the date of start of follow-up was defined as the date of permission to undergo SRS.

Measures

Baseline data (Table 1) were obtained from medical records based on interviews performed by specialized psychiatrists, psychologists, and medical doctors at the GIUUC during the treatment period.

Using the LPR and death registers, we obtained information pertaining to somatic morbidity before and after permission to undergo SRS and time and cause of death after obtaining this permission. More specifically, somatic diagnoses given to the patient from 1977 to January 2013 were investigated. Accordingly, each individual could present with different diagnoses, but multiple contacts with the somatic care system with the same diagnosis only had one outcome before SRS and one outcome after SRS. In addition, data on time and cause of death after permission to undergo SRS were drawn from the death registers until April 2014.

For each diagnosis, specifically chronic heart disease (*ICD-10* diagnoses F400–490), chronic lung disease (*ICD-10* diagnoses J40–47, *ICD-8* diagnoses 490–493), cancer (*ICD-10* diagnoses C00–C97.9, D00–D10.9, *ICD-8* diagnoses 140–209), alcohol-related liver morbidity (*ICD-10* K70–77, *ICD-8* 303–304), or muscle and musculoskeletal diseases (*ICD-10* M80–85, *ICD-8* 720–729), individuals were stratified by diagnostic group membership (ie, had received the diagnosis or had not received the diagnosis) and assigned sex (ie, MtF or FtM).

Mortality was determined by the cause-of-death certificate. Hence, each individual was dead or alive. For death, data related to cause of death were drawn from the death certificate.

Statistics

Statistical analyses were conducted in SPSS 19.0 (SPSS, Inc, Chicago, IL, USA). Clinical variables were analyzed using descriptive statistics. Means and SDs were calculated for

Table 2. Sociodemographics by Male to Female and Female to Male*

	Male to female (n = 58)	Female to male (n = 50)
Primary and secondary education (y), n (%)		
≤11	40 (69.0)	38 (76.0)
12–13 (completion of high school)	16 (27.6)	12 (24.0)
Missing information	2 (3.4)	0
Education beyond primary and secondary school at time of referral, n (%)		
None	29 (50.0)	30 (60.0)
≤3 y or apprenticeship	21 (36.2)	8 (16.0)
≥4 y	5 (8.6)	10 (20.0)
Unknown	3 (5.2)	2 (4.0)
Education beyond primary and secondary school when permission for SRS was granted, n (%)		
None	25 (43.1)	25 (50.0)
≤3 y or apprenticeship	21 (36.2)	13 (26.0)
≥4 y	8 (13.8)	10 (20.0)
Unknown	4 (6.9)	2 (4.0)
Employment at time of referral, n (%)		
Employed	36 (62.1)	31 (62.0)
Unemployed		
Sickness or unemployment benefits	12 (20.7)	7 (14.0)
Social welfare or pension	10 (17.3)	12 (24.0)
Employment when permission for SRS was granted, n (%)		
Employed	32 (55.2)	27 (54.0)
Unemployed		
Sickness or unemployment benefits	5 (8.6)	11 (22.0)
Social welfare or pension	20 (34.5)	11 (22.0)
Unknown	1 (1.7)	1 (2.0)

From Simonsen et al.⁴⁰

SRS = sex-reassignment surgery.

*The χ^2 and Fisher exact tests were conducted but showed no significance ($P < 0.05$).

continuous variables. Frequencies and percentages were generated for nominal and categorical variables. Between-group differences were analyzed using χ^2 test, t-test, and Fisher exact test.

No missing values were found for somatic outcome variables because they were obtained from the register data, where values are present (affected) or absent (unaffected).

RESULTS

Baseline data related to age at referral, permission for SRS, cross-sex hormonal initiation, and years of follow-up after SRS are presented in Table 1.

To investigate the first study aim concerning somatic morbidity before and after SRS, the total number of included

individuals who received a somatic diagnosis was identified (Table 3). As presented in Table 3, 20 FtM and MtF individuals (19.2%) before SRS and 24 FtM and MtF individuals (23.1%) after SRS had somatic morbidity, with no significant difference. Nine individuals (eight MtF and one FtM) had somatic morbidity before and after SRS, resulting in 35 individuals (33.7%) overall who had somatic morbidity. Table 4 lists the specific diagnoses of somatic morbidity.

As presented in Table 4, 25 somatic diagnoses were reported before SRS and 27 diagnoses after SRS from a total of 20 individuals before SRS and 24 individuals after SRS. Nine of the 24 individuals had somatic morbidity before and after SRS. The most common diagnostic category was CVD, affecting a total of 18 individuals, 9 before and 14 after (23 diagnoses) SRS, and 5 of the 14 individuals had CVD before and after SRS. The second most common diagnostic category was muscle and musculoskeletal diseases, with 12 diagnoses, six before and six after SRS, affecting a total of 11 individuals, with only one individual having muscle and musculoskeletal disease before and after SRS.

To investigate differences in somatic morbidity between MtF and FtM individuals, χ^2 test, Fisher exact, and t-test were used. Across diagnostic categories, no significant differences in somatic morbidity between MtF and FtM individuals were found. When comparing somatic diagnoses using χ^2 test, no significant differences between the number of somatic diagnoses given before and after SRS were found.

Concerning the second study aim, cause of death and its relation to somatic morbidity was investigated from after SRS until April 2014. Ten individuals (9.6%; six MtF [10.7%] and four FtM [8.3%]) died from after SRS to April 2014. Mean age at death was 53.5 ± 7.9 years (median = 55.5) for MtF individuals and 53.5 ± 7.3 years (median = 52.5) for FtM individuals ($P > .05$ by t-test). Somatic morbidity (ie, official cause of death) included two suicides (19 and 26 years after SRS, respectively), heart disease ($n = 2$), cancer ($n = 1$), ulcer ($n = 1$), and smoking- and alcohol-related diseases ($n = 4$).

Because the results might be influenced by changes in clinical procedures and guidelines over time and the cultural acceptance of transsexualism, data were checked for systematic differences in permission to undergo SRS from the first 16 years (1978–1994) to the next 16 years (1994–2010). Significantly ($P < .05$) more individuals with transsexualism received permission to undergo SRS from 1995 through 2010 (28 individuals in 1978–1994 and 76 individuals in 1995–2010).

DISCUSSION

We report the first nationwide register-based SRS follow-up study in Denmark of 98% of individuals who officially underwent SRS from 1978 through 2010.

For the first study aim (ie, investigation of somatic morbidity before and after SRS), we found that 19.2% of the cohort had a somatic diagnosis before and 23.1% after SRS. This difference

Table 3. Individuals with Somatic Morbidity Before and After SRS*

Diagnosis, n (%)	Before SRS		After SRS		Before and after SRS	
	Male to female (n = 56)	Female to male (n = 48)	Male to female (n = 56)	Female to male (n = 48)	Male to female (n = 56)	Female to male (n = 48)
Cancer	0	3	2	1	0	1
CVD	5	4	6	8	5	0
Musculoskeletal	3	3	3	3	1	0
Lung	2	1	3	1	2	0
Alcoholic liver	1	3	0	0	0	0
Individuals with somatic diagnosis						
Yes	8 (14.3)	12 (25.0)	12 (21.4)	12 (25.0)	8 (14.3)	1 (2.1)
No	48 (85.7)	36 (75.0)	44 (78.6)	36 (75.0)	48 (85.7)	47 (97.9)

CVD = cardiovascular disease; SRS = sex-reassignment surgery.

*The χ^2 and Fisher exact tests were conducted but showed no significance ($P < 0.05$).

was found not to be statically significant. Further, no significant difference in somatic morbidity between FtM and MtF cohorts was found. For the second study aim (ie, investigation of mortality), no significant difference in mortality between MtF and FtM cohorts was found. Average age at death was 53.5 years, and 10 individuals died after SRS.

For somatic morbidity, CVD was found in 6 MtF individuals (10.7%) and 12 FtM individuals (25.0%). In comparison, 4.4% of assigned men and 3.6% of assigned women older than 35 years in the general Danish population were found to have CVD.⁴¹ In the present study, CVD might have been due to long-term follow-up after HT (16.3 years for MtF cohort, 10.8 years for FtM cohort) as reported by other studies,^{6,10} or the

observed prevalence of CVD might be explained by a correlation between depression and anxiety and CVD as suggested by previous research.^{42,43} Socioeconomic status and CVD are related,^{44,45} and the present study group was characterized not only by anxiety and depression⁴⁶ but also by social marginalization⁴⁷ and difficulties in school, education, and employment.⁴⁰ Hence, these factors could be important underlying mediating and/or moderating mechanisms driving or affecting prevalence rates of CVD in transsexuals, although the design of this study did not enable us to explore this further.

Muscle and musculoskeletal morbidity was found in 11 individuals (10.5%). From 1997 through 2002, 13.9% of the general Danish population was diagnosed with muscle and

Table 4. Number of Somatic Diagnoses*

Diagnosis, n (%)	Before SRS		After SRS	
	Male to female (n = 56)	Female to male (n = 48)	Male to female (n = 56)	Female to male (n = 48)
Alcohol related				
Yes	1 (1.8)	3 (6.2)	0	0
No	55 (98.2)	45 (93.8)	56 (100.0)	48 (100.0)
Cancer				
Yes	0	3 (6.3)	2 (3.8)	1 (2.0)
No	56 (100.0)	45 (93.8)	55 (98.2)	47 (97.9)
Heart				
Yes	5 (8.9)	4 (8.3)	6 (10.7)	8 (16.7)
No	51 (91.1)	44 (91.7)	50 (89.3)	40 (83.3)
Lung				
Yes	2 (1.8)	1 (2.1)	3 (5.4)	1 (2.1)
No	54 (96.4)	47 (97.9)	53 (94.6)	47 (97.9)
Musculoskeletal				
Yes	3 (5.4)	3 (6.3)	3 (5.4)	3 (6.3)
No	53 (94.6)	45 (93.8)	53 (94.6)	45 (93.7)
Positive somatic diagnosis	11	14	14	13

SRS = sex-reassignment surgery.

*The χ^2 and Fisher exact tests were conducted but showed no significance ($P < 0.05$).

musculoskeletal disease by hospital care.⁴⁸ Smoking and excessive alcohol consumption have been linked to low bone mass and increased fracture risk in MtF and FtM individuals,^{49,50} and such lifestyle issues might characterize the present cohort.^{6,46,51,52} However, given the limited number of individuals presenting with skeletal morbidity in this study, more comparable studies are needed to confirm the possible increased risk of skeletal morbidity in this cohort.

Concerning cancer malignancies, five individuals (6.2% of FtM and 3.6% of MtF) were found to have a diagnosis of cancer compared with 2.4% of assigned women and 1.56% of assigned men older than 15 years in the Danish general population.⁵³ Previous studies involving transsexual individuals have found hormone-sensitive tumors.^{16,20,54} Further, in the present study, two deaths were caused by cancer and by leukemia and lung cancer, respectively. However, as in the present study, small samples and the sample design preclude causal inferences regarding relations between treatment of SRS individuals and cancer or cancer-related deaths.

In Denmark, alcohol-related diseases cause 5% of the total number of deaths,⁵⁵ with more alcohol and substance abuse in sexual minority groups.^{51,52,56–58} Four individuals had a diagnosis of alcohol-related diseases before SRS with none after SRS. Further, in the present cohort, two individuals died of the effects of alcohol abuse after SRS. In a previous study on psychiatric morbidity of the present cohort, four diagnoses indicative of alcohol abuse after SRS were found.⁴⁶ Alcohol-related diseases are often the consequence of long-lasting alcohol abuse. Therefore, the actual number of individuals in the present cohort with alcohol abuse could be larger.

Four individuals had a diagnosis indicative of chronic lung disease (3.8%). In comparison, 1.3% of individuals older than 35 years in the in the general Danish population had a diagnosis of severe chronic lung disease.⁵⁹ Lung diseases have, to our knowledge, not been investigated previously in individuals with transsexualism, and therefore we lack and call for comparable studies in which to situate our findings.

Somatic morbidity in the present study group could be due to long-term HT and/or, as suggested by numerous previous studies, influenced by poor mental health, low economic status, social exclusion,⁶⁰ harassment, negative experiences with school⁶¹ and the employment system,^{34,37,62} and discrimination in the health care system.^{46,63,64} Thus, previous studies of the present group have found that 50% of the cohort did not complete further education beyond primary and secondary school. Also, at the time of SRS, only 55% were employed⁴⁰ and 25% presented with psychiatric morbidity before and after SRS.⁴⁶

For the second study aim (ie, cause of death and its relation to somatic morbidity), the study found that 9.6% of the cohort had died at an average age of 53.5 years, with the main cause of death related to smoking and alcohol abuse. The life expectancy of assigned women and men in Denmark is 81.9 and 78.0 years,

respectively. Previous studies of mortality in transsexual individuals in countries comparable to Denmark^{6,10} have found an increased risk of death in transsexual individuals. The present study had a lack of statistical power, and further long-term studies are needed to draw firm conclusions about transsexualism and increased risk of death.

Two individuals in the study group committed suicide 19 and 26 years after SRS, respectively. A Swedish study of SRS individuals (N = 324) found significantly increased mortality from suicide and significantly higher risk for suicide attempts compared with the general Swedish population.⁶ A Dutch study (N = 1,109) of SRS and non-SRS individuals found a high incidence of attempted suicide and completed suicide in the study cohort compared with the general Dutch population.³¹ An Italian study of 163 SRS MtF individuals found that four had attempted suicide before SRS and one had attempted suicide 12 to 18 months after SRS.⁶⁵ A Danish study reported death from suicide in 3 of 29 SRS MtF individuals (follow-up = 6 years).³² Many explanations can be considered for suicide and attempted suicide. One might be regret for undergoing SRS,³² but in the present study suicide occurred more than 19 years after SRS and therefore does not seem to be an immediate consequence of SRS. Because reasons for suicide attempts and manifest suicide often are multifactorial and because of the low incidence in the present study, further research is needed to contextualize these results further.

Limitations

The strength of this study is the unique cohort studied. Thus, on a national basis and over a 30-year period, 98% of all SRS individuals were included. This provides a unique opportunity to assess differences between MtF and FtM individuals on variables for somatic morbidity and mortality. The cohort included only individuals who received permission to undergo SRS during a period with strict criteria for obtaining permission to undergo SRS. Accordingly, the group is highly selected and might not reflect transsexuals per se in Denmark. Although we had a very large cohort for this type of study, some of our statistics had small cell sizes, limited numbers, and thus low statistical power, increasing the chances for type II errors. Because most somatic care in Denmark is provided by general practitioners, an underestimation of the prevalence of somatic morbidity in the study is plausible. Thus, somatic morbidity as presented in this study might be substantially higher.

CONCLUSION

Using a sample comprised of 98% of all individuals who underwent SRS in Denmark from 1978 through 2010, this study found somatic morbidity in 19.1% of the study group before and 23.2% after SRS. Mortality rates were 9.6%, with an average age at death of 53.5 years. No significant differences in somatic morbidity or mortality were found between MtF and FtM individuals. No firm conclusions can be drawn from the

present study, because the present study design does not allow for determination of causal relations between HT or SRS and somatic morbidity or mortality. One can speculate as to whether the increased risk of psychiatric problems and lifestyle issues in sexual minority groups influenced the risk of mortality and CVD in the present study. The findings underline the importance of supporting individuals with transsexualism to contact and be treated in the public health care system and to pay more attention to lifestyle issues in general.

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Exhibit G

ORIGINAL ARTICLE

Long-term follow-up of individuals undergoing sex reassignment surgery: Psychiatric morbidity and mortality

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ABSTRACT

Background: There is a lack of long-term register-based follow-up studies of sex-reassigned individuals concerning mortality and psychiatric morbidity. Accordingly, the present study investigated both mortality and psychiatric morbidity using a sample of individuals with transsexualism which comprised 98% ($n=104$) of all individuals in Denmark. **Aims:** (1) To investigate psychiatric morbidity before and after sex reassignment surgery (SRS) among Danish individuals who underwent SRS during the period of 1978–2010. (2) To investigate mortality among Danish individuals who underwent SRS during the period of 1978–2010. **Method:** Psychiatric morbidity and mortality were identified by data from the Danish Psychiatric Central Research Register and the Cause of Death Register through a retrospective register study of 104 sex-reassigned individuals. **Results:** Overall, 27.9% of the sample were registered with psychiatric morbidity before SRS and 22.1% after SRS (p = not significant). A total of 6.7% of the sample were registered with psychiatric morbidity both before and after SRS. Significantly more psychiatric diagnoses were found before SRS for those assigned as female at birth. Ten individuals were registered as deceased post-SRS with an average age of death of 53.5 years. **Conclusions:** No significant difference in psychiatric morbidity or mortality was found between male to female and female to male (FtM) save for the total number of psychiatric diagnoses where FtM held a significantly higher number of psychiatric diagnoses overall. Despite the over-representation of psychiatric diagnoses both pre- and post-SRS the study found that only a relatively limited number of individuals had received diagnoses both prior to and after SRS. This suggests that generally SRS may reduce psychological morbidity for some individuals while increasing it for others.

KEYWORDS

Follow-up, gender identity disorder, mortality, psychiatric morbidity, sex reassignment surgery (SRS), transsexualism

Since 2002 all individuals in Denmark referred with a diagnosis of transsexualism have been treated at the Sexological Clinic, Gender Identity Unit, University Hospital of Copenhagen (GIUUC).

According to the *International Statistical Classification of Diseases and Related Health Problems* 10th edition (ICD-10) an individual may be diagnosed with transsexualism if: (1) the person has the desire to live and be accepted as a member of the opposite sex, (2) usually a sense of discomfort with, or inappropriateness of one's anatomic sex, and (3) a wish to have surgery and/or hormonal treatment to make the body as congruent as possible with the preferred sex. In the ICD-10, transsexualism is categorized as a subgroup of gender identity disorders (GID) (1).

In Denmark, individuals with GID are commonly referred to the GIUUC under the ICD-10 codes DF 64.0–64.9 (1) by a general practitioner (GP) or

psychiatrist for assessment and clinical evaluation. Assessment and clinical evaluation includes blood sample analyses for chromosome and hormonal abnormalities, physical examination, screening for psychiatric morbidity, psychological testing, and monthly sessions with a psychologist or psychiatrist.

Following assessment and clinical evaluations the process of sex reassignment surgery (SRS starts with cross-sex hormonal treatment for a period of at least 12 months). Subsequently, an application to the Danish Health and Medicines Authority for permission to undergo SRS may follow. SRS may include both castration and genital reconstructive surgery (2). The costs of the treatment are paid for by the public Danish Medical System. Today the Danish legal criteria for castration and genital SRS are: an ICD-10 diagnosis of transsexualism (F64.0), a persistent wish for and understanding of the consequences of castration, and a

minimum age of 18 years. The minimum age for castration at the time of the present study was 21 years. According to national guidelines the evaluation period before being eligible to apply for SRS is a minimum of 1½ years. Since 2014 it has been possible to obtain legal sex change without having received medical treatment by application to the Ministry of Economy and Interior Affairs.

SRS has been practised for more than 60 years and is the internationally recognized treatment to ease gender dysphoria in individuals with transsexualism (3,4). Despite the long history of treatment, few and opposing data on mortality and psychiatric morbidity among sex-reassigned transsexuals are available. Accordingly, whether individuals with transsexualism and GID in general are at increased risk of psychiatric morbidity and/or have higher mortality rates compared with individuals without transsexualism are discussed.

In a Japanese study (5), among 603 individuals with GID (60.3% female to male (FtM); 39.7% male to female (MtF)) attending a gender clinic, current psychiatric morbidity was found among 19.1% of MtF and 12.0% of FtM. On this basis it was concluded that transsexualism was not associated with increased rates of severe comorbid psychological findings. This conclusion was supported by the results of a study by Cole et al. (6) of 435 patients evaluated for hormonal and surgical treatment at a gender clinic in Texas, USA. This study found that less than 10% evidenced problems associated with mental illness. Further, Bandini et al. (7), in an Italian study of 100 individuals with GID found that body uneasiness in GID was not linked to general psychopathology. Finally, concerning mortality, Dutch studies (8–10) among individuals diagnosed with GID have reported that mortality rates post-treatment with cross-sex hormones was no higher than that of the general population, the Dutch studies did not separate individuals who only had cross-sex hormones and those who both had cross-sex hormones and SRS.

In contrast to the studies above, other studies have indicated that individuals with GID may both hold more psychiatric morbidity, besides GID, and higher mortality rates as compared to other populations. In a meta-analysis of 28 studies including a total of 1833 individuals with GID (11) a lifetime prevalence of Axis 1 disorders, specifically depression and anxiety, was found among 71% of included individuals. Furthermore, it was found that GID individuals with a pre-existing severe psychopathology had retained more psychopathological symptoms and had worse outcomes in the domains of social and psychological functioning, post-transition, as compared with GID individuals with no pre-existing psychopathological symptoms (5,12–15).

In a Nordic context, a Swedish study by DHejne (16) of 324 sex-reassigned individuals found a fourfold risk of being hospitalized for sex-reassigned individuals included, due to psychiatric morbidity, as compared to the general population. Further, the risk of all-cause mortality in SRS individuals was found to be three times higher than that of the general population (16).

To date, the only published Danish study of individuals with transsexualism is an interview study including 37 individuals diagnosed with transsexualism who had undergone SRS during 1958–1977 (29 MtF, 8 FtM) (17,18). In this study, no MtF suffered from severe psychiatric morbidity and the vast majority (83%) reported feeling physically better after SRS. However, three MtF were reported deceased by suicide. Among FtM, two FtM were reported as “hospitalized with psychiatric morbidity” after SRS while none were reported as deceased by suicide. Although methodologically and statistically the Danish study suffered from a number of limitations the conclusion, that FtM seemed to overcome post-operative difficulties and function better post-SRS than MtF, is in line with the majority of comparable studies (11,19–22).

More generally, major methodological problems related to the study of psychiatric morbidity and mortality in transsexuals and GID, including many of the studies cited above, include small sample sizes, participant heterogeneity, recruitment and diagnostic biases and inconsistencies (e.g. place of participant recruitment, differences in the diagnostic criteria of GID), high drop-out rates and limited follow-up periods (16).

The aims of the current study were therefore to investigate psychiatric morbidity and mortality using register-based data in a cohort of nearly all (98%) of Danish individuals with transsexualism who underwent SRS during a more than 30-year period (from 1978–2010). Specifically:

Aim 1: To investigate psychiatric morbidity before and after SRS among Danish individuals with transsexualism who underwent SRS during the period of 1978–2010.

Aim 2: To investigate mortality among Danish individuals with transsexualism who underwent SRS during the period of 1978–2010.

Methods

Procedure

The study was approved by the Danish Data Protection Agency and the Danish Health and Medicines Authority. Permission was obtained from the Civil Law Board to

Table 1. Baseline data.

Variables	Male to female <i>n</i> = 56 (%)	Female to male <i>n</i> = 48 (%)
Mean age at referral, years (SD)	30.3 (9.8)	27.0 (8.7)
Mean age at permission for SRS, years (SD)	37.1 (9.7)	32.6 (8.0)**
Mean age at initiating cross-sex hormones, years (SD)	32.0 (9.9)	29.8 (8.4)
Number of sessions at Gender Unit (%)		
1–30	30 (53.6)	23 (47.9)
31–60	20 (35.7)	22 (45.8)
> 60	4 (7.1)	2 (4.2)
Unknown	2 (3.6)	1 (2.1)
Mean length of follow up in years after SRS (SD)	16.38 (7.1)	10.21 (6.1)

SD, standard deviation; SRS sex reassignment surgery.

Pearson χ^2 test: * $p \leq 0.01$, ** $p < 0.001$.

identify names and Social Security numbers of all individuals who had undergone SRS from 1978–2010 and who were treated at the GIUUC.

National registers

Since 1970 the Danish Psychiatric Central Research Register has electronically registered all inpatients treated at psychiatric departments in Denmark. Outpatients have been registered since 1995. The Register contains date of onset and end of treatment and diagnoses of all psychiatric episodes given at the hospitals. Diagnoses were/are coded according to the eighth version of the ICD (1969–1993) or ICD-10 (1994 onwards) (1). Data from 1970 to January 2013 was included in the study.

The Cause of Death Register has recorded all deaths and causes of death in Denmark since 1970. Death events occurring up to April 2014 were included in the study.

Study population

A total of 104 individuals (56 MtF and 48 FtM), diagnosed by a psychiatrist according to ICD-8/ICD-10 criteria of transsexualism (1) at GIUUC were included in the study. All participants underwent castration in the period of 1978–2010 and had permission from the Danish Health and Medicines Authority to undergo SRS. Verification that an individual had undergone castration was done through CPR number (social security number). From this information on, the gender of the person can be easily identified. That is, equal numbers indicate assigned female sex and odd numbers assigned male sex. Accordingly, changes from equal to odd numbers or vice versa of any of the targeted individuals would verify castration. Surgical procedure codes could not be used due to the lack of a specific code for SRS, therefore date of SRS (start of follow-up) was defined as the date of permission to undergo castration. Data was drawn from

both the social security number of the assigned sex, and the reassigned sex. Baseline data (Table 1) were obtained from medical records. These data were obtained based on interviews performed by specialized psychiatrists, psychologists, and medical doctors at the GIUUC during the treatment period.

Measures

We studied psychiatric morbidity before and after permission to undergo SRS. More specifically, we investigated psychiatric diagnoses (gender identity disorders excluded) given to the patient from 1970 up until January 2013. Consequently, each individual could present with several different diagnoses, but multiple contacts with psychiatric care with the same diagnosis only had one outcome pre-SRS and one outcome post-SRS. In addition, time and cause of death following the permission for SRS were included in the study. Data on death were drawn up until April 2014.

For each diagnosis, i.e. anxiety (ICD-10 F40.0–49.0), depression (ICD-10 F30.0–39.0), substance abuse (ICD-8 303–304, ICD-10 F10.0–19.0), personality disorder (ICD-10 F60.0–63.0), neurotic personality (ICD 8 300–309), and psychosis (ICD 8 290–299, ICD 10 F20.0–29.0), individuals included were stratified by diagnostic group membership (i.e. received the diagnosis, did not receive the diagnosis) and assigned sex (i.e. MtF or FtM).

Mortality was studied by the cause of death certificate. Consequently each individual was either dead, data on death certificates were drawn, or not dead.

Statistics

Statistical analyses were conducted in SPSS version 19.0. The psychiatric and clinical variables were analysed using descriptive statistics. Means and standard deviations (SD) were calculated for continuous variables. Frequencies and percentages were generated for

Table 2. Number of psychiatric diagnoses.

	Pre-SRS		Post-SRS	
	Male to female <i>n</i> = 56 (%)	Female to male <i>n</i> = 48 (%)	Male to female <i>n</i> = 56 (%)	Female to male <i>n</i> = 48 (%)
Anxiety ICD-10				
Yes	0	4 (8.3)	6 (10.7)	5 (10.5)
No	56 (100.0)	44 (91.7)	50 (89.3)	43 (89.5)
Depression ICD-10				
Yes	3 (5.4)	3 (6.3)	9 (16.1)	3 (6.3)
No	53 (94.6)	45 (93.7)	47 (83.9)	45 (93.7)
Abuse ICD-10				
Yes	0	2 (4.2)	0	4 (8.3)
No	56 (100.0)	46 (95.8)	56 (100.0)	44 (91.7)
Abuse ICD 8				
Yes	0	1 (2.1)	0	0
No	56 (100.0)	47 (97.9)	56 (100.0)	48 (100.0)
Personality disorder ICD 10				
Yes	0	4 (8.3)	5 (8.9)	3 (6.3)
No	56 (100.0)	44 (91.7)	51 (91.1)	45 (93.7)
Neurotica personalis ICD 8				
Yes	9 (16.1)	8 (16.7)	0	0
No	47 (83.9)	40 (83.3)	56 (100.0)	48 (100.0)
Psychosis ICD 8/10				
Yes	7 (1.7)	7 (4.0)	2 (3.6)	2 (4.1)
No	9 (98.3)	1 (96.0)	54 (96.4)	46 (95.9)
Any psychiatric diagnose				
Yes	19 (49)	29* (41)	22	17

SRS, sex reassignment surgery.

Pearson χ^2 test: * $p \leq 0.01$, ** $p < 0.001$.

Table 3. Individuals with one or more psychiatric diagnoses.

	Pre-SRS		Post-SRS	
	Male to female <i>n</i> = 56 (%)	Female to male <i>n</i> = 48 (%)	Male to female <i>n</i> = 56 (%)	Female to male <i>n</i> = 48 (%)
Individuals with ≥ 1 psychiatric diagnoses (%)	19 (33.9)	10 (20.8)	15 (26.8)	8 (16.7)

Four MtF and three FtM had psychiatric morbidity both before and after SRS.

SRS, sex reassignment surgery.

Pearson χ^2 test: * $p \leq 0.01$, ** $p < 0.001$.

nominal and categorical variables. Between-group differences were analysed using chi-squared tests, t-test and Fisher's exact test.

No missing values were found for the psychiatric outcome variables as they were obtained from register data, where values, in the first place, are either present (affected) or missing (unaffected).

Results

Mean age of participants and baseline data related to age of referral, permission for SRS, sex reassignment hormonal initiation, number of session at GIUUC, and years of follow-up can be found in Table 1. As can be seen from Table 1 significant difference between MtF and FtM was found for mean age at permission to undergo SRS where MtF were found to be significantly older than FtM.

Data related to psychiatric diagnoses pre- and post-SRS can be found in Table 2. Data on individuals with one or more psychiatric diagnoses besides transsexualism/GID can be found in Table 3. As can be seen from the table a total of 29 of 104 individuals were given one or more psychiatric diagnoses pre-SRS while 23 of 104 individuals were given one or more psychiatric diagnoses post-SRS. A total of 22 individuals were diagnosed with psychiatric morbidity pre-SRS but had not received further diagnoses post-SRS to January 2013. Sixteen individuals who were not diagnosed with psychiatric morbidity pre-SRS were diagnosed with psychiatric morbidity post-SRS. Seven individuals received psychiatric diagnoses indicative of psychiatric morbidity both pre- and post-SRS. Psychiatric diagnoses on the seven individuals can be found in Table 4.

In relation to mortality, from post-SRS to April 2014 a total of six MtF and four FtM were deceased (mean age MTF 53.5 (SD 7.9, median 55.5), FTM 53.5 (SD 7.3, median

Table 4. Diagnoses for individuals with pre- and post-psychiatric morbidity.

Individual no.	Pre-SRS							Post-SRS						
	MtF				FtM			MtF				FtM		
	1	2	3	4	5	6	7	1	2	3	4	5	6	7
Anxiety ICD-10					✓			✓	✓				✓	
Depression ICD-10	✓		✓					✓		✓				✓
Abuse ICD-10										✓			✓	✓
Abuse ICD-8				✓							✓			
Personality disorder ICD-10												✓		
Neurotica personalis ICD-8	✓	✓				✓	✓							
Psychosis ICD-8/10		✓		✓		✓	✓						✓	✓

52.5), $p = \text{not significant}$). Causes of death included suicide 2 (19 and 26 years after SRS), smoking and alcohol related diseases 4, heart diseases 2, cancer 1, ulcer 1.

Discussion

We report on the first register-based SRS follow-up study in Denmark including 98% of all individuals in Denmark who have undergone SRS during the period of 1978–2010.

Pertaining to study aim 1, we found that 28% of the study group had been diagnosed with psychiatric morbidity pre-SRS and 22% post-SRS. No significant differences were found between the number of MtF and FtM individuals suffering from psychiatric morbidity pre- and post-SRS, although significantly more psychiatric diagnoses overall were present in the FtM group. Further, approximately 7% of the sample received a psychiatric diagnosis both pre- and post-SRS indicative of psychiatric morbidity both before and after SRS.

These results indicate that overall, individuals undergoing SRS in the Danish public health system hold considerable psychiatric morbidity besides GID both pre- and post-SRS. Using psychiatric diagnoses as an indicator of psychological/psychiatric well-being, our findings did not support previous studies that found FtM to be better functioning than MtF (11,16,23,24). Pre-SRS we did not find significantly more FtM with psychiatric morbidity, but those suffering from psychiatric problems had multiple diagnoses. Seven individuals had psychiatric morbidity pre- and post-inception, indicating that 23 individuals had been cured of psychiatric suffering from the start of the study period until the time of SRS. Seventeen individuals without psychiatric morbidity pre-SRS, were given a psychiatric diagnosis post-SRS. Further studies are needed to explore this issue.

Anxiety, depression, and neurotica personalis constituted the majority of psychiatric diagnoses found in the present study. This finding is in line with comparable studies in the area which have generally found that

transsexualism was associated with anxiety and depression both before and after SRS (15,25,26).

Pertaining to study aim 2, almost 10% of the study population died at a mean age less than 60 years where the life-expectancy of assigned females and males in Denmark is 81.9 years, and 78.0 respectively. Although limited by the number of individuals included in this study, the results may provide some indication that SRS individuals are at increased risk of mortality post-SRS and thus that medical and clinical attention to this area of concern remain important (16,27,28).

In the present study two deaths were due to suicide. Although this number is far too small to come to any conclusions on, it is found in several other studies that individuals with transsexualism have a higher risk of suicidal ideation, attempts, or actual suicides (9,12,21, 29–33). Some studies attribute this to stress caused by stigmatization and gender abuse that may increase the probability of depression, anxiety, and suicide attempts of LGBT minority groups including transsexuals (34–40). In this connection a previous study on this present cohort (41) found an over-representation of problems related to school, education and employment which may explain both the elevated mortality and psychiatric morbidity rates as compared to the general Danish population (42).

Clinically, the results of the present study stress the importance of social and psychiatric counselling, support and follow-up of individuals preparing for or having undergone SRS. This may be especially relevant for the unknown number of self-identified transsexuals or publicly diagnosed transsexuals who do not consult the Danish public health system for SRS but never the less receive SRS abroad at their own expense.

Limitations

The present cohort includes only individuals who received permission to undergo SRS during a period with strict criteria for obtaining permission to undergo SRS. Accordingly, the group is highly selected and may not reflect transsexuals per se in Denmark.

Although we had a very large cohort for this type of study, some of our statistics may still suffer from low statistical power, thus increasing the chance for type II errors. Especially in regard to mortality, the cohort and the number of deaths did not hold enough statistical power to confirm or refute previous study findings.

Since most psychiatric care is provided by GPs and other outpatient care, underestimation of the prevalence of psychiatric morbidity in this study is probable. Thus, the psychiatric morbidity rates presented in this study may actually be substantially higher.

In the present study seventeen individuals were diagnosed with neurotica personalis and 11 individuals were diagnosed with psychosis according to ICD-8 before SRS. Caused by change from ICD-8 to ICD-10 in 1993, we found no ICD-8 diagnoses after SRS. The high incidence of ICD-8 morbidity is most likely due to the clinical aetiology in the 1970s and 1980s that indicated that transsexualism was psychotic or personality disordered in origin (29,43).

Conclusion

Using a sample comprised of 98% of all individuals who have undergone SRS in Denmark during the period of 1978–2010 this study found substantially high rates of psychiatric morbidity among this cohort. No significant differences in psychiatric morbidity or mortality were found between MtF and FtM save for the total number of psychiatric diagnoses where FtM were found to hold a significantly higher number of psychiatric diagnoses overall. Despite the over-representation of psychiatric diagnoses both pre- and post-SRS, the study found that only a relatively limited number of individuals had received diagnoses both prior to and after SRS. This suggests that generally SRS may reduce psychological morbidity for some individuals while increasing it for others.

Disclosure of interest

The authors report no conflicts of interest. The authors alone are responsible for the writing of this paper.

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Exhibit H



Deposition of:
Stephen B. Levine , MD

September 10, 2021

In the Matter of:
Kadel, et al vs. Folwell

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1 IN THE UNITED STATES DISTRICT COURT
2 FOR THE MIDDLE DISTRICT OF NORTH CAROLINA

3 ~~~~~

4 MAXWELL KADEL, et al.,

5 Plaintiffs,

6 vs. Case No. 1:19-cv-272-LCB-LPA

7
8 DALE FOLWELL, in his official
9 capacity as State Treasurer of
10 North Carolina, et al.,

11 Defendants.

12 ~~~~~

13 Video Deposition of
14 STEPHEN B. LEVINE, M.D.

15 September 10, 2021
16 9:05 a.m.

17 Taken at:
18 Veritext Legal Solutions
19 1100 Superior Avenue
20 Cleveland, Ohio

21 Tracy Morse, RPR
22
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1 Q. Okay. And so then were there any
2 external grants to research and publish about
3 the treatment of children or adolescents --

4 A. No.

5 Q. -- with gender dysphoria?

6 Okay. Is that a, "No," when I included
7 the, "Gender dysphoria," as well?

8 A. That is a, no.

9 Q. Okay. Thank you. Okay. So on
10 page 3 of your report -- actually, I'm sorry.
11 It's going to be the bottom of page 4 and to
12 the top of page 5. Your report lists your
13 experience as an expert witness, which we
14 talked about a little bit earlier. I just --
15 I'm wondering if you would confirm this is not
16 an exhaustive list of your experience as an
17 expert witness either via deposition or report.

18 A. I wouldn't want to testify that
19 this is absolutely complete, given the fact
20 that I don't keep a list compiled. This is
21 kind of compiled retrospectively from memory
22 and documents. And so this is the best I could
23 have done on April of 2021 --

24 Q. Understood. Thank you. So --

25 A. -- you might find something else.

1 Q. Was it --

2 A. -- in a commercial building where
3 our clinic was. It was just, you know, a
4 conference room in our clinic.

5 Q. And that was within -- was that
6 within a business --

7 A. It was --

8 Q. -- a psychiatric practice?

9 A. I'm sorry. I interrupted you.

10 It was within The Center For Marital
11 Health, which was a business that I and two
12 other people started and owned and ran. And in
13 that business, we continued the same kind of
14 work we did with the University minus the large
15 number of trainees.

16 Q. You mentioned that after '93, you
17 were not being paid by the University. Were
18 you providing your clinical psychiatric
19 professorship gratuitously?

20 A. Meaning without pay? Yes.

21 Q. Okay. Do you know if, after you
22 moved the clinic away from Case Western
23 Reserve, if Case Western Reserve University
24 Medical School created a separate gender
25 identity clinic?

1 A. Years later they did --

2 Q. Oh, sorry.

3 A. -- I would say, they created a
4 separate clinic perhaps in 2017, 2016.

5 Q. Do you know the name of that
6 clinic?

7 A. I don't think it's in the
8 department of psychiatry. I think it's in the
9 department of pediatrics. And the answer to
10 your question is, no.

11 Q. Does The LGBTQ and Gender Care
12 Program sound familiar?

13 A. No.

14 Q. But have you -- sorry. Have you
15 evaluated any patients through that separate
16 clinic that Case Western Reserve has?

17 A. No. Much to my dismay, that clinic
18 was formed and maintained without any input
19 from me, who I thought was one of the experts
20 in the field.

21 Q. Do you know if they have
22 psychiatrists, within that clinic?

23 A. I -- I'm not knowledgeable about
24 the composition of that clinic. There is a
25 very strong liaison between our department of

1 What do you mean by, "This era"?

2 A. Before 1993.

3 Q. Okay. And what do you mean by,
4 "Occasional"?

5 A. I would say that 95 percent of the
6 patients that we saw were 16 and 17, 18 and up.
7 We could debate what the word, "Child," means,
8 but to me an 11-year-old is a child, even
9 a 13-year-old is a child, especially when my
10 children were 13. And so we -- in the first
11 twenty years, transgender issues were primarily
12 an older teenager and adult, mostly adult
13 issues. In recent years, I would say, 12, 15
14 years, the number of adolescents appearing in
15 gender clinics at our place and everywhere as
16 far as I can see has increased exponentially,
17 especially the number of teenage girls who are
18 declaring themselves trans boys.

19 Q. So how many -- sorry. So the first
20 twenty or so years, you said approximately 5
21 percent of all patients were children.

22 A. Were younger -- on the younger end
23 of the spectrum --

24 Q. Right.

25 A. -- yes.

1 it, you see? But at this moment -- this week,
2 I have one patient that I see weekly, who is a
3 transgender teen. My staff -- if I can be
4 presumptuous to call them, "My staff" -- our
5 staff sees more.

6 Q. And thinking about the last year,
7 approximately how many adult patients did you
8 see -- and let's use your framing of,
9 "Regular." So that could be one, for one
10 followup visit or that could be for more -- how
11 many adult patients did you see for treatment
12 of gender dysphoria?

13 A. Approximately six.

14 Q. And using that same framing of,
15 "Regular," how many children, so under age 11?

16 A. In the last year?

17 Q. Yes, yes. In the last year.

18 A. Zero.

19 Q. How many adolescents in regular
20 treatment for gender dysphoria would you
21 approximate you've seen in the last five years
22 individually, exclusive of your supervision of
23 other clinicians?

24 A. If you ask me the question in the
25 last year, I would have told you five or six,

1 but since you ask it as a five-year period, I'm
2 at a loss to tell you whether it's twelve or
3 fifteen. I --

4 Q. An approximate is fine. Thank you.

5 A. -- let's just say a dozen with an
6 asterisk, very approximate.

7 Q. And jumping a little bit more in
8 terms of time. How about the last ten years?

9 A. Again, using the same asterisk, I
10 would say, double it.

11 Q. Okay. And you said zero people
12 under age 11, so children this last year. What
13 about in the last five years?

14 A. Oh, two years ago, we had this
15 charming little 6-year-old. One of my
16 colleagues specializes in children and I get to
17 hear about these cases. Occasionally I get to
18 meet the parents, but I personally have not
19 delivered a psychotherapeutic care or
20 evaluation directly of a child with the
21 exception of this one person that I was
22 involved with.

23 Q. And that was this last year, you
24 said?

25 A. That was -- I think it was probably

1 two, two and a half years ago.

2 Q. Oh, okay. And what kind of
3 treatment -- I should say, have you referred
4 any of those adolescent patients for additional
5 treatment, besides psychotherapy, for the
6 treatment of gender dysphoria?

7 A. Yes.

8 Q. And what kinds of treatment have
9 you referred them for?

10 A. For endocrine treatment.

11 Q. Okay. And approximately what
12 percentage of those adolescent patients have
13 you referred for endocrine treatment?

14 A. Give me the timeframe of that
15 question, please.

16 Q. Sure. So you said a few moments
17 ago, in the last five years, you saw maybe,
18 asterisk, 12 to 15 adolescent individually
19 yourself. Of those 12 to 15, what would be the
20 approximate percentage you referred for
21 endocrine treatment?

22 A. I'm hesitating to answer the
23 question, because some of those children have
24 been taking testosterone or estrogen
25 surreptitiously from their parents. And while

1 I didn't refer them for the treatment, I was
2 seeing them while they were taking the
3 treatment. So if we're only talking about
4 adolescent -- referrals of adolescents for
5 hormones, I would say a very small percentage
6 of those, say, I guess you would say 10
7 percent.

8 Q. Fair enough. Have you had yourself
9 individually as a clinician, have you had any
10 non-transgender children who you have made a
11 referral for endocrine treatments related to
12 other conditions?

13 A. No.

14 Q. Okay. So then zooming out 30,000
15 foot view of your 48-year career now, would you
16 say overall, you have provided treatment --
17 that is, psychiatric treatment -- to mostly
18 adults experiencing gender dysphoria, gender
19 identity issues?

20 MR. KNEPPER: Objection, form.

21 A. I would say that throughout my
22 career, we should divide my career into the
23 first twenty years where mostly adults were
24 seen by our team and myself. And then we ought
25 to talk about the last ten or fifteen years

1 where the number of adults has diminished and
2 the number of adolescents has increased
3 dramatically.

4 Q. Okay. Thank you. So as a part of
5 your private practice, do you write letters of
6 authorization for endocrine treatments?

7 A. Yes.

8 Q. And do you write letters of
9 authorization for gender affirming surgeries?

10 A. I have. I have not recently,
11 because most of my patients are 13 or 15 or 16,
12 you know.

13 Q. Okay. And I'm sorry. Just by,
14 "Recent," when was the last time you wrote a
15 letter of authorization for a gender affirming
16 surgery for an adult?

17 A. Probably twelve months ago.

18 Q. Okay. And over the course of your
19 career focusing on your treatment of adults
20 experiencing gender identity issues, for what
21 percentage of those patients would you estimate
22 you wrote a letter of authorization for gender
23 affirming surgery for?

24 MR. KNEPPER: Objection, form.

25 A. Again, I would like to put an

1 asterisk to whatever I answer this question as.
2 I have not kept track of those figures. I have
3 written -- I've written or cosigned letters for
4 hormone treatments and for gender confirming
5 surgeries for many people. There were more
6 people in the '70s and '80s than in recent
7 decades. In part as a reflection of my own
8 evolution of understanding of these problems
9 and in part it's a reflection of the demography
10 of patients who are coming to see me. I really
11 would not like to answer that question, only
12 because I don't know if the word, "Fifteen," or
13 the word, "Twenty-five," or the word,
14 "Thirty-five," is more accurate --

15 Q. Understood.

16 A. -- but I can tell you, I have
17 written letters, especially in the early years,
18 for the things that you're making reference to.

19 - - - - -

20 (Thereupon, Deposition Exhibit 2,
21 12/21/2020 Zoom Deposition of
22 Stephen B. Levine, M.D., was marked
23 for purposes of identification.)

24 - - - - -

25 Q. Okay. For the record, I'm showing

1 Q. Do you think as a general matter
2 that it's good for patients who come to DELR
3 for services related to gender dysphoria to be
4 able to have insurance coverage of that care?

5 MR. KNEPPER: Objection, form.
6 Beyond the scope.

7 A. Well, the people who come to DELR
8 are generally coming for evaluation and
9 psychotherapy services. And I believe it's
10 very important that people have access to
11 mental health care and that mental health care
12 for many of our patients are not wealthy,
13 affluent people. And the fees that even
14 masters prepared people charge can become
15 prohibitive. And so I think it's a very nice
16 idea, the psychiatric services, mental health
17 services evaluation and ongoing treatments,
18 with or without medication, it would be nice to
19 be able to cover those things, yes. I think
20 that's a long answer, yes.

21 Q. Understood. And thinking about the
22 treatment that you refer patients out for, the
23 endocrine treatments in particular, do you
24 think it is generally good if you provide
25 authorization for that treatment that the

1 patient be able to afford it?

2 MR. KNEPPER: Objection, form.

3 A. May I say, of course?

4 Q. You may. You may say anything you
5 would like.

6 A. Of course.

7 Q. Thank you. Well, anything you
8 would like within reason.

9 If you make a letter of authorization for
10 a patient for the treatment of gender dysphoria
11 specifically related to a surgical treatment,
12 do you think it is good that they be able to
13 access that treatment that you've authorized?

14 MR. KNEPPER: Objection, form.

15 A. Not to be cagey, I want to talk
16 about one word you just used in that sentence.
17 I need you to understand that historically in
18 our clinic for those 47 years, our clinics
19 for 47 years, we are not in the business and we
20 have never been in the business of recommending
21 surgery or recommending hormones. We recommend
22 a continued evaluation so that we -- the person
23 can make up their mind how to proceed.

24 It is not our knowledge base to know
25 who's going to do better and who's going to do

1 worse and who is not going to have any
2 difference at all with hormones or with
3 surgery. So what we do is we say, we will
4 write a letter of support for endocrine
5 treatment or for hormones if this is what you
6 want. And we say what our concerns are. We
7 tell the endocrinologist and we tell the
8 surgeon what our concerns are and that we
9 see -- we have reservations about this, and
10 these are our reservations, but the patient has
11 decided this is what he or she wants to do.

12 And so we write a letter of support, but
13 I don't -- every time you use the word,
14 "Recommendation," there's part of me that wants
15 to say, no, we do not recommend. We have never
16 recommended. We have not had the knowledge
17 base. We have not had the clinical experience
18 and the knowledge base to say, I'm a doctor. I
19 know this field. This is what I recommend to
20 make you better. We do not talk that way. We
21 do not think that way. And so I may want to
22 always put an asterisk to any sentence that you
23 use the word, "Recommend." I need you to
24 understand that that's where I'm coming from.

25 MR. CHARLES: Thank you,

1 Dr. Levine.

2 Excuse me just a moment. Can you read
3 back my question. I don't recall if I used,
4 "Recommend." I thought I used,
5 "Authorization." I just want to make sure.

6 (Record was read.)

7 MR. CHARLES: If we could just go
8 off the record for a second.

9 VIDEOGRAPHER: Off the record 10:52.

10 (Discussion held off the record.)

11 VIDEOGRAPHER: On the record 10:53.

12 BY MR. CHARLES:

13 Q. Okay. Thank you for that
14 clarification, Dr. Levine. I'll be more
15 careful about using terminology more close to,
16 "Authorization," rather than, "Recommendation,"
17 and I understand your distinction in your
18 practice. So do you, though, think it's good,
19 if you are authorizing a treatment, a patient
20 has said, This is the treatment I would like,
21 and you have done an evaluation and determined
22 that you will write, as you said, a letter of
23 support, do you then, as a practitioner, think
24 it's good that they can access it, that they
25 can afford it?

1 concept of agency and being a doctor, I think
2 is different than the implication of your
3 question.

4 Q. Is the worrisomeness for a
5 patient's future health, is that a reason to
6 deny all medical care for gender dysphoria?

7 A. Absolutely not.

8 Q. Dr. Levine, I'd like to return back
9 to, I believe it's Exhibit 2, the Claire
10 deposition. And please, if you would turn to
11 page 156.

12 A. I'm sorry. 150 what?

13 Q. Page 156. And beginning at line 10
14 on page 156, Dr. Levine, I'll read it, if
15 you'll just follow along, please.

16 Question: "Are you aware that this case
17 concerns an insurance exclusion that is
18 categorical at preventing" --

19 Skipping to line 15.

20 "-- hormones and surgery as a treatment
21 for gender dysphoria?"

22 Answer: "I am aware that your plaintiffs
23 are suing to get coverage for -- that is not
24 provided by their particular insurance. I am
25 aware of that."

1 demonstrate their efficacy. This is the
2 problem.

3 This is the essence of the problem. This
4 is, I think the essence of my testimony with
5 you today. It's not whether I personally as a
6 doctor would like this patient to have
7 insurance to cover their hormones. It's about,
8 is this the right thing to do for this person
9 and can I help the person see clearly what the
10 dangers are and what the benefits are. That's
11 the issue for a doctor, for Stephen Levine as a
12 doctor. I hope that's a cogent answer --

13 Q. It is --

14 A. -- to your question.

15 Q. -- it is cogent. Thank you.

16 Given all of that, is that -- so you just
17 explained, testified that there are
18 complications, some lack of -- and I'm
19 summarizing here, so I will confirm that this
20 is an accurate summary of what you just shared,
21 but I can't possibly repeat all of that. Given
22 all of those concerns that you have, is that a
23 reason to deny all medical interventions to
24 people with gender dysphoria?

25 MR. KNEPPER: Objection, form.

1 A. No, but that's not -- that's a
2 separate question about insurance.

3 Q. Yes, it is a separate question. So
4 now I'm asking: Are those concerns you raised
5 justifications in your mind for denying medical
6 interventions to all people with gender
7 dysphoria?

8 MR. KNEPPER: Objection, form.

9 A. You know, I'm not advocating
10 denying endocrine treatment or surgical
11 treatment. I'm just saying that we as a
12 medical profession need to walk the walk that
13 we talk. We say as a principle of ethics that
14 our interventions should be based upon the best
15 current knowledge, it should be based on
16 science. It should not be based on politics.
17 It should not be based on fashion. It should
18 not be based on civil rights considerations.
19 They should be based on the kinds of studies
20 that I just described to you with predetermined
21 outcome majors that are agreed upon --

22 Q. Sorry?

23 A. -- period.

24 Q. I was --

25 A. I forgot to put the period.

1 Q. That's okay. Did you just say,
2 Dr. Levine, you're not an expert in health
3 insurance?

4 A. I am not an expert in health
5 insurance.

6 Q. Okay. Or what insurance should or
7 should not cover?

8 A. Yes.

9 Q. Do you recall what the insurance
10 billing code typically is for psychotherapy for
11 gender dysphoria? I know it's been a long time
12 since you've accepted commercial insurance, so
13 I'm not sure if the billing codes are the same,
14 but do you recall --

15 A. The billing code is 90837.

16 Q. Okay. Is there a code that you're
17 familiar with that is F64.0?

18 A. That's not a billing -- that's
19 diagnostic code --

20 Q. Thank you.

21 A. -- there's a separate code for
22 diagnosis and a separate code for procedure.

23 Q. I see. So F64.0 is a diagnostic
24 code?

25 A. Yes.

1 VIDEOGRAPHER: Off the record 11:26.

2 (Recess taken.)

3 VIDEOGRAPHER: On the record 11:31.

4 BY MR. CHARLES:

5 Q. Okay. Dr. Levine, in your report,
6 you stated that you had not met with any of the
7 plaintiffs in this case, correct?

8 A. Yes.

9 Q. Okay. And you have not interviewed
10 any of the plaintiffs in this case, correct?

11 A. Correct.

12 Q. And so you are not offering any
13 opinions about the plaintiffs in this case,
14 correct?

15 A. Correct.

16 Q. Okay. And that would include the
17 veracity of their experiences of gender
18 dysphoria, correct?

19 A. Yes, correct.

20 Q. And that would not include the
21 accuracy of their gender dysphoria diagnoses,
22 correct?

23 A. Correct.

24 Q. Okay. You're not offering any
25 opinions about their mental health histories?

1 A. Correct.

2 Q. Nor any of the affects of the
3 gender affirming treatment they may have
4 received?

5 A. Correct.

6 Q. Okay. Thank you. Let's return to
7 your report. I don't know if you have that --

8 A. My report?

9 Q. Yes. You can put away that
10 document in your hand.

11 So if you would, please, turn to page 6
12 of your report.

13 Okay. So on page 6, paragraph a. at the
14 bottom of the page there, Dr. Levine. The
15 report states that this is one of the opinions
16 you're offering, which is, "Sex as defined by
17 biology and reproductive function cannot be
18 changed. While hormonal and surgical
19 procedures may enable some individuals to
20 'pass' as the opposite gender during some or
21 all of their lives, such procedures carry with
22 them physical, psychological, and social risks,
23 and no procedures can enable an individual to
24 perform the reproductive role of the opposite
25 sex." Did I read that correctly?

1 methodology and are capable of critically
2 reviewing the literature. So your statement is
3 true on the most superficial level, but is
4 totally incorrect when it comes to scientific
5 standards of care for issuing guidelines for
6 the medical profession. So I don't know how to
7 answer the question. On the surface, the
8 answer is, yes. And underneath the surface,
9 the answer is, no.

10 Q. So the International Journal For
11 Transgender Health is still a peer-reviewed
12 source, though, right?

13 A. It's peer reviewed by people who
14 make their living supporting transgender care.

15 Q. But it's still peer reviewed,
16 right?

17 A. It's peer reviewed --

18 Q. And as for your --

19 A. -- I think it's peer reviewed.

20 Q. Okay. Understood. And as for your
21 more conservative approach, can you cite to any
22 studies or research that resulted in better
23 outcomes than people who adhere strictly to the
24 WPATH standards of care version 7?

25 A. No. This is part of the problem in

1 evaluation leading to a therapeutic process, it
2 seems prudent, given the fact that we are
3 changing people's bodies, especially teenagers'
4 bodies, and they are not of developmental
5 sophistication yet that court systems or at
6 least one court system thinks they're certainly
7 too young to make these life-altering
8 decisions. So people in SEGM are biased in the
9 direction of being conservative and providing
10 psychotherapeutic evaluations of the child, of
11 the teenager and of their parents, of their
12 family systems to see if we can find a way to
13 help them be informed about what is going --
14 what they think they want to do in their
15 future.

16 Q. And so when you provide letters of
17 authorization for hormones or for surgery, do
18 you do so in accordance with the WPATH
19 standards of care?

20 A. Yes. That is the standard, to
21 provide a letter of recommendation.

22 Q. Okay. So turning back to your
23 report, Dr. Levine. You can go ahead and put
24 away the trial transcript there.

25 A. I'm sorry. Did you say, "Turning

1 Q. Okay. So is a, "Hypothesis," an
2 idea about why something happens, but doesn't
3 provide evidence for why something is
4 happening?

5 MR. KNEPPER: Objection, form.

6 A. A, "Hypothesis," generates the
7 pursuit of evidence.

8 Q. Has social contagion as an
9 explanation for increased cases of gender
10 dysphoria been scientifically proven yet?

11 A. No. But when you seek -- when you
12 see -- actually see patients and talk to them
13 about their friends and hear about the
14 influence of the Internet and the gurus on the
15 Internet who tell 13 and 12-year-old children
16 who are concerned about menses or concerned
17 about breast development or concerned about
18 their bodies changing and then they're told
19 that they're transsexual by somebody that
20 they've never met that they talked to on the
21 Internet, that would be social contagion or
22 social education.

23 Or when you hear about a friend who
24 declares themselves trans and then your patient
25 six months later declares themselves trans, you

1 wonder about the -- the interpersonal,
2 psychological link between best friends in
3 young puberty, young years of puberty and how
4 one can identify with one's friends and that
5 would be a social contagion. Those are 3the
6 kinds of ideas that people like me get when we
7 sit with people week after week talking about
8 their lives. You see, that's not science.

9 But that is clinician and this is the
10 kind of thing that leads to intuition, clinical
11 intuition and that's the source of the
12 generation of the hypothesis. But we think as
13 clinicians, when we hear -- I mean, I don't
14 think I've ever seen a teenager trans person
15 who hasn't been heavily involved and influenced
16 by the Internet, for example, but I have not
17 done studies to document that in a way that
18 would be scientifically acceptable. There are
19 other people who have.

20 And I doubt very much if you'll ever find
21 a clinician on any side of this issue, you see,
22 who would say, oh, no most of my patients have
23 never talked to anyone on the Internet about
24 transgender. The Internet is just part of life
25 today and -- but transgender teenagers spend

1 hours and hours of their time getting counseled
2 or participating with the virtual trans
3 community. That's a hypothesis.

4 Q. So no scientific citation?

5 A. When we use the word, "Scientific,"
6 in the best sense, yes, the answer to your
7 question is, no scientific.

8 Q. Okay. No studies of citations you
9 can point to today to support that hypothesis?

10 A. Oh, I think Lisa Littman's studies
11 are in the literature and/or in press that
12 documents this.

13 - - - - -

14 (Thereupon, Deposition Exhibit 7,
15 "Correction: Parent reports of
16 adolescents and young adults
17 perceived to show signs of a rapid
18 onset of gender dysphoria," Article,
19 was marked for purposes of
20 identification.)

21 - - - - -

22 Q. Okay. For the record, please note
23 I'm showing to Dr. Levine what has been marked
24 as Exhibit 7. "Correction: Parent reports of
25 adolescents and young adults perceived to show

1 signs of a rapid onset of gender dysphoria," by
2 Lisa Littman published March 19, 2019. Have
3 you seen this material before, Dr. Levine?

4 A. I've seen of it. I don't think
5 I've read it.

6 Q. Okay. Were you aware that the Lisa
7 Littman article had to be withdrawn, corrected
8 and republished?

9 A. Yes.

10 Q. Okay. And were you aware that the
11 initial article was based on a survey of
12 parents --

13 A. Yes.

14 Q. -- of purportedly transgender
15 children and the parents were recorded -- I'm
16 sorry. Let me start over. Were you aware that
17 the Littman article was based on a survey of
18 parents who were recruited through some parent
19 groups?

20 MR. KNEPPER: Objection, form.

21 A. I knew it was a survey of parents.

22 Q. Okay. And did you know there were
23 no report-outs from the young adults of those
24 parents in the article?

25 A. It was a report of parents'

1 transitioning. However, it is...important to
2 note that there are other survey items where
3 the parent would have direct access to
4 information about their child and that those
5 answers reflect items that can be directly
6 observed." Did I read that correctly?

7 A. Yes, you did.

8 Q. All right. Your report also cites
9 as support for the social contagion hypothesis
10 to an article from Medscape.com written by
11 Becky McCall and Lisa Nainggolan as support for
12 the social contagion theory. Is that correct?
13 I'm sorry. It's not going to be on this
14 article, Doctor.

15 A. I don't know that article.

16 Q. Okay.

17 A. You haven't asked me a question
18 about this. Did I misunderstand something?

19 Q. No, no. Sorry. We're just --

20 A. You haven't asked my opinions about
21 that, yeah.

22 - - - - -

23 (Thereupon, Deposition Exhibit 8,
24 "Transgender Teens: Is the Tide
25 Starting To Turn?" Article, was

1 marked for purposes of
2 identification.)

3 - - - - -

4 Q. Yeah. So, for the record, I'm
5 showing Dr. Levine what has been marked as
6 Exhibit 8. "Transgender Teens: Is the Tide
7 Starting To Turn?" by Becky McCall and Lisa
8 Nainggolan, April 26, 2021. Dr. Levine, you
9 said you have not reviewed this article before?

10 A. Which one are you referring to?

11 Q. I'm sorry. That one to your left.

12 A. This?

13 Q. Yes. Take your time.

14 A. Have I reviewed it, no. You know,
15 I've seen the picture of Keira Bell. I've seen
16 news reports of this in the past, but they were
17 just news reports, yeah.

18 Q. Do you know if either of the
19 authors of this article is a scientist?

20 A. I have no idea.

21 Q. Okay. Or a psychiatrist?

22 A. (Indicating.)

23 Q. I'm sorry. Could you make your
24 responses verbal? I'm forgetting.

25 A. I have no idea.

1 Q. Okay. Thank you. Have either of
2 them ever treated transgender children or
3 adolescents?

4 A. I would have no idea.

5 Q. Okay. To your knowledge, is the
6 information provided on Medscape.CA subject to
7 peer review?

8 A. I don't know how Medscape works.
9 I've heard there have been retractions, but I
10 don't know how their peer reviewed is made.
11 Perhaps people write in that, This is
12 ridiculous what you've been teaching or what
13 you've been saying, but whether they're peer
14 reviewed or not, I have no idea.

15 Q. So you probably -- I'm sorry. So
16 do you know if this article has been published
17 in a peer-reviewed journal to your knowledge?

18 A. "Transgender teens: Is the
19 Tides" -- that article?

20 Q. Yes.

21 A. I don't know. I don't know this
22 article. I don't know where it's from.

23 Q. Okay. So your report includes a
24 quotation from this article. "The vast
25 majority of youth now presenting with gender

1 multi-continental set of observations from
2 Europe, from Australia, from North America --

3 Q. Okay.

4 A. -- it almost doesn't even need
5 citations it's so clinically apparent.

6 Q. Okay. But there's no citation in
7 your report?

8 A. In my report, yes.

9 Q. Okay. So on page 18, going back to
10 your report, at the bottom of page 18, you use
11 a term, "Transgender Treatment Industry." Is
12 this the first time you have used this term?

13 A. In this report?

14 Q. No.

15 A. You mean, did I ever use it in
16 another report?

17 Q. Yeah, yeah.

18 A. I'm not sure. If this is -- if
19 it's not the first, it might be the second.

20 Q. And where did the term originate?

21 A. I think it -- the term originated
22 from Dwight Eisenhower at the end of his --
23 when he was leaving the presidency in 1952, he
24 warned the people about the military industrial
25 complex and that there was a very comfortable

1 the methods we made reference to before, the
2 efficacy of the treatment and the downsides of
3 the treatment. But because WPATH is an
4 advocacy organization and the scientific
5 establishment of the efficacy of their
6 treatments are not important to them, what they
7 are doing is teaching young mental health
8 professionals and medical professionals as a
9 whole what their ideology is. They say it's
10 scientifically established.

11 I'm here to tell you to the extent that I
12 understand science, it is not scientifically
13 established. In a sense, there is an industry
14 that has different elements that feed each
15 other; that's the transgender treatment
16 industry. I think if we put our heads
17 together, we could find another term.

18 Q. So did you coin that phrase then?

19 A. No --

20 Q. Okay.

21 A. -- no.

22 Q. Have you seen it used before in any
23 peer-reviewed articles?

24 A. Not in a peer-reviewed article.
25 I've seen it used in these kind of expert

1 opinion -- (Indicating.)

2 Q. Okay.

3 A. -- I would -- you know, if I had
4 time and I had a committee of people, I -- I
5 would probably find a different term for it.
6 But I don't mean it in a disparaging way. I
7 mean that this is a group of compassionate
8 people trying to help other people who actually
9 believe that the science has established the
10 best practices when in fact they're not well
11 informed.

12 Q. Do you need a sip of water after
13 that?

14 A. No. I'm just a long-winded guy.

15 I want to add, if I may, that we should
16 make a distinction between education and
17 indoctrination. Education can be based on
18 science. Indoctrination is based on preferred
19 beliefs that, if you allow me to use this term
20 again. The transgender treatment industry is
21 heavy on indoctrination and has declared, if
22 you look at the standards of care, if you don't
23 believe these systems, you're not a
24 competent -- you're not competent to take care
25 of people. That of course is the height of

1 A. No. Their gender dysphoria may be
2 a product, you see, of these other things. For
3 example, if you have someone who has been
4 sexually abused by her stepfather and becomes a
5 trans person in adolescents, we want to talk
6 about the sexual abuse and the process between
7 that person and what fears for the present and
8 the future that has caused the child. And
9 we're not attacking their trans identity.
10 We're trying to help them understand where they
11 came from and what they're coping with and why
12 they're so fearful or so distressed by their
13 body changing.

14 Q. And their gender dysphoria could be
15 separate and apart from that traumatic
16 experience?

17 A. Theoretically it could be, yes.

18 Q. And if it persisted sufficiently
19 enough, you would consider a letter of
20 authorization for --

21 A. Yes.

22 Q. -- hormones?

23 A. Yes.

24 MR. KNEPPER: Objection, form.

25 Q. Okay. If you would, please, turn

1 A. That is correct. And may I add
2 that it's very, very difficult to understand.
3 The natural question would be, how do you
4 compare the general population with the trans
5 people who did not have surgery with the trans
6 people who did have surgery.

7 Q. Thank you, Dr. Levine. That's not
8 my question, though. I just wanted to confirm
9 that was not the control group. You mentioned
10 this study later in your report, page 66
11 beginning at paragraph 74. Do you see that?

12 A. Um-hum.

13 Q. Okay. And basically that -- well,
14 here, let me point you exactly. The sentence
15 starts with, "Similarly," about halfway down
16 the page, third sentence of that paragraph.

17 A. Um-hum.

18 Q. And, as you mentioned, you cite the
19 Dhejne study and I believe -- or I should ask:
20 Is the Denmark study you're referencing the
21 study directly after it --

22 A. The Simonsen study.

23 Q. -- the Simonsen study?

24 A. Yes.

25 Q. Okay. So beginning with the Dhejne

1 study, do you think because that study showed
2 that some people committed suicide after gender
3 affirming surgery that no patient should be
4 able to access gender affirming surgery?

5 MR. KNEPPER: Objection, form.

6 A. That would be illogical.

7 Q. Okay. Dr. Levine, I understand you
8 said that would be illogical, but just to be
9 clear. You're not recommending -- sorry. I'm
10 not using that word. You're not saying that
11 the fact that some people commit suicide
12 following gender affirming surgery means that
13 there should be a ban on access to that
14 surgery. Is that right?

15 A. Not for that reason, no.

16 MR. KNEPPER: Objection, form.

17 Q. Not for that reason. Okay. Are
18 you recommending that there would be bans on
19 gender affirming surgery for any reason?

20 A. I think there are -- you know, I
21 think most prudent people in this field, just
22 to use the example of what you read out loud
23 about the Finland study, a case-by-case basis.
24 That's how doctor need to decide things, but
25 there are many, many reasons to be cautious

1 fashion and to be very hesitant about going
2 forward.

3 Q. But you're not recommending total
4 bans on gender affirming surgery?

5 A. I'm not recommending total bans.
6 I'm aware of the individual circumstances of
7 individual people's lives and their commitment
8 to transgender living. And I don't want to be
9 draconian about this. I want to be
10 compassionate about this.

11 Q. I understand. I appreciate that.
12 I just want to make sure I'm understanding you
13 correctly.

14 - - - - -

15 (Thereupon, Deposition Exhibit 12,
16 "Long-Term Follow-Up of Transsexual
17 Persons Undergoing Sex Reassignment
18 Surgery: Cohort Study in Sweden,"
19 Article, was marked for purposes of
20 identification.)

21 - - - - -

22 Q. So for the record, I'm presenting
23 to Dr. Levine what has been marked as
24 Exhibit 12. "Long-Term Follow-Up of
25 Transsexual Persons Undergoing Sex Reassignment

1 For the 22nd time today, did I read that
2 correctly?

3 A. It's the 23rd time.

4 Q. Oh, okay.

5 A. Yes.

6 Q. I was hoping you weren't counting,
7 but, okay. Did you testify earlier today that
8 the limitation of the Dhejne study is that the
9 controls were not transgender persons who had
10 not undergone gender affirming surgery?

11 A. Yes.

12 MR. KNEPPER: Objection, form.

13 Q. Okay. You can set that aside,
14 Dr. Levine.

15 - - - - -

16 (Thereupon, Deposition Exhibit 13,
17 2017 "On Gender Dysphoria," Booklet
18 From Department of Clinical
19 Neuroscience, Karolinska Institutet,
20 Stockholm, Sweden, was marked for
21 purposes of identification.)

22 - - - - -

23 Q. For the record, Dr. Levine has an
24 exhibit that has been marked as Exhibit 13.
25 "On Gender Dysphoria," by Cecilia Dhejne from

1 ideation in transgender people.

2 A. Well, you know about the
3 Branstrom-Pachankis study and the criticism of
4 the study --

5 Q. But I'm not talking about the
6 study.

7 A. -- and part of the study
8 demonstrated that it increased suicidal
9 ideation and attempts in the first two and a
10 half years after surgery, especially in the
11 first year --

12 Q. Right. Is your testimony --

13 A. -- so I'm not testifying that. I
14 thought you were asking me about this, which I
15 need to comment on, because this is not an
16 accurate depiction of my statement in the
17 reference. (Indicating.)

18 Q. Well, that's not what I'm asking
19 about, Dr. Levine.

20 A. Well, you're reading this and I'm
21 misquoted here. So I don't want you to imply
22 that she is accurately representing my views,
23 because I did not say that gender affirming
24 treatment in general should be stopped. I've
25 never said that. This is an article about

1 at different times have reported that in the
2 large majority of patients, absent a
3 substantial intervention such as social
4 transition and/or hormone therapy, gender
5 dysphoria does not," continue, "through
6 puberty."

7 So there are some children who persist in
8 their asserted gender identity through puberty,
9 correct?

10 MR. KNEPPER: Objection, form.

11 A. Correct.

12 Q. And some who persist in wanting to
13 transition via medical treatments?

14 MR. KNEPPER: Objection, form.

15 A. Yes. Some of the children have
16 learned about medical treatments somewhere
17 along the line and they feel instantly that
18 this is for them.

19 Q. And then looking at paragraph 56,
20 which is on page 41, so just the very next page
21 on the bottom, the second sentence in that
22 paragraph. "I observe an increasingly vocal
23 online community of young women who have
24 reclaimed a female identity after claiming a
25 male...identity at some point during their teen

1 transgender people is individual based, right?

2 A. Well, it's both --

3 MR. KNEPPER: Objection, form.

4 A. -- yes, that's partially true. And
5 ideally that's true, but it's obviously not
6 entirely true. It's why we're here, is it's
7 categorically based.

8 Q. Let me rephrase that. You design
9 treatment for your patients based on what that
10 patient in front of you, what they need, what
11 they want, what you determine -- sorry. Not
12 what you determine, but what you might
13 authorize?

14 MR. KNEPPER: Objection, form.

15 A. What the patient and I discern
16 together.

17 Q. Thank you. Okay. Let's jump to,
18 again, still in your report, page 68.

19 A. We've left 40 and 41? 68.

20 Q. Okay. Looking at the bottom of
21 page 68, Dr. Levine, paragraph 78. It states,
22 "Similarly, the American Psychological
23 Association has stated because approach" --

24 A. Sorry.

25 Q. I apologize.

1 Gender Nonconforming People (2015)."

2 So is that lack of consensus that you
3 discuss a justification to categorically ban
4 social transition for children as a treatment
5 for gender dysphoria?

6 MR. KNEPPER: Objection, form.

7 A. By, "Children," you mean 6 and 7
8 year olds?

9 Q. Those for whom medical intervention
10 is not indicated.

11 A. Is that a reason to ban it?

12 Q. Correct, social transition.

13 MR. KNEPPER: Objection, form.

14 A. The reason to -- so let me qualify
15 that. There's a, yes, answer, there's a reason
16 to ban it. And the reason to ban it is both a
17 developmental and an ethical reason. There
18 have been eleven studies of these cross-gender
19 identity children who are not socially
20 transitioned and the vast majority of them
21 de-transition by the time they're mid
22 adolescents or older adolescents. They become
23 homosexual individuals usually or bisexual
24 individuals, but they are cis gender.

25 So if we take a 6-year-old child and

1 A. -- nor you didn't ask me to comment
2 on that.

3 Q. It was related to what you had said
4 before. So this is related but not related to
5 what we just read. So you can put that aside.

6 A. Okay. But your next question was
7 about puberty blocking hormones, which are not
8 being used for 6-year-old's and 7-year-old's --

9 Q. Correct, yes, a separate group of
10 people.

11 A. -- so we're on a different
12 category.

13 Q. Yes.

14 A. Okay. So you asked me if I think
15 puberty blocking hormones should be used on a
16 case-by-case basis?

17 Q. Correct, yes.

18 A. I don't think so.

19 Q. So that is to say, there are no
20 circumstances you would advocate for a total
21 ban on that intervention?

22 MR. KNEPPER: Objection, form.

23 A. Number one, I've never seen a child
24 where that has come up where I thought it was a
25 good idea. In the cases I've seen, it was like

1 a treatment for the mother's pathology, not for
2 the child. And it's like a warning sign, boy,
3 be careful. You see, if you see one case like
4 that, you wonder -- and it's so conspicuous,
5 you wonder in the next case, if the same thing
6 is going on in a more subtle way.

7 Is the child acting out the ambitions of
8 the mother or the father? I just think
9 prudence -- I think considering the child has
10 not gone through puberty or has not gone far
11 into puberty and puberty brings all kind of
12 psychological, physical and social changes to a
13 child and those changes lead to desistance in
14 many, many children, to put them into a state
15 where all their peers are developing physically
16 and they're going to be poirot (phonetic).

17 And then most of those children have
18 social anxiety problems and they avoid -- they
19 don't have friends, right. And this is going
20 to make them even more different than their
21 peers and it's gone to deprive them of the
22 sexualization of their mind and the discovery
23 of masturbation and the discovery of sexual
24 desire for partners, you see. This is only
25 going to increase the child's difference from

1 her peers or his peers and I don't think this
2 is a prudent idea.

3 And if you wanted me to suggest a ban on
4 anything, it would be a ban on using puberty
5 blocking hormones, especially when the
6 evaluation of those children are focused on the
7 gender dysphoria of the child and not on the
8 background of the child and not on what's going
9 on. So I think that's an answer to your
10 question.

11 If we're going to use these drugs, if
12 we're going to use social transformation of
13 children, if we're going to use puberty
14 blocking hormones, it should only be used in a
15 carefully designed protocol. And follow up has
16 to be guaranteed so in one year and in two
17 years and in three years and before we start
18 giving cross-gender hormones we have data --

19 Q. Sorry.

20 A. -- so the answer to your question
21 is, I would consider banning puberty blocking
22 hormones even for children who have been
23 cross-gender identified for four years to give
24 them a chance to desist, which is exactly what
25 the Dutch protocol did, by the way.

1 Q. Sorry. So you just said you would
2 ban -- you would recommend a ban on --

3 A. If --

4 MR. KNEPPER: Objection, form.

5 A. -- look, I'm a doctor. I'm not a
6 policy maker --

7 Q. I understand, yes.

8 A. -- if you ask me my political
9 opinion about, should we ban this, is that a
10 reasonable thing, I think there's a very strong
11 argument for banning puberty blocking hormones.

12 Q. Okay. And, right. So you're here
13 as an expert offering an expert opinion. So
14 are you separating that from -- like are you
15 saying your political views that you would
16 advocate for bans or are you saying your expert
17 opinion you're offering in this case is you
18 would recommend ban?

19 MR. KNEPPER: Objection, form.

20 A. I would recommend ban. To what
21 extent it's from my politics or from my being a
22 parent or from my being a doctor, I don't know.
23 I would recommend we not use puberty blocking
24 hormones.

25 Q. In Claire, in this case that we

1 Answer: "Where we had a healthy mother
2 and father, an intact family who was
3 psychologically informed and who has -- where a
4 child has come out of toddlerhood acting
5 consistently in a gender atypical fashion, and
6 where the parents are not homophobic..."

7 Question: "The parents are not what kind
8 of people?"

9 Answer: "Homophobic."

10 For the 27th time, did I read that
11 correctly? Did I read that correctly?

12 A. Yes.

13 MR. CHARLES: Okay. All right.
14 Let's go ahead and take a break for a few
15 minutes.

16 VIDEOGRAPHER: Off the record 3:20.

17 (Recess taken.)

18 VIDEOGRAPHER: On the record 3:38.

19 BY MR. CHARLES:

20 Q. So, Dr. Levine, before the break,
21 you were talking about 6 and 7 year olds and
22 you mentioned there were eleven studies. Can
23 you identify which eleven studies from your
24 report you're referring to?

25 A. Cantor, the reference Cantor lists

1 the eleven studies and these eleven studies
2 have been done over probably thirty years.

3 Q. Okay. So Cantor was one review of
4 eleven studies?

5 A. Cantor was a review of the eleven
6 studies. I can't list to you the eleven
7 individual studies. The latest one is written
8 by Singh, S-i-n-g-h. It was published in April
9 of 2021, in the Frontiers of Psychiatry. And
10 that perhaps is the most comprehensive of them.
11 And that's the one that confirms -- that's a
12 study of boys and it confirmed that 12.2, I
13 think percentage of them persisted over a
14 thirteen-year period.

15 Q. So that was one -- that was the
16 Singh study that came out. Is that same study
17 mentioned in the Cantor review?

18 A. (Nodding.)

19 Q. Okay. And you said that
20 established that 12.2 percent of prepubertal
21 boys persisted into adolescents? Okay.

22 A. Yes. This harkens back to the
23 ethical issue that I talked about before. You
24 know, if you know that 88 percent of them are
25 going to persist -- desist, why in the world

1 identified 60,000 case reports world wide on
2 the Internet. See Exposito-Campos..." --

3 A. That is an error, by the way.

4 Q. Sorry. Which part of that is an
5 error?

6 A. That, "60,000," is my error. It
7 should say, "16,000."

8 - - - - -

9 (Thereupon, Deposition Exhibit 17,
10 "A Typology of Gender Detransition
11 and Its Implications for Healthcare
12 Providers," Article, was marked for
13 purposes of identification.)

14 - - - - -

15 Q. Okay. So for the record, I'm
16 showing Dr. Levine what has been marked as
17 Exhibit 17. "A Typology of Gender Detransition
18 and Its Implications for Healthcare Providers,"
19 Pablo Exposito-Campos, 2021. Okay. Have you
20 seen this study before, Dr. Levine?

21 A. Yes.

22 Q. Okay. So on page 1 of this report,
23 about halfway through the very first paragraph
24 in the introduction beginning with, "As a
25 consequence." Do you see that there?

1 important to note that this typology does not
2 suggest two clear-cut categories, for a
3 secondary detransition can lead to a primary
4 detransition" -- oh, sorry. Let me start over.
5 Sorry.

6 Okay. Let me start from a different
7 place, Dr. Levine. The second sentence.
8 "In r/detrans" --

9 And there's an HTTP address --

10 A. Okay.

11 Q. Okay. You see that.

12 -- "a subreddit for detransitioners to
13 share their experiences with more than 16,000
14 members, one can find several stories of people
15 who call their transgender status into question
16 after stopping transitioning due to medical
17 complications or feeling dissatisfied with
18 their treatment results"?

19 Do you know what a, "Subreddit," is,
20 Dr. Levine?

21 A. I believe it's just a division of a
22 larger website where people, you know, with
23 similar interests.

24 Q. Okay. Do you understand this
25 sentence to be suggesting that all 16,000 of

1 those members have offered a story of
2 detransition?

3 MR. KNEPPER: Objection, form.

4 A. I think -- I think it may be true
5 that either they have offered a personal story
6 or they're fascinated because of their own
7 considerations of that story. They're thinking
8 about it themselves, which would be in keeping
9 with the idea that even people who have
10 transitioned begin to doubt whether they made a
11 wise decision and they're considering
12 detransition. I'm not so sure it means that
13 all 16,000. I would have no way of
14 ascertaining that. You know, in my worry, I
15 would lean towards most of them are seriously
16 considering or have detransitioned. And in my
17 skepticism, I would say I'm not sure whether
18 it's 15,000 or 12,000 or 8,000.

19 Q. But you have no way to confirm
20 that --

21 A. I have no way.

22 Q. -- if it's all of them or a few of
23 them or three of them?

24 A. You're absolutely right. I have no
25 way of confirming that.

1 where hormones are safe and surgery is a good
2 thing to do. If a person said that, you know,
3 skeptically, I think that would disappoint
4 certain patients, but how it was said and when
5 it was said in response to what would either
6 determine whether the person is engaged with
7 the mental health professional or leaves the
8 mental health professional. You know, all
9 mental health professionals are not created
10 equal.

11 Q. So it sounds like you're saying it
12 could do harm to that patient?

13 MR. KNEPPER: Objection, form.

14 A. No, I'm not saying that. I'm
15 saying it could be disappointing to that
16 person. What that person did with the
17 disappointment may prove harmful just because
18 of that person or it may prove in fact
19 beneficial.

20 Q. Are you satisfied -- let's orient
21 this question around the patients you've seen
22 in the last 12 months. Are you satisfied that
23 those patients -- actually, sorry. Let me
24 start over. Are you satisfied that the
25 patients you have seen historically for whom

1 you provide letters of authorization for
2 hormones give sufficiently informed consent?

3 MR. KNEPPER: Objection, form.

4 A. From my point of view, I did what I
5 could to reach the standard of having the
6 person internalize and think about, digest,
7 dream about and come back and talk to me about
8 it. That's all I can do. I can't guarantee
9 that if I do what I do that it's going to
10 change your mind or help you steer your ship in
11 a slightly different angle --

12 Q. So --

13 A. -- so I would not write a letter of
14 recommendation if I didn't feel like I did my
15 part. And if the person indicated that they
16 couldn't pay attention to me, I wouldn't write
17 the letter.

18 MR. CHARLES: Understood.

19 Okay. John, finished.

20 MR. KNEPPER: You're finished?

21 MR. CHARLES: I mean, barring --

22 MR. KNEPPER: Barring --

23 MR. CHARLES: We can't tell the
24 future.

25 MR. KNEPPER: I wasn't ready for

1 history and current psychiatric diagnosis, it's
2 more complicated than just the internet.

3 But we need to understand who these
4 children are and how they're different from
5 their peers and what we could possibly do to
6 help them to have a better life. I know some
7 of the conversation today was, we'll help them
8 have a better life by giving them puberty
9 blocking hormones, but that doesn't address --
10 I think it has a risk of harming them further.
11 And it doesn't address the comorbid
12 developmental challenges that these children
13 face.

14 And I'm afraid -- and it's controversial,
15 because I don't have the answer. I'm afraid
16 there's a possibility we're making these
17 children have a worse outcome. And until you
18 can demonstrate to me in a very careful
19 controlled study that separates the autistic
20 from the non-autistic, you see? That separates
21 the kids who come from a family that's intact
22 from a family where there's a single parent.
23 Where you can separate the kids who were
24 sexually abused from the kids who were not
25 sexually abused. I'm not sure puberty blocking

Exhibit I

Stephen Levine
December 21, 2020

IN THE UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF FLORIDA
TALLAHASSEE DIVISION

CASE NO. 4:20-cv-00020-MW/MAF

JAMI CLAIRE, KATHRYN LANE and
AHMIR MURPHY,

Plaintiffs,

vs.

FLORIDA DEPARTMENT OF
MANAGEMENT SERVICES, et al,

Defendants.

ZOOMED DEPOSITION OF STEPHEN B. LEVINE, M.D.

Monday, December 21, 2020

9:30 a.m. - 2:51 p.m.

Via Zoom

Tallahassee, Florida 32308

STENOGRAPHICALLY REPORTED BY:

SANDRA L. NARGIZ
RPR, CM, CRR, CRC, FPR, CCR-GA

Job No. 166551

Stephen Levine
December 21, 2020

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Stephen Levine
December 21, 2020

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6

7

8 (STENOGRAPHER'S NOTE: Exhibits were received
9 premarked electronically; only Exhibits 1, 2, 3, 7,
10 10, 11 and 13 were referred to in deposition.)

10

11

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1 **right?**

2 A No, that is. I think -- we'll quibble
3 over the word only. If you use the word
4 predominantly, I would say they are predominantly
5 taking care of. They are a specialty clinic for the
6 transgender.

7 Q So predominantly treating transgender
8 people, but not 100 percent?

9 A That's my guess.

10 Q Okay. What sorts of treatments do you
11 provide for your patients with gender dysphoria?

12 A Psychiatric evaluation of the patient and
13 the family, the parents and the other siblings;
14 psychotherapy to further the process of
15 understanding this whole phenomenon; recommendations
16 for hormones and occasionally recommendations for --
17 depending on the biologic sex of the patient, for
18 genital or breast surgery.

19 Q How many patients have you recommended
20 hormone therapy for?

21 A You mean over 47 years?

22 Q Let's start with the 47 years, yeah.

23 A I don't know. Can I give you a gross
24 estimate?

25 Q Sure.

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1 to be directed to the surgeon.

2 Q Okay. If a surgeon told you I require a
3 letter for this facial feminization surgery, are
4 there circumstances under which you could see
5 yourself providing a letter, not of recommendation
6 but of authorization, for a person to receive this
7 surgery from the surgeon?

8 A I could see myself under certain
9 circumstances, if I understood the patient's motives
10 and had a lot of time to discover and discuss this,
11 the history and alternative approaches and wondering
12 about the psychology of wanting this, I could see
13 theoretically.

14 That's what I do, you know, as a
15 psychiatrist; I am trying to investigate the meaning
16 of the wish and the solution that the patient is
17 hoping for, the problem the patient is hoping this
18 would be a solution for.

19 And so I want to be able to consider this
20 and have a respectful, mutual, slow dialogue that is
21 slow, meaning multiple sessions, to consider the
22 nuances of this because, you know, all of us have a
23 self-concept of how handsome we are or pretty we
24 are, and most everyone wants to get a little more
25 handsome and a little more pretty and we are -- we

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1 Q Okay.

2 A I believe that if a surgeon is going to do
3 this, he ought to know what I think -- what I know
4 about the person's history and the person's
5 intellectual capacities and the prices they paid for
6 their gender dysphoria already.

7 For example, the loss of a family and no
8 relations to children, or the inability to have a
9 relationship, an intimate relationship with other
10 people. I believe the surgeon needs to have an
11 understanding of the person.

12 I don't have an understanding whatsoever
13 of the techniques of surgery. You see? I am just a
14 psychiatrist. And the psychiatrist -- and the
15 surgeon has very little understanding of how a
16 person got to be in his office. And I believe that
17 the letters of recommendation should capture the
18 humanness of this person and the desperation of this
19 person and the justification that the person uses
20 and the hopes they have for this surgery. But
21 that's Levine, you know.

22 Q I want to show you the WPATH Centers of
23 Care section that discusses letters. This is
24 Exhibit 7 which we are going to put on the screen.
25

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1 (Exhibit 7 was marked for identification.)

2 BY MR. TILLEY:

3 Q Let's go to page 27. It looks like the
4 document page 27, it's .pdf page 33, Bates stamp
5 PL 0450524.

6 You see, Dr. Levine --

7 MS. COLES: Can you read that, Dr. Levine?
8 It looks a little small on my computer.

9 THE WITNESS: I can read it. It says
10 referral for surgery.

11 MS. COLES: Okay. Just making sure.

12 BY MR. TILLEY:

13 Q At the bottom, I am going to start there
14 and then we'll go on to the following page. At the
15 bottom it says, The recommended content of the
16 referral letters for surgery is as follows: 1, the
17 client's general identifying characteristics -- now
18 we are continuing on to the next page -- number 2,
19 results of the client's psychosocial assessment,
20 including any diagnoses.

21 And then it goes on to 3, 4, 5, and 6.

22 Dr. Levine, can you just review those if
23 you can read it and then let me know if you agree
24 with those statements.

25 (Short pause.)

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1 A I don't disagree with the statements, but
2 each of those statements, of course, need to be
3 operationalized by the letter writer. For example,
4 the first one, identifying characteristics,
5 oftentimes identifying characteristics would be like
6 this is a 63-year-old Caucasian veterinarian. But
7 there are many other identifying characteristics
8 that might be included.

9 So you can interpret these things with
10 terse statements or elaborate statements. I favor
11 elaborate statements. For example, I would like to
12 say a divorced father of four, or a roller derby
13 official. I would like to identify him as much as a
14 person as possible. But in the history of medicine,
15 race, age, and nourishment passes for identifying
16 information.

17 So the results of the psychosocial
18 assessment, including any diagnosis. Psychosocial
19 assessment would be the processes in his life
20 history, including any current or past diagnoses,
21 you see. So substance abuse might be a very
22 important part of number 2.; and the duration. So
23 if I am writing a letter, if I am one of two people
24 who have been hired to write a letter for genital
25 surgery, and I might have had three visits with the

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1 not inquiring about your medical history and your
2 psychiatric history. But it may be psychologically
3 beneficial to you and an M.D. may recommend that you
4 do that. And that recommendation would be based on
5 his or her knowledge that you are likely to suffer
6 from seasonal affective disorder, and the treatment
7 is bright lights and sunshine. And sunshine would
8 be far superior because of its luminescence, the
9 number of lumens exposed, than bright lights.

10 BY MR. TILLEY:

11 Q Let's go back just briefly to WPATH. And
12 I know you mentioned you have a more conservative
13 approach. So let me ask you this.

14 Is it fair to say that if you personally
15 believed that you would authorize hormones or
16 surgery for someone with gender dysphoria, someone
17 following the WPATH Standards of Care would also
18 believe that?

19 A Yes.

20 Q Okay. Let's talk about insurance for a
21 little bit. If you recommended that -- if you
22 authorized some form of treatment for gender
23 dysphoria, whether it be hormones or some form of
24 surgery, would you expect that that treatment would
25 be covered by your patient's insurance?

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1 offering an opinion on transgender people accessing
2 sex-specific public places; is that right?

3 A No.

4 Q It's correct that that's not right?

5 A You mean like bathrooms, and so forth?

6 Q Right. You are not making an expert
7 opinion in this case concerning sex-specific spaces;
8 is that correct?

9 A That's right.

10 Q Okay. Let's go to page 13. You say that
11 plaintiffs assert that the WPATH Standards of Care
12 are widely accepted. Do you see that statement?

13 A Please tell me what paragraph it's in.

14 Q Under heading number 4.

15 A Yes. Okay.

16 Q Do you disagree that the WPATH Standards
17 of Care are widely accepted by the major medical and
18 mental health associations?

19 A No.

20 Q Okay. You just think that they are wrong;
21 is that correct?

22 A Yes, and widely accepted doesn't tell you
23 60 percent or 40 percent. It just says widely
24 accepted.

25 Q Okay. Is it -- how would -- how would you

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1 You see?

2 So I am saying, please, let me talk to you
3 about human beings here and how important having
4 ongoing lifelong relations with one's children are
5 and being a grandfather or grandmother, and being
6 connected to a family of origin. I am not talking
7 about categorical bans. I am talking about being
8 smart.

9 BY MR. TILLEY:

10 Q Are you aware that this case concerns an
11 insurance exclusion that is categorical at
12 preventing --

13 MS. COLES: Form.

14 BY MR. TILLEY:

15 Q -- hormones and surgery as a treatment for
16 gender dysphoria?

17 MS. COLES: Form.

18 A I am aware that your plaintiffs are suing
19 to get coverage for -- that is not provided by their
20 particular insurance. I am aware of that.

21 BY MR. TILLEY:

22 Q Do you think that exclusion is
23 appropriate?

24 MS. COLES: Form.

25 A I've already answered that question, I

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1 believe.

2 BY MR. TILLEY:

3 Q What is the answer?

4 A That it's a political decision that varies
5 from state to state, and it belongs to the process
6 of political science and the courts and not doctors.

7 Q And if you yourself were treating them and
8 determined that they understood the risks and you
9 thought the treatment would be psychologically
10 beneficial and provided letters of authorization to
11 them, you would want that treatment to be covered by
12 insurance; is that correct?

13 MS. COLES: Form.

14 A I am an agent of the patient, I want
15 what's best for the patient, and especially if the
16 patient couldn't otherwise afford it, I would wish
17 for my patient to have it, yes.

18 BY MR. TILLEY:

19 Q I know you said you are not about
20 categorical bans, but let me ask you about minors
21 again.

22 Would you support a categorical ban on
23 access to puberty blockers to treat gender
24 dysphoria?

25 MS. COLES: Form.