Complicating Male and Female Brains and Discarding Organizational/ Activational Theory: Behavioral Neuroendocrinology from 2000 to 2019

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My dishonorable mentions are to Dulles International Airport's Port of Entry (the most miserable hours of my travel experiences) and the person who made an AI (read: language-learning model) acknowledgement page generator that I found while trying to discover if acknowledgments pages had any rules (and I think "you have to write your own thank-you's" should be the first and only one). Also, a mention of myself: the length of this thesis is well over requirements, and I cannot even excuse myself by wishing I had more time to write a shorter work.

Thank you, finally, to my mother (for her patience and support), to my sisters (for their love and encouragement), and to my partner (for making me laugh and making me happy).

Chapter 1: Introduction

"Surgeons must be very careful

When they take the knife!

Underneath their fine incisions

Stirs the culprit—Life!*"* [1]

1.0: The Research Question

"Research on sex and the brain has come a long way since 1969."[2]

In 2020, Melissa Hines published a commentary called "Neuroscience and Sex/Gender: Looking Back and Forward," as part of a celebration of 50 years since the founding of the Society for Neuroscience. Intriguingly, the paper discussed research on sex and gender differences from the perspective of a theory being proven insufficient. A paper in 1959, by Phoenix *et al*., had proposed that early androgen exposure "organized" sexually differentiated behavior and that later circulating androgen "activated" that behavior, based on their studies with guinea pigs.[3] However, Hines' conclusion firmly anchored that study in the past:

"The 1959 report of permanent influences of early androgen exposure on later behavior, as well as reports of dramatic sex differences in the avian and rodent brain, corresponded to a historical time when the social roles of men and women differed dramatically. The narratives of that time often referenced inborn systems and hard

[1] Emily Dickenson, "Surgeons must be very careful," *The Poems of Emily Dickenson Edited by R. W. Franklin,* Harvard University Press (Cambridge, Mass.): 1999, republished at URL: poetryfoundation.org/poems/45722/surgeons-must-be-very-careful-156.
[2] Melissa Hines, "Neuroscience and Sex/Gender: Looking Back and Forward," *Journal of Neuroscience 40*(1): 2 January 2020, DOI: 10.1523/JNEUROSCI.0750-19.2019, p. 41.
[3]Charles H. Phoenix, Robert W. Goy, Arnold A. Gerall, and William C. Young. 1959. "ORGANIZING ACTION OF PRENATALLY ADMINISTERED TESTOSTERONE PROPIONATE ON THE TISSUES MEDIATING MATING BEHAVIOR IN THE FEMALE GUINEA PIG." *Endocrinology 65* (3): 369-382. DOI: 10.1210/endo-65-3-369.

> *wiring of the brain. More recent research has demonstrated greater neural plasticity than was imagined 50 years ago, and interactions between hormones and environmental factors in shaping the human brain and behavior."*[4]

Even more intriguingly, many of the papers cited were relatively recent, and other parts of the paper suggested that the study of sex/gender in a neuroscientific framework was an ongoing project on newly shaky ground.

All of this information implied a recent but fairly decisive theoretical shift, in which the organizational/activational theory of Phoenix *et al.* had been contradicted and, at least by some researchers, discarded. I wanted to know how the assumptions Hines spoke about, assumptions about "dramatic sex differences" and "inborn systems and hardwiring of the brain," had been revealed and then discarded. Her commentary placed these ideas squarely in the middle of the last century—was that true? Had they lived on? How had "neural plasticity" and "interactions between hormones and environmental factors" undermined organizational/activational theory? How had the organizational/activational theory become historical, and what might that change reveal about scientific theories and knowledge more broadly? After investigating the organizational/activational theory and doing some additional reading, I decided to rewind the clock twenty years and watch the organizational/activational theory's decline through a survey of journal literature from 2000-2019.

The majority of this introduction by wordcount contains background information. The literature surveyed for this thesis is recent and often technical, using information from fields which have been changing rapidly in the last few decades, and so I decided to err on the side of caution. §1.1 discusses the organizational/activational theory specifically, while §1.2 briefly

[4] Hines, "Neuroscience and Sex/Gender: Looking Back and Forward," p. 41-42.

summarizes some of the historical precursors and background knowledge that will be useful to get the most out of chapters 2-4. In particular, §1.2 attempts to give a multidisciplinary history of sex difference research leading up to and taking place around the year 2000. §1.3 discusses the choice of literature for the survey and §1.4 discusses prior research on sex/gender behavioral differences in neuroendocrinology. Finally, §1.5 lays out the subjects of chapters 2-5.

1.1: What is the Organizational/Activational Theory?

The organizational/activational hypothesis or theory–or, sometimes, dogma–of the sexual differentiation of the brain is, on its face, rather simple. Prenatally, sex-specific hormonal influences–testosterone and estrogen for boys and girls respectively–organize the brain. Later, during and after puberty, circulating hormones (again, testosterone and estrogen) activate those behaviors, leading to the sexual differentiation of behavior. Since the gonads (testes or ovaries) differentiate significantly prior to the brain, the causal mechanism asserted by the theory was that chromosomal sex (the presence of XX or XY chromosomes in mammals) caused the gonads to differentiate, which then, by releasing hormones, caused the differentiation of the brain. While the theory was originally developed to explain sex-specific behaviors like mounting and lordosis in rodents (guinea pigs, initially[5]), rodents were and are one of the most common model

[5] Phoenix *et al.,* "ORGANIZING ACTION OF PRENATALLY ADMINISTERED TESTOSTERONE PROPIONATE."

organisms for human behavior when human tests would be unethical, and the broad strokes of the organizational/activational theory were assumed to apply to humans as well.

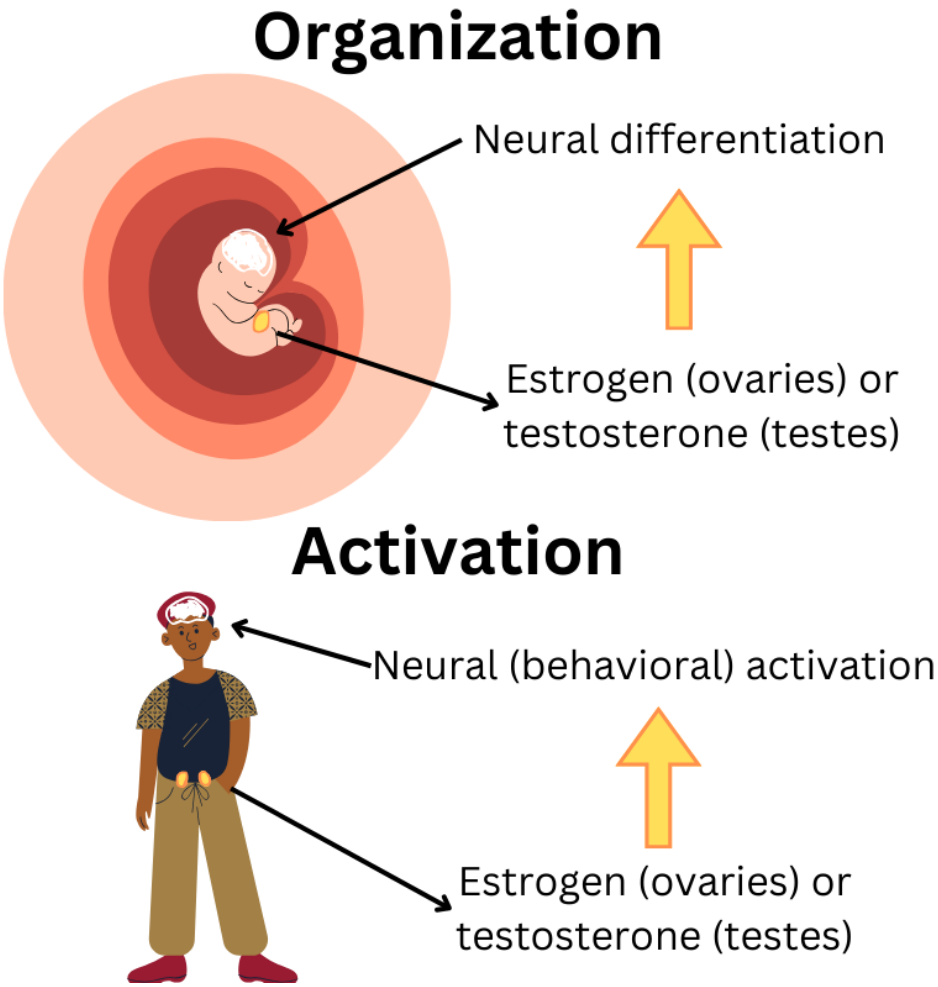

Figure 1. Image description of the organizational/activational hypothesis in its simplest form. (Made by author using Canva)

Like many simple ideas, the devil is in the details. Taken at its most literal, the organizational/activational theory implies an unchanging and sexed mind from birth, only altered by activation after puberty, injury, or disease. While this appears to fly directly in the face of the ability of minds to learn new behaviors and forget old ones, the organization of behavior need only apply to sexually differentiated behaviors, preferences, or desires. (If one could reliably and certainly determine what, if anything, was sexually differentiated in humans, that is.) Gender

identity and sexual orientation are the most consistently differentiated, in humans–those with XY chromosomes and testes tend to prefer women and call themselves men, and those XX chromosomes and ovaries tend to prefer men and call themselves women. Besides these (relatively) straightforward sex-typed aspects, there is also a fluctuating list of additional differentiated characteristics: sex-typed childhood play, empathy, social dominance, physical aggression, and spatial and verbal reasoning, to quickly name the most popular. There are also mental illnesses, neurodevelopmental conditions, and neurological diseases which are more or less commonly diagnosed by sex. Autism, ADHD, and schizophrenia tend to be more commonly diagnosed in and more severe in boys and men, while depression, multiple sclerosis, and anxiety are more commonly diagnosed in and more severe in girls and women. Between these behavioral, psychiatric, and neurological differences, it was hoped, a consistent idea of the 'male' and 'female' brain in humans and for mammals in general would become evident despite the cultural, social, and lifetime effects which obscured the fundamental biology of the (sexed) brain. As my descriptions have perhaps hinted, this hope was not fulfilled as of 2020.

1.2: Underlying or Historical Theories, Ideas, and Assumptions

In addition to the organizational/activational theory, there were–are–a few corollary or otherwise related theories which either fed into or were supported by the larger hormonally-determined structure. The specific hormone believed to be organizing sex-typed human behavior was testosterone, though sometimes the hormone was generalized to 'androgen(s)'. Prenatal androgen was argued to masculinize behavior, and an absence of prenatal androgens caused the fetus to, by default, feminize. More subtle arguments distinguished the axes of masculinization

and feminization, previously shown to be separate in lab experimentation,[6] but masculinization and defeminization were generally considered to go together and thereby collapsed into a single dichotomous spectrum for most purposes. Femininity was the 'default' state, and prenatal androgens masculinized and defeminized the animal's behavior. Finally, researchers argued broadly, "[w]hile the physical differences between males and females are clear, differences in behavioral traits are less clear-cut. However, certain behaviors do show sexual dimorphism in adults."[7]

Another related, though in decline by 2000, theoretical assumption was the lack of neural plasticity after that area was 'complete'. Essentially, significant remodeling to the brain cannot take place after a certain period, although this varies by the structure considered. The visual cortex largely stops developing after 12 months of age in humans, while the prefrontal cortex generally stops developing at about age 25.[8] While the organizational/activational theory argues most explicitly for organizational actions of sex-differentiated behaviors taking place prenatally, perinatal (at or near the time of birth) and neonatal (early infancy) actions are sometimes included in the span of time in which organization might occur. If the organization of sex-typed behavior was assumed to be 'locked in' or 'programmed' at a certain point, neural plasticity was, or could be, a threat to the permanence of that organization.[9]

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[6] Anne Fausto-Sterling, "Chapter 8: The Rodent's Tale" in *Sexing the Body: Gender Politics and the Construction of Sexuality* (New York: Basic Books, 2020). Ebook.

[7] Celina C.C. Cohen-Bendahan, Jan K. Buitelaar, Stephanie H.M. van Goozen, Jacob F. Orlebeke, and Peggy T. Cohen-Kettenis, "Is there an effect of prenatal testosterone on aggression and other behavioral traits? A study comparing same-sex and opposite-sex twin girls," *Hormones and Behavior 47* (2): February 2005, DOI: 10.1016/j.yhbeh.2004.10.006, p. 230.

[8] Gregory Z. Tau and Bradley S. Peterson, "Normal Development of Brain Circuits," *Neuropsychopharmacology* 35 (1): 30 September 2009, DOI: 10.1038/npp.2009.115, p. 147-168.

[9] It was possible at the time that the brain was modular, which would have ensured that the parts of the brain that governed sex dimorphic behavior would not be affected by neural plasticity, but that still would have left two problems. First, some of the theoretically sex dimorphic behaviors, like spatial or linguistic ability and memory, would interact with areas like the hippocampus (learning and memory) which were known or strongly believed to

Discussions and hypotheses about what the various areas of the brain did, functionally and structurally, also played a role. With the advent of functional magnetic resonance imaging (fMRI) in 1990, the brain's structures could be more directly linked to function; that is, changes in oxygenated versus deoxygenated hemoglobin could be used to track oxygen usage in neurons and—theoretically—their activation patterns.[10] fMRIs could also penetrate to the inner areas of the brain, such as the hypothalamus, brain stem, and pituitary gland, which electrodes (placed on the surface of the skull) could not reliably do. A final important point about fMRI measurements is that even a single individual's brain scan required significant computational processing and statistical analysis; the individual units of brain were "voxels", or subdivided three-dimensional areas of the scan, which computational analysis aggregated and smoothed. Examples of individual scans (Fig. 2A, numbered, with two types of stimuli) and composited brain scans for average responses (Fig. 2B, for the two types of stimuli) are given below.

exhibit neural plasticity, meaning that experience could play a significant role in these (again, theoretically) sex dimorphic behaviors. Second, many of the theoretically sex dimorphic behaviors, like aggression or empathetic ability, were not associated with individual areas of the brain and might or might not also be affected by plasticity. Regardless, however, because sex dimorphisms were frequently unlocalized to anatomical regions of the brain or defined as differences in learning and memory, plasticity still posed a theoretical problem for modular ideas of the brain.

[10] David J. Heeger and David Ress, "What does fMRI tell us about neuronal activity?", *Nature Reviews Neuroscience* 3: February 2002, DOI: 10.1038/nrn730, p. 142-151.

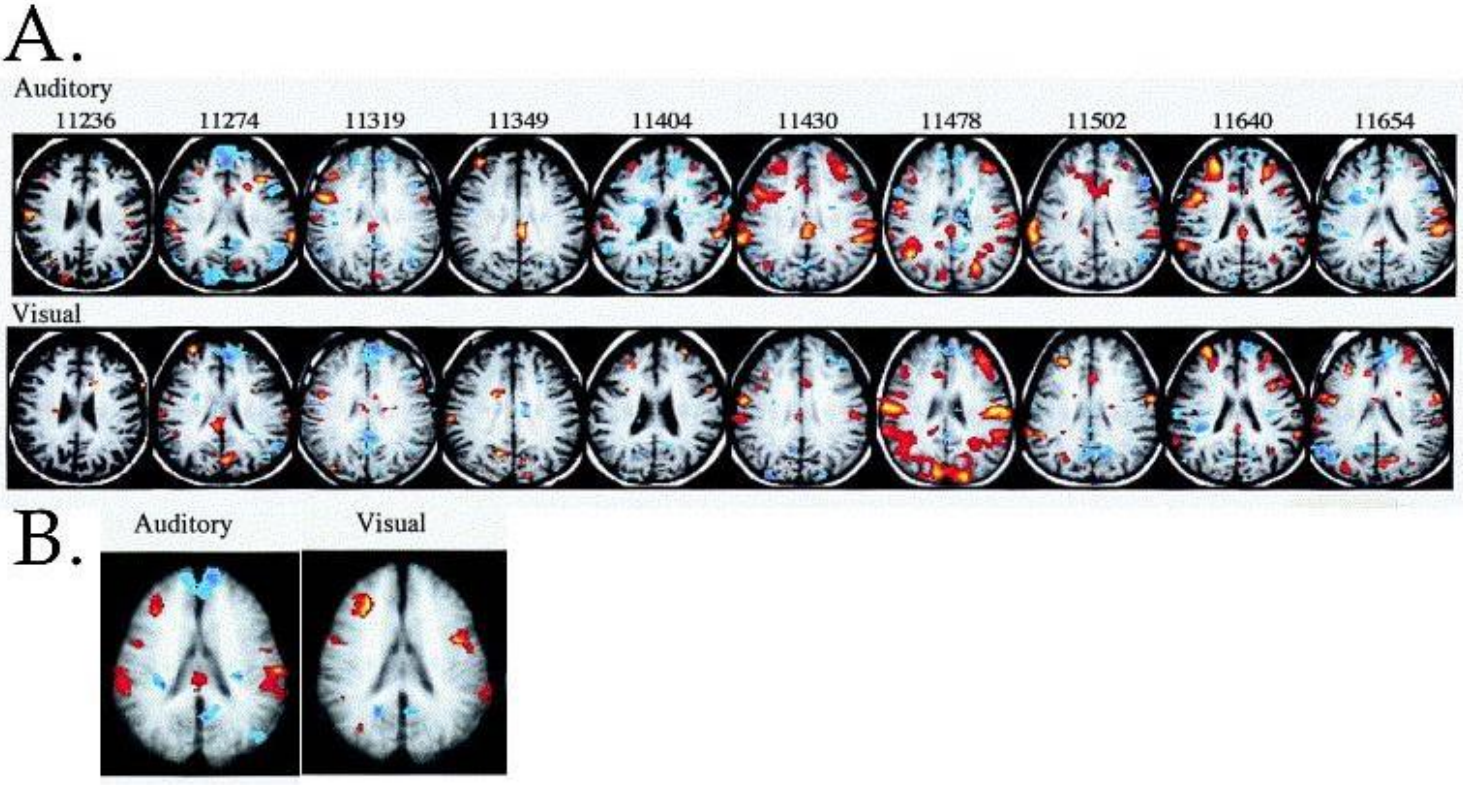

Figure 2. (A) shows representative scans from ten subjects (numbered at top) reacting to an auditory (top) and visual (bottom) stimulus. The blue shows local reductions in oxygenated blood while the red shows local increases in oxygenated blood. In theory, blue areas are currently 'activating' (using oxygen from the blood) while red areas are currently 'resting' (accumulating oxygen due to a lowered level of local activity). Both blue and red are relative to a general 'baseline'. (B) shows examples of composited scans at a depth in the brain that roughly corresponds to a composite of the representative images in (A). Images taken from Alexander A. Stevens, Pawel Skudlarski, J. Christopher Gatenby, and John C. Gore, "Event-related fMRI of auditory and visual oddball tasks,"* Magnetic Resonance Imaging 18*(5): June 2000, DOI: 10.1016/S0730-725X(00)00128-4, p. 495-502, chosen as a representative example of fMRI experimentation in 2000; (A) taken from Figure 3, p. 500; (B) taken from Figure 1, p. 498, z=23 for both images. Images were cropped and rearranged by author in Autodesk Sketchbook.

To explain why and how fMRI scans were used in sex difference research, it may be useful to consider one of the oldest posited sex differences in brain structure and function:

cerebral lateralization and the corpus callosum. The corpus callosum links the left and right hemispheres of the brain, and has a rather odd history bound up in the concept of "functional cerebral asymmetry," which in turn reaches out towards left- and right-handedness, cerebral development, intelligence, sex differences, and homosexuality.[11] The theory of cerebral laterality dates back to Paul Broca, an eminent French anatomist, who in the 1860s associated damage to the left frontal lobe (Broca's area) with a loss of speech capacity, and asserted that most humans were right-handed because they were left-brained.[12] Perhaps because the capacity for language is often considered one of the fundamental human aspects, brain (and hand) asymmetry also became associated with 'higher' cognitive function–indeed, rapidly becoming a marker of European heritage and manliness in the context of France in the 1860s.[13] While Broca himself believed that left-brainedness was a mere happenstance of development and that the right hemisphere could fulfill the same functions (creating left-handed individuals), contemporary and later neuroanatomists argued that the right and left hemispheres of the brain were functionally lateralized in healthy adults, and that this was a feature of humanity and higher thinking. [14] While left-handers could, in theory, simply be reversed right-handers, the belief that left-handedness might have a "pathological origin" remains relevant into the present day.[15]

In the nineteenth century, the left hemisphere contained the human faculty of speech, and thereby became associated with humanity, intelligence, reason, masculinity, white superiority,

[11] Anne Fausto-Sterling, "Chapter 5: Sexing the Brain: How Biologists Make a Difference" in *Sexing the Body*.
[12] Tabea Cornel, "An even-handed debate? The sexed/gendered controversy over laterality genes in British psychology, 1970s-1990s," History of the Human Sciences, 33, no. 5 (September 15, 2020): 138. DOI: 10.1177/0952695120944031.
[13] Anne Harrington, "Nineteenth-Century Ideas on Hemisphere Differences and 'Duality of Mind,'" *Behavioral and Brain Sciences* 8, no. 4 (1985): 620, DOI: 10.1017/S0140525X00045337.
[14] Ibid., p. 620-622; Cornel, "An even-handed debate?", p. 138-139.
[15] Alexandre Jehan Marcori and Victor Hugo Alves Okazaki, "A historical, systematic review of handedness origins," *Laterality* 25, no. 1 (2020): 102. DOI: 10.1080/1357650X.2019.1614597. (Admitting to ambidextrousness seems even more problematic, if only "lower animals" had fully symmetrical brains.)

objectivity, and the conscious self.[16] Largely due to a theory of complementarity, the right hemisphere became the location of animality, emotion, madness, femininity, nonwhite inferiority, subjectivity, and the unconscious or subliminal self.[17] The twentieth century complicates this picture for one key reason: visuospatial reasoning was assigned to the right hemisphere. With the rise of science and engineering as occupations only for the mathematically literate, it was now the right hemisphere associated with masculine spatial superiority and the left hemisphere associated with feminine verbal faculty.[18] What this meant for the other associations varied: some, like "objective" and "subjective", appear to have remained fully intact; others, like "realistic" versus "impulsive", can be reworked into a different gendered paradigm that gives women more self-control than men;[19] and finally, still others, such as "abstract" versus "concrete", reworked older dichotomies ("reason" and "madness") to less judgmental versions.[20] This insistence on a difference between the hemispheres, even if it was no longer so clear what it was (or what it meant), also remained gendered and handed in part because of a quirk of left-handedness: men are more frequently left-handed than women.[21] The argument was no longer that men were (or should be) left-lateralized, but that lateralization in and of itself was a masculine trait that conferred higher performance.[22]

[16] Harrington, "Nineteenth-Century Ideas on Hemisphere Differences," p. 622.
[17] Ibid.
[18] Ibid.
[19] Culturally linked to the phrase "boys will be boys", the belief that girls mature faster, and higher diagnosis rates of boys with ADHD.
[20] Harrington, "Nineteenth-Century Ideas on Hemisphere Differences," p. 622; Fausto-Sterling, Chapter 5: Sexing the Brain: How Biologists Make a Difference" in *Sexing the Body*. I say "less" because "abstract vs. concrete" is not a fully non-judgmental comparison; abstract thinkers would generally be considered "too brilliant to deal with practical matters" if they are incapable of thinking concretely, while concrete thinkers would be considered "incapable of seeing the bigger picture".
[21] Cornel, "An even-handed debate?", p. 138-139.
[22] Fausto-Sterling, Chapter 5: Sexing the Brain: How Biologists Make a Difference" in *Sexing the Body*.

To loop back to the corpus callosum and integrate these points, some of the research during 2000 to 2019 suggested, among other propositions, that women had larger and more developed corpus callosi, that homosexual men were more likely to be left-handed, that both left-handedness and homosexuality arose from deviations in normal development, that women and the left-handed were less lateralized overall, and, with the advent of the fMRI, that language in particular was more or less likely to be lateralized as both a function of sex and as a function of timing over the menstrual cycle. While the explicit history given above was rarely referenced beyond an occasional mention of Broca, the ground covered above may explain—or at least provide some context for—a few of the base assumptions represented across the research. The first assumption was that function and ability followed structure. The larger relative size of the corpus callosum (see Fig. 3) in females[23] was assumed to lead to more or perhaps better use of the corpus callosum, which in turn explained both the decreased language lateralization in women, particularly high-estrogen women, and women's higher linguistic abilities overall. The second assumption was that homosexual men and left-handed individuals suffered from some developmental masculinizing deficiency which made them more female-esque in sexual orientation and functional lateralization respectively. This idea can be expressed as "female as

[23] Debatably, in both MRI and anatomical studies; Fig. 3. See Fausto-Sterling, "Chapter 5: Sexing the Brain: How Biologists Make a Difference" in *Sexing the Body*.

default", which is aligned with ideas of the female mind being less developmentally "human", covered above.

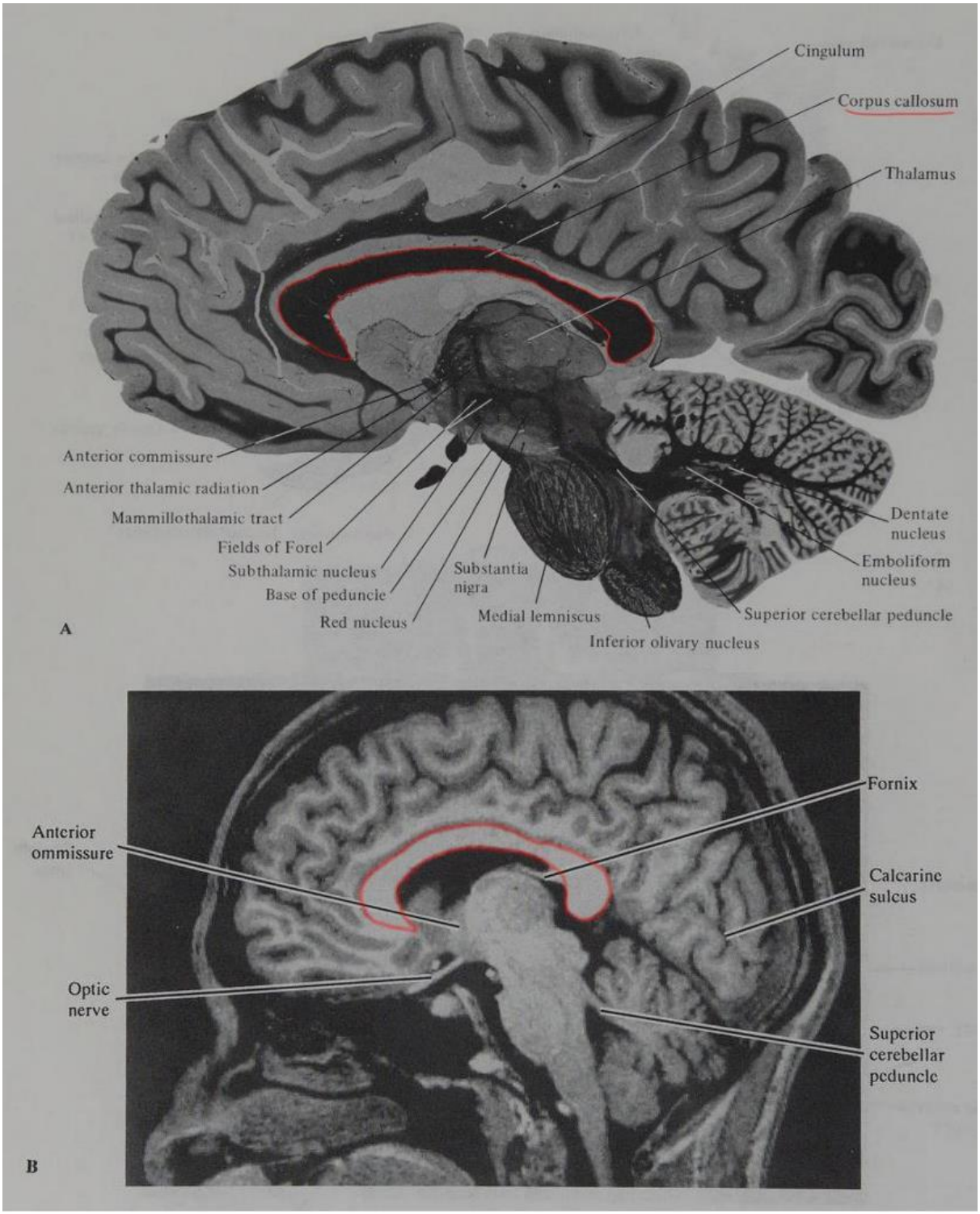

Figure 3. These images are from a 1995 guide to dissection and neuroanatomy, representing roughly the state of knowledge and scanning ability in 2000. (A) shows a dissected brain stained with a reagent that attaches to myelin, a substance which surrounds the axons of some (but not all) neurons in vertebrates. The varying levels of stain reflect the varying levels of myelinated neurons in each area. The corpus collosum is visible as a particularly darkly stained area in this slice through slightly off the center of the brain along a front-back axis. (B) shows roughly the same "slice" in an MRI scan of a living subject. In the MRI, the corpus callosum is defined by the slight darkness separating it from the cerebrum (gray matter) as well as by part of the ventricles of the brain (frontal horn of right ventricle), a black area on the MRI. In both (A) and (B), the corpus collosum has been outlined in red in an image editor. While the corpus callosum is clearer in the dissection than in the MRI, the shape of the brain overall and the corpus callosum specifically is different between examples due to the absence of the skull and spinal fluid in the dissected brain. Additionally, while an MRI would give a living 'slice' of the brain, the corpus callosum was difficult to distinguish from surrounding matter, particularly towards the front of the brain at the underside of the 'hook'. This outline was done to the best of my ability, but is likely inexact. However, as Fausto-Sterling noted in her discussion of attempts to calculate volumes of corpus callosi, a number of these issues are not unique to my attempt. (Sexing the Body, Ch. 5).
Images are taken from Fig. 5-22, p. 138, from Lennart Heimer,* The Human Brain and Spinal Cord: Functional Neuroanatomy and Dissection Guide*, 2nd ed., Springer-Verlag (New York): 1995, accessed via archive.org. Images were cropped and outlined by author in Autodesk Sketchbook.

Additionally, it is also telling that the "femaleness" of the brain in some way interacted with the circulating hormones of the menstrual cycle, as hormonal changes have been most strongly associated with women for the last two hundred years.[24] The "sex hormones", or "sex steroids", generally include the female hormone(s) estrogen(s) and the male hormones androgens (or the solitary hormone testosterone), with the inconsistent addition of progesterone, or "the

[24] On an interesting note, men also have a testosterone rhythm, but it is daily instead of monthly.

pregnancy hormone".[25] There are, however, a few complications to this picture when examining the biochemistry of these steroids (Fig. 4). Firstly, there are actually four progestogens, five androgens, and three estrogens. Secondly, all sex hormones are enzymatically created from cholesterol, which is ubiquitous throughout the body but concentrated in the brain. Thirdly, cholesterol is first made into a progestogen before becoming a corticoid (generally metabolic or inflammatory hormones)[26] or an androgen. Fourthly, androgens can either be made into an irreducible androgen, dihydrotestosterone (DHT) or into an estrogen. Most notably, estradiol and testosterone are separated by the single enzyme aromatase.[27] In addition to noting the chemical similarities between hormones, it is also worth noting that hormones must activate biological pathways through receptors or other cellular responsive mechanisms, after which they can alter the immediate or long-term behavior of cells, organs, and the full organism.[28]

[25] Etymologically, the word "progesterone" derives from "pro-", as in supporting, "gestation", as in pregnancy, and "-one", which is derived from the German word for sterol, "steron".

[26] This alternative avenue for progestogens is relevant for congenital adrenal hyperplasia (CAH), a relatively common cause of ambiguous genitalia in those with XX chromosomes. If an enzyme in the corticoid pathways is nonfunctional or inconsistently functional, the adrenal glands will produce excess progesterone, androgens, and estrogens as a side effect of hyperactivation of the pathways to create corticoids. Corticoids are, among other things, required for stable blood pressure and salt levels, and so the human body tends to prioritize their production.

[27] Removing a methyl group with a few bond rearrangements to make the first carbon ring into a benzene ring. This particular chemical rearrangement did end up being *slightly* a problem in other research, when hormones far in excess of physiological levels were added to model animals or petri dishes to study aging, strokes, and Alzheimer's through cell degradation. Benzene rings are fairly good at staying stable when encountering reactive oxygen species; the other types of carbon ring structures are not. That might be enough for estrogens, particularly at unnaturally high levels, to be better at halting or slowing a chain of mitochondrial decay/failure and cell death than androgens in a way that might or might not translate into bodily systems functioning relatively normally. Beyond this footnote, it was not worth getting into, though, as the other problematic assumptions are either more fundamental or make this issue somewhat irrelevant. See chapters 2 and 3.

[28] The details of this will be covered more thoroughly in chapter 2.

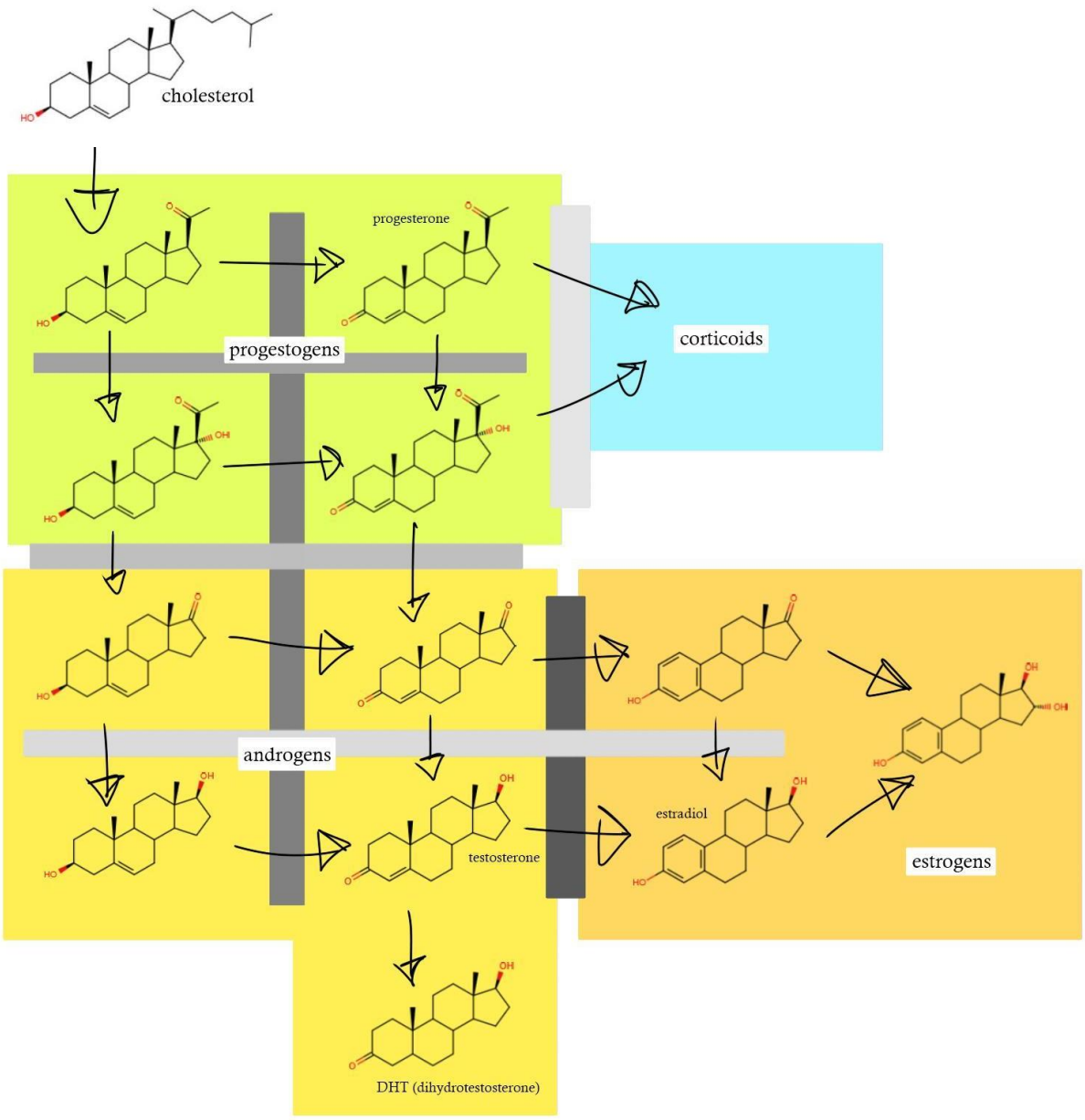

Figure 4. The synthesis pathways of the different sex hormones are depicted here. The progestogens have a green background, the androgens a yellow background, and estrogens an orange background. The different corticoids are collapsed into a single box (blue) for visual convenience. The arrows represent synthesis steps, almost always (except for estriol, the rightmost estrogen) with a single enzyme. Any arrows passing through the same box are using the same enzyme; e.g., the step that makes androgens into estrogens is through the enzyme aromatase regardless of the type of the oxygen group at the other end of the molecule. Arrows without boxes have enzymes not shared with other steps in this pathway. Progesterone,

testosterone, dihydrotestosterone (DHT), and estradiol are individually labelled. Figure generated by the author using the PDB Chemical Sketch Tool and Autodesk Sketchbook.

Hormones were believed, in 2000, to primarily work through genetic mechanisms; that is, to bind to a receptor which in turn caused (or partially caused) some set of genes to be made into messenger RNA (mRNA), known as transcription, and then into proteins, known as translation.[29] The Human Genome Project, which completed its first publicly available draft in 2001, is a foundational event in human genomics. Since then, genetic sequencing has only become simpler, cheaper, and faster,[30] which has allowed geneticists, biochemists, molecular biologists, and cell biologists to begin the task of understanding the vast and complex network of ways that genes are regulated.[31] The word "gene" is not an unquestioned term,[32] but its current practical definition is, roughly, "a DNA sequence which can be made into (part of) at least one polypeptide chain, or protein, or at least one RNA sequence which acts within the cell or organism." Genes, in this framework, include 'traditional' genes which can be transcribed and translated into proteins, as well as sequences such as the ribosomal RNA (rRNA) and the transfer RNA (tRNA) genes, but do not include, for example, mRNAs, micro RNAs (miRNAs), or long non-coding RNAs (lncRNAs), which are generally considered "sequences".[33]

[29] By 2000, there was also some evidence that hormones could work through "rapid" mechanisms, or mechanisms which took place far more quickly than DNA transcription into mRNA and then translation into proteins, though the evidence at that time was indirect. See §2.1 and 2.2.

[30] Alice Maria Giani, Guido Roberto Gallo, Luca Gianfranceschi, and Giulio Formenti, "Long walk to genomics: History and current approaches to genome sequencing and assembly." *Computational and Structural Biotechnology Journal 18*: 2020, p. 9-19; DOI: 10.1016/j.csbj.2019.11.002.

[31] Knowledge that genes *were* regulated dates back to the 1950s and the discoveries of Barbara McClintock about genetic transposition and "controlling elements" in maize, as well as Jacob and Monod's characterization of the *lac* operon in *E. coli* in the 1960s. The details about genetic regulation, however, such as frequency, type, mechanism, and which mechanisms control the expression of which protein-coding sequences, are an area of active investigation.

[32] Petter Portin and Adam Wilkins, "The Evolving Definition of the Term 'Gene'." *Genetics 205*(4): April 2017, p. 1353-1364; DOI: 10.1534/genetics.116.196956.

[33] The distinction appears to be "acts within the cell"; miRNAs appear to have functions in mRNA transcript regulation and lncRNAs in DNA regulation, but tRNAs and ribosomes (including the rRNA in them) have semi-permanent existences within the cell. This is, however, a gray area I am trying to simplify.

Gene regulation was an ongoing area of investigation during this period, evidenced by the creation of the field of epigenetics in approximately[34] 2008. An influential biological[35] definition of epigenetics includes all inheritable changes to gene expression which do not originate with changed genetic sequences.[36] To explain the slight issue with that definition, there are multiple ways that genes' expression can be regulated. Firstly, there is the activation or repression of transcription into mRNA, which the hormonal ligand receptors (estrogen receptors, etc.) can accomplish by binding specific sequences within the nuclear DNA. Secondly, there is RNA-based regulation, by either DNA-RNA interactions such as the silencing of X chromosomes by layered *Xist* RNA, or by RNA-RNA interactions, for example by miRNAs, which prevent certain mRNAs from being translated into proteins. Thirdly, there are the modifications to the DNA itself or the histones, proteins the DNA is wrapped around, by attaching chemical groups, like methyl groups or acetyl groups, that either 'open up' or condense the DNA, allowing or preventing transcription of certain areas. Fourthly, the RNA transcripts are modified between transcription and translation, removing some of the non-coding sequences and sometimes creating multiple alternate transcripts of the same gene by selectively 'splicing' different coding sequences together. Fifth, the cell may sequester RNA sequences to (temporarily) prevent their translation. Finally, the translation of the mRNA into protein by the ribosomes can be hindered or assisted by the shape of the mRNA or various cellular elements. Additionally, there are a

[34] There are precursors and the dating is somewhat in the air; 2008 is the date of the Cold Spring Harbor conference referenced in ft. 36.
[35] There are a handful of other definitions from psychology which share a focus on development; namely, that each 'stage' of life (infancy, childhood, adolescence, etc.) forms the basis for the next, and that the circumstances of one's life formed the 'epigenetic' influences for the later developmental stage. See, for the oldest example, Erik Erikson, *Identity, youth, and crisis*. W.W. Norton (New York): 1968, accessed through the Internet Archive; p. 92-96, 105-107.
[36] Shelley L. Berger, Tony Kouzarides, Ramin Shiekhattar, Ali Shilatifard, "An operational definition of epigenetics." *Genes and Development 23*(7): 1 April 2009, p. 781-783; DOI: 10.1101/gad.1787609.

number of ‘modifications’ which are DNA-protein crosslinks, or proteins covalently bonded to sequences of DNA which prevent their translation or replication. Epigenetics as a subject usually includes the DNA-RNA interactions, or at least those which remain consistent within cell lines like *Xist*, and the chemical modifications to the histones and DNA which allow or prevent access to the DNA, which can in theory be inherited even if they do not last long enough to be inherited, but can include all of the above changes to gene expression if epigenetics includes all of the different effects which can alter gene expression, or additionally include, for example, elements in the cytoplasm of both daughter cells after mitosis that are inherited and can affect gene expression.[37]

Another subject that appears deceptively simple is the place of evolution by natural selection. Evolutionary theory, in its simplest form, begins with pre-existing variation between individuals. Based on the interplay between inherent (genetic, epigenetic, etc.) and environmental (nutrition, climate, etc.) factors, the individuals will develop different phenotypes,[38] which then will, on average, have different reproductive success. Those individuals with more reproductive success will pass their traits on and, in a population, eventually all members of that population will possess the trait(s) that increase reproductive success. This description of evolutionary theory centers selection, including sexual selection, and adaptation. Evolutionary biologists, however, ask questions about specific traits in specific organisms. At the molecular level, it is not clear that much or even most variability is directly

[37] Carrie Deans and Keith A. Maggert, “What Do You Mean, ‘Epigenetic’?” *Genetic 199*(4): April 2015, p. 887-896; DOI: 10.1534/genetics.114.173492.
[38] C. Kenneth Waters, “A Pluralist Interpretation of Gene-Centered Biology.” *Minnesota Studies in the Philosophy of Science*, Vol. 19: Scientific Pluralism, Ed. Kellert, Stephen H.; Longino, Helen E.; Waters, C. Kenneth. University of Minnesota Press (Minneapolis): 2006, p. 190-214.

selected upon, being either neutral or nearly neutral.[39] One model of the development of molecular complexity additionally suggests that nearly-neutral changes over time in cellular factors become the fodder for future creative or additive evolution, which suggests that at least some traits may go through a period of being "invisible" to selective pressures before conditions change and the trait becomes relevant.[40] Traits can additionally be pre-constrained (e.g. vertebrates descended from tetrapods have four or fewer limbs because the fish that tetrapods evolved from had four lobed fins)[41] or otherwise fundamentally limited by tradeoffs (e.g. size of antlers of elk reach a maximum survivable size),[42] be tangentially the result of selection (byproduct traits or genetic linkage),[43] or be significantly affected by chance variation in population genetic variance, such as by genetic drift[44] or founder effects.[45] Different groups within and outside of evolutionary biology put different amounts of emphasis on the likely causes of traits in general.[46] The literature covered by this thesis focused mostly, though not

[39] Tomoko Ohta, "The Nearly Neutral Theory of Molecular Evolution," *Annual Review of Ecology and Systematics 23*: 1992, p. 263-286; jstor.org/stable/2097289. Kimura, *The Neutral Theory of Molecular Evolution*. Jeffrey D. Jensen, Bret A. Payseur, Wolfgang Stephan, Charles F. Aquadro, Michael Lynch, Deborah Charlesworth, and Brian Charlesworth, "The importance of the Neutral Theory in 1968 and 50 years on: A response to Kern and Hahn 2018," *Evolution 73*(1): January 2019, p. 111-114; DOI: 10.1111/evo.13650.
[40] Michael W. Gray, Julius Lukeš, John M. Archibald, Patrick J. Keeling, W. Ford Doolittle, "Irremediable Complexity?" *Science 330*(6006): 12 November 2010; p. 920-921; DOI: 10.1126/science.1198594.
[41] Stephen Jay Gould, "The Evolutionary Biology of Constraint." *Daedalus 109*(2): Spring 1980, p. 44; jstor.org/stable/20024665.
[42] Randolph M. Nesse, "Maladaptation and Natural Selection." *Quarterly Review of Biology 80*(1): March 2005, p. 62-70; DOI: 10.1086/431026.
[43] Elisabeth A. Lloyd, "What a Difference Research Questions Can Make!" *Proceedings and Addresses of the American Philosophical Association 90*: November 2016, p. 129-153; jstor.org/stable/26622942.
[44] R. B. O'Hara, "Comparing the Effects of Genetic Drift and Fluctuating Selection on Genotype Frequency Changes in the Scarlet Tiger Moth." *Proceedings of the Royal Society: Biological Sciences 272*(1559): 22 January 2005, p. 211-217; DOI: 10.1098/rspb.2004.2929.
[45] Jason J. Kolbe, Manuel Leal, Thomas W. Schoener, David A. Spiller, Jonathan B. Losos, "Founder Effects Persist Despite Adaptive Differentiation: A Field Experiment with Lizards." *Science 335*(6072): 2 March 2012, p. 1086-1089; DOI: 10.1126/science.1209566.
[46] For an old critique of adaptationist-centered positions, see S. J. Gould and R. C. Lewontin, "The Spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme." *Proceedings of the Royal Society of London, Series B: Biological Sciences 205*(1161): 21 September 1979, p. 581-598; jstor.org/stable/77447. Cf. Mark E. Olson and Alfonso Arroyo-Santos, "How to Study Adaptation (and Why To Do It That Way)." *The Quarterly Review of Biology 90*(2): June 2015, p. 167-191; DOI: 10.1086/681438, but see also Lloyd, "What a Difference Research Questions Can Make!".

exclusively, on genetic or otherwise innate causal mechanisms for traits that had been selected for and represented adaptations, though not always to the current environment.[47]

This focus on adaptationist explanations, sometimes conceptualized through the "inclusive fitness model", in behavioral neuroendocrinology literature during this period may have been due to the theoretical framework of evolutionary psychology. Evolutionary psychology, at least according to many of its early proponents[48] and observers,[49] "is simply the scientific study of thought, emotion, and behavior, in light of a general adaptationist framework."[50] Evolutionary psychology has received significant criticism from numerous perspectives:[51] anthropological,[52] philosophical,[53] historical,[54] political,[55] psychological,[56] and biological.[57] However, critics, proponents, and observers agree that this adaptationist assumption

[47] This will be discussed further in chapter 4.
[48] David M. Buss, "Evolutionary Psychology: A New Paradigm for Psychological Science." *Psychological Inquiry 6*(1): 1995, p. 1-30; jstor.org/stable/1449568.
[49] Andrew Neher, "Evolutionary Psychology: Its Programs, Prospects, and Pitfalls." *The American Journal of Psychology 119*(4): Winter 2006, p. 519; DOI: 10.2307/20445363.
[50] Douglas T. Kenrick, "Evolutionary Psychology: Resistance is Futile." *Psychological Inquiry 17*(2): 2006, p. 102; jstor.org/stable/20447308.
[51] Some of these criticisms will be covered in more detail in chapter 4.
[52] Giordana Grossi, "A module is a module is a module: evolution of modularity in Evolutionary Psychology." *Dialectical Anthropology 38*(3): September 2014, p. 333-351; DOI: 10.1007/s10624-014-9355-0. Alison Nash, "Are stone-age genes created out of whole cloth? Evaluating claims about the evolution of behavior." *Dialectical Anthropology 38*(3): September 2014, p. 305-332. DOI: 10.1007/s10624-014-9354-1.
[53] Stuart Silver, "Methodological and Moral Muddles in Evolutionary Psychology." *Journal of Mind and Behavior 31*(1, 2): Winter-Spring 2010, p. 65-83; URL: jstor.org/stable/43854267. Subrena E. Smith, "Is Evolutionary Psychology Possible?" *Biological Theory*: 05 December 2019, p. 1-11; DOI: 10.1007/s13752-019-00336-4. Letitia Meynell, "Evolutionary Psychology, Ethology, and Essentialism (Because What They Don't Know Can Hurt Us). *Hypatia 27*(1): Winter 2012, p. 3-27; jstor.org/stable/41328895.
[54] Jonathan Michael Kaplan, "Historical Evidence and Human Adaptations." *Philosophy of Science 69*(S3): September 2002, p. S294-S304; DOI: 10.1086/341853. Gowri Parameswaran, "Are evolutionary psychology assumptions about sex and mating behaviors valid? A historical and cross-cultural exploration." *Dialectical Anthropology 38*(3): September 2014, p. 353-373; DOI: 10.1007/s10624-014-9356-z.
[55] Laurette T. Liesen, "Women, Behavior, and Evolution: Understanding the Debate between Feminist Evolutionists and Evolutionary Psychologists." *Politics and the Life Science 26*(1): March 2007, p. 51-70; DOI: 10.2990/21_1_51.
[56] Neher, "Evolutionary Psychology: Its Programs, Prospects, and Pitfalls," p. 519. Christine R. Harris, "A Review of Sex Differences in Sexual Jealousy, Including Self-Report Data, Psychophysiological Responses, Interpersonal Violence, and Morbid Jealousy." *Personality and Social Psychology Review 7*(2): 2003, p. 102-128; DOI: 10.1207/S1532795PSPR002_102-128.
[57] Elisabeth A. Lloyd and Marcus W. Feldman, "Evolutionary Psychology: A View from Evolutionary Biology." *Psychological Inquiry 13*(2): 2002, p. 150-156; jstor.org/stable/1449175.

is central to those who name themselves evolutionary psychologists. Specifically, evolutionary psychologists assume that human behavior is largely the result of evolutionary processes acting on the brain to generate specific behavioral patterns via fitness-based selection, particularly patterns concerned with learning and memory, language, social interactions, and adaptations to ancestral conditions. To refer to an earlier paragraph, evolutionary psychology also tended to accept modular hypotheses of the brain—that is, much like left- or right-hemisphere containing verbal or spatial reasoning, specific areas of the brains were believed to individually cause or create certain behaviors,[58] though which area was involved might or might not be directly hypothesized. This framework is a subset of the broader argument that the shape and development of the human brain were and are subject to the forces of biological evolution,[59] but the assumed adaptationist explanations of modular behaviors developed in an ancestral social and physical environment is largely[60] unique to the adaptationist evolutionary psychology.

It is worth stating at this juncture that sex differences, specifically their modular evolution in an ancestral period, were an essential component of adaptationist evolutionary psychology.[61] Additionally, adaptationist evolutionary psychology, as outlined by Andrew Neher

[58] Smith, "Is Evolutionary Psychology Possible?", 2019, p. 2. Meynell, "Evolutionary Psychology, Ethology, and Essentialism," 2012, p. 9-10.
[59] Smith, "Is Evolutionary Psychology Possible?", 2019, p. 10. Meynell, "Evolutionary Psychology, Ethology, and Essentialism," 2012, p. 5. Lloyd and Feldman, "Evolutionary Psychology: A View from Evolutionary Biology," 2002, p. 150-155.
[60] Meynell asserts that the assumptions from the narrower evolutionary psychology affect the space of theorizing (and the popular media) even in those who do not accept some of those assumptions. "Evolutionary Psychology, Ethology, and Essentialism," 2012, p. 11.
[61] This may be at least partially incidental; David Buss, cited here and in ft. 42, both enthusiastically promoted adaptationist evolutionary psychology and did a lot of research on mate choice in humans. Buss, "Evolutionary Psychology: A New Paradigm," 1995. Kenrick, "Evolutionary Psychology: Resistance is Rutile," 2006. Neher, "Evolutionary Psychology: Its Programs, Prospects, and Pitfalls," 2006, p. 526-528, 537-538. Grossi, "A module is a module is a module," 2014, p. 338. Liesen, "Women, Behavior, and Evolution," 2007. Silvers, "Methodological and Moral Muddles," 2010, p. 67-68. 71-74. Nash, "Evaluating claims about the evolution of behavior," 2014, p. 317-322. Smith, "Is Evolutionary Psychology Possible?", 2019, p. 7-9. Meynell, "Evolutionary Psychology, Ethology, and Essentialism," 2012. Parameswaran, "Are evolutionary psychology assumptions about sex and mating behaviors valid?", 2014. Harris, "A Review of Sex Differences in Sexual Jealousy," 2003.

in 2006, possesses many of same the meta-theoretical practices as those researching the organizational/activational theory: namely, demonstrations that infants possess a behavior, correlations between biological (genetic or hormonal) differences and cognitive or behavioral differences or deficits, arguments that traits are universal across cultural environments, and attempts to find evidence of behaviors the larger theoretical superstructure suggests should exist.[62] Adaptationist evolutionary psychology and the organizational/activational theory broadly shared many of the same assumptions. Both, for example, assumed that there are universal, consistent, and ancestral cognitive types, often emerging from adaptationist-type evolutionary pressures, which are innate and not (or only somewhat) subject to social or cultural factors. In general, those researching organizational/activational theory existed at a multidisciplinary crossroads, between neuroscience, cell biology, genetics, developmental biology, psychology, cognitive science, evolutionary biology, organic chemistry, behavioral sciences, medicine,[63] and zoology,[64] among others. It is, however, notable that in each case, those researching organizational/activational theory tended to rely on information and theories from those fields which reinforced that there were definite, innate, and universal types of brains, rather than focusing on interindividual variation, indefiniteness, or mechanical complexity.

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[62] Neher, "Evolutionary Psychology: Its Programs, Prospects, and Pitfalls," 2006, p. 551-554. The paper Meynell, "Evolutionary Psychology, Ethology, and Essentialism," 2012, additionally contains an analysis of a paper also covered in the literature in *Hormones and Behavior*, part of a minor debate about gendered toys, male and female play styles, and monkeys. Christina L. Williams and Kristen E. Pleil, "Toy story: Why do monkey and human males prefer trucks?: Comment on "Sex differences in rhesus monkey toy preferences parallel those of children" by Hassett, Siebert and Wallen," *Hormones and Behavior 54*(3): August 2008, DOI: 10.1016/j.yhbeh.2008.05.003, p. 355-358. Janice M. Hassett, Erin R. Siebert, and Kim Wallen. "Sex differences in rhesus monkey toy preferences parallel those of children," *Hormones and Behavior 54*(3): August 2008, DOI: 10.1016/j.yhbeh.2008.03.008, p. 359-364. Melissa Hines and Gerianne M. Alexander, "Monkeys girls boys and toys: A confirmation: Letter regarding 'Sex differences in toy preferences: Striking parallels between monkeys and humans," *Hormones and Behavior 54*(3): August 2008, p. 478-479; DOI: 10.1016/j.yhbeh.2008.05.012. Kim Wallen and Janice M. Hassett, "Determining preference requires measuring preference," *Hormones and Behavior 54*(3): August 2008, DOI: 10.1016/j.yhbeh.2008.05.014, p. 480-481.

[63] Covered in more detail in Chapter 4.

[64] Covered in more detail in Chapter 3.

Finally, organizational/activational theory possessed the inertia of history in 2000. During the second half of the twentieth century, primarily following the work of the psychologist John Money, was that gender was primarily determined by how the parents responded to their child and how the child considered themselves—which the nature of the external genitalia importantly affected.[65] There was no inborn sex—or at least, if there was, the sex could be overwritten by how the child was raised. Therefore, Money convinced the parents of an XY infant who had had their penis removed after a circumcision-related injury to raise the child as a girl; Money compared the new girl to her twin brother, who served as a control.[66] His main opponent was Milton Diamond, who "pursued Money with a determination worthy of Inspector Javert in *Les Misérables*".[67] Diamond eventually tracked down the XY child raised as a girl and discovered that he had undergone gender affirming surgeries and treatments and lived as a man: David Reimer. Unfortunately, both David and the "control" twin, Bruce, committed suicide in the early 2000s, which their parents blamed on the stress which Money's gender therapy had placed on the two.[68] Their suicides painfully emphasized to observers that Money had been wrong and, combined with the testimonies about the abuses and pointlessness of conversion therapies[69] and the calls of intersex activists, convinced most scientists and doctors that sexualities, gender roles, and sexes/genders were set at birth.[70]

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[65] Fausto-Sterling, "Of Gender and Genitals: The Use and Abuse of the Modern Intersexual."
[66] Fausto-Sterling, "Of Gender and Genitals: The Use and Abuse of the Modern Intersexual."
[67] Fausto-Sterling, "Of Gender and Genitals: The Use and Abuse of the Modern Intersexual."
[68] Other factors do appear to have played a role in their suicides: Bruce had schizophrenia and David had recently lost Bruce, faced employment issues, and had separated from his wife. That being said, Bruce and David both alleged sexual, mental, and physical abuse at Money's hands, (As Nature Made Him) which, in addition to the painful gender dysphoria David suffered as a teenager before his parents revealed his medical history, likely did negatively affect both well into adulthood.
[69] For both gender and sexuality.
[70] It is true that LGBTQIA+ activists said and say "born this way"; this, however, can equally be explained as a strategic move to attempt to prevent abuse in contexts where murder is generally unacceptable. If an intersex infant, or indeed any infant, has a gender identity that is inalterable, the path which does no harm is to wait for the infant to

Diamond was not, however, simply any researcher. Diamond's thesis research[71] was done with William C. Young,[72] the most important of the four authors[73] of the 1959 paper which first hypothesized organizational/activational effects.[74] Indeed, Diamond later credited the hypothesis of "the forces of organization and activation… by Phoenix and colleagues" that he believed "held as well for all other mammals including humans and would follow evolutionary forces predisposing them in their functioning sexually as males or females" with justifying his initial challenge to Money in 1965.[75] Diamond was not simply arguing against Money. Diamond was arguing for a theory of sex/gender which asserted inborn distinctions between male and female that emerged from evolutionary forces which predisposed males and females to have different sexualities and sexed/gendered roles—the organizational/activational theory.

grow up enough to tell those around them what their gender is—rather than sex-normalizing surgery for intersex infants or gender conversion therapy for transgender children. If sexuality is inalterable, there is no point in trying to change it—rather than subjecting homosexual, bisexual, or asexual youths and adults to gay conversion therapy or medical intervention to attempt to generate sexual desire. If the possibility of changing sex/gender or sexuality remains open after birth, some individuals will convince themselves that they have lit upon the "right" way to "normalize" people. John Money, in fact, is an excellent example of this behavior: he helped transgender adults medically transition (based on his belief that the psychological and sexual behavior could not be altered in adulthood but bodies could) in part because he believed that the "right" way to "normalize" all people was to rigidly and consistently enforce sexes of rearing in early childhood with the eventual end goal that there would never need to be transgender people again. See Elizabeth Reis, *Bodies in Doubt: An American History of Intersex*, 2nd edition, John Hopkins University Press (Baltimore): 2021, particularly "Chapter 5: Psychology, John Money, and the Gender of Rearing in the 1940s, 1950s, and 1960s," p. 115-152.

[71] "Pacific Center for Sex and Society: Bibliography", *University of Hawai'i Mānoa*, URL: hawaii.edu/PCSS/biblio/index.html, last updated 20 May 2017, accessed 28 January 2024.

[72] Milton Diamond and William C. Young, "Differential Responsiveness of Pregnant and Nonpregnant Guinea Pigs to the Masculinizing Action of Testosterone Propionate," *Endocrinology 72*(3): 1 March 1962, DOI: 10.1210/endo-72-3-429; p. 429-438.

[73] Arnold A. Gerall, another author of the original 1959 paper, argued later that Young originally generated the organizational/activational theory. Arnold A. Gerall, "Recollections of the origins of and reactions to the organizational concept," *Hormones and Behavior 55*(5): May 2009, DOI: 10.1016/j.yhbeh.2009.03.001; p. 567-569.

[74] Phoenix *et al.*, "ORGANIZING ACTION OF PRENATALLY ADMINISTERED TESTOSTERONE PROPIONATE."

[75] Milton Diamond, "Clinical implications of the organizational and activational effects of hormones," *Hormones and Behavior 55*(5): May 2009, DOI: 10.1016/j.yhbeh.2009.03.007, p. 622.

1.3: The Literature

As is evident from the above background information, behavioral neuroendocrinology springs directly from neuroscience, endocrinology, and psychology, but draws from medicine, biology, and chemistry as well as a handful of the social sciences. This multidisciplinary milieu is fruitful for research, but frustrating for a historian and philosopher of science attempting to isolate a representative sample. To glimpse the field as a whole, I chose the scientific journals *Hormones and Behavior*, *Frontiers in Neuroendocrinology*, and the *Journal of Neuroscience*, with the addition of the *eNeuro* journal after 2014. *Hormones and Behavior* is one of the oldest and most influential journals in behavioral endocrinology, and the *Journal of Neuroscience* publishes an incredible volume of peer-reviewed neuroscientific literature. The prestige and sheer number of issues from both journals provides a representative sample of their fields. Far less frequent and more specialized, *Frontiers in Neuroendocrinology* unites neuroscience and endocrinology, while also promising cutting-edge research. Finally, I decided to incorporate the Society for Neuroscience's online, open access (but still peer-reviewed) journal *eNeuro*, because it publishes replication studies and negative results.

	Hormones and Behavior	*Frontiers of Neuroendocrinology*	*Journal of Neuroscience*	*eNeuro*	**Total**
2000	2	2	3	0	**7**
2001	13	0	9	0	**22**
2002	8	1	13	0	**22**
2003	3	1	10	0	**14**
2004	7	1	8	0	**16**
2005	9	5	8	0	**22**
2006	17	7	24	0	**48**
2007	4	1	18	0	**23**
2008	9	17	20	0	**46**
2009	24	14	15	0	**53**
2010	26	3	10	0	**39**
2011	25	12	9	0	**46**
2012	13	6	11	0	**30**

	Hormones and Behavior	*Frontiers of Neuroendocrinology*	*Journal of Neuroscience*	*eNeuro*	**Total**
2013	30	10	12	0	**52**
2014	10	18	10	1	**39**
2015	12	8	10	1	**31**
2016	7	17	17	9	**50**
2017	16	15	16	11	**58**
2018	12	16	13	11	**52**
2019	10	11	12	14	**47**
2020	*4*	*2*	*3*	*3*	***12***
Total	**274**	**167**	**251**	**51**	**729**

Table 1. Papers divided by journal and year.

Between these four journals, I have gained sufficient breadth, specialization, timeliness, and variation to provide an accurate view of the field of behavioral endocrinology overall during this period. Having isolated the journals of interest, a brief skim through titles and occasionally abstracts generated a list of 1,159 papers, which were winnowed to 729 papers upon closer reading. While the research period was limited to 2000-2019, a few papers in 2020 were included due to their editorial or retrospective nature. A citation list of the papers, organized by year, is contained in Appendix 1. Table 1 contains the number of papers included in the final group by journal and by year.

	Research	*Reviews*	*Editorial changes*	*Commentary*	*Abstracts*	**Total**
2000	6	1	0	0	0	**7**
2001	11	10	1	0	0	**22**
2002	16	4	2	0	0	**22**
2003	12	2	0	0	0	**14**
2004	14	2	0	0	0	**16**
2005	16	5	1	0	0	**22**
2006	24	14	2	5	3	**48**
2007	18	3	1	1	0	**23**
2008	22	17	2	5	0	**46**
2009	22	20	1	10	0	**53**
2010	22	12	2	3	0	**39**
2011	19	23	0	4	0	**46**
2012	17	11	0	2	0	**30**
2013	23	23	1	5	0	**52**

	Research	*Reviews*	*Editorial changes*	*Commentary*	*Abstracts*	**Total**
2014	17	19	1	2	0	**39**
2015	15	11	1	4	0	**31**
2016	15	16	11	8	0	**50**
2017	25	20	9	5	0	**59**
2018	16	21	7	8	0	**52**
2019	22	16	2	6	0	**46**
2020	*0*	*1*	*1*	*10*	*0*	***12***
Total	**352**	**251**	**45**	**78**	**3**	**729**

Table 2. Papers divided by type and year.

The papers were chosen by perceived relevance to the topics of 'male and female brains', 'sex differences', 'sex dimorphism', 'sex similarities,' and 'organizational/activational theory', and further narrowed by relevance to human behavioral neuroendocrinology specifically, although studies on model organisms with declared relevance to humans were also included. Additionally, papers that discussed research practices, journal practices, or editorial changes were also read and added to the total. Table 2 provides a breakdown of all papers examined by type and by year. "Research" papers were papers focused on reporting the testing of a specific hypothesis through between one and four tests, while "Reviews" summarized existing research on a topic, often taking a position on what consensus follows from the research or what further research should be done but mostly intended as summaries. "Editorial changes" were papers changing publishing practices at a journal or changing editorial staff. "Commentary" papers discussed trends in the literature, recommended general research practices, or took explicit positions on theories or hypotheses. "Abstracts" contains three abstracts from a conference published in isolation in 2006, included for completeness.

There are a handful of trends visible in these tables I wish to draw attention to. The first is that the years 2000 to 2005 show a relatively low number of publications on sex differences compared to the years that followed, and that there were no commentaries I found in this

literature discussing sex differences, male and female brains, or the organizational/activational theory until 2006. Similarly, while research articles were published relatively consistently, relatively few reviews were published during this early period on the topics that this thesis focuses on, suggesting that research may have been in an exploratory phase.[76] The relative lack of papers on sex differences or the organizational/activational theory in *Frontiers of Neuroendocrinology* before 2005 may also suggest that research on these topics had not diffused through neuroendocrinology enough to be considered a "frontier". The second trend is the sustained increase in commentaries and reviews after 2006.[77] These commentaries and reviews put forward new theories and criticized others' theories or research. For example, puberty became a possible second "organizational" period between 2006 and 2015 rather than a purely "activational" period, discussed more in §2.3, and evolutionary explanations, discussed in §4.1, were proposed most consistently between 2006 and 2015. The final trend, or rather, spike, is the elevated number of editorial changes during 2016, 2017, and 2018. These codifications of and changes to publication practices represents the replication crisis, an event that will be discussed in detail in Chapter 3.

1.4: Philosophical Positions and Prior Literature

> *"They [background assumptions] are neither a priori and indubitable nor a posteriori and open to doubt. In normal conditions, they cannot be the object of any kind of justificatory move. Rather, they constitute quasi-logical, or rather* grammatical,

[76] Supporting this supposition, it is worth recalling that the Human Genome Project's first draft was published in 2001 and fMRIs were relatively new. The change in research patterns may also be related to computational changes, either software or hardware being more accessible or faster, but that is speculation based on how later retrospectives and commentaries characterized changing research practices.

[77] The exception being 2007. I suspect that this may reflect a time-lag of research; reviews and commentaries in 2007 may not have had enough evidence to answer questions or support arguments relating to publications in 2006. This is, again, speculation, and may simply reflect chance.

conditions allowing the concepts involved in the inquiry to make sense. [emphasis in original]"[78]

In 2000, Aristides Baltas put forward a theory of scientific controversy, suggesting that scientific controversies were over different background assumptions[79] held by different groups of scientists. The assumptions could be *surface* assumptions, such as methodological preferences, or *deep* assumptions, such as the nature of the terms in question.[80] As the quote above suggests, I believe "background assumptions" characterizes many of the ideas involved in the changes this thesis engages with. The controversy over 'female' and 'male' brains between 2000 and 2019 and the theory of organization/activation involved assumed answers to questions such as "how much can neurons change" or "what is the standard behavior of female mammals". I therefore assumed that this controversy would mostly take the form of a *deep* controversy in an existing science, dealing with *constitutive* (fundamental to the conceptual system) and *interpretative* (basic to the earlier interpretation of the conceptual system) background assumptions.[81] The question would be to determine how and why previously unstated and unproven assertions about sex/gender, neuroscience, behavior, and biology became illuminated, and which assumptions went without notice.

Additionally, biological theories possess a rather less obvious quirk. To prove a negative is impossible; to prove a negative in biology, doubly so. Individual counterexamples, even numerous counterexamples, can mean little, since strict laws in biology are as rare as hen's teeth.

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[78] Aristides Baltas, "Classifying Scientific Controversies," in *Scientific Controversies: Philosophical and Historical Perspectives*, eds. Peter Machamer, Marcello Pera, and Aristides Baltas, Oxford University Press (Cary): 2000, p. 41, eBook.

[79] In the sense of "idea taken for granted", not the sense of "idea intentionally held with insufficient evidence for the purpose of philosophical or empirical inquiry". See §5.5.

[80] Baltas, "Classifying Scientific Controversies," p. 46-48.

[81] Baltas, "Classifying Scientific Controversies," p. 43, 47-48.

By what metric can any biological model be judged insufficient? What is, in the end, the purpose of a biological model? As Antonio Diéguez has argued, the purpose of biological models is to provide genuine understanding, as understood through the criteria of representational fidelity.[82] Representational fidelity demands models have scientifically founded analogies, a lack of oversimplified abstractions, realistic and useful idealizations, scientific ontologies, analogies between the mechanisms of the system and the mechanisms of the model, and most importantly, "predictions about collateral phenomena [that] do not fail systematically."[83] Put simply, models in biology are attempts to conceive life's complexity without being too wrong in too many important ways.

When considering a typology in biology, the existence of individuals who do not fit within it does not disprove the typology. If chromosomes were destiny, and gonads provided the mechanism by which chromosomes became developmental outcomes, then most humans (and mammals) should reliably be one of two types: XY-male-masculine-gynephilic and XX-female-feminine-androphilic. (Sex-typed behaviors not only included general behaviors but also sexual orientations, i.e. heterosexuality.[84]) Mechanisms might fail; androgen insensitivity might create XY women, for example.[85] One might imagine masculine women or feminine men emerging from insensitivity, underdevelopment, or overexposure in similar fashions. However, the failure

[82] Antonio Diéguez, "When do Models Provide Genuine Understanding, and Why does it Matter?" *History and Philosophy of the Life Sciences 35*, no. 4: 2013, URL: jstor.org/stable/43862216, p. 599-620.
[83] Ibid., p. 612.
[84] To the degree this has historically been justified, it is justified by declaring it "evident" that evolutionary selection pressures push towards heterosexual and thereby child-producing orientations. This is a product of assuming that nuclear, two-parent households producing children of those parents are natural and, separately, that all other familial formations (more or less than two parents, adoption, stepparents, multigenerational households, communal childrearing, etc.) are non-natural.
[85] Androgen insensitivity syndrome is a broader spectrum than simply a 46-XY karyotype and female phenotype, encompassing complete, partial, and mild variants, which may present with male, female, or mixed external genitalia. Complete androgen insensitivity syndrome has the highest likelihood of XY-female-feminine-androphilic for the duration of the person's life.

of those mechanisms (and therefore the model) to function some percentage of the time did not, in and of itself, disprove the existence of two basic types of mammal linked by similar developmental processes.

It is worth noting here that the two types of human animal, XX-female-feminine-androphilic and XY-male-masculine-gynephilic, have never been proven a reliable typology. While XX-female and XY-male are the most common pathways of human development, a significant number of people live outside those strict delineations. Different numbers of sex chromosomes, such as 47-XXY and 45-XO, as well as any nonstandard internal or external genitalia or secondary sex characteristics (facial hair, breasts, etc.), all fit within the broadest definitions of "differences of sexual development". When it comes to the development of human beings, who receive significantly more individual attention than nematode worms or guinea pigs, what precisely counts as "oversimplification" should perhaps be reexamined, given that deviations from XX-female and XY-male are about as common as red hair or green eyes.[86] One might also consider social roles and individuals which cannot be characterized as strictly masculine or feminine, such as non-binary and queer Western communities; the *hijras,*[87] *waria,*[88]

[86] Anne Fausto-Sterling, in *Sexing the Body*, calculated a number of 1.7% of the human population having some type of intersex existence. (Table 3.2) There have been some objections to her categorization, as hypospadias (the urethra does not run to the tip of the penis) and Kleinfelter syndrome (47-XXY) are generally not seen as 'intersex' conditions. To somewhat avoid the debate, I have characterized this group as 'deviations from XX-female and XY-male', but her larger point–that there *isn't* a consistent definition of "intersex", or any of its replacement terms–is accurate. Intersex is often considered "obviously between the sexes", but, to be consistent, the definition *should* be any karyotype/genital/secondary sex feature that is not XX-female standard and XY-male standard. Red hair appears in between 1 and 2 percent of the population (estimates vary); about 2 percent of the population has green eyes (estimates vary). Fausto-Sterling, *Sexing the Body: Gender Politics and the Construction of Sexuality*.

[87] Akiko Kunihiro, "Against Taxonomy and Subalternity: Reconsidering the Thirdness and Otherness of Hijras of Gujarat," *South Asia Multidisciplinary Academic Journal 28*: 2022; DOI: https://doi.org/10.4000/samaj.7819.

[88] Terje Toomistu, "Playground love: sex work, pleasure, and self-affirmation in the urban nightlife of Indonesian waria," *Culture, Health & Sexuality 21*(2): 2019, p. 205-218, DOI: 10.1080/13691058.2018.1459847.

and *nádleehi*;[89] historical roles such as the ancient Sumerian *gala*;[90] as well as the genderless Public Universal Friend from the United States in the late 18th century.[91] Justifying exclusive human heterosexuality seems even more absurd.[92] That "masculine" and "feminine" do not possess consistent non-circular definitions, and that categorical[93] distinctions were not justified at all, provides another level of complexity that this thesis will largely not consider directly but should still be kept in mind. Finally, there is the issue of terminology. I will be somewhat inconsistent in this thesis with the terms "sex", "gender", and "sex/gender" to attempt to capture some of the difficulties of integrating this research after the fact. Researchers generally focused on either "sex", the biological characteristics, or "gender", the social characteristics, but I cannot be consistent because they were not consistent enough for me to be certain which they meant. A more detailed discussion is present in 3.3.

Previous literature on this topic includes *Sexing the Body: Gender Politics and the Construction of Sexuality* by Anne Fausto-Sterling,[94] "Am I My Brain or My Genitals? A Nature-Culture Controversy in the Hermaphrodite Debate from the mid-1960s to the late 1990s"

[89] C. Epple, "Coming to Terms with Navajo Nádleehí: A Critique of Berdache, 'Gay,' 'Alternate Gender,' and 'Two-spirit'," *American Ethnologist 25*: American Ethnologist, 25: 1998, DOI: 10.1525/ae.1998.25.2.267, p. 267-290.
[90] Will Roscoe, "Priests of the Goddess: Gender Transgression in Ancient Religion," *History of Religions 35*, no. 3: 1996, URL: jstor.org/stable/1062813, p. 195-203.
[91] Paul B. Moyer, *The Public Universal Friend: Jemima Wilkinson and Religious Enthusiasm in Revolutionary America*, Cornell University Press (Ithaca, NY): 2015, DOI: 10.7591/9781501701450.
[92] Sexual and romantic relationships between same-gendered partners are extremely common in myth, legend, fable, and history. Achilles and Patroclus, the many loves of Sappho, Catallus' untranslatable poetry, the Chinese euphemism "the passion of the cut sleeve", the Mesoamerican god of male prostitutes and homosexuals (Xochipilli, "flower prince"), etc.
[93] Rather than *n*-dimensional plots of behavior, where *n* is arbitrarily large, to accommodate behaviors associated with femininity and masculinity only in tandem (i.e. cooking (x) in the home (y) with femininity and cooking (x) in a professional context (y) with masculinity) as well as not strict homo- or heterosexuality (bisexuality, asexuality, etc.).
[94] Fausto-Sterling, *Sexing the Body: Gender Politics and the Construction of Sexuality*.

by Cynthia Kraus,[95] and "The sexed/gendered controversy over laterality genes in British psychology, 1970s-1990s" by Tabea Cornel.[96] All three works deal with similar topics to my thesis, but all[97] focus on the period before 2000. Kraus and Fausto-Sterling do acknowledge the effects of external forces on internal scientific practice, though neither treat its theoretical aspects in the way I have above. Additionally, none of the three use the framework of scientific controversy.

This thesis also drew substantially on Des Fitzgerald's book *Tracing Autism: Uncertainty, Ambiguity, and the Affective Labor of Neuroscience*[98] and "Genetics on the neurodiversity spectrum: Genetic, phenotypic, and endophenotypic continua in autism and ADHD" by Polaris Koi.[99] Fitzgerald's book contains a meditation on whether it is possible to fully separate the scientific, medical, cultural, and social spheres when considering the diagnosis (diagnoses?) of autism.[100] One of autism's primary diagnostic characteristics is "difficulty with social situations," making much of the neurological research on the topic an attempt to find a *biological* cause for a diagnosis that is at least partially *social*. Koi's paper adds to this mixed framework by discussing how ADHD and autism are continuous, with a range of possible behavioral and mental presentations, and medical, as diagnosis has consequences for treatment,

[95] Cynthia Kraus, "Am I My Brain or My Genitals? A Nature-Culture Controversy in the Hermaphrodite Debate from the mid-1960s to the late 1990s," *Genserus 86*, no. 1: 2001, p. 80-106.
[96] Tabea Cornel, "The sexed/gendered controversy over laterality genes in British psychology, 1970s-1990s," *History of the Human Sciences 35*, no. 5: 2020, DOI: 10.1177/0952695120944031, p. 138-166.
[97] Fausto-Sterling does use some literature published after 2000, particularly in the epilogue she wrote for the second edition. Still, the majority of her book is historical, and her focus is largely on the 1930s-1990s.
[98] Des Fitzgerald, *Tracing Autism: Uncertainty, Ambiguity, and the Affective Labor of Neuroscience.* University of Washington Press (Seattle): 2017. ProQuest Ebook Central.
[99] Polaris Koi, "Genetics on the neurodiversity spectrum: Genetic, phenotypic, and endophenotypic continua in autism and ADHD," *Studies in History and Philosophy of Science 89*: 2021, p. 52-62, DOI: 10.1016/j.shpsa.2021.07.006.
[100] Fitzgerald, *Tracing Autism*, p. 170-183.

care, and assistance.[101] Koi additionally considers–at some length–if and how categories might usefully be extracted from spectra that are strongly associated with *endophenotypes,* also called *subclinical traits*. In the case of autism and ADHD, impaired sensory processing, hyperfocus on specific interests, and failures of communicative ability or comprehension all occur in many individuals in many contexts, only some of whom are said to have either ADHD or autism. Koi's paper calls for a reconsideration of not only the categories of ADHD and autism but the categories of normality and divergence themselves, to be replaced with a multidimensional spectrum of the endophenotypic traits. While this thesis will only occasionally consider autism or ADHD, my ideas were greatly affected by this complex and multidimensional viewpoint on a (different) socio-scientific interaction between medicine, neuroscience, and people themselves.

1.5: A Map of the Territory

Each of the chapters in this thesis discusses the same material, the literature in §1.3, from a different perspective. Chapter 2 considers how the factual, mechanistic assumptions of organizational/activational theory were undermined, complicated, or disproven between 2000 and 2019, with the organizational/activational theory being (to some degree) discarded around 2015-2016. Chapter 3 then turns to the reasons for this theoretical break: the replication crisis, a methodological and publication controversy (apparently ongoing as of 2023). The assumptions that the replication crisis exposed, I argue, had significantly warped the literature about sex differences generally and the organizational/activational theory specifically. The third and final perspectival chapter, Chapter 4, drills down to assumptions about what types of evidence and what types of critiques were valid within the sphere of organizational/activational theory, and

[101] Koi, "Genetics on the neurodiversity spectrum," p. 55-61.

some of the consequences of those assumptions. Finally, Chapter 5 considers what assumptions can reveal about knowledge, knowers, and the ways knowing happens and is used.

Chapter 2: Factual and mechanistic controversies

2.0: Introduction

In this chapter, the basic assumptions of the organizational/activational model that are internal to the model itself will be discussed. Each section after this one will state a question that had a single answer within organizational/activational theory as it originally existed, an answer that the field would eventually reveal as an unjustified assumption. Those studying the organizational/activational theory in each case eventually came to the opposite conclusion or otherwise were forced to complicate the original straightforward answer. The "traditional" or "classic" model of development[102] argued that development in most mammals[103] follows a

[102]Stephanie V. Koebele and Bimonte-Nelson, Heather A., "Trajectories and phenotypes with estrogen exposures across the lifespan: What does Goldilocks have to do with it?", *Hormones and Behavior 74*: August 2015, p. 86-104, DOI: 10.1016/j.yhbeh.2015.06.009.

[103] There are a few mammals, including *Tokudaia osimensis* (the Amami spiny rat), which have an XX female and X0 male, where 0 denotes the absence of an additional chromosome. (Kuroiwa, A., Handa, S., Nishiyama, C. *et al.* Additional copies of *CBX2* in the genomes of males of mammals lacking *SRY*, the Amami spiny rat (*Tokudaia osimensis*) and the Tokunoshima spiny rat (*Tokudaia tokunoshimensis*). *Chromosome Res* **19**, 635–644 (2011). DOI: 10.1007/s10577-011-9223-6). There is also the Transcaucasian mole vole, in which males and females both have only one X chromosome (both X0). (W. Just, A. Baumstark, A. Süss, A. Graphodatsky, W. Rens, N. Schäfer, I. Bakloushinskaya, H. Hameister, W. Vogel; *Ellobius lutescens*: Sex Determination and Sex Chromosome. *Sex Dev* 1 August 2007; 1 (4): 211–221. DOI: 10.1159/000104771). Finally, there is the duck-billed platypus (*Ornithorhynchus anatinus)*, in which there are ten sex chromosomes which form a sort of molecular chain: XYXYXYXYXY in males, and XXXXXXXXXX in females. (Veyrunes, F., Waters, P. D., Miethke, P., Rens, W., McMillan, D., Alsop, A. E., Grützner, F., Deakin, J. E., Whittington, C. M., Schatzkamer, K., Kremitzki, C. L., Graves, T., Ferguson-Smith, M. A., Warren, W., & Marshall Graves, J. A. (2008). Bird-like sex chromosomes of platypus imply recent origin of mammal sex chromosomes. *Genome research*, *18*(6), 965–973. DOI: 10.1101/gr.7101908.) Outside of mammals, there are a wide range of sex determination systems, including the ZW female/ZZ male in birds, temperature-dependence in some fish and reptiles, XX hermaphrodite/X0 male in *Caenorhabditis elegans* nematode worms, haplodiploidy (a complex interaction between multiple genetic loci used to create the heavily female-biased ant and bee colonies), and the platyfish (*Xiphophorus* genus), which has WY/WX/XX females and YY/XY males. Even for animals which have similarly named systems, this should not necessarily be understood to imply that the animals share evolutionarily similar genetic sequences. For example, the platypus $(XY)_5$ and $(X)_{10}$ systems use chromosomes more sequentially homologous to the avian Z and W chromosomes, despite functioning more like the placental and marsupial mammal XY chromosomes. (See Veyrunes *et al*., 2008, previously in this footnote.) The variations in reproductive anatomy within mammals includes, for example, the possible presences of between zero (monotremes) and two (marsupials) vaginas, the presence or absence of a separate birth canal (marsupials), and either one or two uteruses ranging from simplex (humans) to bipartite (pigs, mice, marine mammals) to duplex (monotremes, rabbits, marsupials). (Kobayashi, A., Behringer, R. Developmental genetics of the female reproductive tract in mammals. *Nat Rev Genet* **4**, 969–980 (2003). DOI:

hierarchical framework. Genes on the sex chromosomes differentiate the gonads, which then send (or fail to send) hormonal signals throughout the developing fetus to develop its phenotypically male or female components, including the genitalia and the brain (organization). The changes in the brain are to specific areas, which then causes behaviors to be more male- or female-typical. After development, the gonads continue to control sex-specific or sex-biased behaviors via gonadal hormones (activation). From this combination of organization and activation, nearly all mammals possessed either a "male brain" or a "female brain" that was relatively insensitive to the environment. Most mammals developed consistent phenotypes in their body, genitals, brain, and behavior, all organized and activated one way or the other. The organizational/activational theory was a simple explanation—but for that simple explanation to hold, the assumed answers to the questions below had to also remain simple and straightforward. As the next sections will detail, the assumed answers were either contradicted or complicated between 2000 and 2019, which eventually caused the organizational/activational theory to disintegrate.

2.1: Where do changes come from and what causes them?

One of the most basic of the assumptions underlying the organizational/activational theory is that the gonads are the primary, though not foundational, location that generates sex differences. In other words, a central assumption of organizational/activational theory was that while genes on the sex chromosomes differentiate the gonads into either testes or ovaries, the gonads then go on to differentiate all other structures in the individual through the release of

10.1038/nrg1225); the hormonal variability between and within mammals was a major object of study during the period covered by this thesis. All of the above further sets aside individual variation—if, for example, the SRY sequence in a human has been translocated onto an X chromosome in a human, they will not necessarily follow the developmental schema of "most mammals". Suffice it to say, after this lengthy footnote, that "development in most mammals" should be understood as a very broad and incomplete generalization.

hormonal substances and that the genes have no further direct effects of their own.[104] This primacy of the gonads and hormones, besides being perhaps slightly chauvinistic in an endocrinological field, provided a straightforward causal line. Sexed genes on sexed chromosomes led to sexed gonads, sexed gonads released sexed hormones, and sexed hormones led to sexed brains and eventually, after puberty, sexed bodies.[105]

While this traditional model remained in some use through at least 2013,[106] several different lines of inquiry from multiple perspectives contradicted it at least in part. Firstly, evidence existed, much of it predating 2000, that some sex differences either preceded gonadal differentiation or reflected genetic differences directly, suggesting that some differences did not emerge from or rely on gonadal steroids.[107] Secondly, studies of those with multiple sex chromosomes (i.e. XXX, XXY, XYY, etc.) and one sex chromosome (Turner's syndrome, X0) suggested that some aspects of brain development relied on or were affected by that additional or decreased chromosomal content.[108] This evidence created the category of "direct genetic

[104] This assertion was intertwined with and underlay the experimental practice of adding hormones to chromosomally female animals in order to phenotypically, particularly behaviorally, masculinize them.
[105] Bodies here referring to the various secondary sex characteristics associated with sex as well as the continuous variables like height that differ on average between the sexes.
[106] E.g. J. M. Goldstein, Handa, R. J., Tobet, S. A., "Disruption of fetal hormonal programming (prenatal stress) implicates shared risk for sex differences in depression and cardiovascular disease", *Frontiers in Neuroendocrinology 35*(1): January 2014, DOI: 10.1016/j.yfrne.2013.12.001.
[107] Geert J. De Vries, Rissman, Emilie F., Simerly, Richard B., Yang, Liang-Yo, Scordalakes, Elka M., Auger, Catherine J., Swain, Amanda, Lovell-Badge, Robin, Burgoyne, Paul S., & Arnold, Arthur P. (2002). "A model system for study of sex chromosome effects on sexually dimorphic neural and behavioral traits." *Journal of Neuroscience*, *22*(20), 9005–9014 DOI:10.1523/JNEUROSCI.22-20-09005.2002.
[108] Jean-Francois Lepage; Hong, David S.; Mazaika, Paul K.; Raman, Mira; Sheau, Kristen; Marzelli, Matthew J.; Hallmayer, Joachim; Reiss, Allan L. "Genomic Imprinting Effects of the X Chromosome on Brain Morphology." *Journal of Neuroscience* 33(19): 8 May 2013, p. 8567-8574; DOI: 10.1523/JNEUROSCI.5810-12.2013. Hong, David S.; Hoeft, Fumiko; Marzelli, Matthew J.; Lepage, Jean-Francois; Roeltgen, David; Ross, Judith; Reiss, Allan L. "Influence of the X-Chromosome on Neuroanatomy: Evidence from Turner and Klinefelter Syndromes." *Journal of Neuroscience 34*(10): 5 March 2014, p. 3509-3516; DOI: 10.1523/JNEUROSCI.2790-13.2014. Reardon, Paul Kirkpatrick; Clasen, Liv; Giedd, Jay N.; Blumenthal, Jonathan; Perch, Jason P.; Chakravarty, M. Mallar; Raznahan, Armin. "An Allometric Analysis of Sex and Sex Chromosome Dosage Effects on Subcortical Anatomy in Humans." *Journal of Neuroscience 36* (8): 24 February 2016, p. 2438-2448; DOI: 10.1523/JNEUROSCI.3195-15.2016.

effects", which bypassed hormonal intermediation to affect cells directly, and became part of the standard "updated" models of sexual differentiation by 2015.[109]

Thirdly, a different group of researchers focused on the word "gonads" rather than "hormones". Given that at least some hormones could be synthesized in part or in whole in the brain, known as "neurosteroids", it was not entirely clear which, if any, sex steroids that affected the brain and behavior were coming directly from the gonads, nor precisely what parts of organization or activation they controlled. While neurosteroids did not become fully canonical during this period in these journals,[110] some researchers argued that the rapid control of behaviors by hormones seemed to require some level of local synthesis or alteration due to the physical separation between the gonads and the brain,[111] and other evidence suggested that neurosteroids specifically were necessary for normal brain function.[112] Another researcher, when scanning human brains for the enzyme aromatase, made "the rather surprising observation that the brain of men has the highest estrogen synthesizing capacity in the male body; and the only peripheral organ with similar capacity is the female ovary during ovulation."[113] In addition, the

[109] Koebele and Bimonte-Nelson, "Trajectories and phenotypes with estrogen exposures across the lifespan," p. 87.
[110] To the point that it was unclear what, precisely, should count as a "neurosteroid", specifically whether the hormone must be locally derived from cholesterol or whether local modifications to circulating hormones with enzymes should also qualify. See Velíšková, Jana and DeSantis, Kara A. "Sex and hormonal influences on seizures and epilepsy." Hormones and Behavior 63(2): February 2013, p. 267-277. DOI: 10.1016/j.yhbeh.2012.03.018.
[111] Cornil, Charlotte A.; Ball, Gregory F.; Balthazart, Jacques. "Rapid control of male typical behaviors by brain-derived estrogens." *Frontiers in Neuroendocrinology 33*(4): October 2012, p. 425-446, DOI: 10.1016/j.yfrne.2012.08.003.
[112] Lu, Yujiao; Sareddy, Gangadhara R.; Wang, Jing; Wang, Ruimin; Li, Yong; Dong, Yan; Zhang, Quanguang; Liu, Jinyou; O'Connor, Jason C.; Xu, Jianhua; Vadlamudi, Ratna K.; Brann, Darrell W. "Neuron-Derived Estrogen Regulates Synaptic Plasticity and Memory." *Journal of Neuroscience 39*(15): 10 April 2019, p. 2792-2809, DOI: 10.1523/JNEUROSCI.1970-18.2019; and Rudolph, Lauren M.; Cornil, Charlotte A.; Mittelman-Smith, Melinda A.; Rainville, Jennifer R.; Remage-Healey, Luke; Sinchak, Kevin; Micevych, Paul E. "Actions of Steroids: New Neurotransmitters." Journal of Neuroscience 36(45): 9 November 2016, p. 11449-11458. DOI: 10.1523/JNEUROSCI.2473-16.2016. Li, Rena.; Singh, Meharvan. "Sex differences in cognitive impairment and Alzheimer's disease." Frontiers in Neuroendocrinology 35(3): August 2014, p. 391; DOI: 10.1016/j.yfrne.2014.01.002.
[113] Biegon, Anat. "In vivo visualization of aromatase in animals and humans." *Frontiers in Neuroendocrinology 40*: January 2016, p. 48. DOI: 10.1016/j.yfrne.2015.10.001.

adrenal glands, above the kidneys, release dehydroepiandrosterone (DHEA) and aldosterone, two testosterone and estradiol precursors that additionally can activate estrogen and androgen receptors.[114] To what degree the adrenal glands and gonads were producing raw materials for the brain's local signaling versus signaling to the brain directly remained something of an open question in 2019.[115]

Finally, an unspoken assumption about hormonal effects on the brain and body was that the presence of the hormone was both necessary and sufficient to directly cause sexual differentiation. However, research in 2013 suggested that, for example, immune cells, like microglia and astrocytes, were required for sexual differentiation in the brain.[116] That research also raised a different question—if immune cells were necessary for sexual differentiation, could environmental influences like a maternal infection that could change immune activity change sexual differentiation? In other words, the evidence suggested that if hormones were organizing all of development, at least some of that organization worked through immune cells—but immune cells respond to more than just hormones, and immune cells might therefore respond to a nonhormonal signal during sex differentiation and change the outcome. Epigenetic modifications similarly represented an intermediate between hormone exposure and permanent changes in genetic expression,[117] but epigenetic mechanisms also do not only respond to sex

[114] DHEA is an effective signaler of the estrogen receptor beta (ERβ) and aldosterone is a weaker signal for the androgen receptor (AR).
[115] Balthazart, Jacques; Choleris, Elena; Remage-Healey, Luke. "Steroids and the brain: 50 years of research, conceptual shifts and the ascent of non-classical and membrane-initiated actions." *Hormones and Behavior 99*: March 2018, p. 1-8. DOI: 10.1016/j.yhbeh.2018.01.002.
[116] Lenz, Kathryn M.; Nugent, Bridget M.; Haliyur, Rachana; McCarthy, Margaret M. "Microglia Are Essential to Masculinization of Brain and Behavior." *Journal of Neuroscience 33*(7): 13 February 2013, p. 2761-2772. DOI: 10.1523/JNEUROSCI.1268-12.2013.
[117] Nugent, Bridget M.; Schwarz, Jaclyn M.; McCarthy, Margaret M. "Hormonally mediated epigenetic changes to steroid receptors in the developing brain: Implications for sexual differentiation." *Hormones and Behavior 59*(3): March 2011, p. 338-344. DOI: 10.1016/j.yhbeh.2010.08.009.

hormones.[118] The existence of these intermediaries provided mechanisms for hormonal sex differentiation but also weakened hormonal primacy. Hormones became (perhaps) necessary but were no longer sufficient, and the influence of the hormone was not direct and might be one signal among many.

More broadly but along the same lines, Donna Maney pointed out in a review that many studies had used changes in behavior associated with changes in genetic sequences of steroid receptors as evidence that the steroid receptors and steroids themselves were contributing to the behavior. The changes in genetic sequence often do affect how and whether the sequence becomes mRNA or a protein, but every level of interaction above that—intermolecular interactions, signaling pathway interactions, cell-cell interactions, tissue-tissue interactions, system-system interactions, and individual-individual interactions—can exaggerate, compensate for, over-compensate for, or blend multiple effects of the same genetic change throughout the body.[119] Did hormonal effects have a uniquely consistent effect or was the picture more complex?

In that review, Maney said, "What do associations between polymorphisms and behavior actually tell us? Answering this question will require us to look under the hood, inside the black box."[120] Adding the words "and (gonadal) hormones" after "polymorphisms" gives a rough sketch of this discussion overall. The classical view was that genetic sex determined gonadal sex, which then hormonally directed the brain and body to their final sex—male or female.[121] Due

[118] McEwen, Bruce S. "Redefining neuroendocrinology: Epigenetics of brain-body communication over the life course." *Frontiers in Neuroendocrinology 49*: April 2018, p. 8-30. DOI: 10.1016/j.yfrne.2017.11.001.
[119] Maney, Donna L. "Polymorphisms in sex steroid receptors: From gene sequence to behavior." *Frontiers in Neuroendocrinology 47*: Oct 2017, p. 47-65. DOI: 10.1016/j.yfrne.2017.07.003.
[120] Ibid., 47.
[121] There is a further complication to this picture covered in more detail in chapter 3, which is the question of which hormones caused masculinization versus feminization. It will be covered in the section about model animals, as it

largely to advances in genetic sequencing, protein characterization, and computational power and scope beginning in the late 1990s, researchers in the early 21st century had the chance to unpack the black boxes, and discovered an ever-expanding series of exceptions, caveats, and contradictions. Not only that, but the center of 'power', in the sense of determining sex, shifted from gonads to genes and from localized to distributed (gonads can be found in an anatomical place—genes are in every cell), which left the determination of sex and gender rather less internally consistent or straightforward than it had been. That pattern is not an isolated incident in this field during this period but is instead emblematic of the general trend toward complexity and contextualization.

2.2: How do hormones behave in the body?

Within the organizational/activational theory, hormones were assumed to act directly, as the last section noted, but also linearly and comparatively across individuals. Linear hormone action assumed that increases in hormone level increased the response in a roughly linear manner: add hormone, see a response; add more hormone, see a stronger response. Hormones were also assumed to be comparable across individuals, in that the same level of, for example, circulating estrogen in two individuals was assumed to have about the same impact on their actions, all else being equal. In a way, it is the phrase "all else being equal" which suggests the nascent problem: was all else actually equal?

Contextualization of hormone's molecular behavior took place at three levels: first, the level of the hormone-receptor interactions; second, the level of cellular behavior as a whole; and third, the systemic level of the body as a whole. The contextualization of hormone-receptor

emerged and remained debated at least in part because different animals seem to masculinize through different hormonal mechanisms, including a probable difference between primates (including humans) and rats or mice.

interactions was based on a few pieces of information, emerging just prior to or during the period 2000-2019. Firstly, there appeared to be at least two types of hormone-receptor outcome: "rapid", which released neurotransmitters or changed cellular activity directly, or "transcriptional", which facilitated the transcription of genes, taking place on different timescales. This meant that, as a 2003 paper noted, hormones "may play numerous and sometimes opposing roles in developing and mature neurons".[122] Secondly, at least some receptors could activate in the absence of their associated hormone, suggesting the hormone-receptor relationship was a bit looser than previously believed.[123] Thirdly, not only were a handful more hormone receptors discovered[124] or hypothesized,[125] the known receptors could be selectively activated, altered, or repressed by the presence of co-factors or scaffold proteins and could also be transcribed and translated into proteins in at least a couple ways each.[126] Fourthly, evidence taken from those with more or less sensitive androgen receptors suggested that the absolute amount of circulating hormone mattered less than the individual's reactivity overall.[127]

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[122] Wong, Jeremy K.; Le, Hoa H.; Zsarnovszky, Attila; Belcher, Scott M. "Estrogens and ICI182,780 (Faslodex) Modulate Mitosis and Cell Death in Immature Cerebellar Neurons via Rapid Activation of p44/p42 Mitogen-Activated Protein Kinase." *Journal of Neuroscience 23*(12): 15 June 2003, p. 4984-4995. DOI: 10.1523/JNEUROSCI.23-12-04984.2003.

[123] Auger, Anthony P. "Steroid receptor control of reproductive behavior." *Hormones and Behavior 45*(3): March 2004, p. 168-172. DOI: 10.1016/j.yhbeh.2003.09.013.

[124] Sellers, Katherine; Raval, Pooja; Srivastava, Deepak P. "Molecular signature of rapid estrogen regulation of synaptic connectivity and cognition." *Frontiers in Neuroendocrinology 36*: January 2015, p. 72-89. DOI: 10.1016/j.yfrne.2014.08.001.

[125] There were a few who hypothesized the existence of a second androgen receptor, e.g. Balthazart, Jacques. "Behavioral implications of rapid changes in steroid production action in the brain [Commentary on Pradhan D.S., Newman A.E.M., Wacker D.W., Wingfield J.C., Schlinger B.A. and Soma K.K.: Aggressive interactions rapidly increase androgen synthesis in the brain during the non-breeding season. Hormones and Behavior, 2010]." *Hormones and Behavior 47*(4-5): April 2010, p. 375-378. DOI: 10.1016/j.yhbeh.2010.02.003.

[126] These variants are known as splice variants and are created during mRNA processing. Sellers, Raval, and Srivastava, "Molecular signature of rapid estrogen regulation," p. 72.

[127] Simmons, Zachary L.; Roney, James R. "Variation in CAG repeat length of the androgen receptor gene predicts variables associated with intrasexual competitiveness in human males." *Hormones and Behavior 60*(3): August 2011, p. 306-312. DOI: 10.1016/j.yhbeh.2011.06.006, as well as Rellini, A. H.; Stratton, N.; Tonani, S.; Santamaria, V.; Brambilla, E.; Nappi, R. E. "Differences in sexual desire between women with clinical versus biochemical signs of hyperandrogenism in polycystic ovarian syndrome." *Hormones and Behavior 63*(1): January 2013, p. 65-71. DOI: 10.1016/j.yhbeh.2012.10.013.

Finally, some researchers began to question whether hormones acted linearly; that is, whether increased levels of the hormone always led to increased activity of the receptor and the pathways or processes it activated (or decreased activity for repressed pathways).[128]

At the level of the cell, many receptors and pathways have thresholds of sensitivity at the upper or lower extents through under- or oversaturation, and, to extend to the cell overall, receptors often show maximum activity around a "median" value through interactions with other signaling pathways or safeguards against overstimulation or overproduction. At a certain point, continuing to increase the levels of, for example, testosterone may decrease the activity of the androgen receptor it activates or offer no additional activation and plateau in effectiveness.[129] Within the cell, there are multiple signaling networks that interact in a networked and complex fashion that cannot be accurately described linearly.[130] This includes the co-factors, scaffold proteins, and hormone-independent activation described above, but also that hormone receptors can activate multiple networks at once[131] and that the same receptor can activate different intracellular pathways that cause the same outcome (in this case, in male versus female mice).[132]

Finally, in the body as a whole, a significant proportion of the circulating hormones are not bioavailable[133] and hormonal release is subject to multiple feedback loops which may up- or

[128] Swift-Gallant, A. and Monks, D. A. "Androgenic mechanisms of sexual differentiation of the nervous system and behavior." *Frontiers in Neuroendocrinology 46:* July 2017, p. 32-45. DOI: 10.1016/j.yfrne.2017.04.003.
[129] Ibid., 41, 35.
[130] Ruf, Frederique; Fink, Marc Y.; Sealfon, Stuart C. "Structure of the GnRH receptor-stimulated signaling network: insights from genomics." *Frontiers in Neuroendocrinology 24*(3): July 2003, p. 181-199. DOI: 10.1016/s0091-3022(03)00027-x.
[131] Amandusson, Åsa and Blomqvist, Anders. "Estrogenic influences in pain processing." *Frontiers in Neuroendocrinology 34*(4): October 2013, p. 329-349. DOI: 10.1016/j.yfrne.2013.06.001.
[132] Koss, Wendy A.; Haertel, Jacqueline M.; Philippi, Sarah M.; Frick, Karyn M. "Sex Differences in the Rapid Cell Signaling Mechanisms Underlying the Memory-Enhancing Effects of 17β-Estradiol." *eNeuro 30*(5): 30 October 2018, p. 1-14. DOI: 10.1523/ENEURO.0267-18.2018.
[133] Matousek, Rose H. and Sherwin, Barbara B. "Sex steroid hormones and cognitive functioning in healthy, older men." *Hormones and Behavior 57*(3): March 2010, p. 352. DOI: 10.1016/j.yhbeh.2010.01.004.

downregulate hormonal synthesis in response to receptor sensitivity.[134] Despite this evidence implying a complex and noisy if not uninterpretable distance between hormone and outcome, multiple different groups of researchers across 2000-2019 compared hormone levels between individuals, presumed linear hormonal activity, or both, with little or no justification.[135] While the assumption of non-contextualized, linear, and straightforwardly causal hormonal action was not explicitly discarded by 2019, researchers working in cellular biology or biochemistry, among other fields, frequently described hormonal behavior in a more complex fashion.

2.3: When does development happen?

The original theoretical timing of the organization of sexually differentiated behaviors was either prenatally or soon after birth,[136] while the activation occurred during puberty and explained the behavioral changes in individuals during that period, such as sexual interest and sexual behaviors. The high point of fetal gonadal testosterone release occurs during weeks 8 and 21 of gestation, and "the critical period of brain differentiation [was] thought to closely follow fetal gonadal differentiation."[137] This hypothesized fetal differentiation remained a possibility but did not remain uniquely important for explaining behavioral changes and neural

[134] Hastings, W. J.; Chang, A. M.; Ebstein, R. P.; Shalev, I. "Neuroendocrine stress response is moderated by sex and sex hormone receptor polymorphisms." *Hormones and Behavior 106*: November 2018, p. 74-80. DOI: 10.1016/j.yhbeh.2018.10.002.

[135] See, for example, Schultheiss, O. C. and Rohde, W. "Implicit power motivation predicts men's testosterone changes and implicit learning in a contest situation." *Hormones and Behavior 41*(2): March 2002, p. 195-202. DOI: 10.1006/hbeh.2001.1745.; Edelstein R. S.; Chopik, W. J.; Kean, E. L. "Sociosexuality moderates the association between testosterone and relationship status in men and women." *Hormones and Behavior 60*(3): August 2011, p. 248-255. DOI: 10.1016/j.yhbeh.2011.05.007.; and McHenry, Jenna; Carrier, Nicole; Hull, Elaine; Mohamed, Kabbaj. "Sex differences in anxiety and depression: role of testosterone." *Frontiers in Neuroendocrinology 35*(1): January 2014, p. 42-57. DOI: 10.1016/j.yfrne.2013.09.001.

[136] Chung, Wilson C. J.; de Vries, Geert J.; Swaab, Dick F. "Sexual Differentiation of the Bed Nucleus of the Stria Terminalis in Humans May Extend into Adulthood." *Journal of Neuroscience 22*(3): 1 February 2002, p. 1027-1033. DOI: 10.1523/JNEUROSCI.22-03-01027.2002.

[137] van de Beek, Cornelieke; Thijssen, Jos H. H.; Cohen-Kettenis, Peggy T.; van Goozen, Stephanie H. M.; Buitelaar, Jan K. "Relationships between sex hormones assessed in amniotic fluid, and maternal and umbilical cord serum: What is the best source of information to investigate the effects of fetal hormonal exposure?" *Hormones and Behavior 46*(5): December 2004, p. 664. DOI: 10.1016/j.yhbeh.2004.06.010.

development. As one early paper noted, some "marked morphological changes in the human brain, including sexual differentiation, may not be limited to childhood but may extend into adulthood."[138] In rough order of human development, the posited additional (re)organizational periods include "mini-puberty", or a period lasting a few months after birth;[139] adrenarche, a period of adrenal activation lasting approximately from ages 3 to 13;[140] puberty, which begins between roughly ages 8 and 14,[141] and the perhaps-separate[142] perhaps-continuous[143] period of adolescence, lasting from puberty until approximately ages 18-25;[144] pregnancy, generally occurring between ages 15 and 55;[145] and menopause, occurring generally between ages 45 and

[138] Chung, de Vries, and Swaab, "Sexual Differentiation of the Bed Nucleus of the Stria Terminalis", 1 February 2002, p. 1031.
[139] Lamminmäki, Annamarja; Hines, Melissa; Kuiri-Hänninen, Tanja; Kilpeläinen, Leena; Dunkel, Leo; Sankilampi, Ulla. "Testosterone measured in infancy predicts subsequent sex-typed behavior in boys and in girls." *Hormones and Behavior 61*(4): April 2012, p. 611-616. DOI: 10.1016/j.yhbeh.2012.02.013.; and Pasterski, Vicki; Acerini, Carlo L.; Dunger, David B.; Ong, Ken K.; Hughes, Ieuan A.; Thankamony, Ajay; Hines, Melissa. "Postnatal penile growth concurrent with mini-puberty predicts later sex-typed play behavior: Evidence for neurobehavioral effects of the postnatal androgen surge in typically developing boys." *Hormones and Behavior 69*: March 2015, p. 98-105. DOI: 10.1016/j.yhbeh.2015.01.002.
[140] Nguyen, Tuong-Vi; McCracken, James T.; Ducharme, Simon; Cropp, Brett F.; Botteron, Kelly N.; Evans, Alan C.; Karama, Sherif. "Interactive Effects of Dehydroepiandrosterone and Testosterone on Cortical Thickness during Early Brain Development." *Journal of Neuroscience 33*(26): 26 June 2013, p. 10840-10858. DOI: 10.1523/JNEUROSCI.5747-12.2013.
[141] Berenbaum, Sheri A.; Beltz, Adriene M. "Sexual differentiation of human behavior: effects of prenatal and pubertal organizational hormones." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 183-200. DOI: 10.1016/j.yfrne.2011.03.001.; and Beltz, Adriene M.; Berenbaum, Sheri A. "Cognitive effects of variations in pubertal timing: is puberty a period of brain organization for human sex-typed cognition?" *Hormones and Behavior 63*(5): May 2013, p. 823-828. DOI: 10.1016/j.yhbeh.2013.04.002.
[142] Walker, Deena M.; Bell, Margaret R.; Flores, Cecilia; Gulley, Joshua M.; Willing, Jari; Paul, Matthew J. "Adolescence and Reward: Making Sense of Neural and Behavioral Changes Amid the Chaos." *Journal of Neuroscience 37*(45): 8 November 2017, p. 10855-10866. DOI: 10.1523/JNEUROSCI.1834-17.2017.; and Sisk, Cheryl L.; Berenbaum, Sheri A. "Editorial for the special issue of hormones and behavior on puberty and adolescence." *Hormones and Behavior 64*(2): July 2013, p. 173-174. DOI: 10.1016/j.yhbeh.2013.07.006.
[143] Holder, Mary K.; Blaustein, Jeffery D. "Puberty and adolescence as a time of vulnerability to stressors that alter neurobehavioral processes." *Frontiers in Neuroendocrinology 35*(1): January 2014, p. 89-110. DOI: 10.1016/j.yfrne.2013.10.004.; and Spielberg, Jeffrey M.; Schwarz, Jaclyn M.; Matyi, Melanie A. "Anxiety in transition: Neuroendocrine mechanisms supporting the development of anxiety pathology in adolescence and young adulthood." *Frontiers in Neuroendocrinology 55*: October 2019. Article no. 100791. DOI: 10.1016/j.yfrne.2019.100791.
[144] Sisk, Cheryl L. and Zehr, Julia L. "Pubertal hormones organize the adolescent brain and behavior." *Frontiers in Neuroendocrinology 26*(3-4): October-December 2005, p. 163-174. DOI: 10.1016/j.yfrne.2005.10.003.
[145] Berenbaum and Beltz, "Sexual differentiation of human behavior," 2011, p. 183.

55.[146] By 2019, it would be more accurate to say that "organizational" effects denoted permanent, or at least long-lasting, changes to the brain or body while "activational" effects were transient and did not cause structural changes,[147] though some definitions of the terms suggested that "activational" effects could become "organizational" given the right circumstances.[148] This proliferation of (re)organizational events made the previous assumption of a singular organizational moment for sex differentiation difficult to maintain, even if not all researchers accepted all of the above candidates.[149]

2.4 What is the natural state of a mind?

As briefly mentioned in Chapter 1, there was a preexisting idea that female was the default or un(der)developed state of a mind. Organizational/activational theory focused on masculinization and defeminization in part because feminization was, though not fully passive, the process the body and brain were believed to undergo with no stimulus.[150] "Demasculinization", the counterpart to defeminization, was explicitly argued to not apply to mammals, as "pre-existing masculine traits have not been demonstrated in mammalian sexual differentiation."[151] Continuing this thread of logic, homosexual men tended to be described as "hypomasculinized" and homosexual women as "masculinized" due to their sexual

[146] Koebele and Bimonte-Nelson, "Trajectories and phenotypes with estrogen exposures," 2015, p. 90.
[147] Spielberg, Schwarz, and Matyi, "Anxiety in transition", 2019, p. 6.
[148] Koebele and Bimonte-Nelson, "Trajectories and phenotypes with estrogen exposures," 2015, p. 90, 96, 98.
[149] Puberty and/or adolescence as an organizational or critical period was the most commonly accepted.
[150] Wilson, Jean D. "Androgens, androgen receptors, and male gender role behavior." *Hormones and Behavior 40*(2): September 2001, p. 358-366. DOI: 10.1006/hbeh.2001.1684. Küppers, Eva; Ivanova, Tatiana; Karolczak, Magdalena; Lazarov, Nikolai; Föhr, Karl; Beyer, Cordian. "Classical and Nonclassical Estrogen Action in the Developing Midbrain." *Hormones and Behavior 40*(2): September 2001, p. 196-202. DOI: 10.1006/hbeh.2001.1671. Maggi, A.; Ciana, P.; Brusadelli, A.; Belcredito, S.; Bonincontro, C.; Vegeto, E. "Are there biological bases for a beneficial effect of estrogens in neural diseases?" *Hormones and Behavior 40*(2): September 2001, p. 203-209. DOI: 10.1006/hbeh.2001.1694. Villa, Alessandro; Della Torre, Sara; Maggi, Adriana. "Sexual differentiation of microglia." *Frontiers in Neuroendocrinology 52*: January 2019, p. 156-164. DOI: 10.1016/j.yfrne.2018.11.003.
[151] Wallen, Kim. "Hormonal influences on sexually differentiated behavior in nonhuman primates." *Frontiers in Neuroendocrinology 26*(1): April 2005, p. 9. DOI: 10.1016/j.yfrne.2005.02.001.

preferences,[152] and the "maternal immune hypothesis of homosexuality" similarly posited that male homosexuality originated from interference with normal male development that prevented the masculinization (or perhaps defeminization) of the relevant region(s) of the brain.[153]

The assumption that being female was the "default" state of development was discarded with two new pieces of evidence and a theoretical shift. Firstly, some women with Turner syndrome (45, X0) possess no or very little ovarian tissue, and behaviorally appeared to have non-sexed personalities, interests, and cognitive abilities,[154] and subsequent testing on female rodents suggested that an absence of ovarian hormones caused those rodents to behave in neither typically male nor typically female ways.[155] Secondly, development in female rodents could be directed toward a male-type path by halting a process, specifically DNA methylation in the preoptic area, implying that at least some areas could possess a male "default" instead.[156] From a theoretical perspective, as described in §2.1, if genes had direct effects on development, then cells could only be said to "default" to their chromosomal sex.[157] Indeed, one paper suggested that female as default had been an assumption with little grounding, "because scientists rarely, if ever, studied the phenotype of a mouse lacking gonadal secretions, so there was no accurate

[152] McFadden, Dennis; Shubel, Erin. "The relationships between otoacoustic emissions and relative lengths of fingers and toes in humans." *Hormones and Behavior 43*(3): March 2003, p. 421-429. DOI: 10.1016/S0018-506X(03)00014-X.
[153] Blanchard, Ray; Zucker, Kenneth J.; Cavacas, Ana; Allin, Sara; Bradley, Susan J.; Schachter, Debbie C. "Fraternal Birth Order and Birth Weight in Probably Prehomosexual Feminine Boys." *Hormones and Behavior 41*(3): May 2002, p. 321-327. DOI: 10.1006/hbeh.2002.1765.
[154] Collaer, Marcia L.; Geffner, Mitchell E.; Kaufman, Francine R.; Buckingham, Bruce; Hines, Melissa. "Cognitive and behavioral characteristics of turner syndrome: exploring a role for ovarian hormones in female sexual differentiation." *Hormones and Behavior 41*(2): March 2002, p. 139-155. DOI: 10.1006/hbeh.2001.1751.
[155] Brock, Olivier; Baum, Michael J.; Bakker, Julie. "The development of female sexual behavior requires prepubertal estradiol." *Journal of Neuroscience 31*(15): 13 April 2011, p. 5574-5578. DOI: 10.1523/JNEUROSCI.0209-11.2011.
[156] Forger, Nancy G.; Strahan, J. Alex; Castillo-Ruiz, Alexandra. "Cellular and molecular mechanisms of sexual differentiation in the mammalian nervous system." *Frontiers in Neuroendocrinology 40*: January 2016, p. 67-86. DOI: 10.1016/j.yfrne.2016.01.001.
[157] Arnold, Arthur P.; Chen, Xuqi. "What does the 'four core genotypes' mouse model tell us about sex differences in the brain and other tissues?" *Frontiers in Neuroendocrinology 30*(1): January 2009, p. 7. DOI: 10.1016/j.yfrne.2008.11.001.

determination of the default phenotype."[158] While female as default was not inherent to organizational/activational theory, it synthesized with the original assumption in §2.3, of prenatal or perinatal organization, to form a collective focus on prenatal androgens as the only or primary sex-determining factor in the development of the brain. Much of organizational/activational theory's research hinged on this connection between the prenatal androgen exposure of individuals and their later sex-typed behavior.

2.5 How does one determine the sex of a brain through behavior?

The organizational/activational theory, at least in its original formation, modeled brain differentiation as following and dependent on gonadal differentiation, and there were a number of behaviors, preferences, or self-identifications posited to be typical of the "male brain" or "female brain". For example, most female humans were assumed to or argued to have a gender identity of "woman" or "girl", and female animals generally were assumed to have a sexual preference toward males or men[159] and sexual role behavior that is receptive.[160] To pause there, however, many versions of the "gendered brain" presumed that each aspect tended to align with the others. For example, a paper published in 2002 stated: "[The Child Psychiatry Program's] patients include boys referred to a specialty clinic within that Program because of pervasive and persistent feminine behavior or repeatedly stated wishes to be girls. Because marked cross-gender behavior in boys is a very strong predictor of adult homosexuality (Bailey and Zucker,

[158] Ibid., p. 7.
[159] McFadden, Dennis. "Sexual orientation and the auditory system." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 203; DOI: 10.1016/j.yfrne.2011.02.001.
[160] Henley, C. L.; Nunez, A. A.; Clemens, L. G. "Hormones of choice: The neuroendocrinology of partner preference in animals." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 151-152; DOI: 10.1016/j.yfrne.2011.02.010.

1995), the feminine boys can be regarded as a prehomosexual group."[161] This characterization combines transgender girls, feminine boys, and homosexual boys into a single group.

This idea of 'alignment' assumed that not only were there gendered brains, but brains generally developed to be entirely one gender or entirely the other. This point becomes evident when one considers the absence of bisexuals, particularly male bisexuals, given the relative volume of literature mentioning or analyzing male homosexuality. That assumption remained relevant in the literature through 2019, as a paper published that year argued that gay men who preferred the "bottom" role in anal sex tended to be less gender-conforming, shorter, and had less body hair, while gay men who preferred the "top" role in anal sex tended to be more gender-conforming, taller, and have more body hair, and that all of this suggested that "bottoms" had experienced low androgens (hypomasculinized) and that "tops" had experienced high androgens (hypermasculinized) during development.[162] That many gay (and bisexual) men do not have a favored role in anal sex[163] or prefer not to participate in anal sex[164] was not mentioned. Any one paper's omission of nonbinary genders, polysexual sexualities, nonconforming gender role behavior, or nonnormative sexual role preferences is reasonable. Their relatively sparse representation in the literature, however, suggests that brains tended to be conceived of as fully one gender or the other, and that different metrics in those brains were believed to normally and naturally align with each other and with the closest heteronormative equivalent

[161] Blanchard *et al.,* "Fraternal Birth Order and Birth Weight in Probably Prehomosexual Feminine Boys," 2002, p. 322.
[162] Swift-Gallant, Ashlyn. "Individual differences in the biological basis of androphilia in mice and men." *Hormones and Behavior 111*: May 2019, p. 23-30. DOI: 10.1016/j.yhbeh.2018.12.006.
[163] Moskowitz, David A.; Hart, Trevor A. "The influence of physical body traits and masculinity on anal sex roles in gay and bisexual men." *Archives of Sexual Behavior 40*(4): August 2011, p. 835-841; DOI: 10.1007/s10508-011-9754-0.
[164] Rosenberger, J.G., Reece, M., Schick, V., Herbenick, D., Novak, D.S., Van Der Pol, B. and Fortenberry, J.D. "Sexual Behaviors and Situational Characteristics of Most Recent Male-Partnered Sexual Event among Gay and Bisexually Identified Men in the United States." *The Journal of Sexual Medicine, 8*: 2001, p. 3040-3050; DOI: 10.1111/j.1743-6109.2011.02438.x.

(female/woman/femininity/male sexual preference/female role or male/man/masculinity/female sexual preference/male role). Within the context of the organization of sexually differentiated behavior, this alignment represented the common forces determining these aspects—chromosomal and gonadal sex, generally thought to be mediated by hormones specifically.

This organization of sex-typed behaviors by consistent underlying factors extended beyond those directly relevant to reproduction. Visual stimuli preferences[165] and focuses[166] were argued to become more male-typical with increased testosterone, towards objects and away from people, possibly related to an increase in spatial ability also associated with testosterone.[167] The "power motive" (as opposed to the "affiliation motive"),[168] status-consciousness,[169] and aggression[170] were also associated with increased testosterone. These less directly reproduction-linked behaviors and sexual orientation were linked together: girls with congenital adrenal hypoplasia, a condition that elevates prenatal and early-life testosterone, had higher rates of

[165] Alexander, Gerianne M.; Wilcox, Teresa; Farmer, Mary Elizabeth. "Hormone-behavior associations in early infancy." *Hormones and Behavior 56*(5): November 2009, p. 498-502; DOI: 10.1016/j.yhbeh.2009.08.003.
[166] Iijima, Megumi; Arisaka, Osamu; Minamoto, Fumie; Arai, Yasumasa. "Sex Differences in Children's Free Drawings: A Study on Girls with Congenital Adrenal Hyperplasia." *Hormones and Behavior 40*(2): September 2001, p. 99-104; DOI: 10.1006/hbeh.2001.1670.
[167] van Anders, Sari M.; Hampson, Elizabeth. "Testing the prenatal androgen hypothesis: measuring digit ratios, sexual orientation, and spatial abilities in adults." *Hormones and Behavior 47*(1): January 2005, p. 92-98; DOI: 10.1016/j.yhbeh.2004.09.003. Driscoll, Ira; Hamilton, Derek A.; Yeo, Ronald A.; Brooks, William M.; Sutherland, Robert J. "Virtual navigation in humans: the impact of age, sex, and hormones on place learning." *Hormones and Behavior 47*(3): March 2005, p. 326-335; DOI: 10.1016/j.yhbeh.2004.11.013.
[168] Schultheiss, Oliver C.; Dargel, Anja; Rohde, Wolfgang. "Implicit motives and gonadal steroid hormones: effects of menstrual cycle phase, oral contraceptive use, and relationship status." *Hormones and Behavior 43*(2): February 2003, p. 293-301; DOI: 10.1016/s0018-506x(03)00003-5. Schultheiss, Oliver C.; Wirth, Michelle M.; Stanton, Steven J. "Effects of affiliation and power motivation arousal on salivary progesterone and testosterone." *Hormones and Behavior 46*(5): December 2004, p. 592-599; DOI: 10.1016/j.yhbeh.2004.07.005.
[169] Newman, Matthew L.; Sellers, Jennifer Guinn; Josephs, Robert A. "Testosterone, cognition, and social status." *Hormones and Behavior 47*(2): February 2005, p. 205-211; DOI: 10.1016/j.yhbeh.2004.09.008.
[170] Benderlioglu, Zeynep; Nelson, Randy J. "Digit length ratios predict reactive aggression in women, but not in men." *Hormones and Behavior 46*(5): December 2004, p. 558-564; DOI: 10.1016/j.yhbeh.2004.06.004. Cohen-Bendahan, Celina C. C.; Buitelaar, Jan K.; van Goozen, Stephanie H. M.; Orlebeke, Jacob F.; Cohen-Kettenis, Peggy T. "Is there an effect of prenatal testosterone on aggression and other behavioral traits? A study comparing same-sex and opposite-sex twin girls." *Hormones and Behavior 47*(2): February 2005, p. 230-237; DOI: 10.1016/j.yhbeh.2004.10.006.

homo-/bisexuality and more male-type behaviors overall,[171] and another study found that non-heterosexuality and spatial ability were correlated with each other and, possibly, prenatal androgens.[172]

Compared to, for example, the prenatal or perinatal timing of organization (§2.3), the sex-typed and hormonally associated index of behavior faced relatively little direct pushback. One paper did argue for a more important role of genetics (compared to hormones),[173] and another argued that sexual motivation in female humans hinged on estradiol, much like many other mammals.[174] In sharp contrast, the link between aggression, status-striving, and testosterone simply faded. One paper suggested that the relationship between aggression and testosterone depended on cultural background[175] and another argued, by studying transgender men's aggression during hormone replacement therapy, that the relationship between testosterone and aggression could not be causal.[176] In addition, a paper in 2019 suggested that, while there might be a link between testosterone, aggression, and status-seeking behaviors, the extant research over the last decades, when aggregated for meta-analysis, simply could not

[171] Hines, Melissa. "Prenatal endocrine influences on sexual orientation and on sexually differentiated childhood behavior." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 170-182; DOI: 10.1016/j.yfrne.2011.02.006. Iijima *et al*., "Sex Differences in Children's Free Drawings", 2001. Pasterski, Vicki; Geffner, Mitchell E.; Brain, Caroline; Hindmarsh, Peter; Brook, Charles; Hines, Melissa. "Prenatal hormones and childhood sex segregation: playmate and play style preferences in girls with congenital adrenal hyperplasia." *Hormones and Behavior 59*(4): April 2011, p. 549-555; DOI: 10.1016/j.yhbeh.2011.02.007.
[172] van Anders and Hampson, "Testing the prenatal androgen hypothesis," 2005.
[173] Ngun, Tuck C.; Ghahramani, Negar; Sánchez, Francisco J.; Bocklandt, Sven; Vilain, Eric. "The genetics of sex differences in brain and behavior." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 227-246; DOI: 10.1016/j.yfrne.2010.10.001.
[174] Wallen, Kim. "Women are not as unique as thought by some: comment on 'Hormonal predictors of sexual motivation in natural menstrual cycles', by Roney and Simmons." *Hormones and Behavior 63*(4): April 2013, p. 634-635; DOI: 10.1016/j.yhbeh.2013.03.009.
[175] Welker, Keith M.; Norman, Rachel E.; Goetz, Stefan; Moreau, Benjamin J. P.; Kitayama, Shinobu; Carré, Justin M. "Preliminary evidence that testosterone's association with aggression depends on self-construal." *Hormones and Behavior 92*: June 2017, p. 117-127; DOI: 10.1016/j.yhbeh.2016.10.014.
[176] Defreyne, Justine; Kreukels, Baudewijntje; T'Sjoen, Guy; Staphorsius, Annemieke; den Heijer, Martin; Heylens, Gunter; Elaut, Els. "No correlation between serum testosterone levels and state-level anger intensity in transgender people: Results from the European Network for the Investigation of Gender Incongruence." *Hormones and Behavior 110*: April 2019, p. 29-39; DOI: 10.1016/j.yhbeh.2019.02.016.

support that the connection existed, nor what specific behaviors testosterone influenced or caused.[177] Overall, however, what caused these behaviors to fall out of the literature around 2016 was the fact that the behaviors had been linked together so strongly in the context of a gendered brain; if the gendered brain did not obviously exist, then the relationship of these behaviors to each other and to a sexed developmental trajectory became significantly less clear.[178]

2.6 How do brains work?

Before considering the gendered or sexed brain, however, it is worth considering the brain *simpliciter*—specifically, a few different, and intertwined, conceptualizations of the brain. The first, with reference to Chapter 1, was the hypothesis that the human brain has hemispheric differences in function, and that this lateralization of function represents an essential human aspect. While some continued to conceive of the brain as having separated functions by hemisphere[179] and that right-handedness was a more typical result of development than left-handedness,[180] this hypothesis faced a few hurdles. Firstly, researchers found that the relationship between the size and activity of the corpus callosum, the structure connecting the two hemispheres, was difficult to relate to either lateralization or ability,[181] and lateralization's

[177] This will be discussed further in Chapter 3. Grebe, Nicholas M.; del Giudice, Marco; Thompson, Melissa Emery; Nickels, Nora; Ponzi, Davide; Zilioli, Samuele; Maestripieri, Dario; Gangestad, Steven W. "Testosterone, cortisol, and status-striving personality features: A review and empirical evaluation of the Dual Hormone hypothesis." *Hormones and Behavior 109*: March 2019, p. 25-37; DOI: 10.1016/j.yhbeh.2019.01.006.
[178] See §2.7.
[179] Hwang, Ren-Jen; Wu, Chi-Hsun; Chen, Li-Fen; Yeh, Tzu-Chen; Hsieh, Jen-Chuen. "Female menstrual phases modulate human prefrontal asymmetry: a magnetoencephalographic study." *Hormones and Behavior 55*(1): January 2009, p. 203; DOI: 10.1016/j.yhbeh.2008.10.008.
[180] Bogaert, Anthony F.; Skorska, Malvina. "Sexual orientation, fraternal birth order, and the maternal immune hypothesis: a review." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 247-248; DOI: 10.1016/j.yfrne.2011.02.004.
[181] Bitan, Tali; Lifshitz, Adi; Breznitz, Zvia; Booth, James R. "Bidirectional Connectivity between Hemispheres Occurs at Multiple Levels in Language Processing But Depends on Sex." *Journal of Neuroscience 30*(35): 1 September 2010, p. 11583; DOI: 10.1523/JNEUROSCI.1245-10.2010.

connection to the activating effects of circulating hormones was at a minimum quite complex.[182] Secondly, and more generally, distributed neural networks became a far more common method of conceptualizing the relationship between structure and function in the brain.

The more 'standard' model[183] that preceded and to some degree merged with distributed neural networks assumed that the brain was a set of functionally distinct and localized "modules," or "anatomically distinct areas of the cortex specialize[d] in processing specific types of information,"[184] as well as possessing areas which serve as "control systems", in which it is "possible to draw a line through anatomical space separating localized control regions or circuits (often the pFC [prefrontal cortex] and striatum) from more basic processing (caudal cortical) regions."[185] This model existed in degrees of strength, i.e. the degree to which different cognitive processes were conceived as having one-to-one correlations with neuroanatomical regions,[186] and had its historical origin in[187] studies of individuals who had received brain damage to specific areas of the brain and had lost their ability to perform certain behaviors[188] or recognize

[182] Bayer, Ulrike; Hausmann, Markus. "Estrogen therapy affects right hemisphere functioning in postmenopausal women." *Hormones and Behavior 55*(1): January 2009, p. 228-234.
[183] By no means unanimous, however. Among others, Justo Gonzalo, Constantin von Monakow, Henry Head, and Kurt Goldstein proposed models of holistic, interactionist, or distributed functional networks in the brain. García-Molina, A. "Justo Gonzalo's groundbreaking contributions to the study of cerebral functional organization." *Neurosciences and History 3*(2): 2015, p. 61-67; https://nah.sen.es/en/139-journals/volume-3/issue-2/293-justo-gonzalo-s-groundbreaking-contributions-to-the-study-of-cerebral-functional-organisation.
[184] Affan, Rifqi O.; Scott, Benjamin B. "Everything, everywhere, all at once: Functional specialization and distributed coding in the cerebral cortex." *Neuron 110*(5): 3 August 2022, p. 2361; DOI: 10.1016/j.neuron.2022.07.006.
[185] Eisenreich, Benjamin R.; Akaishi, Rei; Hayden, Benjamin Y. "Control without Controllers: Toward a Distributed Neuroscience of Executive Control." *Journal of Cognitive Neurosciences 29*(10): October 2017, p. 1684; DOI: 10.1162/jocn_a_01139.
[186] Mahon, Bradford Z.; Cantlon, Jessica F. "The specialization of function: Cognitive and neural perspectives." *Cognitive Neuropsychology 28*(3&4): 2011, p. 147-155; DOI: 10.1080/02643294.2011.633504.
[187] The degree to which the conceptually similar (pseudo)theory of phrenology (localizations on the skull rather than the brain) contributed to this theory's development is unclear, although these similarities have not gone unnoticed; for example, an argument for distributed neural processing titled "Beyond Phrenology: What Can Neuroimaging Tell Us About Distributed Circuitry?" by Karl Friston appeared in the *Annual Review of Neuroscience 22*: 2002, p. 221-250; DOI: 10.1146/annurev.neuro.25.112701.142846.
[188] For example, an inability to speak caused by damage to Broca's area, characterized by Broca.

certain stimuli,[189] as well as damage which altered personality or impulse control.[190] Distributed neural networks entered the literature covered by this thesis in the context of sociality: specifically, that the social behavior of different mammals emerged from an evolutionarily conserved set of structures in which the structures themselves did not control behaviors alone, but their various "weights" (or importance) in the network differed between species and individuals as well as changing over time in response to local conditions.[191] As opposed to social behavior "regulated… by individual brain nuclei,"[192] this "weighting", within individuals, allowed for significantly more (modeled) variation in social behavior as the network could represent far more possible "states" than the possible combinatorial activations of the different localized structures.[193]

This theoretical shift represented a break in the hypothesized relationship between structure and function. While some areas could still be conceived as "central components" of their respective networks,[194] the locations of functionally relevant areas often appeared to be

[189] A possible example is the development of prosopagnosia (an inability to recognize individuals by their faces), which may be caused by damage to the right temporal-occipital lobe. Gainlotta, Guido; Marra, Camillo. "Differential Contribution of Right and Left Temporo-Occipital and Anterior Temporal Lesions to Face Recognition Disorders." *Frontiers in Human Neuroscience 5*: 2011, Article no. 55; DOI: 10.3389/fnhum.2011.00055. There are also areas such as the auditory and visual cortexes which, when damaged, can cause a temporary or permanent failure of perception.

[190] For example, the behavior Phineas Gage exhibited after the traumatic brain injury he suffered to his left frontal lobe.

[191] Goodson, James L.; Kabelik, David. "Dynamic limbic networks and social diversity in vertebrates: from neural context to neuromodulatory patterning." *Frontiers in Neuroendocrinology 30*(4): October 2009, p. 429-441; DOI: 10.1016/j.yfrne.2009.05.007.

[192] Young, Larry J. "The neuroendocrinology of the social brain." *Frontiers in Neuroendocrinology 30*(4): October 2009, p. 425; DOI: 10.1016/j.yfrne.2009.06.002.

[193] Any description of a network of *n* nodes (for example, finite state machines), as opposed to a set of *n* (self-contained) nodes, will (by definition) describe the interactions between the nodes and the effects of those interactions on the state of the system. The self-contained nodes, on the other hand, can be expressed as a series of outputs from each node and the state that output corresponds to. The network model will thereby contain more possible "states" than the combinatorial possibilities of the *n* nodes on their own. Goodson and Kabelik, "Dynamic limbic networks and social diversity in vertebrates," 2009.

[194] For example, the amygdala as "the hub of a distributed corticolimbic circuit [that] mediates recognition and reaction to potential danger." Victor, Elizabeth C.; Sansosti, Alexandra A.; Bowman, Hilary C.; Hariri, Ahmad R. "Differential patterns of amygdala and ventral striatum activation predict gender-specific changes in sexual risk behavior." *Journal of Neuroscience 35*(23): 10 June 2015, p. 8896-8900; DOI: 10.1523/JNEUROSCI.0737-15.2015.

"local networks within and across nodes of the [neural network]"—i.e., not necessarily identifiable with anatomical structures and not always separable by individual signaling molecules.[195] Memorably, one researcher recounted a case study about a sex difference that, based on a combination of reasonable suppositions based on experimental evidence about aggressive behavior in some model animals and the proximity of different cell types releasing or possessing receptors for the molecule vasopressin, was believed to promote male aggression.[196] On further investigation, however, the system was revealed to be not as universal as previously believed (not being present in the model animal that reacts aggressively when given vasopressin in that area) and to constitutively *inhibit* aggression in many of the species that possess the system.[197] Modular models tended to assign single functions to single molecules and single anatomic structures,[198] and researchers working with modular models would sometimes fail to characterize "base" or "resting" states—in other words, the system was presumed to be "off" until perturbed. Distributed models challenged these conceptualizations of how structure had been believed to lead to functional outcomes, and although not all researchers accepted or used distributed models, or retained some pieces of earlier modular models,[199] these shifts exposed new questions relevant to organizational/activational theory, such as: was it true that estrogen

[195] Albers, H. Elliott. "Species, sex and individual differences in the vasotocin/vasopressin system: relationship to neurochemical signaling in the social behavior neural network." *Frontiers in Neuroendocrinology 36*: January 2015, p. 49-71; DOI: 10.1016/j.yfrne.2014.07.001.
[196] Kelley, Aubrey M.; Goodson, James L. "Social functions of individual vasopressin-oxytocin cell groups in vertebrates: what do we really know?" *Frontiers in Neuroendocrinology 35*(4): October 2014, p. 512-513; DOI: 10.1016/j.yfrne.2014.04.005.
[197] Ibid.
[198] Particularly dubious for vasopressin specifically, as it and oxytocin activate each other's receptors with nearly equal efficiency and nerves sensitive to vasopressin and/or oxytocin spatially overlap. Additionally, there are effects during embryogenesis known to be governed by overlapping and interrelated concentration gradients that have multiple effects based on local concentration, such as many of the signals released by HOX genes that control body segmentation, which may be also true of oxytocin/vasopressin in the brain as well.
[199] Such as denoting areas "central components".

and testosterone behaved in a linear manner on delineated anatomical regions in the brain? If so, when, and how, and how could those ideas be tested?[200]

There were two final theoretical shifts taking place across neuroscience that were relevant to the organizational/activational theory. Firstly, neural plasticity and adult neurogenesis, particularly in the hippocampus and hypothalamus, became far more accepted during this period.[201] Neural plasticity and neurogenesis raise the possibility of change, which poses an obvious question for a theory which claims behavior and identity to be "organized": why could the brain not be reorganized later?[202] Secondly and finally, the brain's relationship to the immune system underwent a sea change. "The brain had been thought to be 'immune privileged' and isolated from the immune system because of a lack of key immune proteins in neurons. Recently, immune molecules have been demonstrated to be present in the healthy brain and to play a critical role in brain development."[203] This new role for the immune system created an additional and fully separate avenue for (re)organizational changes that was not only distinct from hormone receptors but also responsive to external conditions—namely, infectious diseases contracted by the mother or the individual themselves. Immune system-mediated sex differentiation could align with the chromosomal and gonadal sex of the individual, but the possibility of their disagreement helped create the theoretical space for brains that were not completely one sex nor the other.

[200] Not to take a position on the question either way; simply to point out that the question was more frequently articulated than it had been before these theoretical shifts.
[201] Gage, Fred H. "Neurogenesis in the Adult Brain." *Journal of Neuroscience 22*(3): 1 February 2002, p. 612-613; DOI: 10.1523/JNEUROSCI.22-03-00612.2002.
[202] See also §2.3.
[203] Holder and Blaustein, "Puberty and adolescence as a time of vulnerability," 2014, p. 102.

2.7 What kinds of brains can exist and what defines them?

The best-established assumption was that there were two types of brains, male and female,[204] matched with the "'feminine' and 'masculine' cognitive sets."[205] Other theorists tied the two together: "Consistent with the established sex differences in cognitive functions, a variety of neurobiological substrates underlying cognition have been show[*sic*] to exhibit sex differences at various levels including in gross neuroanatomy, circuit properties, and molecular and cellular mechanisms."[206] What defined one type of brain versus the other was not always fully consistent with other conceptions,[207] but the idea of discrete behavioral, functional, and structural phenotypes of 'male' and 'female' remained present throughout 2000 to 2019 and, at least according to internal histories, first emerged from behavioral neuroendocrinology's foundational "Battle of the Titans" between Frank Beach and William C. Young in the 1950s.[208] One of the most prescient criticisms of this theoretical structure was the observation that behavioral neuroendocrinology's general "perspective… is that variation is categorical (male vs. female) rather than continuous."[209] Other authors noted that while androgens or male genitalia

[204] Maggi, A. *et al.* "Are There Biological Bases for a Beneficial Effect of Estrogens in Neural Diseases?" 2001, p. 203. Singh, Meharavan; Su, Chang. "Progesterone and neuroprotection." Hormones and Behavior 63(2): February 2013, p. 284-290; DOI: 10.1016/j.yhbeh.2012.06.003.
[205] Pearson, Rebecca; Lewis, Michael B. "Fear recognition across the menstrual cycle." Hormones and Behavior 47(3): March 2005, p. 271; DOI: 10.1016/j.yhbeh.2004.11.003.
[206] Hajali, Vahid; Andersen, Monica L.; Negah, Sajad Sahab; Sheibani, Vahid. "Sex differences in sleep and sleep loss-induced cognitive deficits: The influence of gonadal hormones." *Hormones and Behavior 108*: February 2019, p. 54; DOI: 10.1016/j.yhbeh.2018.12.013.
[207] One review argued that "The female brain is designed to respond in a 'permanently transient' nature," for example. Koebele and Bimonte-Nelson, "Trajectories and phenotypes with estrogen exposures across the lifespan," 2015, p. 91.
[208] McCarthy, Margaret M.; Pickett, Lindsay A.; VanRyzin, Jonathan W.; Kight, Katherine E. "Surprising origins of sex differences in the brain." *Hormones and Behavior 76*: 2015, p. 3; DOI: 10.1016/j.yhbeh.2015.04.013.
[209] This author blames the use of lab animals for this perspective; the use of model animals in this field will be discussed in Chapter 3. Crews, David. "Epigenetic modifications of brain and behavior: theory and practice." *Hormones and Behavior 59*(3): March 2011, p. 395; DOI: 10.1016/j.yhbeh.2010.07.001.

biased humans towards a male identity, the appeared to be neither necessary nor sufficient for a male identity or masculine behavior.[210]

Within the literature covered by this paper, the earlier paradigm of distinct and whole male or female brains collapsed based on two pieces of information, both publicized[211] at the end of 2015. Firstly, information published by Joel *et al.* suggested that human brains "consistently at one end of the 'maleness-femaleness' continuum are rare. Rather, most brains are comprised of unique 'mosaics' of features, some more common in females compared with males, some more common in males compared with females, and some common in both females and males."[212] Secondly, and more internally, researchers within developmental neuroendocrinology discovered that non-neuronal cells, specifically immune cells, were crucial for masculinization in the lab rats they used.[213] Rather memorably, the researchers publishing the immune mechanism of sex differentiation, McCarthy *et al*., stated that:

> *"It is conceivable that all end-points induced by estradiol and/or testosterone would involve the same signal transduction pathway and thus would be predicted to differentiate to the same degree within one individual and to the same degree across individuals if the hormone exposure is equivalent — a sort of "Unified Field Theory" of sex differences in the brain. But no matter how desirable such a scenario might be, it isn't true."*[214]

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[210] Wilson, "Androgens, Androgen Receptors, and Male Gender Role Behavior," 2001, p. 358-366. Bao, Ai-Min; Swaab, Dick F. "Sexual differentiation of the human brain: relation to gender identity, sexual orientation and neuropsychiatric disorders." *Frontiers in Neuroendocrinology 32*(2): April 2011, p. 214-226; DOI: 10.1016/j.yfrne.2011.02.007.
[211] The information in footnote 110 was a review of a number of pieces of recent research from that group.
[212] Joel, Daphna; Berman, Zohar; Tavor, Ido; Wexler, Nadav; Gaber, Olga; Stein, Yaniv; Shefi, Nisan; Pool, Jared; Urchs, Sebastian; Margulies, Daniel S.; Liem, Franziskus, Liem; Hänggi, Jürgen; Jäncke, Lutz; Assaf, Yaniv. "Sex beyond the genitalia: The human brain mosaic." *Proceedings of the National Academy of Sciences 112*(50): 15 December 2015; DOI: 10.1073/pnas.1509654112.
[213] McCarthy *et al.*, "Surprising origins of sex differences in the brain." 2015, p. 3-10.
[214] Ibid., p. 6.

Desirability aside, this type of statement created the space for three broad types of theoretical responses.

Firstly, a 'new'[215] type of sex difference emerged—"latent" sex differences, or sex differences in one or more mechanisms that have no higher level sex differentiated representations.[216] Mosaic brains, in part bolstered by the multiple influences on sex differentiation (§2.1), could more easily have distinct effects that in some way compensated for each other.[217] Secondly, and more generally, the theory of "canalization" became a new way to conceive of sex differences. As the aforementioned immune researchers[218] (and a number of others)[219] argued, the existing sex differences represented "canals" of development, in which the possibility for phenotypic variation from internal and external sources was mitigated and counterbalanced, creating two distinct phenotypic sexes with low internal variability.[220] Indeed, the existence of the differentiated sexes was argued to be necessary for reproductive fitness[221] and species survival.[222] To recall an earlier criticism, however, that there are two distinct phenotypic sexes with low internal variability may be an unrealistic assumption, at least for some species or in some contexts, "in part [because] the organisms studied most [are] typically inbred

[215] Latent sex differences had been proposed in 2004 by Geert J. de Vries, though it was absent from the literature covered by this thesis until approximately 2015. de Vries, Geert J. "Minireview: Sex differences in adult and developing brains: compensation, compensation, compensation." *Endocrinology 145*(3): March 2004, p. 1063-1068; DOI: 10.1210/en.2003-1504.
[216] Oberlander, Joseph G.; Woolley, Catherine S. "17β-Estradiol Acutely Potentiates Glutamatergic Synaptic Transmission in the Hippocampus through Distinct Mechanisms in Males and Females." *Journal of Neuroscience 36*(9): 2 March 2016, p. 2677-2690; DOI: 10.1523/JNEUROSCI.4437-15.2016.
[217] McCarthy *et al*., "Surprising origins of sex differences in the brain." 2015, p. 6.
[218] Ibid., p. 6.
[219] Zup, Susan L.; Madden, Amanda M. K. "Gonadal hormone modulation of intracellular calcium as a mechanism of neuroprotection." *Frontiers in Neuroendocrinology 42*: July 2016, p. 40-52; DOI: 10.1016/j.yfrne.2016.02.003.
[220] McCarthy *et al*., "Surprising origins of sex differences in the brain." 2015, p. 6.
[221] Ibid., p. 5.
[222] Walker, Deena M.; Gore, Andrea C. "Epigenetic impacts of endocrine disruptors in the brain." *Frontiers in Neuroendocrinology 44*: January 2017, p. 1-26; DOI: 10.1016/j.yfrne.2016.09.002.

birds and mammals … the perspective engendered is that variation is categorical (male vs. female) rather than continuous."[223]

Finally, likely in part because of the theoretical and practical complexity involved in the multi-modal sex differentiation but significantly influenced by the theoretical changes covered in all previous sections of this chapter, the relationships between development, structure, function, and behavior became significantly more theoretically complex. After discussing the historical difficulties "connecting neuroanatomical variability with behavioral output,"[224] McCarthy *et al*. referenced "a sentiment expressed by Per Södersten almost 30 years ago – 'The search for morphological sex differences in adult rat brains that are caused by the 'organizing effect of perinatal androgen' and that can be related to sex differences in behavior has not been fruitful and may continue unrewarded' (Södersten, 1987) – a sentiment that arguably still holds true today."[225] Their response, which they were not alone in,[226] argued that the connections between neuroanatomy were context-dependent but still fundamentally dependent on the underlying neuroanatomy, that "[s]election pressure is brought to bear on behavior, not on neuroanatomy directly, and therefore ipso facto if a neuroanatomical endpoint is canalized then it must be having a significant impact on behavior."[227] Overall, discussions in this literature on the types of brains that can exist ended, in 2019, having contextualized and complicated the assumed "male" and "female" brain but without having given up the idea entirely.

[223] Crews, "Epigenetic modifications of brain and behavior," 2011, p. 395.
[224] McCarthy *et al*., "Surprising origins of sex differences in the brain." 2015, p. 8.
[225] Ibid.
[226] Zup and Madden, "Gonadal hormone modulation of intracellular calcium," 2016, p. 49. Walker and Gore, "Epigenetic impacts of endocrine disruptors in the brain," 2017, p. 4.
[227] Evolutionary discussions will be covered in more detail in chapter 4. McCarthy *et al*., "Surprising origins of sex differences in the brain." 2015, p. 8.

2.8 Conclusion

> *"In the fifty years since the organizational hypothesis was proposed, many sex differences have been found in behavior as well as structure*[sic] *of the brain that depend on the organizational effects of gonadal hormones early in development. Remarkably, in most cases we do not understand how the two are related. This paper makes the case that overstating the magnitude or constancy of sex differences in behavior and too narrowly interpreting the functional consequences of structural differences are significant roadblocks in resolving this issue."*[228]

The underlying assumption undermined between 2000 and 2019 was the assumption of simplicity. It would have been simple if sexual differentiation followed a simple trajectory in a limited time frame with a straight line of causality from gonadal hormone to masculinization, creating brains of one type or of the other with features that were causally connected to behaviors that fell cleanly into sexed/gendered categories. Across these aspects, a combination of new discoveries, theoretical shifts, and the self-reflection encouraged by the replication crisis consistently complicated or contextualized the assumption of simplicity. The organizational/activational theory was never disproven; instead, it crumbled under an onslaught of a thousand cuts. That being said, as the last section's discussion of "canalization" suggests, contextualization and complication had an effect but did not fully discredit or render irrelevant the idea of using sex/gender as a reliable behavioral, medical, and biological categorization. The question that remains about this theoretical replacement is a rather simple one: what changed?

[228] The McCarthy text in the last section existed in significant conversation with this text. de Vries, Geert J.; Södersten, Per. "Sex differences in the brain: the relation between structure and function." *Hormones and Behavior 55*(5): May 2009, p. 589, Abstract; DOI: 10.1016/j.yhbeh.2009.03.012.

Chapter 3: Experimental considerations and controversies

3.0: Introduction

What changed for the organizational/activational theory was, in no small part, whether researchers were willing to trust its accuracy after the replication crisis had made their scientific enterprise seem significantly shakier. While some of this chapter discusses specific theories and hypotheses about human and animal development and presents counterevidence, the intent is not to demonstrate that these theories and hypotheses are definitively incorrect. Instead, the intent is to demonstrate that these theories and hypotheses cannot be considered reliably confirmed or disconfirmed due to the publication environment before 2016, the experimental paradigms at play, and the assumptions underlying much of the research done between 2000 and 2019 in behavioral neuroendocrinology overall and on the organizational/activational hypothesis specifically. The realizations on the part of editors and researchers that previous information required closer scrutiny then encouraged further examination of previously accepted ideas. The scope of the replication crisis and the experimental nuances that interacted with it are (incompletely) covered here to give an idea of how widespread and endemic these issues were and to support my later analysis.

3.1: Any experiment's *absence* diminishes *me*, because I am involved in *Science*[229]

Before considering the experimental controversies directly, they must be contextualized. As a note, however, this section examines statistical issues in some depth, and while I have attempted to ensure that the statistical discussion is accessible, statistics is a complex field and removed from most other mathematical disciplines in its methods and theoretical framing.[230] As a broad outline, then, the structure of the statistical side of the replication crisis was this: scientists had two values which, together, quantify their statistical likelihood of being wrong about the existence and magnitude of a real effect. A p-value is a statistical test about the likelihood of false positives (seeing an effect when there is none); statistical power is a statistical test about the likelihood of false negatives (seeing no effect when there is one). Scientists and journals elevated the p-value alone as a central test of rigorousness, which left testing for false negatives by the wayside as well as providing unwarranted assurance about the scope and reliability of results. Scope, because a p-value tests for statistical significance rather than significance in a vernacular sense and so a result may not mean much practically; reliability, because statistical tests cannot detect poor data or bias (as covered more extensively in §3.2). Once the p-value became a central test of scientific rigor as elevated by scientists and journals, journals would preferentially publish statistically significant results. If a statistically significant

[229] A reference to the 1624 work *Devotions Upon Emergent Occasions, and severall steps in my Sicknes* by John Donne, specifically Meditation XVII's introductory section. The original phrase is "any mans *death* diminishes *me*, because I am involved in *Mankinde*". The section title retains the italicization and capitalization of the original, though I have chosen to use standard modern grammar and spelling for readability.

[230] Frank Hampel, "Is Statistics Too Difficult?", *The Canadian Journal of Statistics/La Revue Canadienne de Statistique 26*, no. 3: 1998, DOI: 10.2307/3315772 p. 497-513. W. F. Bodmer, "Understanding Statistics," *Journal of the Royal Statistical Society Series A 148*, no. 2: 1985, DOI: 10.2307/2981942, p. 69-81. David Freedman, "From Association to Causation: Some Remarks on the History of Statistics," *Statistical Science 14*, no. 3: 1999, jstor.org/stable/2676760, p. 243-258.

effect was not evident, researchers were under systemic pressure to generate a statistically significant result, either by reanalyzing data with new hypotheses or adding or removing participants. Collectively, this pressure incentivized publishing papers announcing an effect, false or not, rather than the absence of an effect, false or not, without effectively ensuring that the papers were rigorous overall.[231] Some individual papers would be fraudulent to meet these pressures, but the main effect of this incentive set was that the available published research would appear sound and complete without necessarily being either.

Beginning around January 2016, the *Journal of Neuroscience* and *eNeuro* began publishing papers about the 'replication crisis', an ongoing issue across scientific disciplines[232] about researchers' inability to replicate previously published results. Within the biomedical,[233] psychological,[234] and economics[235] disciplines, fields which largely contain the research published in behavioral neuroendocrinology, replication projects failed to replicate the original

[231] Freedman's paper (ft. 2) predicted this general outcome in 1999, saying: "investigators often try to base causal inference on statistical models. With this approach, *P*-values play a crucial role. The technology is relatively easy to use and promises to open a wide variety of questions to the research effort. However, the appearance of methodological rigor can be deceptive." (p. 255, italics in original)

[232] Monya Baker, "1,500 scientists lift the lid on reproducibility," *Nature 533*: 2016; DOI: 10.1038/533452a, p. 452-454.

[233] Katherine S. Button, "Statistical Rigor and the Perils of Chance," *eNeuro* 3(4): 14 July 2016; DOI: 10.1523/ENEURO.0030-16.2016, p. 1.

[234] B. J. Wiggins and C. D. Christopherson, "The replication crisis in psychology: An overview for theoretical and philosophical psychology," *Journal of Theoretical and Philosophical Psychology 39*(4): 2019, DOI: 10.1037/teo0000137, p. 202-217.

[235] Economics lagged behind psychology and the natural sciences in self-evaluation and replication, see Maren Duvendack, Richard Palmer-Jones, and W. Robert Reed, "What is Meant by 'Replication' and Why Does It Encounter Resistance in Economics?", *American Economic Review 107*(5): 2017, DOI: 10.1257/aer.p20171031, p. 46-51, but an early study in 2015 by two US Federal Reserve economists suggested less than half of the published economics research is replicable. Andrew C. Chang and Phillip Li. "Is Economics Research Replicable? Sixty Published Papers from Thirteen Journals Say 'Usually Not'," *Finance and Economics Discussion Series 2015-083* published by the Board of Governors of the US Federal Reserve System: 2015, DOI: 10.17016/FEDS.2015.083, p. 1-25.

results in approximately half[236] of tested experiments.[237] In *eNeuro,* as the Society for Neuroscience's procedural and theoretical journal, the most consistent explanation for the crisis blamed a widespread lack of statistical literacy among journal staff and scientists.[238] At the level of research, experiments were frequently designed around the confirmation or disconfirmation of hypotheses, specifically null hypothesis significance testing. Two statisticians writing in *eNeuro* explained:

> *"In this approach, we ask a qualitative question: Does this drug influence learning? The data collected is then reduced down to a test statistic and p value, and from these we make a qualitative conclusion: Yes, this drug influences learning. Results are often treated as definitive, so there can be little motivation to conduct replications: Why test the drug again now that it has been shown to work?"*[239]

The main flaw of this approach, besides its seeming definitiveness, is that it gives a single answer when a range of possibilities are more justified by the data.

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[236] With a wide margin of error: as low as a third of published papers and as high as two-thirds. I have chosen the language of fractions to avoid any appearance of false precision that might emerge from numerical percentages. See ft. 9 for the methodology to determine the estimate.

[237] This estimate is based primarily on the clinical trial 'success' rate of approximately 50-60%, from Button "Statistical Rigor," p. 4, the Reproducibility Project: Psychology's estimate of 36% replicability in psychology, Wiggins and Christopherson "The replication crisis in psychology," p. 206, and the economics replication figure of 49% with author assistance from Chang and Li. Behavioral neuroendocrinology takes primarily from psychology and biomedicine, though has some economics-minded researchers as well. Additionally, it is unclear whether 'clinical trial success' should be the metric for biomedicine, as it may represent the best case. Psychology, psychiatry, and neuroscience have an "incredible 85-90%" *a priori* hypothesis confirmation rate, (Button "Statistical Rigor," p. 2) suggesting that neuroscience research may have a replicability problem closer in scale to psychology than preclinical medicine. On the other hand, many of the theorized applications of endocrinological research suggest clinical applications for their work, and so perhaps the clinical trial rates are more applicable. This is all aside from the fact that I have focused on a particular subject, and so broad-scale percentages may or may not be representative of the organizational/activational sex difference research this thesis focuses on.

[238] Button, "Statistical Rigor"; Oswald Steward, "A Rhumba of 'R's': Replication, Reproducibility, Rigor, Robustness: What Does a Failure to Replicate Mean?", *eNeuro 3*(4): 7 July 2016, DOI: 10.1523/ENEURO.0072-16.2016, p. 1-4; Ray Dingledine, "Why Is It so Hard to Do Good Science?", *eNeuro 5*(5): 4 September 2018, DOI: 10.1523/ENEURO.0188-18.2018, p. 1-8; Christophe Bernard, "Changing the Way We Report, Interpret, and Discuss Our Results to Rebuild Trust in Our Research," *eNeuro 6*(4): 1 August 2019, DOI: 10.1523/ENEURO.0259-19.2019, p. 1-3; Robert J. Calin-Jageman and Geoff Cumming, "Estimation for Better Inference in Neuroscience," *eNeuro 6*(4): 1 August 2019, DOI: 10.1523/ENEURO.0205-19.2019, p. 1-11.

[239] Calin-Jageman and Cumming, "Estimation for Better Inference in Neuroscience", p. 2.

To say that a hypothesis is 'statistically significant' based on a p-value less than 0.05 is simply to say that the null hypothesis of no effect is not within the 95% confidence interval. As long as the range of effects supported by the data does not precisely include zero, the range of effects may begin at, for example, 0.1% better at stimulating information retention than a placebo. This cutoff approach marries poorly with the relatively small sample sizes in human research, as small sample sizes widen the confidence interval. For example, as the statisticians in *eNeuro* noted, a 2014 report in *Nature Neuroscience* tested the effects of caffeine on memory in 71 individuals. That *Nature Neuroscience* paper suggested that their evidence showed a statistically significant increase in memory consolidation after caffeine administration, meaning that caffeine improves memory. Reinterpreting the data with estimation statistics, however, revealed that the extrapolated population value could, with 95% reliability,[240] lie anywhere between a 0.2% and a 62% increase in memory consolidation without mathematically being particularly surprising. "The most appropriate interpretation at this point would be very modest: *caffeine probably does not impair memory consolidation*.[italics in original]"[241] "Moreover," as the statisticians point out, "this wide range of possibilities is optimistic, as it is based on uncertainty due only to sampling error with the assumption that all other sources of error and bias are negligible."[242]

One of the other aspects blamed for the replication crisis was endemically low statistical power. Statistical power is the probability that an experimental design will detect a real effect rather than giving a false negative. (p-values and confidence intervals are intended to protect

[240] 95% confidence intervals mean that repeating the same experiment on the same population and creating a 95% confidence interval each time will ensure that the confidence interval contains the population value in 95% of the replicated experiments, assuming all other sources of bias and error are negligible.
[241] Calin-Jageman and Cumming, "Estimation for Better Inference in Neuroscience", p. 3.
[242] Ibid.

against false positives.) Statistical power can only be approximated in hindsight,[243] although experimental designs can reduce sources of variability and increase sample sizes to increase statistical power. Crucially, it is the interaction between p-values (or confidence intervals), statistical power, and the number of true hypotheses within the total number of available hypotheses[244] that determine the percent of statistically significant results which detect real effects and the number of insignificant results which correctly confirm the null hypothesis.[245] Holding the threshold for statistical significance constant, if the statistical power is low, many hypotheses will fail to detect real effects; if the pool of hypotheses has a low number of true hypotheses, the likelihood that any individual statistically significant result is a real effect will be low; and if the number of true hypotheses and the statistical power are both low, the number of falsely significant results may outweigh the number of truly significant results due to the low number of true positives and high number of false negatives.

The effect of publication biases only compounded the above problems. As the editor of *eNeuro* recalled in 2019:

> *"When I reflect on my scientific trajectory, I realize that I became more and more p focused, and anything with a $p < 0.05$ became the truth, not only for my research but also when evaluating that of my colleagues. … Like me, you must be amazed when reading papers from high-profile journals (but not only from these journals) reporting smooth stories, in which everything works, everything is claimed to be significant, and thus true, with a minimal number of experiments. At best, I am skeptical. The conclusions are very*

[243] At least in any field where 'real' effects are not known, such as behavioral neuroendocrinology. Katherine S. Button, John P. A. Ioannidis, Claire Mokrysz, Brian A. Nosek, Jonathan Flint, Emma S. J. Robinson, and Marcus R. Munafò, "Power failure: why small sample size undermines the reliability of neuroscience," *Nature Reviews Neuroscience 14*: 2013, DOI: 10.1038/nrn3475, p. 365-376.
[244] Another value that cannot be known except in hindsight.
[245] Dingledine, "Why Is It so Hard to Do Good Science?", p. 5-6.

> *strong, leaving no room for doubt (otherwise you cannot publish in these journals). This practice has contributed in no small way to today's reproducibility crisis. Here I am not talking about scientists faking results or selecting the results that support a theory; this has always existed. The problem is more profound: we transformed a number that is linked to a probability into a goal to achieve, a definitive answer. In short, we distorted the p value to make it say what it is not designed to say."*[246]

Not only did this practice of publishing only statistically significant results consign an unknown number of false negatives to obscurity and elevate an unknown number of false positives, it also created a new source of error: overinflated effect sizes in small, exploratory studies, or "the winner's curse".[247] Small studies have wide confidence intervals, and so achieving a statistically significant result requires either that the difference between groups be extremely large or, more frequently, that the researchers have an unrepresentative sample that inflates or even creates the apparent differences between groups.[248]

Finally, due to the ease of analysis and the existence of a singular metric of success, "researchers [were] more vulnerable than ever to fooling themselves."[249] Beyond possible cognitive biases, such as "[spinning] narratives out of datasets of questionable relevance[and seeking] patterns in noisy data,"[250] computational power and statistics packages allowed for such data-bending processes as 'hypothesizing after results are known' (HARKing) without making

[246] Bernard, "Changing the Way", p. 1-2.
[247] Button, "Statistical Rigor", p. 2.
[248] Creation of statistically significant results is a probabilistic certainty, i.e. it will happen eventually with enough repetitions. The 95% confidence interval means that, if experiments are repeated measuring the same parameter in the same population and other sources of bias and error are negligible, 95% of the confidence intervals will contain the population value. An individual confidence interval has no guarantee that it does, in fact, contain the population value, even if all other sources of bias and error are or could be fully accounted for or removed, hence "creates." Such is the nature of probability.
[249] Button, "Statistical Rigor", p. 2.
[250] Dingledine, "Why Is It so Hard to Do Good Science?", p. 6

the *post hoc* hypotheses explicit, as well as p-hacking, or searches for statistically significant effects with repeated analyses of the same data set. As one writer pointed out:

> *"There is good evidence that such undisclosed flexibility in analysis is commonplace, both from surveys of research practice (John et al., 2012)*[251] *and by the incredible 85–90% of neuroscience/psychology/psychiatry papers claiming evidence for an a priori hypothesis (Fanelli, 2010b).*[252] *Either a high proportion of researchers are researching redundant questions, where the answer is already known, or they are exploring their data to find a significant result and then hypothesizing afterward (Simmons et al., 2011).*[253]"[254]

In combination, the publication environment leading up to the replication crisis, particularly for researchers with less funding and early in their careers, would most reward (with publication, future career prospects, future grant money, etc.) experiments detecting an effect or difference that passed a threshold of statistical significance.

[251] Leslie K. John, George Loewenstein, and Drazen Prelec, "Measuring the Prevalence of Questionable Research Practices With Incentives for Truth Telling," *Psychological Science 23*(5): May 2012, DOI: 10.1177/0956797611430953, p. 524-532.
[252] Daniele Fanelli, "'Positive' Results Increase Down the Hierarchy of the Sciences," *PLoS ONE 5*(4), ID e10068: 7 April 2010, DOI: 10.1371/journal.pone.0010068, p. 1-10.
[253] Joseph P. Simmons, Leif D. Nelson, and Uri Simonsohn, "False-Positive Psychology: Undisclosed Flexibility in Data Collection and Analysis Allows Presenting Anything as Significant," *Psychological Science 22*(11): 17 October 2011, DOI: 10.1177/0956797611417632, p. 1359-1366.
[254] Button, "Statistical Rigor", p. 2.

3.2: Technologic[255]

"Voxel-based regressional analysis is completely automated; therefore, reliability is no longer a concern."[256]

"Over the next 50 years, approaches used in behavioral neuroscience will more closely resemble the sophisticated methods being used to functionally dissect neural circuits. Computer vision technology will enable fully automated, high-throughput, unbiased behavioral analysis, exponentially pushing the field forward."[257]

An unwarranted faith in technological solutions also compounded the replication crisis' problems. The first quote above was published in 2001 and the second was published in 2020. Both show a general attitude that computational analysis was believed to be more accurate (reliable, unbiased) and more efficient (automated, high-throughput) than human analysis. However, there were multiple papers, particularly after 2016, that suggested that this rosy view of computational outputs was at least somewhat misleading. As brought up in the last paragraph of §3.1, computational power was an important part of reverse-engineering statistically significant results from an existing dataset, or HARKing (Hypothesizing After Results are Known), as running multiple mathematically complex statistical tests became significantly easier as computational power increased. More broadly, while computers are reliable and unbiased in

[255] A reference to the *Daft Punk* song of the same name from the 2005 album "Human After All." The title likely means "technology logic" collapsed into a single word ("techno-logic"). The lyrics are spoken by a computerized voice and list the types of technological maneuvers one can do with digital objects (e.g. "Charge it, point it, zoom it, press it, snap it, work it, quick erase it").

[256] J. C. Pruessner, D. L. Collins, M. Pruessner, and A. C. Evans, "Age and Gender Predict Volume Decline in the Anterior and Posterior Hippocampus in Early Adulthood," *Journal of Neuroscience 21*(1): 1 January 2001, DOI: 10.1523/JNEUROSCI.21-01-00194.2001, p. 199.

[257] Cara M. Altimus, Bianca Jones Marlin, Naomi Ekavi Charalambakis, Alexandra Colón-Rodríguez, Elizabeth J. Glover, Patricia Izbicki, Anthony Johnson, Mychael V. Lourenco, Ryan A. Makinson, Joseph McQuail, Ignacio Obeso, Nancy Padilla-Coreano and Michael F. Wells, "The Next 50 Years of Neuroscience," *Journal of Neuroscience 40*(1): 2 January 2020, DOI: 10.1523/JNEUROSCI.0744-19.2019, p. 103.

the sense that they will accurately perform a computer program's instructions quickly and precisely, their reliability and bias in respect to reality depends, among other aspects,[258] on the dataset(s) used, the instructions given by the programmers, and the relationship between the data and reality.

The datasets used for generating models matter significantly. For example, a paper published in *The Journal of Neuroscience* in 2017 tracked gray matter volume and gray matter density and found that, contrary to a significant proportion of the existing literature, the metrics were often inversely correlated and not functionally equivalent.[259] For example, the fact that men and boys tended to have greater gray matter volume than women and girls had been considered paradoxical; gray matter volume and intelligence were correlated in adults, but there was no intelligence difference between men and women or boys and girls.[260] What these authors noted, however, was that women and girls tended to have a higher gray matter density, which perhaps "compensated for" the difference in volume.[261] Why had this relationship not been noted before and why had gray matter volume and gray matter density been assumed to be correlated or equivalent? The authors suggested that many of the previous studies had "focused on neurodegeneration. Such diseases result in neuronal loss, which causes direct decreases in all

[258] There was a puzzling remark about "motherboard clocks" in one editorial, Christophe Bernard, "Editorial: Code Case – Investigating Transparency and Reproducibility," *eNeuro 4*(4): 10 August 2017, DOI: 10.1523/ENEURO.0233-17.2017, p. 1-1. Various programs use time information to run their simulations, and overclocked components (to get more calculations per second out of processing units beyond default specifications) could perhaps cause random number generation using time data to be less than completely random (e.g. if the overclocked unit was not on an overclock-compatible motherboard), but I did not find specific evidence as to what this was referring to.
[259] Efstathios D. Gennatas, Brian B. Avants, Daniel H. Wolf, Theodore D. Satterthwaite, Kosha Ruparel, Rastko Ciric, Hakon Hakonarson, Raquel E. Gur and Ruben C. Gur, "Age-Related Effects and Sex Differences in Gray Matter Density, Volume, Mass, and Cortical Thickness from Childhood to Young Adulthood," *Journal of Neuroscience 37*(20): 17 May 2017, DOI: 10.1523/JNEUROSCI.3550-16.2017, p. 5065.
[260] Ibid., p. 5070.
[261] Ibid., p. 5070.

gray matter measures."[262] The MRI analysis packages had[263] accurately measured the gray matter volumes or gray matter densities of the brains they had been given. The fault lies not in the reliability of the MRI analysis, but in the choice of the subset of all possible data that had formed the basis of these assumptions about people in general.

In addition, there were the programs for scanning and analysis themselves. The computational programs which calculated regressions and analyzed interrelated areas between subject scans became almost invisible parts of scientific inquiry, as neutral and objective as a photograph.[264] In both quotes above, the (hypothetical, in the second case) programs involved were assumed to be non-factors in the eventual results, simply facilitating the researchers' inquiries. This has been, in practice, a practically uncheckable assumption, both by the researchers and by the editorial staff. While some data has been shared openly, particularly since 2010, most scans for most research papers are not available.[265] Just one scanning analysis software program, the Statistical Parametric Mapping program, which is open source from University College London's Functional Image Laboratory, has a public repository begun in 1994 which has been frequently updated since,[266] is written in a mix of two programming languages (MATLAB and C), and relies on fairly complex mathematical principles.[267] Investigating the program directly would be a daunting task. The fact that these methods are difficult to check directly explains why one of the most popular statistical analysis packages of

[262] Ibid., p. 5071.
[263] Most likely, at least; see the next paragraph.
[264] Lorraine Daston and Peter Galison, *Objectivity*, Zone Books (Princeton, NJ): 2010.
[265] Anders Eklund, Thomas E. Nichols, and Hans Knutsson, "Cluster failure: Why fMRI inferences for spatial extent have inflated false-positive rates," *PNAS 113*, no. 28: 12 July 2016, DOI: 10.1073/pnas.1602413113, p. 7904.
[266] The github repository spm/spm's oldest commit is available at github.com/spm/spm/tree/56569620f8, and dates from March 21, 1994, though significant changes were made in 2005. Some of the changes committed on February 28, 2005 (github.com/spm/spm/tree/ea707eb42c) remain in place to the current version of the software as of December 31, 2023.
[267] Linear algebra, 3D integrals, etc. See www.fil.ion.ucl.ac.uk/spm/doc/books/hbf2/.

functional MRIs had an undetected bug for fifteen years that had been insufficiently compensating for multiple comparisons and overestimating statistical significance, discovered by a group intentionally testing these software packages for their false positive rates using publicly available data.[268] Software obfuscates analysis procedures even as it makes some types of analysis possible, and the general absence of skepticism about the imaging process and the analysis afterwards shows that these techniques had been invisible during most of this period.[269] A 2017 editorial in *eNeuro* suggesting that "caution always needs to be exerted and a technique never taken for granted" is the only implicit or explicit discussion in the four journals about anything approaching this topic.[270]

Finally, there is the relationship between data and reality. A new set of editorial standards published for *eNeuro* in 2017 mentioned that computational studies and modeling papers can fail to reproduce—something that, as the editor of *eNeuro* noted, seems surprising. "After all, they are based on mathematics, and mathematics cannot lie, can they?"[271] This question leads toward why computers are not simply 'reliable'. Mathematics cannot lie—internally, at least. The relationship between any internally consistent system of mathematics and reality, however, is not necessarily so simple. Euclidean geometry is internally consistent, but knowledge about astrophysics post-Einstein suggests it cannot completely describe the physics of the universe.[272]

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[268] Eklund *et al.*, "Cluster failure," p. 7903-7904.
[269] Marina Picciotto, "Analytical Transparency and Reproducibility in Human Neuroimaging Studies," *Journal of Neuroscience 38*(14): 4 April 2018, DOI: 10.1523/JNEUROSCI.0424-18.2018, p. 3375-3376, for example, explicitly discusses how to make neuroimaging more replicable but does not raise the possibility of unreliable techniques.
[270] Christophe Bernard, "Editorial: Experimental Bias in Electrophysiological Studies," *eNeuro 4*(6): 26 December 2017, DOI: 10.1523/ENEURO.0432-17.2017, p. 2.
[271] Bernard, "Editorial: Code Case", p. 1.
[272] Black holes, for example, which severely distort spacetime, are extremely difficult to conceptualize or describe in spacetime without violating Euclid's 5th postulate, the postulate of parallel lines. While non-Euclidean spaces can be 'embedded' in Euclidean spaces, such the non-Euclidean two-dimensional surface of a sphere being embedded in a three-dimensional Euclidean space, non-Euclidean spaces cannot be geometrically projected onto Euclidean ones with the same dimensions without distortion, which is why all flat maps of the earth have some amount of distortion.

Computational tools cannot account for researchers who have made incorrect assumptions about the relevance or representativeness of the data the computer analyses, let alone data violating statistical assumptions such as unbiased sampling or independence.[273] Failures to reproduce are an expected outcome if the final published model required data 'maintenance' or significant unrecorded assumptions.[274] Additionally, consider the second quote at the beginning of the section. While it would be the computer reviewing the hours of footage, humans would have to tell it what visual elements would represent a behavior. The computer's answers are only as good as the program it was given to execute (as well as the data it is given to analyze), and it is always humans who are writing the program (and picking the data).[275] All analyses are built on assumptions, justified or otherwise, and computers, like mathematical systems, can only detect failures in internal logic.[276]

For most of the period covered by this thesis, computational tools generally obfuscated that these assumptions were being made at all. The general lack of statistical literacy mentioned in §3.1 played a significant role, as did researcher assumptions about their data and the impenetrability of the programs, but a full exploration of the logic, or perhaps fallacies, behind this computational trust is beyond the scope of this thesis. Few researchers questioned their tools until larger systemic issues or meta-analyses suggested the tools had been failing for some time. The papers covered here have been the exception rather than the rule, questioning what most

[273] In computation, this concept is often phrased as either "garbage in, garbage out" or more specifically "bias in, bias out".
[274] Marcin Miłkowski, Witold M. Hensel, and Mateusz Hohol. "Replicability or reproducibility? On the replication crisis in computational neuroscience and sharing only relevant detail," *Journal of Computational Neuroscience 45*(3): 31 October 2018, DOI: 10.1007/s10827-018-0702-z, p. 163-172.
[275] Programs 'trained' on data combine both basic problems; humans will be specifying what the 'correct' answers are for the data as well as choosing (and often generating) the dataset that the programs are trained on.
[276] Throwing errors, for example, if one part of a program violates the assumptions of another part.

researchers took for granted. When researchers argued that "reliability is no longer a concern"[277] or that the data would be "unbiased"[278] once data gathering and analysis was made the responsibility of a machine, it suggests that researchers fooled themselves with the assurance that their computational tools could not make a mistake—and so, neither could they.

3.3: Sex and gender troubles[279]

Turning to the definitional issues, the terms 'sex' and 'gender' are generally understood to refer to, respectively, the biological and cultural aspects of male- or femaleness.[280] That generality, however, comes with various caveats. Firstly, neither of these terms are simply defined or uncontested in actual usage, including in the literature considered by this thesis.[281] While sex generally refers to one's genitalia, internal gonads, sex chromosomes, reproductive capacity, and secondary sex characteristics (e.g. facial hair and breasts), many types of individuals (including men with gynecomastia, women with facial hair, transgender people using hormones, those who have had hysterectomies, and those with differences of sexual development) will fall out of binary female/male divisions in some contexts, leading to a recent paper arguing for 'sex contextualism'.[282] Additionally, of course, gender's troublesome nature should require little introduction:[283] gendered self-identification, gendered behaviors, gender

[277] Pruessner *et al.*, "Age and Gender Predict Volume Decline," p. 199.
[278] Altimus *et al.*, "The Next 50 Years of Neuroscience," p. 103.
[279] A reference to Judith Butler's 1988 book *Gender Trouble*; see ft. 55.
[280] Charlene L. Muehlenhard and Zoe D. Peterson, "Distinguishing Between *Sex* and *Gender*: History, Current Conceptualizations, and Implications." *Sex Roles 64*: 2011, p. 791-803, DOI: 10.1007/s11199-011-9932-5.
[281] For example, one paper discussed the gendered identification of male mice lacking the signaling component PAC1 to describe their altered sexual behavior and sociosexual investigation of or aggression toward other mice. While this choice could be justified as social behavior in a non-human species and therefore closer to gender than sex, the authors do not directly justify their choice of terminology. Arnaud Nicot, Timothy Otto, Philippe Brabet, and Emanuel M. DiCicco-Bloom, "Altered Social Behavior in Pituitary Adenylate Cyclase-Activating Polypeptide Type I Receptor-Deficient Mice," *Journal of Neuroscience 24*(40): 6 October 2004, DOI: 10.1523/JNEUROSCI.1910-04.2004, p. 8786-8795.
[282] Sarah S. Richardson, "Sex Contextualism," *Philosophy, Theory, and Practice in Biology 14*(2): 2022, DOI: 10.3998/ptpbio.2096, p. 1-17.
[283] Judith Butler, *Gender Trouble*, 2nd ed., Routledge (New York): 2006.

roles, gendered appearance, secondary sex characteristics, sexuality, and sex/romantic role behavior all played (and play) a role in how one identified to themselves and others, and also how one was identified by others, as the experiences of transgender (and homosexual) individuals demonstrates.[284] There are also those who used the terms functionally as synonyms,[285] and those who used the terms sex/gender or gender/sex to articulate their intertwined aspects,[286] although these uses were both less common.

Secondly, this conceptualization of sex and gender lends itself to dualisms: Cartesian, perhaps,[287] but also the division between nature and nurture, or between biology and culture. In the literature covered by this thesis, that the behavioral differences between men and women were overwhelmingly called "sex differences"[288] rather than "gender differences" implied the differences' natural, biological origin. These differences were, in fact, so 'biological' that sex differences could be consistent across species.[289] Sex, the literature collectively argued, was the substrate that gender was built on top of, and sex limited and delimited the ways that gender could be expressed. Indeed, many aspects of sex or gender frequently argued to be cultural,[290]

[284] Raine Dozier, "Beards, Breasts, and Bodies: Doing Sex in a Gendered World," *Gender and Society 19*(3): June 2005, DOI: 10.1177/0891243204272153, p. 297-316.
[285] Nicot *et al.*, "Altered Social Behavior," p. 8786-8795.
[286] Edward Schiappa, "A brief history of defining sex and gender" in *The Transgender Exigency: Defining Sex and Gender in the 21st Century*, Routledge (London): 2022, DOI: 10.4324/9781003250494-4, accessed through taylorfrancis.com/books/oa-edit/10.4324/9781003250494/transgender-exigency-edward-schiappa, p. 15-34.
[287] Judith Butler, "Sex and Gender in Simone de Beauvoir's Second Sex," *Yale French Studies 72*: 1986, jstor.org/stable/2930225, p. 35-49.
[288] Or, as seen in the next section, dimorphisms.
[289] See §3.7. Cf. Kelly M. Dumais and Alexa H. Veenema, "Vasopressin and oxytocin receptor systems in the brain: Sex differences and sex-specific regulation of social behavior," *Frontiers in Neuroendocrinology 40*: January 2016, DOI: 10.1016/j.yfrne.2015.04.003, which suggested that the underlying mechanisms of the vasopressin and oxytocin receptors were often at least somewhat structurally consistent across species and did tend to mediate sex-typed social behaviors, but which behaviors were different between sexes was not consistent between species. In other words, while the mechanisms of sex differences in mammals did appear to be at least somewhat consistent, the behavioral 'outputs' of those sex differences were more often species-specific.
[290] Schiappa, "A brief history of defining sex and gender" in *The Transgender Exigency*, p. 18, 30.

such as different levels of physical aggression[291] and general masculine[292] and feminine[293] behavior, were attributed to sex-typed biological developmental factors. The explanations that sex difference researchers preferred included genetic factors, prenatal environments, epigenetics, and circulating hormones; socialization tended to be a hypothesis of last resort.[294] Sexual differentiation formed the bedrock of gender differences, creating consistent patterns of sexed (and thereby gendered) behavior that were often visible across species, such as aggression in males and childrearing behavior in females. The literature covered by this thesis carried very little implication that gender differences could inscribe themselves into bodies and brains—excepting the papers suggesting that the brain was plastic and responsive to experience briefly touched on in Chapter 1.

By noting this aspect of the research, we may consider a third caveat. Sex, or the biological identification of bodies by medical professionals or biologists, sex/gender (or gender/sex), or the 'cultural' identification of bodies by others through a combination of secondary sex characteristics and gendered behaviors, and gender, or the self-identification of one's identity in a gendered framework, did (and do) not seem to be either fully separate or fully identical.[295] The difficulty of disentangling sex and gender posed a practical problem for sex difference researchers in behavioral neuroendocrinology: how could one be sure that the sex differences were not, in fact, gender differences? Some researchers opted to study less-socialized infants and young children, some researchers conducted twin studies, some researchers used

[291] Cohen-Bendahan *et al.,* "Is there an effect of prenatal testosterone on aggression and other behavioral traits?"
[292] Pasterski *et al.,* "Prenatal hormones and childhood sex segregation."
[293] Blanchard *et al.*, "Fraternal Birth Order and Birth Weight in Probably Prehomosexual Feminine Boys."
[294] Yan Wang, Haoda Wu, and Zhong Sheng Sun, "The biological basis of sexual orientation: How hormonal, genetic, and environmental factors influence to whom we are sexually attracted," *Frontiers in Neuroendocrinology 55*, ID 100798: October 2019, DOI: 10.1016/j.yfrne.2019.100798, p. 1-14.
[295] Schiappa, "A brief history" in *The Transgender Exigency.*

cross-cultural samples, some researchers studied LGBTQIA+ individuals or those with differences of sexual development, many researchers used model animals, some considered theory-based predictions about evolutionarily-selected sexually divergent behavior,[296] and a few researchers suggested that the magnitude, consistency, and ubiquity[297] of the observed correlation indicated that culture alone could not have produced the effect. Some of the assumptions behind these methodologies will be examined more critically in Chapter 4, but it should be acknowledged that researchers attempting to separate sex and gender (and gender/sex or sex/gender) effects could and can only use indirect methods for compelling ethical reasons. For example, healthy human fetal development can only ethically be non-invasively studied, and non-invasive methods, at least as of 2023, provide relatively low amounts of information. In turn, however, the proposition that any model animal system is appropriately modeling human fetal sexual differentiation could not be confirmed and was also difficult to disconfirm. This necessarily indirect approach required significant assumption 'scaffolding' around the information that could be gathered, as any model or experimental approach worked at a significant experimental remove from the questions about humans that researchers often sought to answer.

3.4: Defining a difference from a distinction[298]

> *"Dimorphic means of two forms, and therefore is a term best kept for those differences that are big and have little overlap. But terminology has a way of slipping and frequent*

[296] Commonly including differing pressures regarding aggression and childrearing. Kimberly H. Cox, Paul J. Bonthuis, and Emilie F. Rissman, "Mouse model systems to study sex chromosome genes and behavior: Relevance to humans," *Frontiers in Neuroendocrinology 35*(4): October 2014, DOI: 10.1016/j.yfrne.2013.12.004, p. 405.
[297] Bradley M. Cooke and Jill M. Weathington, "Human and animal research into sex-specific effects of child abuse," *Hormones and Behavior 65*(4): April 2014, DOI: 10.1016/j.yhbeh.2014.03.004, p. 417; McHenry *et al.*, "Sex differences in anxiety and depression," p. 42.
[298] A reference to the idiom "a distinction without a difference", an idiom that dates at least back to the 19th century (Mark Twain writes it in his essay "Concerning the American Language").

> *use leads to further imprecision. As a result, there has been an increasing tendency to equate 'se x differences' with 'sex dimorphisms' which simultaneously leads to a loss of attention to the all important question of magnitude and reliability. A central theme of this review will be that this process has occurred in the conflating of sex dimorphisms in reproductively relevant physiology and behavior and the attendant neurological substrates, with sex differences in cognition. This has occurred not only in the arena of basic science investigation but in the popular press as well."*[299]

An overlapping definitional issue that was more actively considered within the literature was the distinction, or lack thereof, between difference and dimorphism. The terms "dimorphic" or "dimorphism" were used to describe, among other aspects, levels of aggressive behavior,[300] levels of "sensation-seeking behavior",[301] the ratio of hand width to hand length,[302] and "the age of peak gray matter thickness."[303] All four of these would seem to be sex or gender "differences", as the two genders or sexes exhibit significant phenotypic overlap. In practice, despite the efforts of the above author, "difference" and "dimorphism" generally functioned as synonyms. Consider the following passage:

> *"**Sex differences in the brain**, where they exist, they are rarely absolute**. They imply differences in means, often with considerable overlap between males and females.** Nevertheless, it is critical to explore **sexual dimorphisms in the brain** for their impact*

[299] Margaret M. McCarthy and Anne T. M. Konkle, "When is a sex difference not a sex difference?", *Frontiers in Neuroendocrinology 26*(2): September 2005, DOI: 10.1016/j.yfrne.2005.06.001, p. 85-102.
[300] Benderlioglu and Nelson, "Digit length ratios predict reactive aggression in women, but not in men," p. 559; Cohen-Bendahan *et al.,* "Is there an effect of prenatal testosterone on aggression and other behavioral traits?"
[301] Cohen-Bendahan *et al.,* "Is there an effect of prenatal testosterone on aggression and other behavioral traits?"
[302] James T. Martin and Duc Huu Nguyen, "Anthropometric analysis of homosexuals and heterosexuals: implications for early hormone exposure," *Hormones and Behavior 45*(1): January 2004, DOI: 10.1016/j.yhbeh.2003.07.003, p. 34.
[303] Sisk and Zehr, "Pubertal hormones organize the adolescent brain and behavior," p. 166.

> *and therapeutic implications for disease. This is particularly the case for many neurological and psychiatric diseases. … With that knowledge it is irresponsible to ignore* ***sexual dimorphisms in the human brain****, as insights from* ***these differences*** *may help us better understand the etiology of* ***sex biased diseases*** *and ultimately may lead to better therapeutic approaches. In this special issue we explore* ***the sexually dimorphic nature*** *of several psychiatric and neurological disorders with reviews of both preclinical animal studies and clinical data. [bolding added]"*[304]

It is difficult to conclude that this paragraph was intended to suggest that 'dimorphism' and 'difference' were anything other than straightforwardly synonymous.

Two further definitional issues muddled 'differences' further. The first concerned whether sex differences included effects based on circulating sex hormone levels. If it did, then sex differences would not necessarily be permanent or lifelong. While the existence of transgender or nonbinary people receiving hormone therapy would fall into this category, young children and the elderly often have quite low circulating levels of sex hormones. Do people age into and then out of sex differences? Perhaps "hormonally modulated response[s]"[305] could better characterize these behaviors, though this distinction recalls the question of gonadal centrality to sex considered in §2.1 and the historical trend for "hormonal modulation" to become a significantly less simple phenomenon as discussed in §2.2, 2.3, and 2.6.

The second definitional issue is "sex similarity". In the context of this literature, "sex similarity" could refer to two different phenomena: either (1) a similar or identical (behavioral) endpoint which relies on different physiology/hormones/etc. or (2) a difference between males

[304] Larry J. Young and Donald W. Pfaff, "Sex differences in neurological and psychiatric disorders," *Frontiers in Neuroendocrinology 35*(3): August 2014, DOI: 10.1016/j.yfrne.2014.05.005, p. 253
[305] McCarthy and Konkle, "When is a sex difference not a sex difference?", p. 85.

and females only visible when a stressor is added.[306] The first of these ideas about sex similarities was originally[307] proposed by de Vries in 2004,[308] though only became influential beginning in 2012.[309] The second type of "sex similarity" was also characterized as a "latent sex difference," as mentioned in §2.7, and only emerged in 2015.[310] However, the original defining paper added the alternative definition that "the sexes appear to be similar at baseline but in fact have different neurochemical underpinnings that result in different vulnerabilities or divergent outcomes in response to the same stressor."[311] Therefore, while (2) appears to be simply approaching the same process as (1) from a different angle, beginning with output rather than mechanism, it is not. Working backward from output can allow one to discover a mechanism, but that "different neurochemical underpinnings" are responsible for the "different vulnerabilities or divergent outcomes in response to the same stressor" is an assumption. Specifically, it is the assumption that the trail of causality begins and ends at neurochemistry, which in turn assumes firstly that the brain is practically isolated from the body[312] and secondly that neurochemistry cannot be altered by other factors, which the effects of long-term stress, for example, suggest is

[306] Margaret M. McCarthy, Arthur P. Arnold, Gregory F. Ball, Jeffrey D. Blaustein and Geert. J. De Vries, "Sex Differences in the Brain: The Not So Inconvenient Truth," *Journal of Neuroscience 32*(7): 15 February 2012 DOI: 10.1523/JNEUROSCI.5372-11.2012, p. 2241-2247.
[307] In the 'sphere' of references around the literature directly considered for this thesis. It may have been proposed earlier elsewhere, but de Vries appears to have introduced the idea to the literature I considered directly. See ft. 80.
[308] de Vries, "Minireview: Sex differences in adult and developing brains."
[309] For example, in a student review in 2012, "Interestingly, it has recently been proposed that some phenotypic endpoints that do not show sex differences nonetheless can be affected by different factors in males and females (McCarthy et al., 2012)." (p. 6746) The reference within the quote is to ft. 78. Jiska S. Peper and P. Cédric M. P. Koolschijn, "Sex Steroids and the Organization of the Human Brain," *Journal of Neuroscience 32*(20): 16 May 2012, DOI: 10.1523/JNEUROSCI.1012-12.2012, p. 6745-6746.
[310] McCarthy *et al.*, "Sex Differences in the Brain," p. 2242.
[311] Ibid.
[312] The research on the possible effects of digestive health on mental health will not be covered in detail, being largely too late for this thesis. However, it is worth noting that the microbiome of the gut appears to be both different by sex and somewhat causal in sex-differentiated outcomes, at least as of the time of writing. Minal Jaggar, Kieran Rea, Simon Spichak, Timothy G. Dinan, and John F. Cryan, "You've got male: Sex and the microbiota-gut-brain axis across the lifespan," *Frontiers in Neuroendocrinology 56*, ID 100815, DOI: 10.1016/j.yfrne.2019.100815, p. 1-22.

not the case.[313] That last point is particularly relevant when considering the conclusions made about sex similarities or differences in humans, as it implicitly assumes that women and men have lived and continue to live similar lives or that living has negligible effects on neural structures by cutting the chain of causality at recent, measurable neurochemistry.[314]

3.5: Matter of facts[315]

> *"The phenotype. As scientists, the word 'phenotype' means something different to each of us, depending upon our scientific query at hand. The traditional definition of a phenotype is the outward observable traits or characteristics of an organism that can be measured. Any phenotype to mention is a composite resulting from multiple factors. But, which factors? This is the question that helps drive scientific discovery forward. What factors impact observable, and potentially quantifiable, composites in our world? If we start with the basic description of a mammal, for example, an initial question would certainly be, 'Is it a male or female?' Why ask this question? The answer is that males and females have markedly different phenotypes; sex and hormones matter."*[316]

That these terms—sex, gender, difference, dimorphism—were not used precisely is, in and of itself, nothing particularly unique to behavioral neuroendocrinology. What is noteworthy about the imprecision is that it functioned as a collective "motte and bailey" argument.[317] Motte and bailey arguments refer to the practice of giving a single argument which can have (at least)

[313] Bruce S. McEwen, "Neurobiological and Systemic Effects of Chronic Stress," *Chronic Stress 1*: 2017, DOI: 10.1177/2470547017692328, p. 1-11.
[314] This will be further discussed in Chapter 4.
[315] Reference to the idiomatic phrases "matter-of-fact", meaning a bearing or style that is blunt, straightforward, and primarily factual, and "the fact of the matter", a transition phrase which implies that the next information is not widely known or contradicts previously stated information.
[316] Koebele and Bimonte-Nelson, "Trajectories and phenotypes with estrogen exposures across the lifespan," p. 86.
[317] Maarten Boudry and Johan Braeckman, "Immunizing Strategies and Epistemic Defense Mechanisms," *Philosophia 39*: 2011, DOI: 10.1007/s11406-010-9254-9, p. 149-151.

two interpretations. The "motte" interpretation, like the medieval stone keep built on high ground, refers to the safer and less risky position, while the "bailey" interpretation, like the wide and open area around the motte surrounded by a low wall, is the broader and riskier position. The benefit of such arguments for those making them is that one can retreat from the riskier position to safer intellectual ground in the motte, should the bailey prove indefensible. Consider, for example, the quote at the beginning of this section. To say that sex is a commonly collected variable about mammals, including humans, is an uncontroversial and nearly banal statement. To further state that there are often phenotypic differences between male and female mammals, often related to hormones, is also a rather underwhelming position. "Sex and hormones matter"—well, yes, evidently. Reading slightly more critically, however, the authors are not simply arguing that the concept of sex is useful to describe phenotypes of individual mammals, being a descriptor for many linked phenotypic features that frequently appear together; they are arguing that sex is a basic "factor" that creates phenotypes. To reify sex in this manner strips it of its complexity while disconnecting it from the animals the concept describes. Sex, in this conception, does not simply matter but is instead part of the underlying matter from which individuals are sculpted. The motte is that sex is a matter to study in its differences and variations; the bailey is that sex is a matter of existence from which differences emerge.

Considering "sex differences" as a definitional motte and bailey can help encompass both the fundamental fluidity of its various definitional components and aspects as well as the trend within that fluidity toward an essentialist and dimorphic view of sex. At the motte end of the definition, "sex differences" can encompass any difference between sex/genders, no matter its origin or its magnitude. Marginal differences in lung cancer likelihood between genders in the

United States[318] would fit, as well as the widely differing rates of breastfeeding between genders over the twentieth century. This motte, however, is uncontroversial to an extreme. Judith Butler's 1988 description of gender performativity could also fit within:

> *"It seems fair to say that certain kinds of acts are usually interpreted as expressive of a gender core or identity, and that these acts either conform to an expected gender identity or contest that expectation in some way. That expectation, in turn, is based upon the perception of sex, where sex is understood to be the discrete and factic datum of primary sexual characteristics. … If gender attributes, however, are not expressive but performative, then these attributes effectively constitute the identity they are said to express or reveal. … That gender reality is created through sustained social performances means that the very notions of an essential sex, a true or abiding masculinity or femininity, are also constituted as part of the strategy by which the performative aspect of gender is concealed."*[319]

It is important to note that Butler was not arguing here that primary sexual characteristics, sex, and gender do not exist or do not affect the lives of the people who have them; rather, they argued that saying that any essence of sex or gender existed outside of the social acts which constitute gender denied how sex and gender are given meaning and form. The motte definition of "sex differences" implies nothing about the origins of the differences nor the nature of sex itself, and therefore Butler is not contradicting it here.

[318] "Overall, the chance that a man will develop lung cancer in his lifetime is about 1 in 16; for a woman, the risk is about 1 in 17." American Cancer Society, "Key Statistics for Lung Cancer," *American Cancer Society*, last revised 29 January 2024, accessed 1 February 2024, cancer.org/cancer/types/lung-cancer/about/key-statistics.html.
[319] Judith Butler, "Performative Acts and Gender Constitution: An Essay in Phenomenology and Feminist Theory," *Theatre Journal 40*, no. 4: December 1988, DOI: 10.2307/3207893, p. 527-528.

The motte definition, however, does not reliably describe the usage of "sex differences" overall in the behavioral neuroendocrinological literature. To return to a point made in §3.3, to say that socialization was the explanation of last resort understates the issue. The systemic problem was that experiments were not generally designed to account for culture or socialization, relegating culture to a *post hoc* part of analysis sections rather than an integral part of human lives and their experience of sex/gender. One of the most consistent statements across the literature is that women are diagnosed with depression twice as often as men, and that the sex difference in depression is highest when circulating estrogen is the highest—i.e., during the years the person can bear children.

Only one paper among those considered for this thesis explicitly considered that women and girls may experience different social environments and stressors than men and boys, stating, "One might think that women are more prone to depression and anxiety in part because they are more likely to be victimized by child abuse. … [A]side from sexual abuse, boys and girls are victimized by physical abuse at approximately equal rates in the United States."[320] The review's introduction moves into its main material as follows:

> *"Thus, the overarching question addressed in this review is why girls seem to be more vulnerable than boys to the adverse effects of child abuse. As a framework for considering this question, we offer the following model: Because of a sex difference in the prepubertal brain, girls are more likely than boys to perceive and remember social cues as threatening."*[321]

[320] Cooke and Weathington, "Human and animal research into sex-specific effects of child abuse," p. 417.
[321] Ibid.

This review's introduction discusses and dismisses the possibilities that the different diagnosis rates emerge from socialization pressures about seeking professional help or that different child sexual abuse or other maltreatment rates by gender may be contributing to the higher diagnosis rate among women.[322] The review ignores or does not consider the possibility that doctors may be better able to diagnose depression in one gender versus the other[323] or the possibility that the adolescence and adulthood of men and women could be substantively different in a fashion that affects their likelihood of a later diagnosis of depression. Instead, the cause of the difference is located prior to the circumstances of peoples' lives and within the essences of the people involved, implicitly (or, in this case, quite explicitly) denying the relevance of culture and circumstance to sexed/gendered data.

The bailey definition is that sex differences may be revealed by circumstance but that these differences inhere in all people from the earliest hours of development, as sex is an

[322] There are compelling reasons to believe that limiting the scope of relevant child maltreatment to 'physical abuse' paints an extremely unrepresentative picture of the data from a gendered perspective specifically, one not justified by the authors' statements or by the available data. The data on child sexual abuse from the early 2010s suggested that 15% (95% confidence interval (CI) 9-24%) of girls and 8% (95% CI 4-16%) of boys experienced child sexual abuse worldwide, and when the question is narrowed to 'forced intercourse', the ratio changes to 9% (95% CI 6-14%) of girls and 3% (95% CI 1-9%) of boys. J. Barth, L. Bermetz, E. Heim, S. Trelle, T. Tonia, "The current prevalence of child sexual abuse worldwide: a systematic review and meta-analysis," *International Journal of Public Health 58*: 2013, DOI: 10.1007/s00038-012-0426-1, p. 469-483. Additionally, while the data is difficult to parse, in North America girls appear to be significantly more likely to experience psychological/emotional abuse and neglect, the latter being by far the most common type of child abuse in the United States. Gwenllian Moody, Rebecca Cannings-John, Kerenza Hood, Alison Kemp, and Michael Robling, "Establishing the international prevalence of self-reported child maltreatment: a systematic review by maltreatment type and gender," *BMC Public Health 18*, ID 1164: 2018, DOI: 10.1186/s12889-018-6044-y, p. 1-15; Children's Bureau (Administration for Children and Families, Administration on Children, Youth and Families) of the US Department of Health and Human Services, Tables 3-9 and 3-10, *Child Maltreatment 2019:* 2021, available from acf.hhs.gov/cb/research-data-technology/statistics-research/child-maltreatment. North American data was focused on due to the researchers' use of US data; data was chosen from 2019 to be pre-pandemic.

[323] Discussed in §4.4; however, anger is a significant part of male-typical emotional expression and actively discouraged for female-typical emotional expression in modern Western culture, which could have consequences for how women and men express their experience of depression. Combining 'traditional' with a diagnosis which places more emphasis on anger, substance abuse, and risk-taking as symptoms of 'atypical' (male-type) depression found roughly equal rates of depression in men and women. Lisa A. Martin, Harold W. Neighbors, and Derek M. Griffith, "The Experience of Symptoms of Depression in Men vs Women: Analysis of the National Comorbidity Survey Replication," *JAMA Psychiatry 70*(10): 2013, DOI: 10.1001/jamapsychiatry.2013.1985, p. 1100-1106.

essential aspect of all selves. This extreme bailey definition was by no means a unanimous position among the literature considered for this thesis. To return to the definitional instability, however, the facts that "sex" and "gender" had unclear definitional boundaries, that "difference" and "dimorphism" were functionally synonymous in much of the literature, and that "sex similarity" was belatedly defined as a non-obvious difference in mechanism rather than similarity of outcome helped support this collective motte and bailey by providing the space for multiple definitions to exist within what appeared to be roughly the same term. Without this instability in the components of the phrase and the related concepts, "sex difference" could not have supported these different interpretations. Finally, it must be noted that organizational/activational theory in general, and those researching with that framework, tended to assume the bailey definition, or a definition closer to the bailey than the motte, due to the "organizational" half of the theory, which assumed a similar essential and inherent origin for sex-typed behaviors.

Motte and bailey arguments have one final aspect: that the speaker would prefer to defend the bailey if they can. The literature in these four journals consistently hypothesized genetic, hormonal, and developmental explanations for broader societal trends among humans while only inconsistently considering possible historical or cultural explanations for those trends, preferring to locate the causal mechanisms within the biological or developmental parts of individuals rather than the social parts of groups or the interface between. *De jure*, "sex differences" could have been anything; *de facto*, only biological and developmental factors mattered. By failing to consistently consider many if not most aspects of lived experience related to sex when attempting to explain observed differences between sexes or genders,[324] sex

[324] A point expanded upon in chapter 4.

difference researchers in behavioral neuroendocrinology habitually defended the bailey position that social factors were generally non-explanatory for sex differences between humans, retreating to the motte of social/biological interaction when necessary. As the last chapter discussed, the simple positions that supported organizational/activational theory decayed over the period 2000-2019 as the evidence undermined them, though they did not disappear entirely. Similarly, social factors began to creep into and around sex difference research, particularly in the last period during the replication crisis (2016-2019),[325] but "sex differences" as a primarily biological and developmental set of ingrained behavioral and neural differences remained influential.

Returning to §3.1, the factors underlying the replication crisis are particularly relevant when considering sex differences in research, both its multifaceted definitions and as part of the organizational/activational theory. The null hypothesis when researchers examined sex differences, including organizational or activational differences, was that there were no differences between genders.[326] The general tendency not to publish research with a p-value above 0.05, i.e. when the data failed to disconfirm the null hypothesis, meant that research failing

[325] Virginia W. Huynh, Shu-Sha Angie Guan, David M. Almeida, Heather McCreath, and Andrew J. Fuligni, "Everyday discrimination and diurnal cortisol during adolescence," *Hormones and Behavior 80*: April 2016, DOI: 10.1016/j.yhbeh.2016.01.009, p. 76-81; Judy L. Cameron, Kathie L. Eagleson, Nathan A. Fox, Takao K. Hensch, and Pat Levitt, "Social Origins of Developmental Risk for Mental and Physical Illness," *Journal of Neuroscience 37*(45): 8 November 2017, DOI: 10.1523/JNEUROSCI.1822-17.2017, p. 10783-10791; Maria Teresa Ferretti and Lisa A. M. Galea, "Improving pharmacological treatment in brain and mental health disorders: the need for gender and sex analyses," *Frontiers in Neuroendocrinology 50*: July 2018, DOI: 10.1016/j.yfrne.2018.06.007, p. 1-2; Luca Liberale, Federico Carbone, Fabrizio Montecucco, Cathérine Gebhard, Thomas F. Lüscher, Susanne Wegener, and Giovanni G. Camici, "Ischemic stroke across sexes: What is the status quo?", *Frontiers in Neuroendocrinology 50:* July 2018, DOI: 10.1016/j.yfrne.2018.05.001, p. 3-17; Anna Strandqvist, Lisa Örtqvist, Louise Frisén, Agneta Nordenskjöld, Agneta Herlitz, and Anna Nordenström, "No difference in cognitive performance or gender role behavior between men with and without hypospadias," *Hormones and Behavior 109*: March 2019, DOI: 10.1016/j.yhbeh.2019.02.004, p. 64-70; Tessa E. S. Charlesworth and Mahzarin R. Banaji, "Gender in Science, Technology, Engineering, and Mathematics: Issues, Causes, Solutions," *Journal of Neuroscience 39*(37): 11 September 2019, DOI: 10.1523/JNEUROSCI.0475-18.2019, p. 7228-7243.

[326] Or that there were no differences between those with differences of sexual differentiation (DSDs) or who were intersex and those without DSDs or who were not intersex.

to find sex differences was less likely to be published at all.[327] Furthermore, as sex difference research had small sample sizes due to the difficulty of human (and animal) research, research finding effects likely often suffered the "winner's curse": real effects were exaggerated in magnitude, and phantom effects were generated by chance. In addition, because sex difference research, like much other research, tended to be framed in terms of "does the difference exist" rather than "what kind of difference may exist", sex differences were discussed without addressing the possible magnitude of differences suggested by the evidence and without appropriately cautious language reflecting the confidence interval.

To reinforce this point, the United States' National Institutes of Health implemented a policy, beginning in January 2016, that all clinical and preclinical research must include sex or gender in study design or explain their absence.[328] A review discussing this policy pointed to past results: "When sex differences are looked for, they are often found, so we are likely to be on the cusp of learning about a host of new sex and gender differences in the brain."[329] While the factors underlying the replication crisis do not explain the focus on sex differences, these aspects of how the replication crisis happened do help explain why sex differences received far more research support than the absence of sex differences did during the period 2000-2019, especially before 2016. The exaggerated number and size of sex differences generated by that research

[327] One of the earliest publications to argue "no evidence for sex differences" on the basis of failing to disconfirm the null hypothesis (i.e. p-values > 0.05), at least within the articles covered for this thesis, was an article published in *eNeuro* in 2016 as part of an effort to specifically publish negative results as the replication crisis became a central point of discussion in the neuroscientific journals. Jaime A. Willett, Tyler Will, Caitlin A. Hauser, David M. Dorris, Jinyan Cao, and John Meitzen, "No evidence for sex differences in the electrophysiological properties and excitatory synaptic input onto nucleus accumbens shell medium spiny neurons," *eNeuro 3*(1): 4 February 2016, DOI: 10.1523/ENEURO.0147-15.2016, p. 1-12.
[328] US National Institutes of Health, "Consideration of Sex as a Biological Variable in NIH-funded Research," NIH Notice Number: NOT-OD-15-102, *US National Institutes of Health*: 9 June 2015, grants.nih.gov/grants/guide/notice-files/not-od-15-102.html.
[329] Forger *et al.*, "Cellular and molecular mechanisms of sexual differentiation in the mammalian nervous system," p. 67.

environment then helped bolster organizational/activational theory's apparent relevance and kept the bailey definition of 'sex difference' plausible but never quite sturdy enough to gain universal acceptance—at least until the replication crisis began undermining the validity of the existing research beginning in 2016.

3.6: Who measures the measurements?[330]

The replication crisis did not only affect the available data. New methodologies and frameworks were often published in the context of new data or research they helped facilitate, the "success" (i.e. results with a p-value < 0.05) of the methodology proving the technique's effectiveness. Within the context of organizational/activational theory-guided research, the 2D:4D ratio, or the ratio between the lengths of the second finger and the fourth finger, serves as a useful example of methodological controversy. In the early 2000s, the 2D:4D ratio became established as a measurement exhibiting a sex difference,[331] closer to 1.0 in females and 0.9 in males,[332] that studies of those with congenital adrenal hyperplasia (CAH), a congenital condition in which the adrenal glands produce too little cortisol and (often) too much of some androgens,[333] suggested may be a useful marker of prenatal androgen exposure. However, the

[330] A reference to "Quis custodiet ipsos custodes?", or "Who watches the watchmen?", originally written in the Roman poet Juvenal's *Satires,* Satire VI, lines 347-348. This part of the *Satires* is part of an ongoing debate about originality, as there are two different manuscripts which have slightly different versions, but *quis custodiet ipsos custodes* appears to be common between the two. Georg Luck, "The Textual History of Juvenal and the Oxford Lines," *Harvard Studies in Classical Philology 76*: 1972, DOI: 10.2307/310987, p. 217-231.

[331] Dennis McFadden and Erin Shubel, "Relative Lengths of Fingers and Toes in Human Males and Females," *Hormones and Behavior 42*(4): December 2002, DOI: 10.1006/hbeh.2002.1833, p. 492. The cited research is largely from the period 2000-2002. Some antecedents, dating as far back as 1875, appear to have formed the basis for the 2000s research, but the 2D:4D measurement's application to organizational/activational theory does not seem to be much older than 2000.

[332] McFadden and Shubel, "The relationships between otoacoustic emissions and relative lengths of fingers and toes in humans," p. 421.

[333] CAH is more complicated than discussed here. There are multiple forms and varying degrees of severity that the condition can exhibit, related to the specific adrenal cortisol-producing enzyme that has an altered level of function; the only unifying characteristic is that cortisol levels are low, at least early in development, and the size and activity of the adrenal glands are elevated in direct consequence to the low cortisol. The hormones showing altered levels

link between 2D:4D and prenatal androgens was not simply based on those with CAH. It was based on the idea that women with CAH, bisexual and lesbian women, girls and women with autism or ADHD, girls or women with better spatial navigation skills, those with more lateralized hand performance, and women with low inhibitions, for example, all expressed masculinized phenotypes, [334,335] being based in "behaviors that are either sexually dimorphic or ascribed to the actions of sex steroids [and] correlate[d] with 2D:4D."[336] Another researcher hypothesized that elevated androgens might be altering the activity of developmental HOX genes,[337] which would perhaps even explain the tendency for researchers to find lower 2D:4D ratios in male than female mice and baboons as well as humans.[338]

There was some early pushback against this focus on digit ratios and links between it and behavior. Firstly, some researchers noted that "the sex difference in 2D:4D accounts for only 6%–9% of the total variance in digit ratios (Coolican and Peters, 2003),[339] which presumably represents the maximum possible contribution of prenatal androgens."[340] A few other researchers failed to find correlations between different measures of masculinization believed to be affected

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can but do not always include cortisol, aldosterone, some androgens, and some estrogens. While aldosterone is the most directly threatening steroid, as low levels can cause vomiting, dehydration, and death, both androgens and estrogens may be present in elevated or insufficient quantities in male and female children, which may cause, for example, XY-female phenotypes, infertility, precocious or delayed puberty, and menstrual irregularity. CAH is often considered an intersex condition because, due to the excess androgens that often but do not always accompany CAH, many girls with CAH exhibit ambiguous genitalia from birth. Researchers in behavioral neuroendocrinology tended to focus on the virilizing effects of excess androgens on girls and women with CAH, especially those types of CAH which were not characterized by dangerously low levels of aldosterone.

[334] McFadden and Shubel, "Relative Lengths of Fingers and Toes in Human Males and Females," p. 492.

[335] Benderlioglu and Nelson, "Digit length ratios predict reactive aggression in women, but not in men," p. 559.

[336] Ibid.

[337] Martin and Nguyen, "Anthropometric analysis of homosexuals and heterosexuals," p. 36.

[338] Benderlioglu and Nelson, "Digit length ratios predict reactive aggression in women, but not in men," p. 558.

[339] J. Coolican and Michael Peters, "Sexual dimorphism in the 2D/4D ratio and its relation to mental rotation performance," *Evolution and Human Behavior 24*(3): May 2003, DOI: 10.1016/S1090-5138(03)00010-2, p. 179-183.

[340] van Anders and Hampson, "Testing the prenatal androgen hypothesis," p. 96.

by prenatal androgens,[341] including between the 2D:4D ratio and spatial abilities or sexual orientation.[342] The researchers involved, however, tended to argue that perhaps multiple masculinization events may be at play and so the correlations between the 2D:4D ratio and any other masculinization measure may require larger sample sizes to detect—not disputing the 2D:4D ratio directly but suggesting it may have limited usefulness.

In 2014, a researcher discussed the use of 2D:4D to link ADHD and fetal testosterone in decidedly mixed terms but while still fundamentally accepting the arguments underlying the usefulness of 2D:4D research:

> *"Work in clinical and healthy human populations has hinted that exposure to elevated androgen levels during embryogenesis (and maybe also into adulthood) could predispose to male-specific behaviour patterns and to ASDs [autism spectrum disorders] (Baron-Cohen et al., 2011, Hines, 2008). Many of the same arguments and experimental paradigms used in this ASD research are equally applicable to ADHD.*
>
> *A commonly used surrogate index of fetal testosterone exposure is the ratio of the length of the index finger (2D) to the length of the ring finger (4D), with higher levels of in utero exposure being associated with lower 2D:4D ratios (Breedlove, 2010). However, the value of this measure as an index of prenatal androgen exposure in humans is somewhat controversial (Berenbaum et al., 2009)."*[343]

In 2015, Melissa Hines' laboratory published some of the first arguments directly against the use of the 2D:4D ratio in the literature covered for this thesis, arguing that the sex differences in

[341] McFadden and Shubel, "The relationships between otoacoustic emissions and relative lengths of fingers and toes in humans."
[342] van Anders and Hampson, "Testing the prenatal androgen hypothesis."
[343] William Davies, "Sex differences in Attention Deficit Hyperactivity Disorder: Candidate genetic and endocrine mechanisms," *Frontiers in Neuroendocrinology 35*(3): August 2014, DOI: 10.1016/j.yfrne.2014.03.003, p. 339-340.

2D:4D ratios are quite small, that 2D:4D ratios' relationships to predicted measures of masculinization (such as sexual orientation) are inconsistent, and that direct evidence supporting 2D:4D as a measure of prenatal androgen exposure is not particularly robust.[344] In part, this likely reflected Hines' support of a different measure of prenatal and early-life androgen exposure, anogenital distance measurements,[345] but in a commentary published in *Science* in 2019 about the critics of the 2D:4D ratio, Hines credits the consistent failure of her undergraduate students to replicate existing 2D:4D research in her classes as changing her mind.[346] The distance between Hines' personal experience of failed replication and the literature is visible in her discussion of 2D:4D's unreliability in her lab's 2015 paper, since the literature simply did not contain enough evidence of failed replications in basic 2D:4D research in 2015—likely, at least in part, reflecting the publication environment leading up to the replication crisis. The only example of failed replications she cites is a failure to relate mutations in the androgen receptor in men[347] to their 2D:4D ratios,[348] which called the 2D:4D ratio into question primarily indirectly.

In 2020, S. Marc Breedlove's lab, perhaps the lab most responsible for characterizing and promulgating the use of the 2D:4D ratio as a measure of prenatal androgen in the research covered by this thesis, wrote a commentary for the 50th anniversary issue of *Hormones and Behavior* titled "Through a glass, darkly: Human digit ratios reflect prenatal androgens,

[344] Pasterski *et al.*, "Postnatal penile growth concurrent with mini-puberty predicts later sex-typed play behavior," p. 103-104.
[345] Ibid.
[346] Mitch Leslie, "The mismeasure of hands?" *Science 364*(6444): 7 June 2019, DOI: 10.1126/science.364.6444.923, p. 925.
[347] Specifically $(CAG)_n$ repeats, which make the androgen receptor less efficient at communicating the presence of androgen.
[348] Pasterski *et al.,* "Postnatal penile growth concurrent with mini-puberty predicts later sex-typed play behavior," p. 103-104.

imperfectly."[349] The title is taken from the King James Version of the First Letter to the Corinthians,[350] 'glass' translating the koine Greek ἐσόπτρου (*esoptrou*). That ἐσόπτρου may have referred either to a mirror (primarily reflective) or lens (primarily transparent) is, quite accidentally, telling—windows, glasses, are always both reflective and transparent, and this paper reflects its creators at least as much as it offers a view onto the research it describes. The commentary accused the scientific community overall and *Hormones and Behavior* in particular of unjust prejudice against the 2D:4D, particularly focusing on the *Science* commentary mentioned above as evidence that the predictive power of the 2D:4D ratio is being unjustly ignored and minimized due to, the authors argue, the low-tech nature of the method and the difficulty of interpreting the (they admit) noisy data.[351] Through their reading of the *Science* commentary and their arguments that the extant research overwhelmingly supported the relationship between 2D:4D ratios and prenatal androgen, the researchers decry that the then-editor of *Hormones and Behavior* stated in the aforementioned commentary that they would not publish papers that relied on the 2D:4D ratio as a proxy for hormones.

Some specific sentences from this commentary are worth highlighting because they give insight into the thought process of researchers in this context. Firstly: "There are no cultural barriers to measuring fingers [as opposed to anogenital distance], and so the use of 2D:4D is a case of 'searching where the light is.'"[352] The quote is a reference to an old joke about a drunkard looking for his keys under a streetlamp. A policeman asks the drunkard if he lost his keys under the light, and the drunkard replies no, he lost the keys in the park, but 'this is where

[349] Ashlyn Swift-Gallant, Brandon A. Johnson, Victor Di Rita, and S. Marc Breedlove, "Through a glass, darkly: Human digit ratios reflect prenatal androgens, imperfectly," *Hormones and Behavior 120*, ID 104686: April 2020, DOI: 10.1016/j.yhbeh.2020.104686, p. 1-5.
[350] 1 Corinthians 13:12 (King James Version).
[351] Swift-Gallant *et al*., "Through a glass, darkly," p. 2.
[352] Ibid., p. 3.

the light is.'[353] This explanation, however, does not address one of the main criticisms of 2D:4D made in the *Science* commentary—that it may or may not reflect prenatal androgens well enough to be useful, regardless of its measurability. If 2D:4D was sufficiently noisy and anogenital distance measurements sufficiently reflective of prenatal androgens, then 2D:4D could require an unworkable number of measurements to detect effects as accurately as a small number of anogenital measurements. Secondly: "In the meantime, if you compare groups of people and find no difference in their digit ratios, then, just as with any other null finding, you might consider whether your sample was large enough. At the least, you should conduct a power analysis to determine an appropriate sample size to detect a difference *between* the sexes…. If you can detect the sex difference and if you also see a difference between groups *within* a sex, then the only way to gain confidence that the difference is real is to publish the results and see if others can replicate them. [italics in original]"[354] On a practical level, this is likely how the leadup to the replication crisis unfolded from the perspective of the scientists themselves. Null results faced further questioning; significant results were sent off for publication.

Unsurprisingly, a criticism of *Hormones and Behavior*'s editorial staff published in *Hormones and Behavior* received a response from the editorial staff of *Hormones and Behavior*. The editorial staff argued that they did not publish research relying on digit ratios alone because it was not necessarily relevant to hormones, as the relationship between digit ratios and hormone

[353] Noam Chomsky famously described scientific inquiry as being forced to search where the light is. Robert F. Barsky, *Noam Chomsky: a life of dissent,* MIT Press (Cambridge, Mass.): 1998, p. 95. There are a number of variations of the joke and its origins are muddled. A newspaper comic, *Mutt and Jeff*, published a version of the joke in June 1942 where the policeman helps the drunkard search under the lamp for his lost quarter in the last panel. Presumably, the more straightforward version (without the policeman joining in as a subversion) is older than that. Manuela Battaglia and Mark A. Atkinson, "The streetlight effect in type 1 diabetes," *Diabetes 64*(4): April 2015, DOI: 10.2337/db14-1208, p. 1081-1082.

[354] Swift-Gallant *et al*., "Through a glass, darkly," p. 3.

levels was already somewhat weak.[355] As evidence, they indicated a paper published in *Hormones and Behavior* that measured both testosterone and digit ratios and related both to behavior, which was published after the commentary in *Science*.[356] The editors also brought up their own experiences to explain their skepticism of digit ratio-based research. The editors had, in 2008, conducted research on facial measurements and facial measurements' possible relationships to behavior, both in the individuals and in others' reactions. After publication, however, the research got rather out of their control:

> *"We speculated early on that this marker might be related to pubertal androgens in men, but we had, and still have, no evidence for this speculation. It was simply speculation. Yet the literature began using the measure as a marker of testosterone (prenatally, pubertally, adult levels, depending on the study), often citing our paper (Carré and McCormick, 2008)*[357] *as the basis for the assertion without acknowledging that we had made a speculation, not an assertion, and that we had no data in this paper or any other to make such an assertion."*[358]

The editors of *Hormones and Behavior* here sharply diverge from the Breedlove laboratory, as the Breedlove laboratory suggested that their earliest paper on the topic[359] being "cited over 500 times" was part of an argument in support of its research's fundamental accuracy, rather than, as

[355] Cheryl M. McCormick and Justin M. Carré, "Facing off with the phalangeal phenomenon and editorial policies: A commentary on Swift-Gallant, Johnson, Di Rita and Breedlove (2020)," *Hormones and Behavior 120*, ID 104710: April 2020, DOI: 10.1016/j.yhbeh.2020.104710, p. 3.
[356] Ibid., p. 1.
[357] Justin M. Carré and Cheryl M. McCormick, "In your face: facial metrics predict aggressive behaviour in the laboratory and in varsity and professional hockey players," *Proceedings of the Royal Society B: Biological Sciences 275*(1651): 22 November 2008, DOI: 10.1098/rspb.2008.0873, p. 2651-2656.
[358] McCormick and Carré, "Facing off with the phalangeal phenomenon and editorial policies," p. 3.
[359] Terrance J. Williams, Michelle E. Pepitone, Scott E. Christensen, Bradley M. Cooke, Andrew D. Huberman, Nicholas J. Breedlove, Tessa J. Breedlove, Cynthia L. Jordan, and S. Marc Breedlove, "Finger-length ratios and sexual orientation," *Nature 404*: 30 March 2000, DOI: 10.1038/35006555, p. 455-456, from Swift-Gallant *et al*., "Through a glass, darkly," p. 3.

the editors argued about their own research, a vastly inflated number helping to drive the skepticism of the measurement.[360]

Perhaps most fundamentally, the two groups of writers in *Hormones and Behavior* disagreed on what the presence of a significant result means. The Breedlove laboratory argues that, "[i]nterestingly, there is an asymmetry of logic in interpreting studies of amniotic levels of hormone and behavior. Findings that measures of amniotic androgen *do* significantly correlate with a behavior, *despite* the many limitations of the amniotic snapshot, cannot be readily dismissed. [italics in original]"[361] The editors of *Hormones and Behavior*, however, pointed out that the Breedlove laboratory implicitly assumed that publication bias against null results had not been a relevant factor. Re-analyses of their evidence became less convincing (smaller effect sizes and losing statistical significance) when redone with techniques intended to compensate for publication bias.[362] The Breedlove laboratory was also implicitly assuming that the 2D:4D correlation is not confounded. The original *Science* commentary and the editors of *Hormones and Behavior* both pointed to research suggesting that larger hand sizes lower the 2D:4D ratio; when hand size is controlled for, "the male-female difference in digit ratios flipped—men now had higher values."[363] It may in fact be true that higher testosterone is correlated with lower 2D:4D ratios, but studying 2D:4D ratios in isolation may be obscuring the cause behind the correlation (i.e. hand size being larger as a result of higher testosterone), if a shared cause exists

[360] Swift-Gallant *et al*., "Through a glass, darkly," p. 3.
[361] Ibid.
[362] There is a meta-analytical technique, the "fail-safe" analysis, which estimates the number of null findings required to render the data as a whole non-significant, that the Breedlove lab cited. However, this analysis appears to be less than universally agreed with, as the editors of *Hormones and Behavior* noted.
[363] Leslie, "The mismeasure of hands?" The Breedlove lab did attempt to dismiss this argument in their commentary (Swift-Gallant *et al*., "Through a glass, darkly," p. 2), but did not address the basic contention of correlation being confounded by a shared cause.

at all.[364] "Thus, although [the Breedlove laboratory] are convinced that digit ratios have some link to prenatal hormones in humans, many won't be, based on the available evidence."[365]

3.7: The best-laid schemes o' mice an' men gang aft agley[366]

As noted in §3.3, testing for human sex differences required indirect methods for ethical reasons. The most popular method to detect sex differences in humans was to test for sex differences in model animals, particularly rats. Mice,[367] other rodents,[368] non-ape primates,[369] sheep,[370] and a handful of other model organisms ranging from quail[371] to the nematode worm *Caenorhabditis elegans*[372] also appeared in research and reviews. As §3.3 also mentioned, this usage of model animals required significant assumption 'scaffolding': specifically, that results observed in the model animals could be extrapolated to humans. The degree of extrapolation desired from animal studies was variable; for some, characterizing model systems would

[364] Stephen Jay Gould described an analogous problem correlating arm and leg length as an example in *The Mismeasure of Man*; arm and leg length are correlated, but it would seem obviously odd to argue that arm length was driving leg length or vice versa rather than a shared factor driving both. In contrast, many things are correlated without a causal relationship at all; anything that was increasing before 1986 and decreasing from then to December 2023 would be correlated with distance to Halley's comet "but even the most dedicated astrologer would not discern causality in most of these relationships." Stephen Jay Gould, Section: "Correlation, Cause, and Factor Analysis" in Chapter 6: "The Real Error of Cyril Burt: Factor Analysis and the Reification of Intelligence," *The Mismeasure of Man*, 2nd ed., W. W. Norton and Company (New York): 2006, Ebook.
[365] McCormick and Carré, "Facing off with the phalangeal phenomenon and editorial policies," p. 3.
[366] A quote from the Robert Burn poem *To a Mouse*, written in 1785 and in Scots. In English, the lines read: "The best-laid schemes of mice and men/Go oft awry".
[367] L. Christine Turtzo, Chad Siegel, and Louise D. McCullough, "X Chromosome Dosage and the Response to Cerebral Ischemia," *Journal of Neuroscience 31*(37): 14 September 2011, DOI: 10.1523/JNEUROSCI.0621-11.2011, p. 13255-13259.
[368] Melissa M. Holmes, Bruce D. Goldman, Sharry L. Goldman, Marianne L. Seney, and Nancy G. Forger, "Neuroendocrinology and sexual differentiation in eusocial mammals," *Frontiers in Neuroendocrinology 30*(4): October 2009, DOI: 10.1016/j.yfrne.2009.04.010, p. 519-533; Phoenix *et al.*, "Organizing Action of Prenatally Administered Testosterone Propionate."
[369] Albers, "Species, sex and individual differences in the vasotocin/vasopressin system," p. 56; Tony M. Plant, "Neuroendocrine control of the onset of puberty," *Frontiers in Neuroendocrinology 38*: July 2015, DOI: 10.1016/j.yfrne.2015.04.002, p. 84-85.
[370] Plant, "Neuroendocrine control of the onset of puberty," p. 84-85.
[371] Albers, "Species, sex and individual differences in the vasotocin/vasopressin system," p. 55.
[372] Satoshi Suo, Kazuki Harada, Shogo Matsuda, Koki Kyo, Min Wang, Kei Maruyama, Takeo Awaji and Takashi Tsuboi, "Sexually Dimorphic Regulation of Behavioral States by Dopamine in *Caenorhabditis elegans*," *Journal of Neuroscience 39*(24): 12 June 2019, DOI: 10.1523/JNEUROSCI.2985-18.2019, p. 4668-4683.

primarily be valuable to determine how biological systems work in general.[373] Researchers in behavioral neuroendocrinology frequently, however, performed 'preclinical' research, i.e., research explicitly designed to demonstrate possible clinical (human) utility, as well as directly extrapolating results between model organisms, most commonly rats or mice, and humans.[374] Preclinical research and extrapolation relied on the assumptions that sexed aspects of humans and model animals were consistent and that the environments and social aspects of the model organisms were not significantly affecting observed behaviors or sex differences. One paper even stated, after detailing results from papers using rat models, "[b]ecause there are no environmental or sociocultural factors to consider, these differences must be attributable to biological differences between the male and female."[375]

The idea that research could be straightforwardly extrapolated between rats or mice and humans had many objections raised against it within behavioral neuroendocrinological literature. Firstly, rats and mice do not have a childhood, or a delay of reproductive development, comparable to humans,[376] and only primate species are known to undergo adrenarche, or increased adrenal activity between ages 3 and 13 in humans.[377] The length of adolescence in humans is additionally far longer than in other primates.[378] Rats and mice are relatively non-social, focusing on "direct reproduction [as] virtually the only route to reproductive success,"[379]

[373] Rebecca M. Shansky and Catherine S. Woolley, "Considering Sex as a Biological Variable Will Be Valuable for Neuroscience Research," *Journal of Neuroscience 36*(47): 23 November 2016, DOI: 10.1523/JNEUROSCI.1390-16.2016, p. 11817-11822; see also Lisa Eliot and Sarah S. Richardson, "Sex in Context: Limitations of Animal Studies for Addressing Human Sex/Gender Neurobehavioral Health Disparities," *Journal of Neuroscience 36*(47): 23 November 2016, DOI: 10.1523/JNEUROSCI.1391-16.2016, p. 11828.
[374] Eliot and Richardson, "Sex in Context: Limitations of Animal Studies."
[375] Tracey J. Shors, Chadrick Chua, Jacqueline Falduto, "Sex Differences and Opposite Effects of Stress on Dendritic Spine Density in the Male Versus Female Hippocampus," *Journal of Neuroscience 21*(16): 15 August 2001, DOI: 10.1523/JNEUROSCI.21-16-06292.2001, p. 6292.
[376] Plant, "Neuroendocrine control of the onset of puberty."
[377] Nguyen *et al.*, "Interactive Effects of Dehydroepiandrosterone and Testosterone on Cortical Thickness."
[378] Spielberg *et al.*, "Anxiety in transition: Neuroendocrine mechanisms," p. 1.
[379] Holmes *et al.*, "Neuroendocrinology and sexual differentiation in eusocial mammals," pg. 519.

while humans often act for the benefit of the in-group rather than only their direct offspring as well as adopt children from unrelated couples.[380] Rats and mice require estrogen in order to masculinize neural structures during development, while there is no evidence for, and some correlational data against, any masculinization due to estrogen in humans or other primates.[381] The brain structures considered critical to mating behavior in most model animals, particularly the medial preoptic area of the anterior hypothalamus, may or may not be relevant to human or primate sexual behavior at all.[382] Mating behaviors are one of the most common model systems "for dissecting the hormone-brain-behavior bidirectional loop",[383] but humans are not consistently dependent on hormones for sexual behavior in the way that most organisms are.[384] X inactivation in mice is significantly more complete (i.e. fewer genes escape inactivation) than in humans.[385] Microglia from the brains of mice show sex differences thought to be related to

[380] I do not wish to overstate this point, simply to note that all known groups of humans showed or show some level of in-group cooperation not limited to direct relatives, something not true of many other mammals. See, for example, Robert Kurzban and Daniel Houser, "Experiments investigating cooperative types in humans: A complement to evolutionary theory and simulations," *PNAS 102*(5): 21 January 2005, DOI: 10.1073/pnas.0408759102, p. 1803-1807. Adoption appears sporadically but consistently in history, religion, folklore, and mythology (ex. *Oedipus Rex,* Hammurabi's Code, Sita in the *Ramayana*) as well as possibly in pre-history; see Manuel Lozano-García, Cláudia Gomes, Sara Palomo-Díez, Ana María López-Parra, and Eduardo Arroyo-Pardo, "The Study of Adoption in Archaeological Human Remains," *Genealogy 7*(2), no. 38: 2023, DOI: 10.3390/genealogy7020038, p. 1-19.
[381] David Puts and Natalie V. Motta-Mena, "Is human brain masculinization estrogen receptor-mediated? Reply to Luoto and Rantala," *Hormones and Behavior 97*: January 2018, DOI: 10.1016/j.yhbeh.2017.07.018, p. 4; see also Severi Luoto and Markus J. Rantala, "On estrogenic masculinization of the human brain and behavior," *Hormones and Behavior 97*: January 2018, DOI: 10.1016/j.yhbeh.2017.07.017, p. 1-2.
[382] Gert Holstege, Janniko R. Georgiadis, Anne M. J. Paans, Linda C. Meiners, Ferdinand H. C. E. van der Graaf and A. A. T. Simone Reinders, "Brain Activation during Human Male Ejaculation," *Journal of Neuroscience 23*(27): 8 October 2003, DOI: 10.1523/JNEUROSCI.23-27-09185.2003; p. 9191. This study is, quite frankly, not particularly strong due to its small sample size (n = 12). It does refer to some other research on the topic which suggests similarly, and, like much else under discussion, the relevance of the area meaningful in model animals was never proven to be meaningful in primates or humans. The most responsible conclusion is of a lack of knowledge.
[383] Margaret M. McCarthy, "Probing the neural circuits of sex and aggression with precision genetics: Commentary on "Estrogen receptor alpha is required in GABAergic but not glutamatergic, neurons to masculinize behavior" by Wu and Tollkuhn," *Hormones and Behavior 95*: September 2017, DOI: 10.1016/j.yhbeh.2017.07.006, p. 1; see also Melody V. Wu and Jessica Tollkuhn, "Estrogen receptor alpha is required in GABAergic, but not glutamatergic, neurons to masculinize behavior," *Hormones and Behavior 95:* September 2017, DOI: 10.1016/j.yhbeh.2017.07.001, p. 3-12.
[384] Olivia Le Moëne and Anders Ågmo, "The neuroendocrinology of sexual attraction," *Frontiers in Neuroendocrinology 51*: October 2018, DOI: 10.1016/j.yfrne.2017.12.006, p. 55-58.
[385] Turtzo *et al.*, "X Chromosome Dosage and the Response to Cerebral Ischemia."

epigenetic regulation, but post-mortem analysis of human microglia found few transcriptional differences between sexes.[386] Some results finding sex differences do not translate between mice and rats,[387] or even between different strains of rats.[388] Even universal systems within mammals, such as the vasopressin- and oxytocin-reactive and -expressing neurons, can and frequently do show sex differences in many species, but these sex differences are (1) not always present and (2) not always consistent when present (i.e. different species may possess sex differences in the vasopressin and oxytocin systems but connect differently to adjacent systems or act in opposing ways).[389]

Despite this scattershot of disanalogous aspects and complexities, sex differences were assumed to apply to model animals and humans in similar ways unless proven otherwise. This assumption pushed some scientists to reject an older theory about human sexual desire in women: that human women, unlike all other known female mammals, rely on testosterone to increase sexual desire.[390] Others drew a direct line between all behaviors showing sex differences across species:

> *"In animals, behaviors that show sex differences seem to be susceptible to the influences of sex hormones (Hines, 2009):*[391] *sex hormones are known to affect sex-typed behavior, but its effects on gender identity are for obvious reasons impossible to study in animals.*

[386] Villa *et al.*, "Sexual differentiation of microglia," p. 160.
[387] Velíšková and DeSantis, "Sex and hormonal influences on seizures and epilepsy," p. 268.
[388] Amandusson and Blomqvist, "Estrogenic influences in pain processing," p. 342.
[389] Albers, "Species, sex and individual differences in the vasotocin/vasopressin system."
[390] Wallen, "Women are not as unique as thought by some"; see also James R. Roney and Zachary L. Simmons, "Hormonal predictors of sexual motivation in natural menstrual cycles," *Hormones and Behavior 63*(4): April 2013, DOI: 10.1016/j.yhbeh.2013.02.013, p. 636-645.
[391] M. Hines, "Gonadal hormones and sexual differentiation of human brain and behavior," in *Hormones, Brain and Behavior,* Part IV, 2nd ed., edited by Donald W. Pfaff, Arthur P. Arnold, Anne M. Etgen, Susan E. Fahrbach, and Robert T. Rubin, p. 1869-1909; Elsevier Academic Press, DOI: 10.1016/B978-008088783-8.00059-0.

> *Because the largest of all psychological sex differences in humans is gender identity (Hines, 2009),*[392] *sex hormones likely contribute to its development."*[393]

This logical leap requires the assumption that behavioral sex differences emerge from the same hormonal organization and activation across species, rather than other sources like genetics or socialization in one species versus another. Still others drew a direct line between behaviors, or their nearest approximations, in humans and model animals, implicitly assuming that similar behaviors emerged from similar sources.[394]

The assumption of sex differences remaining consistent between animal models and humans extended to the choice of animal models to use. A mouse model system of prenatal Zika infections' effects on behavior used four strains to test if prenatal Zika infection could have later neurobehavioral effects in humans.[395] Two strains showed few behavioral effects of prenatal Zika infection, one strain showed effects across behaviors that were similar between sexes, and one showed significant effects on behavior in female mice only.[396] The authors focus almost exclusively on the strain that showed sex differences in their discussion section without demonstrating its unique relevance as a model in humans, and further speculate that ADHD and impulsive behaviors, including later drug addiction, may be more common in girls than boys who experienced prenatal Zika infections.[397] The authors assumed that the presence of sex differences in one strain's response made that Zika model more relevant to the potential human

[392] Ibid.
[393] Thomas D. Steensma, Baudewijntje P. C. Kreukels, Annelou L. C. de Vries, Peggy T. Cohen-Kettenis, "Gender identity development in adolescence," *Hormones and Behavior 64*(2): July 2013, DOI: 10.1016/j.yhbeh.2013.02.020, p. 291.
[394] McHenry *et al.*, "Sex differences in anxiety and depression," p. 47.
[395] Abigail Snyder-Keller, Laura Kramer, Steven Zink, Valerie J. Bolivar, "Mouse Strain and Sex-Dependent Differences in Long-term Behavioral Abnormalities and Neuropathologies after Developmental Zika Infection," *Journal of Neuroscience 39*(27): 3 July 2019, DOI: 10.1523/JNEUROSCI.2666-18.2019, p. 5393-5394.
[396] Ibid., p. 5400.
[397] Ibid., p. 5401-5402.

consequences without presenting any evidence or acknowledging, explicitly or implicitly, that the choice of which results to focus on and extrapolate to human behavior was a choice.

The final assumption is that the behaviors exhibited by model animals were not contextual to a (particular) lab environment. One paper, comparing human and rodent literature on the hypothalamic-pituitary-adrenal axis, suggested that:

> *"If it were possible to control for the same number of variables in humans as it is in rodents (e.g. diet, housing conditions, etc.) perhaps consistently higher levels of cortisol would be observed in women compared to men. Alternatively, the neuroendocrine response in female rodents may simply be different than it is in women. … [E]xploring the molecular mechanisms underlying this sex difference in rodents could prove to be clinically relevant."*[398]

Despite the last sentence, either of these possibilities undermines the clinical relevance of rodent models. The latter possibility could still leave rodent models marginal clinical utility as a drug testing system for specific cases and could lend some insight into the effects of, for example, cortisol on neural systems in general, but without knowledge of which parts of the hypothalamic-pituitary-adrenal axis differ between species, the models could be as misleading as they are useful with only trial and error in clinical trials to determine their relevance. The former possibility suggests that any model animal system could fail to have any clinical relevance whatsoever, because human behavior cannot be ethically controlled to that extent.

Model animal systems are artificially limited, both in their environment and in their genetics. Strains exist to generate specific and consistent biological and behavioral backgrounds

[398] Debra A. Bangasser and Rita J. Valentino, "Sex differences in stress-related psychiatric disorders: Neurobiological perspectives," *Frontiers in Neuroendocrinology 35*(3): August 2014, DOI: 10.1016/j.yfrne.2014.03.008, p. 306.

against which tests can be run; in the process of creating the experimental technology, individual variation is minimized.[399] On top of that, animals from the same litter will share more environmental aspects than genetically similar individuals from different litters, which can affect the similarity of their behavior or biology.[400] Additionally, a commentary in 2002 pointed out that laboratory conditions decrease the survival of adult neurons because of their "relatively deprived conditions,"[401] and a review of sexually differentiated primate behavior noted that some behaviors, particularly juvenile aggression, were "very situationally variable, occurring in some environments and not in others [98].[402],[403] These aspects all suggest that the clinical relevance of sex differences in cortisol regulation in inbred laboratory mice in invariant environments could pale in comparison to the clinical relevance of the greater individual variation between humans—genetically, epigenetically, prenatally, environmentally, and socially. In fact, there is no particular reason to believe that sex would remain a reliable clinical marker of any relevant biological information in the general population with this additional variability.

The question of whether experiments using model animals can be extrapolated to human contexts is a thorny one and highly dependent on context. Model animals are limited, yes, but intentionally so: performing experiments means also limiting variation outside of the variables of interest in order to draw reliable conclusions. The ability to characterize biological systems in an

[399] J. M. Koolhaas, S. F. de Boer, C. M. Coppens, B. Buwalda, "Neuroendocrinology of coping styles: Towards understanding the biology of individual variation," *Frontiers in Neuroendocrinology 31*(3): July 2010, DOI: 10.1016/j.yfrne.2010.04.001, p. 307-321.
[400] Donald R. Williams, Rickard Carlsson, and Paul-Christian Bürkner, "Between-litter variation in developmental studies of hormones and behavior: Inflated false positives and diminished power," *Frontiers in Neuroendocrinology 47*: October 2017, DOI: 10.1016/j.yfrne.2017.08.003, p. 154-166.
[401] Elizabeth Gould and Charles G. Gross, "Neurogenesis in Adult Mammals: Some Progress and Problems," *Journal of Neuroscience 22*(3): 1 February 2002, DOI: 10.1523/JNEUROSCI.22-03-00619.2002, p. 621.
[402] Kim Wallen, "Nature Needs Nurture: The Interaction of Hormonal and Social Influences on the Development of Behavioral Sex Differences in Rhesus Monkeys," *Hormones and Behavior 30*(4): December 1996, DOI: 10.1006/hbeh.1996.0042, p. 364-378.
[403] Wallen, "Hormonal influences on sexually differentiated behavior in nonhuman primates," p. 10.

organism is unlikely to first describe *Homo sapiens*, a multicellular, slowly developing eukaryote with multiple microbiomes and interacting and specialized organ systems that is the last member of its genus and diverged from its nearest relatives at least 6 million years ago.[404] Practicing on less complex and idiosyncratic cases could help biology develop the frameworks that will hopefully, someday, describe humans. However, what an individual model organism's reliably observed behavior reveals about humans, or even concepts like 'sex', is far less clear.

In 2016, a review admitted "a discomforting truth": giving female rodents testosterone or estradiol to organizationally masculinize them did not make those female rodents unambiguously, or even mostly, male on the level of RNA transcription or epigenetic markers.[405] The review argued that this suggested that male and female phenotypes were "canalized"—essentially, relatively stable against disruption, with most individuals becoming either male or female.[406] Not only is this argument suggesting, as discussed in §3.5, that sex lies below

[404] The population that would eventually diverge into bonobos and chimpanzees seems to have diverged from the population that would become the *Homo* genus at least 6 million years ago, though estimates as high as 8 million years ago and as low as 4 million years ago have been published in the last 15 years. The other hominid species move between genii as new paleontological information is discovered and theoretical frameworks change, but present scientific information suggests two other genii (*Australopithecus* and *Paranthropus*) as well as a number of other species in *Homo* have gone extinct. Regardless, *Homo sapiens* has no close extant evolutionary relatives. Kay Prüfer, Kasper Munch, Ines Hellmann, Keiko Akagi, Jason R. Miller, Brian Walenz, Sergey Koren, Granger Sutton, Chinnappa Kodira, Roger Winer, James R. Knight, James C. Mullikin, Stephen J. Meader, Chris P. Ponting, Gerton Lunter, Saneyuki Higashino, Asger Hobolth, Julien Dutheil, Emre Karakoç, Can Alkan, Saba Sajjadian, Claudia Rita Catacchio, Mario Ventura, Tomas Marques-Bonet, Evan E. Eichler, Claudine André, Rebeca Atencia, Lawrence Mugisha, Jörg Junhold, Nick Patterson, Michael Siebauer, Jeffrey M. Good, Anne Fischer, Susan E. Ptak, Michael Lachmann, David E. Symer, Thomas Mailund, Mikkel H. Schierup, Aida M. Andrés, Janet Kelso, and Svante Pääbo, "The bonobo genome compared with the chimpanzee and human genomes," *Nature 486*: 2012, DOI: 10.1038/nature11128, p. 527-531; Rui Diogo, Julia L. Molnar, and Bernard Wood, "Bonobo anatomy reveals stasis and mosaicism in chimpanzee evolution, and supports bonobos as the most appropriate extant model for the common ancestor of chimpanzees and humans," *Scientific Reports 7*, ID 608: 2017, DOI: 10.1038/s41598-017-00548-3, p. 1-8; Hans P. Püschel, Ornella C. Bertrand, Joseph E. O'Reilly, René Bobe, and Thomas A. Püschel, "Divergence-time estimates for hominins provide insight into encephalization and body mass trends in human evolution," *Nature Ecology and Evolution 5*: 2021, DOI: 10.1038/s41559-021-01431-1, p. 808-819; but see also the "Matters Arising" in response to that article in 4 July 2022 (*Nature Ecology and Evolution 6,* p. 1090-1091) and the response in the same publication (p. 1092-1094).

[405] Forger *et al.*, "Cellular and molecular mechanisms of sexual differentiation in the mammalian nervous system," p. 80.

[406] Ibid.

phenotypes rather than being part of an eventual phenotype, it implies that the binary categorizations "male" or "female" reflect biological forces in rodents and in humans. If developmental and evolutionary forces keep most organisms in most species either unambiguously phenotypically male or unambiguously phenotypically female, and if organisms that model human development also develop this way, then the most plausible conclusion based on this evidence is that human development would also show developmental, biological, evolutionarily selected canalization into two sexes. While the conclusion might or might not be true, the argument's coherence relies on the premise that the development trajectories of model organisms meaningfully reflect the development of human organisms. Perhaps model organisms do reflect humans in this aspect, but there is an absence of supporting evidence due to the inability to directly investigate or manipulate human development for ethical reasons and a wealth of disanalogies between humans and their models. The "model" part of "model organism" was never proven nor even justified: so why had it been assumed?

That assumption may have been, at least in part, a result of the factors creating the replication crisis. As the editor of *eNeuro* asserted in 2019, quoted in §3.1, conclusions had to be strong in order to be published in the highest-prestige journals.[407] As the p-value requirements tended to amplify sex differences and silently discard sex similarities, so too might the requirement for bold and interesting conclusions have warped the published research. Additionally, an early commentary about the replication crisis asked:

> *"If there is an explicit or implied claim of broad relevance, is it reasonable to respond to a failure to replicate by saying that "you didn't do the experiment the same way" (meaning that the findings are conditional and apply only in a particular set of*

[407] Bernard, "Changing the Way", p. 2.

> *experimental conditions)? This highlights another trend; the missing 'caveats' section in Discussions. For any novel finding based on a limited dataset, it would be more prudent to say that the findings* might *only apply in highly constrained circumstances until proven otherwise. [italics in original]*"[408]

Are model organisms' behavioral patterns good reflections of human behavioral patterns? Can this research be extrapolated from model animals to humans? Why might these results fail to translate? These caveats and questions would have undercut claims of clinical applicability and human relevance. The framework within which behavioral neuroendocrinology operated implicitly assumed that "sex" can be decontextualized from species and environment and remain transcendently meaningful when describing behavior, developmental trajectories, neuroanatomy, and endocrinological states. While criticism of model organisms' uses in research did exist throughout the period 2000-2019, it made little impact even after 2016, perhaps due to a lack of other similarly useful experimental technologies.

3.8: What piece of work is a woman[409]

A further unique, or at least extremely unusual, aspect of *Homo sapiens* not mentioned in the previous section is that humans menstruate. Primates (including humans), apes, (most) Old World monkeys, four species of bats, the elephant shrew, and the common spiny mouse are the only placental mammals known to menstruate.[410] Although most organisms undergo estrous

[408] Steward, "A Rhumba of 'R's': Replication, Reproducibility, Rigor, Robustness," p. 3.
[409] Reference to Shakespeare's *Hamlet*, act II, scene 2. The original, as in the Second Quarto (1604): "What piece of work is a man! how noble in reason, how infinite in faculty, in form and moving, how express and admirable in action, how like an angel in apprehension, how like a god; the beauty of the world; the paragon of animals. And yet to me what is this quintessence of dust?" William Shakespeare, "Hamlet (Modern, Quarto 2)," *Internet Shakespeare Editions*, ed. David Bevington, University of Victoria: last modified 11 January 2019, internetshakespeare.uvic.ca/doc/Ham_Q2M/scene/2.2/index.html.
[410] Nadia Bellofiore, Fiona Cousins, Peter Temple-Smith, Hayley Dickinson, and Jemma Evans, "A missing piece: the spiny mouse and the puzzle of menstruating species," *Journal of Molecular Endocrinology 61*(1): July 2018, DOI: 10.1530/JME-17-0278, p. R25-R41.

cycles instead, both menstrual and estrous cycles involve hormonal fluctuations which, based on the theory of hormonal activation, were expected to affect sex-typed behaviors. Some researchers found that hormonal contraceptives, which alter levels of circulating hormones to prevent pregnancy by preventing ovulation, sperm movement through the cervix, and possibly fetal implantation in the womb,[411] may affect fear processing[412] and spatial ability scores.[413] Other researchers argued that "[t]he menstrual cycle with its changing hormone levels provides an elegant natural paradigm"[414] and compared the same subjects at different points of the menstrual cycle in an attempt to isolate the direct effects of the differing levels of estrogen, progesterone, and testosterone.[415]

Far more commonly, however, menstrual cycles were described *post hoc* as a possible factor introducing variability into the data,[416] causing contradictions between studies,[417] or a

[411] Roberto Rivera, Irene Yacobson, and David Grimes, "The mechanism of action of hormonal contraceptives and intrauterine contraceptive devices," *American Journal of Obstetrics and Gynecology 181*(5): November 1999, DOI: 10.1016/s0002-9378(99)70120-1, p. 1263-1269.
[412] Pearson and Lewis, "Fear recognition across the menstrual cycle."
[413] van Anders and Hampson, "Testing the prenatal androgen hypothesis."
[414] Jill M. Goldstein, Matthew Jerram, Russell Poldrack, Todd Ahern, David N. Kennedy, Larry J. Seidman and Nikos Makris, "Hormonal Cycle Modulates Arousal Circuitry in Women Using Functional Magnetic Resonance Imaging," *Journal of Neuroscience 25*(40): 5 October 2006, DOI: 10.1523/JNEUROSCI.2239-05.2005, p. 9309.
[415] Goldstein *et al.,* "Hormonal Cycle Modulates Arousal Circuitry in Women"; Jill M. Goldstein, Matthew Jerram, Brandon Abbs, Susan Whitfield-Gabrieli, and Nikos Makris, "Sex Differences in Stress Response Circuitry Activation Dependent on Female Hormonal Cycle," *Journal of Neuroscience 30*(2): 13 January 2010, DOI: 10.1523/JNEUROSCI.3021-09.2010, p. 431-438.
[416] Elke Smith, Jessica Junger, Katharina Pauly, Thilo Kellermann, Joseph Neulen, Christiane Neuschaefer-Rube, Birgit Derntl, Ute Habel, "Gender incongruence and the brain – Behavioral and neural correlates of voice gender perception in transgender people," Hormones and Behavior *105*: September 2018, DOI: 10.1016/j.yhbeh.2018.07.001, p. 19-20.
[417] Guillén Fernández, Susanne Weis, Birgit Stoffel-Wagner, Indira Tendolkar, Markus Reuber, Stefan Beyenburg, Peter Klaver, Jürgen Fell, Armin de Greiff, Jürgen Ruhlmann, Jürgen Reul and Christian E. Elger, "Menstrual Cycle-Dependent Neural Plasticity in the Adult Human Brain Is Hormone, Task, and Region Specific," *Journal of Neuroscience 23*(9): 1 May 2003, DOI: 10.1523/JNEUROSCI.23-09-03790.2003, p. 3794; Belinda Pletzer, Selina Jäger, and Stefan Hawelka, "Sex hormones and number processing. Progesterone and testosterone relate to hemispheric asymmetries during number comparison," *Hormones and Behavior 115*, ID 104553: September 2019, DOI: 10.1016/j.yhbeh.2019.07.001, p. 2.

possible source of inapplicability of the results of that paper.[418] One paper discussed their results from a large fMRI study attempting to determine if there were sex differences in emotionally-valenced memory as follows:

> *"We could identify corresponding patterns in fMRI during encoding regarding the interaction between sex and valence category on picture ratings. However, it was not possible to show corresponding patterns between behavior and fMRI for the subsequent memory effect during encoding. We cannot rule out the possibility that the lack of valence-category-specific sex differences in brain activity might have been influenced by the heterogeneity of the females group concerning their use of birth control methods, as well as admixture of women in different stages of their cycle as reported in literature for several cognitive domains (Rumberg et al., 2010;*[419] *Bonenberger et al., 2013;*[420] *Marecková et al., 2014*[421]*)."*[422]

That paper, however, fell at a critical hurdle, as the male and female data sets had nearly identical standard deviations in their reported data.[423] Presumably, if menstrual cycles and birth

[418] David A. Puts, Lauramarie E. Pope, Alexander K. Hill, Rodrigo A. Cárdenas, Lisa L. M. Welling, John R. Wheatley, S. Marc Breedlove, "Fulfilling desire: Evidence for negative feedback between men's testosterone, sociosexual psychology, and sexual partner number," *Hormones and Behavior 70*: April 2015, DOI: 10.1016/j.yhbeh.2015.01.006, p. 20.
[419] Bastian Rumberg, Anneke Baars, Jochen Fiebach, Mark E. Ladd, Michael Forsting, Wolfgang Senf, and Elke R. Gizewski, "Cycle and gender-specific cerebral activation during a verb generation task using fMRI: Comparison of women in different cycle phases, under oral contraception, and men," *Neuroscience Research 66*(4): April 2010, DOI: 10.1016/j.neures.2009.12.011, p. 366-371.
[420] Martina Bonenberger, Rebecca C. Groschwitz, Daniela Kumpfmueller, Georg Groen, Paul L. Plener, and Birgit Abler, "It's all about money: oral contraception alters neural reward processing," *NeuroReport 24*(17): December 4, 2013, DOI: 10.1097/WNR.0000000000000024, p. 951-955.
[421] Klara Marečková, Jennifer S. Perrin, Irum Nawaz Khan, Claire Lawrence, Erin Dickie, Doug A. McQuiggan, Tomáš Paus, and the IMAGEN Consortium, "Hormonal contraceptives, menstrual cycle and brain response to faces," *Social Cognitive and Affective Neuroscience 9*(2): February 2014, DOI: 10.1093/scan/nss128, p. 191-200.
[422] Klara Spalek, Matthias Fastenrath, Sandra Ackermann, Bianca Auschra, David Coynel, Julia Frey, Leo Gschwind, Francina Hartmann, Nadine van der Maarel, Andreas Papassotiropoulos, Dominique de Quervain, and Annette Milnik, "Sex-Dependent Dissociation between Emotional Appraisal and Memory: A Large-Scale Behavioral and fMRI Study," *Journal of Neuroscience 35*(3): 21 January 2015, DOI: 10.1523/JNEUROSCI.2384-14.2015, p. 933.
[423] Ibid., Table 2, p. 924; Figure 4, p. 930.

control were introducing variability and no similar processes introduced variability among those incapable of menstruating, the female data should have shown noticeably more variability than the male data. The absence of a justification based on the data collected in the study suggests that this *post hoc* argument about possible variation introduced by menstrual cycles and birth control could be used to explain any unexpected non-significant or variable results about women's behavior.

The standard assumption during the period 2000-2019 was that menstrual cycles and birth control represented "extraneous variables" which obscured "underlying relationships" in the data.[424] However, other researchers argued that "the circadian rhythm of testosterone [in males] has a stronger effect on behavior than the menstrual cycle."[425] "Further," as an article recommending experimental changes in hormonal research noted in 2017,

> *"the perception that sources of hormonal variation in women are problematic for study designs is especially unfair given that researchers do not exclude male participants on account of the variety of factors that reliably impact testosterone levels in men – body fat,*

[424] van Anders and Hampson, "Testing the prenatal androgen hypothesis," p. 93.
[425] Cohen-Bendahan *et al.,* "Is there an effect of prenatal testosterone on aggression and other behavioral traits?", p. 235.

relationship status, and parental status (Burnham et al., 2003,[426] *Gray et al., 2002,*[427] *Pasquali, 2006,*[428] *van Anders and Goldey, 2010,*[429] *Vermeulen et al., 1999*[430]*).* "[431]

Perhaps one of the most interesting criticisms, however, came from a student review[432] of a 2014 experiment considering whether estradiol levels correlated with an increased tendency to choose an immediate small reward over a later large reward.[433] The review pointed out that even if changed estradiol levels caused the change in reward evaluation, the effect may not hold up outside of the laboratory—though the student did still argue in favor of researchers adding menstrual cycle effects to future experimental designs.[434]

What the analytical framework of "menstrual cycle and/or birth control effects" provided was a reason not to engage with the possibility that the hypothesized effects did not actually exist or were extremely small. For example, consider a hypothetical case where the extant literature suggested that women generally differed from men but differed inconsistently between experiments (sometimes with a higher *x* and sometimes with a lower *x*) in a hormonally linked

[426] T. C. Burnham, J. Flynn Chapman, P. B. Gray, M. H. McIntyre, S. F. Lipson, P. T. Ellison, "Men in committed, romantic relationships have lower testosterone," *Hormones and Behavior 44*(2): August 2003, DOI: 10.1016/S0018-506X(03)00125-9, p. 119-122.
[427] Peter B. Gray, Sonya M. Kahlenberg, Emily S. Barrett, Susan F. Lipson, Peter T. Ellison, "Marriage and fatherhood are associated with lower testosterone in males," *Evolution and Human Behavior 23*(3): May 2002, DOI: 10.1016/S1090-5138(01)00101-5, p. 193-201.
[428] Renato Pasquali, "Obesity and androgens: facts and perspectives," *Fertility and Sterility 85*(5): May 2006, DOI: 10.1016/j.fertnstert.2005.10.054, p. 1319-1340.
[429] Sari M. van Anders and Katherine L. Goldey, "Testosterone and partnering are linked via relationship status for women and 'relationship orientation' for men," *Hormones and Behavior 58*(5): November 2010, DOI: 10.1016/j.yhbeh.2010.08.005, p. 820-826.
[430] A. Vermeulen, S. Goemaere, and J. M. Kaufman, "Sex hormones, body composition and aging," *The Aging Male 2*(1), DOI: 10.3109/13685539909003178, p. 8-15.
[431] Kathleen V. Casto, Smrithi Prasad, "Recommendations for the study of women in hormones and competition research," *Hormones and Behavior 92*: June 2017, DOI: 10.1016/j.yhbeh.2017.05.009, p. 191.
[432] Stephanie J. Dimitroff, "Phasic Estradiol Levels and Bias for Immediate Rewards," *Journal of Neuroscience 34*(37): 10 September 2014, DOI: 10.1523/JNEUROSCI.2377-14.2014, p. 12239-12240. The Journal Club section of the *Journal of Neuroscience* exclusively published reviews by graduate students and postdoctoral fellows of other scientific papers.
[433] This concept is known as "delayed discounting."
[434] Dimitroff, "Phasic Estradiol Levels and Bias for Immediate Rewards."

trait. If this pattern was placed in the context of the replication crisis, that multiple papers found statistically significant effects in different directions but no or few papers found no statistically significant effect would suggest that the statistically significant effects had been generated by sampling bias. The statistically significant but contradictory papers' domination of the available literature followed from a publication environment that preferentially published statistically significant papers that made dramatic claims (e.g. "what we believed about *x* was wrong all along"). An explanation relying on menstrual cycle effects, however, would suggest that women exhibited higher variability in the measured trait than men as a result of the menstrual cycle's hormonal changes and differing hormonal contraceptive formulations.

Furthermore, this assumption about the unique difficulty of studying women or female animals in part due to their hormonal variability had important consequences in other research contexts. Much research habitually excluded women[435] and female animals from study designs, a trend so ubiquitous that, as briefly mentioned in §3.5, the National Institutes of Health (NIH) created a policy which made inclusion of both male and female animals the default for any preclinical research that wanted to receive NIH funding after January 2016.[436] Other researchers limited hormonal research to women using hormonal contraception, arguing that the choice was "a strength of our methods, as it should reduce the influence of cyclic fluctuations in ovarian hormones."[437]

The benefits of the argument that menstrual cycles caused unexpected, contradictory, or nonsignificant results covered all outcomes—at least for the researchers involved. Perhaps the argument would convince funders of the necessity of further and likely more methodologically

[435] Casto and Prasad, "Recommendations for the study of women in hormones and competition research," p. 190.
[436] US National Institutes of Health, NIH Notice Number: NOT-OD-15-102.
[437] Puts *et al.*, "Fulfilling desire: Evidence for negative feedback between men's testosterone," p. 20.

complex (i.e. expensive) study to examine the "underlying relationships" that menstrual cycles had obscured.[438] Should that research never be done, however, the argument would remain an option for any future research on the topic, perhaps ensuring that it could be published even if it was nonsignificant or difficult to straightforwardly interpret. It is also worth noting that even if menstrual cycles were responsible for contradictory or nonsignificant results, actual interest in characterizing women's menstrual cycles was minimal to nonexistent. Few studies, often with smaller numbers of participants, used the "elegant natural paradigm" of hormonal menstrual changes;[439] far more studies simply cut women from experimental designs. Menstrual cycles and hormonal contraceptive effects may accurately explain why results seemed or were contradictory or confusing, but their frequent invocation does not reflect evidence, as researchers did not generally gather evidence on the topic, but instead reflects assumptions that both supported researchers' institutional interests and covered for researchers' inability or unwillingness to examine the structural issues of the replication crisis.

3.9: Conclusion

> *"The observation is what it is, a fact, but its interpretation depends on the conceptual framework we are using. This framework is based on what we think we know at time* t. *Since our understanding of phenomena is constantly evolving, it is not surprising to find numerous examples in science when the most appropriate data interpretation had to wait years/decades following the initial observations. We all accept this, because this is how Science progresses. But there are more insidious traps in data interpretation, e.g.,*

[438] van Anders and Hampson, "Testing the prenatal androgen hypothesis," p. 93.
[439] Goldstein *et al.*, "Hormonal Cycle Modulates Arousal Circuitry in Women," p. 9309.

> *confounding factors that we are not aware of, or interpretations that we take for granted. [emphasis in original]"*[440]
>
> *"Consider that you have done everything right, as outlined in the 10 easy steps to modeling. You framed the question precisely, had specific testable hypotheses, choose the right toolkit, implemented the model, fit it to data, selected the right number of parameters/the best model, cross-validated your results, and compared your best model to alternatives from the literature. Does that mean your model is a good model? In fact, what are the criteria of a good model?"*[441]

The wider impact of built-up methodological imprecision, unexamined assumptions, inconsistent definitions, and systems which focus on an unrepresentative subset of the data left researchers unable to trust the literature. One paper in 2017 recounted that a repeatedly and consistently attested sex difference disappeared when researchers compensated for the overall difficulty of the task and ensured equal numbers of (supposedly) male-biased and female-biased tasks to complete, acidly recommending "that research investigating the influence of biological sex become more methodologically rigorous and theory-driven".[442] A 2019 review of research studying behaviors possibly affected by interactions between testosterone and cortisol, or the Dual Hormone hypothesis, admitted defeat: "Without clear definitions of what constitutes support, or lack thereof, for the Dual Hormone hypothesis, it is nearly impossible to evaluate the literature as a whole."[443] In most aspects, the bare facts of organizational/activational theory had

[440] Bernard, "Editorial: Experimental Bias in Electrophysiological Studies," p. 1.
[441] Gunnar Blohm, Konrad P. Kording, and Paul R. Schrater, "A How-to-Model Guide for Neuroscience," *eNeuro 7*(1): 11 February 2020, DOI: 10.1523/ENEURO.0352-19.2019, p. 9.
[442] K. Suzanne Scherf, Daniel B. Elbich, and Natalie V. Motta-Mena, "Investigating the Influence of Biological Sex on the Behavioral and Neural Basis of Face Recognition," *eNeuro 4*(2): 26 April 2017, DOI: 10.1523/ENEURO.0104-17.2017, p. 1.
[443] Grebe *et al.*, "Testosterone, cortisol, and status-striving personality features," p. 27.

already significantly changed by 2016, which is when ideas of consistently gendered brains finally collapsed. In an environment where researchers were already questioning whether the research they had taken for granted really could be wholly trusted, it seems that the researchers extended their questions to the theories, definitions, and methodologies they had also simply assumed to be true. That being said, there are parts of the research that most researchers did not investigate: the assumptions that supported organizational/activational theory's meaningfulness to lived experiences and to other disciplines.

Chapter 4: Adjacent disciplines

4.0: Introduction

> *"Any hesitation over the externality of Science was supposed to thrust us willy-nilly into 'mere social construction.' I maintain that it is fairly easy to escape the menacing choice between the reality of the external world and the prison of the social world. … Salvation through Science comes only in a world deprived in advance of any means to become moral, reasonable, and learned. But in order for this theory of Science to take the place of an explanation about the work of the sciences, a no less absurd theory of the social world has to take the place of analysis of public life."*[444]

The general topic of this thesis is the process of collecting and creating knowledge as a collective enterprise. Chapter 2 was about the process of the organizational/activational theory's decay, while Chapter 3 described the proximate methodological issues that likely provided the impetus for more researchers to reexamine the available evidence more carefully. Chapter 4 will discuss the assumptions which likely doomed organizational/activational theory's human applications at its conception. Assumptions have been a constant presence, asserting how bodies work or what measurements mean, but the assumptions in this chapter excluded alternative explanations for observations supporting organizational/activational theory. What united these alternative explanations is that they used historical or sociological information or theorizing to suggest that sexed/gendered differences were fully contingent on context or at least significantly altered by that context. Many of these assumptions went without notice, and all went without protest until 2014 at the earliest. However, each assumption reveals some aspect that behavioral

[444] Bruno Latour, *Politics of Nature: How to Bring the Sciences into Democracy*, Translated by Catherine Porter. Harvard University Press (Cambridge, MA): 2004, ebook accessed through ProQuest, p. 16-17.

neuroendocrinologists working on or adjacent to organizational/activational theory did not recognize they had assumed. Considering organizational/activational theory as a built structure, Chapter 2 examined the flaws in the stones and mortar and how only mild weather had noticeably eroded it, Chapter 3 described the ways in which the walls and roof had never been properly supported or designed with stable proportions, and Chapter 4 will examine the cracks in the foundation and argue that the house had been built on a fault line.

4.1: Assumed Theory—My Thinking Has Evolved

> *"It is easy to invent a selectionist explanation for almost any specific observation; proving it is another story."*[445]

Organizational/activational theorists and behavioral neuroendocrinology as a discipline assumed a specific version of evolutionary theory to apply to human evolutionary history, a theory that was primarily selective and adaptationist. Evolution by natural and sexual selection, as used across biology, theorizes that from a varying population, some of the varying genotypes and phenotypes have varying reproductive success—i.e. some of the individuals are more likely to have viable offspring than others. Natural and sexual selection are the processes by which individuals are more or less likely to survive to have offspring or to be selected as a mate. The pool of variation is generated randomly,[446] but selection is non-random.[447] When selection is acting directionally in response to local conditions, such as lengthening giraffes' necks over time

[445] Motoo Kimura, *The Neutral Theory of Molecular Evolution*, Cambridge University Press (Cambridge, UK): 1983, p. xiv.
[446] At least, maybe. See J. Grey Monroe, Thanvi Srikant, Pablo Carbonell-Bejerano, Claude Becker, Mariele Lensink, Moises Exposito-Alonso, Marie Klein, Julia Hildebrandt, Manuela Neumann, Daniel Kliebenstein, Mao-Lun Weng, Eric Imbert, Jon Ågren, Matthew T. Rutter, Charles B. Fenster, and Detlef Weigel. "Mutation bias reflects natural selection in *Arabidopsis thaliana*," *Nature 602*: 12 January 2022, DOI: 10.1038/s41586-021-04269-6, p. 101-105. However, see the "Matters Arising" response, as well as the substantive correction to the methodology.
[447] T. Ryan Gregory, "Understanding Natural Selection: Essential Concepts and Common Misconceptions," *Evolution: Education and Outreach 2*: 09 April 2009, DOI: 10.1007/s12052-009-0128-1, p. 156-175.

to reach higher leaves, the trait that was selected is considered an adaptation. However, modern versions of evolutionary theory include non-adaptationist forces as well.[448] Genetic drift refers to events which reduce the genetic composition of a population randomly, i.e. non-selectively, generally through the deaths of a large but arbitrary part of an original population (bottleneck events) or due to the relatively abrupt geographic isolation of a subset of an original population (founder effects). In addition, selection can only act on a population's extant traits, and every step in an adaptation is also being selected on within its evolutionary context. Each precursor of what biologists eventually call an adaptation must either be beneficial or neutral[449] to the members of the species that possess it.

Organizational/activational theory could not grapple effectively with non-adaptationist or imperfectly adaptationist evolution and contained possible evolutionary misconceptions.[450] Behavioral neuroendocrinology as a discipline often referred to evolutionary explanations, specifically the explanation of the natural selection or sexual selection of a trait, to explain what were believed to be universally common and fundamentally sexed/gendered psychological structures. For example, about sex-typed cognition: "Sexually dimorphic cognitive abilities have been hypothesized to have evolutionary origins, with male-specific demands for hunting early in our species conferring an advantage for those with better visuospatial abilities, and more female-specific demands for socialization and child-rearing conferring an advantage for those with

[448] Somewhat of a sidetrack but also in play is that humans are mutualists with a number of species—the domesticated species of plants and animals—and human evolution may be only fully comprehensible when studied with the mutualistic species which depend on humans and which humans depend on.
[449] I should say, beneficial or neutral *enough* to allow the members with the adaptation to proliferate for long enough that further mutations and recombination can occur.
[450] Gregory, "Understanding Natural Selection."

superior language abilities (Hines, 2011;[451] Alexander and Wilcox, 2012[452]).”[453] Evolution by natural selection was hypothesized to have selected for those males with better visuospatial abilities and those females with better linguistic (and social) abilities due to their (hypothesized) non-overlapping roles in early hominid societies. Besides the assumptions about neurological structure[454] and early hominid societies[455] required for this hypothesis, evolution was assumed to be acting selectively and adaptively, acting differently on males and females, and to have become a more or less universal division within humanity (“sexually dimorphic”).

It is the universality of these traits, and their sex-typing, which may represent a misconception about evolution. If psychology and neurology are sex-typical in the majority of humans across all populations based on heritable factors, then there are two general possibilities in an evolutionary framework: high selective pressure or low population variability. The first possibility is that variation is (or was) being consistently selected out of human populations through natural or sexual selection; the second possibility is that variation within humanity is low to nonexistent due to historical bottleneck events and other results of genetic drift.[456] It is perhaps

[451] Melissa Hines, “Gender Development and the Human Brain,” *Annual Review of Neuroscience 34*: July 2011, DOI: 10.1146/annurev-neuro-061010-113654, p. 69-88.
[452] Gerianne M. Alexander and Teresa Wilcox, “Sex Differences in Early Infancy,” *Child Development Perspectives 6*(4): December 2012, DOI: 10.1111/j.1750-8606.2012.00247.x, p. 400-406.
[453] Kerstin J. Plessen, Kenneth Hugdahl, Ravi Bansal, Xuejun Hao, and Bradley S. Peterson, “Sex, Age, and Cognitive Correlates of Asymmetries in Thickness of the Cortical Mantle Across the Life Span,” *Journal of Neuroscience 34*(18): 30 April 2014, DOI: JNEUROSCI.3692-13.2014, p. 6300.
[454] It is not clear why better visuospatial abilities must be ‘traded off’ with better verbal abilities as traits.
[455] It is not clear to what degree early hominid societies divided their labor up by sex in this fashion, nor the rigidity of these boundaries. One could also insist that spatial ability must be linked with remembering gathering areas for food over long periods of time, and navigating to these areas, and verbal ability must be linked to the group organization for hunting.
[456] A recent paper suggests that around the hypothesized time of human chromosomal fusion (chromosome 2 in humans is a combination of two chromosomes in all other primate and ape species) about 900,000 years ago, humans went through a ‘severe population bottleneck’. Bottleneck events are severe reductions in gene variety, generally due to an abruptly reduced population because of the death of population members or because of a smaller group becoming a founder population in a migration. Wangjie Hu, Ziqian Hao, Pengyuan Du, Fabio Di Vincenzo, Giorgio Manzi, Jialong Cui, Yun-Xin Fu, Yi-Hsuan Pan, and Haipeng Li, “Genomic inference of a severe human bottleneck during the Early to Middle Pleistocene transition,” *Science 381,* no. 6661: 31 August 2023, DOI:

unsurprising that behavioral neuroendocrinology papers making evolutionary arguments relying on selection pressure for or against sex-typed psychological or neurological features assume the first possibility rather than the second, but that human populations display relatively low neutral genetic diversity compared to other species would point to the second possibility's relevance.[457] Researchers did not justify their choice to assume the priority of selective mechanisms. More generally, however, evolutionary theory tends to emphasize the diversity within populations and seeks explanations for why variation does not exist. "Typic" or "essentialist" visions of biology frequently engender misinterpretations of evolutionary theory. The idea of "sex-typed" cognition based in evolutionary adaptation requires explanation. In particular, an explanation is required as to how the sex-typed division of labor (specifically *this* sex-typed division of labor, and no other) so completely outcompeted alternative possibilities that sex, a variable believed to apply to all humans, possesses a consistent cognitive "type" rather than, at best, a vague trend that should be nearly undetectable below all other influences on cognitive development.[458]

Additionally, understanding what is and is not an adaptation in humans, and further why that adaptation might exist, may be impossible in humans for lack of evidence. Humans have only a few related extant species, each relatively distant in evolutionary time and ecological niche (chimpanzees, bonobos, orangutans, gorillas, etc.). Because humans possess many unique traits among living species, comparisons cannot address human traits of interest (sophisticated

10.1126/science.abq7487, p. 979-984. Similar arguments about human population bottlenecks in later periods, including the 'out of Africa' transition and the Eurasian to Americas transition at the Bering Strait, have also been made. The 1000 Genomes Project Consortium, "A global reference for human genetic variation," *Nature 526*: 2015, DOI: 10.1038/nature15393, p. 68-74.

[457] Ellen M. Leffler, Kevin Bullaughey, Daniel R. Matute, Wynn K. Meyer, Laure Ségurel, Aarti Venkat, Peter Andolfatto, and Molly Przeworski, "Revisiting an Old Riddle: What Determines Genetic Diversity Levels within Species?" *PLOS Biology 10*(9): 11 September 2012, DOI: 10.1371/journal.pbio.1001388, Table 1 and 2.

[458] Gregory, "Understanding Natural Selection," specifically "Typological, Essentialist, and Transformationist Thinking," p. 171-172.

toolmaking, language, bipedalism, etc.). Modern researchers are left with cases like malarial resistance and sickle cell anemia or adult lactose tolerance—i.e. discrete genetic changes that affected specific subpopulations of humans, which may become universal but are not yet and may never be so.[459] Furthermore, these traits are by far the exception rather than the rule; most variation occurs within populations rather than between them. Those studying the evolution of humanity's physical characteristics can at least compare fossils, but the psychological and neurological architecture of Pleistocene humans or other members of the *Homo* genus cannot reliably be reconstructed. Did humans and humans' ancestors neurologically and/or psychologically adapt during the last six million years? Almost certainly—but an absence of evidence about the psychological differences between ancestral population(s) (assessed at different times) and the modern population prevents a clear understanding of what changed, let alone why.[460] Even such theoretical staples as "Man the Hunter," argued in the first quote in this section, cannot be proven and face significant contradictory evidence from the past and present.[461]

These problems can be viewed as manifestations of a larger problem: that evolutionary arguments as applied to humanity in specific are historical arguments—not in their methodologies or in their data, but at their core. Max Weber once argued, when considering how and whether the social sciences and history could still be objective, that: "In the exact natural sciences, "laws" are as important and meaningful as they are *universal*; but for knowledge of

[459] Jonathan Michael, Kaplan, "Historical Evidence and Human Adaptations," *Philosophy of Science 69*(S3): September 2002, DOI: 10.1086/341853, p. S294-S304.
[460] Subrena E. Smith, "Is Evolutionary Psychology Possible?" *Biological Theory*: 05 December 2019, DOI: 10.1007/s13752-019-00336-4, p. 1-11.
[461] Abigail Anderson, Sophia Chilczuk, Kaylie Nelson, Roxanne Ruther, and Cara Wall-Scheffler, "The Myth of Man the Hunter: Women's contribution to the hunt across ethnographic contexts," *PLOS One 18*(6): 28 June 2023, DOI: 10.1371/journal.pone.0287101.

historical phenomena in their concrete conditions, the general laws, because they are the emptiest of content, are as a rule also the most worthless. As a *generic* term becomes more comprehensively valid—extends its *scope*—it increasingly sends us *away* from the richness of reality, because *in order* to collect the largest possible number of phenomena, to be as abstract as possible, it must contain *nothing.*"[462] The general laws of evolution (natural selection, sexual selection, genetic drift, and so on) outline the generic forces acting on all species at all times. These general laws, however, can only generate a list of the possible types of practically infinite forces that shaped human evolution. The rules of evolutionary development cannot reveal to what degree these forces affected the history of the species of interest: humans. When considering the sex differences humans may or may not possess, all evolutionary arguments on the topic are fundamentally historical arguments about human evolution. The lack of available information makes these arguments difficult or perhaps impossible to substantiate but does not change their historical character.

At present, there is simply insufficient evidence of the specifics of human evolutionary history to make substantiated evolutionary (historical) claims about specific modern aspects of human behavior. For example, a 2017 review paper in *Hormones and Behavior* stated that:

> *"There may have been selection for women to distribute their sexual behavior more evenly across the ovulatory cycle. Perhaps doing so enabled ancestral women to obtain good genes for offspring by copulating contingently outside of the pair-bond while*

[462] „Für die exakte Naturwissenschaft sind die „Gesetze" um so wichtiger and wertvoller, je *allgemeingültiger* sie sind, für die Erkenntnis der historischen Erscheinungen in ihrer konkreten Voraussetzung sind die allgemeinsten Gesetze, weil die inhaltleersten, regelmäßig auch die wertlosesten. Denn je umfassender die Geltung eines *Gattungs*begriffes—sein *Umfang*—ist, desto mehr führt er uns von der Fülle der Wirklichkeit *ab,* da er ja, *um* das Gemeinsame möglichst vieler Erscheinungen zu enthalten, möglichst abstrakt, also inhalts*arm*sein muß. (p. 54) Max Weber, „Die „Objektivität" sozialwissenschaftlicher und sozialpolitischer Erkenntnis," *Archiv für Sozialwissenschaft und Sozialpolitik 19,* no. 1: 1904, p. 22-87. (translation mine) For length reasons, text that was originally spaced (e.g. U m f a n g) is italicized in both the citation and the translation.

> *retaining non-genetic benefits (e.g., food provisioning) conferred by a pair-bond partner (Thornhill and Gangestad, 2015).*[463] *Indeed, in one study, women were especially self-assertive toward their partners during the periovulatory phase, and reported engaging in more behaviors that resist male vigilance and mate guarding, especially if they reported greater attraction to men other than their partners (Gangestad et al., 2014)*[464]*.''*[465]

It cannot be proven that human women engaging in sexual behavior regardless of fertility is a selected adaptation. Further, did hominids consistently, or consistently enough, form pair-bonds that selection would act on that structure? What is a "good" gene in the various contexts of hominid evolutionary history, and how would female hominids or primates distinguish those with "good" genes by their external traits? Do women in relationships who engage in extra-pair copulation in the present do so with men with "good" or "better" genes? (Is that one study representative of most women's behavior?) Most importantly, how do these authors know that this biological and psychological aspect, assuming it exists, was not a result of genetic drift? Which of the general laws are meaningful in this case, and which are not? The larger problem was not that these were historical arguments, but rather that the historical character of this research was unrecognized. Universal laws were being used to generate insights about a specific case: humans.

Despite a general absence of evidence, a possible reason for the popularity of evolutionary arguments about the origins of organization and activation is the explanatory power

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[463] Randy Thornhill and Steven W. Gangestad, "The Functional Design and Phylogeny of Women's Sexuality," in *The Evolution of Sexuality*, eds. T. Shackelford and R. Hansen, Springer: 2015, DOI: 10.1007/978-3-319-09384-0_8, p. 149-184.
[464] Steven W. Gangestad, Christine E. Garver-Apgar, Alita J. Cousins, and Randy Thornhill, "Intersexual conflict across women's ovulatory cycle," *Evolution and Human Behavior 35*(4): July 2014, DOI: 10.1016/j.evolhumbehav.2014.02.012, p. 302-308.
[465] Natalie V. Motta-Mena and David A. Puts, "Endocrinology of human female sexuality, mating, and reproductive behavior," *Hormones and Behavior 91*: May 2017, DOI: 10.1016/j.yhbeh.2016.11.012, p. 25.

that evolution by natural and sexual selection seemingly provides for organizational/activational theory's inherent aspects. For example, one paper, about endocrine disrupting chemicals as pollutants, argued:

> *"**The organizational effects of steroids set the stage for numerous neurobiological processes, some of which are not manifested until childhood, adolescence, or adulthood.** Our understanding of the importance of hormones in brain sexual differentiation is nearly a century old, with evidence that this is established in the embryo through early postnatal life (Phoenix et al., 1959,[466] Barraclough and Gorski, 1961,[467] Petrusz and Flerko, 1965,[468] Morris et al., 2004,[469] Kudwa et al., 2006,[470] Sodersten et al., 2014[471]). While most of the prior evidence was accumulated from rodent work, **the human brain is also structurally and functionally sexually dimorphic** (Ehrhardt and Meyer-Bahlburg, 1981,[472] Wizemann and Pardue, 2001,[473] Cahill, 2006[474]). **Sex differences in reproductive behavior and strategies (e.g. parental behavior, pair***

[466] Phoenix *et al.*, "Organizing Action of Prenatally Administered Testosterone Propionate."
[467] Charles A. Barraclough and Roger A. Gorski, "EVIDENCE THAT THE HYPOTHALAMUS IS RESPONSIBLE FOR ANDROGEN-INDUCED STERILITY IN THE FEMALE RAT," *Endocrinology 68*(1): 1 January 1961, DOI: 10.1210/endo-68-1-68, p. 68-79.
[468] B. Flerkó, P. Petrusz, L. Tima, "On the mechanism of sexual differentiation of the hypothalamus. Factors influencing the 'critical period' of the rat," *Acta Biologica Academiae Scientiarium Hungaricae 18*(1): 1 January 1967, PMID: 5892876, p. 27-36.
[469] John A. Morris, Cynthia L. Jordan, and S. Marc Breedlove, "Sexual differentiation of the vertebrate nervous system," *Nature Neuroscience 7*: 2004, DOI: 10.1038/nn1325, p. 1034-1039.
[470] A. E. Kudwa, V. Michopoulos, J. D. Gatewood, E. F. Rissman, "Roles of estrogen receptors α and β in differentiation of mouse sexual behavior," *Neuroscience 138*(3): 27 March 2006, DOI: 10.1016/j.neuroscience.2005.10.018, p. 921-928.
[471] Per Södersten, David Crews, Cheryl Logan, and Rudolf Werner Soukup, "Eugen Steinach: The First Neuroendocrinologist," *Endocrinology 155*(3): 1 March 2014, DOI: 10.1210/en.2013-1816, p. 688-695.
[472] Anke A. Ehrhardt and Heino F. L. Meyer-Bahlburg, "Effects of Prenatal Sex Hormones on Gender-Related Behavior," *Science 211*(4488): 20 March 1981, DOI: 10.1126/science.7209510, p. 1312-1318.
[473] US Institute of Medicine's Committee on Understanding the Biology of Sex and Gender Differences, *Exploring the Biological Contributions to Human Health: Does Sex Matter*, eds. Theresa M. Wizemann and Mary-Lou Pardue, National Academy Press (Washington, D.C.): 2001.
[474] Larry Cahill, "Why sex matters for neuroscience," *Nature Reviews Neuroscience 7*: 2006, DOI: 10.1038/nrn1909, p. 477-484.

> ***bonding, etc.) are necessary for the perpetuation of the species, as they increase the likelihood of successful reproduction and offspring survival. Therefore, the process of sexual differentiation of the brain is critical for species survival*** *(Dulac and Kimchi, 2007[475]). Even subtle changes in brain organization and activation from disruption by EDCs [endocrine disrupting chemicals] can have profound effects on reproductive strategies, physiology, and behaviors, with potentially devastating effects on a population living in a contaminated area (Crews and Gore, 2011,[476] Crews and Gore, 2012[477]). [emphasis added]"*[478]

The bolded sentences together connect the inherence of sex-typed attractions and behavior with evolutionary adaptations that species, including humans, require for their survival. If these behaviors are (or were) necessary for human survival, that necessity would explain organizational/activational theory's rigidity (beginning in almost all people during early

[475] Catherine Dulac and Tali Kimchi, "Neural mechanisms underlying sex-specific behaviors in vertebrates," *Current Opinion in Neurobiology 17*(6): December 2007, DOI: 10.1016/j.conb.2008.01.009, p. 675-683.
[476] David Crews and Andrea C. Gore, "Life Imprints: Living in a Contaminated World," *Environmental Health Perspectives 119*(9): 13 May 2011, DOI: 10.1289/ehp.1103451, p. 1208-1210.
[477] David Crews and Andrea C. Gore, "Epigenetic synthesis: a need for a new paradigm for evolution in a contaminated world," *F1000 Biological Reports 4*(18): 2012, DOI: 10.3410/B4-18, p. 1-6.
[478] Deena M. Walker and Andrea C. Gore, "Epigenetic impacts of endocrine disruptors in the brain," p. 4. Another paper, Andrea C. Gore, Amanda M. Holley, and David Crews, "Mate choice, sexual selection, and endocrine-disrupting chemicals," *Hormones and Behavior 101*: May 2018, DOI: 10.1016/j.yhbeh.2017.09.001, p. 3-12, similarly argued that "Virtually all aspects of reproduction rely upon the complementarity of male and female anatomy and behavior…. This theme of reciprocity also exists within an individual, namely, the masculine-feminine dichotomy and the capacity to manifest the behavior of the "opposite" sex depending upon the hormonal and social context (Crews, 2010)." (p. 5-6), that "Any disruption of sexual cues through shifts in the trajectory of sexual characteristics could change the perception of the quality of a potential mate. This is devastating for an individual, and if it happens within a large number of individuals within a population could have dire consequences, including population declines or even extinction." (p. 5), and finally that "EDCs during sensitive periods can alter the timing of underlying patterns of gene expression leading to characteristics that are inconsistent with the genetic or gonadal sex. Such 'transgender' individuals present confusing cues to potential mates. … Thus, EDC-altered females, if they were receptive to courtship of uncontaminated males, would have a significant impact on the population — unless males were able to detect and preferentially breed with unaffected females." (p. 9-10) While this paper used a more environmental conservation-focused perspective, it uses human terminology (incorrectly, the animals would probably be more properly described as intersex) currently associated with a moral panic related to beliefs about the "male-female dichotomy" and relies on disgust-based language like "uncontaminated" and "unaffected" as well as fear-based language like "extinction".

development and leading inexorably to sex-typed behaviors) and universality (occurring in all people at all times).

These are assumptions, it is worth reemphasizing, that the theories involved do not necessarily imply and that evidence does not necessarily support. Organizational/activational theory as applied to humans, and often as applied across mammals if not a wider slice of the animal kingdom, was only compatible with a highly selected sex difference consistent across many species with very little internal variability and across nearly all humans' neural development and structure. Evidence that humans did evolve in this highly selected and adaptationist way, and further that the traits were universally selected in the ways hypothesized above, is unlikely to appear. Organizational/activational theory is fundamentally inflexible. If hominids did not universally sex-type their work, if there were advantages to cognitive flexibility rather than rigidity, if genetic drift significantly influenced the traits present in the modern human population, if early hominid societies provided different pressures on early hominid evolution, or if the rapid neural expansion of the primates and hominids in some way disrupted the sex-typed behaviors of earlier ancestors, among other possibilities, the rigidity of organizational/activational theory's sex-typed behaviors and attributes becomes significantly more difficult to defend.

Evolutionary theory generally assumes population variability, increased by mutation and recombination[479] and decreased by selection, but generally present. Organizational/activational theory assumes population invariability—even transcendent invariability across multiple species. The organizational/activational theory requires a specific vision of humans' evolutionary past,

[479] The process when making eggs/sperm of "mixing around" the genes that person got from their parents, so that traits are (generally) unlinked and their future child may possess any combination of the heritable traits available.

one that requires a narrow interpretation of evolutionary theory that is, additionally, contradicted by multiple pieces of research. In practice, there are not two types of brain in humans.[480] Human evolutionary history is under significant dispute, but evidence from foraging societies[481] and archeological evidence[482] suggests that the sexual division of labor might have emerged after or with settled agriculture—i.e. around ten to fifteen thousand years ago.[483] More problematically, organizational/activational theory is really only compatible with narrow variability among humans and hominids maintained by relentless selective pressure to end up with one—and only one—way for sexes/genders to be. Could those working within organizational/activational theory have taken into account the variability of life, the variations within species, the fluidity of gender/sex that a population-forward (rather than type-forward) view of evolutionary theory could imply? Those researchers could have taken such a view, but at least some of organizational/activational theory likely would have shattered on impact. So, much like the other contexts discussed in this chapter, evolutionary theory and evolutionary evidence was used shallowly and selectively, to the degree it is accurate to call these assertions "evolutionary theory" at all.[484] Evolution was assumed to be acting for the reinforcement of organization and activation, despite the mismatch between evolutionary theory as used by evolutionary biologists and as used by behavioral neuroendocrinology during this period.

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[480] Joel *et al.* "Sex beyond the genitalia: The human brain mosaic."
[481] Anderson *et al.*, "The Myth of Man the Hunter," and Madeleine J. Goodman, P. Bion Griffin, Agnes A. Estioko-Griffin, and John S. Grove, "The Compatibility of Hunting and Mothering among the Agta Hunter-Gatherers of the Philippines," *Sex Roles 12*: 1985, DOI: 10.1007/BF00287829, p. 1199-1209.
[482] This paper can determine that 90% of the Neanderthals were likely tanning hides right-handedly; gender did not appear to play any meaningful role in who was tanning hides. Carles Laleuza Fox and David W. Frayer, "Non-dietary Marks in the Anterior Dentition of the Krapina Neanderthals," *International Journal of Osteoarchaeology 7*(2): March 1997, DOI: 10.1002/(SICI)1099-1212(199703)7:2<133::AID-OA326>3.0.CO;2-4, p. 133-149.
[483] Alba Masclans, Caroline Hamon, Christian Jeunesse, and Penny Bickle, "A sexual division of labour at the start of agriculture? A multi-proxy comparison through grave good stone tool technological and use-wear analysis," *PLOS One 16*(4): 2021, DOI: 10.1371/journal.pone.0249130.
[484] See the paragraph about universality being a possible misconception above.

4.2: Assumed Sociology—The Lived Consequences of History

> *"In a nutshell, this is the take-home message: Ignoring other possibilities or interpretations may be the main source of fallacies."*[485]

As discussed in the last section, organizational/activational theory relied on assumptions about how mammals broadly and humans specifically had evolved. To return to one of the quotes above, however, it is worth noting that the discussion of women's sexual behavior across the menstrual cycle moved seamlessly from an evolutionary assertion about an observation to evidence of modern behavior.[486] That seamlessness, generally shared across behavioral neuroendocrinology, is in itself an argument about the past. What behaviors, preferences, and abilities in modern humans are continuous with humans' unrecorded evolutionary past? As little evidence about that past is particularly solid, to assume that some characteristic of modern humans is continuous with that past is also to assume that the origin of that characteristic lies largely within individuals and not meaningfully due to or affected by social structures or recent history. Through this assumption, behavioral neuroendocrinology as a field participated in many sociological arguments, but generally without meaningfully engaging with processes or information gathered by sociologists, anthropologists, archeologists, social historians, or any other types of social sciences.

Another way of stating a central question is this: is a biological framework the most useful framework to explain the behavior in question, or might other frameworks—historical, sociological, psychological, economic, political—provide a better lens? Additionally, even if biological frameworks are in effect, how much do they affect people's behavior compared to

[485] Christophe Bernard, "On Fallacies in Neuroscience," *eNeuro 7*(6): 10 December 2020, DOI: 10.1523/ENEURO.0491-20.2020, p. 1.
[486] Motta-Mena and Puts, "Endocrinology of human female sexuality," p. 25; see ft. 22 above.

those other aspects? Is it a marginal effect or a centrally important one? A direct effect or an indirect effect? For example, evolutionary frameworks, a type of biological framework, consider genetic spread through a population by selection or chance survival of individuals, but do not (at least, not without modification) consider why an individual's genetic success in a larger society may not be entirely within their control for reasons other than being outcompeted. Many societies practice or practiced arranged marriages, in which one partner, most commonly the woman, often has limited agency. Genes may not be as locally important as less evolutionarily selected aspects like money, prestige, or power that can also be inherited. More to the point, however, the economic and social networks of families that are linked through an arranged marriage would give the future children an advantage that was inherited but not evolutionary or biological.[487] Free choices freely chosen on the basis of mutual desirability may not always be an accurate assumption, particularly when discussing mating, partner choice, and the social structures governing and affecting both in humans.

On a larger level of magnification, the literature shows a general unwillingness to question why cross-cultural similarities exist. As obvious sexed/gendered universals would provide evidence that organizational factors worked across humanity, that unwillingness is perhaps unsurprising but contrary to evidence all the same. Consider, as an example, why the nuclear model of the family in which the father produces and the mother reproduces is so apparently ubiquitous.[488] The belief that nuclear, heterosexual families are universal because

[487] While occasionally individuals will put forward the possibility that social or economic achievement represents genetic superiority in some fashion, there is only disconfirming evidence for this proposition. Histories of kings and aristocrats and magnates show people, no better or worse than the members of other classes of their day or of this day. There are many possible explanations for worldly success, but the genetic similarity of all people means that genes are a poor explanation.

[488] Motta-Mena and Puts, "Endocrinology of human female sexuality," contains several particularly egregious examples. Additionally, see the quote cited in ft. 10.

they are natural or freely chosen is flatly ahistorical. Nuclear heterosexual families were not freely chosen but colonially imposed—on the settlers[489] and, far more violently and harshly, on the colonized. Colonial populations in the Americas,[490] Africa,[491] Asia,[492] and Polynesia[493] were all dehumanized and subjugated through the lens of their real or imagined differences from an ideal of white heteronormative nuclear families. Sociologist Haley McEwen, for example, argues that "nuclear family supremacy functioned as a form of cultural imperialism, which attempted to destroy indigenous societies, beliefs, and knowledge systems through the reeducation of

[489] For example, the 'weakness' of white male homosexuality and the necessity of subjugating white females to the home in the cult of domesticity. Daniel J. Walther, "Racializing Sex: Same-Sex Relations, German Colonial Authority, and Deutschtum," *Journal of the History of Sexuality 17*, no. 1: January 2008, URL: jstor.org/stable/30114367, p. 11-24; Adele Perry, "'Fair Ones of a Purer Caste': White Women and Colonialism in Nineteenth-Century British Columbia," *Feminist Studies 23*, no. 3: Autumn 1997, DOI: 10.2307/3178383, p. 501-524.

[490] Maile Arvin, Eve Tuck, and Angie Morrill, "Decolonizing Feminism: Challenging Connections between Settler Colonialism and Heteropatriarchy." *Feminist Formations 25*, no. 1: Spring 2013, URL: jstor.org/stable/43860665, p. 8-34; Richard Phillips, "Settler colonialism and the nuclear family," *Canadian Geographies/Géographies canadiennes 53*(2): 1 June 2009, DOI: 10.1111/j.1541-0064.2009.00256.x, p. 239-253; Sandy O'Sullivan, "The Colonial Project of Gender (and Everything Else)," *Genealogy 5*(3), ID: 67: 2021, DOI: 10.3390/genealogy5030067.

[491] Niara Sudarkasa, "'The Status of Women' in Indigenous African Societies," *Feminist Studies 12*, no. 1: Spring 1986, DOI: 10.2307/3177985, p. 91-103; Rebecca Hodes, "Kink and the Colony: Sexual Deviance in the Medical History of South Africa, c. 1893–1939." *Journal of Southern African Studies 41*(4): 2015, DOI: 10.1080/03057070.2015.1049486, p. 715-733; Sylvia Tamale, "Confronting the Politics of Nonconforming Sexualities in Africa," *African Studies Review 56*(2): 2013, DOI: 10.1017/asr.2013.40, p. 31-45; Eileen Jensen Krige, "Woman-Marriage, with Special Reference to the Lovedu. Its Significance for the Definition of Marriage," *Africa: Journal of the International African Institute 44*, no. 1: January 1974, DOI: 10.2307/1158564, p. 11-37; Bharat Mehra, Paul A. Lemieux, and Keri Stophel, "An Exploratory Journey of Cultural Visual Literacy of 'Non-Conforming' Gender Representations from Pre-Colonial Sub-Saharan Africa," *Open Information Science 3*: 2019, DOI: 10.1515/opis-2019-0001, p. 1-21; Haley McEwen, "Inventing Family: Colonial Knowledge Politics of 'Family' and the Coloniality of 'Pro-family' Activism in Africa," *Africa Today 67*, no. 4: Summer 2021, DOI: 10.2979/africatoday.67.4.03, p. 31-49.

[492] J. Y. Chua, "The Strange Career of Gross Indecency: Race, Sex, and Law in Colonial Singapore," *Law and History Review 38*(4): 2020, DOI: 10.1017/S073824801900052X, p. 699-735; Rovel Sequeira, "Don't Ask, Won't Tell? Sexual Science and the Case Biography of Sodomy in Colonial India," *Modernism/modernity 29*, no. 1: January 2022, DOI: 10.1353/mod.2021.0075, p. 145-168; Philip Howell, "Race, space, and the regulation of prostitution in colonial Hong Kong," *Urban History 31*, no. 2: 2004, DOI: 10.1017/S0963926804002123, p. 229-248; Maryna Romanets, "'Orients' of the Mind: Deviance, Sexual Enlightenment, and True Love in Fredericks's *Degenerate Empress*, Vynnychuk's *Zhytiie haremnoie* (*Life in the Harem*), and Parker's *Roxelana & Suleyman*," *Canadian Review of Comparative Literature/Revue Canadienne de Littérature Comparée 44*(1): March 2017, DOI: 10.1353/crc.2017.0006, p. 95-110.

[493] Margaret Mishra, "'Your Woman is a Very Bad Woman': Revisiting Female Deviance in Colonial Fiji," *Journal of International Women's Studies 17*(4), Article 5: 2016, URL: vc.bridgew.edu/jiws/vol17/iss4/5, p. 67-78; Jenny Coleman, "The 'Inferior' Sex in the Dominant Race: Feminist Subversions or Imperial Apologies?" *Feminist Review 102*(1): 1 November 2012, DOI: 10.1057/fr.2012.13, p. 62-78.

indigenous people into Western gender roles and gendered divisions of labor within public and private spaces."[494] Additionally, as anthropogeographer Richard Phillips noted, "There has… been a tendency to mistake this hegemonic complex of power relations for a natural and inevitable social arrangement, and equally to underestimate the importance of other socio-sexual formations."[495] To claim, even implicitly, that the nuclear heterosexual family is natural is to deny the historical events which violently enforced that model of sexual and social morality for hundreds of years and thereby to base one's theories on a false foundation. If a single natural or innate human family structure exists, any claims about it must grapple with the fundamental evidential issues caused by the intentional suppression and destruction of so many societies and individuals by colonial-capitalist projects, which this literature does not seem to recognize it could or should attempt.

An alternative manifestation of this sort of selective blindness, in which internal factors are focused on to the complete or functional exclusion of external factors, is the extrapolation of some hormonal or organizational factors to cover all possible aspects. There is a definite link between hormonal changes during puberty, menopause, childbirth, and menstrual cycles and some types of mood disorders.[496] However, as one paper about anxiety and adolescence noted, "Although pathological anxiety tends to onset during adolescence, this trend does not occur equally across both sexes. Indeed, a wealth of evidence indicates that rates of anxiety are approximately equal pre-adolescence, become significantly higher in females during

[494] McEwen, "Inventing Family: Colonial Knowledge Politics of 'Family'," p. 32.
[495] Phillips, "Settler colonialism and the nuclear family," p. 240.
[496] Premenstrual dysphoric disorder, postpartum depression and psychosis, mood swings and hormonal crashes during puberty and menopause.

adolescence, and remain so into adulthood (Altemus et al., 2014[497]).”[498] Mood disorders (i.e., anxiety and depression) are significantly higher in women overall,[499] and earlier[500] puberty in girls is associated with worse mental health outcomes and higher likelihood of conflict with authorities.[501] From these pieces of information, and while generally ignoring that late puberty in boys was also associated with long-term negative mental health outcomes,[502] multiple reviews and papers suggested that organizational and activational estrogen “cause girls to be more vulnerable than boys”[503] or that “[t]estosterone may act during development and/or adulthood to dampen stress responsiveness.”[504] Another paper suggested that hormonal factors might explain the larger number of eating disorders during and after puberty in girls and women.[505]

Sari van Anders, one of the only exceptions to this general rule, noted that “frequently, phenomena particular to humans is ironically ignored in the name of ‘biologism’ (an ideological commitment to ignoring non-biological phenomena).”[506] The paper discussing puberty and eating disorders offers a clue about how researchers can near but not quite engage with relevant societal information when it notes that “[t]raditional theories of risk focused on the psychosocial effects (e.g., increased body dissatisfaction) of the physical changes associated with puberty (e.g., increased adiposity) and their potential consequences for eating disorder development

[497] Margaret Altemus, Nilofar Sarvaiya, C. Neill Epperson, “Sex differences in anxiety and depression clinical perspectives,” *Frontiers in Neuroscience 35*(3): August 2014, DOI: 10.1016/j.yfrne.2014.05.004, p. 320-330.
[498] Spielberg *et al.,* “Anxiety in transition: Neuroendocrine mechanisms,” p. 3-4.
[499] McHenry *et al*., “Sex differences in anxiety and depression,” p. 42.
[500] Not ‘precocious’ puberty, or puberty beginning before about age 8; most children begin puberty between ages 8 and 14.
[501] Julia A. Graber, “Pubertal timing and the development of psychopathology in adolescence and beyond,” *Hormones and Behavior 64*(2): July 2013, DOI: 10.1016/j.yhbeh.2013.04.003, p. 262-269.
[502] Ibid.
[503] Cooke and Weathington, “Human and animal research into sex-specific effects of child abuse,” p. 417.
[504] McHenry *et al*., “Sex differences in anxiety and depression,” p. 52.
[505] Kelly L. Klump, “Puberty as a critical risk period for eating disorders: A review of human and animal studies,” *Hormones and Behavior 64*(2): July 2013, DOI: 10.1016/j.yhbeh.2013.02.019, p. 399-410.
[506] Sari M. van Anders, “Beyond masculinity: Testosterone, gender/sex, and human social behavior in a comparative context,” *Frontiers in Neuroendocrinology 34*(3): August 2013, DOI: 10.1016/j.yfrne.2013.07.001, p. 207.

(Fornari & Dancyger, 2003[507]).”[508] However, the blended experience of puberty/adolescence is not merely a set of physical or mental changes which may interact with society—society affects the experience of undergoing physical and mental changes. Indeed, the researchers quoted above about anxiety and adolescence displayed an awareness of this dynamic, but only for the elevated rates of anxiety displayed by women with polycystic ovary syndrome (PCOS), which “remain confounded by the physical consequences of PCOS (e.g., excess hair growth) that could reasonably lead to increases in anxiety in these patients.”[509]

As Niva Piran notes, girls beginning to menstruate “internalize the stigma and comply with the social etiquette of not making the period a material presence in the social environment…, a disempowering process, [and] they experience… *embodied demotion* by the lack of social support, accommodation, or attunement to their needs in managing menstruation[italics in original]”[510] in addition to the stress of being “entrusted with the responsibility of preventing pregnancy without the social conditions of safety”.[511] Appearing to others as (more like) a woman can also cause “metadehumanization”—the experience of being aware that one is being objectified and stereotyped, with corresponding dehumanization in turn of the group stereotyping them.[512] The power dynamics associated with patriarchal structures, in

[507] Victor Fornari and Ida F. Dancyger, “Psychosexual development and eating disorders,” *Adolescent Medicine 14*(1): February 2003, p. 61-75.
[508] Klump, “Puberty as a critical risk period for eating disorders,” p. 400.
[509] Spielberg *et al.*, “Anxiety in transition: Neuroendocrine mechanisms,” p. 7.
[510] Niva Piran, “The Menarche Journey: Embodied Connections and Disconnections,” in *The Palgrave Handbook of Critical Menstruation Studies,* eds. Chris Bobel, Inga T. Winkler, Breanne Fahs, Katie Ann Hasson, Elizabeth Arveda Kissling, Tomi-Ann Roberts. Palgrave-Macmillan (Singapore): 2020, DOI: 10.1007/978-981-15-0614-7_18, p. 209-210.
[511] *Ibid.*, p. 205.
[512] Tina Chevallereau, Florence Stinglhamber, Pierre Maurage, and Stéphanie Demoulin, “My Physical Appearance at the Center of Others’ Concerns: What are the Consequences for Women’s Metadehumanization and Emotions?” *Psychologica Belgica 61*(1): 2021, DOI: 10.5334/pb.558, p. 116-130; Nour Kteily, Gordon Hodson, Emile Bruneau, “They See Us as Less Than Human: Metadehumanization Predicts Intergroup Conflict via Reciprocal Dehumanization,” *Journal of Personality and Social Psychology 110*, no. 3: March 2016, DOI: 10.1037/pspa0000044, p. 343-370.

turn, explain why those boys late to "become men", or those who have hypogonadal conditions,[513] may develop mental health issues for reasons beyond their relative dearth of testosterone—as homosexual or effeminate men can attest, being 'imperfectly' male can result in an experience of embodied demotion.[514] Finally, despite a conference attempting to define why women were more susceptible to stress,[515] a 2016 paper in *Hormones and Behavior* noted that, based on available evidence, women and ethnic minorities were not more vulnerable to discrimination but more discriminated against.[516]

Beyond van Anders, who proposed a non-masculinity-based theory of testosterone levels and changes (and used the term "gender/sex"), there were two striking exceptions to this collective blindness. The first was Dr. Bruce McEwen, who prefaced his review about stress with a note about his fruitful collaboration with his "brother, Dr. Craig McEwen, a Sociologist at Bowdoin College."[517] The second was a group of clinicians working in Switzerland concerned about the—likely observed—poorer outcomes for women after suffering an ischemic stroke and unwilling to blame sex hormones for women "arriv[ing] later at the emergency department".[518] These exceptions prove the rule and suggest that without external pressures (familial or work-based) or a willingness to incorporate external ideas (as appears to be true of van Anders), the

[513] McHenry *et al.*, "Sex differences in anxiety and depression," p. 42-43.
[514] For example: "Though homosexual men (and androphilic male-to-female transsexuals) tend to show male-typical category specificity, on average they have a variety of feminine traits, including occupational and recreational interests, patterns of movement and speech, and a preference for male sex partners (Bailey, 2003)." David Sylva, Adam Safron, A. M. Rosenthal, Paul J. Reber, Todd B. Parrish, J. Michael Bailey, "Neural correlates of sexual arousal in heterosexual and homosexual women and men." *Hormones and Behavior 64*(4): September 2013, DOI: 10.1016/j.yhbeh.2013.08.003, p. 675. The reference (Bailey, 2003) is to the heavily criticized book *The Man Who Would be Queen: The Science of Gender-bending and Transsexualism* written by the last author of the paper.
[515] Jill B. Becker, Lisa M. Monteggia, Tara S. Perrot-Sinal, Russell D. Romeo, Jane R. Taylor, Rachel Yehuda, Tracy L. Bale, "Stress and Disease: Is Being Female a Predisposing Factor?" *Journal of Neuroscience 27*(44): 31 October 2007, DOI: 10.1523/JNEUROSCI.3565-07.2007, p. 11851-11855.
[516] Huynh *et al.*, "Everyday discrimination and diurnal cortisol during adolescence."
[517] McEwen, "Redefining neuroendocrinology: Epigenetics of brain-body communication over the life course," p. 8.
[518] Liberale *et al.,* "Ischemic stroke across sexes: What is the status quo?", p. 10.

underlying trend was to locate causes within the individual. This occured to the point that a review of the long-term effects of child abuse primarily focused on women's greater vulnerability to depression.[519]

Sex is a relatively easy variable to collect—most cultures label children and adults by sex using clothing, behavior, government documents, etc.—and is categorical. "Sex" is, in fact, a suspiciously easy variable, as almost everyone who has ever lived can be neatly slotted into one of two categories. Returning to the point borrowed from Weber earlier in this chapter (§4.1), the more universal a law, the emptier its concepts. Humans differ in brains and behavior, almost all humans can be described as possessing one sex or the other, and some variables can be empirically associated more with one sex or the other. Deciding that sex is, however, a useful or meaningful way of categorizing people by how they behaviorally and neurologically differ (and what those uses and meanings are) would require some further evidence, and that evidence would require engaging with these larger historical and sociological realities. Instead, the trend in the research was to ignore the larger structures individuals live within to locate the cause within those displaying the effect.

This problem is not a new one. In 1990, the *New England Journal of Medicine* published the paper "Racial Differences in Susceptibility to Infection by *Mycobacterium tuberculosis*".[520] The association between African-Americans, or Africans generally, and tuberculosis infection is at least as old as knowledge of the tuberculosis bacillus. Quite tellingly, the connections in the United States between tuberculosis and the white urban poor, particularly Catholics and Southern

[519] Cooke and Weathington, "Human and animal research into sex-specific effects of child abuse."
[520] William W. Stead, John W. Senner, William T. Reddick, and John P. Lofgren, "Racial Differences in Susceptibility to Infection by *Mycobacterium tuberculosis*," *New England Journal of Medicine 322*: 15 February 1990, DOI: 10.1056/NEJM199002153220702, p. 422-427.

and Eastern European immigrants, have largely been left in the early decades of the twentieth century as these populations became racially "white".[521] A similar belief about greater susceptibility to tuberculosis and Native Americans had proven to be completely explanatorily useless as early as the 1930s and definitively by the 1950s: "Indians were not a race apart but a marginalized group afflicted with a disease of poverty."[522] The 1990 paper found that in racially integrated nursing homes and prisons, black individuals are twice as likely to become infected as white individuals; this difference was put down to a heritable vulnerability that is greater in black populations than white populations.[523]

Race, however, is neither precisely a genetic, societal, economic, or spatial variable, instead being a cluster of these properties. As a 2001 commentary in the *American Journal of*

[521] Claudia Maria Calhoon, "Tuberculosis, Race, and the Delivery of Health Care in Harlem, 1922-1939," *Radical History Review* (80): Spring 2001, URL: muse.jhu.edu/article/30173, p. 104.
[522] Christian W. McMillen, "'The Red Man and the White Plague': Rethinking Race, Tuberculosis, and American Indians, ca. 1890-1950," *Bulletin of the History of Medicine 82*, no. 3: Fall 2008, URL: jstor.org/stable/44448614, p. 639.
[523] While an exact discussion of 'real' causes is impossible to do (particularly as an external investigation of nursing home and prison conditions 35 years ago is difficult to say the least), if black neighborhoods had higher rates of tuberculosis than white neighborhoods as the authors state (saying that tuberculosis infection rates at entry were also higher among black people than white people), presumably black people in nursing homes and prisons would frequently receive visitors and objects from black neighborhoods, possibly explaining their higher rates of infection. Also, black individuals in an integrated space, even if it were perfectly integrated, would presumably have lived being black—with black family members, in officially or unofficially segregated neighborhoods, etc.—their whole lives up to that point. The effects of socioeconomic status, undiagnosed asthma or other pulmonary diseases, or environmentally poorer conditions (e.g. poor neighborhoods being near interstate highways, industrial locations, large farms with pesticide spraying, etc.) may be more relevant than genetics. Finally, the assumption that the black and white people in prisons and nursing homes are being watched equally closely for symptoms or treated with equal care is perhaps an unrealistically rosy belief about nursing home and prison staff in the United States, mostly from Arkansas, in the 1980s. In particular, Arkansas passed a state law in 1999 that required local coroners to examine all nursing home deaths specifically after a county coroner found six deaths via negligence in nursing homes which had not been reported, (Chisun Lee and A. C. Thompson, "Gone Without a Case: Suspicious Elder Deaths Rarely Investigated," *ProPublica*: 20 Dec 2011, URL: propublica.org/article/gone-without-a-case-suspicious-elder-deaths-rarely-investigated, accessed 10 January 2024) and Arkansas, in 1994, was also the last state to stop selling prisoners' blood plasma, a deeply corrupt scheme that cross-infected prisoners (due in part to bribery of officials and a lack of care on the part of medical staff) and infected at least 42,000 Canadians with hepatitis C and HIV from the improperly screened blood (Suzi Parker, "Blood Money: An Arkansas prison-plasma business protected by Clinton cronies led to a scandal that almost toppled the government -- of Canada," *Salon.com*: 24 December 1998, URL: salon.com/1998/12/24/cov_23news/, accessed 10 January 2024). Suffice it to say that data from Arkansas nursing homes and prisons in the 1980s and 1990s has some localized problems which undermine the authors', or indeed any, interpretation.

Epidemiology noted, "a person's race/ethnicity is fixed prior to his/her measured social, physiologic, and psychological status; all of these measurable factors are downstream of the exposure in a racially stratified society,"[524] recommending that researchers focus on the "more specific measures… for which racial/ethnic status is acting as a rough surrogate."[525] The general failing in both cases is that sex/gender and race/ethnicity *as assessed by others* affect how people are treated in the world for the entirety of those people's lives through multiple mediators and in overlapping ways.

By failing to account for or actively avoiding the information about the material differences in the lives of boys and girls or women and men and then theorizing about the biological origins of different behaviors, these researchers are assuming that these lived and experienced differences are nonexistent or unimportant to explain why the behaviors are different between groups. Building on that assumption, a further assumption is that these differing biological mechanisms should be co-opted in therapies, by "individualizing" medicine by sex, by utilizing sex as a biological variable in research, or, more drastically, treating sexual assault trauma by stimulating neurogenesis in "[f]emales" using the "fortunate… opportunity to learn one set of new and effortful behaviors in their lifetime – those related to caring for offspring during motherhood."[526] These therapies, however, cannot address systemic or social causes of mental health problems, and these frameworks cannot acknowledge the lives or societies that people come from and go back to—no one, after all, is only their sex or gender. By consistently failing to include the nuances and variety of human experiences or the historical

[524] Jay S. Kaufman and Richard S. Cooper, "Commentary: Considerations for Use of Racial/Ethnic Classification in Etiologic Research," *American Journal of Epidemiology 154*(4): 15 August 2001, DOI: 10.1093/aje/154.4.291, p. 293.
[525] Ibid., p. 297.
[526] Tracey J. Shors and Emma M. Millon, "Sexual trauma and the female brain," *Frontiers in Neuroendocrinology 41*: April 2016, DOI: 10.1016/j.yfrne.2016.04.001, p. 91.

origins of existing social structures, behavioral neuroendocrinologists working within the organizational and activational framework assumed one particular interpretation of the world without evidence or justification; a version of the world, furthermore, that has never been true.

4.3: Assumed Generalizability—Women Thinking Scientifically

> *"The point that troubles Wittgenstein is manifestly not that Pascal has made a mistake in her description of how she feels. Nor is it even that she has made a careless mistake. Her laxity, or her lack of care, is not a matter of having permitted an error to slip into her speech on account of some inadvertent or momentarily negligent lapse in the attention she was devoting to getting things right. The point is rather that, so far as Wittgenstein can see, Pascal offers a description of a certain state of affairs without genuinely submitting to the constraints which the endeavor to provide an accurate representation of reality imposes."*[527]

One of the most common themes in the behavioral neuroendocrinology literature was the difference between men and women's spatial and linguistic abilities. Explanations, mechanisms, and associated traits varied: hemispheric dominance,[528] man-as-hunter and woman-as-social,[529]

[527] Harry G. Frankfurt, *On Bullshit*, Princeton University Press (Princeton): 2005, p. 31-32.
[528] Kathleen Wermke, Anja Quast, and Volker Hesse, "From melody to words: The role of sex hormones in early language development," *Hormones and Behavior 104*: August 2018, DOI: 10.1016/j.yhbeh.2018.03.008, p. 206-207.
[529] Plessen *et al.*, "Sex, Age, and Cognitive Correlates of Asymmetries in Thickness of the Cortical Mantle Across the Life Span," p. 6300; David C. Geary, "Sex differences in social behavior and cognition: Utility of sexual selection for hypothesis generation," *Hormones and Behavior 49*(3): March 2006, DOI: 10.1016/j.yhbeh.2005.07.014, p. 273-275; Rebecca Knickmeyer, Simon Baron-Cohen, Peter Raggatt, Kevin Taylor, and Gerald Hackett, "Fetal testosterone and empathy," *Hormones and Behavior 49*(3): March 2006, DOI: 10.1016/j.yhbeh.2005.08.010, p. 282-292.

prenatal androgens,[530] aggression and sensation-seeking (for males),[531] homosexuality improving one's skill in the other sex's 'domain',[532] Y chromosome effects,[533] oral contraceptive use,[534] 2D:4D ratios,[535] autism-as-male and depression-as-female (see §4.4), and so on. With only very few exceptions, men and male model animals, generally rats, were observed to have higher spatial reasoning abilities; women, however, had greater verbal and social intelligence. Results generally fit within this framework or merely failed to confirm it; only one paper, in 2000, suggested that women had greater mathematical or spatial memory.[536]

Behavioral neuroendocrinologists collectively believed that there were "biologically caused differences between the… 'feminine' and 'masculine' cognitive sets,"[537] and, furthermore, that at least some of the preferences for social stimuli for infant girls and mechanical stimuli for infant boys were biologically based.[538] "Men outperform women on many

[530] Aitziber Azurmendi, Francisco Braza, Aizpea Sorozabal, Ainhoa García, Paloma Braza, María R. Carreras, José M. Muñoz, Jaione Cardas, and José R. Sánchez-Martín, "Cognitive abilities, androgen levels, and body mass index in 5-year-old children," *Hormones and Behavior 48*(2): August 2005, DOI: 10.1016/j.yhbeh.2005.03.003, p. 187.
[531] Peter A. Bos, Jaak Panksepp, Rose-Marie Bluthé, and Jack van Honk, "Acute effects of steroid hormones and neuropeptides on human social–emotional behavior: A review of single administration studies," *Frontiers in Neuroendocrinology 33*(1): January 2012, DOI: 10.1016/j.yfrne.2011.01.002, p. 17-35; this association was related to the man-as-hunter and women-as-social evolutionary arguments.
[532] van Anders and Hampson, "Testing the prenatal androgen hypothesis," p. 92-98; see also, Cooke and Weathington, "Human and animal research into sex-specific effects of child abuse," p. 420, where the authors suggest that "the capacity to empathize may vary in accordance with sexual orientation, not gender," arguing that straight women and gay men are more empathetic and straight men and lesbian women are less empathetic.
[533] Victoria N. Luine, "Estradiol and cognitive function: Past, present and future," *Hormones and Behavior 66*(4): September 2014, DOI: 10.1016/j.yhbeh.2014.08.011, p. 604.
[534] Ramune Griksiene, Rasa Monciunskaite, Aurina Arnatkeviciute, Osvaldas Ruksenas, "Does the use of hormonal contraceptives affect the mental rotation performance?" *Hormones and Behavior 100*: April 2018, DOI: 10.1016/j.yhbeh.2018.03.004, p. 29-38.
[535] van Anders and Hampson, "Testing the prenatal androgen hypothesis."
[536] Sarah J. Duff, and Elizabeth Hampson, "A Beneficial Effect of Estrogen on Working Memory in Postmenopausal Women Taking Hormone Replacement Therapy," *Hormones and Behavior 38*(4): December 2000, DOI: 10.1006/hbeh.2000.1625, p. 262-276.
[537] Pearson and Lewis, "Fear recognition across the menstrual cycle," p. 271.
[538] Gerianne M. Alexander and Janet Saenz, "Early androgens, activity levels and toy choices of children in the second year of life," *Hormones and Behavior 62*(4): September 2012, DOI: 10.1016/j.yhbeh.2012.08.008, p. 500-504.

spatial tasks, including mental spatial rotation (Voyer et al., 1995[539]), while women outperform men on verbal tasks such as verbal fluency (Heinzel et al., 2013[540]) and verbal memory (Munro et al., 2012[541]; Murre et al., 2013[542]).”[543] From this basis, one paper investigated the possibility that “sex differences in occupational interests are due, in part, to prenatal androgen influences on differential orientation to objects versus people.”[544] The authors of that paper went on to argue that “[t]his work is relevant to the broad question of women’s underrepresentation in STEM careers because it points to a hormonally-influenced psychological characteristic—interest in working with objects versus people—that underlies vocational interests.”[545] Another paper noted even more straightforwardly that “[d]ifferences in quantitative abilities have received the most

[539] D. Voyer, S. Voyer, and M. P. Bryden, “Magnitude of sex differences in spatial abilities: A meta-analysis and consideration of critical variables,” *Psychological Bulletin 117*(2): 1995, DOI: 10.1037/0033-2909.117.2.250, p. 250-270.
[540] Sebastian Heinzel, Florian G. Metzger, Ann-Christine Ehlis, Robert Korell, Ahmed Alboji, Florian B. Haeussinger, Katja Hagen, Walter Maetzler, Gerhard W. Eschweiler, Daniela Berg, Andreas J. Fallgatter, and the TREND Study Consortium, “Aging-related cortical reorganization of verbal fluency processing: a functional near-infrared spectroscopy study,” *Neurobiology of Aging 34*(2): February 2013, DOI: 10.1016/j.neurobiolaging.2012.05.021, p. 439-450.
[541] Cynthia A. Munro, Jessica M. Winicki, David J. Schretlen, Emily W. Gower, Kathleen A. Turano, Beatriz Muñoz, Lisa Keay, Karen Bandeen-Roche, and Sheila K. West, “Sex differences in cognition in healthy elderly individuals,” *Aging, Neuropsychology, and Cognition 19*(6): 2012, DOI: 10.1080/13825585.2012.690366, p. 759-768.
[542] Jaap M. J. Murre, Steve M. J. Janssen, Romke Rouw, and Martijn Meeter, “The rise and fall of immediate and delayed memory for verbal and visuospatial information from late childhood to late adulthood,” *Acta Psychologica 142*(1): January 2013, DOI: 10.1016/j.actpsy.2012.10.005, p. 96-107.
[543] M. LaClair, M. Febo, B. Nephew, N. J. Gervais, G. Poirier, K. Workman, S. Chumachenko, L. Payne, M. C. Moore, J. A. King, and A. Lacreuse, “Sex Differences in Cognitive Flexibility and Resting Brain Networks in Middle-Aged Marmosets,” *eNeuro 6*(4): 1 July 2019, p. 1-19; DOI: 10.1523/ENEURO.0154-19.2019.
[544] Adriene M. Beltz, Jane L. Swanson, and Sheri A. Berenbaum, “Gendered occupational interests: Prenatal androgen effects on psychological orientation to Things versus People,” *Hormones and Behavior 60*(4): September 2011, DOI: 10.1016/j.yhbeh.2011.06.002, p. 315.
[545] Beltz *et al*., “Gendered occupational interests: Prenatal androgen effects,” p. 316.

attention because of the large sex differences in choice of professional careers in natural science and mathematics favouring males (Halpern et al., 2007[546]).”[547]

On closer inspection, however, this theory of biologically-based differences in STEM employment becomes rather less coherent. The Mental Rotations Test (MRT) is a test designed in 1978[548] to maximize the measurability of sex differences in mental rotation tasks, which involves visualizing an object and rotating it around an axis while comparing it against a set of possibly matching objects (Fig. 5A). A similar test, the Clock Rotation Test, also tests for mental rotation ability but does so within a 2D framework, and, in the harder variant, also shows similar sex differences to the MRT (Fig. 5B, 5C).[549] However, as of a study done in 2013 attempting to validate a link between MRT performance and estradiol levels, not only was it not “known why the MRT evokes such a large sex difference,” “only one study has asked which feature of the MRT underlies its variation over the menstrual cycle.”[550] That study also noted that “[i]t is informative theoretically that the effects of the ovarian cycle reported for mental rotation are *not* seen for many other types of cognitive functions, which exhibit either no relation to the cycle or, in a few cases, such as verbal fluency or certain memory-related functions (e.g., Hampson and

[546] Diane F. Halpern, Camilla P. Benbow, David C. Geary, Ruben C. Gur, Janet Shibley Hyde, and Morton Ann Gernsbacher, “The Science of Sex Differences in Science and Mathematics,” *Psychological Science in the Public Interest 8*(1): August 2007, DOI: 10.1111/j.1529-1006.2007.00032.x, p. 1-51.
[547] Linda Ahrenfeldt, Inge Petersen, Wendy Johnson, and Kaare Christensen, “Academic performance of opposite-sex and same-sex twins in adolescence: A Danish national cohort study,” *Hormones and Behavior 69*: March 2015, DOI: 10.1016/j.yhbeh.2015.01.007, p. 123.
[548] Steven G. Vandenberg and Allan R. Kuse, “Mental Rotations, a Group Test of Three-Dimensional Spatial Visualization,” *Perceptual and Motor Skills 47*(2): 1978, DOI: 10.2466/pms.1978.47.2.599, p. 599-604.
[549] David W. Collins and Doreen Kimura, “A Large Sex Difference on a Two-Dimensional Mental Rotation Task,” *Behavioral Neuroscience 111*(4): August 1997, URL: oce.ovid.com/article/00001975-199708000-00019, p. 845-849.
[550] Elizabeth Hampson, Na’ama Levy-Cooperman, and Jennifer M. Korman, “Estradiol and mental rotation: Relation to dimensionality, difficulty, or angular disparity?” *Hormones and Behavior 65*(3): March 2014, DOI: 10.1016/j.yhbeh.2013.12.016, p. 239.

Morley, 2013;[551] Maki et al., 2002[552]), show improvement at high not low levels of estradiol."[553] In other words, while "language skills appear to consistently favor women,"[554] the only type of mathematical skill that is found to have a significant bias in favor of men are some types of spatial skills, the largest and most consistent effects found with spatial rotation tests.[555]

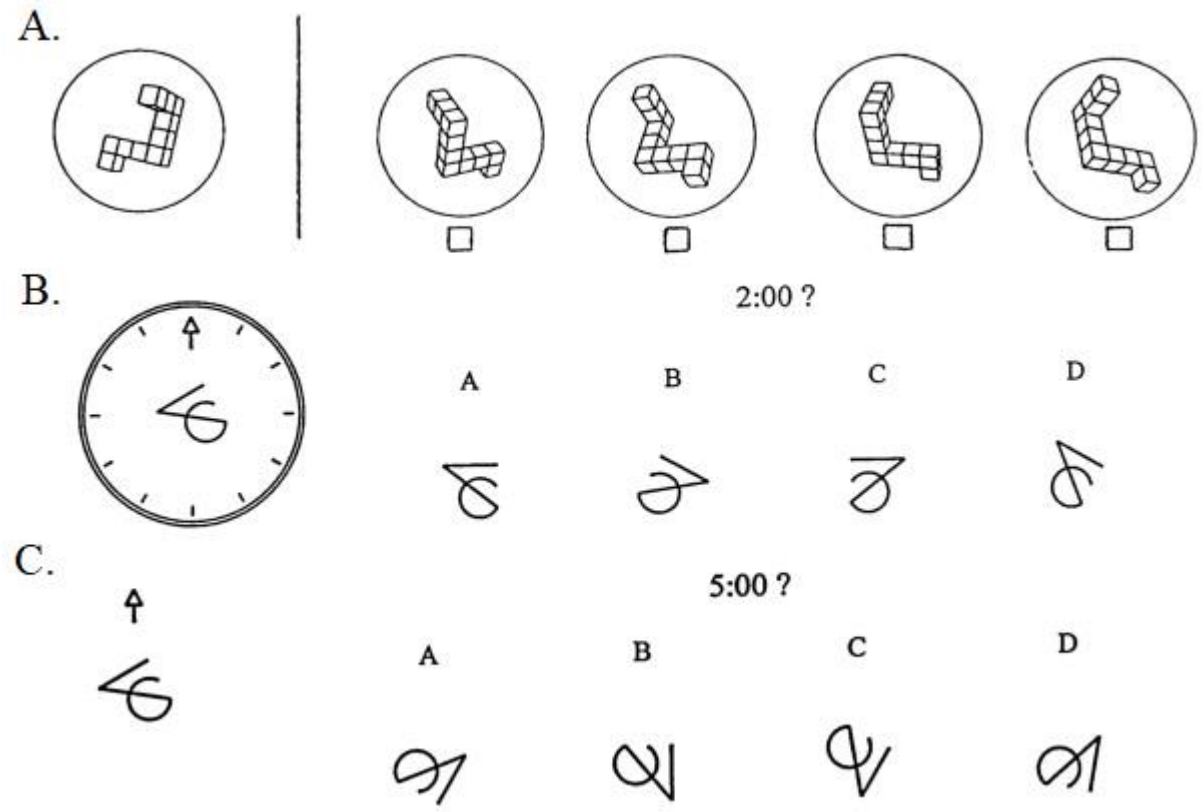

Fig. 5A: Vanderburg and Kuse Mental Rotation Test Example (1978); 5B: Collins and Kimura Easy Clock Rotation Test (1997), note the presence of the clock face around the symbol; 5C:

[551] Elizabeth Hampson and Erin E. Morley, "Estradiol concentrations and working memory performance in women of reproductive age," *Psychoneuroendocrinology 38*(12): December 2013, DOI: 10.1016/j.psyneuen.2013.07.020, p. 2897-2904.
[552] Pauline M. Maki, Jill B. Rich, and R. Shayna Rosenbaum, "Implicit memory varies across the menstrual cycle: estrogen effects in young women," *Neuropsychologia 40*(5): 2002, DOI: 10.1016/S0028-3932(01)00126-9, p. 518-529.
[553] Hampson *et al.*, "Estradiol and mental rotation," p. 239.
[554] 10.1523/JNEUROSCI.0475-18.2019, p. 7233-7234
[555] For example, "other aspects of spatial cognition reveal female advantages (e.g., object identity memory), or no gender differences (e.g., object location memory)." Ibid., p. 7234.

Collins and Kimura Hard Clock Rotation Test (1997). Taken from Hampson et al., "Estradiol and Mental Rotation," p. 241.

Even accepting that these spatial rotation tests measure natural ability,[556] auxiliary assumptions are required to believe that sex differences in ability explain any large percentage of the difference in employment in STEM fields. Firstly, while spatial rotation abilities are required for some STEM careers that show a male bias, such as mechanical engineering,[557] spatial rotation abilities are also required for biochemistry,[558] a STEM career that shows a female bias,[559] and not at all required for computer science, which shows a male bias.[560] Secondly and relatedly, and again assuming that sex-typed cognitions are large and consistent enough to affect employment, it is not at all clear that spatial rotation ability specifically should affect one's ability to do most mathematical or scientific operations. One would have to assume that spatial rotation ability is so necessary that it not only dwarfs the contributions of skill in all other non-rotative mathematical domains (arithmetic, algebra, statistics, much of calculus, much of

[556] Which we perhaps should not—mental rotation tasks show "reversals to female advantages… when mental rotation tasks are framed as 'art tasks' rather than 'math tasks,'" Ibid., p. 7234, and similar cultural, societal, or psychological pressures could equally lower the male scores in language abilities.
[557] In the United States in the academic year 2020-21 within mechanical engineering/mechanical technology/technician, bachelor's degrees were awarded to 30,234 men and 5,990 women. (Table 318.30 in Cristobal de Brey, Anlan Zhang, and Sarah Duffy, *Digest of Education Statistics, 2021* (NCES 2023009), National Center for Education Statistics, Institute of Education Sciences, US Department of Education). In the European Union in 2021 in category F0715: Engineering and engineering trades, Mechanics and metal trades, bachelor's degrees or equivalents were awarded to 35,772 men and 5,633 women (Eurostat, "Graduates by education level, programme orientation, sex and field of education," *Eurostat,* DOI: 10.2908/EDUC_UOE_GRAD02.)
[558] Differentiating between stereoisomers, a distinction that is highly relevant given that biochemical processes can often only process one stereoisomer or the other, is a nearly identical exercise to the Vanderberg Mental Rotation Test, requiring complex structures to be rotated and compared.
[559] In the United States in the academic year 2020-21 within biochemistry, bachelor's degrees were awarded to 4,155 men and 5,199 women (Table 318.30 in de Brey *et al.*, *Digest of Education Statistics, 2021*). In the European Union in 2021 in category F0512: Biochemistry, bachelor's degrees or equivalents were awarded to 4,539 men and 9,120 women (Eurostat, DOI: 102908/EDUC_UOE_GRAD02).
[560] In the United States in the academic year 2020-21 within computer and information sciences and support services, bachelor's degrees were awarded to 81,921 men and 22,953 women (Table 318.30 in de Brey *et al., Digest of Education Statistics, 2021*). In the European Union in 2021 in category F061: Information and Communication Technologies, bachelor's degrees or equivalents were awarded to 60,933 men and 15,409 women (Eurostat, DOI: 102908/EDUC_UOE_GRAD02).

geometry, linear algebra, etc.), but also makes irrelevant the contributions of all other types of ability, such the ability to write, read, speak, synthesize and analyze information, think logically, manage assistants, memorize images or facts, describe observations, design experiments, organize groups, have relevant background experience or knowledge, or compensate for a lack of innate ability with, as the "Easy" Clock Test implicitly suggested,[561] external guideline systems or computational tools.

Spatial rotation got expanded to the whole of mathematical and scientific ability as well as employment rates in a way that was never really supported by the evidence and came with more contradictions than support. There is perhaps a difference on average between men and women in some cognitive areas which is down to genetics or hormones. However, by attempting to explain different rates of participation in fields of employment by gender with this research, the researchers, by and large, show a fundamental carelessness with a specific aspect of the truth. Behavioral neuroendocrinologists, particularly the organizational/activational theorists researching this question, were not actually seeking to answer the question "Is our research relevant?", because the researchers had already assumed that the answer must be "yes". Why would spatial rotation alone be relevant? What jobs actually require spatial rotation, and what are the gender ratios within these fields? Do these assertions, in fact, translate into reality? The analysis sections were arriving at a predetermined answer, and so the individual facts no longer mattered as much as the conclusion. As §4.2 noted, an absence of justification can reflect an assumed continuity. In this case, the assumption seems to be that basic research on spatial reasoning was primarily or even somewhat explanatory for STEM employment, supported by the

[561] Differences in ability between either men and women or women at different stages of their menstrual cycle disappeared when the clockface was available as a reference to assist with the task of mentally rotating the figure; compare Figure 5B (Easy) with 5C (Hard).

additional assumption that spatial ability means STEM and verbal ability means all other areas of employment and knowledge. It is the collective failure to grapple with the complexities of lived experience, in this section and §4.2, that prevents the organizational/activational theory and behavioral neuroendocrinology more generally from explaining lived experience.

4.4: Assumed Reification—Diagnosing Psychiatry with Biological Disease

> *"The disease model, I would argue, makes the following four claims about mental disorder. First, it maintains that the causes of mental disorders are to be found in disturbances or abnormalities of biological structures, functions, or processes. … Second, for each disorder there is a specific etiology, a single common abnormality that determines the nature or form of the disorder. Third, disorders are discrete categories such that affected individuals differ qualitatively from the unaffected. Disorders correspond to real, categorically distinct entities, rather than simply being pragmatically useful ways of sorting people into groups with similar features…. Diagnosis is … crucial, a matter of identifying the kind that the individual exemplifies, involving an inference from observed clinical features back to an underlying type. Considering the individual primarily as an instance of a kind is vital because each kind is a potent source of inductive inferences about cause, pathological mechanism, clinical expression and course, and response to treatment. Fourth and finally, the disease model implies that mental disorders are not deeply culture- or time-bound, what Reznek (1991)*[562] *terms the 'universality thesis.' Historical and cross-cultural variations are generally minor and incidental, because the expressions of mental disorders are primarily outgrowths of*

[562] L. R. Reznek, *The Philosophical Defense of Psychiatry*, Routledge (London): 1991.

biological processes and 'the social and psychological dimensions of sickness are seen as epiphenomenal' (Kleinman, 1988, p. 143)[563]*."*[564]

The assumptions covered so far in this chapter excluded alternative explanations for observed sexed/gendered differences. In this case, however, the exclusion of alternatives is the starting point. As the quote above discusses, treating psychiatric diagnoses as fundamentally biological entities located the diagnoses within the patients themselves, reifying[565] the psychiatric diagnoses as biological. This reification immediately excluded possible sociological reasons for differing rates of prevalence or of diagnosis. From there, organizational/activational theorists readily noted that some psychiatric diagnoses displayed a gender/sex bias. Using this observed difference in rates of diagnosis, organizational/activational theorists, and behavioral neuroendocrinology more broadly, argued that the gender/sex bias meant that the etiology of those psychiatric disorders was tied up with sex. The assumption that diagnoses were fundamentally biological entities was what allowed the epidemiological characteristics of these diagnoses to be incorporated into organizational/activational theory at all. After the diagnoses had been incorporated, the internal assumptions of organizational/activational theory[566] then guided the theorists' use of those diagnoses.

For example, autism and depression were sex-typed counterpart mental health diagnoses. For autism diagnoses, the diagnostic imbalance was 4 phenotypic males for every phenotypic female, and "[a]mong individuals diagnosed with an ASD [autism spectrum disorder] without any physical or cerebral abnormalities as measured by MRI the ratio has been estimated to be as

[563] A. Kleinman, *Rethinking Psychiatry,* Free Press (London): 1988, p. 143.
[564] Nick Haslam, "Psychiatric Categories as Natural Kinds: Essentialist Thinking about Mental Disorder," *Social Research 67,* no. 4: Winter 2000, URL: jstor.org/stable/40971424, p. 1033-1034.
[565] Reification by assignment, perhaps; the abstract entity was assigned to and incorporated into the individuals it described in order to make it concrete.
[566] As discussed in chapter 2 and to a degree in chapter 3.

high as 23:1 (Miles and Hillman, 2000[567]).”[568] Depression diagnoses, by contrast, had a gendered ratio of approximately 2 phenotypic females for every phenotypic male.[569] These gender disparities became an initial piece of evidence, as multiple researchers posited that “[t]he male dominance in ASD suggests that sex hormones could play a role in the diagnostic gap between males and females,”[570] while others stated that “the increased susceptibility of females to depression has been well documented.”[571] The evidence also included studies of infants who had had elevated fetal testosterone levels, which were associated “with poor social relationships and the development of restricted interests… (Knickmeyer et al., 2005)[572]…[,] reduced frequency of eye contact at 12 months of age (Lutchmaya et al., 2002),[573] reduced vocabulary size from 18 to 24 months (Lutchmaya et al., 2001),[574] and reduced scores on the Empathy Quotient (EQ) with

[567] Judith H. Miles and Richard E. Hillman, “Value of a clinical morphology examination in autism,” *American Journal of Medical Genetics 91*(4): 10 April 2000, DOI: 10.1002/(SICI)1096-8628(20000410)91:4<245::AID-AJMG1>3.0.CO;2-2, p. 245-253.
[568] Sara M. Schaafsma and Donald W. Pfaff, “Etiologies underlying sex differences in Autism Spectrum Disorders,” *Frontiers in Neuroendocrinology 35*(3): August 2014, DOI: 10.1016/j.yfrne.2014.03.006, p. 255.
[569] Cynthia L. Bethea, Nick Z. Lu, Chrisana Gundlah, and John M. Streicher, “Diverse Actions of Ovarian Steroids in the Serotonin Neural System,” *Frontiers in Neuroendocrinology 23*(1): January 2002, DOI: 10.1006/frne.2001.0225, p. 42.
[570]Amer Moosa, Henry Shu, Tewarit Sarachana, and Valerie W. Hu, “Are endocrine disrupting compounds environmental risk factors for autism spectrum disorder?” *Hormones and Behavior 101*: May 2013, DOI: 10.1016/j.yhbeh.2017.10.003, p. 15.
[571] Tracy L. Bale and Wylie W. Vale, “Increased Depression-Like Behaviors in Corticotropin-Releasing Factor Receptor-2-Deficient Mice: Sexually Dichotomous Responses.” *Journal of Neuroscience 23*(12): 15 June 2003, DOI: 10.1523/JNEUROSCI.23-12-05295.2003, p. 5296.
[572] Rebecca Knickmeyer, Simon Baron-Cohen, Peter Raggatt, and Kevin Taylor, “Foetal testosterone, social relationships, and restricted interested in children,” *The Journal of Child Psychology and Psychiatry 46*(2): February 2005, DOI: 10.1111/j.1469-7610.2004.00349.x, p. 198-210.
[573] Svetlana Lutchmaya, Simon Baron-Cohen, and Peter Raggatt, “Foetal testosterone and eye contact in 12-month-old human infants,” *Infant Behavior and Development 25*(3): 2002, DOI: 10.1016/S0163-6383(02)00094-2, p. 327-335.
[574] Svetlana Lutchmaya, Simon Baron-Cohen, and Peter Raggatt, “Foetal testosterone and vocabulary size in 18- and 24-month-old infants,” *Infant Behavior and Development 24*(4): 18 April 2001, DOI: 10.1016/S0163-6383(02)00087-5, p. 418-424.

increased Systematizing Quotient (SQ) at 96 months (Auyeung et al., 2006;[575] Chapman et al., 2006)[576].”[577]

From this information and the assumptions of organizational/activational theory more broadly, researchers argued that “human and animal data …[that] suggest that biological and genetic factors also significantly contribute” to the development of the underlying behaviors[578] or more forcefully insisted[579] that “[t]hese sex differences are seen in multiple diverse countries and cultures, suggesting a biological basis.”[580] Autism was thereby connected to androgens, particularly prenatal androgens due to frequent childhood diagnosis of autism or autism spectrum disorders, and, perhaps due to ideas of female-as-default,[581] researchers argued that depression emerged from a greater organized and/or activated sensitivity of stress feedback systems connected to low levels of testosterone and which were therefore more common in women.

[575] Bonnie Auyeung, Simon Baron-Cohen, Emma Chapman, Rebecca Knickmeyer, Kevin Taylor, and Gerald Hackett, “Foetal testosterone and the child systemizing quotient,” *European Journal of Endocrinology 155* Supplement 1: November 2006, DOI: 10.1530/eje.1.02260, p. S123-S130.
[576] Emma Chapman, Simon Baron-Cohen, Bonnie Auyeung, Rebecca Knickmeyer, Kevin Taylor, Gerald Hackett, “Fetal testosterone and empathy: Evidence from the Empathy Quotient (EQ) and the ‘Reading the Mind in the Eyes’ Test,” *Social Neuroscience 1*(2): 2006, DOI: 10.1080/17470910600992239, p. 135-148.
[577] Moosa *et al.,* “Are endocrine disrupting compounds environmental risk factors for autism spectrum disorder?” p. 15.
[578] Klump, “Puberty as a critical risk period for eating disorders,” p. 400. The quote itself is taken from a paper about eating disorders but well expresses the general opinion of the papers about the hormonal origins of autism and depression, as well as eating disorders, depression, oppositional and defiant behaviors, ADHD, and so on.
[579] As the quote above suggests.
[580] Margaret Altemus, “Sex differences in depression and anxiety disorders: Potential biological determinants,” *Hormones and Behavior 50*(4): November 2006, DOI: 10.1016/j.yhbeh.2006.06.031, p. 534. While it might be churlish to point out, this sentence has no citation, and so I will not speculate about the identities of the “multiple diverse countries and cultures” to which the author refers. More substantively, please see §4.2’s discussion of the history of the nuclear family for a discussion as to why ‘cultural similarities’ surrounding gender roles and familial structures should not necessarily be taken at face value.
[581] See §2.4.

The androgen theory of autism, proposed and researched by Simon Baron-Cohen,[582] argued that (1) autistic people displayed an impaired ability to socialize, imagine, verbalize, and empathize,[583] (2) that females show higher ability at these tasks,[584] and therefore that (3) autism can be described as "an extreme of the male brain (Baron-Cohen, 2002[585])."[586] In turn, researchers building from these theories asked, "Is the predominantly female disorder of depression (2:1 female to male ratio, Hammen, 1997)[587] a characteristic of the extreme female brain?"[588] On a theoretical level, that this theory recapitulates the philosophical and traditional dichotomy between masculine reason and feminine emotion[589] reproduced in a scientific context[590] had been noted well before this thesis.[591] On a factual level, the characterization of autistic people as lacking the ability to empathize (and imagine)[592] has been disputed and disconfirmed by further research and theorizing,[593] including by autistic people themselves,[594] and, eventually, by Simon Baron-Cohen.[595]

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[582] A highly influential figure and commonplace in this research—Baron-Cohen is an author in all five papers in ft. 129 to 133, cited in the same paragraph in ft. 134.
[583] Knickmeyer *et al.*, "Fetal testosterone and empathy," p. 282-283; Pearson and Lewis, "Fear recognition across the menstrual cycle," p. 267-269.
[584] Knickmeyer *et al.*, "Fetal testosterone and empathy," p. 282-283; Pearson and Lewis, "Fear recognition across the menstrual cycle, p. 267-269.
[585] Simon Baron-Cohen, "The extreme male brain theory of autism," *Trends in Cognitive Sciences 6*(6): 1 June 2002, DOI: 10.1016/S1364-6613(02)01904-6, p. 248-254.
[586] Knickmeyer *et al.*, "Fetal testosterone and empathy," p. 283.
[587] Constance Hammen, *Depression*, part of *Clinical Psychology A Modular Course*, ed. Chris R. Brewin, Psychology Press (East Sussex): 1997.
[588] Pearson and Lewis, "Fear recognition across the menstrual cycle, p. 270.
[589] Christine A. James, "Feminism and Masculinity: Reconceptualizing the Dichotomy of Reason and Emotion," *International Journal of Sociology and Social Policy 17*, no. 1-2: 1997, DOI: 10.1108/eb013296, p. 129-152.
[590] Stephanie A. Shields, "Gender and Emotion: What We Think We Know, What We Need to Know, and Why It Matters," *Psychology of Women Quarterly 37*(4): December 2013, DOI: 10.1177/0361684313502312, p. 423-235.
[591] For example, Cordelia Fine's *Delusions of Gender* and Gina Rippon's *The Gendered Brain* (UK) or *Gender and Our Brains* (US).
[592] Anat Kasirer, Esther Adi-Japha, and Nira Mashal, "Verbal and Figural Creativity in Children With Autism Spectrum Disorder and Typical Development," *Frontiers in Psychology 11*, ID 559238*:* 2020, DOI: 10.3389/fpsyg.2020.559238, p. 1-15. The research here is less established, but as with empathy, the actual barrier appears to be in communication, verbal or non-verbal, explicit or implicit—autistic people are both empathetic and imaginative, but have a combination of difficulty communicating their empathy and imagination to others in a form comprehensible to neurotypical individuals, some difficulty understanding the cues of neurotypical people (though,

Turning to depression as female-typed, reviews examining depression tended to suggest that women had an organizational and activational sensitivity of the hypothalamic-pituitary-gonadal (HPG) and hypothalamic-pituitary-adrenal (HPA) axes. "Although historically sex differences in medical and psychiatric disorders were largely ignored,[596] more recent research has focused on the neurobiological underpinnings of sex differences in vulnerability and resilience to stress and its related disorders."[597] In particular, "the sensitivity of the individual to stress"[598] or "dysregulation of the stress response"[599] could be a precipitator of "abnormalities in the regulation of the hypothalamic-pituitary adrenal axis,"[600] and women, particularly after puberty, exhibited "a greater magnitude and duration of HPA response to different stressors

as conceptions of the 'double empathy problem' (ft. 594) suggest, likely not as much difficulty as neurotypical individuals have understanding the cues of autistic people), and lower rates of internalizing non-verbal cues.

[593] Catherine J. Crompton, Danielle Ropar, Claire V. M. Evans-Williams, Emma G. Flynn, and Sue Fletcher-Watson, "Autistic peer-to-peer information transfer is highly effective," *Autism 24*(7): October 2020, DOI: 10.1177/1362361320919286, p. 1704-1712; Yu-Lun Chen, Laura L. Senande, Michael Thorsen, and Kristie Patten, "Peer preferences and characteristics of same-group and cross-group social interactions among autistic and non-autistic adolescents," *Autism 25*(7): October 2021, DOI: 10.1177/13623613211005918, p. 1885-1900; Sue Fletcher-Watson and Geoffrey Bird, "Autism and Empathy: What are the real links?" *Autism 24*(1): January 2020, DOI: 10.1177/1362361319883506, p. 3-6.

[594] Damian E. M. Milton, "On the ontological status of autism: the 'double empathy problem'," *Disability and Society 27*(6): 2012, DOI: 10.1080/09687599.2012.710008, p. 883-887; Rebekah C. White and Anna Remington, "Object personification in autism: This paper will be very sad if you don't read it," *Autism 23*(4): May 2019, DOI: 10.1177/1362361318793408, p. 1042-1045.

[595] Ido Shalev, Varun Warrier, David M. Greenberg, Paula Smith, Carrie Allison, Simon Baron-Cohen, Alal Eran, and Florina Uzefovsky, "Reexamining empathy in autism: Empathic disequilibrium as a novel predictor of autism diagnosis and autistic traits," *Autism Research 15*(10): October 2022, DOI: 10.1002/aur.2794, p. 1917-1928.

[596] This statement does not come with an accompanying citation. The work of Sigmund Freud on psychosexual development and penis envy, the diagnosis of "hysteria," and that homosexuality-as-diagnosis was conceived largely as a failure of gender/sexed normality in the patient and thereby an illness of the mind would seem to contradict this statement. See also the work of Laura Hirschbein. Laura D. Hirschbein, "Science, Gender, and the Emergence of Depression in American Psychiatry, 1952-1980," *Journal of the History of Medicine and Allied Sciences 61,* no. 2: April 2006, URL: jstor.org/stable/24632297, p. 187-216 and Laura Hirschbein, "Sex and Gender in Psychiatry: A View from History," *Journal of Medical Humanities 31*: 2010, DOI: 10.1007/s10912-010-9105-5, p. 155-170.

[597] Bangasser and Valentino, "Sex differences in stress-related psychiatric disorders," p. 303.

[598] Bale and Vale, "Increased Depression-Like Behaviors in Corticotropin-Releasing Factor Receptor-2-Deficient Mice," p. 5295.

[599] Alon Chen, Eric Zorrilla, Sean Smith, David Rousso, Coree Levy, Joan Vaughan, Cindy Donaldson, Amanda Roberts, Kuo-Fen Lee and Wylie Vale, "Urocortin 2-Deficient Mice Exhibit Gender-Specific Alterations in Circadian Hypothalamus–Pituitary–Adrenal Axis and Depressive-Like Behavior," *Journal of Neuroscience 26*(20): 17 May 2006, DOI: 10.1523/JNEUROSCI.3955-05.2006, p. 5500.

[600] Altemus, "Sex differences in depression and anxiety disorders," p. 534.

(Rhodes and Rubin, 1999)[601].”[602] Testosterone, in contrast, might exert a protective effect against depression, as higher testosterone levels were associated with reduced vulnerability to trauma[603] and men with hypogonadism or being treated with androgen-depleting drugs for prostate cancer reported depressive and anxious symptoms or disorders that were improved with testosterone therapies.[604]

While these explanations for sex-biased diagnosis rates fit well within the organizational/activational framework, the evidence supporting these theories was never particularly strong. The relationship between autism and being a phenotypic male primarily rested on studies connecting prenatal androgens to later observed behavior, relying on the pre-existing association between organizing androgens acting prenatally to predispose a fetus to male-type behavior[605] to fully connect the two. Prenatal androgens in human infants are, however, quite difficult to measure accurately. A study in *Hormones and Behavior* in 2004 considered the various methods of measuring fetal testosterone, noting that the different methods used to that point did not correlate well, and additionally that “the main access of androgens into

[601] Michael E. Rhodes and Robert T. Rubin, “Functional sex differences (`sexual diergism') of central nervous system cholinergic systems, vasopressin, and hypothalamic–pituitary–adrenal axis activity in mammals: a selective review,” *Brain Research Reviews 30*(2): August 1999, DOI: 10.1016/S0165-0173(99)00011-9, p. 135-152.
[602] Chen *et al.,* “Urocortin 2-Deficient Mice Exhibit Gender-Specific Alterations,” p. 5500.
[603] There are some theoretical issues with this paper in particular, discussed in ft. 322 in §3.5. Cooke and Weathington, “Human and animal research into sex-specific effects of child abuse,” p. 416-417.
[604] That there are many reasons that hypogonadal men, some with HIV, and those with prostate cancer might respond positively to testosterone treatments appears to not occurred to the authors; as testosterone therapy might be interpreted as “giving one their masculinity (back)” as well as “ensuring patients may live as normal a life as possible”, which would both increase positive feelings in the patients, the causative role of testosterone in these cases seems less clear than the authors imply. McHenry *et al.,* “Sex differences in anxiety and testosterone,” p. 42-43. See also: Susanne Fischer, Ulrike Ehlert, and Rita Amiel Castro, “Hormones of the hypothalamic-pituitary-gonadal (HPG) axis in male depressive disorders – A systematic review and meta-analysis,” *Frontiers in Neuroendocrinology 55*, ID 100792: October 2019, DOI: 10.1016/j.yfrne.2019.100792, p. 1-9.
[605] For example, in Phoenix *et al.,* “ORGANIZING ACTION OF PRENATALLY ADMINISTERED TESTOSTERONE PROPIONATE.”

the amniotic fluid is by fetal urine (Judd et al., 1976;[606] Schindler, 1982)[607].”[608] The relationship between fetal urine hormone levels and fetal blood hormone levels is mediated by the kidneys, and while there is likely some relationship between the two, the strength of the relationship is too dangerous to the fetus to directly investigate. The prenatal androgens in the fetus’s body cannot be measured, and, as discussed in §2.2, the responsiveness of any individual depends on the responsiveness of the receptor as well as the levels of the circulating hormone. Because of these realities, the informational content of comparisons of fetal testosterone levels between male fetuses is, at best, highly limited.

For depression diagnoses, there were four pieces of additional circumstantial evidence implicating circulating estrogen as a risk factor for depression. Firstly, the sex differences in depression diagnoses “is not manifested until puberty,”[609] although this shows an absence of compensation for the societal shifts and experiences of puberty for girls as discussed in §4.2. Secondly, the onset of menopause (surgically or naturally) is also correlated with depression, although this correlation is also possibly affected by changes in self-conception and social evaluation concomitant with the loss of reproductive ability. Thirdly, postpartum women can experience depression and psychosis[610] “coincident with the precipitous decline in E [estrogen] and P [progesterone],”[611] although an absence of social support after childbirth or a traumatic

[606] Howard L. Judd, Jill D. Robinson, Philip E. Young, and Oliver W. Jones, “Amniotic Fluid Testosterone Levels in Midpregnancy,” *Obstetrics and Gynecology 48*(6): December 1976, ISSN: 0029-7844, p. 690-692.
[607] Adolf E. Schindler, “Origin of Human Amniotic Fluid,” *Monographs on Endocrinology 21*: 1982, DOI: 10.1007/978-3-642-81656-7_2, p. 2-4.
[608] van de Beek *et al*., “Relationships between sex hormones assessed in amniotic fluid,” p. 668.
[609] Bethea *et al.*, “Diverse Actions of Ovarian Steroids in the Serotonin Neural System,” p. 42.
[610] As well as postpartum psychosis, which is what the quote (ft. 611) is about; however, postpartum psychosis is a complex phenomenon and appears to be, at least in some cases, better treated as a severe and unprecedented bipolar episode possibly triggered by the stresses—particularly the sleeplessness—of childbirth and caring for an infant. Lauren M. Osborne, “Recognizing and Managing Postpartum Psychosis: A Clinical Guide for Obstetric Providers,” *Obstetrics and Gynecology Clinics of North America 45*(3): September 2018, DOI: 10.1016/j.ogc.2018.04.005, p. 455-468.
[611] Bethea *et al.*, “Diverse Actions of Ovarian Steroids in the Serotonin Neural System,” p. 42.

birth experience, for example, may also be risk factors for postpartum depression in particular.[612] Fourthly, the most concrete link between estrogen and mood is premenstrual dysphoric disorder (PMDD), a mood disorder linked to the menstrual cycle.[613] "These women are improved by elimination of ovarian estrogen (E) and progesterone (P) …, and readministration of either E or P in a nonfluctuating manner will precipitate the mood disorder."[614]

Each of these pieces of evidence is confounded. The shared confounding factor is that each event (puberty, menopause, childbirth) comes with societally mediated and often relatively abrupt changes in lived experiences and embodiment, often experienced negatively as discussed in §4.2. One might similarly note that observations of administered testosterone reducing depressive symptoms in male patients is also confounded by the social mediation of medical restoration of "manhood".[615] In addition, while estrogen and progesterone levels are implicated as a causal agent for PMDD, the accuracy of extrapolating that to all other women[616] with depression is speculative, given that the proximate causes of PMDD, a diagnosis which includes less emotional symptoms like marked changes in appetite and pain, were (and remain) unclear.[617]

For both autism and depression, there was a marked absence of robust evidence that hormones, acting organizationally or activationally, are etiological factors in diagnosis. There are correlations between each diagnosis and other variables, but causation cannot be justified. While autism as extreme male brain and depression as feminine vulnerability both suffered from other

[612] Dorothy K. Y. Sit and Katherine L. Wisner, "Identification of Postpartum Depression," *Clinical Obstetrics and Gynecology 52*(3): September 2009, DOI: 10.1097/GRF.0b013e3181b5a57c, p. 456-468.
[613] Bethea *et al.*, "Diverse Actions of Ovarian Steroids in the Serotonin Neural System," p. 42.
[614] Ibid.
[615] Manhood is in quotations here because testosterone or androgens being a molecule of manhood is a fraught relationship; see chapter 2.
[616] Or, more properly, all of those who were assigned female at birth.
[617] Andrea J. Rapkin and Erin I. Lewis, "Treatment of Premenstrual Dysphoric Disorder," *Women's Health 9*(6): November 2013, DOI: 10.2217/WHE.13.62, p. 537-556.

evidentiary deficits, the first failing in each case was to accept that diagnoses existed outside of—biologically below—society. Indeed, each section of this chapter has described how researchers working on organizational/activational theory, and to some degree other behavioral neuroendocrinologists, denied the social and historical aspects of the evidence they wished to use. The excluded alternative explanations suggested that observations of sex differences are, or at least might be, marking aspects of humanity that are contextual and contingent rather than enduring or essential. "Science", to return to the quote that opened this chapter, is supposed to exist outside of social factors—and so, social factors were removed by assuming the social world was secondary to the biological world.

4.5: Assumed Power—Great Responsibilities and Good News

> *"Although the world of truth differs absolutely, not relatively, from the social world, the Scientist can go* back and forth *from one world to the other no matter what: the passageway closed to all others is open to him alone. In him and through him, the tyranny of the social world is miraculously interrupted when he leaves, so that he will be able to contemplate the objective world at last; and it is likewise interrupted when he returns, so that like a latter-day Moses he will be able to substitute the legislation of scientific laws, which are not open to question, for the tyranny of ignorance. Without this double interruption there can be no Science…, no Western conception of public life. [emphasis in original]"*[618]

Latour's description of the intentional separation of the social world and natural world explores the lowest and most fundamental assumption underlying all the others that this chapter has discussed. This is the assumption: there exists a social world and a natural world; the natural

[618] Latour, *Politics of Nature,* p. 11.

world is external to and unaffected by the social world, but not *vice versa*, and the scientist can speak for the natural world to the social world. From this foundation, all else follows. The evolution of humanity is not a historical question for which there shall never be enough evidence, but a scientific one which scientific laws can answer (§4.1). The behaviors of today reflect the biological consequences of development, not the happenstances of modern history (§4.1, 4.2, 4.3. 4.4). Medical diagnoses describe biology, so medical diagnoses can be treated as inputs for biological thought (§4.4). Indeed, all people are biological, and a biological distinction marks the groups of interest, so biological etiologies must be a meaningful framework to apply (§4.2, 4.3). For example, "[w]hile learning and enculturation could contribute to the sex difference in emotional processing, this begs the question as to where such patterns of enculturation would originate, if not from an original 'biological' predisposition."[619] Finally, the social world cannot affect the natural world, so understanding the social world is unnecessary—except insofar as understanding the social world will help to communicate the "legislation of scientific laws"[620] to the public. This section will focus on how efforts to publicize the findings of organizational/activational theorists, behavioral neuroendocrinologists, and neuroscientists overall without actually engaging with the social elements means that those findings are unrepresentative of reality.

Even if the social world and natural world could be separated, any scientist studying humans, directly or by proxy, cannot separate these worlds. A critical step of the scientific

[619] Cooke and Weathington, "Human and animal research into sex-specific effects of child abuse," p. 420. Using roughly the same logic, it could also be argued that the German palate is biologically predisposed to beer and the French palate to wine based on their relative consumption in each nation. Equally, if inheritance is stripped from this argument, one might argue that because fields are generally plowed in straight lines, but sailboats move with or tack against the wind, that therefore human farmers possess a biological predisposition to linear movement while sailors only move relatively to outside forces.
[620] Latour, *Politics of Nature,* p. 11.

process(es) is publication, and it is there that the problems begin. One review discussing possible reasons for the male bias in autism spectrum disorder (ASD) diagnoses noted that:

> *"**Many studies document the higher prevalence of ASD in boys than in girls. One contributing factor may be an influence of gender on diagnosis**. … [T]he gendered social environment is different between the sexes, which may lead to perception of the same social deficit being categorized as shy in girls and unresponsive in boys, contributing to the diagnostic bias towards males (Goldman, 2013).[621] Additionally, the tests used to diagnose an individual may be biased towards the male specific pathophenotype, and may not include the aspects necessary to diagnose a girl with a mild form of ASD. **Moreover, because of the awareness of the sex bias in ASD incidence, ASD is interpreted as a male disorder (Baron-Cohen, 2002),[622] making it more likely for clinicians to diagnose boys with the disorder, as they are a risk group, than girls.** Indeed, one study reports that girls are diagnosed with an ASD less frequently than boys with the same autistic trait scores (Russell et al., 2011).[623] This is supported by a study that found girls who met the case definition of ASD as established by the Autism and Developmental Disabilities Monitoring (ADDM) Network for ASD surveillance were less likely to have a formal ASD diagnosis than boys (Giarelli et al., 2010).[624] The finding in*

[621] Sylvie Goldman, "Opinion: Sex, gender and the diagnosis of autism—A biosocial view of the male preponderance," *Research in Autism Spectrum Disorders 7*(6): June 2013, DOI: 10.1016/j.rasd.2013.02.006, p. 675-679.
[622] Baron-Cohen, "The extreme male brain theory of autism."
[623] Ginny Russell, Colin Steer, and Jean Golding, "Social and demographic factors that influence the diagnosis of autistic spectrum disorders," *Social Psychiatry and Psychiatric Epidemiology 46:* 2011, DOI: 10.1007/s00127-010-0294-z, p. 1283-1293.
[624] Ellen Giarelli, Lisa D. Wiggins, Catherine E. Rice, Susan E. Levy, Russell S. Kirby, Jennifer Pinto-Martin, and David Mandell, "Sex differences in the evaluation and diagnosis of autism spectrum disorders among children," *Disability and Health Journal 3*(2): April 2010, DOI: 10.1016/j.dhjo.2009.07.001, p. 107-116.

> *several studies that girls are diagnosed later than boys (Mandell et al., 2010;[625] Rhoades et al., 2007;[626] Shattuck et al., 2009[627]) supports the hypothesis that girls are more likely to be underdiagnosed than boys. Furthermore, it may result in later intervention of[*sic*] ASD in girls leading to worse ASD scores later in life compared to boys.* ***These observations indicate that, although boys are more vulnerable to develop[*sic*] ASD than girls, clinicians should not treat being a boy as a leading factor for a diagnosis of ASD****. [bolding added]"*[628]

The authors are describing a self-fulfilling prophecy, at least for those girls who did not receive an autism spectrum diagnosis.[629] They were not boys, and boys get autism spectrum disorder far more often, so those girls could not have autism spectrum disorder; Q.E.D.

While clinicians are perhaps the most likely to respond to scientific findings in a reflexive way, as a point of professional practice and adherence to the latest available information, the problem is broader. "Reflexive public predictions" are predictions which influence the behavior of the public to the point that the truth value of the prediction depends, at least in part, on the publicization of the prediction itself.[630] A simple example of this idea is that an economist might

[625] David S. Mandell, Knashawn H. Morales, Ming Xie, Lindsay J. Lawer, Aubyn C. Stahmer, and Steven C. Marcus, "Age of Diagnosis Among Medicaid-Enrolled Children With Autism, 2001-2004," *Psychiatric Services 61*(8): August 2010, DOI: 10.1176/ps.2010.61.8.822, p. 735-852.
[626] Rachel A. Rhoades, Angela Scarpa, and Brenda Salley, "The importance of physician knowledge of autism spectrum disorder: results of a parent survey," *BMC Pediatrics 7*, ID 37: 2007, DOI: 10.1186/1471-2431-7-37.
[627] Paul T. Shattuck, Maureen Durkin, Matthew Maenner, Craig Newschaffer, David S. Mandell, Lisa Wiggins, Li-Ching Lee, Catherine Rice, Ellen Giarelli, Russell Kirby, Jon Baio, Jennifer Pinto-Martin, and Christopher Cuniff, "Timing of Identification Among Children With an Autism Spectrum Disorder: Findings From a Population-Based Surveillance Study," *Journal of the American Academy of Child and Adolescent Psychiatry 48*(5): May 2009, DOI: 10.1097/CHI.0b013e31819b3848, p. 474-483.
[628] Schaafsma and Pfaff, "Etiologies underlying sex differences in Autism Spectrum Disorders," p. 265.
[629] It is also worth wondering if any boys got diagnoses who 'should not' have, for similar reasons.
[630] Matthew Kopec, "A More Fulfilling (and Frustrating) Take on Reflexive Predictions," *Philosophy of Science 78*, no. 5: December 2011, DOI: 10.1086/662266, p. 1249-1259; The idea was first characterized by Roger C. Buck, "Reflexive Predictions," *Philosophy of Science 30*, no. 4: October 1963, URL: jstor.org/stable/186067, p. 359-369.

believe that a bank is about to fail and publicize the idea, which then causes a run on the bank,[631] which causes the bank to fail. The bank might or might not have failed anyway, but the prediction was the cause of how the bank did fail. While sex differences are not exactly a prediction, discussions of sex differences that are accessible to the public may similarly affect public behavior. Research and meta-analyses since the 1980s have indicated that sex/gender differences in empathy and other tasks are manipulable by changing the salience of various motivations, including a desire to exhibit gendered behavior aligned with the self-conception of the individual.[632] That research would suggest that any discussion of sex differences would have the potential to affect the behavior of the public if sufficiently well-publicized.

The organizational/activational theory of sex differences did reach the public. The originator of the androgen theory of autism, Simon Baron-Cohen, also wrote a book on the topic intended for the lay public: *The Essential Difference: Male and Female Brains and the Truth About Autism,*[633] published in 2003. The book was received relatively positively at the time[634] and was commercially successful enough for Baron-Cohen to author or co-author multiple other

[631] Withdrawal of money from accounts by many account holders simultaneously, generally implied to be somewhat irrational. As banks are loaning entities and take money deposited by one account and loan it to another, they generally do not have sufficient capital to repay all accounts, or even many of their accounts, at the same time; without other lines of credit, the bank will fail. Such collapses were common in the United States prior to the market reforms during and after the Great Depression.

[632] Charlotte S. Löffler and Tobias Greitemeyer, "Are women the more empathetic gender? The effects of gender role expectations," *Current Psychology 42*: 2023, DOI: 10.1007/s12144-020-01260-8, p. 220-231.

[633] Simon Baron-Cohen, *The Essential Difference: Men, Women, and the Extreme Male Brain,* Allen Lane (London): 2003.

[634] *The Guardian* in particular had a highly positive review. David Adam, "His 'n' hers," *The Guardian,* 16 May 2003, URL: theguardian.com/books/2003/may/17/featuresreviews.guardianreview6. The review that appeared in *Cognitive Neuropsychiatry* politely disagreed with Baron-Cohen's thesis being all-encompassing but largely seemed to appreciate attention and money being drawn to autism research: H. D. Ellis, "Book Review," *Cognitive Neuropsychiatry 10*(1): 2005, DOI: 10.1080/13546800344000273, p. 73-75. Contrast the review appearing in *Phenomenology and the Cognitive Sciences,* which argued instead that Baron-Cohen's reasoning was sloppy and his fundamentals were unsound due to a flawed idea of intelligence. Niel Levy, "Book Review: Understanding Blindness," *Phenomenology and the Cognitive Sciences 3*: 2004, URL: philarchive.org/rec/LEVBRU, p. 315-324.

books for lay audiences.[635] Even without that book, however, books like *Brain Sex: The Real Difference Between Men and Women*[636] as well as the books *The Female Brain* and *The Male Brain*[637] also suggested to the public that there were inborn male and female differences in the brain and in behavior—and, crucially, suggested to the public what those differences were.[638] The trouble, given the reflexive nature of gendered/sexed behaviors, is that the success of these books may undermine the inherent nature of the very differences they claim to characterize. The more people knew about these perhaps-inherent sex differences before research began, the less that straightforward research into these perhaps-inherent sex differences can be trusted to reflect the biological rather than the social.

Unfortunately, unlike the bank example above, one cannot "look at the books," as it were, in order to determine what the "real" population differences in behavior between sexes/genders might have been if these books had never been published. Regardless, however, the sex/gender differences described in these books may exist and may even be meaningful on a population level, but the research is untrustworthy in the most literal sense—it cannot be trusted to reflect

[635] Simon Baron-Cohen, *Zero Degrees of Empathy: A New Theory of Human Cruelty*, Allen Lane (London): 2011; Simon Baron-Cohen, *The Pattern Seekers: A New Theory of Human Invention*, Allen Lane (London): 2020; Peter Myers, Sally Wheelright, and Simon Baron-Cohen, *An Exact Mind: An Artist with Asperger Syndrome*, Jessica Kingsley Publishers (London): 2004.
[636] Anne Moir and David Jessel, *Brain Sex: The Real Difference Between Men and Women*, Harmony: 1989.
[637] Louann Brizendine, *The Female Brain*, Broadway Books: 2006; Louann Brizendine, *The Male Brain: A Breakthrough Understanding of How Men and Boys Think*, Harmony: 2011.
[638] There is also *The Man Who Would Be Queen: The Science of Gender-Bending and Transsexualism* by J. Michael Bailey, Joseph Henry Press: 2003, a book which, for the first time, brought sustained attention to Ray Blanchard's typology of transgender women. Ray Blanchard, another figure represented in the literature for this review, suggested that transgender women were either (1) fundamentally male homosexuals taken to a natural conclusion of femininity or (2) autogynephiles, or those who possessed a sexual interest in having a female body, who are often "really" straight men (transgender lesbians). This typology is inaccurate and poorly reflects the range of transgender women/nonbinary women-adjacent people. While much criticism has (for good reasons) focused on the flaws of autogynephilia as a description, it is worth noting that (1) posits a sort of femininity event horizon, where sufficiently feminine-typed sexuality (androphilic) and feminine behavior leads to wanting to be a woman or maybe just being one. As a conclusion of organizational development of male and female brains, however, (1) follows. The androgen that was organizing a male brain and male external phenotype did not fully masculinize the brain, and so men ended up with female-typed behaviors and/or identities: homosexuality, femininity, and transgender women.

reality. As noted above, gendered/sexed effects can appear and disappear when the salience of gender/sex in the experimental context is manipulated. Organizational/activational research, however, repeatedly failed to consider social or historical explanations in various contexts for at least two decades. By neglecting the social elements of sex/gender in favor of biological explanations, the actual salience of social beliefs about sex and gender in the existing research is unknown and unknowable. Any research studying humans that seeks to bypass rather than account for social factors will be the same type of untrustworthy for the same types of reasons.

So, to return to Latour's description of the "interruptions," the scientist cannot depart the social world and arrive at the objective world when studying human behavior or the aspects of human existence which are affected by human behavior.[639] The other interruption is when the scientist returns to the social world with the facts and laws from the objective world—a characterization that many neuroscientists would seemingly agree with. A 2016 article in *eNeuro,* one of a handful sprinkled throughout the pages of the journals of the Society for Neuroscience,[640] attempted to convince the scientist-readers of the journal to become science communicators as well.[641] Despite acknowledging that knowledge can be formed through "discovery, creation, [and] synthesis"[642] and suggesting "conversation with society at large",[643] the author's idea of conversation is a one-way enterprise of knowledge transfer: "sharing",[644] in its kinder form, and "dissemination"[645] elsewhere. "[T]he real questions are how will they know

[639] E.g. rates of lung cancer, which are not a human behavior but frequently lie downstream of smoking rates and workplace hazards.
[640] Another example: David M. Eagleman, "Why Public Dissemination of Science Matters: A Manifesto," *Journal of Neuroscience 33*(30): 24 July 2013, DOI: 10.1523/JNEUROSCI.2556-13.2013, p. 12147-12149.
[641] E. Paul Zehr, "With Great Power Comes Great Responsibility—A Personal Philosophy for Communicating Science in Society." *eNeuro 3*(5): 1 September 2016, DOI: 10.1523/ENEURO.0200-16.2016, p. 1-6.
[642] Ibid., p. 5.
[643] Ibid.
[644] Ibid., p. 6.
[645] Ibid., p. 5.

it [scientific knowledge], what is the medium through which they are ready to know it, and how do I translate the scientific messages into a comfortable message for them?"[646]

The author later quotes the first comic book in which the character Peter Parker or Spiderman ("Spider-Man", in 1964) appeared. What is notable about that example is that Peter Parker fails to act and then his uncle is killed by a criminal that Spiderman could have stopped.[647] The comic ends with the words "with great power there must also come—great responsibility!", the message being that the moral—superheroic, even—act when one has the power to help others is to do so. The author synthesizes that phrase with a paraphrase of Frances Bacon, "knowledge is power," as follows:

> *"It is time to accept that our efforts to create and generate scientific knowledge put great power into our hands. This also obliges us to exercise great responsibility. For that knowledge to have any value, it is our responsibility to affect the largest audience by communicating as widely as we possibly can."*[648]

If knowledge is power, and with great power comes great responsibility, to conclude that responsibility is measured by affecting as many people as possible requires the assumption that one is spreading good, true, useful knowledge—what might in Greek be the *euangelion*, the good news, that, through its translation of *gōdspel*, eventually became the word "gospel".

It would be reductive to claim that this advocate of science communication believes in the manner that an evangelist for a religion believes, and that is not the point of this comparison. The aspect that is shared, however, is the assumption that what is being communicated is right, true, and good—that giving the knowledge (the news) to another is inherently to bestow a boon

[646] Ibid., p. 6.
[647] Or, at least, that is what the character of Peter Parker believes.
[648] Zehr, "With Great Power Comes Great Responsibility," p. 6.

on them. The beneficence of the scientist's actions is assumed. By implication, then, this one-way communication of knowledge is the only type of responsibility that scientists have that they do not fulfill. Alternatively, if their knowledge of the natural world is imperfect, it is better than (more true and real than) anything in the social world and thereby still a boon. The social world cannot educate the scientist, and to listen would jeopardize the scientific entry to the natural world. The social world might be spoken to, but listening to it or understanding it is unnecessary.

This is the flawed foundation: there exists a social world and a natural world; the natural world is external to and unaffected by the social world, but not *vice versa*, and the scientist can speak for the natural world to the social world. From this assumption, all else follows. What should the scientist know to speak about the nature of the world or of the people and creatures in it? Is it scientists' responsibility to learn from the past? Hormones being the central explanatory factor of both mental illnesses and sex-typed development has been proposed and argued for and used in medical practice before, in the 1920s through 1950s, with many of the same basic fallacies, overconfident predictions, and overemphasis of sex-as-predictor that appeared in the literature used for this thesis.[649] Hundreds of papers in the literature covered in this thesis implicitly or explicitly recommended using sex as a predictor[650] or hormones as a treatment[651] in

[649] Hirschbein, "Sex and gender in psychiatry: a view from history."
[650] One of the most straightforward of these is: "it's pretty difficult to find any single factor that's more predictive for some of these diseases than gender." This quote from Thomas Insel, head of the US National Institute of Mental Health in 2005 speaking to *Science*, was requoted in 2014 by the editorial introduction to an issue of *Frontiers in Neuroendocrinology* focusing on "Sex differences in neurological and psychiatric disorders." Constance Holden, "Sex and the Suffering Brain," *Science 308*(5728): June 2005, DOI: 10.1126/science.308.5728.1574, p. 1574. Young and Pfaff, "Sex differences in neurological and psychiatric disorders," p. 253.
[651] The possible treatments were for, among other things, lack of sexual desire, multiple sclerosis, Turner syndrome, and cognitive decline during aging. Even when randomized controlled trials suggested that estrogens and progesterone have no particular benefits for post-menopausal women and may cause harm, researchers simply modified their recommendations to emphasize that treatment should be started *at* menopause. Jill M. Daniel, "Estrogens, estrogen receptors, and female cognitive aging: The impact of timing," *Hormones and Behavior 63*(2): February 2013, DOI: 10.1016/j.yhbeh.2012.05.003, p. 231-237.

medical practice, and only one paper mentioned this previous attempt at all.[652] When, and how, and to whom is it the scientists' responsibility to listen? What power, or knowledge, do the scientists actually possess—the power to comprehend the world, or the knowledge to create the world, or both, or neither?

4.6: Conclusion

> *"[W]e shall be told calmly that one must be very careful 'not to mix the sublime epistemological questions'—on the nature of things—'with the lowly political questions'—on values and the difficulty of living together. It's really so simple! If you try to loosen the trap by shaking it, it will close more tightly still, since you will be accused of seeking to 'confuse' political questions with cognitive ones! ... That you are abandoning all criteria for judging what is true and what is false! ... Those who have split public life into Science and society through a sophism are going to accuse you of sophistry! You will die of hunger or suffocation before you have gnawed through the bars of the prison in which you freely locked yourself up."*[653]

What is an assumption? An assumption is a belief that evidence has no bearing on. It lives both above proof and below disproof—it is so evident it requires no evidence, and individual contradictions cannot budge it from its foundational position. Latour's frustrated description, above, of attempting to engage with such unjustified and yet inaccessible beliefs, in

[652] Maurand Cappelletti and Kim Wallen, "Increasing women's sexual desire: The comparative effectiveness of estrogens and androgens," *Hormones and Behavior 78*: February 2016, DOI: 10.1016/j.yhbeh.2015.11.003, p. 178-193. I would like to point out that Cappelletti and Wallen suggest only that supraphysiological doses of hormones in women would not increase sex drive, rather than the framework of hormones as a treatment being flawed. Also, this one paper's mention is as many times as the Hippocratic theory of the humors is mentioned in the literature reviewed for this thesis. Bos *et al.,* "Acute effects of steroid hormones and neuropeptides on human social–emotional behavior," p. 17, 31.

[653] Latour, *Politics of Nature,* p. 15-16.

addition to the amusement and catharsis it contains,[654] may explain why the assumptions persist. To operate without them requires explanation; to argue with them directly, particularly by bringing in alternative forms of evidence, is often a frustrating and tedious task. New types of evidence often need to be justified, vocabulary may need to be borrowed, invented, or defined, and at its most extreme, new theoretical structures may need to be invented in order to engage with the assumption at all—and after all that, only some of the criticism that will meet these new ideas will be reasoned or reasonable.

However, while each of these sections has been about assumptions, this chapter has also been about consequences. These conclusions and analyses deny that deviations from the norm happen naturally (§4.1), provide a view of human existence that is at best severely limited and at worst implicitly denies past destruction (§4.2), generally work backward from present conditions and justify the status quo (§4.3), then build on these flawed foundations to synthesize new theories that are doomed from inception (§4.4), and finally propagate these theories to a society which is listening (and might fulfill or nullify the prophecy) but which will not be listened to or understood (§4.5). Medicine and science take place in a world that is political. Attempts to ignore that reality will take a position, frequently adopting those positions for which invisibility is an advantage—white supremacy, patriarchy, neo-colonialism, cis- and heteronormativity, and so on. Returning to Latour once more, at no point in recorded memory were the questions "on the nature of things" and those "on values and the difficulty of living together" actually unmixed. That assumption was the fault line, and nothing built on it was ever sound.

[654] Catharsis for me, at least.

Chapter 5: Conclusion

5.0: Introduction

"It is time to explain myself—let us stand up."[655]

Between 2000 and 2019, the organizational/activational theory went from ascendant to a crumbled edifice. The final blows were the replication crisis, the immune research which proved without a doubt that gonadal hormones could not be the only actor in sex differentiation, and Joel *et al.*'s neuroscientific study which failed to find consistent structures in male versus female brains which reliably distinguished the two, arriving at the end of 2015 and the beginning of 2016 as topics in the journals covered in this thesis. Chapter 2 described the accumulation of contradictory facts that eventually eroded organizational/activational theory's foundational assumptions. Next, chapter 3 went into the varied statistical and methodological issues which, prior to the replication crisis, likely prolonged the organizational/activational theory's relevance. Finally, chapter 4 covered the foundational assumptions which obscured certain types of social evidence from being known at all. This concluding chapter will attempt to consider what the trajectory of the organizational/activational theory might suggest about knowledge and knowers, as well as how people come to know things through communication and the complexity of justified beliefs.

5.1: Situated assumptions and partial knowledge

"*The basis of any discipline is not the answers it gets, but the questions it asks. As an exercise in the anthropology of knowledge, this paper stems from asking a simple*

[655] Walt Whitman, "Song of Myself" (1892 Version), section 44. This is when in the 52-part poem of himself that Whitman begins to end. The lines after this that I quite enjoy are "Do I contradict myself?/Very well then I contradict myself,/(I am large, I contain multitudes.)" and "I too am not a bit tamed, I too am untranslatable,/I sound my barbaric yawp over the roofs of the world."

> *question: what were the females doing while the males were out hunting? It was only possible to ask this question after I had become politically conscious of myself as a woman. … Though there have been women anthropologists for years, it is rare to be able to discern any difference between their work and that of male anthropologists. Learning to be an anthropologist has involved learning to think from a male perspective, so it should not be surprising that women have asked the same kinds of questions as men. But political consciousness … leads to reexamination and reevaluation of taken-for-granted assumptions."*[656]

Scientific theories are underdetermined by the information available.[657] While scientific theories will be consistent with observations, available evidence, and logic,[658] the theories that become accepted require background assumptions about the world and the measurement apparatuses involved, as well as assumptions about what cognitive values—simplicity, accuracy, scope, fruitfulness, consistency[659]—can or should decide between competing theories. I would argue, however, that not all sciences and theories are equally underdetermined. Behavioral neuroendocrinology cannot, for ethical reasons, run many experiments which would allow

[656] Sally Slocum, "Woman the Gatherer: Male Bias in Anthropology" in *Toward an Anthropology of Women,* ed. Rayna R. Reiter, Monthly Review Press (New York): 1975, p. 49.
[657] Helen Longino, "Chapter 7: How Values Can Be Good for Science," in *Science, Values and Objectivity*, eds. Peter Machamer and Gereon Wolters, University of Pittsburgh Press (Pittsburgh): 2004, p. 131-135. Accessed through ProQuest Ebook Central.
[658] On the assumption here that there are no distorting factors. One might recall Lysenko's hold on the USSR's biological sciences for a rather straightforward counterexample, or nineteenth-century craniometry for a slightly more complex example. While these might fall under pseudoscience instead, I am hesitant to name them so here because "science" as experienced in the moment is governed by what different scientific experts and authorities endorse—that is the purpose of having expertise or authority. That this type of science is pseudoscience may be stated by individuals in the moment but is in general only collectively understood as such in hindsight.
[659] Thomas Kuhn's list of epistemic values that are decided between. Mauro Dorato, "Chapter 3: Epistemic and Nonepistemic Values in Science," in *Science, Values and Objectivity*, eds. Peter Machamer and Gereon Wolters, University of Pittsburgh Press (Pittsburgh): 2004, p. 58. Accessed through ProQuest Ebook Central. Compare Longino's alternative feminist cognitive values from Helen E. Longino, "Cognitive and Non-Cognitive Values in Science: Rethinking the Dichotomy," in *Feminism, Science, and the Philosophy of Science, Volume 256*, eds. Lynn Hankinson Nelson and Jack Nelson, Kluwer Academic Publishers (Dordecht): 1996, p. 39-58.

researchers to more effectively investigate the processes involved; those investigating the biological and developmental origins of human behaviors must therefore apply more assumptions than those investigating geology or organic chemistry.

Those who study the origins of sex differences through a biological lens have another set of assumptions to deal with. When considering how unjustified assumptions might enter research contexts, it can be tempting to concentrate on the active choices scientists make—to pursue avenues of investigation, to decide between theories, to determine what is and is not evidence, and to attempt to apply their research to specific societal, practical, or political ends.[660] From a wider perspective, however, scientists are not merely the active choices they make, but embodied knowers[661] working in fundamentally social systems of learning, peer review, and communication.[662] About sex differences specifically, it would be at best implausible to state that any researcher or scientist has had no exposure to societal beliefs about sex and gender (and sex/gender) from their earliest days of life onwards, applied to both themselves and others.

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[660] Dorato, "Epistemic and Nonepistemic Values in Science." There are a few reasons I do not particularly agree with this philosopher's perspective, nor do I agree that the author has demonstrated that there are "no serious objection[s] to the neutrality of the cognitive content of scientific hypotheses." p. 75. In particular, categories which have some biological elements, like race/ethnicity, sex/gender, sexuality, neurodivergence, and disability, have historically been biologically and socially enmeshed categories that have, again historically, been assumed to be biologically relevant until proven otherwise. It is all well and good to "claim that whenever nonepistemic values are the essential factor determining the acceptance of a hypothesis, we are facing an instance of *poor* science or of an *unreliable* piece of knowledge," (p. 57, italics in original) but in this case, science could not address these issues for the majority of the nineteenth and twentieth centuries at minimum for all five above categories (if not into the eighteenth and twenty-first centuries as well). When the poor science is acceptable to the status quo or the powerful or when unreliable knowledge should incline the scientists to refrain from theorizing, science is or can be unreliable for extended periods and scientists do not consistently recognize science's unreliability at the time. As time goes to infinity, cognitive content in scientific hypotheses may go to 100%; however, given that humans generally live less than a century and science needed at least two to begin to dispose of its prejudices, that mismatched timescale remains a "serious objection".

[661] Donna Haraway, "Situated Knowledges: The Science Question in Feminism and the Privilege of Partial Perspective," *Feminist Studies 14*, no.3: Autumn 1988, DOI: 10.2307/3178066, p. 575-599.

[662] Peter Machamer and Lisa Osbeck, "The Social in the Epistemic," in *Science, Values and Objectivity*, eds. Peter Machamer and Gereon Wolters, University of Pittsburgh Press (Pittsburgh): 2004, p. 78-90. Accessed through ProQuest Ebook Central.

Indeed, one might note that the scientists researching these topics are very likely to have lived within the same, or highly similar, social contexts of sex and gender, largely being from normative (heterosexual, cisgender, able-bodied, neurotypical, etc.) and Western contexts in one historical moment. If fallacies emerge from failing to consider alternative explanations, as the editor of *eNeuro* suggested,[663] the perspectives that the researchers possess will limit the number of explanations that can be conceived: fewer types of perspectives, fewer alternatives, and more fallacies. "[A]lthough the traditional definition of *objectivity* has been integrally bound to truth, the operationalizing of this concept has always had a social or collective dimension. That is, what any group takes to be objective is that on which there has been sufficient intersubjective agreement among observers (see, for example, Kerlinger 1986)[664]."[665] Collectively, the fewer different standpoints—identity-based, framework-based, knowledge-based, and socially-based—that are present in the group, the fewer types of criticism can be made and the less likely certain types of error will be found.

To return to the idea of embodiment, Haraway spoke about seeing from "[t]he standpoints of the subjugated," and suggested that these standpoints "are knowledgeable of modes of denial through repression, forgetting, and disappearing acts".[666] It is at this point I should own up to my own partial perspectives, or at least the ones I can.[667] My own experience in research and writing this thesis has been one of running headlong into the assumptions made

[663] Bernard, "On Fallacies in Neuroscience."
[664] Fred N. Kerlinger, *Foundations of Behavioral Research*, 3rd ed., Holt, Rinehart, and Winston (New York): 1986.
[665] Machamer and Osbeck, "The Social in the Epistemic," p. 88.
[666] Haraway, "Situated Knowledges," p. 584.
[667] I am bisexual, non-gender conforming, a woman, experienced abuse, and was diagnosed with an emotional disorder rather than a learning disorder in part based on my female phenotype and so prevented from accessing medications, assistance, and self-understanding for much of my life. Being neurodivergent, bisexual, non-gender conforming, a woman, and a survivor of trauma made certain assumptions in the literature evident by the mere fact that I did not share those assumptions, not helped by the fact that the researchers' assumptions were additionally contradicted or undercut by my own experiences.

about people like me or those I have known. For example, my standpoint of having once been a preteen girl with an early but not precocious puberty who knew other pre-teenage girls allowed me to wonder at the ignorance displayed by the researchers about why depression rates for girls but not boys sharply increased during puberty, particularly for those with early puberties. Depressive symptoms seem a fairly reasonable response to sexual harassment or sexually charged remarks from adults and then other children[668] and an absence of social support surrounding menstruation, let alone the effects of puberty with more than the usual feelings of isolation due to earlier timing or compounded stressors. Being a teenage boy also seemed stressful, but it, at least apparently, came with few additional restrictions and more freedoms; being a teenage girl came with fewer freedoms and more restrictions.[669] The researchers of these papers are not more or less situated than I am, when I emphasize the social experiences of going through puberty for those assigned female at birth as opposed to the hormonal changes of going

[668] A quick and non-exhaustive list of my own examples: a family friend suggested I'd get quite a lot of tips if I worked as a waitress wearing such a short skirt (a skirted bathing suit worn among family and family friends) at age 11; cat-called at ages 10 and 13 in Philadelphia and New York City, respectively; my pediatrician looked me up and down and loudly muttered "jailbait" at age 14. Most of those girls I knew had heard of the 'trick' to call friends if you were being followed and how to hold keys between your fingers as makeshift 'claws' by age 13 at the latest. Messaging from older men and women tended to emphasize that men and boys were fundamentally untrustworthy and would sexually assault girls and women given the slightest chance—but also often gave no assistance or comfort to anyone who had been assaulted. I find it difficult to overstate the isolating effect of these aspects in combination, as half of one's fellow humans (nearly all those assigned male at birth) were designated threatening, while the other half (assigned female at birth) may or may not have had these types of experiences or understood them in the same ways. The elevated "depression" rate seemed less of a mental illness within the girls triggered by circumstance and more an eminently understandable reaction of the girls to stressful circumstances beyond their control in multiple ways, and that no researchers so much as considered the possibility suggested that these types of experiences were invisible to them.

[669] Part of the isolation explained above is that people, institutions, places, and times would become unsafe. For example, while teenagers were both discouraged from being out late, boys were discouraged because they were believed to *create* trouble; girls were believed to find and be victims of trouble. There is also the aspect of being assigned female at birth once expressed by Adrienne Rich, in her poem "Natural Resources": "4. *Could you imagine a world of women only,/*the interviewer asked. *Can you imagine//a world where women are absent.* (He believed/he was joking.) Yet I have to imagine//at one and the same moment, both. Because/I live in both. *Can you imagine,*//the interviewer asked, *a world of men?*/(He thought he was joking.) *If so, then,//a world where men are absent?/*Absently, wearily, I answered: Yes." *The Fact of a Doorframe: Poems Selected and New 1950-1984*, W. W. Norton (New York): 1984, p. 258.

through puberty for those assigned female at birth; however, I would suggest that any discussion of the increased rates of depressive symptoms in girls versus boys at puberty that did not consider at least *both* of these two types of influences would be woefully incomplete.

In addition, I wish to highlight another aspect of having a situated perspective that is less about "I am" and more about "I have learned" or "I know others who are or know". My undergraduate degrees were in molecular biology, which gave me the context to question some of the material and methodological assumptions, and twentieth century history, which allowed me to question some of the underlying assumptions about the nature of the modern social world, such as the universality and naturality of the nuclear family—all aside from getting a degree in the history and philosophy of science, which gave me other contexts to use. Among my family members and friends are a computer scientist, an active member of the disability community, a public health worker, a number of LGBTQIA+ individuals, an entomologist, an archeologist, a historian, a handful of engineers, as well as a few others, and their understandings and perspectives were invaluable in the process of writing this thesis. As Slocum declared about anthropology and which describes many human endeavors, "It is our task … to create a 'study of the human species' in spite of, or perhaps because of, or maybe even by means of, our individual biases and unique perspectives."[670]

As mentioned in [section]4.3, there were only few exceptions to the general non-engagement with social factors, falling into two broad cases: those who participated in the medical system directly, or those individual cases who came in with or sought out information and contacts external to their research field. One neuroscientist noted that, at least in their experience and the experiences of those they asked, that junior scientists in particular were

[670] Slocum, "Woman the Gatherer," p. 50.

encouraged not to communicate in non-traditional outlets—and this same neuroscientist noted that they benefited from public outreach "by establishing collaborations with colleagues outside of my immediate field".[671] The general picture of behavioral neuroendocrinology—and neuroscience as a whole—is of an insular field with few contacts with other natural sciences, let alone more distant disciplines like the social sciences or humanities. This thesis did not generate new criticisms but imported existing criticisms from other fields—statistics, computer science, sociology, evolutionary biology, molecular biology, etc.—which would have been significantly less novel if these fields were in consistent and meaningful contact. The statistical criticisms were, at some length,[672] imported into neuroscientific contexts, but few other distinct perspectives were incorporated into neuroscience or behavioral neuroendocrinology during this period.[673]

Another way to think about the privilege of partial perspective and standpoint epistemologies is in terms of time. Part of the reason the general lack of interdisciplinary contacts in behavioral neuroendocrinology and neuroscience broadly is concerning is that no one person can know everything, nor should any researcher individually be expected to find all the

[671] Susana Martinez-Conde, "Has Contemporary Academia Outgrown the Carl Sagan Effect?" *Journal of Neuroscience 36*(7): 17 February 2016, DOI: 10.1523/JNEUROSCI.0086-16.2016, p. 2080.

[672] While most modern discussions of statistical meta-scientific research begin with Ioannidis' paper in 2005, John P. A. Ioannidis, "Why Most Published Research Findings Are False," *PLOS Medicine 2*(8), e124: 2005, DOI: 10.1371/journal.pmed.0020124, the authors in *Journal of the American Medical Association* in 1966 flagged the improper use of statistical methods in medical research and its effect on that research in the longer term. Stanley Schor and Irving Karten, "Statistical Evaluation of Medical Journal Manuscripts," *Journal of the American Medical Association 195*(13): 1966, DOI: 10.1001/jama.1966.03100130097026, p. 1123-1128.

[673] Besides the previously discussed papers, one of the other exceptions was an interdisciplinary collaboration between some neuroendocrinologists and cancer researchers, which forced the neuroendocrinologists to take non-gonadal, local hormone synthesis seriously as some cancers, particularly prostate cancer, could not be understood without it. It is worth reminding the reader that this thesis's primary source documents were 729 scientific papers and that I can not only recall the interdisciplinary papers specifically, I can count them on two hands. H. Bobby Fokidis, Hans H. Adomat, Geetanjali Kharmate, Elham Hosseini-Beheshti, Emma S. Guns, and Kiran K. Soma, "Regulation of local steroidogenesis in the brain and in prostate cancer: Lessons learned from interdisciplinary collaboration," *Frontiers in Neuroendocrinology 36*: January 2015, DOI: 10.1016/j.yfrne.2014.08.005, p. 108-129.

issues in their own research, much less the research of everyone else. While no individual could or should be expected to know everything, the group should at least know *of* all the types of knowledge the world possesses.[674] However, even this bare bones understanding requires time—time to understand what knowledge is out there, time to understand what knowledge you possess, and time to bridge different viewpoints. Is there knowledge limited to individuals, or types of lives? Philosophically, that may or may not be true.[675] Practically, however, one would need several lifetimes to learn and unlearn the assumptions and reflexes of each perspective before moving on to the next—a difficult task, when one's own perspective is generally no small amount of trouble to comprehend.[676] That task would also be fundamentally Sisyphean, as new standpoints would always be created, contemporary standpoints translated, and old standpoints recovered from the past. Becoming an expert in all subjects, too, would be an impossible endeavor. It is far easier, instead, to learn how to listen and how to ask, to understand one's own blind spots, by proxy if necessary, and to understand the value of admitting ignorance publicly.[677]

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[674] Presumably, the opportunity to cultivate interdisciplinary contacts or at least to understand who to speak to in order to understand a topic is part of the purpose of a university with multiple faculties over a wide range of learning. In practice, the actual usage of such resources and connections in the literature covered by this thesis is inconsistent and minimal.

[675] Either answer is rather strange to consider, but perhaps it operates in gradations, that some knowledge is easier or more difficult to access from different starting standpoints. Regardless, I leave it to others to discuss this question.

[676] For example, Haraway notes that no standpoint is unproblematic or immune from questioning. "To see from below is neither easily learned nor unproblematic, even if 'we' 'naturally' inhabit the great underground terrain of subjugated knowledges. The positionings of the subjugated are not exempt from critical reexamination, decoding, deconstruction, and interpretation; that is, from both semiological and hermeneutic modes of critical inquiry. The standpoints of the subjugated are not 'innocent' positions. On the contrary, they are preferred because in principle they are least likely to allow denial of the critical and interpretive core of all knowledge." Haraway, "Situated Knowledges," p. 584.

[677] While Socrates often is paraphrased saying "I am the wisest man alive because I know only that I know nothing," none of Plato's surviving literature, at least, has Socrates saying this. Instead, as in the *Apologies* (section 21d), Socrates notes that his interlocutor believes that they know something (in this case, about goodness) but they do not actually seem to know anything after Socrates engages them in a dialogue, while Socrates knows that he does not know anything about that thing. The main point of Socrates (and Plato) is better stated as that it is wise (possessing *sophos*, *σοφός*) to know what one does not know.

5.2: Scientific noise and signaling relevance

> *"This even-handed weighing of evidence is essential to good science, where our analytic procedures must be capable of both ruling in and ruling out effects (and of reserving judgment due to an uninformative sample. … In practice, though, null hypothesis testing with* p *values is often used as though it can only demonstrate effects. This is like having a neural network that can only express LTP [long-term potentiation]; noise will eventually saturate the system.[emphasis in original]"*[678]
>
> *"Information society is verging on noise society, a state in which the information, meant to convey knowledge, ends up losing the ability to speak at all. Our culture becomes taciturn without being silent, moving towards a noisy muteness."*[679]

The introductory quote of the last section was written by an anthropologist in the 1970s, and very little of the material of that section is particularly novel—excepting, perhaps, the emphasis on time as a central reason to take standpoint epistemology seriously as a method to discover previously unquestioned assumptions. However, that lack of novelty itself points to some larger questions. Why have these criticisms not been incorporated? While this thesis has the benefit of hindsight, not all of these criticisms went unspoken at the time. However, these criticisms either required the replication crisis or significant and unmistakable contradictory evidence to *begin to be* incorporated or remained unacknowledged within the literature as of 2019. For statistics in particular, the answer appears to be that mentors and other senior scientists had been passing on incorrect information about p-values, specifically that a low p-value was

[678] Calin-Jageman and Cumming, "Estimation for Better Inference in Neuroscience," p. 7.
[679] Torbin Sangild, "The Aesthetics of Noise," ed. Pelle Krøgholt, Datanom: 2002, URL: ubu.com/papers/noise.html.

definitive while high p-values required explanation.[680] More broadly, however, this thesis has been supporting the claim that requiring research to be relevant or rewarding researchers for relevance will inevitably cause the system to generate noise at least as fast if not faster than it generates any sort of usable information.

While this thesis has discussed many sorts of assumptions, the assumptions in general had the effect of expanding the relevance, importance, and permanence of a single system: the effect of gonadal hormones on behavior, particularly sexed/gendered behavior. Examples of this skew range from the resistance to the importance of neurosteroids and ignorance about signaling pathways, through the asserted transcendence of sex across mammals (or even all of *Chordata*), to the evolutionarily selected and psychiatrically actionable histories of sexed/gendered cognitions. The assumptions and their deployments in introductions, discussion sections, and conclusions often functioned as assertions of applicability for the research itself. It is highly improbable that almost all basic research in any field is truly applicable in the relatively near term—and if it is, that it is applicable to the problems that the researchers believed it applicable to. While I suspect the reasons for this insistence on relevance, understood as applicability or importance to present problems, emerge primarily from funding and employment pressures and structural incentives,[681] it is enough to note that these pressures and incentives are encouraging researchers to give similar signals of relevance.

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[680] Calin-Jageman and Cumming, "Estimation for Better Inference in Neuroscience," p. 7; also, see §3.6.

[681] For a positive opinion which emphasizes working with existing healthcare networks, see Cecilia Vindrola-Padros, "Can We Re-Imagine Research So It Is Timely, Relevant and Responsive? Comment on 'Experience of Health Leadership in Partnering with University-Based Researchers in Canada: A Call to 'Re-Imagine' Research'," *International Journal of Health Policy and Management 10*(3): 2021, DOI: 10.34172/ijhpm.2020.43, p. 172-175; for a negative opinion which emphasizes the incorrectness of applied research as a concept, particularly in biology, David Botstein, "Why we need more basic biology research, not less," *Molecular Biology of the Cell 23*, no. 21: 2012, DOI: 10.1091/mbc.E12-05-0406, p. 4160-4161. For the less obvious benefits of applicability, one might consider the repeated emphasis placed on science communication and the external benefits of having one's research

The presence of a signal, however, is made meaningful by the fact that it is not always present. Otherwise, it is merely a characteristic of the group.[682] Very few of the researchers in this literature stated that their research was fundamentally flawed due to an issue discovered after research had begun, or that their research would perhaps never be applicable to humans, or that the effects discovered were so vanishingly small as to be irrelevant to most situations—the main exception being those papers which failed to confirm an existing effect attested in the research. Not all research will be relevant, and unless researchers can be honest that they have no reason to believe their research will be relevant or admit after the fact that they were incorrect that their research will be relevant to the question at hand (and not receive a negative consequence), the signal of relevance in research will trend towards meaninglessness over time because all researchers will seek to ensure the presence of the signal. The replication crisis was precipitated by statistical failings, but it was also fueled by an arms race of relevance and importance between researchers and between journals. If research *as a whole* is to be applicable, individual researchers cannot expect to discover anything of note—but unless individual researchers are no longer judged by their individual impact, discovering which pieces of research are applicable will remain a laborious and expensive process of discarding false leads.

5.3: Collecting facts and collective ignorance

"Which would be better, what sticks or what falls through?

promulgated to a lay audience, like speaking engagements, books, funding, a raised profile in the university setting, etc.

[682] In biology, this signal to meaningfulness question is raised by creatures which have evolved to appear to be poisonous, venomous, or otherwise unpleasant by mimicking the poisonous, venomous, or unpleasant animal but are generally harmless and palatable. Examples include the clearwing moths, which are bee and wasp mimics, the scarlet kingsnake, which mimics the venomous coral snake, and the common hawk-cuckoo (or brainfever bird) which mimics the shikra, or little banded goshawk. Such a tactic only works because other sources of food for the predators they seek to avoid are available; otherwise, either the predator would be selected to accurately distinguish between the dangerous animal and the mimic, or the dangerous animal would be selected to further distinguish itself from the mimic—both changes which would expose the mimic to predation.

Or does the choice itself create the value?"[683]

"é seo go léir a thabhairt faoi ndeara is áit a dhéanamh dó id' chroí gan pléascadh

é seo uile is an móta Normannach a chonac is mé ag gabháil na slí,

áit éigen faoin dtuath in aice le Cill Mhhaighneann i gContae na Mí,

An Obair. Sin í an obair. Sin í an obair nach éasca.

--

to take it all in, to make room in your heart without having your heart burst,

to take in not only this but that Norman motte and bailey

I passed near Kilmainham or thereabouts,

a place called Nobber. That's the task. An obair. *A task that's far from easy."*[684]

One of the Irish phrases for "to know" is "*a fhios a bheith ag*", or "the knowledge is on (possessed by)". Rather than using the English word "know", which simultaneously implies perception,[685] comprehension,[686] and recognition,[687] it may be more useful to consider beliefs, of which knowledge (as justified true beliefs)[688] is a subset, as things which must be collected and held onto. This adjusted perspective on knowing can rather easily be translated into assumptions—those beliefs carried without being justified and often without even the awareness that they are being carried—as well as standpoints—the accumulated experiences and studies of a lifetime, held on to. Understanding knowing as requiring the collection of beliefs, however, can

683 Seamus Heaney, "The Riddle," *The Haw Lantern*, Faber and Faber (London): 1987, p. 51.
684 Nuala Ní Dhomhnaill, "An Obair/The Task," *The Fifty Minute Mermaid*, translated by Paul Muldoon, Gallery Books (Loughcrew): 2007, p. 22-23.
685 For example: "he knew something was not quite right", "she knows about her parents' divorce."
686 For example: "they knew French and Dutch", "physician, know thyself."
687 For example: "I've never met your father, but I know your mother", "humans know right from wrong" (recognize right and recognize wrong and can distinguish the two), "he knew his old shipmate even through the years that had passed" (he recognized a friend after years apart).
688 Gettier cases will be discussed in the next section.

also clarify some of the other evidential issues this thesis engaged with. The methodological issues covered in chapter 3 artificially limited the evidence available to be collected, and so skewed the possible conclusions that one could make with the evidence available; the failings of researchers to adequately engage with other types of evidence covered in chapter 4 amounts to a biased collection of some types of evidence and not others and generally without their context, which skewed the possible conclusions again. In addition, it is useful to consider the point brought up in [section]5.2 about noise. Traditionally, it is darkness which is seen as the enemy of knowing—but light, too, can blind. What does it mean to try and collect knowledge if one is drowning in facts and assertions and arguments, each contributing to a crescendo of static in which only the loudest and most dramatic statements can win out, and even then only for a moment?

Before considering that question, I would like to return to chapter 2, in which I quoted researchers searching for simplicity who mourned that their investigation of immune differentiation in the brain disproved that hormone exposure was 'enough', either between individuals or within one individual, to explain all sex differentiable factors in "a sort of 'Unified Field Theory' of sex differences in the brain. But no matter how desirable such a scenario might be, it isn't true."[689] Simplicity, however, is not a cognitive value which is valuable no matter its degree. While certainly researchers can 'overfit' explanations to data, oversimplification is no less a failure of explanation. When discussing humans in a collective sense, the history of prejudices and stereotypes suggests that oversimplification is the more present danger. One might also ask what "simplicity" is in relation to—ideally, the prior beliefs of the researchers and

[689] McCarthy *et al.*, "Surprising origins of sex differences in the brain," p. 6.

the state of the world should be at least roughly aligned, but in practice such things vary.[690] The research on sex/gender and sexuality covered for this thesis, in particular, was overburdened with self-fulfilling prophecies and unjustifiable assumptions, from which the 'simplest' explanations tended to rely on conflations of male-masculine-man-gynephilic-dominant and female-feminine-woman-androphilic-submissive.

Noise, or more properly the presence of an overwhelming number of signals, poses a particular problem for knowers. To go on a brief tangent, those who 'hunt' for evidence of ghosts often use technologies to see the unseen world[691]—often ones which either can be manipulated to give fraudulent results, as in spirit photography,[692] or those which can be read into, like the radio static in which snatches of speech can be formed into the words of the dead—described by one scholar as "Rorschach audio".[693] When any conclusion can be generated from the available evidence, the noise will be sifted through—but, when done using preexisting assumptions, the eventual conclusion reached will be as much a reflection of the knower as the knowable. To return to Latour, the social and scientific cannot be separated in part because that separation allows those who study, use, and practice science to forget that scientists cannot truly leave

[690] Longino, "Cognitive and Non-Cognitive Values in Science: Rethinking the Dichotomy," p. 52-53. See also her discussion of "External Consistency or Conservatism vs. Novelty", p. 51-52, which I to some degree consider below when I discuss "entanglement."

[691] Colin Dickey, "The Broken Technology of Ghost Hunting," *The Atlantic*, 14 November 2016, URL: theatlantic.com/science/archive/2016/11/the-broken-technology-of-ghost-hunting/506627/.

[692] Nancy M. West, "Camera Fiends: Early Photography, Death, and the Supernatural," *The Centennial Review 40*, no. 1: Winter 1996, URL: jstor.org/stable/23740730, p. 170-206. The spirit photographs made by their first creator, Mumler, in the nineteenth century are, most likely, some variety of double exposure—though the details remain, and will likely forever remain, unknown. All technologies of investigation, on some level, promise to show things as they are, not as they appear to be to our limited human faculties—"the camera never lies"—but trick photography proves that though the camera might faithfully reproduce what it sees, the photographer has no such restrictions.

[693] Known by those who believe as the "Electronic Voice Phenomenon," with the additional alternative name "thanatophonics." Joe Banks, "Rorschach Audio: Ghost Voices and Perceptual Creativity," *Leonardo Music Journal 11*: 2001, URL: jstor.org/stable/1513432, p. 77-83; Manuel Cirauqui, "Thanatophonics: From White Noise to Forensic Radio," *PAJ: A Journal of Performance and Art 35*, no. 2: May 2013, URL: jstor.org/stable/26376128, p. 20-25.

themselves and the assumptions they carry at the door with their coats. Perception in noisy environments is fallible, and recognition even more so; it will be the beliefs that are on—held by—those thinking about a problem that will guide their answers.

To finally return to the question asked in the first paragraph, the knowers discussed in this thesis are, for the most part, acting collectively on agreed-upon beliefs. Collective knowing, when it has a lack of standpoint diversity, has not been tested as much as the collective nature of the knowledge would imply. However, it is also worth considering the idea that the base assumptions and the evidence that supports them also have a reinforcing effect on each other. Fausto-Sterling described the relationship between apparently but not truly disparate ideas as knots in a macramé weaving: "the connecting threads secure individual knots within the larger structure, even though a single knot may not be all that strong."[694] I prefer to describe the ideas as *entangled*, however, as macramé is an art while I am uncertain of the level of intent involved here. To construct this tangle, the assumptions select the evidence available to be collected; then, from a noisy environment, any beliefs may be generated or selected, and so, searching for simplicity, the ideas that require the least explanation or which fit into existing paradigms will be collected and held on to. The process then repeats. Indeed, chapter 4 was about how adjacent fields were yanked and pulled into alignment or ignored entirely based on their ability to become part of the larger, entangled theoretical structures. Contradictions fell through the system until the whole dynamic was questioned, as the replication crisis, the immune research, and the brain scans of Joel *et al.* in combination did.

Understanding knowers as maintaining and collecting their beliefs in a continuous process, affected by their starting points (intellectual and lived), can help explain why

[694] Fausto-Sterling, *Sexing the Body*, Fig. 5.6.

information was known but not used, why alternatives were published but left no trace in the collective, and why decades-old critiques can explain recent events. While belief collection *can* generate bias, I wish to re-emphasize that knowledge being a thing that might or might not be on one is also trying to consider the way that knowledge is incorporated and then used. In this case, because contradictions and alternative standpoints could not join the tangle and could also not disentangle these reinforcing assumptions, they could not be collected and join the knowledge on the researchers—unless the researchers entered with the alternative standpoint or could not, as the immune researchers and those aware of Joel *et al.* could not,[695] deny the contradictions any longer. Perception and recognition are not enough; it matters more whether the knowledge is on them when they are thinking about a problem. Do they apply their knowledge? Which types? When? How? What do they think their audience believes? (Is there a penalty for those who have to explain more to their audience?) What knowledge is collected and held onto by the people who engage in research collectively and, hopefully, collaboratively? What, in the end, governs whether researchers and scientists can even incorporate information or beliefs into their standpoints—and whether they will?

5.4: How to fool and be fooled

> *"The truth is out there, but lies are inside your head."*[696]
>
> *"The first principle is that you must not fool yourself—and you are the easiest person to fool."*[697]

[695] McCarthy *et al.,* "Surprising origins of sex differences in the brain," p. 6.
[696] Terry Pratchett, *Hogfather,* Corgi Books (UK): 2006, p. 234.
[697] Richard P. Feynman, "Cargo Cult Science", Caltech's 1974 Commencement Address. URL: calteches.library.caltech.edu/51/2/CargoCult.htm.

So far, I have left the concept of "knowledge" as being equivalent to "justified true belief". Unfortunately, there is a quite well-known tension between "justified" and "true"—specifically, the tension articulated by Gettier,[698] in that unjustified true beliefs (epistemic good luck) and justified false beliefs (epistemic bad luck) are both possible. The general pattern of a Gettier case, or epistemic (good) luck, is that the knower has advanced a justification for their belief, and that their belief is true, but the true belief does not follow from the justification, generally because some part of the justification is incorrect or does not follow.[699] That gap between justification and truth is also quite relevant to the scientific papers published during this period. The statistical issues, overstated applicability, methodological problems, and incautious use of medical and psychological terminology[700] are the gaps in the justification that make these arguments potential Gettier cases. The conclusions may be true, but this research cannot justify that belief.

Stepping back, however, a generated piece of knowledge that begins with "one may divide humans into groups" and ends with "these groups will display average differences" can be a true belief *regardless of the justification between these two statements*—for example, when

[698] Edmund L. Gettier, "Is Justified True Belief Knowledge?" *Analysis 23*, no. 6: June 1963, DOI: 10.2307/3326922, p. 121-123. See also Linda Zagzebski, "The Inescapability of Gettier Problems," *The Philosophical Quarterly 44*, no. 174: January 1994, DOI: 10.2307/2220147, p. 65-73.

[699] Say, for example, (A) that someone is traveling through the countryside and sees a number of houses dotting the landscape. (B) They suppose that the people who live there must be wealthy, as the houses are quite grand, and, having occasion to visit one of the houses, (C) believes that the people who live in that house will be extremely well-off. (A) is the initial observation, (B) is the justification, and (C) is the final belief that can be proven either true or false. In general, the fastest way to make (C) either an unjustified true belief or a justified false belief is to make the house the person finally tests their hypothesis on exceptional in the context described by (B). Either the area is awash with movie set facades and/or shady real estate scams or tax dodges and the person has visited the only inhabited building in a hundred miles (unjustified and true), or the area is generally a wealthy if fairly ordinary neighborhood, but the person has chosen a "house" which was actually built to be a museum in which no one has ever lived (justified and false).

[700] And the other issues discussed in chapters 2-4.

dividing humans by race, nationality, diet,[701] and, of course, sex/gender. Given that there are many justificatory trains available for sex/gender (sex hormones, other circulating molecules, neurodevelopmental patterns, prenatal influences, chemical environments, social environment, social pressures on behavior, learned changes to neurobiology, social pressures on answers provided in experimental contexts, etc.) and that in general these explanations are not used together, at least some of the justifications attempting to explain the origins of sex differences are systemic Gettier cases—describing true phenomena but unjustified. I say "systemic Gettier cases" because Gettier cases in their original form are one-off thought experiments, but this dynamic of true belief without a correct justification also describes, at least potentially, the answers to these larger research questions across multiple disciplines investigated by at least hundreds of researchers over well more than the two decades this thesis covered.

For example, one can understand race science, i.e. the research supporting the theory that human racial groups differed in ways which explained their differing life outcomes, as a Gettier-type justification addressing a true belief. It is true, for example, that after chattel slavery was ended within the United States[702] African-Americans as a group had generally shorter lifespans, were less likely to be educated, had lower incomes, and so on. Race science justified this true belief as a biological consequence of African heritage; sociological arguments justified this true belief as a social consequence of the poorer conditions African-Americans lived in.[703] This

[701] Vegetarian versus non-vegetarian diets are a common point of dispute for being perhaps-more healthy, or perhaps-an effect of a social determinant of health like wealth or social class, or both, or something else.

[702] This refers to the period between the ratification of the 13th Amendment to the US Constitution on 6 December 1865, which made African-Americans citizens of the United States, and the writing of this thesis in 2024. While slavery hardly ended worldwide, or even within the United States, the widespread chattel slavery of the preceding three centuries did largely halt in 1865.

[703] This is, of course, a simplification of the historical trajectory. For example, it is true that those of African descent, more specifically sub-Saharan African descent, are more likely to have sickle-cell anemia due to inherited factors and the relative historical prevalence of malarial disease. This is, however, likely the exception which proves

historical example does suggest that poor justifications and better or good justifications can in some way be separated, as race science is largely defunct. While a couple of population-level differences are (sickle-cell anemia/malarial resistance)[704] or might be[705] inherited, most population-level differences between European-Americans[706] and African-Americans in the United States emerge from systemic and social factors—poorer living conditions, poorer economic prospects, higher likelihood of encountering state or non-state violence, higher likelihood of experiencing disadvantage due to prejudice, etc. Race science represents over a hundred years of work that justified the existing racial social order under the auspices of scientific authority to produce only misery and nothing of worth, so one would hope that scientists are trained differently or have in some way benefited from the lessons of history to prevent such moral and epistemic travesties.

However, the professional magician and skeptic James Randi argued in 2005 that scientists were not actually less vulnerable to being fooled but were far easier to fool than just anyone off the street.[707] Conmen generally try to justify some belief, a belief they hope the audience will accept as true, through the use of trickery and sleight-of-hand. Scientists, at least in

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the rule, as socioeconomic factors and prejudice far better explain most of the lived experiences of African-Americans over the past two centuries than inherited factors.

[704] However, it is worth recalling the point made in §4.2—race/ethnicity is not genetics, and it is important to recall that race is a relatively insensitive proxy for what is actually of interest—in this case, the prevalence of a genetic mutation.

[705] I have no particular ideas about what these would be, but they may exist.

[706] This is more complex than it at first appears, given the discrimination against various groups now seen as "European" but who would not necessarily have been "White" in the socioeconomic sense—those from the Iberian peninsula, what is now Italy, the island of Ireland, Finland, much of the historically Catholic or Orthodox areas of Central, Eastern, and Southeastern Europe, and all European Jews were at various times characterized as racial "others" by the normative white, Protestant, and Anglo-Saxon United States population. That being said, "whiteness" has generally collapsed into one group for most population-level purposes in the United States.

[707] James Randi, "James Randi's Swift," 20 October 2006, URL: archive.randi.org/site/jr/2006-10/100620sentient.html. I would also point out that Randi, when discussing spiritual healing in that newsletter, correctly noted the existence of publishing bias and results-oriented incentives in both the scientific and public spheres that would propagate positive results and squash negative results.

Randi's experience, tended to assume that they 1) had a grasp on the natural world exceeding that of others, 2) could not be deceived by someone who (they believed) was not their intellectual equal in logical thought, 3) would not be fooled if they took sufficient care to examine the materials or process, and 4) had a grasp on the human elements involved. Conmen and magicians could then use these four beliefs to fool the scientist, by directing the scientist's attention to those parts of the trick that will not expose the trick being run.[708] That the scientists have a grasp on the natural world exceeding that of magicians and conmen is probably true, but the scientists are still human—and magicians and conmen succeed and fail based on their grasp of the human elements of interactions. Conmen can make a human trick look like a natural miracle by allowing the scientist to believe that the interaction lies squarely within the scientist's domain of expertise when it is the human, interpersonal elements which are more relevant. While the conmen and magicians manipulate the salience and visibility of different information to seem to alter the world, it is not this manipulation alone that fools the scientist. The scientist *also* must eliminate this alternative, ideally without thinking about it: 'Something about my view on the matter obscured my vision; I was fooled, though I do not know how, and this is not magic.' The scientist tricks themself by believing they have eliminated or controlled for the complexities, the hidden mechanisms, and the human elements. Intelligence is not a cure-all to these problems; it may, in fact, be a hindrance. The scientist knows, after all, that they are intelligent, learned, and skilled. Why would that scientist not turn that intelligence, learning, and skill to use by fortifying their own conclusion and undermining any objections, particularly if they believe that they could not have been fooled?

[708] Randi's example was to state that he was using an ordinary spoon—the scientist would invariably investigate the spoon, but the spoon was always ordinary and the trick would lie somewhere else.

Donna Haraway once described nature as a trickster, the Coyote of some southern US native American folklores, suggesting that those investigating the world should "give up mastery but keep searching for fidelity, knowing all the while that we will be hoodwinked."[709] In other words, a justification of an observation or set of data may in fact be correct, but not only is there no way to know, it is more likely than not that the chain of justification is flawed somehow. That being said, I do not wish to claim that all flawed justifications are equally poor, nor that there are no potential changes that might alleviate the worst of these epistemic failings. While scientists could take a position of reflexive distrust toward bold claims and generally decline to amplify them or believe them, that would be an effort at compensation rather than a solution and may, at an extreme, lead to calcification of the discipline as new ideas cannot enter to be discussed.[710] Straddling the systemic and individual levels, the space and context to understand one's own standpoint and take advantage of the standpoints of others should be encouraged and facilitated. To know one's own biases requires the space to express those biases before they become an epistemic problem.[711] To understand the limits of one's experience requires glimpsing some of

[709] Haraway, "Situated Knowledges," p. 593-594.

[710] While the situations are not quite analogous, geology around 1900 had calcified in this way. Radiometric dating, evolutionary theory, and species ranges (including fossil ranges) were not accepted as evidence for the age of the earth and plate tectonic shifts in geology, and geology collectively clung to an age for the earth (Kelvin's) that biologists and physicists (among others) suggested could not be accurate as well as rejected plate tectonics until they flatly could not anymore. Lawrence Badash, "The Age-of-the-Earth Debate," *Scientific American 261*, no. 2: August 1989, URL: jstor.org/stable/24987366, p. 90-97; Philip England, Peter Molnar, and Frank Richter, "John Perry's neglected critique of Kelvin's age for the Earth: A missed opportunity in geodynamics," *GSA Today 17*, no. 1: January 2007, DOI: 10.1130/GSAT01701A.1, p. 4-9; Robert P. Newman, "American Intransigence: The Rejection of Continental Drift in the Great Debates of the 1920's," *Earth Sciences History 14*, no. 1: 1995, URL: jstor.org/stable/24137198, p. 62-83; Richard Conniff, "When Continental Drift Was Considered Pseudoscience," *Smithsonian Magazine*, June 2012, URL: smithsonianmag.com/science-nature/when-continental-drift-was-considered-pseudoscience-90353214.

[711] I am recalling a former chemistry teacher who described the halogens (Group 17/VIIa) as "wanting" electrons and the alkali metals (Group 1/Ia) as "wanting" to "give up" their electrons, before stopping and saying that atoms could not want anything and that this was not proper scientific terminology. However, he continued, while he (and various testing organizations) would deduct points for students using that terminology on a test, that should not stop anyone from thinking that way, as long as they did not fool themselves into believing that the atoms really did feel

the depth and breadth of human experiences in time and space, and to access the expertise of others requires recognizing and respecting those disciplines as well as understanding that their expertise may be relevant.

Within the framework outlined in this conclusion, which might be imagined as a set of knowers with their own histories and assumptions collecting knowledge (or at least beliefs) to answer questions about the experienced world, there might additionally be a few tricks to start understanding when systemic Gettier cases might be the structure of the question. One suggestion I can offer is that it might be worth asking someone critical of the concepts and definitions in use if there is some systemic flaw to be wary of that might be a problem for the train of justification.[712] Another similar suggestion is to discuss one's own understanding of various entities with those who study those entities more directly—biochemistry's understanding of hormone receptors and cell signaling pathways, for example, changed significantly between 2000 and 2019 in ways that were inconsistently reflected in the organizational/activational research. (These two suggestions, however, do cost time and effort and require some familiarity with other fields, and so may be more systemic than at first they appear.) Another possible question, to return to entanglement, is: what other beliefs about the world, and the nature of evidence, are linked to this justification, and can this justification actually stand on its own?[713] If

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things. In other words, the self-conscious use of a biased and inaccurate framework may have some utility—but more useful, at least for me, was suddenly understanding that I had already been thinking of the elements as having agency as a shorthand for electronegativity without ever really noticing until he said something. It is unlikely I am alone in this tendency, as agency is habitually attributed to all sorts of organisms, quasi-living entities (e.g. viruses), particles (e.g. molecules), and processes (e.g. evolution) which do not obviously or flatly cannot act with intention in the way expressed by the verbiage attached to them. Without that teacher stating to the class the sort of caution they should try to exercise, I am unsure when or how I would have noticed my own biases.

[712] The argument of the critic might not be relevant, or the scientist might not agree with it, but not seeking out any possible issues seems somewhat shortsighted, given the general access to research and scholarship universities and research institutions pay for.

[713] To a point, at least. Reexamining the ability of scientists to investigate the world due to phenomenological debates about humans' ability to access "reality", if such a thing exists, may be less useful for practicing researchers who presumably have different questions they wish to examine.

there are many linked pieces of evidence pointing to the same conclusion, are any of those pieces of evidence strong enough to stand without the theory linking all of that evidence together?

Finally, how many ways are there to go from the definition of the question to the true belief? In the case of sex/gender differences, there are a number of preexisting chains of justification (listed in the second paragraph in this section). Even had evidence been sufficient to eliminate a particular type of social influence on gender identity, one should return to Randi's description of how scientists are easier to fool. That particular justification may be incorrect, but to reflexively eliminate all other possibilities besides one's own (the conman's, who in this case is also the scientist) preferred justification is one of the ways scientists fool themselves. While I would argue that the universality of sex-typed assumptions certainly helped these patterns persist in behavioral neuroendocrinology, the patterns themselves are more broadly applicable, as my use of historical and contemporary examples has demonstrated. In the case of the organizational/activational theory, the researchers were systemically failed by their institutions and repeatedly fooled themselves, unable to see that they had assumed simplicity where there was complexity. In the end, they were unable to justify their conclusions at all because of the possibilities left unaccounted for and the gaps between what they could support and what they argued.

5.5: Conclusion

> *"When being serious, when not exploring deliberately simplified models, the theoretical physicist differs from the novelist in thinking that maybe the story might be true. Perhaps there is some analogy with the historical novelist. If the action is put in the year 1327, the Pope must be located in Avignon, not Rome. The serious theories of theoretical physicists must not contradict experimental facts. If thoughts are put into the mind of Pope John*

> *XXII, then they must be reasonably consistent with what is known of his words and actions. When we invent worlds in physics we would have them to be mathematically consistent continuations of the visible world into the invisible…even when it is beyond human capability to decide which, if any, of those worlds is the true one. [ellipsis in original]"*[714]

Throughout this thesis, I have mostly spoken about assumptions as failures, but assumptions can also be necessary. When information is inaccessible and it is unclear if or when the information will be knowable, assumptions can bridge the gap.[715] When it is unclear even what information will be meaningful to answer a question, starting with what one can observe is a reasonable choice.[716] The mistake made by scientists is not making the assumption, but forgetting (or never knowing) that it is an assumption. Assumptions are like scaffolding: they allow for something to be built, but they are not steady ground.

Organizational/activational theory was adopted around 2000 at an unfortunate crossroads of inertia (there are naturally men and women), ignorance (of how humans develop and decide who they are), and assumption (behavioral neuroendocrinology can understand this with a few years of work—after all, we have fMRIs and the Human Genome now). I suspect that these are the three basic elements of how scientists—and all researchers—can fool themselves into believing they know something when they do not: inertia to keep the theorizing going in a

[714] J. S. Bell, "Six possible worlds of quantum mechanics," in *Speakable and Unspeakable in Quantum Mechanics*, 2nd ed., Cambridge University Press (Cambridge, UK): 2004, p. 195.
[715] Chapter 2 and §3.7, 3.8.
[716] There are many criticisms of the morphological species concept, or the idea of defining a species by its features, but it is also the first species concept and what scientists fall back on in cases with low information (extinct species, species with low observational data, species without genomic data, etc.) because sometimes there is nothing else to work with.

consistent direction, ignorance to provide space for endless further possibilities and caveats, and assumptions to paper over all the gaps.

The last recommendation I have, and the least satisfying, is Socrates' definition of *sophos*: knowing the outlines of one's own ignorance.[717] To paraphrase and extend Bell's quote above, when we invent stories to explain the world, we create those stories because we believe that they might be true. The stories might be consistent continuations of the known into the unknown, but Bell's point is that consistency alone is not enough to make the imagined worlds true. Any of the imagined worlds might be the real one, but resorting to imagination means that it is yet "beyond human capability to decide which, if any, of those worlds is the true one."[718] The task, *an obair*, is to hold the imagined world and make room for all the other worlds that might be—a task that's far from easy.

[717] The relevant section in Plato's "Apology" is 21b through 21d and the first half of 21e. Plato, "The Apology of Socrates," translated by Benjamin Jowett and adapted by Miriam Carlisle, Thomas E. Jenkins, Gregory Nagy, and Soo-Yong Kim, *Harvard Center for Hellenic Studies*, URL: chs.harvard.edu/primary-source/plato-the-apology-of-socrates-sb.
[718] Bell, "Six possible worlds of quantum mechanics," p. 195.

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Appendix 1: Citations of Journal Literature

Citations are organized chronologically by year and then alphabetically by first author.

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