# Securing Cisgendered Futures: Intersex Management under the "Disorders of Sex Development" Treatment Model

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*In this critical, feminist account of the management of intersex conditions under 2006's controversial "Disorders of Sex Development" (DSD) treatment model, I argue that like the "Optimal Gender of Rearing" (OGR) treatment model it replaced, DSD aims at securing a cisgendered future for the intersex patient, referring to a normalized trajectory of development across the lifespan in which multiple sexed, gendered, and sexual characteristics remain in "coherent" alignment. I argue this by critically analyzing two ways that intersex management has changed between OGR and DSD: 1) regarding sex-assignment recommendations for three patient populations and, 2) with the prenatal treatment of pregnant individuals at risk of conceiving a fetus with congenital adrenal hyperplasia with the steroid hormone dexamethasone. I conclude that like OGR before it, DSD also unjustifiably presumes that typical genitalia are necessary for cisgendered development. However, unlike OGR, it appeals to the empirically inadequate, theoretically suspect, and biologically determinist model of gender development known as brain-organization theory. Given this, I conclude that the treatment of intersex people under DSD continues to be driven by problematically heterosexist and transphobic assumptions regarding the value and normalcy of cisgendered life, while practically and discursively constituting it as such.*

In this article, I provide a critical, feminist account of the medical management of intersex conditions under the controversial "Disorders of Sex Development" (DSD) treatment model introduced in 2006. I argue that just like the "Optimal Gender of Rearing" (OGR) treatment model that it replaced, DSD aims at securing a specifically *cisgendered future* for the intersex patient, and is thus, practically speaking, a suite of medical interventions aimed at the clinical production of *cisgendered lives*. By *cisgendered future* I mean something far more expansive than one in which individuals identify with the sex they were assigned at birth. Rather, I use this term to refer to a

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normalized trajectory of development across the lifespan in which multiple sexed, gendered, and sexual characteristics remain in dynamic but "coherent" alignment. These variables include primary sex characteristics (specifically, external genitalia), secondary sex characteristics (from the development of breasts, to sex-typical fat distribution, to hair-growth patterns), gendered characteristics including identity, behavior or role, patterns of cognition, nonsexual gendered desires (such as toy and occupation preference), as well as sexuality. I use the term *cisgendered* rather than *cisgender* specifically to: 1) differentiate my critical description from self-identification as cisgender, transgender, or nonbinary, and so on, and 2) to emphasize the discursive and material constitution of this trajectory of development as cisgendered via the treatment model, insofar as it presumes the normative and descriptive normalcy of cisgendered lives and materially normalizes individuals on the basis of that presumption. In this sense, contemporary intersex management both reifies the normalcy of cisgendered life and materially constitutes it as such.[1] Further, in the cases under consideration here, it does all of this without the patient's informed consent.

To make this argument, I critically analyze two ways in which intersex management has changed between OGR and DSD: 1) with regard to sex-assignment recommendations for three patient populations and 2) with the introduction of prenatal treatments for pregnant individuals identified as at risk of conceiving a 46 XX fetus with congenital adrenal hyperplasia with the steroid hormone dexamethasone (a practice often referred to as "fetal dex"). On the basis of this analysis, I derive a series of conclusions. First, although DSD inherits from OGR both its aim *and* the empirically unjustified assumption that typical genitals are necessary for "normal" cisgendered development, it is underwritten by a different model of gender development (Creighton et al. 2014). Specifically, unlike OGR, which was practically dominated by a social-constructionist model of gender development, DSD is practically dominated by a biologically determinist model known as brain-organization theory, which holds that brains are "organized" into feminine or masculine patterns by prenatal hormone exposure. Brain-organization theory has received a good deal of critical attention in the last decade or so, pointing to many ontological and empirical reasons for skepticism (Fine 2010; Jordan-Young 2010).

Thus, I conclude that just like OGR before it, DSD suffers from a complex, theoretically suspect, and empirically inadequate account of gender development as its foundation. As a result, clinicians are just as hamstrung under DSD with regard to providing "evidence-based recommendations" regarding not only *which* sex to assign, but also *how* to assign it in terms of which surgical procedures to use, and their timing (Houk and Lee 2010, 4506; Creighton et al. 2012; Lee et al. 2012; Creighton et al. 2014). Further, this means that the primary justifications for intersex management under DSD remains social rather than based on clinical evidence about human biological function. Clinical treatment of intersex patients thus continues to be underwritten by problematically heterosexist and transphobic assumptions regarding the value and normalcy of cisgendered life—without consideration of the ways in which this suite of therapies itself contributes to the reification of cisgendered life as normal and valuable (bolstering transphobia and heterosexism in the process). In this

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sense, the very same norms cited as demanding interventions like fetal dex and sex assignment are affected through their practical constraint on the range of sexed, gendered, and sexual futures available to those with intersex conditions.

## From "Optimal Gender of Rearing" to "Disorders of Sex Development"

In 2006, the American and European pediatric endocrine associations published a special article titled "Consensus Statement on Management of Intersex Disorders" (hereafter Consensus Statement) proposing the first official revision in both the treatment model and the system of nomenclature for these conditions since John Money and his colleagues Joan and John Hampson introduced their "Optimal Gender of Rearing" (OGR) treatment model in the 1950s (Lee et al. 2006). Underwriting OGR was Money's complex account of gender development, which held that "variables of sex" such as "chromosomal sex, gonadal sex, internal and external morphologic sex and hormonal sex (prenatal and pubertal)" could be independent of one another (Money 1995, 21).[2] For Money, the development of gender was a "multistage process that relied on multiple attributes of biological sex and social variables but that could not be said to derive from these exclusively" (Karkazis 2008, 53). However, for a large portion (95%) of patients studied by Money and his colleagues at Johns Hopkins in the early 1950s, gender identity corresponded to sex of rearing—or rather, gender socialization (Rubin 2012, 888). This led Money and the Hampsons to identify sex of rearing as being of particular importance to gender development—perhaps even more so than biological variables given the stability of gender once learned (Karkazis 2008, 52–54). Further, Money and his colleagues saw normalized genitalia as key to gender development—despite accumulating a good deal of contradictory evidence from the patients they were studying.[3] Genitals that did not match a child's assigned sex would impede the child's identification with their assigned sex and gender and, further, might cause parents to "waver in their commitment to raising the child in the assigned gender" (57–58). The prioritization of sex of rearing over other variables in sex-assignment—including those of biological sex—and the importance placed on normalized genitalia for both the child being socialized and those doing the gendered socialization meant that Money's complex account of gender development boiled down to a social-constructionist model organized around what intersex scholar Morgan Holmes refers to as Money's "genital determinism" (Holmes 2008, 69; Karkazis 2008, 52–54).

By the time DSD was introduced in 2006, the ethics, politics, and science underlying the OGR treatment model had been the subject of almost twenty years of intense, often scathing critique by both feminist academics and intersex activists (Kessler 1990; Fausto-Sterling 1993; Chase 1999; Fausto-Sterling 2000; Chase 2013).[4] Critics from these intimately entangled communities began raising concerns in the early 1990s, focusing in particular on the performance of medically unnecessary genital normalizing surgeries that were (and remain) generally accompanied by sterilizing gonadectomies, on infants and children unable to provide informed consent—often

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at the expense of later sexual function. They questioned the positioning of the medical treatment of intersex conditions as exceptional, legitimating practices that routinely violated bioethical principles of respect for autonomy, beneficence, nonmalfeasance, and justice. They pointed out that these surgeries were (and remain) necessarily experimental given that the kind of clinical trials that might provide quality evidence regarding the efficacy of the techniques involved and their ideal timing —trials that would involve randomly assigning medically unnecessary genital surgeries, performed on differing schedules to infants—are ethically prohibited. Furthermore, they called into question the presumed naturalness of physical sex dimorphism that constituted intersex conditions as pathologies requiring treatment, as well as the heterosexism underlying treatment recommendations and outcome analyses that valued "aggressiveness and sexual potency for boys and passiveness and reproductive/sexual-receptive potential for girls" and according to which homo or bisexuality and instability of gender identification constituted bad outcomes (Dreger and Herndon 2009, 204).

Both feminist academics and activists (particularly those associated with the Intersex Society of North America [ISNA]) often forwarded a type of natural-variation account regarding intersex bodies, based fundamentally on the notion that "although certain intersex-related conditions (such as salt-[wasting] associated with Congenital Adrenal Hyperplasia) can be life threatening and thus may require intervention, intersexuality itself is not pathological and thus does not require medical treatment" (Spurgas 2009, 99). It was because intersex conditions generally, and ambiguous genitalia in particular, threatened social norms about sex, gender, and sexuality that structure so much of daily existence that they were pathologized, they argued, and this pathologization justified their treatment with unsuccessful, experimental surgeries and secrecy, causing immense physical and psychological harm.

The Consensus Statement cites progress made in "diagnosis, surgical techniques, understanding psychosocial issues, and [in] recognizing and accepting the place of patient advocacy" as motivating the revision in treatment model and nomenclature it lays out (Lee et al. 2006, e488). It reclassifies intersex conditions under the umbrella category "disorders of sex development," and assigns a DSD-specific diagnosis (to be used in conjunction with one's condition-specific diagnosis) that references one's total chromosome number, specific "sex chromosome" makeup, and, in some cases, gonadal makeup (for example, an individual with Complete Androgen Insensitivity Syndrome (CAIS) would now also receive the diagnosis 46 XY testicular DSD). Beyond the shifts in sex-assignment recommendations I explore below, the treatment model outlined within the Consensus Statement is not that different substantively from OGR. Its surgery recommendations, however, are certainly the most conservative ever issued publicly by a group of clinicians, stating that surgery should be limited to "cases of severe virilization," that functional outcomes be emphasized over aesthetic, and cautioning that although "it is generally felt that surgery that is performed for cosmetic reasons in the first year of life relieves parental distress and improves attachment between the child and the parents; the systematic evidence for this belief is lacking" (Lee et al. 2006, e491).[5]

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Clinical adoption of the DSD treatment model among pediatric specialists in intersex management internationally was so rapid and so near total that one of the Consensus Statement co-authors characterized its swift usurping of OGR as "a quiet revolution in medicine" only four years after its introduction (Hughes 2010, 160). However, it is now clear that DSD has not brought about a reduction in the frequency with which genital normalizing surgeries are performed on infants unable to provide informed consent, despite the Consensus Statement's call for moderation. Clinical experts in the field, as well as researchers in the humanities drawing on interviews with clinical experts, have found that surgery continues to be positioned as a necessary part of optimal care under DSD, and is performed as frequently as it was before, and may even have increased (see Karkazis 2008; Creighton et al. 2014; Feder 2014; Davis 2015; Crary 2017; Kirkland 2017).[6]

Despite the Consensus Statement's qualifiers regarding the lack of evidence for surgery, it does nothing to dispute the notion that typical genitalia are in fact essential for normal gender development. It makes clear that genitalia cannot be too atypical, or "severely viriliz[ed]"—however that line is drawn—as surgery is still recommended in these cases, despite the acknowledged lack of evidence. Moreover, it does nothing to disrupt the pursuit of cisgendered life as the primary aim of this suite of interventions.

Thus the primary aim of intersex management and the presumption that typical genitalia are essential to its achievement have not changed between OGR and DSD (as attested to by the stubborn persistence of genital normalizing). However, the model of gender development concretized through and guiding the use of the suite of interventions encapsulated by the treatment model has. Instead of the social-constructionist model distilled out of Money's more complex account, clinicians now turn to the biologically determinist account known as brain-organization theory in order to predict gender under DSD. In the following two sections, I critically analyze two ways in which intersex management has changed between OGR and DSD—with regard to sex assignment for three patient populations and with the introduction of fetal dex—and show that both of these changes are justified via appeal to brain-organization theory. Moreover, in addition to providing further evidence for my account of cisgendered futures, I detail the effects of this shift in model in terms of its reprioritization of physical sex characteristics in predicting gender, and the larger reconstitution of biomedical definitions of normal sex and gender that it achieves.[7]

## Shifts in Sex Assignment Recommendations

There are clear indications within the clinical literature of shifts in recommendations regarding sex assignment under DSD for three particular patient populations.[8] The first group is those patients with 46 XY cloacal exstrophy, a condition associated with the protrusion of the abdominal organs through the abdominal wall such that infants can be born with their intestines and bladder exposed (Meyer-Bahlburg 2005). The second are those 46 XY patients with micropenis—defined as a penis smaller than

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2.5 standard deviations below the mean—that is specifically the result of nonhormonal factors (Hatipoğlu and Kurtoğlu 2013, 217).[9] Finally, the third group is those 46 XX patients with congenital adrenal hyperplasia (CAH), whose genitalia are considered to be "severely masculinized" (Houk and Lee 2010). CAH is the most common intersex condition (excluding hypospadias), and is associated with the decrease or complete loss of activity of an enzyme involved in steroidogenesis, the endocrine cascade or multi-step set of processes via which cholesterol is converted by enzymes into biologically active hormones such as cortisol, androgens, and estrogens (Roughgarden 2004; Miller and Auchus 2011). This enzyme deficiency leads to the accumulation of substrates that are then converted to androgens by active enzymes in the environment, leading to an increase in androgen production and the "masculinization" of XX individuals over the lifespan, beginning in utero (Miller and Auchus 2011).

Whereas OGR recommended female sex assignment for all three of these patient populations, clinicians increasingly advocate for assigning all of these groups male under DSD, specifically via appeals to brain-organization theory. The rationale for assigning the first two groups—46 XY patients with cloacal exstrophy and those with micropenis—female under OGR had to do with the limitations of phalloplasty techniques, the social-constructionist model of gender development underwriting OGR, as well as heteronormative assumptions regarding both masculinity and femininity. It often was—and remains—too difficult to build a penis capable of "voiding in a standing position and achieving straight erections and proper ejaculation that will permit suitable sexual intercourse" (Creighton et al. 2012, 603). It is comparatively much easier, however, to create an aesthetically typical vagina capable of penetration (if not sensation) (603). Thus, heteronormative assumptions regarding the capacities necessary to normal cisgendered life as either a man or a woman and technological limitations to actualizing those capacities—which, importantly, are rendered limitations only by those heteronormative assumptions—led clinicians to default toward female assignment for these patients. This makes sense when assumptions and technological capacities are read against the social-constructionist model of gender development that presumes the necessity of typical genitalia for normal cisgendered development, and prioritizes gender socialization over all other contributing variables. With regard to the third patient population—"highly masculinized" 46 XX patients with CAH—their XX chromosomes coupled with the relative ease of vaginoplasty meant that clinicians saw themselves as *repairing* these latter patients' bodies rather than changing them in some way; surgery and hormone therapy for these patients restored them to being the girls that they both should have been, and really were, all along (see Karkazis 2008, 106–08).

Under DSD, however, there has been a clear shift in clinical practice to assign 46 XY patients male. As 2016's "Global Disorder of Sex Development Update since 2006: Perceptions, Approach and Care" (Global Update hereafter) clearly indicates,

> The previously widespread routine assignment of 46, XY newborns with markedly hypomasculinized genitalia as females has given way to *more*

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> *detailed considerations of biological factors* [emphasis added]. . . . Physicians are now more likely to suggest male assignment of 46, XY newborns who presumably had *normal-male prenatal androgen levels* with *nonhormonal genital malformations* [emphasis added], such as cloacal exstrophy of the bladder. (Lee et al. 2016, 169)

It goes on to explicitly invoke brain-organization theory in warning clinicians amid this "trend to assign most 46, XY DSD patients as male" that "individualized caution must be taken with male assignment based on *evidence of androgen responsiveness and CNS androgen exposure during fetal life* [emphasis added]" (169). The implication here is that 46 XY patients with atypical genitalia should receive male assignment only if there is evidence of "typical" androgen exposure (and response) in utero (that is, their underlying etiology is not hormonal in nature). Clinicians are being cautioned not to let themselves get carried away amid this "trend," and assign as male any 46 XY infants who do *not* exhibit evidence of being responsive to androgens and having been exposed to it in utero.

Given that the technical limitations plaguing phalloplasty under OGR persist under DSD, one could interpret the move toward male assignment for 46 XY patients with cloacal exstrophy and micropenis as the expansion of those futures constituted as normally cisgendered under the new treatment model (as one reviewer suggested). Indeed, we might read this as an easing of unjustified assumptions regarding the necessity of typical genitalia to being a "normal" boy or man, or of heteronormative assumptions regarding what capacities genitalia must have to be valued, and/or considered typical. Although such an expansion might be an unintended side effect of this shift in clinical practice, it is not the primary intention. Indeed, heteronormative concerns persist under DSD; for example, Global Update advises that "for those with 46, XY and a micropenis, male assignment is preferable for most regardless of penis size except for those with partial [androgen insensitivity syndrome]" (Lee et al. 2016, 169). Nonetheless, it goes on to note that "the issue of penile length persists" in managing 46 XY patients with micropenis, as many patients will have "insufficient penile length to achieve penetrative intercourse" (169). Further, it emphasizes the importance of considerations regarding penile length and the capacity for penetrative intercourse in deciding how to treat these patients—ostensibly with regard to sex assignment—cautioning that "[i]t is clear that the same care plan cannot be applied to all individuals with a given diagnosis/syndrome and severity," in light of these issues (169). Beyond this, the fact that the general frequency with which genital normalization is performed has persisted (if not increased) under DSD indicates that the presumption that typical genitalia are essential to the development of normal cisgendered life continues to hold sway in general—it is only members of these populations who seem to avoid the knife.

The apparent relaxing of norms regarding the necessity of typical genitalia for these patients is the result of the discursive and practical ascension of brain-organization theory as the dominant model of gender development within DSD. Brain-organization theory presumes that the well-established sex-differentiating effects of

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hormones on genitalia are mirrored within the brain, giving rise to "masculinized" or "feminized" brains. As reflected in my account of cisgendered futures, the masculinizing and feminizing effects of hormones posited by the theory are quite expansive. Indeed, as Rebecca Jordan-Young writes in *Brain Storm: The Flaws in the Science of Sex Differences*, brain-organization theory presumes that hormone exposure generates neurological "permanent masculine or feminine patterns of desire, personality, temperament, and cognition" (Jordan-Young 2010, xi). Hormones are thus responsible for not only the development of one's identity as cisgender (or transgender, or nonbinary, and so on), but also "a very wide range of differences related to gender and sexuality—in humans these include everything from spatial relations, verbal ability, or math aptitude, to a tendency to display nurturing behaviors, to sexual orientation" (21–22).

As a result, although typical genitalia—and physicians' capacity to produce them given an infant's genital tissue and the surgical techniques available—are still presumed to be an important component to a cisgendered future, prenatal hormone exposure and receptivity come to take priority as *the most important* characteristics to consider in sex assignment under DSD. This is why the Global Update's recommendations make a distinction between those who have hormonal etiologies and with "nonhormonal genital malformations such as cloacal exstrophy of the bladder" and specifies that physicians are only more likely to suggest male assignment for 46 XY patients *if* they have a nonhormonal etiology that indicates "normal-male prenatal androgen levels" (Lee et al. 2016, 169). Cloacal exstrophy is an excellent example of a nonhormonal intersex condition and is included under the umbrella category of intersex conditions only because the condition frequently involves atypical development of the urogenitals (often in a "bifid" manner where they are split into two equal parts) (Meyer-Bahlburg 2005). The Consensus Statement equivocates on the issue of treating these patients, simply noting that those "reared female show variability in gender identity outcome, but >65% seem to live as female"—a statistic that does not imply practice would or should move away from (already normative) female assignment (Lee et al. 2016, e491). However, Valerie Arboleda, David Sandberg, and Eric Vilain point to a 2004 report of "self-initiated gender-change in a cohort of patients with 46 XY cloacal exstrophy" as initiating a shift in pediatric urologists' recommendations regarding sex assignment, citing a 2011 survey of American pediatric urologists in which 79% of respondents recommended male assignments for 46 XY cloacal exstrophy, "with 97% identifying *'brain imprinting' by prenatal androgens* [emphasis added] as an important factor in their decision" (Arboleda, Sandberg, and Vilain 2014, 611). This is also why the Global Update specifically excludes those with partial androgen insensitivity syndrome from its recommendation that all 46 XY patients with micropenis be assigned male, "regardless of penis size," and cautions that physicians assign such patients male based only on "evidence of androgen responsiveness and CNS androgen exposure during fetal life" (Lee et al. 2016, 169). Those with hormonal etiologies (like partial androgen insensitivity syndrome) cannot be presumed to have had typical androgen exposure or responsiveness during fetal life, raising questions about the masculinization of their brains, and thus clinicians' ability to

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successfully secure a masculinely cisgendered future for them in my more expansive sense.

In this way, a reconstitution of the range of gendered life normalized as cisgendered has been achieved, and formally codified through DSD. The dominance of brain-organization theory within the model and the wide range of physical and behavioral effects it attributes to hormones proliferates the number of characteristics that must cohere in order for one to "count" as cisgendered beyond a mere alignment between the sex one is assigned at birth and one's gender identity—if it ever really was merely that.[10] Assigning 46 XY patients with "nonhormonal genital malformations" male under DSD does mean potentially assigning them to trajectories of development that deviate from that normalized as cisgendered—insofar as they may not ever have aesthetically or functionally typical genitalia (and hence, could be read as an expansion of the range of life constituted as normally cisgendered, just for these populations). However, genitalia are only one variable of many that makes up gendered life under DSD—and the correlation presumed by brain-organization theory between multiple neurologically based characteristics of the gendered self (for example, identity, behavior, nonsexual desires, and sexuality) justifies prioritizing them over genitalia. Indeed, if a more global coherence among these variables is the aim, then it is better to have a typically cisgendered boy/man with slightly atypical genitalia than an atypically gendered girl/woman with aesthetically typical genitals. Who knows what surgical advances in phalloplasty might be made? Further, these patients' presumed capacity to produce and respond to typical levels of androgens ensures their future development of "coherent" secondary sex characteristics—development that could always be supplemented with hormone replacement therapy if needed. In this sense, although the range of life constituted as normally gendered might have expanded somewhat for those 46 XY patients with nonhormonal etiologies under DSD, it has formally narrowed in general under DSD, insofar as it normalizes a high level of coherence across a wide range of sex, gendered, and sexual variables. Additionally, the treatment model places greater trust in advances in knowledge-production, and the technical capacity to normalize the body, than in sociopolitical capacity to accept a range of sexed and gendered life—something noted by Ellen Feder (Feder 2014).

The dominance of brain-organization theory under DSD means that those 46 XY patients with atypical genitalia, but presumably typical prenatal androgen exposure/receptivity, now receive male rather than female assignment on the basis of their (presumably) masculinized brains. It also means that the third patient population—those 46 XX CAH patients with "severely masculinized" genitals—now receive male rather than female assignment, also on the basis of their (presumably) masculinized brains, as indicated by their physically masculinized genitalia. This is why, despite the Consensus Statement's replication of OGR's recommendation that "markedly masculinized 46 XX CAH infants be assigned female," there is a good deal of evidence to suggest that clinical practice leans toward male assignment (Lee et al. 2016, e491). In "Approach to Assigning Gender in 46 XX Congenital Adrenal Hyperplasia with Male External Genitalia: Replacing Dogmatism with Pragmatism," Consensus

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Statement co-authors Christopher Houk and Peter Lee note that "gender assignment in contemporary DSD management encourages consideration of multiple factors . . . recognizing that some degree of gender predetermination may have taken place in some types of patients," before going on to argue specifically for male assignment for these patients (Houk and Lee 2010, 4502). Though they acknowledge that outcome evidence for their proposal "is incomplete," they argue there are nonetheless "several lines of evidence which support it" (4503). In addition to citing studies showing that there appears "to be a high risk of gender dysphoria" in markedly masculinized patients assigned female "regardless of karyotype," they also note "additional, albeit indirect support" from a single study revealing gender transition (if not dysphoria) to be relatively rare among the few (33) markedly masculinized XX individuals assigned male who have been studied (4503–04). Finally, they identify the avoidance of genital surgery and "the irrevocable loss of sensitive genital tissue" as an additional benefit of male assignment in these cases, pointing out that it will "offer more options for the adult DSD patient who would logically have a better outcome with surgical reassignment from male to female than vice versa" (4504).

Equivocating evidence, avoidance of surgery (and maintaining options via this), as well as avoiding dysphoria seem like prima facie good reasons for male assignment in these cases—assuming that male assignment here does not involve surgical interventions of any kind (such as phalloplasty). These are good reasons for avoiding surgery for all intersex patients. However, Houk and Lee invoke brain-organization theory both implicitly and explicitly in their arguments for assigning severely masculinized 46 XX patients male. Their reference to a high risk of gender dysphoria for markedly masculinized patients assigned female "regardless of karyotype" (that is, "sex chromosomes"), for example, is a thinly veiled appeal to brain-organization theory and its assumption that the developmental effects of hormones on the genitals is mirrored in the brain. The prenatal androgen exposure responsible for the "marked masculin[ization]" of these patients is presumed to have similarly masculinized their brains, such that they are at a high risk of gender dysphoria—despite their XX chromosomes. Thus, the hormonal masculinization of the gendered self can be in a sense read through the severity of the masculinization of the genitalia—hence their specification that only "markedly masculinized" 46 XX CAH patients be assigned male. More explicitly, Houk and Lee identify "androgen-induced masculinization of the fetal brain" as appearing to be "important in the DSD patient exposed to male typical levels of testosterone during fetal life, including masculinized 46, XX DSD patients in whom such exposure produced male genitalia" (Houk and Lee 2010, 4506). Furthermore, they cite "high levels of fetal androgen exposure" as a key justification for the move to male assignment for 46 XY cloacal exstrophy patients under DSD, and argue that "similar consideration be given to the 46, XX CAH child with functional male genitalia whose existence implies *a high degree of masculinization of the brain*" (4506; emphasis added).

The Global Update's recommendations echo those of Houk and Lee (Lee et al. 2016, 169, citing Lee, Houk, and Husmann 2010, which also argues for male assignment for these patients). Further, both Houk and Lee 2010 and the Global Update

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underscore the multiplicity of gendered and sexual variables that must align for a future to be considered properly cisgendered (beyond a mere alignment between one's sexed body and one's gender identity). For example, one study finds that highly "masculinized" CAH women were more likely than the control group to be nonheterosexual, and to have "male-dominant occupations, a greater interest in rough sports and motor vehicles," before concluding that "46, XX newborns with marked genital masculinization are more likely to show marked masculinization of behavior" (Lee et al. 2016, 169).

Thus, in all three of these patient populations—46 XY patients with cloacal exstrophy, 46 XY patients with nonhormonal micropenis, and those "severely masculinized" 46 XX patients with CAH—changes in treatment are not motivated exclusively or even primarily by a desire to avoid surgery and its attendant complications, or a relaxing of heteronormative assumptions regarding genitalia, but to produce stably cisgendered subjects, and secure a future free of gender ambiguity, dysphoria, and/or transition. Prenatal administration of "fetal dex" attempts to do the same thing by intervening even earlier in the developmental process, by preventing the "masculinization" of 46 XX fetuses with CAH in utero.

## Fetal Dex

Prenatal administration of dexamethasome to those identified as at risk of conceiving a fetus with CAH (either through genetic screening or having already had a child with classic CAH) for the specific purpose of preventing the "masculinization" of 46 XX CAH fetuses in utero was first introduced in France in 1978 and in the USA in 1986 by endocrinologist Maria New (Meyer-Bahlburg et al. 2012, 103). Alice Dreger, Ellen Feder, and Anne Tamar-Mattis write that the practice is considered by many specialists as constituting *the* standard of care when treating women who might give birth to a child with CAH (Dreger, Feder, and Tamar-Mattis 2012, 278). A glucocorticoid steroid twenty-five times more potent than cortisol, fetal dex *will not cure CAH*. Rather, the goal is specifically to prevent the overproduction of androgens in only those CAH-affected fetuses with XX chromosomes, and thus prevent the masculinization of both their genitalia *and* their brains. That is, it aims at preventing the development of those "highly masculinized" 46 XX CAH infants that clinical practice is now shifting toward assigning male on the basis of that masculinization discussed above.

Because the human genitalia and reproductive structures differentiate very early on in fetal development (between seven and seventeen weeks), the masculinization process this treatment seeks to avoid begins prior to clinicians' ability to test whether the fetus is affected—that is, before we can test whether the fetus has both XX chromosomes *and* CAH (Warne, Grover, and Zajac 2005, 23; Yiee and Baskin 2010). As a result, fetal dex must be administered to a pregnant woman as soon as she learns she is pregnant (ideally five to six weeks' gestation) and is then discontinued if the fetus is found to have XY chromosomes or if it is found to have XX chromosomes

but tests negative for CAH, which accounts for 7/8 of all cases (Warne, Grover, and Zajac 2005, 23).[11] Although the atypically low cortisol production associated with CAH results in the production of atypically high levels of androgens in *all* affected fetuses, it is not considered a problem for those XY fetuses with CAH as such exposure does not carry the risk of genital or gender ambiguity (that is, "incoherent" masculinization of the body and mind characteristic of a noncisgendered future) as it does for those with XX chromosomes. Thus, the futures of such fetuses are conceived of as already securely cisgendered. The administration of fetal dex to prevent masculinization of 46 XX CAH infants has been lauded as "an excellent example of pharmacological therapy during pregnancy" and a "paradigm of prenatal diagnosis and treatment" (Rosner et al. 2006, 803; Nimkarn and New 2010, 5).

Despite this, in recent years concerns have been raised regarding the ethics of fetal dex. Because the administration of fetal dex to avoid masculinization in 46 XX fetuses with CAH is an off-label use of the steroid, it has never been clinically trialed. Thus, despite its standardization within intersex management, and its promotion as safe (particularly to those pregnant women who receive it), clinicians have no real idea of the effects of the prenatal administration of dexamethasone—which exposes the developing fetuses to 60–100 times the normal level of glucocorticoids—on either the fetus or the mother (Dreger, Feder, and Tamar-Mattis 2012, 281). Fetal dex can nonetheless be administered for this use due to its categorization as a category C drug by the FDA, meaning: "[a]nimal reproduction studies have shown an adverse effect on the fetus and there are no adequate and well-controlled studies in humans, but potential benefits"—in this case, securing a cisgendered future for an infant—"may warrant use of the drug in pregnant women despite potential risks" (FDA Pregnancy Categories—CHEMM n.d.). Indeed, animal studies, as well as retroactive studies of human children treated with fetal dex, have raised serious concerns regarding its use. These studies have reported correlations between prenatal exposure to fetal dex and increased risk for heart disease and diabetes (Kelly et al. 2012), as well as cognitive delays in both animals and humans (Dreger, Feder, and Tamar-Mattis 2012, 281; Hirvikoski et al. 2012; Meyer-Bahlburg et al. 2012). In fact, there have been so many problems identified with fetal dex that American endocrinologists Walter Miller and Selma Feldman Witchel published a "clinical opinion" piece in the May 2013 issue of the *American Journal of Obstetrics and Gynecology* urging obstetricians to abstain from treating their eligible pregnant patients with fetal dex, and to prevent their being treated with it by other clinicians involved in their patients' care. Miller and Witchel argue that the risks of this intervention far outweigh the benefits of "ameliorating genital virilization" for about 80–85% of affected fetuses, citing evidence from human and animal studies that show that:

> first-trimester dexamethasone decreases birthweight; affects renal, pancreatic beta cell, and brain development; increases anxiety; and predisposes to adult hypertension and hyperglycemia, in addition to retroactive human studies showing that first-trimester dexamethasone is associated with orofacial clefts, decreased birthweight, poorer verbal working memory, and

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> poorer self-perception of scholastic and social competence. (Miller and Witchel 2013, 355)

Furthermore, Swedish researchers performing the only controlled and prospective (rather than retrospective) study of fetal dex to have ever occurred announced in 2012 that they stopped recruiting new patients into the study in 2010 after "severe adverse events" were noted in the treatment group on follow-up, such as developmental delay, hydrocephalus, and severe mood fluctuations (Hirvikoski et al. 2012, 1882). The Swedish team concluded that the use of fetal dex is unethical not only in daily medical practice, *but even in the context of clinical trials*. Given that the risks of fetal dex are inadequately understood *and* the fact that 7/8 of those fetuses exposed "do not benefit from the treatment *per se*," they argue that it is "globally" unacceptable that fetuses at risk for CAH "are still treated prenatally with [dex] without follow-up" (1882). Further, they stress their position that if any clinicians are going to continue to administer fetal dex for this purpose, then "the minimal requirement should be" that it occurs *only* in the context of clinical trials "including long-term follow-up of all treated individuals," wherein parents are given "thorough information . . . about the potential risks and uncertainties, in addition to the benefits of this treatment" (1882). However, given the already evidenced risks, and the cessation of their own study, it is clear that even this fails to reach the standards of ethical research for this group.

One of the acknowledged goals of fetal dex is preventing the development of—and thus, avoiding the surgery to treat—anomalies of the urogenital sinus in CAH-affected XX fetuses. Most commonly, the urethra and vagina are joined, potentially increasing the risk of repeat infections. Dreger, Feder, and Tamar-Mattis argue, however, that this is a secondary goal, pointing to articles by New—the original and one of the primary proponents of fetal dex—to argue that the primary aim of treatment is to ensure that "CAH-affected female fetuses . . . develop in a more female-typical fashion than they otherwise might" (Dreger, Feder, and Tamar-Mattis 2012, 280). Fetal dex aims at securing a cisgendered future for the fetus—and at foreclosing potential noncisgendered futures—by preventing the development of both ambiguous genitalia *and* so-called "behavioral masculinization" in the form of tomboyishness and lesbianism or bisexuality (280). Further, Dreger, Feder, and Tamar-Mattis identify brain-organization theory as playing a key role in the rationale behind the use of fetal dex, identifying its aim as "engineer[ing] the CAH-affected female fetus's hormonal system to be typically female" in the face of concerns regarding its potential development along "a more masculine pathway *neurologically and genitally*" (281; emphasis added). Furthermore, they underscore the expansive, developmental nature of the cisgendered future clinicians aim to secure, quoting Saroj Nimkarn and Maria New's rationale that

> Without prenatal therapy, masculinization of external genitalia in females is potentially devastating. It carries the risk of wrong sex assignment at birth, difficult reconstructive surgery, and subsequent long-term effects on quality of life. *Gender-related behaviors, namely childhood play, peer*

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> *association, career and leisure time preferences in adolescence and adulthood, maternalism [interest in being a mother], aggression, and sexual orientation become masculinized in 46, XX girls and women with 21HOD deficiency* . . . . Genital sensitivity impairment and difficulties in sexual function in women who underwent genitoplasty early in life have likewise been reported. We anticipate that prenatal dexamethasone therapy will reduce *the well-documented behavioral masculinization* and difficulties related to reconstructive surgeries. (Nimkarn and New 2010, 9; emphases added)

Dreger, Feder, and Tamar-Mattis note that Nimkarn and New seem particularly concerned with XX CAH women failing to be heterosexual wives and mothers, having raised the issue in multiple publications and at multiple events (Dreger, Feder, and Tamar-Mattis 2012, 281).[12] Whether one is using fetal dex or surgical sex assignment, one is trying to produce girls who are girls across the lifespan, and then appropriately feminine, heterosexual women, in both body (sex) and mind (gender/sexuality). Codified within and reconstituted through the DSD treatment model is a narrowing of the range of life constituted as normally cisgendered (and an expansion of the abnormally noncisgendered). Sex assignment and fetal dex are part of a suite of interventions aimed at securing heternormatively cisgendered futures for intersex patients under DSD, and thus, simultaneously, the foreclosing of all other potential trajectories of gendered development.

## Reifying the Normalcy of Cisgendered Life

The production of bioscientific models that constitute while naturalizing binary models of sex and gender has been central to the history of intersex medicine (Dreger 1998; Fausto-Sterling 2000; Germon 2009; Mak 2012; Rubin 2012). Beyond controversially reclassifying intersex conditions as "disorders of sex development," DSD also achieves a reconstitution of the field of normally and abnormally gendered life, narrowing that trajectory of development defined as normally cisgendered for most patient populations (except for 46 XY patients with nonhormonal etiologies). This is the result of the discursive and practical ascension of brain-organization theory as the model of gender development underwriting DSD, via which clinicians attempt to predict gender in light of the infant's physical sex characteristics—notably now, the nature of their underlying etiology and evidence regarding prenatal androgen exposure/responsiveness—and the suite of therapeutic and surgical interventions available to them. Importantly, prediction is something of a mischaracterization here. Rather, clinicians attempt to predict which of those two trajectories of normalized as either masculinely cisgendered or femininely cisgendered can be most easily and coherently secured on the basis of the evidence and techniques available to them—a guess regarding a kind of developmental "best fit."

Further, the theoretical and practical dominance of brain-organization theory under DSD means that—just like OGR before it—it suffers from a complex,

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theoretically suspect, empirically inadequate account of gender development as its foundation. As Jordan-Young writes, the theory is underwritten by a two-part hypothesis: 1) the assumption "that male-typical sexual orientation or gender identity will correlate with other male-typical physical or psychological traits (and vice versa for female-typical sexual orientation and gender identity)," and 2) the assumption that those traits correlate because "both are influenced by hormones during the critical period of development" (Jordan-Young 2010, 38). At its core, then, is a presumption that the effects of hormones on genitalia are mirrored in the brain, despite multiple, deep disanalogies between the two structures. First, human brains and behaviors are nowhere near as dichotomous as genitalia. Unlike human brains, human genitalia can be reliably sorted into those that are male-typical and female-typical by observers who do not know the sex of the human they came from (49). On the other hand, the level of physical and/or structural sexual dimorphism exhibited by human brains remains the subject of live debate, as does the question of whether there are "distinctively gendered patterns of brain function" (Jordan-Young 2010, 49; Rippon et al. 2014). Beyond this, there is far more overlap among than divergence between genders "on the majority of social, cognitive, and personality variables," as well as evidence to suggest what differences are found are nowhere near as fixed as the theory posits, given the brain's unique "permanent plastic[ity]" (Rippon et al. 2014, 4). Second, human genitalia and human brains have very different developmental periods. Whereas the former differentiate very early on in fetal development (between seven and seventeen weeks' gestation), human brains are grossly underdeveloped at birth, and their lengthy developmental period is a markedly dynamic one, characterized by high levels of developmental plasticity in response to both biological and social inputs (Jordan-Young 2010; Yiee and Baskin 2010). The deeply interactionist nature of neurological—and subsequently, of gender—development is something feminist critics have argued is inadequately theorized or accounted for within brain organization research, undermining the research design (and thus, the empirical adequacy) of studies on it (Fine 2010; Jordan-Young 2010).

Most of those researching brain-organization theory think of themselves as "interactionists," insofar as they grant that the social plays a role in gender development, but they disagree about the relative importance of the social vs. the natural in terms of hormone exposure (Jordan-Young 2010, 8). Further, Jordan-Young argues that brain-organization theory is, at best, a "biosocial" rather than "interactionist model," because it fails "to account for how physical and social variables *work in tandem*" (8; original emphasis). That is, it presumes an additive, linear model of social and natural (hormonal) inputs leading to later behavioral outputs, without considering the way in which these inputs might interact or how that interaction might affect development. A properly interactionist model would "suggest that the *character* (not just the amount) of biological influence is affected by specific aspects of the environment, and vice versa" (8; original emphasis). And there is, indeed, evidence that these inputs do interact—for example, animal studies have shown that fatherhood can bring about a reduction in testosterone in males that varies with the level of parenting/physical contact (Gettler et al. 2011). Gina Rippon and her colleagues draw on

Anne Fausto-Sterling's use of "entanglement" to characterize the dynamic (Fausto-Sterling 2000), interactive, and socially dependent nature of the brain's development (Rippon et al. 2014), and the fact that "the social phenomenon of gender is literally incorporated, shaping the brain and endocrine system, becoming 'part of our cerebral biology'" (Kaiser et al. 2009, 57).

Fascinatingly, despite appealing to it, clinical experts in intersex management seem keenly aware of both the theoretical and empirical inadequacy of brain-organization theory, and its inability to ground practical recommendations. The clinical literature is full of warnings that, for example, "understanding of the effect of *in utero* androgens on human central nervous system (CNS) development remains inadequate to provide clear guidelines for gender assignment" (Houk and Lee 2010, 4506). As Global Update makes plain at some length:

> A biomarker of gender identity is not (yet) available. Although a number of studies have published differences in central nervous system (CNS) structures between transgender and cisgender adults, these studies use a variety of brain-imaging (or cadaver-sectioning) techniques; the findings are heterogeneous and lack replication; and where there are structural differences, they usually overlap to a considerable degree between transgender and cisgender samples, so that they are not yet useful for individual gender categorization. Moreover, our current knowledge of the structures and functions of the CNS underlying gender identity is insufficient to read MRIs for the presence of a specific gender identity. Even if at some point in the future such an interpretation of MRI findings should become possible for individuals at later stages of cognitive development, it is questionable that the brain of a newborn is developed enough for the prediction of gender identity years later, given the gradual development of critical sex-dimorphic aspects of the CNS. (Lee et al. 2016, 168)

As a result, clinicians are just as hamstrung under DSD as they were under OGR with regard to providing "evidence-based recommendations" on sex assignment for intersex infants (Creighton et al. 2012). Moreover, they are also keenly aware that this paucity of evidence extends to recommendations regarding how to assign sex in terms of which surgical procedures to use, and their timing (Creighton et al. 2012). In 2012, the *Journal of Pediatric Urology* published a special issue featuring articles generated from a multidisciplinary meeting of experts in intersex management in Annecy, France earlier that year. In an article dealing specifically with surgery, the authors are clear that "there is a serious lack of data to provide adequate guidance as to the best timing and surgical approach" (Creighton et al. 2012, 603). Indeed, they note that not only are there "no long-term studies to directly compare the merits of individual procedures," but also that "there are unlike[sic] to be so in the future" (608).

Given this, I conclude that DSD remains as socially (rather than empirically or biologically) motivated as OGR was before it. Indeed, like OGR, DSD carves out a discursive and material field of life as normally cisgendered only to then, like OGR,

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appeal to that definition of normal cisgendered life as requiring the interventions the treatment model encapsulates. DSD both determines and produces life as cisgendered, affecting the descriptive and normative normality of cisgendered life it assumes.

Assuming the normality of cisgendered life is not only ethically questionable, but it fails to reflect reality. As multiple recent academic studies and more popular articles have noted, "sex and gender diversity" is increasing, at least in the US context, where the DSD treatment model was developed (Flores et al. 2016; Davis 2018). The number of adults who identify as trans has doubled in the United States in the last ten years (from 0.3% to 0.6%), and those numbers are even higher among adolescents.[13] Further, as technological advances increase—including our knowledge about the "molecular genetics of gonadal development and neurological sex differences"—the number of those who count as having bodies that are neither typically male nor female will increase (Rosario 2009; Clune-Taylor 2016). Indeed, there appears to be a disconnect between those futures and lives imagined within and actualized through the DSD treatment model, and those projected by current trends. Such speculations aside, it remains unclear that one's gendered future is something that others should have authority to govern, no matter how well-intentioned their interventions may be. Those interventions are not only necessarily experimental, irreversible, and often sterilizing ones that put patients (and potentially those who gestate them, in the case of fetal dex) at a plethora of unknown risks, but are also performed without the person's informed consent.

## Notes

Earlier versions of this article were presented as part of the Feminist Research Speaker Series in the Department of Women's and Gender Studies at the University of Alberta in Edmonton, October 2015; as part of the Works-in-Progress Series in the Program in Gender and Sexuality Studies at Princeton University, Princeton in October 2016; and at the Humboldt-Princeton Symposium: Gender, Sexuality, Queer, and Trans Studies Write Back at Humboldt University, Berlin in June 2017. I wish to thank the organizers of these events and fellow participants for their generative comments. Special thanks to Cressida J. Heyes, J. R. Latham, Judith Butler, Regina Kunzel, Gayle Salamon, and *Hypatia*'s anonymous referees for their incisive criticisms and support. Finally, I thank Princeton University for funding this research through my position as a Postdoctoral Research Associate in the Program in Gender and Sexuality Studies, and wish to recognize my Research Assistant at Princeton in particular, Jean Bellamy, for her excellent work.

1. A full account of my concept of "cisgendered life" is beyond the scope of this text, and of the specific arguments I make within it. However, for the purposes of this article, I shall note that my understanding of cisgendered life is indebted to Foucault's arguments regarding the historical emergence of the concept of *bios* or "life, itself" in the work of naturalist Georges Cuvier (Foucault 1970; Mader 2011). Foucault argues that this new transcendental concept of life, and its attendant notion of biological function, signals a rupture between the classical episteme of natural history as the science of natural beings and the modern episteme of biology as a science of living forms, and the conditions of possibility for both biology and biopower. In this sense, although "cisgendered lives" are

individual, embodied instantiations of "cisgendered life," the latter refers to a norm greater than the sum of those parts.

2. Money has often been reductively portrayed as a strict social-constructionist—and maligned for this view. In fact, however, he was—at least initially—an interactionist in a manner revolutionary for his day (Diamond 1965; Zucker 1996; for an in-depth review of challenges to Money's position as a social constructionist, see Karkazis 2008, 63–80).

3. For more on the contradiction between Money's conclusions and his data, see Feder 2014, chapter 1. For further critical analysis of Money's views in general see Downing, Morland, and Sullivan 2015.

4. For more on the DSD treatment model as a reaction to academic and activist challenges to the management of intersex conditions under OGR, and an attempt to reassert medical authority over intersex bodies in their aftermath, see Davis 2015.

5. As Milton Diamond and Jameson Garland point out, there have been no studies "support[ing] the belief that gender variant children require early genital surgery for societally favored gender development" (Diamond and Garland 2014, 3).

6. As Katrina Karkazis notes, "most surgical articles take for granted that surgery will be performed (and hence simply describe how to do it)," and this is echoed in a trend noted in the clinical literature to focus on surgical outcomes (Karkazis 2008, 134). Further, there are some indications that the frequency with which surgeries are performed has perhaps increased under DSD. For example, Sarah Creighton and her colleagues note an increase in operations on the clitoris performed in the United Kingdom on those under fourteen since 2006 according to data from the National Health Service (NHS) (though the authors do caution that "collection of the most basic surgical data is poor and very variable") (Creighton et al. 2014, 38).

7. Although one might be tempted to read DSD's appeal to brain-organization theory as a break from Money's social-constructionist account of gender development enshrined in OGR, or from the larger history of the management of and research on intersex patients, this would be a mistake. As Rebecca Jordan-Young details, both Money and intersex management history played a central role in—as I would frame it—the historical emergence of brain-organization theory as a *discipline* (that is, as a field of knowledge/power), following its initial formulation in 1959. For an account of this history, see Jordan-Young 2010, chapter 2.

8. Clinical practice regarding intersex conditions is notoriously heterogeneous for a variety of reasons, from the universally acknowledged paucity of evidence supporting interventions performed, to the absence of a centralized database tracking procedures, to the subjective nature of clinical assessment itself (indeed—clinicians may vary widely in their opinions regarding how large a clitoris needs to be to require surgical normalization, for example). Beyond this, there was historical variation in the ways in which OGR was taken up both within and outside of the United States (see, for example, Mak 2012 as a counter to the somewhat unified narrative offered by Dreger 1998; Germon 2009; and Reis 2009). As a result, I have limited myself here to changes in practice for which there is clear evidence in the clinical literature, citing that evidence where appropriate. I thank an anonymous reviewer for this helpful point/corrective.

9. Micropenis is associated with a variety of conditions/underlying etiologies that can be grouped/divided in a variety of ways (for example, Nihal Hatipoğlu and Selim Kurtoğlu

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divide them into the categories "insufficient testosterone secretion," "testosterone activation defects" (such as partial androgen insensitivity syndrome), "developmental abnormalities" (of which cloacal exstrophy is an example), "idiopathic," and "in conjunction with other congenital malformations" (Hatipoğlu and Kurtoğlu 2013, 220).

10. Indeed, even those of us who are comfortable in our identity as cisgendered are well aware of the potential social and material risks associated with challenging gendered norms. For example, despite my comfort with my identity as a cisgender woman, I may nonetheless suffer consequences for exhibiting traits gendered as masculine, such as being ambitious, intelligent, confident, or assertive. Hence my specification re: clinical codification.

11. CAH being an autosomal recessive condition, only one-quarter of fetuses will be positive for CAH and only half of those will be XX fetuses, leaving the total likelihood of having an XX CAH positive fetus at one-eighth.

12. For example, Dreger, Feder, and Tamar-Mattis report that at a 2001 meeting of parents of CAH-affected children organized by the CARES Foundation, New showed a photo of an XX infant with CAH and ambiguous genitalia and stated, "The challenge here is . . . to see what could be done to restore this baby to the normal female appearance which would be compatible with her parents presenting her as a girl, with her eventually becoming somebody's wife, and having normal sexual development, and becoming a mother. And she has all the machinery for motherhood, and therefore nothing should stop that, if we can repair her surgically and help her psychologically to continue to grow and develop as a girl" (New, quoted in Dreger, Feder, and Tamar-Mattis 2012, 282).

13. Joanna Almeida and her colleagues found that 1.2% of high-school students in Boston identified as trans (Almeida et al. 2009), and Nicole Rider and her colleagues found that 2.7% of youth in Minnesota identified as trans or gender nonconforming (Rider et al. 2018; see also Flores et al. 2016).

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