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Sex Genes: A Critical Sociomaterial Approach to the Politics and Molecular Genetics of Sex Determination

How should the social sciences engage with the materiality of "nature"? The literatures of both the social studies of science and gender studies have wrestled with this question in their analyses of the production of scientific knowledge. In examining the production or consumption of scientific knowledge, these literatures have demonstrated how production and consumption are social and cultural activities. Within this shared terrain, however, many differences emerge both within and between these two literatures on the questions of how to theorize about the social in the scientific and about the scientific in the social, and how to create a language that does not separate science from society.

One topic explored has been the biological explanations for differences between males and females. Biologists and social scientists have proposed explanations for behavioral differences, and debates abound. In this article I do not discuss theories of or data on behavioral differences. Instead, I explore research on the material production of males and females in molecular genetic research on sex determination.

I address the question of how the social sciences should engage with the materiality of nature—in this case, the molecular genetics of sex determination. I employ a critical sociomaterial approach to social scientific engagements with the biological sciences. The sociomaterial approach encompasses the poststructuralist view that meanings are not inherent in events, phenomena, and things. That is, it assumes that humans attribute meanings to things through complex interactions based within specific locations in

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society, culture, and history. For example, the meanings attributed to nature—how nature is read—differ depending on its reader's location in time and place (see, e.g., Williams 1985; Strathern 1992). This approach also builds on feminist and sociocultural studies of science that have argued against the neat divide between nature (as nature in the raw) and culture (as social discourses and meanings). To emphasize this coproduction of nature and culture, Donna Haraway uses the term "material-semiotic practices" (1991, 208) to refer to the production process and "natureculture" (2003, 1) to refer to its product.

Despite this poststructural understanding of the mediation of natureculture, a material world does at times assert itself in ways that make us take notice (Haraway 1991; Fausto-Sterling 2000). Some anthropologists have used the term *biophysicality* (Goodman and Leatherman 1998; Escobar 1999) to describe such occurrences, while sociologists of science Bruno Latour (2000) and Michel Callon (1986) refer to the material world as comprised of nonhuman actants and treat them as ontologically on par with human actants.

Given that interventions by the material or biophysical world are acknowledged, the question arises: how does one recognize and deal with the actions of biophysicalities (or nonhuman actants) if they are always mediated by culture? To address this question, I use a critical sociomaterial approach to show how the materiality of sex is produced. I reexamine experimental research investigating the "*SRY*" and "*DAX-1*" genes, the so-called sex-determining genes, in mice and humans.[1]

A critical sociomaterial approach allows the examination and reanalysis of the social and historical production of material knowledge. It assumes that what is taken to be material must be investigated and should not be accepted at face value. It also requires multiple readings of the same data from different sociocultural perspectives or frames of reference. This approach builds on the theoretical efforts of, among others, feminist theorist

[1] *Sry* stands for sex-determining region Y gene, the gene that sits on the Y chromosome and is currently considered to be the gene that initiates male sex characteristics. *Dax-1* is the name for the dosage-sensitive sex reversal adrenal hypoplasia gene 1, Xp21, a transcription factor involved in adrenal cortex development and gonadotropin secretion. It has been widely accepted as an "antitestis" or ovary-determining gene because patients with a duplication or "double dose" of *Dax-1* had features of XY sex reversal, a condition in which individuals have the chromosomes of males but the physical attributes of females. For the same gene sequence, the agreed-upon notation in research articles is the italicized and lowercase *Sry* or *Dax-1* for the mouse gene, the italicized and capitalized *SRY* or *DAX-1* for the human gene, and the italicized and capitalized with quotation marks "*SRY*" or "*DAX-1*" for the gene in multiple species.

Haraway (1991), anthropologist Arturo Escobar (1999), philosopher Sandra Harding (1998, 2001), and the practical efforts of social movements around the globe to incorporate perspectives of actors not usually included in the production of science. These varied perspectives produce new knowledge and add dimensions to what Western science calls nature.

Thus, my reanalysis of "*SRY*" and "*DAX-1*" experiments is made in the context of multiple perspectives on sex. I examine human actions in sex determination by analyzing the research methods and interpretations of geneticists as well as the efforts of sex-gender theorists and transgender activists to theorize and remake sex. By analyzing the genetic experiments using multiple perspectives, I provide an alternative reading of the materiality of sex. That is, this reexamination of research on molecular genetic developmental processes provides a focus on the complex sets and pathways of events that produce material sex. These multiple pathways and multiple experimental outcomes could explain variations in human physiological phenotypes that sometimes do not fit neatly into the binary sex categories, male and female. Just as previous studies of human behavior, physiology, endocrinology, and chromosomes have met with difficulties in finally elucidating the source of male-female sex differences, so too have recent attempts to ascertain sex differences at the level of genes met with complexities and ambiguities. My reanalysis of genetic research further substantiates previous knowledge of sex as diverse and variable.

I also find that human and molecular geneticists used their own sociohistorically located normative definitions of sex in their experimental designs and analytic frames, thereby setting the stage for reproducing their own taken-for-granted categories of sex. Yet, even under those conditions, the material world intervened. New molecular genetic technologies produced new data that could have led researchers to new insights about sex development. However, new signals read through old frames can be discounted: in their conclusions researchers decided to ignore data that contradicted their initial assumptions.

This study refers to such ignored data as an "awkward surplus." Here, a critical sociomaterial reexamination of the awkward surplus suggests a different research conclusion from that reported by the scientists. This approach attends to unanticipated research results that experimenters recognized as problematic or awkward and that they thus ignored in their final conclusions. This critical sociomaterial approach provides a way to reexamine unexpected experimental data using different frames of reference and data from other sources. For example, social scientists, using knowledge of social movements (feminism, gay and lesbian movements, queer theory, intersexual and transsexual activism) and social and cultural

theory, literally can see differently when examining the work of geneticists and other scientists in the production of the science of sex. Further, the concept of awkward surplus provides science studies with a way of talking about materiality that does not deny human mediation but also acknowledges material agency. More generally, reexaminations of experimental material provide opportunities for natural scientists, social scientists, and other parties to approach research differently and collaboratively to produce new explanations.

Theoretical and historical frames of the sex-gender distinction

The sex-gender distinction has been the foundation of gender theory since the 1970s.[2] In their attempts to decouple biology from behavioral differences between the sexes, feminists in the 1970s and 1980s embraced the term *gender* to argue that behavioral differences between girls and boys and women and men were gendered.[3] That is, these differences were constructed within specific cultural and historical contexts (Scott 1988) and through specific technologies (see, e.g., de Lauretis 1987; Lorber 1994). Gendered differences, it was noted, are not uniform but situationally produced and interactionally accomplished (see, e.g., Kessler and McKenna 1985; West and Zimmerman 2002).[4] The term *gender* was also used to speak about sexuality in ways that did not assume or enforce heterosexuality (Rubin 1975). In this period, then, gender became socially constructed, while sex remained in the realm of nature and was left to biologists.

In the 1980s and 1990s, some feminists began to challenge this culture-nature division. Some studied the effect of hierarchies of power on the production of biological models of the body (e.g., Fausto-Sterling 1985; Hubbard 1990; Bordo 1993) and battled biological determinism by arguing that biological knowledge itself was gendered. Critics of gendered and raced knowledges argued that humans attribute meanings to nature through complex interactions based within specific locations in society, culture, and history—that nature is read differently depending, among other things, on the differential positions of its oh-so-human readers.[5]

[2] This is not intended to be a complete discussion of the history of gender theory, feminism, or gender and science.

[3] The term *gender*, as separated from *sex*, originated in John Money and Anke A. Ehrhardt's (1972) studies of hermaphrodites.

[4] For work on the idea of gender as process, see, e.g., Ferree, Lorber, and Hess (1999) and Butler (2004).

[5] See, e.g., Rose 1983; Smith 1987; Trinh 1987; Haraway 1989; Russett 1989; Schiebinger 1989; Laqueur 1990; Strathern 1992; Glenn 1999; Duster 2003.

The 1980s and 1990s also saw more explicit challenges to the feminist embrace of the sex-gender, qua nature-society, split. Historian of science Evelyn Fox Keller (1987), for example, argued against the dualities of sex and gender and of nature and science. Such dualities, she maintained, gave gender unlimited cultural plasticity and made science a set of relativist, interested constructions. In place of these polarities, Keller proposed that a multiplicity of differences could produce varied ways of doing science, each of which could be legitimate. Differences do not have to be reduced to those between male and female, where males and females produce diametrically opposed kinds of science. Nor must one choose universalism as the polar alternative and the only legitimate science. Instead, Keller suggested that there are many different possible kinds of sciences. Feminist theorist and historian Haraway (1988) similarly argued for "situated knowledges" produced by those with particular stakes in those knowledges.

Other feminist writers deconstructed the production of sex. Philosopher Judith Butler (1993) argued that it was incumbent on feminists to show how sex itself is discursively produced under historically located regulatory regimes of gender. Haraway argued more broadly that "bodies . . . are not born; they are made. . . . The various contending biological bodies emerge at the intersection of biological research, writing, and publishing; medical and other business practices; cultural productions of all kinds, including available metaphors and narratives; and technology" (1991, 208). Anne Fausto-Sterling (2000) presented concrete examples of the discursive production of bodies—specifically bones, brains, hormones, and genitalia—by medical and biological professionals.[6] Noting the conflation of the terms *sex* and *gender* in popular discourse, Joan Wallach Scott argued that "the conflation in ordinary usage of sex and gender can be considered a correction of the 'mistake' that treats sex and nature as transparent entities outside of 'culture'; instead, both gender and sex have to be understood as complexly related systems of knowledge" (1999, 72).[7]

In this article I take up the challenge of Keller, Butler, Haraway, Scott,

[6] Historian of science Diana Long Hall's (1976, 92–94) research on sex hormones in endocrinology demonstrates how novel biological practices and technologies in the 1920s changed and disturbed established representations of sex differences. For more recent work on the history of the intersection of hormone research and sex disciplining, see Oudshoorn (1994), Clarke (1998), and Fausto-Sterling (2000, chaps. 6, 7, 8). For an interesting challenge to feminist critiques of sex hormone research, see Roberts (2000).

[7] Feminist theorists Moira Gatens (1996) and Elizabeth Grosz (1994) also argue that the early division between sex and gender was useful for its purposes at that time but that this division now serves to keep feminists attending to social gender and to cede their authority over biological sex to biology.

and Fausto-Sterling. I show how the materiality of sex is produced in genetic sex-determination research, and I propose alternative knowledge practices and outcomes. A study of the production of the materiality of sex requires more than an examination of the shaping of sex via gendered understandings of scientists; it requires more than a study of the perception of sex in the minds of humans. Both have been necessary but are not sufficient. The study of the production of the materiality of sex also requires the engagement of social scientists in the production of biological sex. It requires our being in on the design and not just in quality control. I propose, then, that feminists and social scientists go beyond simply accepting or critiquing the products of science to engaging in the actual production of science. I begin by exploring molecular genetics research on sex-determination genes.

Do genes determine sex? Analysis of research on the molecular genetics of sex determination

If social scientists are to engage scientific research, it is incumbent on us to understand the sociotechnical processes that generate knowledge. Scientific knowledge is the outcome of socially situated production, where the social and technical are one process. Social scientific analysis of scientific research requires attending to all aspects of scientific knowledge production, including the daily laboratory practices that produce data and conclusions, the production of scientific articles, the media's selective reporting of some research results and not others, and the interested audiences and consumers of the knowledge produced (who are ever present throughout the production process, not simply at the last step). My investigations included all four aspects, but here I present the experiments that produced genetic knowledge about sex determination. I include the uncertainties, ambiguities, guesses, assumptions, omissions, and exclusions that were part of that knowledge production.[8]

Of mice and men: The design of male sex-determination genetic experiments

The search for the male-determining gene began in the 1980s in David Page's laboratory at the Whitehead Institute for Biomedical Research, which is affiliated with the Massachusetts Institute of Technology. Page's laboratory produced a "male gene" that was first named the *ZFY*, or zinc

[8] Since the 1970s scholars in the social studies of science have explored how scientific knowledge is marked by its situation and process of production.

finger Y, and later renamed the *TDF*, for "testis-determining factor" (Page et al. 1987, 1091).[9]

Page and his colleagues' experiments on what they thought might be the testis-determining gene are significant because they set the research protocol for all subsequent studies of male- and female-determining genes. This protocol first studies someone who has been selected for study after having presented him or herself to physicians because of a problem. In this case, Page and colleagues identify these individuals as "sex-reversed" because of their "abnormal" (1091) chromosomal constitutions, where their "sexual identities [are] at odds with their chromosomal makeup" (Roberts 1988, 21). Researchers then develop transgenic animal models of these "variations from the norm" (Roberts 1988, 21) and use them to study and describe the "normal" developmental pathway.[10]

Researchers in Page's laboratory used DNA from XX male human patients (or males with two X chromosomes instead of the usual XY chromosomes) and a female with a chromosomal constitution of "46,X,t(Y,22)(p11.2;q110)" (Page et al. 1987, 1094), which Page states represents a "reciprocal translocation between Y and autosome 22" (1094). According to David Baltimore, then director of the Whitehead Institute, "This is a classic use of very rare human genetic *defects* to find something very important about biology" (quoted in Roberts 1988, 21; emphasis added). Page states, "The key to the whole endeavor rests with certain exceptions to the rule that Y is sex-determining. . . . XX males were the most important exception" (quoted in Roberts 1988, 21). Leslie Roberts, a writer for *Science*, goes on to say that "XX males appear entirely normal . . . until they try to have children and are found to be sterile. Page reasoned that these men [with XX chromosomes] must contain a piece of Y chromosome, attached to one of their X chromosomes, that does not show up under light microscopy" (1988, 21).

The next step was to attempt to confirm the *ZFY*'s properties in mouse experiments. This did not go well. In December 1989, a team of scientists working at the Medical Research Council National Institute for Medical

[9] See Fausto-Sterling (1989) for an early critique of Page's research. Again, *Zfy* equals the mouse gene, capitalized *ZFY* the human gene, and "*ZFY*" the gene in multiple species.

[10] I use quotes around the term *normal* to refer to the construction of the "normal" through the construction of the "abnormal" developmental pathway. I discuss the simultaneous construction of the normal and the "pathological" later in this article. Transgenic animals or organisms are products of genetic manipulation. Their genetic material (nuclear deoxyribonucleic acid [DNA]) has been altered using recombinant DNA techniques that allow the movement of DNA from one organism into another. These DNA transfers are sometimes from a different species, sometimes from the same species.

Research and the Imperial Cancer Research Fund in London announced that *Zfy* (the mouse gene) did not produce testes in mice (Palmer et al. 1989; Kolata 1990). The hunt was on again to find the male-determining gene.

The maleness gene found

In July 1990 and May 1991, Peter Koopman, Peter Goodfellow, Robin Lovell-Badge, and their colleagues made a big splash with news of a new candidate, *Sry*, for the male-determining gene. They published their male gene research results in the journal *Nature*. Their approach to studying the male gene was similar to Page's: select sterile human males with XX chromosomes, find a gene common to them, then develop a transgenic mouse model to confirm (or contest) that that gene is involved in producing testes. A 1991 article titled "Male Development of Chromosomally Female Mice Transgenic for *Sry*" (Koopman et al.) announced that their *Sry* gene in the mouse model could turn XX female mice embryos into males.[11]

A close reading of the 1991 article by Koopman and his colleagues, however, tells a more ambiguous story. In the first experiment of the study, a number of fertilized eggs were injected with the *Sry* DNA sequences. The eggs were then transferred to the uteruses of female mice to develop, and this produced 158 viable embryos. Eight of these turned out to be XX mouse embryos with *Sry* incorporated into their DNA. Six of these eight were called female and two male.

In the second experiment, fertilized eggs were again injected with *Sry* DNA sequences, and the resulting embryos were transferred to the uteruses of female mice to develop. Ninety-three animals grew to term. Of these ninety-three, three were transgenic XX mice that had incorporated the *Sry* gene into at least one of their X chromosomes. Of the three *Sry* transgenic XX mice, two were females that produced viable eggs and reproduced. The third was called an XX male. It produced no sperm and was infertile.[12] The term *male* was applied because the animal had testes, although the testes were only 22 percent the size of normal male mouse testes. Human geneticist Giovanna Camerino, when commenting on this experiment, said, "Size doesn't matter. What is important is that [the

[11] Again, *Sry* indicates the mouse gene, *SRY* the human gene, "*SRY*" the same gene in multiple species.

[12] Koopman and colleagues' explanation for the mouse's sterility is that "the presence of two X chromosomes in a male mouse always results in sterility. . . . It was therefore not surprising that the sex-reversed transgenic mouse m33.13 was also sterile" (1991, 119).

mouse] acted as a male when put in a cage with female mice."[13] That is, the transgenic mouse tried to mate with the females. This single transgenic male intermouse (my term) was the pride of Koopman and colleagues' experiment, and its photograph was displayed on the front covers of *Nature*, *Science*, and the *New Scientist* and on the front pages of the *New York Times* and the *Boston Globe*.

To summarize the two experiments by Koopman and colleagues: In the first experiment there were three times as many XX females carrying *Sry* (six) as XX males carrying *Sry* (two). In the second experiment, there were twice as many XX females carrying *Sry* (two) as XX males carrying *Sry* (one). The *Sry* gene appeared to produce many more females than males, but still the gene became the poster "boy" of male-determining genetics.

Interestingly, the Koopman and colleagues (1991) article frequently referred to this fabricated *Sry* XX mouse as "normal." That is, the mouse exhibited "normal" size and weight, "normal" copulatory behavior (i.e., "he" copulated with females four times in six days), "normal" populations of Leydig cells, a "normal" reproductive tract (even though it did not produce sperm), and "normal" production of anti-Müllerian hormones and testosterone.[14]

More interesting, though, are the *Sry* females produced in the experiment by Koopman and his colleagues. Like the male mouse, the genome of these mice had also incorporated the *Sry* gene, and yet they displayed female physical characteristics. However, Koopman and colleagues treated these cases as anomalies that did not complicate the finding that *Sry* produces males:

> A further two XX transgenics, m32.10 and m33.2, showed an external female phenotype, yet both carried many copies of *Sry*. These mice have produced offspring and so have functional reproductive tracts and ovaries. They also provide further evidence, along with the transgenic XX female fetuses, that f741 [*Sry*] does not always cause sex reversal. Although there could be subtle rearrangements of the *Sry* gene making it non-functional, the possibility of this occurring in all these cases is remote. There are two more probable

[13] Interview with Giovanna Camerino, professor of human genetics, University of Pavia, Italy, October 10, 2000.

[14] Leydig cells produce the hormone testosterone when stimulated by another hormone. The anti-Müllerian hormone is a protein that inhibits the development of the ducts in a male embryo. If not inhibited, these ducts develop into the upper vagina, cervix, uterus, and oviducts. The ducts disappear as the male develops.

> explanations. First, these females could be mosaic for the transgene, with only a small proportion of the cells making up the somatic portion of the genital ridge carrying functional *Sry* gene copies. Analysis of XX <-> XY chimaeras suggests that females or hermaphrodites develop if less than about 30% of cells are XY. Secondly, the expression of the transgene could be affected by the position at which it integrates. Except for a few cases where locus-controlling regions are present, expression of transgenes almost always depends on their chromosomal location. These two alternatives can be examined by breeding from the adult XX transgenic females. Mouse m33.2 has not yet produced transgenic offspring. However, m32.10 has transmitted the transgene to female offspring, suggesting that it is not mosaic. (Koopman et al. 1991, 120)

In other words, Koopman and colleagues offer two explanations for the occurrence of *Sry* female mice. The first argues that the mice might be mosaics—mice that have incorporated *Sry* into some cells (perhaps less than 30 percent) but not into others. However, not only is one mouse (m32.10) a fertile and probably nonmosaic *Sry* female; she also initiated a new and genetically unique strain of mice that produce *Sry* females (Koopman et al. 1991, 120). This means that she incorporated *Sry* into her germ cells and passed on the *Sry* gene to her offspring. If *Sry* is the male-determining gene, how then can a reproductive female mouse carrying *Sry* in her cells still be a female? Here Koopman and colleagues pose a second explanation—that this particular *Sry* mouse is female rather than male because *Sry* is integrated in a position along the X chromosome that somehow prevents it from being expressed. This conjecture requires further research, since Koopman and colleagues could provide no evidence to support it.

It is not unusual for scientific experiments to raise more questions than they answer. Indeed, it is the norm. Why, then, did the article by Koopman and colleagues begin and conclude with the bold statement that *Sry* is sufficient to produce maleness? "It is now shown that *Sry* on a 14-kilobase genomic DNA fragment is sufficient to induce testis differentiation and subsequent male development when introduced into chromosomally female mouse embryos" (Koopman et al. 1991, 117).

Analyzing studies of genetic sex determination allows us to highlight the interpretations made by scientists in the process of experimentation. The experiments by Koopman and colleagues produced one XX-*Sry* sterile mouse with 22 percent–size testes (classified male) and three female-classified XX-*Sry* mice, one of which reproduced other females carrying

the *Sry* gene. Although *Sry* researchers noted that these different outcomes of the same gene did not fit with their original hypotheses, they still interpreted their results as confirming their initial hypothesis that *Sry* was the male-determining gene.

Examining the details of Koopman and colleagues' (1991) article also provides an opportunity to make other interpretations. One could, for example, raise an alternative plausible explanation for the experiments' complicated results: that is, that the presence of *Sry* females is evidence that genetic sex determination is more complex than the researchers claimed and that it involves interaction between many genes as well as other possible factors (e.g., ribonucleic acid, mitochondrial DNA, particular proteins in the area, or other epigenetic elements and events).[15] If these females have the *Sry* gene, could there be other genes or other factors that might be guiding the embryo toward femaleness? What is maleness; what is femaleness? Do genes determine sex? Or are things more complicated?

Of mice and women: Female sex-determination genetic studies

The dominant scientific view of sex determination from earlier in the twentieth century was that an embryo is female until something triggers a change that leads to the development of male testes (Jost 1953). As many feminist writers have pointed out, the development of females appears to be discussed by biological and medical texts in terms of passivity—in the absence of an active trigger required to induce male development, an embryo develops ovaries, a female secondary sexual characteristic (see, e.g., Martin 1991; Fausto-Sterling 1993a).[16] Early *Sry/SRY* experiments were based on this same assumption: embryos develop into female organisms if they lack the *Sry* gene to trigger the onset of male secondary sexual characteristics. Testes and ovaries distinguish males from females in this experimental world of human and molecular genetics. However, experiments in the 1990s countered this truism by presenting evidence for a separate gene involved in female sex determination.

In August 1994, Barbara Bardoni, working in Camerino's laboratory, and her collaborators reported finding a gene region on the X chromo-

[15] This complexity applies to even a limited definition of epigenetics. See the special issue of *Science* on epigenetics (Riddihough and Pennisi 2001), especially the exchange about the devolution of the term (Wu and Morris 2001).

[16] However, Cynthia Kraus finds that Drosophila sex determination research "does *not* provide a good example of androcentrism—but, rather, provides a counter-example" (2000, 152). She uses this case to argue for a reconsideration of feminist critiques of science.

some in the *DSS* (dosage sensitive sex reversal) region two doses of which are powerful enough to disrupt normal testis development in the presence of "SRY" (Bardoni et al. 1994, 500).[17] In an article titled "A Dosage Sensitive Locus at Chromosome Xp21 Is Involved in Male to Female Sex Reversal," published in the science journal *Nature Genetics*, Bardoni, Camerino, McCabe, and their colleagues propose a female-determining sex gene that operates at about the same time in the development of the embryo as the *SRY* gene. The embryo, they argue, is destined to become a male unless a gene in the *DSS* region counters the effect of *SRY*: "A group of four [human] patients found to have a working *SRY* gene nonetheless exhibited varying degrees of feminization, an event that should not happen if the maleness gene were the dominant determinant of gender. Three of the four displayed feminine external genitals, while the fourth had ambiguous genitals. All had been raised as girls" (Bardoni et al. 1994, 497). In these cases, a section of the X chromosome was doubled, giving them a double dose of the *DSS* gene. Two copies of a gene in the *DSS* region of the X chromosome can help push the fetal gonads, which have the potential to become either ovaries or testes, to become ovaries. Thus, an extra dose of the gene in males would undermine the efforts of the *SRY* factor to build testes. In a follow-up study (Swain et al. 1996), Camerino and colleagues proposed that a gene in the *DSS* region called "*DAX-1*" was responsible for undermining the "*SRY*" gene's action.

Of mice, humans, leakiness, and complexity

Researchers at Larry Jameson's laboratory at Northwestern University (e.g., Yu et al. 1998) subsequently conducted studies on *Dax-1* from which they argued that *Dax-1* is not a female-determining gene. Jameson and his colleagues reported that disabling the *Dax-1* gene in female mouse embryos did not prevent these embryos from developing into mice with ovaries. Moreover, they reported that male mouse embryos with disabled *Dax-1* genes became sterile. Their conclusion was that "*DAX-1*" is not an ovary-determining gene but rather has a critical role in spermatogenesis, the generation of sperm.

Camerino accepts the Jameson laboratory's claims for its mouse model but not for humans. She believes that species differ in their genetics of sex determination. *Sry/SRY*, she argues, acts very differently in mouse and man in the timing of the expression of the gene. Camerino further

[17] See also Dabovic et al. 1995; Graves, Camerino, and McLaren 1995; Zanaria et al. 1995; Swain et al. 1996.

contends that interactions between human *SRY* and *DAX-1* also differ from those between mouse *Sry* and *Dax-1*. Subsequent studies have shown that sex-determination genetics also differ between organisms in different phyla, thus reinforcing Camerino's position on mouse-human differences (Goodfellow and Camerino 1999).

Camerino's late 1990s studies have pointed to the vital role of *DAX-1* in sex determination in humans. In 1999, after Camerino's research on *DAX-1* raised questions about *SRY*'s power to transform embryos, Koopman (1999) hypothesized that the embryo did not develop into a male because the *Sry* mouse gene may be just one trigger in a series of steps that transform the XX embryo into a male mouse. Other possible explanations were that "*SRY* may act to repress genes that activate the female pathway of development, or to repress the repressor of the male pathway" (Koopman 1999, 840–41), or that "*DAX-1*" represses "*SRY*'s" action (Goodfellow and Camerino 1999).

Goodfellow and Camerino (1999) propose a hierarchic cascading view of sex determination, where *SRY* and *DAX-1* in humans act as triggers at the top of the hierarchy of a series of genes and activities necessary to the development of sex (here defined as ovaries and testes). Thereafter, many other events occur in the process of the organism's sex determination—for example, other genetic switches turn on or off during the embryo's development. These different genes and their expressions generate subsequent genetic actions, and a cascade of genetic switches and expressions produce the organism's final sex characteristics.

But there are more complications in sex determination and more questions than answers. Some scientists argue for proliferation in genes of promoter regions, structural genes, different forms of proteins from the same gene, and so on that complicate the picture of sex determination (Goodfellow and Camerino 1999). There is a long list of genes that are suspected of being involved in sex determination, and this list gets longer every year. In addition to *SRY* and *DAX-1*, these include *Wilm's tumor 1*, or *WT-1*, whose expressed protein has several different splicing alternatives and produces up to twenty-four different forms of the protein; *SF-1*, which is a nucleohormone receptor that is expressed in the hypothalamus, pituitary, gonads, and adrenals; and *Sox-9*, which is similar to *Sry*.[18] Then there are the interactions among the genes. As Camerino says, "*Everybody has found interaction of everything with everything*. With different results, etc., [sex determination] is complex, and the genetic term

[18] See also Parker, Schimmer, and Schedl 1999.

is leaky. Leaky. This is a prokaryotic genetics term.[19] It means that things are not that stable. They are not something strongly determined."[20]

Camerino believes that "*DAX-1*" is a female sex-determination gene high up in the hierarchy of sex determination (higher than *Sox-9*, *SF-1*, etc.) as *SRY* is high up in the hierarchy of sex determination for males (Goodfellow and Camerino 1999). Although this has not yet been demonstrated, she believes that future experiments could prove it to be true. In the meantime, Camerino calls "*DAX-1*" an antitestis gene because it has been shown that a double dose of it can turn off "*SRY*." The interactions among all these genes and proteins contribute to the instability, or "leakiness," in sex determination.[21]

What is sex? How is it determined? Does "*SRY*" cause males to develop? Does "*DAX-1*" cause females to develop? Does a cascade of molecular elements and interactions determine sex? At this writing it is thought that "*SRY*" and "*DAX-1*" are key genes that act initially to trigger male or female development in an embryo. However, it is believed that other genes also are needed to continue development toward male or female. These genes interact with one another, and the interactions can lead to other events. One possibility is that they could lead to hermaphroditic combinations of characteristics. Another possibility is that different cells in the same embryo have different genes, which then lead the embryo to develop into a hermaphroditic body. These embryos are called mosaics. At this point, genetic studies point to more complex interactions and unanswered questions rather than to any clear answers. These complex interactions are part of the leakiness of genetics.[22]

Do humans determine sex?

In the experimental arena of sex determination, molecular and human geneticists are the arbiters. But do genes and geneticists determine human sex identity? Physicians, psychiatrists, parents, courts, prison officials, and at one time the International Olympics Committee have all taken positions on human sex determination, often with little contest. Recently, social scientists, feminist theorists, queer theorists, and gay rights, intersexual,

[19] Prokaryotes are organisms like bacteria whose DNA is not enclosed in a nucleus. Eukaryotes are usually multicellular organisms whose DNA is encased in a nucleus.

[20] Interview with Camerino.

[21] Ibid.

[22] Ibid.

and transsexual activists have attempted to gain authority in debates about sex determination.

Intersexual social movement

The sex-determining gene experiments discussed were based on studies of human patients who exhibited genitalia and reproductive organs that did not fit neatly into standard categories of male and female. Often classified as intersexuals, people with sexually indeterminate bodies have become both subjects and objects of research and activism in the last ten years. Medical and research professionals have often treated intersexuals as residuals—people whose bodies do not fit commonly understood sex categories and need to be managed, explained, or made to fit into one or the other category. Recently, however, intersexuals have begun to organize to contest the medical definitions of their bodies and to work toward building collective identities to differentiate themselves from standard male and female categories and to establish intersexuality as a new and standard category of sex identity.

In the United States, medical practices have been used to manage intersexual infants and to surgically and chemically mold them to fit dimorphic sex categories (Dreger 1995). It has been common for doctors to "fix" sexually ambiguous babies soon after birth by surgically creating either male or female genitalia to accord (when possible) with internal reproductive organs. Sociologist Suzanne J. Kessler (1990) finds that decisions about which sex to assign to an infant were made primarily on the basis of what she calls aesthetic concerns, such as the length of the penis. If doctors guessed that the infant's penis was destined to be too small, then female genitalia were constructed. However, physicians saw their work as merely restoring the person's "natural" sex to him or her and, along with parents, regularly made decisions about these matters with the intention of protecting children from psychological damage. Kessler argues that these physicians displayed a "failure of imagination" (1990, 26) in attributing their decisions to nature: "Rather than admit to their role in perpetuating gender, physicians 'psychologize' the issue by talking about the parents' anxiety and humiliation in being confronted with an anomalous infant" (1990, 25).

Gender reassignment has not necessarily produced happy outcomes in adults, and some have organized themselves into the Intersex Society of North America (ISNA), which is based in San Francisco. In the late 1990s, ISNA member and founder Cheryl Chase and her colleagues generated a social movement to halt surgical practices on infants or at least to insist on more discussion before infants are transformed into one or the other

sex. Members of ISNA marched on medical schools to halt sex reassignment surgeries and published newsletters and press releases to educate the public about intersexuality. They have been the subject of *Nova* programs aired by the Public Broadcasting Service and of articles in major newspapers. In an October 14, 1996, press release titled "Intersexed Decry American Genital Mutilation," the ISNA compared intersexual infant surgery to African genital mutilation (see Chase 1996).

Chase and her ISNA colleagues have produced their own versions of naturalist baselines and categories to resist the medical practices that have pathologized and transformed their bodies.

> Intersex specialists are busily snipping and trimming infant genitals to fit the Procrustean bed that is our cultural definition of gender. . . . Surgical and hormonal treatment allows parents and physicians to imagine that they have eliminated the child's intersexuality. Unfortunately, the surgery is immensely destructive of sexual sensation as well as one's sense of bodily integrity. Because the cosmetic result may be good, parents and physicians complacently ignore the child's emotional pain in being forced into a socially acceptable gender. The child's body, once violated by the surgery, is again and again subjected to frequent genital examinations. Many "graduates" of medical intersex corrective programs are chronically depressed, wishing vainly for the return of body parts. Suicides are not uncommon. Some former intersexuals become trans-sexual, rejecting their imposed sex. (Chase 1996, 1)

By violating the natural body in their pursuit of a socially normal child, Chase contends, physicians and parents actually produce pathology.

Chase is a major protagonist in *Sexing the Body*, written by feminist biologist Fausto-Sterling (2000). Fausto-Sterling uses contemporary and historical biomedical scientific research on intersexuals and sexology to argue for multiple sex categories. In 1993 she published a provocative op-ed piece in the *New York Times* proposing that humans should have five sex categories rather than two (Fausto-Sterling 1993b). She argues that there is a physical continuity between the sexes of male and female, and rather than make bodies and persons fit into just two categories, male and female, she proposes that additional categories be embraced by medicine and society.[23]

[23] On third sexes, see, e.g., Serena Nanda (1989), who writes on the Hijras in India, and Gilbert Herdt (1996), who writes on Two-Spirit people (formerly called berdaches) in the United States.

Alice Domurat Dreger, Fausto-Sterling, Kessler, and the ISNA have made a difference. Intersexuals now have more support if they choose to speak out about their physiologies. Physicians do not automatically perform surgery on infants with some conditions, and parents are more involved in deciding whether or not to surgically transform infants with ambiguous genitalia into males or females (see, e.g., Navarro 2004). Nevertheless, two sex categories still dominate the choices and frames for physicians, parents, and scientists.

Transsexual activism

In their debates about biology and sex identity, many transsexuals insist on dichotomies but not those determined by anatomy or physiology. They argue that their physical bodies are not "natural" and that they instead feel more "naturally" to be members of the sex that does not accord with their genitalia. That which is usually taken as natural, the body, becomes unnatural, while that which is usually assumed to be socioculturally produced, gender, becomes natural. In this way, they argue differently from Chase and others who use bodies and biology to argue against dichotomies. Some transsexuals argue against the male-female dichotomy and for a wide range of gender identities, but they also argue for the naturalism of gender (e.g., Roughgarden 2004). Other feminist writers have argued that body and behavior are not separate entities and instead that materiality and gender identity are codetermined (e.g., Butler 1993). They argue against trying to adjust the body to fit an ideal gendered identity and for the complex and varied possibilities of the body—that is, for a transsexual position that speaks from outside the boundaries of the sex-gender binary.[24] Transsexuals, then, are not homogeneous in their positions regarding sex-gender dichotomies and naturalistic explanations for gender and sex identity. Despite or perhaps because of this heterogeneity, transsexuals contest the simplistic sex-gender, natural-social dichotomies in ways that emphasize the discursive construction of bodies and identities.

Analysis of data and discussion

What is sex? Will genetics be the final authority in answering this question? Sex gene experimenters have argued that "*SRY*" is an active element in the development of testes and that "*DAX-1*" is an active element in the development of ovaries. As stated earlier, to explain the ambiguities in *Sry*

[24] See also Stone 1991; Bornstein 1994; Bolin 1996; Feinberg 1998; Stryker 1998. For a history of transsexuality, see Meyerowitz (2002).

experimental outcomes on mice, some researchers have argued that in addition to *Sry* a cascade of other genetic and nongenetic factors and interactions are necessary to determine sex. But they do not question the assumption that testes indicate males and ovaries indicate females.[25] In contrast, some intersexual and transsexual activists, feminist theorists, and social scientists have contested this medical definition of sex. Although their definitions of sex are heterogeneous, transsexuals agree among themselves that possessing testes or ovaries does not determine their sex identities. Intersexual activists, biologist Fausto-Sterling, and psychologist Kessler use the existence of phenotypic features like ambiguous genitalia and reproductive organs as evidence that sex is not a male-female dichotomy. Using feminist and social scientific perspectives in light of research on transgender social movements, I now present an analysis of two processes through which sociocultural frames entered into the design of the sex-determination experiments I have presented above, and I examine how these frames influenced the analysis of the resulting data.

Experimental design: The normal defines the pathological and the pathological defines the normal

The *Sry* and *Dax-1* mouse experiments show that human and molecular geneticists used their own definitions of what constituted normal sex and pathological sex to design their scientific investigations. Despite their differences, both *Sry* and *Dax-1* researchers set up their initial experiments defining sex as a binary. They built this assumption into their experiments by choosing patients who presented themselves in the clinic with what were considered nonstandard sex phenotypes. In the mid-1980s, Page's laboratory used DNA from XX male human patients who were impotent (Page et al. 1987). Koopman and colleagues (1991) began with sterile male human patients with XX chromosomes whose common gene was used to develop a transgenic mouse model. In the early 1990s, Camerino and her colleagues (Bardoni et al. 1994; Zanaria et al. 1995) used data from female human patients with a "working *SRY* gene who nonetheless exhibited varying degrees of feminization" (Angier 1994, C1). In the

[25] An exception is Melanie Blackless et al. (2000), who argue against binarism even at the level of chromosome composition, not just gonads and reproductive organs. Phoebe Dewing et al. (2003) find differential gene expression between the developing brains of male and female mouse embryos and hypothesize that gonadal hormones may not be the only influence on male-female sex differences in brain development and behavior. This research should be carefully examined.

language of Camerino and her colleagues, "the double dosage of DSS in individuals with Xp duplications and a functional SRY gene . . . hampers repression of the ovarian pathway, leading to gonadal dysgenesis and phenotypic sex reversal" (Bardoni et al. 1994, 500).

These researchers' choices of patients for their studies set the parameters for their definitions of normal sex to be males or females who can heterosexually reproduce. The researchers would classify any variation from this to be pathological. However, as sociologists and historians have argued, classifications, categories, and taxonomies of scientific and medical knowledge are produced within specific historical situations. Further, categories of normal or healthy and pathological or ill are historically coconstituted categories, defined only in relation to each other (Canguilhem 1978). There is no normal without a pathological and vice versa. Michel Foucault (1970, 1978) argues that such classifications and taxonomies of scientific and medical knowledge constitute a map of the power relations of the particular time period and also have the power to normatively govern the ways humans act and feel.

Biological categories and classifications, then, are not natural, value free, or innocent. Sex categories in particular operate within socially prescribed systems of meaning. Human and molecular geneticists use their own sociohistorically located normative definitions of sex to design their experiments on sex determination. As a result, new molecular genetic experiments on sex determination do not challenge the previously determined socially defined categories. Instead, they give material form to socially defined ideas. By selecting particular human bodies in the design of their sex-determination experiments, these geneticists have reproduced their own taken-for-granted categories of sex.[26]

The genetic experiments I have presented are producing particularistic, not universalistic, knowledge. However, because of the power held by science and medicine in our world, the two sexes—male and female—are once again rendered natural and original, this time through the *Sry* and *Dax-1* mouse experiments. But power is a process that is never finalized. Just as feminists, queer theorists, and transgender activists are attempting to transform definitions of sex, this study challenges this power by showing how human and molecular geneticists insert normative societal assumptions into their scientific practices.

[26] See Hacking (1992) and Fujimura and Chou (1994) on self-authenticating practices in laboratory sciences.

Experimental data analysis: In search of the male-determining gene

Sry mouse experiments incorporated yet another set of assumptions: they focused on the male-determining factor rather than on the female. Hypothesizing that a gene common to XX men induces embryos to develop as males, the *Zfy* and *Sry* mouse studies were designed in an attempt to find that gene. The researchers found a version of that gene and inserted it into XX female mice to see if it would transform the females into males. When Koopman and colleagues (1991) produced a mouse with a small penis, they concluded that they had found the male-determining gene. They acknowledged that many more XX embryos had incorporated the *Sry* gene and developed as females rather than males, including one reproducing female that gave birth to female offspring carrying the *Sry* gene. However, in their frame of reference—the focus on male sex determination—the researchers relegated the female *Sry* mice to the status of anomalous data and omitted them from their published conclusions.

The researchers' focus on finding male sex determinants is in line with the history of sex-determination research. As stated earlier, it has been assumed that an embryo is female until something triggers a change, causing the development of male testes (Jost 1953). Thus, sex-determination research has been structured to search for the determination of the male phenotype (Eicher and Washburn 1986). Eva M. Eicher and Linda L. Washburn note that "the genetics of testis determination is easier to study [than ovary determination] because human individuals with a Y chromosome and no testicular tissue, or with no Y chromosome and testicular issue, are relatively easy to identify" (1986, 329).[27] While some experiments have countered the idea of passive female sex development, the idea of active female sex development has not entered easily or consistently into the literature (Fausto-Sterling 2000, 346). The research of Camerino and her colleagues on *DAX-1* joins this minority tradition, although it still represents sex as a binary male-female dichotomy. The field of sex determination is dominated, however, by *Sry* research and continues in the vein of early twentieth-century ideas.

Examining the awkward surplus from new frames of reference

The *Sry* mouse studies employed new molecular transgenic technologies to investigate the details of sex development in mice. The introduction of these new technologies made new signals possible. These new signals

[27] This point further distinguishes the research of Camerino and her collaborators on determinations of female sex.

could have led researchers to new insights about sex development. I show, however, that new signals read through old frames are not seen.

One fascinating aspect of empirical scientific research is its ability to surprise researchers with unanticipated results. Although philosopher and historian of science Thomas Kuhn argues that the paradigmatic frame of normal scientific practice does not aim at novelty and even suppresses it, he also acknowledges that it often yields "pre-novelties" (1962, 5–6) in the form of anomalies. Kuhn also argues that anomalies must be recognized—that is, recognized as new knowledge and not as errors or noise. Kuhn suggests that it is usually not the paradigmatic practitioners who recognize anomalies as novel, but instead it is the new generation of researchers, or even researchers from another field, who can see novelty because they are not immersed in the governing paradigm.

Anomalies can, in Kuhn's schema, lead to the production of both new knowledge and a new paradigmatic order, a new form of normal science. However, in Kuhn's discussion the sources of the differences in perception required to recognize novelty remain within the science community, albeit in a different generation or discipline. Historian of science Nancy Stepan (1993) goes beyond Kuhn to argue that paradigms are not just limited by a scientific community's set of theories and practices but also by social and cultural metaphors. In contrast to Kuhn's intellectualist explanation that a paradigm changes with the accumulation of a critical mass of anomalies that cannot be explained by the paradigmatic frame, Stepan argues that it is often through social, political, or economic changes in society that both scientists and citizens come to see that cultural metaphors have governed how we perceive reality and that they no longer apply.

The data produced by the *Sry* and *Dax-1* mouse experiments, the questions raised about sex/gender by transgender and feminist activists, Kuhn's discussion of anomalies, and Stepan's 1993 revision of Kuhn's ideas together suggest that there may be data that tend to be ignored because they do not fit the frames of reference of their observers. Considering this awkward surplus, I argue that the introduction of new frames of reference may illuminate results of experiments that have been ignored in the investigation's conclusions.[28] In this way, the concept of awkward surplus can aid in the

[28] This use of *frames of reference* is taken from sociologist Erving Goffman's (1986) argument that humans develop and use frames of interpretation to organize and make sense of the events, activities, and phenomena to which they attend in everyday life. Goffman's frames allow us to think of scientists as acting through their formal and tacit scientific training and also through their sociocultural contexts and experiences.

rereading of experimental conclusions and thereby produce alternative interpretations with different social consequences.

Reexaminations of study results such as those presented here provide opportunities for natural scientists, social scientists, and other parties to attempt to work differently and collaboratively to produce new explanations. Using the notion of awkward surplus, social scientists and social activists can fill a role similar to that of scientists from another field, those whom Kuhn sees as potential innovators—people who can see anomalies as sources of novel ideas and findings because they bring different assumptions to the table. With respect to the *Sry* and *Dax-1* studies presented here, I apply my knowledge and skill in understanding social frames of meaning to explore whether, when, where, and how these frames affected the researchers' scientific perception. As Haraway argues in "Situated Knowledges" (1988), other actors with stakes in a problem should be involved in studying it.

In examining *Sry* experimental data, I am attempting to salvage the experimental results that sex gene researchers first acknowledged and then chose to ignore. That is, although the researchers (Koopman et al. 1991) noted that some mice did not perform according to their expectations, they failed to conduct further experiments to try to make sense of these anomalous results. Koopman and colleagues chose instead to continue to construct their follow-up experiments as if *Sry* caused maleness in mice. Their subsequent studies presented additional complexities and ambiguities that the scientists could not explain. One researcher, Camerino, continually referred to some of the results as "bizarre."[29] Although researchers attempted iterations to make the results fit their original assumptions, these subsequent experiments did not answer their questions, and they decided to wait for "better" experiments.[30] *Better*, I argue here, refers to experiments that will yield results that make sense to them within their frames of reference.

After identifying an awkward surplus of results in the data, my next step was to explore new interpretations. By reviewing the data without thinking about sex as a binary category, I saw that the last fifteen years of research on "*SRY*" and "*DAX-1*" have provided much evidence for complexity in the genetics of sex determination. Recent experiments have raised the possibility of a proliferation of genes in promoter regions of the chromosome, of structural genes, and of different forms of proteins being produced by the same gene, all of which complicate the question

[29] Interview with Camerino.

[30] Ibid.

of sex determination. There is by now a long list of genes suspected of being involved in sex determination, and this list grows longer every year. If we also consider the interactions among these genes, sex determination at the genetic level is steadily increasing in complexity. When we add the interactions of genes with various proteins, developmental pathways, cell signaling pathways, and many other parts of cellular, organismal, and environmental parts and processes that are fast becoming the territory of a new field called "systems biology" (Fujimura 2005), the complexity of sex determination escalates even more.

A key characteristic of complexity is instability. Using a term first developed in the field of prokaryotic genetics, Camerino argues that sex determination is "leaky," by which she means unstable or not strongly determined.[31] That is, there is no single pathway through which sex is genetically determined. Indeed, there may be many pathways with multiple different genes involved in each pathway. And although Camerino believes that there is a hierarchy of pathways with "*SRY*" and "*DAX-1*" involved at the top of the hierarchy, this argument must be verified.

In contrast to the geneticists' view, I suggest that a feminist, social scientific, or transgender analysis might consider the many sex variations as resulting from multiple developmental pathways that involve genetic, protein, hormonal, environmental, and other agents, actions, and interactions. These variations need not be represented as outliers, residuals, anomalies, or pathologies in a binary system. Instead, a reanalysis of *Sry* and *Dax-1* mouse research shows that genetics can produce phenotypic variations suggesting that sex is a fluid concept, not a binary concept incorporating only the conventionally gendered sexes of male and female.

In summary, the concept of awkward surplus is useful, first, to help us attend to unanticipated results that are recognized as problematic or awkward by experimenters and are thus ignored in their conclusions. Second, the concept provides an opportunity to reexamine unexpected experimental results either by using different frames or perspectives or by reexamining them in conjunction with data from other sources. Third, the examination of awkward surpluses provides a space where scientists and social scientists can work together in the production of new knowledge.

Who adjudicates the awkward surplus?

In addition to the interpretations of geneticists in the original *Sry* mouse study (Koopman et al. 1991) and my reanalysis, there may be other in-

[31] Ibid.

terpretations. The designation of awkward surplus and possible multiple explanations of what the awkward surplus means raise other epistemological and methodological questions. How do we decide which interpretations are valid? If prescribed systems of meaning frame our very perceptions of matter, is my alternative interpretation not just as situated in particular sociocultural assumptions as those of the biologists I study? With respect to the concept of awkward surplus in particular, how do we adjudicate whether an awkward surplus provides useful or useless information? And who should adjudicate?

Answers to these questions in the social studies of science, medicine, and technology are many and are heatedly debated. Some science studies scholars argue that our job is not to decide what is valid knowledge but to study how each possibility fares in the struggle for scientific authority. These scholars prefer to descriptively analyze scientific practice and struggles for authority without taking normative positions on knowledge outcomes (e.g., Lynch 2001). However, other science studies have also shown that many nonscientists have already intervened in the making of science. Religious groups have asserted their agendas, sometimes supporting the programs of particular scientists (Shapin and Schaffer 1985) and sometimes intervening against the programs of particular scientists through control of research-funding processes of government agencies such as the National Institutes of Health (NIH) and the National Science Foundation (Borenstein 2004). Private industrial concerns have inserted their agendas through their in-house research or through institutional funding of research in private institutes and research universities (Krimsky 2003). Governments have also selectively influenced the development of scientific knowledge in particular directions (MacKenzie 1993; Eden 2003). Beyond these overt exercises of political power in the making of knowledge, social studies of science have demonstrated the introduction of political and cultural agendas into scientific research through subtle and unintentional processes. Indeed, as Stephen Jay Gould (1981), Stepan (1993), and Hall (1976) argue, throughout history it has been difficult to separate scientific efforts from commonly accepted cultural knowledge.

Given the past and present roles of power and partiality in the production of knowledge, feminist scholars of science in particular argue that science analysts should play a part in the struggle for authority by taking positions and supporting some knowledge claims over others. Haraway (1988) argues that those who have the greatest stakes in a knowledge claim should act collectively to produce that knowledge. Harding (1998) has provided epistemological arguments for the production of new kinds

of knowledge by participants who are not professional Euro-American scientists. Scientists themselves take heterogeneous positions. Some argue that science should police itself, while others argue that there is a place for nonscientists in scientific knowledge production.

However, the epistemological frames of Haraway (1988) and Harding (1998) still leave us with the questions of who qualifies as a stakeholder in a particular problem and how those stakeholders who are not professional scientists can participate in the making of science. For instance, the Bush administration's conservative religious policy makers and backers argue—and have acted upon the view—that they have a stake in scientific research. They have taken up positions on stem cell research and influenced NIH decisions about which projects to fund.[32] In the case of sex-determining gene research, I argue that intersexuals should have some authority in the making of knowledge of sex. However, the Bush administration could similarly argue that the religious ultraright should also have a place at the table. Is adjudication possible, or is it simply a battle of wills and power? In the battle of power-knowledge (Foucault 1980), barriers to participation are usually high.

The problem of who should and can authorize science is a question that appears to be answerable only historically (e.g., Fujimura 1998). Nevertheless, some science studies scholars are attempting to wrestle with this problem prospectively in epistemological terms and practical terms.[33]

A critical sociomaterial approach

This analysis of sex-determination research demonstrates the critical sociomaterial approach to the study of science, a theoretical approach that incorporates ideas and lessons from feminist theory and the social studies of science. I have included an analysis of science that incorporates the sociocultural frames of reference of researchers who have stakes in and perspectives on a particular scientific problem. I call for social scientific or feminist analyses of science to include an examination of the production of the materiality that supports scientific claims. I propose that feminist social scientists and activists should include the exploration of

[32] On February 18, 2004, over sixty leading scientists—Nobel laureates, leading medical experts, former federal agency directors, and university chairs and presidents—signed a statement voicing their concern over the misuse of science by the Bush administration. The Union of Concerned Scientists has a Web site that solicits signatures of additional U.S. scientists in support of this effort. See http://www.ucsusa.org/scientific_integrity/interference/scientists-signon-statement.html.

[33] On epistemological approaches, see, e.g., Haraway (1988), Barad (1998), Harding (1998), and Longino (2001). On practical approaches, see, e.g., Rosser (2000).

the materiality of sex in their analyses. The biology of sex is too important to leave to biologists alone because they usually are not trained to attend to and analyze how sociocultural frames influence their own experimental processes. This critique is exactly what feminist, social scientific, and humanist analyses can provide. Their different frames of reference may suggest new interpretations of evidence and even new experimental designs.

The methods for analyzing the material production of science include reading research articles in search of data that could be meaningful in a frame or context of analysis different from that of the original experimenters and/or observing scientists at work producing scientific knowledge in the laboratory or the field and identifying and examining awkward surpluses of data that do not fit within the researchers' frames of reference. This analytical approach requires an epistemological argument for the claims made in the new analysis and a discussion of the proponents' stakes in their role as knowledge makers.

Conclusion

I have employed a critical sociomaterial approach to reexamine scientific mouse experiments on sex-determining genes, especially *Sry* and *Dax-1*. I have provided a critique of the investigations and an analysis of some of the investigators' awkward surplus data. This approach to science incorporates theoretical efforts to move beyond reading society onto nature and reading nature onto society. It does not impose sociological categories onto the natural sciences, nor does it impose biological categories onto the social sciences. Instead, it argues for a collaboration that gains from different expertises.

The results of this reexamination demonstrate that the design and analysis of molecular genetic experiments are inhabited by sociocultural meanings and understandings. In the case of genetic sex determination, scientists used the social categories of "normal males" and "normal females" to design their experiments and protocols, and they reproduced these categories in their experimental processes.

My reexamination of research in sex determination also shows an awkward surplus of data that researchers ignored in their conclusions from the *Sry* mouse experiments. They did not view some experimental results as findings because those results did not fit their cultural expectations.

In contrast, from the perspective of feminism and social science as well as of research on transgender movements, I suggest that these residual data provide significant information on the actions of sex genes.

Instead of viewing the results as bizarre, I suggest reinterpreting the residual data to illuminate genetic instability (leakiness) and possible multiple pathways of sex development as explanations for the variations in body phenotypes that do not fit the binary male-female norms. *Sry* and *Dax-1* mouse experimental results that fall outside the experimenters' frames of reference may be legible within other frames. Sex may be highly variable and more fluid than geneticists (and many of the rest of us) anticipate.

I argue for the examination of the awkward surplus in scientific data as a valuable research tool. Reconsideration of data and conclusions would use frames of reference different from those of the original experimenters, frames taken from other actors and realms of life.

The concept of awkward surplus provides science studies with a way of engaging with material agency. Even within the cultural framing of understandings of nature in a particular period, we find biological outcomes that stand clearly outside scientists' abilities to control or explain them. The concept of awkward surplus provides a theoretical and methodological framework for thinking about anomalous results when meaning has not quite become fixed.

Awkward surplus is also useful for thinking about how feminist and other social theorists and activists can participate in creating knowledge about materiality. The work of transgendered activists and some feminist theorists to promote the acceptance of variations in bodies and the normalization of their own bodies can be useful in the production of molecular genetic research. Scientists, too, must have an opportunity to cross the divide. They can use the work of feminists, queer theorists, and transgender activists to think creatively about their own research surplus and their accepted protocols for producing knowledge. The awkward surpluses of scientific data indicate complexities that fall outside the structures of scientific paradigms and some social frames of meaning.

With respect to sex itself, these readings of novel data suggest that the variations in and complexities of sex development raised by feminist analysts at the levels of human behavior, bodies, hormonal systems, embryos, cells, and chromosomes are replicated at the level of genes. Sex, even at the genetic level, is a sociomaterial process and product.

Given this conclusion, my study of the production of the materiality of sex joins arguments in feminist studies for the collapsing of the sex-gender (qua biology-society) distinction. Instead of treating sex as biological and gender as social, I argue that sex, like gender, is a sociomaterial product. Sex-determining gene research and the political actions of transgendered activists introduce moments of ambiguity and transgression that

disturb the dichotomies of male-female, sex-gender, and nature-culture. Highlighting the social aspects of sex contests assumptions about gender and sex and thereby about the sex-gender split.

My investigation is an argument for broadening our social imaginaries—our definitions and understandings—of the material, the natural. A critical sociomaterial view of sex integrates sociocultural and historical investigations of the production of the material (e.g., the complexities and variations of sex physiologies and genetics) with diverse social imaginaries about sex and bodies proposed by feminists, queer theorists, intersexuals, and others. In this approach, we study and juxtapose the actions and interactions of social activist groups, social theorists, biologists, bodies, and genes in order to understand the collective, contentious, contradictory, and interactive crafting of sex in humans.

I do not mean to argue that the natural should be the foundation for substantiating, explaining, or changing existing gendered arrangements in society. Social imaginaries should be enough for promoting an acceptance of diversity. Historical examples of efforts to use natural differences to justify social hierarchies provide yet another reason for eschewing biology as foundational for social practices. The recent rise of evolutionary psychology is the latest in such efforts to produce natural arguments for social practices and hierarchies.

Nevertheless, demonstrations of the sociomaterial production of sex, the Möbius strip production of sex, are useful for maintaining our awareness that natural categories are also social categories. Further, even as our current language of analysis maintains the division between the natural and the social, the point of a critical sociomaterial approach is to move in the direction of a language where there is no division, where we are always conscious that the natural and the social are not separated.

For example, we need to think of the categories male and female not as representing stable, fundamental differences but as already and always social categories. They form a set of concepts, a set of social categories of difference to be deployed for particular purposes. Ergo, what counts as male and female must be evaluated in their context of use. The categories male and female, like the categories men and women, may be useful for organizing particular kinds of social investigation or action, but they may also inhibit actions.

A critical sociomaterial approach that joins awkward surpluses from the laboratory with the experiences of people in the world opens up opportunities to challenge the taken-for-granted scientific categories that help to construct or maintain definitions of similarity, difference, and pathology. This is particularly important today, when new biotechnologies are being

used to link disease and behavioral genes with particular social categories of race and ethnicity.

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