State of the Field Review

THE NEW GENETICS OF SEXUALITY

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SAME SEX ATTRACTION: Your genetic score is above average.
—"How Gay Are You?" App, GenePlaza

In 2019 a "genetic app" titled "How Gay Are You?" was published on GenePlaza (Maxmen 2019), immediately following the publication of a headline-making genetics study on same-sex sexual behavior (Ganna et al. 2019; Belluck 2019). In response to widespread outrage, the app was quickly discontinued (Bellenson 2019). But, rather than a rogue misapplication of responsible science—as the study's authors and others decried it—this debacle is emblematic of the pitfalls inherent in the new era of sexual genetics. In this essay we introduce the central methods, concepts, and key terms in the field of sociogenomics and related genetic sciences. We then review high-profile claims from this field that posit genetic theories of gender and sexuality, or that analyze gender and sexuality as variables in the characterization of brain, psychiatric, and medical conditions. Last, we characterize the conceptual, methodological, social, and ethical questions opened by this new frontier for an interdisciplinary audience, emphasizing the gap between the sociogenomic imaginary and what the data can and do currently show. Our goal is to build on existing critical perspectives (Clare, Grzanka, and Wuest 2023) by translating and contextualizing highly technical developments in the new era of genetics research and to invite scholars to engage with the issues it raises.

Since the rise of "sexual science" and sexology in the West around the turn of the twentieth century (Amidon 2008; Mancini 2010; von Krafft-Ebing 2013), clinicians and life scientists have searched for evidence of sexuality in the body. This has included looking at finger digit ratios (Holmes et al. 2021; Swift-Gallant

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et al. 2021), waist-to-hip ratios (Hughes and Gallup 2003; Reiches et al. 2018), facial structure (Skorska et al. 2015), phrenological characteristics (Janssen 2015), hormone levels (Evans 1972; Meyer-Bahlburg 1979), and genes and brain structures (LeVay and Hamer 1994; Votinov et al. 2021). More recently, high-profile research groups have investigated the existence of genetic influence on same-sex sexualities, using massive genomic datasets to go beyond searches for a "gay gene" to complex, many-gene models of genetic contributions to sexuality. Although these scientists have claimed to find genetic components associated with same-sex sexual behaviors (Ganna et al. 2019), even by their own standards, these findings hold limited explanatory power, reproducibility, or conclusive evidence of a biological basis for sexual orientation. Studies like these fall under the rapidly growing field known as sociogenomics (Mills and Tropf 2020), in which ever-advancing scientific techniques and volumes of human biological and social data have generated new frontiers in the search for the biological underpinnings of sexuality.

Broadly, the field of sociogenomics seeks to elucidate the genetic basis of social behaviors and to situate them in an evolutionary perspective. This project is not entirely new; in many ways, sociogenomics extends previous endeavors from classical behavioral genetics, neuropsychiatry, and racial eugenics to isolate the biological underpinnings of who people are and why they do what they do (Bliss 2018; Panofsky 2014).

However, recent increases in the volume of data available for sociogenomics research, in combination with the development of new statistical methods, have ushered in a "paradigm shift" in these fields (Matthews and Levy 2022). Under this new paradigm, researchers conceptualize variation in complex human behaviors as resulting from the aggregate of many tiny, piecemeal contributions from across the genome. By sequencing the DNA of populations and correlating allele variation with certain behaviors or life outcomes—often ascertained by self-report on surveys—geneticists can develop a *polygenic risk score* (PRS) value for every individual, which aims to predict their probability of displaying a specific trait or behavior. In the burgeoning field of precision medicine, individual risk scores are seen as an indispensable tool that, one day, will steer population-specific or personalized clinical interventions that take the entirety of one's genetics and social experience into account (Lewis and Vassos 2020).

This shift has implications across multiple domains, with scientists applying similar large-scale genomic methods to traits ranging in social salience from heart disease or cancer (Arking and Chakravarti 2009; Sud, Kinnersley, and Houlston 2017), to intelligence (Loo et al. 2012; Zabaneh et al. 2018), to risk-taking (Clifton et al. 2018; Li et al. 2020), to sexual behavior (Ganna et al. 2019; Polimanti, Wang, et al. 2017; Terracciano et al. 2011). These research programs

have caused significant controversy, with proponents arguing they will unlock key discoveries undergirding the human experience (Harden 2021) and detractors critiquing the ethics of applying these techniques to stigmatized behaviors and thereby treating them as biologically rooted. Of particular concern is the possibility that polygenic score (PGS) with shared genetic architecture (see "Correlating Gender/Sexuality Traits" below) will contribute to harmful beliefs about the relatedness of social, behavioral, and health phenomena, for example, by correlating the likelihood of developing schizophrenia with contracting HIV (Wang et al. 2017), or of exhibiting "risk-taking behaviors" with one's number of lifetime sexual partners (Polimanti, Wang, et al. 2017; Polimanti, Zhao, et al. 2017).

Some readers may wonder if these critiques of the use of PRS and genetic essentialism might be at odds with gay and trans rights movements that have often turned to "born this way" arguments in political fights for recognition, from gay marriage to gender-affirming updates of birth certificates. This is a complicated issue that merits longer discussion (Wuest 2023). Without dismissing these rhetorics and the advocates and organizations using them, we maintain that it is critical to attend to the tenuous nature of these essentialist claims, as they flatten and erase the variability of human sexuality and gender, and can be reappropriated by eliminationist or eugenic movements. It remains a fully open question whether deploying essentialist arguments for the sake of securing more rights and protection from the state for marginalized groups may actually incur more risk of violence, rather than less.

Here we provide an overview of the central methods, assumptions, and epistemological limitations of sociogenomics and related genetic sciences. We also situate these developments in political context and highlight opportunities for future research programs in light of the shifting nature of genetic claims making. We believe queer studies scholars should be aware of how this new science is carving categories of sex, gender, and sexuality into a new frontier of predictive genetic science, which stands at the intersection of the long-standing search for the biological bases of human variation and recent profit-driven investment in genetic data science and in clinical and consumer genetic services.

The Science of Polygenic Risk

The full DNA sequence, or genome, of an organism is made up of many distinct loci, or physical locations where specific genes can be found in most individuals within a population. At each locus, there are different sequence variants that may be present, called alleles. Variation in each individual's combination of alleles can contribute to phenotypic, or observable trait, differences. Alleles can differ

from each other by as little as a single DNA base pair substitution (out of the thousands that make up a gene)—called a single nucleotide polymorphism (SNP)—or as much as a full deletion of the locus.

While the concept of a single gene for every trait has persisted in popular culture (Carver et al. 2017; Nelkin and Lindee 2004), human geneticists predict that most complex traits are *polygenic*, such that many alleles at many different loci each have a small effect on the trait. This framework was originally developed over a hundred years ago to reconcile models of genetic inheritance between discrete traits, such as eye color, and continuous traits, such as height (Fisher 1919; Visscher and Goddard 2019), both of which can be polygenic, but this theory could not be brought to bear on complex human social traits until recently, with the availability of large genomic datasets.

Owing to the small effect size of each SNP, finding the polygenic basis of a complex trait requires significantly more data than locating a single gene that controls a trait by itself. Datasets amenable to these inference problems became feasible only over the last decade, after the development of next-generation DNA sequencing technologies and the subsequent aggregation of large numbers of human genomes, both through nonprofit research initiatives, such as the UK Biobank—which contains approximately five hundred thousand genome sequences from adults in the UK—as well as through commercial entities, such as 23andMe, which holds over twelve million sequences from people around the globe (23andMe 2023; Richardson and Stevens 2015).

With these datasets, geneticists are able to conduct genome-wide association studies (GWASs), which look at correlations between the presence of different SNPs and trait values. Because of the relative ease with which a GWAS can be done, they have quickly become a staple in the field of human genetics, with GWASs conducted on scores of traits ranging from celiac disease to age of menarche (Visscher et al. 2017). However, GWAS results can be cumbersome to interpret. Importantly, while GWAS results are often described as identifying the genetic components of a trait, in fact GWASs cannot determine if any particular SNPs are themselves causal for a trait. While a GWAS can estimate the "effect" of a particular indicator SNP on a trait of interest, this estimate is known to not be the true biological contribution of that allele. Rather, it is an aggregate measure of the combined effect of all variants that are linked, either physically along the genome or statistically via nonrandom association, to that indicator SNP. These interpretive complications helped motivate the creation of single-value scores, or indexes, which summarize the influence of an individual's genome on their phenotype.

These predictive indexes for trait liabilities sum estimated SNP effects from association studies to calculate a single score that aims to describe the total

genetic propensity of an individual to have a certain trait (reviewed for nonhuman systems in Crouch and Bodmer 2020). In humans, a similar approach is used to calculate a "polygenic score," a single value that represents an estimate of the contribution of an individual's genotype to their phenotype (reviewed in Rosenberg et al. 2019). This score is also called a "polygenic risk score" or "polygenic index" (PGS, PRS, or PGI, respectively).

One of the key issues in current PGS research is "portability," or how much predictive ability a PGS calculated in one sample (e.g., "Europeans" or "Utah Mormons") has in another. With the popularity of the UK Biobank, geneticists have naturally become interested in investigating whether its European bias limits its applicability to other populations (Privé et al. 2022). Portability issues are known to arise between different "ancestry groups" and are conceptualized as a downstream result of random genetic variation that arises owing to geographic isolation between populations. While most geneticists recognize that genetic ancestry and social definitions of race and ethnicity are not identical, and there is strong consensus that geneticists should use language that avoids the implication that race/ethnicity is a proxy for biological propensity for social and behavioral traits (Gravlee 2009; Kaplan and Fullerton 2022; Mohsen 2020), many geneticists and public translators of science still use the term *race* or *genetic races* to describe these groups, contributing to race-essentialist understandings of group differences. Portability has also been considered between sexes, with some studies opting to run separate GWASs on "males" and "females" in the search of distinct differences in genetic architecture between them (Bernabeu et al. 2021). Exploring the implications of this widespread assumption of sex-specific genetic architecture in GWAS studies is a current focus of our research at the GenderSci Lab.

In the context of gender and sexuality, PGS studies face additional issues resulting from sampling practices, both ethical and technical (Richardson et al. 2019). It is standard practice in GWASs to remove individuals from the dataset whose karyotype, or chromosomal makeup, (as determined by sequencing) does not conform to what is expected of their self-reported "sex" (e.g., XY chromosomes for "males"). In addition, many sequencing approaches—such as the SNP genotyping used by 23andMe—will only detect the presence or absence of a Y chromosome, which will fail to discriminate between possible sex chromosome karyotypes with nonstandard ploidy, such as in people with Klinefelter syndrome (XXY). This approach leads to systematic under- or misrepresentation of transgender and gender-diverse populations, which is only further exacerbated by data curation practices for the surveys accompanying these genetic studies. For example, Ganna et al.'s 2019 GWAS for same-sex sexual behavior systematically removed individuals who identified themselves as transgender or nonbinary from

their dataset. While inclusion is not necessarily the goal, these practices embed a binary conceptualization of sex and build the exclusion of trans individuals into the bedrock of genomic science (Epstein 2008). Ultimately, of course, a fundamental issue is that these methods rely on researchers' definitions of the trait of interest, a critical avenue for the introduction of social and cultural assumptions and biases into the science. Importantly, geneticists are not trained to critically evaluate surveys, and the translation of the survey answer and genetic variant correlation to genetic control of a real trait is usually not scrutinized as heavily as the genomics itself—a point to which we return below.

Glossary Box

Term	Definition
Genome	The total genetic information in an individual, a copy of which is present in almost every cell.
Locus	A locus is a physical location in the genome, sometimes used synonymously with gene.
Allele	An allele is a particular variant, or copy, of a gene. There may be many different alleles for the same gene, but they are all found at the same locus.
Single-nucleotide polymorphism (SNP)	An SNP is a variant in which a single base pair of DNA has been changed. Alleles might differ from each other by as little as a single SNP.
Phenotype	The observable characteristic or trait of an individual.
Polygenic trait	A trait that is influenced by multiple genes at different loci. A polygenic trait can be discrete, such that many genes influence the likelihood of a particular categorical outcome, or continuous.
GWAS (genome-wide association study)	A study design in which, using a large amount of sequence data from many individuals, a phenotype or trait is correlated with the presence or absence of different genetic variants (for example, SNPs).
Polygenic risk score, Polygenic score, Polygenic index	All three of these terms (abbreviated PRS, PGS, or PGI) refer to the same quantity, which integrates allele effect sizes ascertained from a GWAS study to calculate an individual's genetic propensity to have a certain trait from their genotype.
Pleiotropy	When a single gene produces multiple traits or effects. This can occur because the gene influences two distinct pathways ("horizontal" or "true" pleiotropy) or because the gene has an effect that itself generates a downstream signal ("vertical" or "spurious" pleiotropy).
Effect size	The estimated impact that a particular allele has on some trait of interest.

Pressing Issues in Current Polygenic Research

Weak Claims and Spurious Results

Almost universally, polygenic scores do not explain even close to the majority of the phenotypic variance observed, and their component signals are often not individually statistically significant. In Ganna et al.'s 2019 study claiming a "complex genetic architecture" of sexuality, for example, only three of five initially identified SNPs were reproducible in a follow-up replication analysis, and each explained only approximately 1 percent of the variation in sexual behavior; overall, the authors estimate the full contribution from genetics to the phenotype of "same-sex behavior" to be as low as 8 percent. In a 2017 GWAS on male same-sex sexual behavior (Sanders et al. 2017), no individual SNP was statistically significant, yet the paper reports several "promising regions" of SNP clusters. In another GWAS claiming to "provide a genetic basis" of why sexually active individuals with schizophrenia are more likely to engage in risky sexual behaviors and contract HIV (Wang et al. 2017), the authors even concede that their results have a low significance level and small effect sizes. In light of extremely minimal findings even by the standards of genetics research, one may find it baffling that this research continues and is lauded as revealing fundamental truths of human sexuality.

But the celebratory posture toward such minor findings may be explained by the fact that it is often taken for granted in genetics research that certain results "should" exist, and that better scientific methods, tools, and datasets will, in time, reveal them. For example, because sexual minorities often experience worse mental health outcomes than other social groups, a common hypothesis in sociogenomics is that there must be shared genetic variants that contribute to "risky" sexual behavior and psychiatric conditions (Polimanti, Zhao, et al. 2017; Wang et al. 2017; Ganna et al. 2019). These types of assumptions allow weak claims or statistically insignificant results to be framed as illuminating, while simultaneously fueling uncritical calls to collect more, or "more diverse," data from individuals with gender-expansive identities and non-European ancestry in order to confirm the findings. The question of mechanism—that is, through what biological pathway the identified genes of interest actually influence the phenotype—is typically suspended or proffered as a thin hypothesis. Authors may vaguely point to physiological tissues and systems the identified genes play a role in, such as excitatory neurons (Karlsson Linnér et al. 2019) or thyroid hormonal pathways (Sanders et al. 2017), which frequently appear in the discussion section without deeper interrogation.

The authors of these studies often caution against causal interpretations of their findings, particularly owing to possible social and cultural influences on

the phenotypes they study, and flag the need for future research that might integrate history or culture via gene-by-environment analysis. Nonetheless, current methods approach sexuality and gender as genetically influenced in a universal way through time and across social contexts, deferring any grappling with the complexity of gender and sexuality as social phenomena. Gene-by-environmental interactions and population stratification (differences in allele frequencies between populations), both essential for engaging the social, are well understood to complicate polygenic score analysis (Rosenberg et al. 2019). Even complicated social practices such as parenting are reframed as biological factors detectable through GWAS, with genes imagined to act across generations via "genetic nurture." Concerns about these weaknesses and other methodological limitations of polygenic scores have been so far held at bay by researchers who claim that more expansive, inclusive datasets will allow for greater control of environmental variables and open the door to portability across populations. But it is not at all evident that more and more data will address the fundamental questions of methodology and interpretation that pursuing the genetics of social and behavioral traits related to human sex, gender, and sexuality brings.

Correlating Gender/Sexuality Traits with Behavioral and Psychiatric Conditions

Many investigations in sociogenomics are interested not only in determining the genetic basis of a particular trait but also in how that "genetic architecture" overlaps with other traits (Bulik-Sullivan et al. 2015). These analyses aim to demonstrate shared genetic control between two traits by way of genetic correlations, which describe the statistical relationship between GWAS results on different traits or sample populations. These genetic correlations are often offered as a potential signal of some true genetic phenomenon, such as direct pleiotropy, in which one gene impacts multiple traits. However, two traits may appear to be genetically correlated because one impacts the other at a nongenetic level, a phenomenon known as vertical pleiotropy. While some authors do acknowledge this complexity, it is usually sidelined in favor of stronger, genetic claims. For example, in one analysis of "risk-taking behavior" (Strawbridge et al. 2018), the authors reported that risk-taking had a significant genetic correlation with obesity. Although the authors do state that "there are likely to be a range of potential mechanisms linking risk-taking behavior with obesity," this small disclaimer is paired with strong claims implicating pleiotropy of risk-taking behavior and obesity through genetic control of "brain regions involved in cognition, learning, and reward" (9). Even when social factors are acknowledged, strong biological interpretations of genetic correlation results can lead to startling conclusions. For example, in a study of

same-sex sexual behavior (SSB), the authors interpret their finding of a negative genetic correlation between alleles associated with same-sex sexual behavior and number of children to claim that "SSB-associated alleles are overall reproductively detrimental in the contemporary British population," and they note that their results "predict that SSB-associated alleles will gradually decline" as a result of increased contraceptive use decreasing the evolutionary "need" for same-sex sexual behavior (S. Song and Zhang 2023: 2–3). By reporting genetic correlations between traits related to sex, gender, sexuality, and reproduction and other traits as a signal of shared genetic architecture or biological pathways between these phenotypes, these studies leave open unfounded interpretations that there exists rigorous evidence of essential biological underpinnings and associations between, for example, sexuality and mental health outcomes. These omissions could lead to irresponsible speculation in policymaking, governance, and social provisioning, as well as contribute to social stigma and perpetuate stereotypes.

Because of the impracticality of assessing the genetic correlation of a trait of interest with every other trait for which GWAS data exists, the set of traits that are tested as potential candidates for sharing genetic architecture are selected by the researchers and research institutions conducting and funding these analyses, perhaps because they simply seem interesting or probable, based on unspecified assumptions. For example, in Ganna et al.'s analysis of the polygenics of same-sex sexual behavior, the traits tested for genetic correlations were overwhelmingly either those commonly classified as psychiatric conditions (such as schizophrenia, bipolar disorder, or anxiety) or social attributes with overtly negative valences (such as loneliness or neuroticism). Similarly, in a GWAS study of "risky behaviors," lifetime number of sexual partners and age of first intercourse were interpellated with not only other risky behaviors but also psychiatric conditions (Karlsson Linnér et al. 2019). While it is not necessarily the case that reporting correlations between sexuality and more "positive" PGSs such as "leadership ability" (Z. Song et al. 2022) would lead to more equitable GWAS research, it is clear that the preselection of variables for genetic correlation analyses implies unstated background assumptions about the quality or value of the traits of interest, restricting the space of possible outcomes and shaping the narrative of genetic relationships between traits to match the preconceptions of the researchers conducting these studies. In this way, rather than uncovering a true genetic correlation between highly social traits, researchers are reinscribing harmful, socially produced assumptions about those with vulnerable or stigmatized identities and experiences.

Bluntness of Categories

Research using PRS and GWAS presents gender/sex and sexuality categories as self-evident, flat, and extendable across cultural boundaries. Terms such as *same-sex attraction* are used to agglomerate diverse and personal human experiences, immediately diminishing their multidimensionality. The concept of same-sex sexuality as a coherent, single trait depends on the idea that the individuals all experience sexuality in the same way, as highlighted in a recent *GLQ* roundtable (Clare, Grzanka, and Wuest 2023). Worryingly, quantitative statistical results are built on binary assumptions that pit heterosexuality and homosexuality as opposed parts of a whole. In their study of "male sexual orientation," Sanders et al. (2017: 3) contextualize their work using this dichotomy, stating that

> the continued genetic study of male sexual orientation should help open a gateway to other studies focusing on genetic and environmental mechanisms of sexual orientation and development. Detectable genetic variants predisposing to homosexuality would have alternative alleles, which would necessarily predispose to heterosexuality, thus contributing to understanding of both typical heterosexual and minority homosexual orientations.

Similarly, categories of sex are positioned as opposed, universal, and binary, with a cottage industry of analyses of sex differences in the genetics of obsessive-compulsive disorder, depression, and even post-traumatic stress disorder—all without consideration of the variation within sex-classes, or a single mention of gendered social variables that might contribute to observed sex differences (Nievergelt et al. 2019; Khramtsova et al. 2019; Trzaskowski et al. 2019).

Researchers use sex-related variables without acknowledging how dependent these terms are on their context or, often, even defining them at all. The terms are taken to be self-explanatory, building in researchers' preconceptions of sex and gender, while overlooking how factors like socioeconomic status, geography, and racial stratification may affect them. Even in Ganna et al.'s (2019: 3) study, which was lauded for engaging LGBTQ+ organizations and providing a descriptive text box on gender/sexuality, the authors operationalize same-sex sexual behavior as having even one same-sex encounter in the life course, while acknowledging that their study excludes transgender, intersex, and other queer persons, while hoping "that this limitation will be addressed in future work."

The Clinic and the Market

The generation of polygenic scores for sexual- and gender-identity-related traits is occurring within a context permeated by commercial market incentives and a vision, underwritten by major private and public medical research funders, of personalized medicine grounded in routine bedside genomic testing (Eyal et al. 2019; Juengst et al. 2016). For example, the largest studies investigating genetic correlates of same-sex sexual behavior (Ganna et al. 2019; Zietsch et al. 2021) used data from the UK Biobank and 23andMe, and were funded by the National Institute of Child Health and Human Development specifically "to investigate the genetics of sexual orientation" with a running total of nearly USD $4 million (Sanders and Martin 2022; Sanders 2022). Meanwhile, the direct-to-consumer (DTC) genetic testing market size surpassed USD$3 billion in 2022 (Swain and Subodh 2023), and the full US genetic testing market size is projected to reach USD$10.29 billion by 2027 (*Fortune Business Insights* 2023). For reference, this is just over the current market size for acetaminophen (the drug in Tylenol), which stands at $9.8 billion (Future Market Insights 2023).

Although PGS scores have shown at best limited success in advancing clinical treatment for a small set of diseases (Lewis and Vassos 2020), some behavioral geneticists hold out the promise that sociogenomics will also help social scientists identify effective interventions for social problems. For example, at a National Institutes of Health roundtable titled "The Promise and Perils of Social and Behavioral Genomics," Daniel Benjamin, a senior author of a GWAS of educational attainment, speculated that work identifying genetic markers could help control for differences in the genetic makeup of the subjects in expensive experiments, such as those testing the effect of free preschool on lifetime educational attainment, in order to ensure the most precision possible (Lee et al. 2018). The downstream goal of this approach would be to "better understand the origins of inequality" (Braudt 2018: 12), a goal that behavioral geneticists believe will be more achievable by fusing genetic data and social science.

The ultimate endpoint of PGS studies is explicitly imagined as a set of tests available to consumers on the market or in the clinic, allowing the estimation of individualized risk scores in furtherance of precision medicine (Lewis and Vassos 2020). In this vision, polygenic scores deliver actionable, predictive estimates of health risk, which might be used to recommend behavioral modification or pharmaceutical treatment (Balogh, Pulay, and Réthelyi 2022; Foley, Corvin, and Nakagome 2017; Mishra et al. 2022). Controversially, they might also be used in embryo selection through preimplantation genetic testing (Forzano et al. 2022;

Karavani et al. 2019; Treff et al. 2022). Existing evidence on DTC genetic testing suggests that, even if the use of PGS in clinical settings is heavily regulated or cautioned against, these tests will become available and popular in commercial markets for "off label" uses (Au 2022).

In short, investigations in clinical genetics and sociogenomics together form a pipeline for claims about the etiology of complex behavioral phenotypes, as well as a possible future in which nonnormative genders and sexualities can be predicted, anticipated, and "treated" accordingly.

Consent, Privacy, and "Beneficent" Science

As sociogenomic research advances, we can anticipate that geneticists, IRBs, and research study participants will face varied justificatory rationales for scientific studies entangling the study of health-related outcomes with social justice aims, while raising risks to vulnerable groups. Particularly when faced with decision points in sociogenomic research related to gender and sexuality, a central concern is research participant consent. For example, in the UK Biobank (2020) consent form, participants were told that their samples would be used for "health-related research purposes." Could participants have reasonably expected that their sexuality would fall under this purview? Applications of data collected for health research to sociogenomic studies of sex-, gender-, and sexuality-related traits raise questions about the legitimacy and scope of "broad" consent in genomics research, which allows data to be used for unspecified future research in both US and UK data governance regimes (Hallinan and Friedewald 2015; Holm and Ploug 2019).

Sociogenomics researchers have proven remarkably successful in skirting these ethical concerns by framing the science as inherently desirable, beneficent, and health-promoting research that is in the best interest of vulnerable populations. Many GWAS studies of social traits such as sexuality deploy a justificatory framework centered around the possibility of using such studies to develop treatment for the harms from stigmatized behaviors, such as personalized treatment to minimize harm based on an alleged genetic link between "risky sexual behavior" and "alcohol dependence," or between HIV and schizophrenia with that same "risky sexual behavior" (Polimanti, Wang, et al. 2017; Wang et al. 2017).

Notably, the primary level of evaluation by IRBs is based on risks to individuals. Because individual genetic data is generally anonymized, risk to individuals is considered minimal, even though it may have potentially dramatic consequences for entire vulnerable populations. As it stands, the predominant focus of IRBs on individual risk of harm is unequipped to grapple with community-wide

implications of sociogenomics for LGBTQ+ populations. For this and other reasons, IRBs and other stakeholders lack adequate ethical frameworks for research in this field. These ethical standards must be grounded in historical and social understandings of genders and sexualities—as identities, as subjectivities, as oppressed classes—rich enough to account for the possible harms of polygenic research, beyond individuals.

The Need for Critical Intervention from Gender and Sexuality Scholars

This research raises crucial questions ripe for analysis by scholars of gender and sexuality. Rigorous, deep work on the underlying assumptions and social structures driving sociogenomics research, and on the ways in which social-cultural beliefs are naturalized or challenged by sociogenomics discourse, is vital in order to prevent harm to vulnerable populations and challenge underlying biases in scientific practice. Our hope is that this essay can catalyze, and serve as a resource for, critical interventions by gender and sexuality scholars, who have expertise directly relevant to this area of emerging scientific research.

There is now a well-developed critical discourse, integrated into advanced training in genomics and medicine, on the care that must be taken in designing, analyzing, interpreting, and communicating claims about genetic differences between racialized human populations. However, claims about sex and gender differences have not received the same scrutiny, and critical engagement by scientific stakeholders regarding these axes of social difference remains sorely lacking. Queer studies scholarship must intervene and expand the scope of these conversations, as well as identify the impacts of this research program for feminist politics and for gender and sexual minorities. Specifically, there is a need for feminist, science and technology studies (STS), and critical race scholars to analyze polygenic methods in the context of big data genomics and rapid commercialization of this industry, and to trace the ways in which sex, race, and other forms of difference are co-constructed in this field of scientific practice.

One touchstone comes from feminist and critical race STS scholars, such as Alondra Nelson (2016), Jenny Reardon (2009), and Kim TallBear (2013), who explore how the politics of genetics research is embedded within broader power structures. This field of scholarship argues that genetics research actively influences the social and cultural politics of race and ethnicity. While polygenic research practices at once ensure that gendered and sexualized subjects are "fixed in place . . . to enable technoscientific development" (Benjamin 2016: 145), they are also methods for "making up people" (Hacking 1986), and we anticipate that

new scientific practices will also function generatively to facilitate new identities and self-understandings.

Similar to racial genetic discourses, research on the genetics of gender- and sexuality-related traits appeals to "the natural" to justify particular visions for classifying human identities and behaviors (Fausto-Sterling 2000; Wuest, forthcoming; Richardson 2013). The appeal to nature establishes sexuality as innate and fixed, despite most researchers' agreeing that sexuality is a combination of social, biological, and environmental factors. This research joins a long-standing tradition of claiming that minority groups are biologically different from "the norm." This is the ghost of eugenicist histories (Subramaniam 2014), which utilized genetic research to characterize LGBTQ+ people as genetically inferior and justified forced sterilization or genocide of LGBTQ+, disabled, immigrant, Black, Latinx, and Indigenous people in the United States (Terry 2013; Terry and Urla 1995; Stern 2019; Briggs 2002; Roberts 2014; Ostrowsky 2020).

The proliferation of precision medicine, sociogenomics, and the geneticization of social categories relies on a complex political economy of public, private, and nonprofit research and venture capital funding (Bryant 2018; New York Genome Center 2023). The skyrocketing demand for DTC genetic tests and incorporation of PRS into more aspects of society suggests that social groups will be increasingly emmeshed in financialized, biopolitical governance regimes predicated on modes of biological citizenship (Petryna 2004; Rose and Novas 2005). These developments warrant analyses of the undergirding institutional arrangements, policies, and deployments of capital shaping this process, as well as attention to new inequities and asymmetries it might produce.

In sum, the new age of polygenic science is poised to bring shifts in how human sexual and gender diversity is conceptualized, measured, and pinned to the body/biology in scientific research. Like past historical eras, sociogenomics research maintains that the drivers of human sexual experience and gendered behavior may be located in the body; only now, these influences are detected as piecemeal contributions from throughout the genome, interpellated with other behaviors and health conditions, using increasingly advanced statistical techniques. Despite being riddled with methodological and epistemological problems, these studies receive immense amounts of funding, generate considerable public and scientific hype, and are used to justify the need for ever-expanding research. Although GWASs are explicitly noncausal in nature and are generally run on extremely specific samples—such as only white European populations that exclude transgender and intersex persons—and despite producing results with low effect sizes or statistical significance, they are lauded as unveiling universal

truths of the human experience. As genetic science and capital interests continue to converge, scholars of gender and sexuality must attend to these developments, both to critically interrogate the knowledge they are claimed to produce, as well to understand the productive power of these forces in crafting new gender and sexual subjectivities. As measures of polygenic risk for behavioral traits continue to proliferate, so too must our concepts, critiques, and theories for making sense of this new science of sex itself.

Note

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