

# Homunculus in the Hormones?<sup>1</sup>

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How is it that we, as humans, come to be who we are? It is, of course, one of the most ancient questions, and it may signal a curiosity about individuality (How do I, in particular, come to have these ideas, skills, bodily traits, and desires?) as well as about groups (Why are these things living and those dead? Why are these beings human and those dogs? Why is *this kind* of human different from *that kind*?). Aristotle proposed that all living beings must have a “soul” – an active principle that animates and directs the development of the organism, without which the living being would be lifeless as a mineral. The soul gave both life itself, and the specific form of the organism. This explanation satisfied plenty of people for a remarkably long time. But during the seventeenth century, learned people became more and more likely to expect that theories about the natural world should be based on evidence, and the idea that development was directed by the soul created a serious problem. Namely, no one had ever seen a soul, nor had any idea where, specifically, it might be, nor how, exactly it might work. In short, the soul was too abstract and metaphysical an idea for the increasingly materialist beliefs of Western science.

And so, because no one could see how matter could take on new forms all by itself, the idea of preformationism began to take hold. In short, this was the idea that organisms develop from smaller versions of themselves. The fundamental form of the organism was always already there. The biologist/historian Clara Pinto-Correia (1997) has written a lovely history that fleshes out all the details of the interesting and still relevant history of preformationism, which she defines as “the assumption that the primordial organism already contains

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inside itself all other organisms of the same species, perfectly preformed, miniscule though they might be”. That idea now seems quaint, but it is not really so far removed from what she describes as “the more sophisticated version of the model”, pre-existence, “in which the primordial organism contains only the basic blueprints of all the related organisms to come” (Pinto-Correia, 1997: xxi).

A big argument among preformationists concerned the role of eggs and ovaries versus the role of sperm and testes. ‘Spermists’ were certain that the rudimentary human could be found in sperm, and ‘ovists’ were convinced that the egg was the true address of the tiny pre-human. The famous naturalist Antoni van Leeuwenhoek, a spermist, famously reported how his microscope allowed him to ‘see’ these tiny creatures in sperm, and he called them “little men” (in Latin: homunculus). The homunculus is still with us, as it turns out. Scientists no longer ‘see’ them in sperm, or in ova – but I argue in this paper that a theory of development that is currently quite popular is a variation on the broad theme of pre-existence. This theory, brain organization, holds that ‘sex hormones’ transform the initially sex-neutral matter of the brain, causing the brain to take on traits that are timelessly masculine or feminine. Instead of literal ‘little men’, the entity that is now thought to be transmitted across time, from one generation to the next, is masculinity or femininity – a popular way of understanding fundamental categories of human nature. Rather than curled inside the sperm, as the ‘spermists’ believed, or tucked inside the ovum, as ‘ovists’ did, the imaginary timeless ‘little men’ and ‘little women’ of brain organization theory are encased in testosterone and estrogen.

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This chapter encapsulates the main arguments of my book *Brain Storm* (Jordan-Young, 2010), in which I present the first systematic and synthetic analysis of all the studies applying brain organization theory to humans from introduction of the theory in 1959 until the first decade of this century. I begin by explaining the theory itself and the basic study designs, and then describe my analytic method of using “symmetry principles” to evaluate how well the theory is supported by the evidence that has emerged from the many hundreds of studies in humans. To anticipate my conclusion, I argue that there are so many gaps and contradictions in the research on brain organization in humans that the theory is not supported by a coherent body of evidence. Current acceptance of this theory as a ‘fact’ of human development is at best premature.

For the purposes of this chapter, I present a very concise summary of the main types of human brain organization studies, five core assumptions that drive the research, and three fundamental ways that the overall evidence from research on brain organization fails to support the theory by conventional scientific standards. I conclude with a few notes about the different levels on which we can understand the discrepancies and gaps in brain organization research, especially considering whether it is necessary to choose between identifying scientific errors, or pursuing an analysis that demonstrates the fundamentally social and contingent nature of scientific knowledge. This tension poses a particular risk when I use other kinds of research on human sex-typed traits in order to contrast those with the vision of sex/gender embedded in brain organization theory. In *Brain Storm*, I used such contrasts in a variety of ways, chief among them to show that all evidence does not, in fact, converge in support of brain organization theory. I want to be clear here that it was never my intention to suggest that one of these *other* ways of knowing human sex/gender is in some way fully ‘true’ or can escape all the baggage of embedded gender ideology, incomplete modeling and data, and other flaws that bedevil brain organization research. Yet some research approaches do seem more promising than others to me. Everything in my own research on sexuality, my knowledge of history and feminist STS, and life experiences prepare me to see sexuality and gender as complex, contingent, and historically changeable – in short, not very likely to be the kind of phenomena that might be characterized as ‘traits’ that emerge out of masculine or feminine substances. So with that disclaimer out in the open, let’s take a closer look at brain organization research.

## THE THEORY OF BRAIN ORGANIZATION

“If you’re going to reproduce bisexually, you need different genitalia, you need different gonads, too, and you need different internal organs. But the brain controls these things, so you need – I think you need a different brain.”

Brain Organization Researcher (‘Dr. I’), August 20, 1998

To examine brain organization theory, I analyzed all the human studies that scientists have used to test this theory, and I also identified the most widely-cited and influential scientists in the world who conduct such studies. My main interest was in talking to them about the technical details of their studies, but I

also I asked them to explain the theory in the most concise way they could, and I asked them what stimulated their initial interest in the theory. ‘Dr. I’ is one of those scientists<sup>2</sup> – a world-renowned neuroscientist who has been doing studies related to this theory for decades. The way he framed it, in a sexually reproducing species, the brain is a sort of accessory reproductive organ. To him, the existence of a male brain versus a female brain is not an open question that one should investigate, but is instead a logical requirement of sexual reproduction itself. Brain organization theory is appealing to ‘Dr. I’ and others who think like him because it has the characteristic of ‘parsimony’, meaning that it is the simplest explanation that covers a great number of phenomena. Brain organization theory is an excellent example of a parsimonious theory, because it builds a single unifying explanation of sex development, encompassing both behavior and the physical/physiological body. The key actor for both the body and behavior (via the brain) would be steroid hormones.

To skim quickly over a great deal of history that is covered nicely in many other places (e.g. Oudshoorn, 1994; Sengoopta, 2006; Van den Wijngaard, 1997), the first century of hormone research – predating both the term ‘hormones’ and the biochemical insights and technologies that would allow precise isolation and identification of these substances – was focused on questions of sex. The steroids that people still today tend to think of as ‘sex hormones’ were put together into groups based not on having similar chemical structures, but based on their abilities to affect characteristics associated with masculinity (the hormones classified as ‘androgens’) or those affected with femininity (the ‘estrogens’). Nelly Oudshoorn (1994) has shown how a certain ideology of binary, oppositional sexes created a research framework that not only shaped the experiments in early endocrinology, but repeatedly blocked evidence that contradicted this ideology. Three major expectations included that: 1) these chemicals would be sex-specific (present only in healthy individuals of one sex or the other, but not both); 2) they would be antagonistic (androgens counteracting the effects of estrogens); and 3) they would be fundamentally ‘for’ development of sexual characteristics, as opposed to being involved in a wide range of functions for both sexes.

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2 | Following standard ethnographic practice, in both the book and this chapter I refer to all scientists I interviewed by pseudonyms rather than their real names. I promised them confidentiality when I conducted the interviews, in order to make them feel comfortable expressing scientifically unpopular views, or critiquing colleagues or work that they wouldn’t want to publicly oppose.

By the 1950s, experiments in developmental endocrinology had demonstrated that steroids play a very powerful role in the differentiation of reproductive and genital structures. It was during this time that more systematic experimental methods (e.g., using control groups as well as experimental groups) and advanced research techniques (e.g., the ability to conduct surgeries extremely early in animal development) allowed Alfred Jost and others to solve what had been a longstanding puzzle in sexual development: how do fetuses go from being sexually ‘neutral’ – that is, structurally indistinguishable by sex, from the macroscopic level, at least – to being visibly and functionally either ‘male’ or ‘female’? Jost built on experiments going all the way back to the father of experimental endocrinology, the Viennese scientist Eugen Steinach, trying to show that steroids, and in particular androgens, seemed to play the decisive role. If any animal is exposed to a sufficient quantity of androgens during the critical period in its life when genital and reproductive differentiation takes place (a period that is the same for every individual in a given species), that animal will develop the reproductive structures of a male. In the absence of androgens, ‘female’ structures would develop. Jost concluded that the female pathway is the *default direction*, which happens in the absence of any gonadal hormones. This led Jost and others to assert that female development is ‘passive’. This idea, which enjoyed an uncanny fit with social ideas about the assumed (proper and natural) passivity of women, reigned for nearly fifty years before developmental biologists came to their senses and apparently remembered that there is no such thing as ‘passive’ development: there are *always* mechanisms to be explained. It’s simply the case that the developmental mechanisms for female reproductive structures seem to be somewhat different than for males – they aren’t under the obvious control of gonadal steroids, and they may be more directly related to genetic mechanisms (see e.g. Hughes, 2004; Yao, 2005). And while it is true that ‘androgens’ like testosterone (especially after conversion to dihydrotestosterone) are crucial for development of male-typical structures, it’s also the case that some aspects of male reproductive tract development are in fact controlled by estrogen, the supposedly ‘female’ hormone (for a review, see Vincenzo et al., 2009). This already shows that the Jost Paradigm is too simple in holding that with androgens, development is masculine, and without androgens, development is feminine.

In 1959, William Young and colleagues extended the Jost Paradigm to explain the development of masculine and feminine *behavior*, by suggesting that brain development follows a path that is basically similar to the development of genitals: the brain begins as sex-neutral in all individuals (regardless of

chromosomes), but it develops as either masculine or feminine depending on whether androgens are present during a critical period (Phoenix et al., 1959). They based their thinking on experiments in guinea pigs that had been castrated and treated with hormones at very early stages of life. Those animals who were treated with androgens, regardless of their chromosomal sex, were more likely to display ‘male-typical’ sexual behaviors, meaning that they would readily mount other animals. ‘Depriving’ developing animals of androgens during the same critical period resulted in a basically ‘female-typical’ pattern, meaning that they would more readily perform the arched-back lordosis response, and allow other animals to mount them. From the very first research report on brain organization, Young and his colleagues suggested that this same process could explain human sex-typed behavior.

One of the most important things to understand about brain organization theory is that it cannot be tested experimentally in humans. Doing so would require interventions in human development that are not only unethical and against both international law and virtually all professional codes of ethics, but logistically so complex and expensive as to be utterly unrealistic. For example, to mirror even the most basic, early experiments on guinea pigs, scientists would need to perform surgery on human fetuses to remove the testes or ovaries during a very early period of development, and then randomly assign the pregnant women who were still carrying these castrated fetuses to receive standardized ‘masculinizing’ or ‘feminizing’ hormone regimens. Then these experimentally manipulated fetuses would have to be tracked all the way into adulthood to see how their gender and sexuality turned out – under ‘blinding’ conditions, of course, meaning that neither the scientists nor the families nor the experimental subjects themselves would know what kind of treatment they got. There’s no need to go into all the other details of this nightmarish-fantasy research: it’s an evil idea, and it’s simply not possible. As a result, you can’t look at the results of studies on brain organization in the same way that you would if they were experiments. You have to consider the evidence differently, and more holistically.

With experiments, scientists control the circumstances of knowledge production as much as possible, ideally varying just one thing at a time in order to observe how elements relate to one another. This is a rough, or idealized, description, but it works as a contrast to the sorts of studies scientists have to do when they can’t experiment. Non-experimental studies are called ‘observational research’ or ‘quasi-experiments’. In this sort of work, there is even more room for interpretation, and there can never be a single definitive study.

Instead, scientists have to piece together various studies that use different approaches, and therefore have different strengths and weaknesses. But in piecing together those different studies, it is important to pay attention to how the different elements that scientists are working with either line up (are symmetrical) or are different. That is how I conducted symmetry analyses of brain organization research. While the work of examining more than 300 studies in detail was painstaking, the idea behind it was rather simple: when scientists say that androgen exposures in early development lead to masculine gender and sexuality, what precisely do they mean by ‘masculine gender and sexuality’? Do these things mean the same thing in various different studies? What about the other phenomena that scientists link to early hormone exposures, like heterosexuality or homosexuality, or ‘sex-typed interests’? In my book, and in the few examples below, I show that the way scientists approach these phenomena are so profoundly different as to make the whole body of research extremely incoherent.

First, though, it’s useful to understand the kinds of studies scientists have used to test the theory in humans. Their first strategy was to study people they considered ‘experiments of nature’: those who were known to have had hormone exposures that were unusual for their genetic sex. They studied people with intersex conditions in which either the level of exposure to particular hormones, or the way that the body responded to hormones, was not typical. The most common intersex conditions that scientists have studied include genetic females with the classical form of congenital adrenal hyperplasia (CAH), in which there is high production of androgens during fetal life; androgen insensitivity syndrome (AIS), in which a genetic male produces androgens, but the tissues do not respond to them; and 5-alpha reductase deficiency, in which an enzyme that is necessary for converting testosterone to dihydrotestosterone is missing, so that genetic males without this enzyme are born with ambiguous looking genitalia, but develop a much more ‘masculine’ physique at puberty. Once scientists have identified a group of people with intersex conditions to study, they compare this group with non-intersex people of the same assigned sex/gender (i.e. both intersex and ‘control’ subjects must be reared in the same gender). In epidemiology, studies that compare outcomes among two groups who have had different earlier exposures are called cohort studies, so that’s the term I’ll use to describe these studies.

By 1967, reports began to appear that suggested girls and women who had been exposed to a high level of androgens in the womb were, indeed, more ‘masculine’ than other girls and women (Ehrhardt and Money, 1967; Ehrhardt,

Evers, and Money, 1968; Ehrhardt, Epstein, and Money, 1968), and the researchers attributed this to the effects of ‘sex hormones’ on the developing brain (see Jordan-Young, 2010: 32–35 and 69–73).

A second research strategy was introduced in the early 1970s: start from the other end of development, by comparing people with patterns of gender or sexuality that scientists considered ‘sex-reversed’ to people that they considered ‘normal’. This is a classic ‘case-control’ design, where scientists look at people with different outcomes and then look for some kind of evidence that the two groups have had different earlier exposures. Many studies along these lines begin by recruiting groups of gay men and heterosexual men for comparison. The scientists then take measurements of other physical or psychological traits that they believe are also affected by early hormone exposures – things like the relative length of different fingers, left- versus right-handedness, or personality traits that are considered masculine versus feminine. If they find that gay men and straight men are, on average, also different on any of these other traits, the scientists infer that early hormone exposures may have influenced both sexual orientation and the other trait(s) (see Jordan-Young, 2010: 38–48 and chapter 5, *passim*).

So to recap the two main sorts of studies, brain organization studies of intersex people group subjects according to the hormonal *inputs* into their development; studies of gay, lesbian, and trans people begin with developmental *outcomes* that scientists consider sex-reversed, then look backwards for evidence that their fetal hormone exposures were different than those of cisgender heterosexuals.

Though they are rarely, if ever, explicitly enumerated, it’s worth identifying a number of underlying assumptions within brain organization theory research: 1) the brain is (*must be*) sexually dimorphic; 2) ‘male’ and ‘female’ are distinct categories; 3) ‘masculine’ and ‘feminine’ are sets of simple, common-sense traits; 4) since reproduction is the purpose of sexual differentiation, heterosexual desire and behavior is the aim of brain organization; and, it follows from the fourth assumption that 5) homosexuality is a ‘cross-sex’ trait which, above all other aspects of personality or behavior, indicates that brain organization is ‘sex reversed’ – meaning that it has taken a different path from the sexual differentiation of the rest of the body.

Even the originators of the theory were well aware that not all of these elements were true, especially in humans but even in non-human species. For example, the fact that there is some overlap in the supposedly dimorphic sexual behavior was the reason that they were so careful to use untreated control ani-

mals of both sexes (Phoenix et al., 1959: 182). That is, hormonally untreated, normal females of guinea pigs and the other small mammals will mount other animals to some extent, and males of these same species also allow themselves to be mounted. Young's team may not have been aware of the extent to which these behaviors varied, because exploring and documenting the phenomena related to animal behavior or human behavior was not their real interest. From the beginning, the things that interested biological scientists about sex were all 'how' questions, not 'what' questions. In other words, they were firmly focused on *how* sex develops, but did much less thinking about *what* precisely 'sex' is. As a result, they incorporated more or less wholesale the folk ideas about sex that were popular in the times and places where they have worked. While the details in these folk ideas have varied in interesting ways that turn out to be important for understanding how the research fits together over time, one of the most enduring assumptions in scientific work on hormones and sex development is that sex is binary (male versus female), and it is a 'package deal' – sex-linked traits of the body, all aspects of personality or behavior that are coded as 'masculine' or 'feminine', and everything about erotic desire and practice are all understood to *flow from* and also *reveal* a single underlying masculine or feminine nature.

In humans, the 'behavioral phenotypes' that are linked to this theory span virtually every domain that has been thought to differ between girls and boys in childhood, or men and women in adulthood. Sexuality was always of special interest, given the origins of the theory and the underlying notion that brain organization served the ultimate purpose of reproduction. The aspects of sexuality that scientists linked back to early hormone exposures included sexual orientation, libido, types of sexual acts, and patterns of becoming sexually aroused (e.g. Allen and Gorski, 1992; Ehrhardt, Evers, and Money, 1968; Kester et al., 1980; LeVay, 1991; McIntyre, 2003; Money, Ehrhardt, and Masicca, 1968; Yalom, Green, and Fisk, 1973; see Jordan-Young, 2010: chapter 6, *passim*). They also linked what they called 'core gender identity' to hormones, meaning that they believed hormones to affect one's fundamental sense of self as male or female (or, as they sometimes acknowledged, as neither or both) (e.g. Berenbaum and Bailey, 2003; Ehrhardt, Epstein, and Money, 1968; Gooren and Cohen-Kettenis, 1991; Zucker et al., 2001; see Jordan-Young, 2010: 257–64 for an alternate reading of the evidence). Scientists have further asserted hormonal influence on 'gender role', a catch-all category that encompassed any behavior more common to, or thought to be appropriate for, one sex versus the other – things ranging from playing with dolls, building toys,

and love of sports to care in personal appearance, relationship ideals, hobbies and occupational aspirations (e.g. Berenbaum and Hines, 1992; Henderson and Berenbaum, 1997; Money and Ehrhardt, 1972; Pasterski et al., 2005; see Jordan-Young, 2010: chapter 8, *passim*). They also linked cognitive skills to early brain organization, and were initially confident that ‘general intelligence’ or IQ increased with androgen exposures (Ehrhardt and Money, 1967; Money and Lewis, 1966). Researchers dropped that claim fairly early, when it became clear that IQ doesn’t actually differ between males and females, but continued to tie specific skills like mental rotation ability or verbal fluency to sex-typed hormone exposures early in development (e.g. Grimshaw, Sitarenios, and Finegan, 1995; Rahman et al., 2003; Sanders and Ross-Field, 1986; see Jordan-Young, 2010: chapter 4, especially 70–71, also 179–80).

Brain organization theory immediately transformed the research paradigm for sexual development, and hormones were thenceforth understood to play two distinct roles: an ‘organizing’ role and an ‘activating’ role. The organizing role, which hormones could only play during certain early critical periods of development, was about a permanent transformation of the brain from sex-neutral to either male or female, so that the behaviors that would eventually be expressed would consistently follow this sex-typing. The activating role, which circulating hormones play during puberty and adulthood, was about the specific timing and extent to which these ‘latent’ behaviors would get expressed. A good analogy is thinking about trains that run along particular tracks in the countryside. The ‘organizing’ force is like laying down the tracks, and the ‘activating’ force is akin to the power that makes the trains run. You can lay tracks allowing trains to pass through particular towns, but without power, the train will never get there. Likewise, all the power in the world will not make a train pass through a town that isn’t on the rail route.

Many of the longstanding puzzles in hormone research stem from the fact that it is not possible, in either humans or experimental animals, to find consistent correlations between hormones and behaviors. For example, many scientists tried but failed to find a relationship between intra-sex variations in observable sex-typed behavior, on the one hand, and circulating hormones, on the other. It was also not possible to consistently achieve behavioral changes by manipulating circulating hormones. Unfortunately, the history of endocrinology shows many examples of scientists and doctors attempting to change men’s sexual orientation from homosexual to heterosexual by giving them testosterone and other androgens, and these attempts are a part of the research tradition that fed into brain organization theory (for a review of many such

studies, see Meyer-Bahlburg, 1977). The ‘organizing’ hypothesis allowed an escape hatch from the difficulty posed by the failure of such interventions. Once the theory was introduced, the inability of scientists to show a correlation between behaviors or psychological traits and adult levels of circulating hormones could be dismissed as irrelevant. With the brain organization theory, scientists simply hypothesize that sex atypical traits or behaviors mean that something unusual *must have happened* with hormones in the early organizing period – even though that ‘something’ could no longer be directly seen or measured. This opened up a great many avenues for research designs that might yield indirect evidence of those earlier hormone effects. Scientists studied literally any aspect of behavior or psychology and temperament among intersex people, and if it seemed to differ from the non-intersex people to whom they are compared, then these differences have been overwhelmingly attributed to hormones. This continues to be the case in spite of longstanding arguments that the rearing experiences of intersex people are often dramatically different (Doell and Longino, 1988; Karkazis, 2008), that the medical and psychological interventions to which they are routinely subject are often traumatizing and in any case are certainly consequential (Jordan-Young, 2011; Minto et al., 2003; Morland, 2011), and that many other physical and physiological variables (like outward appearance, ‘mood hormones’, and metabolism) are also different in some intersex conditions, so attributing group differences to ‘prenatal sex hormones’ is an unacceptably narrow scientific interpretation of the data (see Jordan-Young, 2010: chapter 9, especially 240–57).

Within a few years it was not possible to even be taken seriously in the field without affirming the theory (Van den Wijngaard, 1997). As the 1970s rolled into the 1980s and beyond, though, this theory increasingly became regarded as a simple fact of development, folded into the background assumptions of research rather than being explicitly stated. This, of course, makes it all the more difficult, and all the more important, to step back and ask *how*, precisely, we know that this is the way things work? What is the evidence for this theory?

One way to answer this question is to approach it from a ‘within science’ perspective, focusing on methods and rules of evidential support. Elsewhere (Jordan-Young, 2010 and 2011), I have demonstrated three fundamental ways that the studies on brain organization in humans fail to provide convincing support for the theory according to the internal rules of science. First, many of the studies do not meet conventional standards of scientific research. For example, rules of statistical testing are routinely violated (e.g., by doing too

many comparisons in the same study, or by using the wrong kind of statistical tests for the study design). Another important violation of scientific principles is that studies that do not support the theory are routinely ignored in the literature, while those studies that strongly support the theory are cited very heavily, even though the latter are the smallest and least reliable studies. Collectively, such errors amount to what I have called “loading the dice” in favor of the theory.

The second major failure of brain organization theory is an interpretive problem: scientists routinely favor the explanation that hormones exert a direct organizing effect on the brain, which in turn directly affects behavior. Yet, as noted above regarding studies of people with intersex conditions, there are often many other (and, I would argue, more plausible) explanations for the small differences that scientists sometimes observe between people whose early hormone exposures have been different.

The third major failure is what I have termed a “lack of symmetry” between different studies of the same phenomenon. For example, dozens of studies that supposedly show how early hormone exposures affect sexual orientation do not actually add up to a coherent conclusion, because the studies use different and even outright contradictory definitions of and measures for sexual orientation. The same is true of studies that supposedly link early hormone exposures to other aspects of sex/gender psychology, such as ‘feminine’ or ‘masculine’ sexual styles, non-sexual interests (e.g., toys, occupations, hobbies), and personality traits that are supposedly gendered, such as aggression. Thus, I’ve argued that even from a strictly ‘empiricist’ or conventional scientific perspective, it’s time to drop this theory and move on to more interesting and complex ways of studying human development.

## CONCLUSION

The empirical critique – the part of my work that might understandably be mistaken for a sort of naïve feminist empiricism (Harding, 1993) – is only one facet of my analysis. In this project, I have tried to work on multiple analytic fronts simultaneously, in order to find points of connection with the widest range of readers, including everyone from the scientists who do these studies, to people who read about them in the daily news, to students of gender and critical science and technology studies (STS). In doing so, I know that I risk annoying readers from each of these groups by not directing myself more

fully or consistently to their interests and points of view. But I disagree with the notion that you cannot simultaneously be thoughtful about the adequacy of specific data and analytic methods for addressing particular questions, and at the same time maintain the perspective that *all* scientific knowledge is fundamentally social and partial (Haraway, 1988; Harding, 1993; Longino, 1990 and 2002).

Like all scientific objects, the human ‘traits’ that scientists study in connection with brain organization are contingent (Barad, 2007; Mol, 2002). The specific patterns of sexuality, cognition, emotion, and behavior that scientists ‘know’ through their research, and which their studies tie to specific kinds of hormone exposures, are not timeless, static, stable objects, but emergent phenomena, produced through the intra-action of the living beings who are studied, and the specific scientific practices that scientists employ in their research. These intra-actions characterize the psychology research that shows ‘gendered’ traits to be distributed in a mosaic way across individuals, rather than being actually sex-dimorphic, just as surely as they characterize brain organization research. Annemarie Mol and others who closely follow scientific practices (e.g. Franklin and Roberts, 2006; Martin, 1994; Murphy, 2006; Rapp, 1999) fracture the self-confident narrative of singular scientific objects and simple cause-effect relationships into “partialities, positionalities, complications, tenuousness, instabilities, irregularities, contradictions, heterogeneities, situatedness, and fragmentation – complexities” (Clarke, 2005: xxiv). Mol has observed that “this turns doing anthropology into a philosophical move” (2002: 32).

If I have not also turned the study of practices in brain organization research into a philosophical move, then I have failed in one of my aims. The point of delving into such detail into these studies, examining how the measures work, how the phenomena being examined shift from study to study, is precisely to highlight such contingencies. It is also to point out that there are different levels and degrees of multiplicity. Scientific standards such as those that suggest legitimate approaches to measurement, statistical procedures, and selection of experimental subjects and comparison groups cannot simply be thrown out because STS has already shown that science is contingent anyway. Most of us doing STS do it because of a healthy respect for, even a love of, empirical research. The most thrilling STS analysis doesn’t consist in pointing out ‘flaws’ but in pointing out interesting and seemingly impossible inconsistencies in excellent, even brilliant work (see Mol, 2002 for what is perhaps my favorite example of such an analysis).

In the final chapter of *Brain Storm*, I looked beyond brain organization research and contrast the model of development implicit in that theory with other, more interactive and contingent, models of development. I also contrast the implicit models of femininity and masculinity in brain research with the models of masculinity and femininity that are found in other kinds of studies, such as social psychology, or research on learning interventions related to cognitive skills that show sex/gender imbalances. What is the point of contrasting brain organization research with other forms of research on sex/gender? It is not to assert that one form is clearly 'true': even controlled experiments, if they could be done on human brain organization, would not yield pristine and simple knowledge. On this point, I think my argument in *Brain Storm* was faulty, or at least insufficiently clear; in several places, I appealed to the evidence from other kinds of research as if that evidence were 'naked fact'.

So here I wish to be clearer: scientists' ideas as well as the concrete methods and tools that they use work together with their human subjects to produce particular versions of reality. In the end, it is necessary to notice but then move beyond the knowledge that different scientific approaches yield divergent information about the 'same' phenomena (say aggression, or verbal abilities, or highly specific skills like 3-D mental rotation). It is necessary, finally, to ask which kinds of interventions in the world do different research projects make possible, and what interventions do they preclude (Longino, 2012)? This makes us responsible for making much more explicit the connections between scientific projects and social investments, and it is particularly crucial when the subject of research is human differences.

What sort of interventions are made possible or precluded by the project of brain organization research? This question brings us back to the dear little homunculus, and it is why I closed my book with a call to turn away from research that seeks the cause of female or male 'essential natures'. Brain organization research is deeply invested in female and male as the 'basic' human division, not just for purposes of reproduction but in general. It enshrines a particular worldview that treasures the sex/gender binary, and invites interventions to further stabilize sex/gender as singular within individuals. It is basically a backwards-looking theory, holding the individual as a 'finished product' of sorts and seeking the seeds of the individual's nature in 'masculine' or 'feminine' chemicals at the right (or wrong) time and place. It is a research project that is persistently (some might argue obsessively) focused on 'deviations' from the supposedly correct and healthy masculine or feminine type, which is the most extremely stereotyped form. This research orientation blunts

interventions that might reimagine gender, or those that would refocus attention on other ways of understanding human traits and potential.

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