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Biological Bodies: Defining Sex in the Modern Era

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Biology

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Often in science we find our biggest questions in our unexamined rules and assumptions. This is especially true with definitions and categories. Questions such as 'what is a species?' or 'what is biological sex?' seem straightforward at first. After all, we have theories and definitions that can be simplified and understood in layman terms. But when examined with a critical eye we realize how little of the foundational assumptions on which we base our theories, research, legislation, and even our mundane activities, are fully understood, even if they have been accepted as facts.

Biological sex is one of these unquestioned assumptions which has risen to become a fact. Biological sex is defined as "either of the two major forms of individuals that occur in many species and that are distinguished respectively as female or male especially on the basis of their reproductive organs and structures" and, alternately, as "the sum of the structural, functional, and behavioral characteristics of organisms that are involved in reproduction marked by the union of gametes and that distinguish males and females" (Merriam-Webster, 2017). Or, in evolutionary terms, as "producing large, immobile gametes or small, mobile gametes" (Fausto-Sterling, 2000b). Underlying these definitions is the assumption that evolution created male and female, built by unique internal chemistry to show dimorphic bodies. With these bodies they procreated, joining egg and sperm and fusing chromosomes to create offspring mirroring one of them in sex, chosen by the Y chromosome and shown by genital appearance upon birth (Callahan, 2009; Lorber, 1993).' Or so it is said.

Biological sex is largely accepted at face value, unquestioned by scientists or the public. It is so ingrained into our culture that it has been used to do everything from cornering the children's toy market to justifying the exclusion of women in STEM fields (Butler, 1990; Fausto-Sterling, 2000a). After all, who would question what we can clearly see? Boys have penises, testes, seminiferous tubules, testosterone, and rampant body hair while girls have vaginas, clitorises, ovaries, uteruses, estrogen, fallopian tubes, and breasts– plain and simple (Fausto-Sterling, 2000a; Hird, 2004; Stoller, 1968). But, what about the times when it isn't plain and simple? What about the times when babies are born with

empty scrota, or genitals too little to be a penis but too big to be a clitoris, or when a child grows up with a vagina but discovers upon puberty that they have XY chromosomes, or when a father of four wakes up from surgery to be told that he has a uterus (Addison & Taylor-Alexander, 2016; Ainsworth, 2015; Hird, 2004)? What becomes of these people under the definition of biological sex? More importantly, what becomes of biological sex when its parameters fail to be as unquestionable as we claim them to be?

Biological sex encompasses many elements which are sometimes referred to as all-or-nothing traits of sex, and at other times are referred to additively, with the sum of differently valued parts adding up to being one sex or the other (Ainsworth, 2015; Costello, 2016; Freidman, 2013). These elements are chromosomal sex, genital sex, gonadal sex, hormonal sex, secondary sex, brain sex, somatic cellular sex, and sex identity, with three of these eight elements observable in some way by the naked eye (genital, gonadal, and secondary) and four other elements that are claimed or proven to be measurable. Sex identity, however, is only discernable by asking the individual in question (Arboleda, Sandberg, & Vilain, 2014; Fausto-Sterling, 2012; Gayharpoon, 2017; Jordan-Young, 2010). These elements are the tools with which we measure and prescribe biological sex, but they are also the tools with which we police it.

Individuals whose elements of sex do not match the all-or-nothing definition of biological sex or do not additively place them in the sexes we call 'male' or 'female' are reprimanded or corrected for their transgressions of 'the rules of biological sex' (Costello, 2016). In addition, many corollaries of biological sex, such as sex complementarianism, the idea that males/men and females/women are fitted 'compliments' to one another with opposing roles, bodies, strengths and weaknesses, separation of the sexes, sex hormones, and the effect of hormones on brain/personality have widespread implications for medicine, law, and society at large (Butler, 1990; Hird, 2004; Roberts, 2000; Stoller, 1968). With such widespread effects on our social, scientific, and medical lives understanding and questioning biological

sex becomes not just an important scientific inquiry but a dire social issue (Lorber, 1993; Unger, 1979). We can no longer afford to take biological sex at face value; it is time we deconstructed and analyzed it from an interdisciplinary angle, leaning heavily on the scientific and largely biological framework on which it stands but without neglecting psychology, neuroscience, sociology, endocrinology, genetics, medicine, and gender & women studies.

A scientific fact is "any observation that has been repeatedly confirmed and accepted as true; any scientific observation that has not been refuted" (Dictionary.com, 2017). With this in mind, I will look at studies, observations, definitions, ideas, and common assumptions which surround biological sex and at the social, legal, and medical implications which follow to see how our definition of sex measures up to both scientific law and theory. I will examine in depth at each of the proposed elements of biological sex, questioning the validity of each by carefully analyzing studies and think-pieces on the topic. Data from all kingdoms will be included, as biological sex is a term which is used not just to describe humans or even vertebrates, but everything from plants and fungi to mussels, fish, and insects. Special consideration will be given to cases such as the ones that were raised in my first rhetorical postulate, those of the intersex individuals whose diverse experiences and lives have been the subject to extensive scientific scrutiny. Lastly, I will delve into sociology and gender and women's studies which are affected by our understanding of biological sex and which study its rhetoric and history.

Chromosomal sex

Humans have 23 pairs of chromosomes within the nucleus of each cells of these, one pair is of particular interest, the 'sex chromosomes'. These chromosomes come in two varieties, an X and Y. The Y chromosome, half the size of the X chromosome, primarily contains genes which contribute to what we would call 'male' development. It is found in most every designated male at birth (DMAB) individual,

most commonly in the form of an XY pair, and aids and facilitates the growth of many elements of sex (Fausto-Sterling, 2012; Lercher, Urrutia, & Hurst, 2003). Unlike the Y, the X chromosome is found in every human, XX being most common for designated female at birth (DFAB) individuals, and at least one copy is necessary for a person to live as it is home to a variety of genes. Despite this, it is considered the female chromosome in order to be seen as a compliment to the Y chromosome (Lercher et al., 2003).

The X chromosome, while being called the 'female chromosome', is not connected to 'female traits' in the same way that the Y is connected to 'male traits'. Many genes for these traits are found on the other 22 pairs, called autosomes, instead of being localized on the X, which seems to play a greater role in tissue-specific genes (Lercher et al., 2003) . In this way, the role of the X is more analogous to the role of an autosome than it is to the Y. And XX individuals have the same amount of expressed X-linked genes as XY individuals, due to the formation of a Barr body in each cells. The Barr body is a randomly deactivated X chromosome, condensed to an unusable form to regulate the amount of X-linked genes active in the cell at a time. In addition to this, the X is known to contain several genes relating to sperm creation and development as well as having an arguably greater effect on XY individuals due to the increased occurrence of X-linked disease (Lercher et al., 2003; Wang, McCarrey, Yang, & Page, 2001)

In contrast, the Y chromosome has been found to house genes which specifically contribute to 'male' development (van Anders, 2013). Specifically the *SRY* gene, located on the Y chromosome, is responsible for testicular differentiation and development (Ainsworth, 2015; van Anders, 2013). Individuals who lack the *SRY* gene will not undergo testicular differentiation (van Anders, 2013). The Y is deemed the 'sex determining factor' in humans, drosophila, and many mammals, because the Y of heterochromasomal individuals is the only chromosome passed down to offspring that varies in most cases, as XX individuals can only pass on an X. This can be seen in the haploid karyotypes of sperm, with each typically carrying either an X or a Y (Callahan, 2009; Richardson, 2002). This, however, is far from the only way to determine the chromosomal sex of an organism. In the platypus, each individual has ten

'sex chromosomes', with 'females' having all X and 'males' having five of each in an alternating pattern (Bagemhil, 1999; Fausto-Sterling, 2012). In birds, which have an Z-W chromosomal system, females are the heterochromosomal sex (Bagemhil, 1999; Fausto-Sterling, 2012). In reptiles various species-specific temperature ranges determine the sex of the offspring (Bagemhil, 1999; Fausto-Sterling, 2012). Microorganisms lack what we would call 'sex chromosomes' all together (Bagemhil, 1999; Fausto-Sterling, 2012).

In humans the XY/XX system can vary in many ways. Intersex individuals with chromosomal variations can be born with X0, XXY, XXX, XYY, XXXX, XXXXX, XXXY or, XY/XXY or XX/XXX mosaic chromosomes (O'Neil, 2013). These variations lead to changes in the phenotype of other elements. Changes in the gonadal sex element can occur in X0 karyotypes, where the ovaries are underdeveloped and sterile, in XXX, XXXX, XXXXX, and XX/XXX karyotypes, where early-onset ovarian menopause can occur, and in XXY, XXXY, and XY/XXY karyotypes, where testes and prostate glands are smaller than average (Callahan, 2009; O'Neil, 2013) Intersex variations are not only chromosomal: they can be hormonal, genital, or otherwise. Nor are intersex variations particularly rare: the number of babies born that vary from common conception of 'male' and 'female' has been calculated to be around one in every 100 births and the number that are given 'normalizing surgery' calculated to be close to one in every 1,000 births (Ainsworth, 2015; Hird, 2004; ISNA, 2008a). These statistics are comparable to the frequency of redhead births, which occur in 1-2% of the world population (ISNA, 2008a)

But the X and Y are only two out of 46 chromosomes in the human body and their role extends beyond that of reproduction and sexual elements. The necessary role of the X chromosome contains many autosome-like genes that play a role in non-sexual functions. Conversely, many genes located on autosomes can play vital roles in elements of biological sex and reproduction, such as the *FOXL2* gene located on chromosome 3 which facilitates in the division, upkeep and regulation of granulosa cells.(US Deartment of Health & Human Services, 2017).

Gonadal Sex

All fetal humans start their gonadal development with a urogenital ridge that forms a pre-gonadal structure which is identical in all fetuses (Fausto-Sterling, 2012). Then, if the fetus has a *SRY* gene on the Y and a *SOX9* gene on the chromosome 17, the *SRY* will produce a chemical product that binds to *SOX9*. If this occurs in the correct sequence, the pre-gonadal structure will develop as testes. If not the fetus will follow what we call a 'female' pattern of development, but without ovaries (Arboleda et al., 2014; Fausto-Sterling, 2012). Our knowledge of which genes activate and facilitate ovarian development is limited thus far to *FOXL2, WNT4, β-catenin* signaling and *R-spondin1* (Matson et al., 2011; Tomaselli et al., 2011). *FOXL2*, located on chromosome 3, and *WNT4*, on chromosome 1, are needed for ovarian development and for the other elements of 'female' development. Mice which lack either of these two genes develop as 'males'. But without *R-spondin1*, located on chromosome 1, XX mice and humans will develop as 'males' in both gonadal and genital elements despite lacking the *SRY* gene. (Fausto-Sterling, 2012). In this way gonadal determination is better seen as a game of 'rock-paper-scissors' than as a two-pronged, direct pathway.

I have already mentioned that changes in chromosomes can lead to changes in gonadal phenotype, but this is not the only means by which variation can arise. Changes in hormonal phenotype or environment during development have, in the past, been theorized to affect gonadal phenotype. This is unsubstantiated, however, because testicular differentiation occurs in utero, independent of androgen exposure, under the influence of the *SRY* gene (Arboleda et al., 2014; Fausto-Sterling, 2012; Uhlenhaut et al., 2009). Variation in differentiation and development can occur in intersex individuals resulting in underdeveloped gonads, indeterminate gonads, such as ovotestes, or gonads which share various degrees of 'mixed' phenotypic expression or levels of function (Arboleda et al., 2014; ISNA, 2008b). In addition, changes in phenotype and function can occur outside of the context of intersex

individuals. Polycystic ovary syndrome, testicular and ovarian cancer, menopause, sterility, gonadal dysgenesis, abnormal or incomplete gonadal growth, and surgery are just a sampling of possible sources of non-intersex variation (Costello, 2016; Fausto-Sterling, 2012; Hird, 2004; ISNA, 2008b).

Many forms of intersexuality are associated with gonadal sex and reproductive variations. XY Gonadal Dysgenesis leads to the development of gonadal streaks, minimally present gonadal tissue which does not produce steroid hormones and is often removed later in life (University of California, 2015). XXY karyotypes most often result in sterility while X0 karyotypes lose ovarian function before puberty and cause their oocytes to become inviable (Ainsworth, 2015; University of California, 2015). Gonadal development can yield ovotestes, which contain a mixture of testicular and ovarian tissues. Or it can yield one testicle and one ovary, this is known as 'true gonadal intersexuality' (TGI) (Arboleda et al., 2014; ISNA, 2008b). In these cases, only one tissue type can typically function (Arboleda et al., 2014; University of California, 2015).

While these variations all complicate an already intricate system, the story does not end at so-called 'sex differentiation'. Rodent model studies have shown that *FOXL2*, in addition to playing a role in fetal gonadal development, has an important role in ovarian upkeep (Uhlenhaut et al., 2009). In a 2009 experiment by Uhlenhaut et al., deletion of *FOXL2* in adult XX mouse ovarian follicles lead to upregulation of testis-specific genes, including target gene *SOX9*. The upregulation began transdifferentiation from adult ovarian tissues (theca and granulosa cells) to what appeared to be testicular tissues (sertoli-like and leydig-like cell lineages) (Uhlenhaut et al., 2009). These lineages underwent spermatogenesis and produced testosterone (Uhlenhaut et al., 2009). This entire process occurred independently of a Y chromosome or *SRY* gene. Global deletion of *FOXL2* in mice, via null or *LACZ* replacement, leads to fetal ovarian dysgenesis instead of gonadal sex reversal (Uhlenhaut et al., 2009). Thus, in adult XX bodies, *FOXL2* plays a role in prevention of transdifferentiation from ovarian tissues to testicular tissues and upregulating ovarian tissues and function, while in fetal bodies it helps in

ovarian differentiation (US Deartment of Health & Human Services, 2017). In human XX adults with a heterozygous *FOXL2* mutation, infertility is often observed, but a human homozygous loss-of-function mutation of *FOXL2* has yet to be observed (Uhlenhaut et al., 2009).

A similar process has been observed in XY mice, where loss of the *DMRT1* transcription factor 3 in adult sertoli cells using Dhh-cre or Sf1-cre activated *FOXL2* and allowed transdifferentiation of sertoli cells into granulosa cells (Matson et al., 2011). And, while XX *FOXL2* null mice will be born with ovarian dysgenesis, XY *DMRT1* null mice are born with testes, although these testes which will later undergo transdifferentiation (Matson et al., 2011; Uhlenhaut et al., 2009). So, while *FOXL2* plays two roles in mice, those of fetal gonadal differentiation and adult upkeep via repressing testes-promoting genes and activating ovary-promoting genes, *DMRT1* seems to only have a role in upkeep, repressing multiple ovary-promoting genes and activating testes-promoting genes. However, in human fetal XY individuals that lack *DMRT1,* transdifferentiation is observed prior to birth (Matson et al., 2011). The mechanistic difference between mice and humans that causes this variation is currently unknown, but may be due to the longer gestation of humans or due to lacking hypothetical genes which would be redundant with *DMRT1* in mice (Matson et al., 2011). Nonetheless, as *SRY/SOX9* and *FOXL2, WNT4, β-catenin* signaling, and *R-spondin1* can be imagined as playing 'rock paper scissors' in early human gestation, *DMRT1* and *FOXL2* can be imagined to be doing the same throughout mouse adulthood. This metaphor can also apply for *DMRT1* and *FOXL2* function later in human gestation, and possibly later in adult life in the case of *FOXL2.* In this way, the permanence of gestational gonadal determination and our more recent concept of its differentiation and upkeep is more complicated than previously thought.

Hormonal Sex

Sex hormones. The phrase conjures up images of high school biology, of a textbook drawing showing a hypothetical human body split down the middle and arrows pointing to where the body's

parts are ‘feminized’ and ‘masculinized’ and the tiny architects that are to blame: estrogen and testostrone, the quintessential hormonal building blocks of human sex difference. But this phrase and this vision of estrogen and testosterone is an outdated oversimplification of scientific reality that amounts to misinformation. In a study of high school science text books, the term “sex hormones” was present in all texts sampled, as was the impression of each sex having its own, singular hormone; estrogen for females and testosterone for males (Nehm & Young, 2008). The texts also all failed to discuss other functions of these hormones outside of sex-and-reproductive function. The use of texts containing the phrase “sex hormone” has been associated with misconceptions about the functions and locations of these hormones. (Nehm & Young, 2008).

In reality these hormones, more correctly known as “steroid hormones”, play many roles in all bodies, including in the developing fetus. There are two types of steroid hormones, corticosteroids and gonadal steroids. These break down further into groups based on the receptor types that they bind to: glucocorticoids, mineralocorticoids, which are corticosteroids produced in the adrenal cortex, and androgens, estrogens, and progestogens, which are gonadal steroids, produced in the gonads or placental tissue (Edocrine Society, 2017; Tulane University, 2014). In the fetus, these steroid hormones most often take one of two paths. One option is for Anti-Mullerian hormone to degenerate the paramesonephric ducts that would otherwise become the uterine ducts, uterus, cervix, and upper vagina (Fausto-Sterling, 2012). This is typically followed by testosterone spurring reconstruction of the mesonephric ducts to the vans deferens, epididymis, and seminal vesicles. Otherwise, maternal and fetal estrogen encourage the paramesonephric ducts to develop while the mesonephric ducts degenerate (Fausto-Sterling, 2012).

Androgens are defined primarily by their ability to bind to androgen receptors; they are the first anabolic steroids and are the compounds from which all estrogens are made (van Anders, 2013). While testosterone is the most well-known androgen, dihydrotestosterone (DHT) and androstenedione are of

equal importance to the body. All bodies have varying amounts of androgens, which spike during puberty. In individuals with testes, Leydig cells are the main producers of androgens, but they are also produced by ovaries, adrenal glands and fat cells, with these three being the sole suppliers of androgen production in individuals without testes (Tulane University, 2014). In addition to the formation of the ducts, vesicles and other structures which carry sperm and the degradation of other ducts, androgens, in concord with Anti-Mullerian hormone, also support sperm production and testicular movement to the scrotum (van Anders, 2013). Upon reaching puberty, large amounts of androgens can lead to increased muscle development, decreased adipocyte storage with most fat localized on the waistline, voice deepening, hair growth, and other effects (Tulane University, 2014; van Anders, 2013). In pregnant individuals androgens have a potential role of preventing premature uterine contractions (Tulane University, 2014).

Estrogens, in contrast, are defined by their ability to bind to estrogen receptors. In all bodies they are found in lower concentrations than androgens, though they perform many important functions (Edocrine Society, 2017; Roberts, 2000). Estrogens can be both organic and synthetic. The four naturally occurring forms of estrogen are estradiol, estriol, estrone, and estetrol, the latter of which is only produced during pregnancy. Structural effects of estrogens include the formation of mammary tissue, increased and localized adipocyte storage in the hips and thighs, uterine and endometrial growth, maintenance of blood vessels and skin, and increased bone growth (Edocrine Society, 2017). In addition, estrogen plays roles in the synthesis of binding proteins, aids in blood coagulation and platelet adhesion, increases fluid and salt retention, helps prostate gland development, increases pheomelanin, aids spermatogenesis, and supports alveoli (Edocrine Society, 2017; Schulster, Bernie, & Ramasamy, 2016). Lastly, estrogens are vital to menstruation and pregnancy, in conjunction with progestogens (Edocrine Society, 2017). While the effects of estrogens are widespread, and many are vital to all bodies, focus is placed so heavily on their role in menstruation, lactation, and pregnancy that other effects of estrogen

go unnoticed and under researched, much like non-reproductive and non-'sex-linked' effects of testosterone (Roberts, 2000).

When it comes to behavior, it has been thought that hormones directly affect certain behaviors, such as aggression, sexual behavior, and nurturing (Jordan-Young, 2010; van Anders, 2013). Rodent studies where XY rats were exposed to estrogens in utero showed less sexual behavior, mounting, and behaved 'femininely', fighting with other rats less than the control group (Jordan-Young, 2010). Similar studies in XX rats that were exposed to high levels of androgens in utero found that they showed less 'female sexual behavior', known as lordosis, or arching, and showed more 'masculine' behaviors such as aggression (Arboleda et al., 2014; Fausto-Sterling, 2000a; Jordan-Young, 2010; Roberts, 2000). These types of results are often extrapolated, claiming that 'testosterone = masculine behavior' and 'estrogen = feminine behavior' for all mammals. These claims are unfounded and ignore several factors. First, mouse neurobiology and human neurobiology are very different, making extrapolation of mouse behavior data to human behavior shaky at best (Fausto-Sterling, 2012; Jordan-Young, 2010). Second, in this study, and many other studies like it, the factor of environment is either ignored or reduced (Jordan-Young, 2010; Roberts, 2000). This is not as glaringly important with rodents as it is with humans, but a 2012 study XY rats showed that sexual experience changes the AR-positive receptor cells present in the brain, and another study cross fostering rats and lemmings found that the rats would mate with males and females of both their own species and the species they were raised with (Bateson, 1983; Swaney, Dubose, Curley, & Champagne, 2013). Therefore, the environment's effects on rodent behavior are great enough that we cannot simply ignore them. Third, it conflates mating behavior with human sexual behavior, which is much more instinct-driven in rodents and is not known to involve the same conscious thought, reasoning, and abstract communication skills that play a role in human sexual behavior (Jordan-Young, 2010). Fourth, it treats mating position as a sex-linked trait while animals of many species have been observed in the wild taking sexual positons counter to what is expected (Bagemhil,

1999; Fausto-Sterling, 2012; Jordan-Young, 2010). And, as with mating behavior vs human sexual behavior, we cannot ignore that humans can reason and choose their positioning based on things other than hormones (Fausto-Sterling, 2012; Roberts, 2000). Hormone studies with XX individuals who were exposed to high androgens in utero have found only weak, often conflicting, correlations between androgen exposure and sexual behavior with no conclusive evidence (Arboleda et al., 2014; Jordan-Young, 2010). There has been no conclusive evidence that hormones create sexually dimorphic behavior. Most studies working to that end have found data that conflicts with other studies or only demonstrates a small variation in mean with large amounts of overlap (Fausto-Sterling, 2012; Jordan-Young, 2010).

Hormone levels vary from person to person, each existing on what is called a range of reaction (Roberts, 2000). Range of reaction assumes that the phenotype of an organism depends both on genotype and the environment. Each organism is born with a genotype which allows them to produce a certain range of amounts of each hormone (Jordan-Young, 2010; Roberts, 2000). Maybe person A can produce between low and medium amounts of testosterone and person B can produce between medium and high amounts. Person B's ability to produce much more testosterone than person A does not mean that they always will be producing to their full capacity; environment also plays a role. For example, XY individuals that are regularly exposed to infants often have lower testosterone than those who are not, and pregnant XX individuals have higher testosterone than XX individuals who are not pregnant (Fausto-Sterling, 2012; Roberts, 2000; van Anders, 2013). So, if person A has environmentally heightened testosterone production and person B has environmentally lowered testosterone production then, despite their different ranges of possibilities, they could both be producing the same amount of testosterone. So it is possible for both XY and XX bodies to have steroid hormonal ranges that span from very high to very low levels of production and for any type of bodies to overlap in their current level of production even if their ranges are not the same. This is of specific statistical interest

because most if not all claims of 'XX having less testosterone' or 'XY having less estrogen' are based on the average production of a sample group of people whose hormones are in flux, whose ranges of production are unknown, and is based on a difference which, while it may be statistically significant, fails to tell the whole story (Jordan-Young, 2010).

The body is in constant interaction with its external and internal environment. We are constantly being shaped by our environment through the chemicals we intake, the stimuli we experience, and the changes our bodies. We are in contact with our external environments for our entire lives, taking it in to our bodies and reacting to stimuli starting when we are in utero. Chemicals, ranging from vitamins and medications to pollutants and food, are always passing through our bodies. Some chemicals, such as medically introduced hormone supplements or birth control, have a direct and clear effect on our hormonal balance while others, such as the foods we eat and the plastics we handle, have a less obvious effect.

Many plastics, fertilizers, pesticides, and other industrial chemicals have been found to contain xenoestrogens and other types of endocrine-disrupting chemicals (EDCs) (Diamanti-Kandarakis et al., 2009; Fucic et al., 2012). EDCs are defined by the U.S. Environmental Protection Agency (EPA) as "an exogenous agent that interferes with synthesis, secretion, transport, metabolism, binding action, or elimination of natural blood-borne hormones that are present in the body and are responsible for homeostasis, reproduction, and developmental process". EDCs can affect the amount of steroid, and other, hormones present in the body (Diamanti-Kandarakis et al., 2009). Xenoestrogens have been correlated to an increased risk of oestrogen related cancers while other EDCs have been linked to effects on male and female reproduction, breast development, neuroendocrinology, thyroid, metabolism and obesity, and cardiovascular endocrinology (Diamanti-Kandarakis et al., 2009; Fucic et al., 2012). In addition, phytoestrogens, which are found in plants such as soy, have been found to have both

estrogen-linked benefits, such as a lowered risk of osteoporosis, and risks related to its status as an EDC which depend on the age and health of the individual (Jefferson & Wendy, 2010).

Other factors that can affect hormonal balance include changes within the body itself. As organisms age, their hormone levels fluctuate, with spikes of estrogen that help regulate pubertal growth in all bodies (Delemarre-van de Waal, van Coeverden, & Rotteveel, 2001). In most XX bodies estradiol and androstenedione are related to height velocity during growth spurts and in most XY bodies levels of testosterone and estradiol are related to growth spurt height velocity (Delemarre-van de Waal et al., 2001). Throughout the menstrual cycle estrogen and testosterone are in a cyclic flux, starting day one with extremely low levels of both and climbing until week three of the cycle when ovulation occurs and both decrease sharply as progesterone takes over until the cycle starts over (Lichterman, 2009). During pregnancy, estrogen plays a central role in preventing miscarriage and supports fetal hormones and development (University of Maryland at Baltimore, 1997). Then, during menopause, estrogen, progesterone, and testosterone levels decrease to average around 10% of what was normal pre-menopause (Lichterman, 2009). For most XY individuals, testosterone levels decrease at around 1% a year starting around age 30, while estrogen levels rise starting in middle age as more testosterone is converted to estrogen (Severson, Barclay, & Kim, 2015).

Lastly, environmental influences such as stress levels, parental status, and social interactions can influence steroid hormones. In XX young adults, self-perceived psychological stress predicts of relative estradiol levels, with high stress correlated to lower estradiol than non-stressed XX young adults on the same menstrual day (Roney & Simmons, 2015). By contrast, XY mice exposed to acute sensory stressors express higher testosterone levels than unstressed peers (Armario & Castellanos, 1984). And, while parenthood may be stressful to some, it has been shown that XY individuals who have children experience a dip in testosterone that is not seen in childless peers, with a higher relative amount of time spent caring for the children being correlated with a greater testosterone decrease (Gettler, McDade,

Feranil, & Kuzawa, 2011). Lastly, social interactions, such as being around someone who is crying, can affect steroid hormone levels, at least for XY individuals who are around an XX individual who is crying (Gelstein et al., 2011). The tears of XX individuals lower testosterone levels of XY individuals who smell them, showing just one way that our interactions effect our hormonal balance (Gelstein et al., 2011).

In these ways, our external and internal context effects steroid hormonal balance, which in turn affects health, phenotype, and bodily function. It is a complex and cyclical pattern that reaches far beyond the oversimplified mantra that 'men are full of testosterone and women are full of estrogen' and changes not only throughout one's lifespan but from minute to minute.

Genital Sex

As gonadal sex, both the gonads and their associated ducts, is being formed, external genital sex is also being formed. During the indifferent stage, XX and XY fetuses have an identical phallus that can develop into a penis if there are influential levels of androgens or into a clitoris if there are influential levels of estrogens (Fausto-Sterling, 2012). Simultaneously, the labioscrotal swellings and urogenital folds either fuse under the influence of androgens to form the scrotum and penile shaft or remain unfused with influential estrogens and become the outer and inner vaginal lips (Fausto-Sterling, 2012). But, the precise roles of these hormones on genital sex formation and their effective dosages and mechanisms are largely unknown at this point, with further research needed in these areas.

This basic roadmap does leave room for variation. For example, in XY individuals whose testes do not form, or form incompletely, there is not sufficient testosterone to undergo penile and scrotal formation, resulting in vaginal, labial, and clitoral development or indeterminate genitalia (AIS Support Group UK, 1997). In individuals with XY chromosomes and Complete or Partial Androgen Insensitivity Syndrome (CAIS or PAIS), androgen receptor cells in the body do not bind to testosterone, making the testosterone in the body unusable (Arboleda et al., 2014; University of California, 2015). These

individuals are born with XY chromosomes, testes, and genitalia that match or closely match that of a vagina, labia, and clitoris, or with indeterminate genitalia, and are often assigned female at birth (Arboleda et al., 2014; Callahan, 2009; Fausto-Sterling, 2012; University of California, 2015). Other times hormone synthesis, and not testicular formation or receptor function, is responsible. Four enzymes responsible for testosterone synthesis: 17-ketosteroid reductase, 17-alpha-hydroxylase, 3-beta-hydroxysteroid dehydrogenase, and 17-beta-hydroxysteroid dehydrogenase, and one enzyme , 5-alpha reductase, responsible for converting testosterone to a usable form, have been observed to malfunction or be absent (AIS Support Group UK, 1997). If any one of these enzymes does not function or is absent an XY fetus will develop a vagina, labia, and clitoris alongside their internal testes, and if there is a partial malfunction then intermediate genitalia will form (AIS Support Group UK, 1997). Other times we only have are descriptors rather than a diagnosis, such as 'aphallia', the lack of a penis but fully formed testes, scrotum, and ducts, or 'micropenis', a phallus below 2.5 standard deviations of what is of mean phallus length, and 'hypospadias', where the urethral opening is along the shaft of the phallus or at the base of the penis itself (ISNA, 2008b).

In addition, 17-ketosteroid reductase, 17-alpha-hydroxylase, 3-beta-hydroxysteroid dehydrogenase, and 17-beta-hydroxysteroid dehydrogenase are also needed for development of internal reproductive structures such as the uterus, cervix, and upper vagina (AIS Support Group UK, 1997). Other genital variations of XX individuals include those which are highlighted by complete or partial vaginal absence while still having fully or partially formed ovaries, uteri, and ducts, such as Mayer Rokitansky Kuster Hauser (MRKH) Syndrome and Vaginal Atresia (AIS Support Group UK, 1997). Other times chemically androgen-effected external genitalia form. Progestin Induced Virilization (PIV) is a variety of XX phenotypes produced by the effects of introduced progestin in utero, as in the 1950-60's it was incorrectly believed to prevent miscarriage (Arboleda et al., 2014; Hird, 2004; ISNA, 2008b). Progestin is metabolized into androgens by the body, and while all individuals with PIV have ovaries and

a uterus or uterine tract, the produced androgens cause phenotypes ranging from only an enlarged clitoris to a lack of vagina, complete phallus, and fused labia (ISNA, 2008b). Another type of androgen-affected XX external genitalia is those caused by Congenital Adrenal Hyperplasia (CAH), the most common form of genital variation in XX individuals (ISNA, 2008b). CAH is a malfunction in the production of cortisone by the adrenal glands, instead producing an androgen precursor which is metabolized into androgens and causes a similar range of phenotypes as PIV (Arboleda et al., 2014; ISNA, 2008b). Because CAH is metabolic, androgens are produced throughout life and individuals with CAH can show characteristics such as dense body hair, a receding hairline, deepened voice, and enhanced muscular formation after puberty (ISNA, 2008b). But, just as in some XY individuals, at times all we have are have is a descriptor, in this case 'clitoromegaly', an enlarged or elongated clitoris, which can occur in conjunction with a diagnosis or with an unknown cause (ISNA, 2008b).

But what is the dividing line between a large clitoris and a small penis? In the United States the dividing line is called a Genital Grid, or Phall-o-meter, colloquially; a small ruler used to measure the phallus and determine whether the doctor will tell the waiting parents "it's a boy" or "it's a girl" in times of uncertainty (Fausto-Sterling, 2000a; Hird, 2004). By the Genital Grid's definition a clitoris is any phallus between the lengths of 0.2 and 0.7 centimeters and a penis is any phallus between the lengths of 2.5 and 4.5 centimeters (Hird, 2004). Any phallus between 0.75 and 2.25 centimeters is considered 'ambiguous' and doctors will either use what other internal or external elements of sex they can observe to choose what to sex the baby as, or, more likely, simply prescribe the infant genital surgery to sculpt a medically acceptable clitoris, as taking away material is easier done then adding, and tell the parents that they will raise the child as a girl (Fausto-Sterling, 2000a; Hird, 2004). Not only this, but if other elements of sex 'disagree' with an otherwise 'acceptable' length of phallus, for example an XX infant with ovaries, a vagina, and a 3 cm phallus, the phallus will be surgically removed (Callahan, 2009; Costello, 2016; Fausto-Sterling, 2000a).

I would like to take a moment for a quick disclaimer. In this paper, when I speak of medical practices and cultural responses to certain phenotypes, I am only able to speak in terms of Western culture and practice. Both response and medical procedure will vary from culture to culture and practice to practice, more so than I am able to accurately portray in one paper. For that reason I remind my readers that all of my perspectives on cultural response and medical practice is strictly in Western society, mostly the United States of America.

With that in mind, I would like to talk about these genital surgeries once more. These surgeries, performed within the first few months of life and often needing one or more subsequent reconstructions later in life, are done without the consent of the child, and often without the fully informed consent of the parents (Costello, 2016; Hird, 2004; ISNA, 2008b). Moreover, functionality of the sensory nerves within the phallus is not taken into consideration when performing surgery, as the 'acceptability' of the cosmetic appearance is seen as having paramount importance (Callahan, 2009; Costello, 2016; Hird, 2004). This means that many, if most, individuals who have surgery performed on their 'abnormal', but fully functional, genitals will lose sensation, orgasmic function, and/or the ability to derive physical pleasure from genital manipulation (Costello, 2016; Hird, 2004). This is mirrored in those DFAB individuals who partially or completely lack a vagina. Vaginoplasty, while not as commonly done on infants as phallic surgery, is seen as necessary for those infants which have functional ovaries and uteri so that menstruation may occur (Creighton, 2001). In individuals who do not need a vagina for menstruation, the choice to create or expand a vagina is merely for the sake of heterosexual penetration, without any means for sensation or pleasure during intercourse or masturbation (Costello, 2016; Creighton, 2001; Hird, 2004). In addition, while dilators are meant to be used first before surgery is considered and it is strongly advised to wait until adolescence so that some type of informed consent can be given, often these surgeries are performed on young children (Creighton, 2001). Due to these and other reasons, there is increasing evidence of patient dissatisfaction with their surgical outcome and

a call from both intersex individuals and some medical practitioners to reevaluate their policies on cosmetic genital surgery in the case of phenotypic variation (Costello, 2016; Creighton, 2001).

In these ways and more, bodies can exhibit a large array of natural genital phenotypes, which are sometimes further altered by physicians. The external genitals, maybe more so than any other element of sex, exist on a spectrum of phenotypes which can range from 0 centimeters to 3 centimeters with the urethral opening anywhere from the tip of the phallus to the base and with any amount of, or lack of, fusing of the developed labioscrotal folds. In this we see natural variation on a sliding scale, not two true forms and many 'mistakes'.

Secondary Sex

When puberty occurs both androgens and estrogens spike and the development of many secondary sex characteristics (SSCs) begins. For some, high levels of androgens can begin to allow production of facial hair and increased muscle development, for others estrogens can start the growth of mammary tissue and begin the menstrual cycle. But in all bodies, puberty is followed by some increased amount of thick body hair and body odor. These changes differ from body to body. Some teens who are expected to develop breasts will barely develop any defined tissues while others which are expected to grow facial hair never do. This can be said for every SSC, even menstruation. While menstruation is a part of the reproductive function of individuals with uteri and ovaries, and therefore is not an SSC in the strictest sense, its timing, cultural implications, and how it is viewed give it enough of the hallmarks of a SSC that I feel it is fitting to talk about it in this section. And like all other SSCs, it is variable, alterable, and not always present in every individual culturally or biologically expected to have it.

The variability of SSCs is easily observable by taking any random sample of adults from the population. A sample of XX individuals will have different breast sizes, from nonexistent to DDD,

different hip widths of a similar extremity in range, menstrual cycles with various days of flow, from 3 to 7 in most cases, and various schedules, ranging from a 27 to 35 day cycle under most cases. The same amount of variation can be seen in random samples of XY individuals, with shoulder width following a similar range as XX hip width, and larynx size, and therefore voice depth, ranging from very small and high to very large and low. The alterability of these characteristics is also easy to observe. Beards and body hair can be shaved, waxed, trimmed, and tweezed. Muscles can be built or let to atrophy. Steroids and supplements can be taken to alter musculature. Birth control can alter or regulate the menstrual cycle. Cosmetic surgery can remove, reduce, or enlarge breasts. Plastic surgery can alter face shape and structure. There are almost limitless ways that SSCs can be changed, either temporarily or permanently, to alter the sexed expression of an individual's body. And, through alteration or naturally occurring variation, all of these SSCs are optional. Body and facial hair, certain fat deposits, defined musculature, large larynx, wide hips, breasts, all of these can be eliminated either by virtue of never developing them or through deterioration or permanent medical alteration. Even the menstrual cycle, by sterility, removal of the ovaries, or tube tying, can be eliminated.

Due to their intense variability, none of these traits are considered to be, strictly speaking, necessary when deciding if someone is 'male' or 'female'. One can be 'female' without breasts or wide hips, or 'male' without an enlarged larynx or wide shoulders, both in the medical and social senses of the terms. Likewise, in some instances, one can be considered a member of one sex despite showing select traits from another. This goes beyond the famous 'bearded ladies' and extends to things such as gynecomastia (male breast development), and atypical muscular development or fat deposition.

Because of this, SSCs, being a complex and often fluid part of bodily composition and expression, are often of little to no importance in medical determination of 'biological sex' (Addison & Taylor-Alexander, 2016; Hird, 2004). Their role in the social function of sex, how it is falsely conflated with gender and thus is used to perform one's 'correct' bodily expression of both, is far more important

(Fausto-Sterling, 2000b; Morris, 2011; Unger, 1979). This is seen most clearly when talking about trans individuals. When someone who is trans comes out invasive questions about their anatomy soon follow. When they will get top surgery, implants, an Adams apple shave, voice training, hormone therapy, laser hair removal? When will they ensure that they have the SSCs that society says that they should have? To diverge from this prescription, to say that one will be keeping their chest flat or big, their voice deep or high, or their body hair shaven or unshaven, is looked upon harshly. When society says that a trans person must change their body in ways that they may or may not want to in order to 'truly be' their selves- that is society's policing of sex via SSCs.

Brain Sex

The study of brain difference was pioneered in 1959 when an experiment on mating behavior in guinea pigs proposed that the prenatal hormonal environment affected the brain's structure to produce sexual behaviors (Jordan-Young, 2010). It would later be proposed that the brain was 'organized' in utero so that it was 'primed' to have a particular response to steroid hormones. This proposition is now called the neurohormonal theory (Jordan-Young, 2010; Roberts, 2000). This theory gave rise to a branch of research that looked to find evidence of brain organization, the supposed structural differences which are thought to be created by hormones in utero and produce sexed behaviors. Neurohormonal theory is driven by three assumptions: hormones will be sex specific, functions of hormones will be limited to, or at least primarily contributing to, sex and reproduction, and hormones will be antagonistic towards each other (Jordan-Young, 2010). As I already explained in the Hormonal Sex section, these three assumptions are unsupported by evidence and are countered by studies and observations dating back as far as the 1920's (Edocrine Society, 2017; Jordan-Young, 2010; Roberts, 2000; Tulane University, 2014). Yet scientists use neurohormonal theory to frame and produce research to this day.

Before continuing this section I want to consider the history and politics that are so closely married to the topic of brain sex. When we, as scientists, speak on a topic we often consider it in a vacuum and ignore it's historical and political context. For neurohormonal theory and brain organization research this context is sex, the intercourse and sexuality definition thereof, and heteropatriarchy, the cultural normalization and value of heterosexuality and men over homosexuality and women which gives heterosexual men more cultural power than other groups (Butler, 1990; Fausto-Sterling, 2000a; Glumshoe, 2017; Jordan-Young, 2010). Studies in the 1990's and early 2000's compared the brains of heterosexual men and women to homosexual men and women, under the pretense that homosexual behavior was a 'cross sex' behavior. They believed the brains of homosexual men would resemble those of heterosexual women and vice versa because, by being attracted to men, they were showing a 'female' trait (Adkins & Leonard, 1996; Arboleda et al., 2014; Butler, 1990; Jordan-Young, 2010). The standard for what is 'female' behavior and 'male' behavior was, and always has been, rooted in heteropatriarchal views. But when brain organization research came into the limelight, the conversation shifted from "what girls and boys should do" to "what boys and girls do naturally" (Jordan-Young, 2010). Thus, when giving out teaching methods, encouragement, roles, or the subtle influence of parental guidance, the methods never changed- just the reasoning. Science nicely lined up to what was already established, giving a place for STEM exclusion, the glass ceiling, and other forms of misogyny to root. At the same time it allowed transphobia and homophobia a new platform, queer people were no longer just sinners, now they were also sick and mutated. They were people who could be 'cured' or prevented from being born, with the help of the right hormones. Because these conversations still occur today, we must keep in mind both the foundation that this branch of study is built upon and the high stakes of the impact of its results.

Despite the large claims made on the basis of neurohormonal theory, there is little firm evidence to back them up. Current data is of extremely limited use due to the way that human studies

are performed. Neurological experiments using animal models are insufficient for making large claims , more so than other fields of research, due to the differences between model organisms and humans in brain structure and experience (Fucic et al., 2012; Jordan-Young, 2010). This is why studies with human subjects are vital. However, due to ethical reasons, a true experiment with the necessary controls and manipulations to see if A causes B is impossible in human subjects. What is left is a network of quasi-experiments, weak correlational studies trying to instead see the effect of A on B. These quasi-experiments rely on large amounts of interconnected data all fitting together and pointing to the same conclusion (Jordan-Young, 2010). The fit of these studies, as well as the care and precaution put into their set-up, is what allows them to be of value, not the additive nature of having many replicates (Jordan-Young, 2010). A good example of this method is the network of quasi-experiments that link early human steroid hormone exposure to genital development (Jordan-Young, 2010). These experiments show dose-dependency, directional results, and results of many different experiments that agree with those that were performed before them (Jordan-Young, 2010). This type of fit is not seen in the quasi-experiments testing neurohormonal theory.

Before looking for a link between human steroid exposure and genital development, scientists performed animal studies which allowed for true manipulation and controls. Mammalian genital structure is largely conserved, as is genital function, unlike behaviors and areas of neurogenesis which are often species-specific (Bagemhil, 1999; Jordan-Young, 2010). This means that using animal studies of supposed hormonal effects on behavior and brain organization to draw possible human implications is far more difficult, complex, and ambiguous than it is for genital structure (Joel et al., 2015; Jordan-Young, 2010). With this complexity comes the need for excess caution before making any claims, especially any that may have societal effects.

This need for care extends to the ways we measure our variables. Jordan-Young calls measurements “vehicles through which assumptions travel in studies without being tested” and this is

because the ways that we measure our variables determines what we are actually studying (Jordan-Young, 2010). The methods and measurements that scientists use can lead to mismeasure, where internal bias leads to misreading of objective measurements. In the past, mismeasure has revealed scientist bias in ways such as seeing the brains of African people or women as being smaller than those of Europeans or men, even when observers were looking at the same brains (Jordan-Young, 2010). Measurements can also lead to misattribution, where our measurement tool has no connection to what we are trying to measure (Jordan-Young, 2010). When a researcher sets up a measurement tool, they are creating a working definition of the target trait. If one were to use the proportion of 'boy's toys' vs 'girl's toys' that a child chose to play with as the measure of 'masculinity' or 'femininity', then not only is one saying that toy choice is a trait that can be sexed, but also that some toys are inherently more appealing to one group of children but not another and that the preference of these toys is innate and linked to sex (Fausto-Sterling, 2000b, 2012; Jordan-Young, 2010). That is a lot of assumptions packed into one measurement. These assumptions are built on social biases, gendered thinking, and societal values that change from culture to culture (Fausto-Sterling, 2012). They also ignore the social factors which may influence toy choice, removing the ability to discern any biological origin (Fausto-Sterling, 2012). This makes setting up a quasi-experiment to find a small amount of possible link between steroid hormones and differences in brain and behavior increasingly difficult.

Most studies use behavior to assign brain sex, because no known brain structure exists that is sexually dimorphic (Joel et al., 2015). Categorical placements, like sexing a trait, require dimorphism and internal consistency (Joel et al., 2015; Jordan-Young, 2010). But no dimorphic physical difference has been found that allows a researcher to look at a brain, in the flesh or via MRI or CT scan, and determine if that brain belonged to a male or female (Joel et al., 2015; Jordan-Young, 2010). Even touchstones, such as one sex having more or less grey matter, are more population averages than true dimorphic traits (Joel et al., 2015). This is due to a lack of internal consistency. It is fully possible for any person to

have any range of traits that can be found in the human brain to the point that it is impossible to derive a sex from these traits. In fact, most brains are better thought of as 'mosaics' of traits that were once thought of as 'male' or 'female'. A study using MRI scans of 1,400 brains showed large amounts of overlap in previously 'sexed' characteristics of white matter, grey matter, and neural connections, with most brains being very mixed in their phenotypes of 'sexed' brain matter (Joel et al., 2015). So, no matter the sample type, MRI, or analysis method there has been no sign of dimorphism or internal consistency of sexed differences in brain structure.

Behavior is a more common metric, assumed under neurohormonal theory to have an unknown physical root connecting steroid hormones and behavior. The types of behavior that are examined to find potential sexual dimorphism include sexuality, sexual orientation, and typed interests (Jordan-Young, 2010). Sexuality, in this case, is the way in which one experiences sexual urges and desires, not the attraction towards another individual (Arboleda et al., 2014; Jordan-Young, 2010). It was once understood that 'masculine' sexuality includes frequent thoughts of sex, forward behavior, acting on one's own desires, and becoming aroused to visual stimuli (Arboleda et al., 2014; Jordan-Young, 2010). Inversely, 'feminine' sexuality is seen as passive, indifferent, with infrequent thoughts of sex, and arousal primarily to physical stimuli (Arboleda et al., 2014; Jordan-Young, 2010). By this concept, 'masculine' sexuality is focused on the genitals and physical pleasure, with or without a partner, while 'feminine' sexuality is focused on romance and emotional pleasure, always needing a partner (Arboleda et al., 2014; Jordan-Young, 2010). With the freedom for DFAB people to speak up about their own sexualities and the further emergence of DFAB people in the sciences, this idea was quickly realized to be false. Many DFAB people experience visual arousal, masturbate, have frequent sexual desires, or exhibit other supposed 'masculine' traits of sexuality (Butler, 1990; Fausto-Sterling, 2012; Jordan-Young, 2010). Likewise, DMAB people can experience the inverse. But, these preconceived ideas of sexuality remained until as late as 1982, when John Money and his team, in a follow up on a study of CAH XX

individuals, said that subjects reported experiencing erotic response to both physical touch and visual stimuli and experienced pleasure genitally (Ehrhardt & Money, 1967; Jordan-Young, 2010). Despite Money's previous categorization of this as 'masculine' in previous studies, in this case it was marked as "normal female sexuality" (Jordan-Young, 2010). This finding aided Money in his conclusion that CAH XX individuals are behaviorally indistinguishable from control XX individuals despite their early androgen exposure (Ehrhardt & Money, 1967; Jordan-Young, 2010).

Sexual orientation, to whom or to what traits a person is sexually attracted, is another outdated facet of sexed behavior under neurohormonal theory. Under the heteropatriarchal concept of sex, it is 'male' to be attracted to 'women' and 'female' to be attracted to 'men', with assumed neurohormonal origins of this attraction (Fausto-Sterling, 2000a; Jordan-Young, 2010). Thus, when observing an individual in the context of the neurohormonal theory, one would say that attraction to women is a "male" trait, which points toward masculinized structures in the brain formed by androgens, and vice versa (Fausto-Sterling, 2000a; Jordan-Young, 2010). This is again troubled by the nuance of definition and context. How does a researcher measure attraction? Relying on self-reporting of identity ignores how fluid identity can be. Simply reporting past sexual encounters erases other factors that can influence encounters other than attraction. But reporting attraction depends on how attraction is framed. Are we equating heterosexual men to homosexual women because both desire men or homosexual men to homosexual women because both transgress heterosexual norms? Are they attracted to masculinity or femininity? Certain genitals? What about attractions to trans, nonbinary, or intersex people? There is no best metric, though some are better than others, and what metric a researcher chooses decides what they are actually studying. But, when a researcher says that they are studying sexual orientation and hormones, we have to be sure that they are doing so with an inclusive and accurate measuring tool, that they are looking at every variable, and that care is taken to be respectful in their study. Even then there is no guarantee that another researcher who uses a different

metric will be studying the same thing, even if they also claim to be studying the connection of hormones to sexual orientation. Many times two studies that claim to be studying the same thing use such different measures and framework that comparing the two datasets would be like comparing apples to oranges.

In what studies we do have, using CAH XX individuals as the focus, some have found these individuals to have higher rates of homosexual fantasy and behavior than their non-CAH family members (Jordan-Young, 2010). But most studies do not find these rates to be any higher than the general population of non-CAH XX individuals (Jordan-Young, 2010). This is because the rate of self-reported homosexual fantasy and behavior in family members of CAH XX individuals is often lower than average, meaning that CAH XX individual seem to have more instances of homosexual fantasy and behavior by comparison (Jordan-Young, 2010). Larger, more comprehensive, studies of homosexuality in CAH XX individuals have found no reason to conclude that these individuals are more likely to display homosexual tendencies than any other individual, regardless of early androgen exposure dose or timing (Jordan-Young, 2010).

Sex-typed interests, mostly in younger children, is another way that masculinity and femininity have been measured in the context of the neurohormonal theory. This metric relies on two assumptions: first that sex-typed interests are in some way innate and; second, that these supposed innate interests are related to the neurohormonal theory (Jordan-Young, 2010). However, a researcher can do little to detect an innate component to interests because of the confounding influence of socialization (Fausto-Sterling, 2012; Jordan-Young, 2010). As early as its day an infant is bombarded by sensory cues and is sexed and gendered by those around them. When a child with a penis is born, every family member gives the new parents blue onesies, camo blankets, toy cars, and Legos. A child with a vagina is given pink onesies, princess dresses, dolls, and house sets. This presents a possible sensory bias. If a child lives in a pink room and has pink toys and eats out of pink bottles a child, as soon as they

are able to see color, is likely to develop a positive association with the color that is most prevalent in its life (Fausto-Sterling, 2012). This is not an innate preference, it is conditioned. This is also an example of the cultural influence of possessions. By saying "these things are for you" and giving only 'manly' or 'girly' options, a parent implicitly tells their child that "those other things are not for you". This becomes more overt when a child is screaming in the store because he wants a Barbie doll and his parents tell him no because "that's a girl toy". This concept is extended to play type. Reprimanding girls for being too rowdy while sending boys outside to "blow off steam" shapes the actions of children based on our social standards and rules. From romance and self-adornment being 'feminine' and high energy play and utility in clothing being 'masculine', most of the traits that researchers use to determine sexed interests fall into cultural norms that are enforced in children, by parents, other adults and the media, since birth (Jordan-Young, 2010). Additionally, the cultural reasoning behind why these traits are considered 'masculine' or 'feminine' make them scientifically arbitrary. For these reasons sex-typed behavior is not a metric that can be reliably used in testing neurohormonal theory.

Further examining the hypothetical link between brain structure and behavior, scientists are studying differentiation of pregnancy-associated progenitor cells (PAPCs) in maternal tissues (Bianchi, Zickwolf, Weil, Sylvester, & DeMaria, 1996). When a fetus is carried in utero, a bidirectional cellular exchange occurs between fetus and parent (Bianchi et al., 1996; Zeng et al., 2010). If this fetus is XY, the cells exchanged will go from an XY body to an XX body and vice versa. In rats it has been observed that these PAPCs can travel to the brain and differentiate into neurons, living in the brain for up to 7 months postpartum (Zeng et al., 2010). These rats with XY neurons have not yet been tested for differential behavior, to my knowledge, but in humans XY PAPC cells have been found in the blood of postpartum XX individuals whose last pregnancy was 27 years prior to testing (Bianchi et al., 1996; Zeng et al., 2010). Studies of deceased XX individuals have found neurons with XY genotypes, presumably originating in the same way (Hird, 2004; Jordan-Young, 2010). This is a further example of how the brain can change

throughout the lifespan and another possible challenge to the idea that changes in neural structure can lead to changes in sexed behavior.

Overall, we see no clear dichotomy, with internal consistency, in behavior. And the differences in averages that we do see cannot be separated from the culture, context, and socialization in which they were formed. When we look at physical structure the results are the same: no dichotomy, no internal consistency, and confounding variables that are impossible to separate from the variable we intend to observe. The brain is the most dynamic organ in the human body; it is plastic and is praised for being so (Fine, Jordan-Young, Kaiser, & Rippon, 2013). The idea that genes create brain pathways has been long overturned and the neurohormonal theory is showing signs of one day following suit. The pathways in our brains are built by Experience-Dependent Plasticity (EDP), the way that our experiences grow, maintain, and prune the neural connections our neurons (Fine et al., 2013). Despite EDP having a robust foundation and its effects in many areas, such as learning or personality, being widely studied, studies on EDP's effects on sexed behavior are few and far between. The great plasticity of human brains is well known. It is the very cornerstone on which our brains are built, to the point that many argue it is what makes us human (Fine et al., 2013; Jordan-Young, 2010). Yet it is still ignored in the fields of human biological sex and psychological gender studies, with neurohormonal theory overshadowing it (Fine et al., 2013; Jordan-Young, 2010). With the data that we do, or rather do not, have, I would like to see an end to the idea of physical brain sex and the equal treatment of EDP studies of sexed behavior compared to neurohormonal theory. Thereby allowing carefully created studies and their fit to determine which model is closer to what is observed. As it stands, there is no evidence of physical brain sex and no known biological origin to culturally sexed behavioral variations.

Cellular Sex

Cells can be divided into haploid cells and diploid cells (Klazema, 2014). Diploid, or somatic, cells have two copies of each chromosome. Haploid, or germ, cells have only one copy of each chromosome (Klazema, 2014). Somatic cells make up almost every cell in the body, from skin to neurons to organs, whereas the only germ cells are gametes produced from the germ line in either the ovaries, testes, or parts of the ovotestes (Arboleda et al., 2014; Klazema, 2014; Tomaselli et al., 2011). Some scientists have proposed that, because only sperm cells have differences based on their X or Y chromosome, with each variant having different speeds and peak survival environments, they are the only cells we can say are sexed (Hird, 2004). Others maintain that XY and XX somatic cells may have sexed differences, in how they react to internal stimuli, or otherwise (Hird, 2004; Tannenbaum, Schwarz, Clayton, de Vries, & Sullivan, 2016).

Whether they are individually sexed or not, somatic cells are another way that biological sex can be further complicated- through a condition known as chimerism (Bianchi et al., 1996). Chimerism occurs when an organism has cells originating from two zygotes; most commonly when two zygotes fuse in utero (Bianchi et al., 1996). This results in a body that carries two genetic codes and can manifest as an individual with organs with different genomes, two blood types, or mixed gonads and/or genitalia (Bianchi et al., 1996; Pritchard, Wick, Slonim, Johnson, & Bianchi, 2012). Not all XX/XY chimeras are intersex, and many chimeras live most, or all, of their life without knowing about their chimerism. This leads to the condition being seen as rarer than it actually is. In addition, most chimeras that originated from XY/XX zygotes will have only one zygote's cells in their reproductive organs and genitalia, with manifestations of their chimerism being found in heterochromatic eyes, patchy skin tone, duel blood type, or lying undetected in having one or more internal tissues or organ stemming from a different cellular line than the rest of the body (Bianchi et al., 1996; Pritchard et al., 2012). Most chimeras do not know that they are chimeric until something goes wrong, like failing a maternity test or needing an organ transplant. That means that any individual could possibly be partially made of cells that originated

from a different zygote than the one that created their blood, saliva, and other typical targets of DNA testing. And these other cells may not have the same XX or XY chromosome combination that they assumed made up their entire body.

Other forms of chimerism, called micro-chimerisms, include organ transplants, blood transfusions, bone marrow transplants, skin grafts, and PAPCs (Bianchi et al., 1996; Loubiere et al., 2006; Pritchard et al., 2012). Many cell types have been found having crossed as PAPCs, including immune lineages, mesenchymal stem cells, and placental-derived cells (Pritchard et al., 2012). As many as 75% of pregnancies can end with the parent carrying fetal immune line cells that can persist in the body for years with XY PAPC cells being found in the blood of one individual 27 years post-pregnancy (Bianchi et al., 1996; Loubiere et al., 2006). Stem cells transferred in this way can differentiate in the body into different cell types, including organ cells and neurons, as mentioned in the Brain Sex section (Bianchi et al., 1996; Zeng et al., 2010). One can also receive microchimeric cells from an older sibling, twin sibling, miscarriage, or vanished twin in utero. All of these methods of receiving extra-zygotic cells can result in mixed XX/XY genotypes in the recipient (Bianchi et al., 1996; Khosrotehrani et al., 2003).

Haploid cells, also known as germ cells, germ line cells, or gametes, receive special attention when talking about biological sex. Gametes are seen as part of the social and medical definitions of biological sex and are the sole focal point of the evolutionary definition of biological sex (Ah-king, 2010; Hird, 2004). But not all organisms, or even all individuals, produce gametes (Bagemhil, 1999; Hird, 2004). Microorganisms have no analogous structure to the gamete, a strictly multicellular structure needed t for macroorganisms to reproduce (Bagemhil, 1999; Hird, 2004). In organisms that do use gametes, it is not uncommon for an individual to be unable to produce gametes for one of many reasons. Agenesis of the gonads prevents any gametes from being produced and can occur for many, typically chromosomal, reasons (Arboleda et al., 2014). Infertility based on improperly coded or absent gamete-building genes, lack of one or more structures needed, or an improper environment for protein folding can lead to an

agamic state, or at least a state without functioning gametes (Miyamoto et al., 2012; Singh & Schimenti, 2015). Menopause naturally ends gamete retention (Lichterman, 2009). Chemical infertility, through hormonal changes or external chemicals, and surgical infertility can also lead to agamic states, either accidentally or purposefully. All of these occurrences can lead to an organism without functioning gametes. What sex would these individuals be assigned under the evolutionary definition of biological sex? Or would they be sexless? Would one's sex change after menopause? Surgery? Discovering one's infertility? It seems silly to think of, but if we define sex by gametes these are the questions that need to be asked.

In the medical and social definitions of sex, gametes seem to be almost inconsequential, more of an afterthought compared to gonadal or genital sex. Compared to somatic cells and cell structures, gametes make up an extremely small portion of our bodies. If a human has an estimated 37.2 trillion cells in their body and an estimated 2 million gametes, as estimated for ova in a fetal XX individual, then gametes only make up 0.000000119331742% of their cells (Eveleth, 2013; Silber, 2017). These cells are also only relevant in terms of reproduction or reproductive health, with the majority of people not thinking about their eggs or sperm unless they are trying to have a baby or avoid having one. Considering that the designation of sex has lifelong medical and social implications it may not be fitting for something as small and voluntarily removable as gamete status be part of the definition.

Ideology

Ideas about biological sex have changed throughout time, following scientific discovery and societal ideals in tandem. During the pre-enlightenment era, women were seen as the same as men, just inverted, with the vagina being seen as the same structure as the penis (Hird, 2004; Laquer, 1990). The

separation that was called ‘sex’ was more analogous to what we today call gender, a behavioral dichotomy more than the bodily factors (Hird, 2004). The one bodily sex was said to have varying levels of ‘seed’ or ‘fluid’ inside that determined if someone turned out ‘male’ or ‘female’ and the ‘seed’ levels were said to be in a constant warlike battle over the body (Hird, 2004). As for the bodily form, a separate issue than ‘male’ or ‘female’ at the time, heat was said to have determined if genitals were internal or external. The genitals were two forms of the same organ, which would escape the body if it had more ‘vital heat’ to form the penis or remain inverted and form the vagina if the body was cold enough (Hird, 2004). Until the 18th century, even while knowing of bodily differences, behavior was still what decided sex and records show cases of DFAB children developing an elongate phallus and thusly changing behavior and social position without trouble or remaining in their ‘female’ behavior and role (Hird, 2004). Bodies were in a more acceptable state of flux because of the one-sex model. It did not matter what body a person had as much as if they correctly performed their social sex, ie; ‘gender’ (Hird, 2004).

In the 19th century post-enlightenment, the ability to dissect cadavers spurred a shift from a gender-based sex to a biological sex based in what were called ‘fixed patterns of difference’ (Hird, 2004; Laquer, 1990). The body was no longer a whole package, it was a collection of parts which needed to be classified (Hird, 2004). These new ideas put ‘males’ and ‘females’ on separate tiers, never to touch and separated into a ‘perfect working system’ where they were different and complementary (Costello, 2016; Hird, 2004). This ‘sex complementarianism’ was fueled by religious views and meant to back heteronormativity and patriarchy under the guise of what is ‘natural’ (Costello, 2016; Hird, 2004). In doing so the female body became a perfectly created ‘baby maker’ that was fitted and subservient to men (Costello, 2016; Hird, 2004). This social effect on the interpretation of data is seen most clearly in the past descriptions of sexed skeletal structure. On average the skull of a DFAB person has a larger skull-to-body ratio than the average DMAB person (Hird, 2004). This was interpreted as ‘’females’ skulls

are more childlike and so they are less rational and intelligent' when it could have been just as easily interpreted as increased intellect via more brain space (Hird, 2004). The interpretation of the vagina as analogous to the penis also persisted after the enlightenment, despite no longer thinking it to be an inside-out version thereof (Laquer, 1990). This thought, which aligned with previously held beliefs, persisted until developmental studies allowed scientists to discover that the penis is analogous to the clitoris (Hird, 2004; Laquer, 1990). Thus, interpretations from and use of past data requires a historical analysis of what biases and assumptions went into that data. This continues today as biases, agendas, and historical context are still used to build and interpret scientific studies of biological sex. Science of sexual difference has, since its conception in the enlightenment, been used to further the ideas and constructs of society and guise them as 'natural' and, therefore, indisputable (Costello, 2016).

An ideology is system of ideas and ideals, typically one that forms the foundation of a policy or political action (Lorber, 1993). Unlike a scientific fact, which exists whether we discover it or not and does not require any help to be upheld, an ideology is a man-made system with categories and boundaries that need to be maintained and enforced to remain in place (Costello, 2016; Lorber, 1993). Natural bodies break the supposed rules of biological sex often and researchers are steadily finding new ways that bodies continue to break these rules. But many people do not want these rules to be broken. That is why corrective surgery for intersex people exists; it is why misconceptions about steroid hormones abound; and it is why transgender people, both on and beyond the binary, have been disenfranchised for decades. Nature does not uphold biological sex, humans do. When people call intersex bodies 'unnatural', or the connotative use of 'mutated', it is a sure sign of ideology. As Costello put so eloquently, "Truly unnatural things do not happen… if ever you want to know when to suspect an ideology is at work, you can be sure it is the case when someone tells you something is unnatural and should not occur. Because here's the thing about true natural laws: they function whether you want them to or not" (Costello, 2016). Nonetheless, people cling to ideologies very strongly, claiming that any

violation will do away with the whole system, causing it to crumble (Costello, 2016). In the same way, people rebel against having their ideologies shaken most strongly when they do not think it is ideology and instead think that it is fact (Costello, 2016). So, if biological sex can crumble because an intersex person exists without surgically being forced into one sex, or because of the existence any one of the many forms of sexual element variations, then it isn't really a scientific fact- it is an ideology.

But why does biological sex being an ideology matter? It's a system that works for a large percentage of the population and covers most cases well enough. Why change the system if it works well enough? I'd say it is because 'well enough' is not the goal of either science or medicine. In both fields if a researcher or a medical practitioner can do better, whether it be in the pursuit of knowledge, practice, understanding, or care, if they can do better it is wrong not to. The system of biological sex has widely reaching effects. Its influence extends from research protocol, where the US National Institute of Health (NIH) mandates that sex be included as a biological variable in all NIH-funded studies, to social documentation, what areas individuals can access, and what types of treatment they will receive (Fausto-Sterling, 2000b; Tannenbaum et al., 2016). For many, the coverage of the system of biological sex is not 'well enough'. For intersex individuals, whose births are treated as taboo cases of socio-medical emergency and are subjected to cosmetic 'normalization' surgery before an age where they can give informed consent, this system is not 'well enough'. For binary and nonbinary transgender individuals, who are prevented from living as their selves, on paper or in their day to day lives, because biological sex is used as a weapon against their existence, it is not 'well enough'. For DFAB individuals, systematically having their bodily rights curbed and standards of medical care sacrificed because of 'scientifically' backed acts of sexism, it is not 'well enough'. Biological sex fails many people. It fails them medically, psychologically, socially, and politically-- all with the assumption that these failures are scientific and natural and that there is no way of changing them. But if biological sex is understood as an ideology and not as scientific fact then these failures cease to be acceptable. If science no longer

upholds biological sex, then the only thing that holds it in place and allows it to hurt and to fail our vulnerable populations is us.

Moving Forward

If sex is an ideology and not fully supported by scientific data, is it at least a good rule? I would say no, in its current form it is not. If there is an exception to every element of a rule then it is hardly a rule to begin with. In this paper I have highlighted many exceptions for each element of sex's rules. For chromosomal sex we have XO, XXY, XXX, and many other combinations which are exceptions to the rule of 'XX or XY'. For gonadal sex we have ovotestis, mixed expression of a single ovary and testicle in the same individual, gonadal streaks, and surgically removed gonads as exceptions to the rule of 'ovaries or testicles'. For cellular sex we have chimerism, microchimerism, and various types of sterility as exceptions to the 'XX or XY cells' and 'ova or sperm' rules. For every rule that the ideology of biological sex offers there is an exception and a person alive today who embodies that exception. For some exceptions, such as chromosomal sex, these people may be a minority but a system that works for a majority is not the same as a system that works for everyone. These minorities, when added together, form a large and diverse group that are not fully described, or protected, under the rules and ideology of biological sex. And, even if their numbers were small, a majority is not everyone. Our medical and societal constructs are imperfect and in need of changes if they cannot cover everyone. So, our current system of sex is far from a rule. It is a suggestion at best.

In both physical and psychiatric medicine, changes in the concept of sex would have widespread implications. The first would be paperwork. The inclusion of sex in paperwork should be changed, as well as how we keep track of what body parts a patient has and needs to keep healthy. For trans, nonbinary, and intersex patients, this small step can have immeasurable benefit towards their mental wellbeing and comfort. For some, this comfort may be the difference between choosing to seek medical

help, instead of abstaining from it (American Psychological Association, 2015; G R Bauer, Pyne, Francino, & Hammond, 2013). This goes doubly so for psychiatric care, where respect for identity can be the difference between a positive outcome and borderline malpractice, as is seen in its strongest form in so-called conversion therapy (American Psychological Association, 2015; Nemoto, Bödeker, & Iwamoto, 2011). For trans, nonbinary, intersex, and cis people, the biggest failure of current methods of including sex in paperwork is the assumptions it brings into patient care. When a patient is marked as being 'female' their doctor turns their attention in one direction. Have they had a breast exam? Pap smear? What contraceptive methods are they using? Are they pregnant? While at the same time other questions are put away. Have they had a prostate exam? Are they at risk for cardiac disease? Are they satisfied with their sexual health? These questions, like research questions and methods, are vehicles for assumptions. Just because someone checks 'male' does not mean that they do not need a breast exam or different options for contraceptives. Just because someone checks 'female' does not mean that they are not at an elevated risk for cardiac disease or that reproductive function is the goal of their care. Furthermore, these categories do not mean that one can assume what body parts someone has or what is normal for them. For an intersex individual, having indeterminate genitals may be their normal. For a DFAB person who chose to remove their ovaries, that is their normal. By caring for the bodies that patients have instead of the bodies we assume they have, or the bodies we think that they should have, we can offer a higher standard of care for everyone who seeks medical care.

Socially and politically, the reconfiguration of policy to include the recognition of trans, nonbinary, and intersex people will further improve the standard of living, and ease dysphoria or unrest that sex-based documentation causes, for many (G R Bauer et al., 2013; Rhodes, Bethell, & Bondy, 2006). Offering a more inclusive system would not only be an act of respect but could, for some, be a lifesaver. Individuals who do not feel accepted and affirmed in their identity are at a far higher risk of committing suicide than their cisgender counterparts. Any small ways we can decrease this risk can have

a large impact (Greta R Bauer, Scheim, Pyne, Travers, & Hammond, 2015; Yadegarfard, Meinhold-Bergmann, & Ho, 2014). Changes would also allow a slow start in the right direction towards curbing discriminatory rhetoric, as science would no longer stand by and offer sound bites of paraphrased biology which act by separating groups further. With scientific findings no longer upholding ideology, or simplistic rhetoric, a social and societal change in attitude towards these groups would fall to personal and religious beliefs. These beliefs are not ours as scientists or policy makers to change, but we can no longer to offer ideology, inconclusive data, and over simplifications of complex biological processes which can be misconstrued as facts that support these beliefs.

It is important to ask: in having a system of sex, both as ideology and as supposed biology, what are we trying to accomplish? First, as is the goal of many systems in scientific fields, the system of sex is meant to categorize. It is meant to provide clean lines between groups, saying that people with certain parts and elements belong in one group and those without those parts and elements belong in the other group. In creating these groups categorization also works in reverse, saying that if one is in a group and fails to have certain parts of elements then one must either switch groups or be 'fixed' so to fit the group description. It takes a system of benign cataloguing and, because it does not describe all known variation and becomes both social and political, turns it into a system of distinct boxes that everyone is expected to fit into. There is also the goal of verbal and social precision, having a way for others to understand exactly what is meant when one is communicating ideas with words or nonverbal cues. In a culture that works to maintain heterosexuality and its associated norms, patriarchy, cisgender, and the nuclear family, being able to easily convey one's genital status, reproductive role, and place in the heteropatriarchical hierarchy is important. The system of biological sex do all of this in several ways. By verbally saying "I am male", or the colloquial, gendered offshoot "I am a man", what a person is really saying "I have a penis, I produce sperm, I do not carry a fetus, I am most likely attracted to people who do, and I am in a place of social power". In addition, they are implicitly accepting associated social roles

and ways of being that are expected of the category. Nonverbally, this is more often communicated via secondary sex elements, mode of dress, and behavior, all of which are seen as shorthand for the phrase "I am male" and everything that entails. The same goes for documentation. On identification cards such as passports, birth certificates, and driver's licenses the category of sex serves as another way to identify an individual beyond the headshot photograph because the 'F' or 'M' implies bodily traits that one should expect to see. For TSA x-rays and scans, telling the computer that someone passing through the scanner is 'male' leads the computer to expect organic material in the crotch region and not flag it as a potentially suspicious item, such as smuggled substances or material. For medical professionals the 'M' and 'F' are shorthand for what type of care they will give. All of these purposes not only attempt to offer precision, but also try to maintain order. This strict order is what makes fluidity in the system difficult, or impossible. There is no room in the system for people who do not fit into the standard 'M' or 'F' unless we make room. There are hurdles for trans people in the form of state-mandatory surgeries that must be performed before one can change one's documented sex, and mandated psychiatric screening before these expensive, uninsured surgeries are possible. These assumptions, imprecise attempts as precision, acts of gatekeeping, and intolerance towards variance make the system we have unable to accomplish the goals that it was set up to accomplish, let alone leave room for expansion and improvement.

So, since our current system is failing to accomplish its goals of meaningful categorization, communicative precision, and maintaining order-- what should be done instead? For categorization, I would suggest a duel system of scientific observation and social self-identification. Science, no matter what, will continue to categorize. It is the job of scientists to sort out what they see and to try to make sense of it. I am not saying that we should bar that. But, what is observable and known should take precedence over pet theories. If data contradicts what is expected, it should go without saying that, as scientists, we should be excited and intrigued by this, not quick to ignore it or shut it down. By doing so

we ignore the diversity, complexity, and fluidity that exists in the world we are trying to document. In saying that divergences in form or function of the body are mutations, in the connotative sense, we forget that they are also mutation in the denotative sense. They are variations from which genetic diversity enters our gene pool; the changes that occur naturally and are just as random of a scramble as anyone else's genotype. They are as natural as anyone else. Our obsession with categorization should not be so militant that we forget that nature is dynamic and fluid and that our boxes will never quite capture the whole picture. This is why self-identification is so important. Observation can find every way that human bodies can vary, but even if every physical mix and match option had a name, which they do not, it would still not capture the internal sense of self that each body is married to. This sense of self, which is how a human orients themselves in their body and in the world, is important because for most everyone it is a vital part of their life whether they know it or not. Having this sense of self respected is a key part of one's mental health and wellbeing and essential in any reformed system of sex (Yadegarfard et al., 2014).

One of the ways that sense of self and social categorization can both be satisfied is for the addition of a "neither" or "none of the above" category in the sex and/or gender categorization systems of all documentations. This would allow the system to always be accommodating to each individual, as it means that the system is not the definition of what options are available. But for this addition to be meaningful it must come with true fluidity within the system. This means that the ability for one to change their documented sex should be easy, affordable, and accessible. The process should be without high fees, mandatory psychiatric letters, surgeries, or proof of 'living as X' for any amount of time, all of which act as gatekeepers to people who cannot access healthcare or cash, who do not wish to change their body, or who do not wish to change lifestyle. A record of past identities and the cost of printing new documents and ID cards should be all that is needed to change sex in a truly fluid system.

Lastly, in place of attempted precision, a system of true communicative precision in both medical records and day to day written and verbal language is needed. This means that if a person enters a medical office, instead of marking a paper to say they are male or female, they would check what organs and physical structures they have. If a patient marks that they have a vagina, then a doctor can ask if they have had a pap smear recently. If they have a prostate they can ask if they have had a prostate exam, and so on. This true precision of language allows for doctors to care for the bodies that patients have without involving a system of sex that allows assumptions and dysphoria to propagate. This extends to common language. As I have been, in this paper, using phrases such as "DMAB" and "DFAB", or "people with penises", or using the terms "men" and "women" only in a way that includes trans men and women by default. This allows what I am actually saying to be fully understood, without use messy biological shorthand, unless referring to the system of shorthand in itself. Mindfulness in saying what is meant allows for personal exchange, literature, and medical care to be inclusive, caring, and precise.

I do not advocate for the erasure of sex from our society. It would be unrealistic to do so. I only advocate for the reclassification and redefinition of sex from a biological fact to an ideology, a human-made category that we have control over and can change so to better include those who currently do not fit into the system. Changes should be made in the form of self-identification, the addition of 'none of the above' or similar categories, accessible fluidity within the system, and true communicative precision. These changes and ideas stem from both the immense variation observed in the human body that violate the 'facts' of biological sex in one or more ways, and the ways that sex matches the definition of ideology. Bodies, structures, and hormones exist- there was never any doubt that they do- but they do not exist in the boxes and rules that we set up for them. The system of sex that we have in place is not any more natural than the 24-hour time schedule. Just because we realize that a 24 hour time structure is arbitrary doesn't mean that we don't recognize that the sun, day, and night exist, only

that the system doesn't exist naturally. Variance, plasticity, and fluidity are natural and messy, this is why we try and find order in them. But in our attempt to organize we cannot, as scientists who live in a social world and as social beings who live in a political world, lose sight of the bigger picture. The bodies we are trying to categorize are also people. They are not just parts, but a whole which will always be greater than the sum. If our science and our politics are not taking this into account then they are failing. We owe it to our fellow human beings to be kind and cautious in our interpretations and applications of results, and we owe it to nature to remember that it will always be more complex than a textbook can summarize.

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