

Plasticity and Programming: Feminism and the Epigenetic Imaginary

The “living being does not simply perform a program,” writes French feminist philosopher Catherine Malabou in a 2016 *Critical Inquiry* essay on the new science of epigenetics (435). Epigenetics, she argues, “unsettle[s] the equation between biological determination and political normalization” (432). For feminists, the science of epigenetics holds a “power of resistance,” offering political possibility through the “deconstruction of program, family, and identity,” “fractur[ing] the presumed unity of the political subject” (438). Similarly, in her 2014 “Politics Materialized: Rethinking the Materiality of Feminist Political Action through Epigenetics,” theorist Noela Davis proffers epigenetics as a resource for feminists working to “rethink matter, its vibrancy, dynamism and agency” (63). Feminist and queer theories that understand bodies as “performative processes of materialization rather than given or inert bases to be worked on by cultural activity” (65), she argues, may find empirical support in the new science of epigenetics.

Epigenetics is a new, methodologically diverse, controversial, and fast-moving scientific field attached to powerful progressive and reconstructive social and political imaginaries—that is, philosophies of life and materiality—at the intersections of the life and social sciences. Malabou and Davis exhort feminists to approach epigenetics as a grounding for feminist theory, suggesting that epigenetics might serve feminist theories of nonbinary, plastic, and diversity-affirming conceptions of sex and gender. Cultivating biological data as a source with which to imagine feminist futures and produce alternative conditions of possibility for political action, feminist new materialists such as Malabou and Davis seek to break down the distinction between the political and material and to build feminist theory out of the blood, guts, brains, and epigenomes that make up what they conceptualize as the very materiality of scientific investigations. The “thinking with” the wetware of biology that new materialist theorists seek to cultivate, however, raises worries of a decontextualized approach to the posits of biology and an uncritical

Thank you to Tess Consoli and Andrew Mazzaschi for editorial assistance and to Robyn Bluhm, John Dupré, Ginger Hoffman, colleagues at the Wesleyan Center for the Humanities, and the journal’s anonymous reviewers for valuable comments on earlier versions of this essay.

[*Signs: Journal of Women in Culture and Society* 2017, vol. 43, no. 1]

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embrace of a material grounding for political action. The case of epigenetics makes this particularly clear.

Using methods from history and social studies of science, this article contributes to the empirical literature on the heterogeneous transformations wrought by the science of epigenetics across the life sciences. The first part of the essay analyzes the entry of epigenetics into theories and modes of inquiry in research on sex differences in the mammalian brain and in social behavior. This is a classically charged field of study with deeply contested core assumptions and a history of ideological deployment within wider social debates about male and female natures (Fine 2010; Jordan-Young 2010). Attending to history and to local, situated practices in the epigenetics of sex in the brain significantly recasts the widely perceived relationship between epigenetics and plasticity-affirming biologies. I demonstrate that in contrast to a reigning discourse that associates epigenetics with biological plasticity related to social and environmental context, current research on sex differences in the mammalian brain understands epigenetics as a mechanism by which genes and hormones produce and maintain sexual dimorphism. Rather than making sexual phenotypes more complicated, or making them less determinant and more variable, epigenetic factors, in this prevailing model, work to fix and direct dimorphic development by encoding binary patterns of gene expression in the brain.

The observation that epigenetics has, in practice, only replicated and amplified deterministic and binary models of the biology of sex differences in the brain and behavior does not, of course, imply that feminists should abandon the plasticity- and diversity-affirming potential of epigenetics. But it does highlight the importance of careful and critical attention to the unfolding meanings of epigenetics research in context and practice before embracing epigenetics as a foundation for feminist theoretical and empirical claims. Taking up feminist provocations regarding the possibilities for epigenetics to inform feminist theory, the second part of this essay considers the opportunities for and dilemmas of feminist engagements with the still-nascent field of epigenetics. Intellectual exchange between epigenetics and feminist theory, I argue, cannot be based merely on a posture of fascination or on the recognition of certain shared proclivities. Epigenetic science is contested, and the political imaginaries attached to it are plural and complex. Hence, I argue for the importance of feminist empiricist approaches to epigenetic science, including critically assessing the strength of scientific claims relevant to the interaction of gender and epigenetic mechanisms; strategically challenging the methods, concepts, and empirical findings of epigenetics research; and submitting feminist propositions (including those regarding the concept of plasticity itself) to test.

Plasticity and programming: Epigenetics, sex, and the brain

Epigenetics refers to molecular mechanisms that regulate DNA expression in response to genetic and nongenetic cues. Studies of epigenetic mechanisms arrived coincident with transformations in the life sciences in the aftermath of the completion of the major genome projects. Increasingly over the past decade, biomedical researchers have turned from the question of DNA sequence to that of DNA regulation. Common epigenetic mechanisms include methylation, acetylation, microRNAs, and histone modification, all of which function as molecular cofactors that repress or activate DNA expression. DNA microarray technology allowing researchers to easily assess the presence of epigenetic regulation in particular tissues and regions of the genome has facilitated the rapid movement of epigenetic research into nearly every field of the biomedical sciences.

Iconic epigenetic studies involve riveting claims of dramatic changes in epigenetic markings as a result of environmental exposures. Rat pups stimulated by maternal licking gain epigenetic markings at key loci involved in stress regulation and become low-anxiety adults (Szyf et al. 2005). Members of poorer classes in Glasgow, Scotland, exhibit lower global methylation levels, a common measure of epigenetic regulation, than do the wealthy (McGuinness et al. 2012). A series of Scandinavian studies suggest that a grandparent's experience of famine may be recorded in the epigenome, altering descendants' health even without changes to the DNA itself (Bygren, Kaati, and Edvinsson 2001). Emphasizing the reactivity and responsiveness of the body to social and environmental influences, such findings make it seem plausible that epigenetics could offer a rich and provocative theoretical frame for understanding gender-sex at the level of the body.

In the spring of 2015, a new epigenetic study suggesting a thrilling and intellectually challenging rewriting of long-held paradigms of profound and fixed sex and gender differences in the biology of the brain appeared in the journal *Nature Neuroscience*. The study, "Brain Feminization Requires Active Repression of Masculinization via DNA Methylation" (Nugent et al. 2015), used the emerging tool kit of epigenetics to examine sexual differentiation in the preoptic area (POA) of the rat brain. A locus of great interest and contention among scholars of sex, gender, and the brain, the POA is implicated in sex differences in rodent reproductive behavior and is claimed to be among the most sexually dimorphic regions of the mammalian brain.

The study seemed to challenge received understandings of sex differences in the POA at several levels. In apparent contrast to a view of male and female brains as dimorphically hardwired during early development, the study found that epigenetic mechanisms permitted the POA to be "highly modifiable," exhibiting "rapid" alternations in levels of epigenetic markers in re-

sponse to “changes in excitability” (Nugent et al. 2015, 690). Against the long-held view that femaleness is the default in the absence of masculinizing factors that repress feminization, the authors argued that epigenetic mechanisms reveal that brain “feminization is an active and ongoing repression of masculinization and thus reversible” (696). They concluded that this finding is “evidence for the duality of the brain, with some arguing for the simultaneous presence of both male and female circuits or phenotypes” (696). Finally, at odds with a history of focus on documenting and quantifying sex differences in the brain, the authors positioned epigenetics as a mechanism that can function to equalize levels of gene expression between males and females. While 70 genes showed sex differences, the other 381—which they termed “convergence genes”—maintained equal levels of expression with the aid of epigenetic markers (695).

The study, from the laboratory of neuroendocrinologist Margaret McCarthy, could be read as suggesting the possibility of much greater dynamism and variability in sex and gender phenotype in the mammalian POA than had previously been appreciated. It hinted that epigenetics may reveal that differences once understood as hardwired are perhaps more accurately understood as trip-wired, held in place by delicate and contingent epigenetic processes subject to reversal. Such plasticity-affirming, gender-bending findings speak to the highest hopes of feminist scholars for the new science of epigenetics: that epigenetics may counter traditional ideological conceptions of male and female differences by documenting a diversity of sexual phenotypes and that epigenetics may provide methods for studying the biological embodiment of gender by yielding mechanisms for the environmental and social mediation of sex.

Yet the authors themselves, and the scientific community, did not embrace this vision. In the scientific and public discussion that followed the publication of the study, scientists interpreted its findings as further confirmation of the long-standing paradigm of hardwired and dimorphic sex differences in the brain. As the study’s lead author, Bridget Nugent, said, “my hope is that these studies have taken us one step closer to fully understanding how and why males and females are so different” (in *Epigenie* 2015). A 2015 article in *The Scientist* titled “Female Brain Maintained by Methylation” (Azvolinsky 2015) framed the study as explaining how “differences in male and female rodent sexual behavior are programmed during brain development” and as offering further evidence of how “male hormones unleash the male program.” The article asserted that “these latest findings suggest that there may be more sex differences in the rodent brain than previously thought.” Sex-differences researcher Geert de Vries said of the study: “our understanding that the female state of the brain is the default still stands.

What changes now, because of this study, is our thinking as to how that default state is preserved” (in Azvolinsky 2015). In short, researchers saw the study as corroboration for long-standing models of the brain’s stringent maintenance of sexual dimorphism and as the discovery of new sex differences in the previously unexplored realm of epigenetic markings rather than as a diversity- and plasticity-affirming conceptual framework for understanding the materiality of sex and gender.

Close analysis of the dynamics of the integration of epigenetics research into recent basic biological studies of sex, gender, and sexuality illuminates why. Very broadly, there are two explanatory projects in epigenetics-centric sciences. One examines how epigenetic mechanisms play an integral and generalized role in the unfolding of human development, mediating between hormones and genes to execute developmental processes such as growth, brain development, puberty, menopause, and aging. This is presently most recognizable as a research program in developmental genetics. The second explanatory project looks at how, in response to environmental cues, discrete epigenetic mechanisms at particular sites on the genome can causally contribute to individual phenotypic outcomes, including those conceptualized as pathology, such as high anxiety or obesity. This work is currently most legible as a research program at the intersection of environmental and genetic epidemiology.

These two projects—the study of the long-lasting effects of early epigenetic programming and the study of the short-term or fleeting role of epigenetic mechanisms in the plastic mediation of environmental cues and gene expression throughout the life course—reflect an already well-established explanatory framework for approaching the biology of sex that makes room for developmental plasticity as well as stability.¹ Originating at the turn of the twentieth century with the discovery of sex hormones and sex chromosomes, this explanatory framework posited a model of sex in which genes and chromosomes determine initial sexual fate and gonadal hormones such as estrogens and androgens govern sexual differentiation and secondary sexual traits (Richardson 2013).

Hormones, the twentieth-century avatars of sexual plasticity, function by conveying biochemical messages from cell to cell, leading to changes in

¹ The alignment of the explanatory project of epigenetics with the activational-organizational model has its historical origins in the emergence of molecular epigenetics at the interstices of endocrinology and genetics. Studies of gene expression in relation to sex hormones played an important and proximate role in the conceptual origins of studies of epigenetic mechanisms such as methylation in the 1980s. An early example is Saluz, Jiricny, and Jost (1986).

gene expression and metabolic function. Mid-twentieth-century studies of sex hormones in relation to rodent sexual differentiation of the gonads and brain led to the now-dominant activational-organizational explanatory framework for the biology of sex. According to this model, sex hormones such as androgens and estrogens have two causal repertoires. An organizing effect occurs during the development of an organism and irreversibly primes or programs tissues to respond in certain set ways to later physiological events. Sexual differentiation of reproductive organs during early fetal life is an excellent example of this. An activating effect is one produced by circulating hormones in the mature organism. For instance, adrenalin increases heart rate, and estrogen levels trigger the release of luteinizing hormone in women, inducing ovulation.²

In the twentieth century, researchers advanced a linear model of genetic and hormonal sex determination in which the presence of the Y chromosome and testosterone during early development masculinized a body plan that was by default female. Then, throughout the life course, sex hormones regulate genes to maintain sexual differentiation and sex-related functions. Today, epigenetic mechanisms form the linchpin of an even more dynamic model in which hormones and genes interact throughout the life course to regulate sex-differentiating processes. Yet the received explanatory framework of organization and activation, and of programming and plasticity, persists.

Epigenetics first entered basic biological sex research at the turn of the twenty-first century, during a paradigm shift in the life sciences—a move from studies that assume a few determinant factors to more nonreductionist, postgenomic approaches that invite a wide repertoire of cofactors in biological explanations.³ In 2012, geneticist and neuroscientist Arthur Arnold coined the term “sexome” to describe this postgenomic understanding of the biology of sex (Arnold and Lusia 2012). Arnold and colleagues describe the sexome as an interactive, dynamic network composed of many sex-biased factors, including epigenetic ones, involved in all life processes. The terminology of the systems sciences—“complexity,” “pulsating networks,” “dynamic interactions,” and “emergence”—pervades new epigenetics-centric models of the biology of sex:

We envision the function of cells, tissues, and individuals to be controlled by complex intersecting causal pathways, in which specific phys-

² While the relevance of the activational-organizational model for understanding the sexual differentiation of the human reproductive organs is well established, its application to human gender and sexuality in the brain remains critically contested (Jordan-Young 2010; Joel 2012).

³ On “postgenomics,” see Richardson and Stevens (2015).

ical events cause changes in other events. . . . In this analogy, functional gene networks pulsate with activity, with specific nodes increasing and decreasing in their activity, stimulating and inhibiting each other, creating a dynamic net of interactions that lead to emergent phenotypes (such as heart rate, fat and energy metabolism, etc.). Sex differences in gene networks, and in the phenotypes that they control, are created when the activity of some nodes is greater in one sex than in the other; the sex differences in network functions are caused by sex-specific factors acting in the network. The totality of sex-biased factors in the network comprise the sexome. (Arnold, Chen, and Itoh 2012, 70)

Adherents contrast a “traditional” view of genetic and hormonal determination of brain sexual differentiation with their “modern” view, in which many cofactors, including epigenetic ones, orchestrate sex-specific processes throughout the brain:

[The] traditional view, seductive in its simplicity, must now be replaced. Sufficient new evidence has accumulated to warrant a shift away from the old serial model and toward a more complex and nuanced model in which numerous sex-specific factors, hormonal, genetic and epigenetic, act in parallel to cause or eliminate sex differences in the brain and other tissues, by mechanisms that frequently are region specific and heterogeneous in terms of their intracellular mechanisms and mode of cell-to-cell communication. The modern view emphasizes a diversity of proximate mechanisms and an interaction of multiple sex-specific factors in many brain regions. (McCarthy and Arnold 2011, 677)

In this picture of sex in the brain, hormones, genes, and experiences play parallel and interacting roles to create and maintain sex differences. Epigenetic mechanisms comprise a key element of postgenomic models of the biology of sex. As visualized in figure 1, in such a model long-lasting sex-specific epigenetic modifications in early mammalian brain development result in “sexually dimorphic epigenomes.”

Over the past decade, epigenetic mechanisms have emerged as an explanatory resource in studies of the biology of sex and the brain in two principal ways. First, epigenetics provides a new tool kit for elucidating the molecular mechanisms of hormone-gene regulation. Take, for instance, the estrogen receptor α (ER α) gene, a major target of current research on the epigenetics of sociosexual behavior regulation in the brain. Changes in levels of expression of the ER α gene alter sensitivity to estrogen. A substantial line of present research examines how epigenetic mechanisms such as methylation and his-

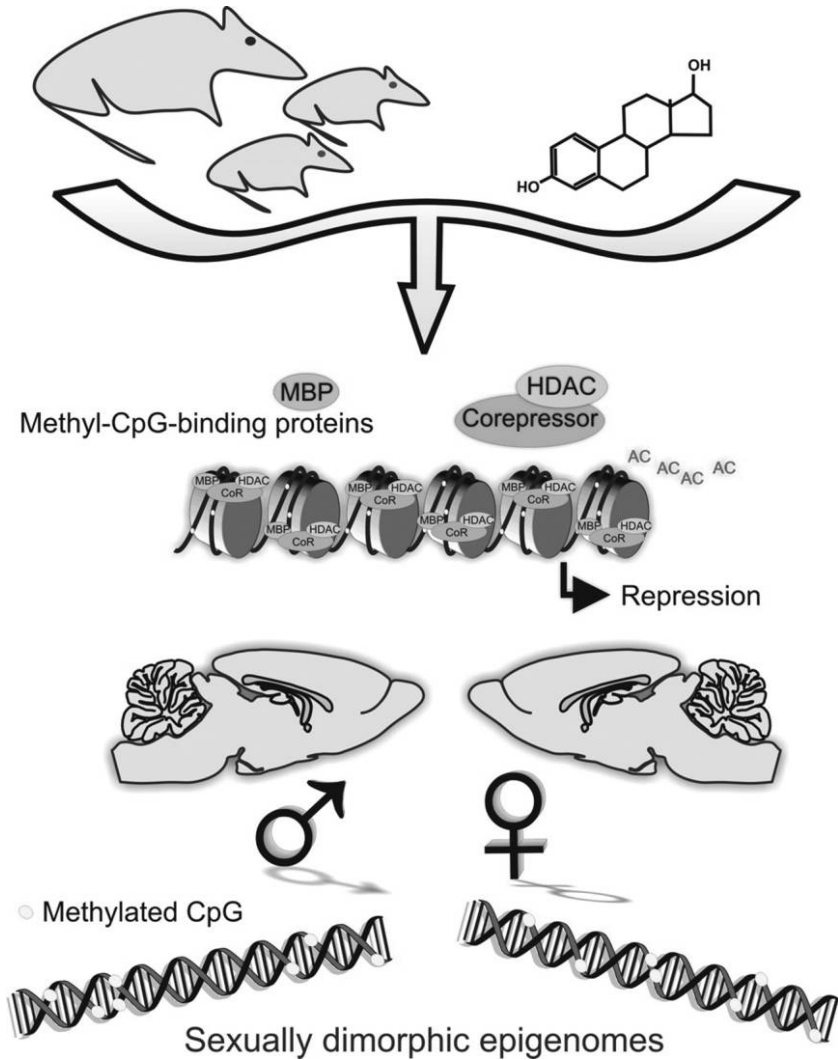


Figure 1 Sexually dimorphic epigenomes. “Emerging evidence suggests sex differences in at least four related parameters: (1) DNA methylation patterns, (2) methyl transferases, (3) methyl-binding proteins, and (4) corepressor proteins, all of which can contribute to lasting differences in the brain and behavior” (McCarthy et al. 2009, 12817). © 2009 by the Society for Neuroscience. Reprinted with permission. A color version of this figure is available online.

tone acetylation of the $ER\alpha$ gene vary between males and females over developmental time and in different regions of the brain. In mice, sex differences in methylation and histone acetylation at the $ER\alpha$ gene caused by exposure to the steroid estradiol have been observed in the POA and mediobasal hypothalamus during early development. Altering exposure to estradiol during the prenatal period changes the rate of DNA methylation,

in turn altering levels of ER α gene expression in males and females (Matsuda 2014). Another hormonal target of epigenetic investigation, oxytocin, is involved in lactation and also theorized as a central component of human social and emotional regulation and bonding. Oxytocin has sex-specific effects in males and females. Researchers are investigating how epigenetic mechanisms mediate sex differences in fetal and infant response to maternal stress or separation (Alves et al. 2015). Correlating early epigenetic programming with measures of oxytocin and variation in social behavior in adults, these studies position epigenetics as a link between early adversity, hormonal changes, and sex differences in adult behavior.

Second, epigenetic mechanisms supply a new hypothetical mechanism to fill in causal “black boxes” in explanations of the biology of sex, gender, and sexuality in the brain and social behavior. In explanatory schema, the visual icon of a black box represents a hypothetical causal mechanism with inner workings that may be obscure, controversial, or complex, connecting an input and an output (Latour 1987). Take, for example, black-box epigenetic explanations of the timing of female puberty. Scientists believe that puberty is ultimately caused by changes in the gonadotropin-releasing neurons located in the hypothalamus, but they lack a clear picture of how the body decides when to initiate puberty. Epigenetics is filling this explanatory gap. A 2013 *Nature Neuroscience* article (Lomniczi et al. 2013) demonstrating that modification of epigenetic mechanisms can advance or delay the timing of puberty, for instance, postulated epigenetics as a “biological regulatory system that meets [the] requirements” necessary to explain “how inherited, permanent changes in DNA sequence can regulate gene expression dynamically while also imposing an encompassing level of coordination and transcriptional plasticity on the gene networks involved” (281). Acknowledging that puberty involves a “cellular network” and that “no isolated pathway or cellular subset is solely responsible for the neuroendocrine control of puberty,” the researchers embrace epigenetics as an “encompassing” framework to bring coherence to a network-like phenomenon in sexual development (281).

Epigenetics has also delivered an evocative black-box causal mechanism for theorizing the biological development of homosexuality. “More than a decade of molecular genetic studies have produced no consistent evidence for a major gene, or other genetic marker, contributing to male homosexuality” (354), begins a 2012 paper in the *Quarterly Review of Biology* by evolutionary biologist William Rice and colleagues. Epigenetics, they propose, adds “the missing component” (344). They hypothesize that “canalizing epimarks” (344) alter the fetus’s sensitivity to testosterone, causing homosexuality. The term “canalization”—historically employed to describe the

emergence of organization and fixed states from complex, plastic, non-terminate systems—is here applied to suggest one way in which the biological plasticity of epigenetics can serve as a mechanism for inducing redundant, overdetermining direction in biological development. Rather than seeking a gene variant, this model locates the origins of homosexuality in epigenetic modifications to gene regulation. As visualized in figure 2, here hypothetical epigenetic mechanisms function as a black box to realize a causal model of homosexuality as a simple deviation from typical dimorphic sexual development.

In 2015, Eric Vilain and Tuck C. Ngun presented research at the American Society of Human Genetics seeking to validate Rice’s model (Ngun et al. 2015; see also Ngun and Vilain 2014). Their as-yet-unpublished paper, which has since attracted serious empirical critiques calling into doubt the soundness of the study (Yong 2015), claimed to find epigenetic markers that accurately predict the homosexual twin in mixed-sexual orientation monozygotic twins 67 percent of the time. Still new and highly controversial, research seeking the mechanistic basis of sexual orientation in deviations in the epigenetic development of the sexed soma provides an example of how, rather than disrupting biological determinisms, the materiality and theoretical re-

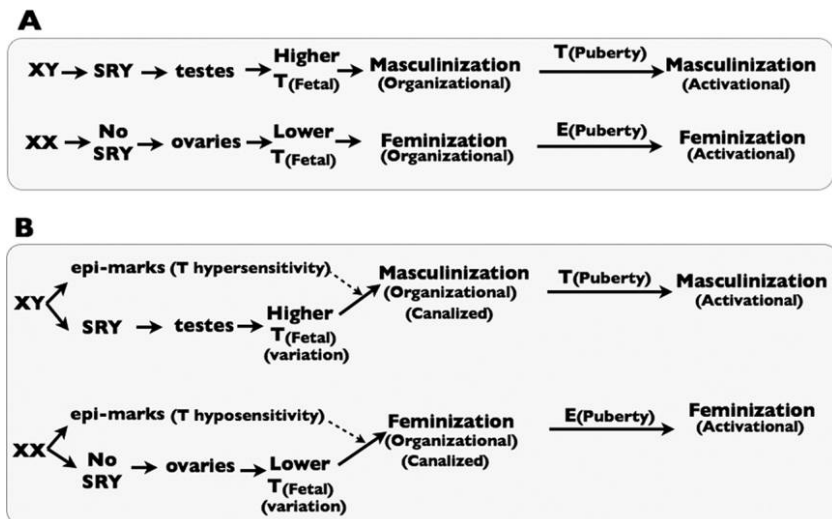


Figure 2 An epigenetic theory of homosexuality. “The classical view of sexually dimorphic development (A) is that higher androgen levels in XY fetuses and adults masculinize sexually dimorphic traits and lower androgen levels in XX fetuses and high estrogen in adults feminizes development. Our analysis (B) indicates that androgen signaling includes an additional component: it is canalized by epi-marks that are produced during the embryonic stem cell stage of development” (Rice, Friberg, and Gavrilets 2012, 346). © 2012 by the University of Chicago Press. Reprinted with permission.

sources of epigenetics may just as easily expand scientists' ability to position traces of biological sexual difference in relation to social categories of normalcy and abnormalcy (see Terry 1999; Fausto-Sterling 2000).

In sum, while epigenetics-centric models of sex reformulate the "stuff" that makes up sex, they do not represent a radical break from the explanatory projects of the twentieth-century sciences of sex. In research on epigenetic mechanisms in sex, gender, and sexuality in the mammalian brain and social behavior, studies focus on the elucidation of the local role of epigenetic mechanisms in organizational and activational effects of steroid hormones at particular genetic loci and hormone receptors. The epigenetic effects under investigation are at well-defined genetic loci implicated in sex differences, such as the estrogen and oxytocin receptors. In black-box biology that theorizes the development of sociosexual behavior, such as puberty or sexual orientation, epigenetics is principally an explanatory resource for describing the canalization and maintenance of sex differences.

In these models, epigenetic mechanisms serve to canalize and fix sex differences mediated primarily by genes and hormones. For the geneticist, epigenetic mechanisms explain gene action. They explain how gene expression responds to environmental cues, including but not limited to sex hormones. For the endocrinologist interested in sex differences, epigenetic mechanisms help explain how, in response to environmental cues, hormones permanently organize or actively mediate gene expression. The continuity between twentieth-century research programs and twenty-first-century epigenetics-centric sciences of sex can be seen in the persistence of the "programming" metaphor in epigenetic science.⁴ Hence, a typical finding of an epigenetics-sex study follows a template: a positive result is one that shows the organizing, determining, or programming effect of an epigenetic mechanism in establishing sex differences. "Testosterone acts via epigenetic processes, in particular the regulation of histone acetylation, to direct sexual differentiation of the brain," write McCarthy and her colleagues (2009, 12819). Similarly, the review article "Epigenetic Changes in the Estrogen Receptor α Gene Promoter: Implications in Sociosexual Behaviors" concludes that "environmental differences during development are programmed in the brain as a different pattern of epigenetic marks, and that this leads to differences in neuroendocrine and behavioral characteristics after maturity" (Matsuda 2014, 4).

Appreciation of the explanatory role of epigenetic mechanisms within the sciences of sex, gender, and sexuality in the brain yields three insights discor-

⁴ A carryover from informatics discourse pervasive in twentieth-century genetics, the programming metaphor has been widely and critically dissected in science studies literature on genomics; see Godfrey-Smith (2000), Kay (2000), and Keller (2000).

dant with feminist new materialist hopes for epigenetics. First, while researchers acknowledge the importance of environmental cues in epigenetic regulation, within the study of sex differences in the brain, epigenetics is not presently conceptualized as a source of ongoing plasticity in sex and gender itself. Rather, epigenetic mechanisms function as powerful and overdetermining agents in the canalization and programming of sex differences—not despite, but because of, their plasticity. In current research on brain sex, gender, and sexuality, epigenetics is understood as a ubiquitous and flexible adaptive mechanism that reliably works in a dynamic, plastic manner to enforce binary sex-differentiated pathways within the body. Sex differences in epigenetic regulation stabilize and overdetermine binary sexual dimorphism in the brain. In this field, environmental cues are largely restricted to other molecular factors—hormonal and genetic—in the immediate biological milieu, not social and cultural contexts. These epigenetic mechanisms are typically themselves genetically determined, meaning that they are understood as biological agents, the existence and function of which are ultimately encoded in the DNA, and which are designed to be reliably deployed within the organism to maximize adaptive aims.

Second, epigenetic plasticity is understood as itself sexually dimorphic. Inquiry is currently focused on epigenetics as a source for the elucidation of the biology of sex-specific responses to the environment, not on how environmental exposures create variation in sex-stereotyped behavior in males and females. Male and female epigenetic plasticity in response to the environment is itself theorized as sexually dimorphic (Champagne 2013). Studies of epigenetic mechanisms that link early social environment to adult brain and behavior suggest that the epigenetic pathways involved differ depending on the sex of the infant. Take, for instance, the much-celebrated link between maternal-infant licking and grooming behavior and the epigenetic programming of the brain pathways implicated in stress and anxiety in rats. This finding is more pronounced in males, as dams (female parents) lick and groom male offspring more often than female ones.

Third, epigenetics-centric research models render sexual dimorphism ubiquitous in the molecular architecture of the body. Sex-biased epigenetic mechanisms are theorized as mediating sex-specific processes at the interface of sex steroids and the genome in early development and throughout the life span. Epigenetics research thus postulates sex as a much broader part of the substructure of gene-environment interaction. In explanatory models in the biology of sex that invoke epigenetics, sex and gender become ubiquitous processes, not localized to gross regions of sexual dimorphism (see de Vries and Forger 2015). In the twentieth-century linear model of sex, sex inhered only in sex-specific elements such as the XX and XY chromosomal comple-

ments or in reproductive organs such as the testes and ovaries. In post-genomic models, the whole body is imbued with networked processes that are sexed. Conceptualizing any process that involves genes or hormones as sexed, postgenomic and epigenetics-centric research programs greatly multiply the sites, signs, and signifiers of binary sex in the body. Rather than the environment becoming a resource to explain sexual variation, sex differences become an expanded explanatory resource for illuminating biological variation.

In these many ways, epigenetics, insofar as it is a plasticity-affirming biology, is not in any necessary way akin to feminist imaginaries of the gender-plastic body. In epigenetics, forms of biological plasticity walk in lockstep with programming and its affiliated determinisms and binaries. Plasticity is often associated with malleability and variation and programming with hard-wiring and permanence. But historically, these two concepts are polysemic and need not be oppositional, or even mutually exclusive, within biological explanations of sex. Plasticity can be programmed, and programming can be plastic. Indeed, in the field of brain sex research, epigenetics inherits well-trod modes of operationalizing plasticity within the programming discourses of the hormonal and genetic life sciences. As such, epigenetics' move to the center of sex-difference research appears poised to reiterate, and even amplify, essentialist and thoroughgoing notions of biological sex differences, rather than to complicate or soften them.

Feminism and the epigenetic imaginary: Critical contestations and empiricist approaches

Feminist theorists and scientists embrace wide-ranging human variation in sex, gender, and sexuality and reject essentialist and binary conceptions of human difference. Using empirical and material methods to study gendered embodiment, they seek to illuminate the role of social and cultural context in shaping human bodies. Plasticity is central to the feminist call for a biology less focused on fixed sex differences. Feminist historian and philosopher of science Evelyn Fox Keller, for example, in her 2010 *The Mirage of a Space between Nature and Nurture*, urged scientists to shift their central question from one of the extent of biological determination to one of “degree of phenotypic plasticity”: “How malleable is a given trait, at a specified developmental age?” (75).

Feminist scientists and science studies scholars have devised innovative theories and research methods to study the sexed and gendered plasticity of bodies and biologies. In her recent contributions to the study of the embodiment of gender-sex in early child development, for instance, biologist

and feminist theorist Anne Fausto-Sterling employs dynamic systems theory to model and empirically study the iterative play of sex and gender in critical developmental transitions (Fausto-Sterling, Garcia Coll, and Lamarre 2012). Similarly, in her current research on testosterone, gender, and aggression, feminist social neuroendocrinologist Sari van Anders draws on gender theory to create novel experimental designs to study how gendered social interactions may alter hormonally mediated behavioral processes stereotypically linked to sex differences in modes of wielding power (van Anders, Steiger, and Goldey 2015).

The question of how gender—the social norms and expectations associated with masculinity and femininity—is corporealized within the biological, sexed body has been foundational to feminist theory since its inception. In her 1898 treatise *Women and Economics: A Study of the Economic Relation between Men and Women as a Factor in Social Evolution*, Charlotte Perkins Gilman argued that the social conditions of subjugation had created “rudimentary female creatures” ([1898] 1970, 164), weak in body and servile in mind. Gilman contended that sexual inequality between the sexes was produced and maintained by social factors working in interaction with the body and biology. With greater equality, she predicted, women would grow physically larger, stronger, and more agile (73–74). Three-quarters of a century later, Joanna Russ’s feminist science fiction classic *The Female Man* (1975) presented a protoepigenetic parable imagining a meeting between four women with identical genomes but from different time periods:

Look in each other’s faces. What you see is essentially the same genotype, modified by age, by circumstances, by education, by diet, by learning, by God knows what. Here is Jeannine, the youngest of us all with her smooth face: tall, thin, sedentary, round-shouldered, a long-limbed body made of clay and putty; she’s always tired and probably has trouble waking up in the morning. Hm? And there’s Joanna, somewhat older, much more active, with a different gait, different mannerisms, quick and jerky, not depressed, sits with her spine like a ruler. Who’d think it was the same woman? There’s Janet, hardier than the two of you put together, with her sun-bleached hair and her muscles; she’s spent her life outdoors, a Swedish hiker and a farmhand. You begin to see? . . . We ought to think alike and feel alike and act alike, but of course we don’t. *So plastic is humankind!* (Russ 1975, 163–65; emphasis added)

In *The Female Man*, differently gendered social and cultural contexts produce striking variation in embodied sex and gender comportment. Most recently, the BBC America science fiction series *Orphan Black*, praised for its

scientific acumen and feminist vibe, features a single actress who plays different versions of contemporary women—and a transgender man—with identical genomes. The show is a gender-bending phantasmagoria, showing the clones living wildly divergent lives: timid brainiac and lesbian Cosima; uptight, straitlaced soccer mom Alison; the transgender character Tony; and, needy, mentally deranged Helena, among others. The diversity of Western gender performativity—embodied, but firmly delinked from genomic endowment—is on impressive display. The show’s writers regularly invoke epigenetics as an explanation for this rainbow of characters, as in one episode in which the female child character, Kira, is teasingly advised not to eat too much “salted fish” or she will “end up with a beard”: “It’s epigenetics, monkey. It’s a proven fact” (*Orphan Black* 2015). These compelling visions of gender plasticity form an imaginary that has historically animated feminist intrigue with plasticity-affirming biological theories of all sorts, including epigenetics.

For feminist theorists, then, epigenetics is not only a set of scientific propositions, data, methods, and practices. It is also a discursive imaginary advancing heterogeneous future-oriented visions. This is true not only within feminist discourse but in a variety of other arenas within and beyond the life sciences. This imaginary is evident in the increasing displacement of the visual symbol of the genetic age, the double helix, by a new iconography of diagrammatic spaces showing genomes, human bodies, and physical and social environments in dynamic interaction over time and space. Epigenetics in these renderings is not a mere mechanism—a methyl group nestled against a double helix, say—but an arrow within often messy and variegated causal spaces. Epigenetics connects history and future, mother’s exposures and child’s outcomes, social policy and individual bodies. In these explanatory schemas, epigenetics is not reducible to any particular molecular mechanism: it is a causal resource in a much more expansive and theoretical sense. Over the past decade, epigenetics has entered several streams of scholarly and public intellectual discourse beyond the sciences, from Deepak Chopra’s New Age pronouncements that with meditation and good nutrition “you can change your genes” (2015) to click-bait headlines such as “Why You Should Be Worried about Your Grandmother’s Eating Habits” (Rank Lev 2014).

Within the academy, social scientists see epigenetics as a potential tool for the empirical study of biosocial interactions and as a corrective to the bald determinisms of the genetic era. A 2013 *Annual Review of Sociology* essay by Hannah Landecker and Aaron Panofsky situates epigenetics as representative of a “renegotiation and reconfiguring of the biological, the social, and their interrelation” (353) in the postgenomic moment. They describe epigenetics as “connecting social regulation to gene regulation in new and newly

direct causal ways” (335), opening a “‘critical window’ for engagement” (337) between the social sciences and biology. More broadly, they suggest that epigenetics offers a generative new “heuristic” (346) for sociological theory. As they write, “the logic, temporality, and findings of epigenetics could be productively incorporated into both research and theory in sociology without requiring sociologists to learn how to do molecular biology. It can be a mode of thought, a form of attention, or a resource with which to build hypotheses and models” (346).

Yet, as Landecker and Panofsky also note, the political and social implications of epigenetics are contested (2013; see also Lock 2015). Science studies and bioethics scholars have raised concerns about the renewal of dangerous and stigmatizing forms of somatic determinism with the rise of epigenetics (Waggoner and Uller 2015). The notion that early social context can mark the body, programming it for life, presents a specter of biologically marked, disadvantaged populations, contributing to class and race stigma (Mansfield 2012). The central role of pregnant women in epigenetic science, as vectors for intergenerational epigenetic programming, raises worries of enhanced mother blaming and increased surveillance and regulation of maternal bodies (Richardson et al. 2014; Richardson 2015). Discourses of epigenetic plasticity encourage individuals to see themselves as protectors and cultivators of their own epigenetic health, inviting neoliberal discourses of health optimization as individual responsibility.⁵

A nascent and unsettled science, epigenetics has also provoked intense contestations within the life sciences over the validity of its findings and conceptual frameworks.⁶ Leading scientists express concern that epigenetics is overhyped and charge that methodological issues cloud its central empirical claims. Only a brief summary of these issues is possible here. Findings of correlations between human phenotypes and epigenetic patterns are difficult to interpret. Epigenetic marks may be a cause of the phenotype; they may also be a result of it. Epigenetic mechanisms, particularly in the broadest sense of the term—as the apparatus of gene regulation—are now presumed to be relevant to any biological process. But omnirelevance must not be mistaken for power or precision in the mechanism itself. Changes in the epigenome (or the set of epigenetic markers at any region of interest in the genome) indicate system-level changes. These changes may be of potentially high biological

⁵ For a development of this argument in the case of neuroplasticity discourse, see Pitts-Taylor (2010).

⁶ Science studies scholars have documented extensive contestations among scientists about the validity of epigenetics findings and the revolutionary nature of epigenetics with respect to genetics; see Towlinski (2013) and Pickersgill (2016).

import, or they may be part of regulatory systems so complex and redundant that any one change cannot be clearly read as correlated or causally linked to any particular event. The effects of epigenetic perturbations are extremely small and often hard to detect and replicate (Francis 2014). Epigenetic studies overwhelmingly focus on methylation, but methylation and other singular epigenetic factors interact with many other cofactors that are presently poorly understood.

There are further limitations when considering humans in particular. The visually dramatic effects of epigenetic markers in cases of meta *epi*-alleles, such as the mouse *agouti* locus, at which changes induced by methylation in the maternal diet grossly alter the hair color and body size of offspring, have not been apprehended in humans. Indeed, in humans, what constitutes a normal or abnormal epigenome is still not fully intelligible (Greally 2015). Similarly, whether epigenetic marks acquired in early life are truly permanent in humans is not well understood. Despite the popular fascination with claims of transgenerational epigenetic inheritance, to date, only a few untested examples of truly transgenerational epigenetic inheritance have been discovered, and these are in nonhuman organisms, mostly plants (Heard and Martienssen 2014).

The case of epigenetics and sex differences adds to this picture of epigenetics as an emergent science accompanied by plural biosocial imaginaries and empirical and conceptual contestation. While some invoke epigenetics as a grounding for plasticity-affirming feminist theories, analysis of epigenetic approaches as they are deployed within the on-the-ground language, claims, and cognitive and social practice of this particular area of present-day science yields a different imaginative horizon. Epigenetics adds a new mechanism to biological explanations of sex that have long accepted a complementary repertoire of deterministic, programmed biological processes and dynamic, plastic biological systems. On close inspection, epigenetic approaches appear continuous with historically well-entrenched models of hardwired sexual dimorphism in the brain.

Presently, epigenetics does not affirm feminist theories of the plasticity of gender-sex. The possibility that epigenetics might, as new materialist theorist Elizabeth Wilson urges, help feminists “build conceptual schemata about the body that are astute both politically and biologically” (2004, 86), at best remains latent in epigenetic science. None of this, of course, renders impossible the prospect that epigenetics might in the future become a resource for feminist studies of the development of gender-sexed bodies. But it does suggest that such an aim will not emerge organically from the material facts of the science. To explore the full potential of epigenetics for feminist theories of the body and biology, feminist theorists must contest the ontological and

epistemological commitments of mainstream research in this field, critically analyzing pretheoretical conceptual commitments within epigenetic research on sex and formulating alternative research questions.

Epigenetics offers experimentally manipulable mechanisms for the study of the interaction of social context and biology. Hence, it offers plausible empirical and conceptual resources for feminist scientists and theorists of gendered bodies and biology. By asking different questions of the data and methods of epigenetics, feminist empiricist approaches might explore an alternative picture of the postgenomic biology of sex and contribute to the reorientation of the field. Feminist theory might inspire questions such as these: How might epigenetic mechanisms interact continuously with genetic and hormonal factors rather than fixing sex differences at an early stage of development? What happens when social and broader environmental factors are included alongside hormonal and genetic ones as potential cofactors in the epigenetic apparatus of sex? Rather than hypothetical flowcharts ending in male and female epigenomes (fig. 1), might scientists consider models that include multiple canalizing trajectories, leading to, as Fausto-Sterling (1993) once famously wondered, five genders-sexes rather than two?⁷

Studies of the methylation status of the previously discussed ER α gene offer an intriguing model for imagining engagements between the materiality of epigenetic mechanisms and feminist theories of the body and biology. In rodents, the level of methylation at ER α in the sex-differentiated POA varies over developmental time in relation to exposures to social and environmental factors. A female that has experienced simulated maternal grooming as an infant yields higher ER α methylation levels than one that has not. An adult female that resided, in the uterus, between two female littermates exhibits higher methylation at the ER α receptor than one that grew between two males (Matsuda 2014). Such findings suggest the possibility of studies that creatively proliferate social and environmental exposures, the genes involved, and the regions of the brain and body of interest in order to experimentally assess the plasticity of sexed and gendered phenotypes and explore the epigenetics of mammalian sexual variation over life history.

Whether the potentiality of expansive sex and gender plasticity will be fully empirically explored in epigenetics research, however, will not be determined

⁷ Landecker (2014), for example, points out that while there have been voluminous studies of the effects of maternal behavior on the fetal epigenome, scientists have not examined the epigenetic changes induced in women by pregnancy. A research program examining the epigenetics of women's diverse reproductive histories might yield empirical evidence relevant to the question of sex- and gender-related biological plasticity and variation over developmental time among women.

only by the material facts of the science itself. To develop epigenetics as a resource for feminist reconstructions of scientific models of the biology of sex and gender, feminist science scholars must examine the causal-explanatory model of sex, as well as the discursive and political imaginaries, in which epigenetics research is embedded in practice. While scientific observations constrain the extent and range of possible biological plasticities at sex-related epigenetic loci, the direction of research and its theoretical extension within gender studies depends on strategic contestations in the politics of gender and science—contestations over which questions are asked, which methodologies are adequate to ask them, and how gender-sex is conceptualized.

These sorts of contestations might revise and contribute to our understandings of how the “scene” (Wilson 2004, 69) of socially mediated gender plays out in the body. But equally, the outcome may be that feminist theorists learn that plasticity, multifactoriality, complexification, and the contingency of determinants of sex/gender/sexuality is a less useful conceptual tool kit than it is often imagined to be. Indeed, there may be a need for revised and more nuanced feminist appraisals of both plasticity-affirming and programming-centric models in biology. Perhaps plasticity-affirming biologies do not produce a picture of a spectrum but rather one of the overdetermination of sexual binaries in the body. Testing feminist intuitions about plasticity might provide an impetus for deepening and reframing feminist investment in plasticity-affirming conceptual frameworks and aversion to programming-centric ones—and even for retheorizing the distinction between plasticity and programming altogether.

Conclusion

Responding to what Margaret Lock (2013) has called “the lure of the epigenome” from the perspective of feminist science studies, this essay has analyzed the integration of epigenetics research into the subfield of the scientific study of the core biological pathways related to sex, gender, and sexuality in the brain in the post–Human Genome Project era. Today, epigenetics is an expanding explanatory resource in the basic biology of brain and behavioral sex. In coming years, researchers will almost certainly find epigenetic mechanisms correlated with many hormonal and developmental processes, and they will find sex differences in those epigenetic processes themselves.

While epigenetics researchers embrace the importance of environmental inputs in biological systems, this does not lead them to affirmations of diversity and variation in sex and gender. Instead, epigenetics functions as a redundant or overdetermining mechanism carrying out and maintaining the persistence of classic, well-established hormonal processes implicated in sex

differences. Though a substantial conceptual divide persists between feminist theories of gender plasticity and dominant models of the epigenetic canalization of sexual dimorphism in the brain, future research programs to test hypotheses related to the plasticity of gender-sex may yet blossom. Thus far, however, the embrace of epigenetic mechanisms has not entailed the acceptance of greater plasticity and variation in sex and gender among scientific researchers. Instead, epigenetic research has more often reinforced and extended deterministic and binary conceptions of sex differences in brain and behavior, sustaining the difference paradigm of biomedical research on sex and gender robustly established in the twentieth century (Fausto-Sterling 2000; Epstein 2007; Richardson 2013).

Making sense of the striking divergence between feminist imaginaries and scientific researchers' appraisals of the conceptual potentialities perceived in epigenetic research on sex requires appreciating that plasticity-affirming biologies are situated within the history of twentieth- and twenty-first-century sciences of sex, which have been deeply committed to a reigning metaphor of programming and to reductionist and determinist explanations. The case of epigenetics and research on sex differences in the brain, furthermore, shows the importance of attending to field-based specificities as science studies scholars map transformations in theory and practice introduced by the new science of epigenetics. In each scientific field, the explanatory reach of epigenetics emerges temporally through local practice and contestation, and it does so against the backdrop of received intellectual frameworks. Epigenetics is not only a material mechanism but also a fluid imaginary functioning diversely across heterogeneous social spheres. Plasticity and programming are not necessarily opposites but are often two sides of the same coin. As new plasticity-affirming biologies arise, their materialities will not speak for themselves; feminist science scholars are called on to critically contest the discursive, ontological, empirical, and methodological terms of epigenetic science itself.

*Department of the History of Science and
Committee on Degrees in Studies of Women, Gender, and Sexuality
Harvard University*

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