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Beyond a Shared History: A Biosocial Perspective on Sociogenomics and Racism in Germany

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Abstract Recent advances in sociogenomics offer new opportunities to integrate genetic and epigenetic measures into social science research on human lifespan development. Now, German social science cohorts have followed suit with this global trend. We anticipate that the integration of genetic measures into German social science cohorts is likely to be met with hesitation and dismay. Historically, racialized pseudo-science disguised as genetic research was used to justify the political exploitation, oppression, and genocide conducted by colonial and Nazi Germany regimes. In response, German institutions and social sciences actively avoided race-related research. However, avoiding the intersection of socially constructed race and genetics may stall the deconstruction of enduring racial discrimination and the identification of racialized social inequalities. Recent survey studies show that half of the German population still believe in the existence of biologically distinct human “races” and that racism is rampant. This article is aimed at providing a biosocial

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perspective on sociogenomics and racism in Germany. First, we discuss the biologicistic construction of race that became prevalent in colonial and Nazi Germany. We argue that racist legacies are sources of social inequality in contemporary German society. We further review recent human genomic science that clearly demonstrates that there is no biological basis to socially constructed race. Second, we propose a biosocial perspective that integrates how genes “get out of the skin” and racism “gets under the skin”. Transactional genetic effects, which involve human behavior and interactions between people in society, are expected to depend on environmental inequalities tied to systemic racism. We summarize recent sociogenomics studies using polygenic indices and epigenetic profile scores showing that a) genes contribute to complex human traits and b) the expression of genetic variation is affected by socioeconomic and racialized inequality. Finally, we offer a roadmap toward race-critical biosocial research that breaks with the historically informed avoidance of race to reconstruct race-critical concepts, datasets, and scientific systems.

Keywords Genetics · Racism · Gene–environment interplay · Epigenetics · Germany · Social determinants of health

Jenseits einer verknüpften Vergangenheit: Eine biosoziale Perspektive auf Soziogenomik und Rassismus in Deutschland

Zusammenfassung Der Beitrag beleuchtet die jüngsten Fortschritte in der Soziogenomik und deren Potenzial, genetische und epigenetische Faktoren in die sozialwissenschaftliche Forschung zur menschlichen Entwicklung zu integrieren. Aus einer neuen biosozialen Perspektive auf Soziogenomik und Rassismus in Deutschland wird die Wechselwirkung zwischen Genen, menschlichem Verhalten, sozialen Interaktionen und Umweltfaktoren, insbesondere systemischem Rassismus, untersucht. Durch die Zusammenfassung aktueller soziogenomischer Studien wird verdeutlicht, dass Gene zu komplexen menschlichen Merkmalen beitragen und dass der Ausdruck genetischer Unterschiede von sozioökonomischen und rassifizierten Ungleichheiten beeinflusst wird. Im Beitrag wird außerdem die historisch vorherrschende biologische Konstruktion von „Rasse“ im kolonialen und nationalsozialistischen Deutschland behandelt und aufgezeigt, wie dieses Erbe soziale Ungleichheit in der modernen deutschen Gesellschaft beeinflusst. Die aktuellsten Erkenntnisse der Humangenomik werden herangezogen, um eindeutig festzuhalten, dass es keine biologische Grundlage für das Konzept von „Rasse“ gibt. Abschließend bietet der Beitrag einen Ausblick auf rassismus-kritische biosoziale Forschung in Deutschland. Diese Forschung strebt an, Konzepte, Datensätze und wissenschaftliche Systeme rassismus-kritisch zu überprüfen und neu zu gestalten.

Schlüsselwörter Genetik · Rassismus · Gen-Umwelt-Zusammenspiel · Epigenetik · Deutschland · Soziale Determinanten der Gesundheit

1 Introduction

Recent advances in **sociogenomics**¹ offer new opportunities to integrate genetic and epigenetic measures into research on human behaviors, health, and well-being. For example, sociogenomics researchers have identified genetic variants correlated with traits commonly investigated by social and developmental scientists, such as educational attainment, body mass index, and sexual behavior (Ganna et al. 2019; Okbay et al. 2022; Pulit et al. 2019). A catalyst for sociogenomics research has been the addition of biological measures to big-data cohort studies in the USA, UK, Netherlands, Norway, and other countries (Mills and Tropf 2020). In short, “the genetic data revolution is underway” and science is rapidly advancing (Martschenko 2022, p. 717). Now, German social science cohorts, such as the Socioeconomic Panel Study and TwinLife, have followed suit with this global trend (Koellinger et al. 2023; Mönkediek et al. 2019). We anticipate that the integration of genetic measures into German social science cohorts is likely to be met with hesitation and dismay (Burt 2022; Mills and Tropf 2020). There are good reasons for this wariness.

Disguised as “genetic research,” racialized pseudo-science was used to justify the political exploitation, oppression, and genocide conducted by colonial and Nazi Germany regimes. In response, German institutions and social sciences actively avoided race-related research for several decades. Perhaps unsurprisingly, this race-evasive approach has not deconstructed racist beliefs; recent survey studies show that almost half (49%) of the German **population** still believe in the existence of

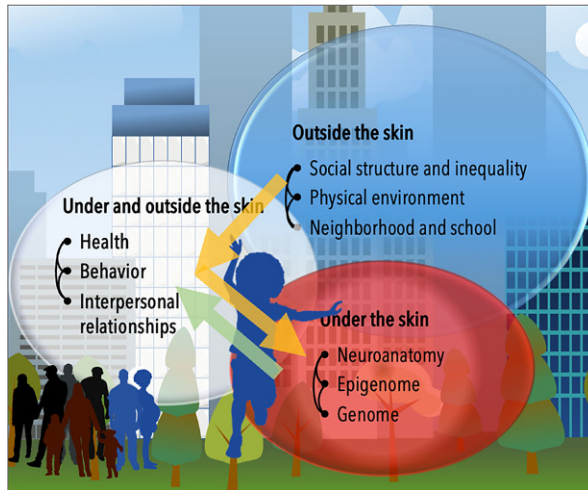


Fig. 1 A biosocial perspective frames the biological and the social as interdependent and mutually constituting forces. *Outside the skin* social inequality becomes measurable *under the skin* by influencing our proximate environments, behaviors, and interpersonal relationships (yellow arrows). *Under the skin* genetic differences influence not only our biological attributes but also our behaviors and relationships via gene-environment interplay (green arrow). Systems of social inequality impose a hierarchy on genetically influenced individual differences, which in turn affects the development of transactional traits

¹ Bolded terms are included in our glossary.

“human races” (DeZIM 2022). Moreover, nascent survey studies document racial discrimination in German public social systems, including health care, education, housing, and employment, that need to be further investigated as potential sources of population disparities in health and well-being (Aikins et al. 2021).

Although the arrival of sociogenomics in Germany carries the risk of misapplication, a biosocial perspective offers an opportunity to counteract pervasive **bio-deterministic** notions about how genes may influence life course processes. Biosocial research is “a broad concept referencing the dynamic, bidirectional interactions between biological phenomena and social relationships and contexts, which constitute processes of human development over the life course” (see Fig. 1; Harris and McDade 2018, p. 2). It transcends the dichotomy between distal and proximate levels of analysis, and frames the biological and the social as interdependent and mutually constituting forces. Biosocial research provides a lens through which the effects of both genes and **racism** on human development can be integrated.

Our article comprises three sections. First, we present a shared history of genetics and racism in Germany that led to race-evasive social science. We argue that racist legacies are sources of **social inequality** in contemporary German society. Moreover, we briefly review recent human genomic science that clearly shows that there is no biological basis to socially constructed race. In Sect. 2, we summarize recent sociogenomics studies using **polygenic indices** (PGI) and **epigenetic profile scores** indicating that (1) genes matter to human development and (2) the phenotypic expression of genetic variation is affected by socioeconomic and racialized inequality. We present a biosocial perspective that contextualizes effects of genes on human development within larger systems of racialized and economic social inequality. In Sect. 3, we offer a **race-critical** roadmap that strengthens (1) race-critical biosocial research and (2) a race-critical scientific system.

2 The Biologistic Construction of Race

2.1 A Shared History of Genetics and Racism in Germany

Black German scholars have long argued that understanding contemporary racism in Germany requires reviewing the history of European colonialism in Africa and other parts of the world (El-Tayeb 2001; Kilomba 2008; Oguntoye et al. 1992). In 1884–1885, the German chancellor von Bismarck invited American and European colonizers to formalize the partitioning of Africa. This resulted in the German occupation of territories in Togo, Cameroon, “German East Africa,” and “German South-West Africa,” and the first genocide of the twentieth century in today’s Namibia, where, in 1904–1908, Lothar von Trotha ordered the killing of thousands of Herero and Nama, partially in concentration camps (Aikins 2008; Olusoga and Erichsen 2010; Zimmerer and Zeller 2008).

A continuum of racist ideologies can be traced extending from the colonial construction of **white** biological supremacy to genocidal theories and practices (Bauche 2021; Mohsen 2020). The biologistic construction of race and racial hygiene first emerged during German colonialism. Eugen Fischer introduced the rediscovery of

Mendel's work on genetic inheritance in pea plants into the field of **physical anthropology**. The "white" or "Aryan race" was posited to possess superior intelligence, personality characteristics, and various health advantages compared with other "human races." German colonies were used as "laboratory conditions" to further develop eugenic and racial hygiene ideology in physical and psychological health (Grosse 2000). Fischer studied descendants of Dutch settlers and local Khoikhoi women of the Rehobother community as research subjects in German South-West Africa to establish the heredity of "racial characteristics" and "race" as a biological construct (Bauche 2021; Lipphardt 2012; Plümecke 2013).

In Germany, Fischer and his colleagues conducted studies on several hundred German-born children of white mothers and Black fathers from African colonial troops, whom the French stationed in post-WWI occupied Germany (Weindling 2022). Fischer later became the director of the notorious Kaiser Wilhelm Institute of Anthropology, Human Heredity, and Eugenics, which further expanded the construction of biological race and racial purity that became the cornerstone of Nazi eugenic ideology. In 1937, the Nazi regime forcibly sterilized these German-born children of white mothers and Black fathers, who were derogatively called "Rhineland Bastards," to prevent future racial impurity of the desired "Aryan" German nation (Weindling 2022).

The academics Otmar von Verschuer, Emil Abderhalden, and Josef Mengele were involved in institutionalizing racialized pseudoscientific theories by conducting dehumanizing racist experiments for medical and genetic research (Bauche et al. 2022). Mengele, a doctor from Auschwitz-Birkenau, was particularly notorious for his experiments on prisoners during the Holocaust. The biological construction of race and racial hygiene culminated in the eugenic policies of Nazi Germany that led to the systematic murder of millions of people during the Holocaust.

Unfortunately, racial violence did not end with Nazi Germany. Examples include heightened racial violence against refugees, "foreigners," and/or "un-German" people in the years following German reunification, murders by the National Socialist Underground organization (2000 to 2011), and anti-Semitic and racially motivated terrorist attacks in Halle (2019) and Hanau (2020) (Karakayalı et al. 2017; Karapın 2002; Schellenberg 2013). Anti-Black, anti-Semitic, anti-Muslim, anti-Asian, and anti-Roma and Sinti racism present in Germany today is historically tied to the scientific theories of white superiority and racial purity stemming from colonial and Nazi Germany (DeZIM 2022).

2.2 Race Evasiveness in Contemporary Germany

In an explicit attempt at preventing future state-commissioned racial atrocities, German political and scientific institutions were made "race evasive." We use the term **race evasiveness** to describe the deliberate avoidance of addressing race, racialization, and racism. This concept acknowledges that such avoidance is not passive but purposeful, reflecting an effort to uphold a status quo that favors whiteness. Race evasiveness can manifest in various ways, from actively avoiding discussions of race to downplaying the importance of race and racism (Annamma et al. 2017; Bonilla-Silva 2013). Race-evasive approaches in Germany obscure racism temporar-

ily, socially and geographically. For example, racism is commonly restricted to Nazi policies, the right-wing parliamentary party “Alternative for Germany,” British and French colonialism, or viewed as a US-specific issue (Bojadžijev et al. 2017). Consequently, there is a lack of data on the experiences of racialized groups (Ahyoud et al. 2018).

Recently, an amendment to remove or replace the term “*Rasse*” (race) in the German constitution has been proposed (Froese and Thym 2022). Capturing the present meaning of “*Rasse*” is difficult, because it is avoided in public discourses owing to its association with Nazi Germany and far-right political groups (Erbach et al. 2023). Conversations about the term “*Rasse*” predominantly revolve around historical definitions that reference biological categorizations, origin, cultural background, or national background (Kattmann 2017; Lipphardt 2019).

However, efforts to replace the German term “*Rasse*” underestimate the adaptability of the concept of race. It is its vagueness and ambiguity that make it a persistent framework (Bauche 2021). By upholding an elusive discourse in which “neither race has to be explicitly addressed nor does racism have to be offensively defended” (Bojadžijev 2015, p. 276), race-evasive patterns are sustained across generations. This is well captured by Balibar’s concept of “racism without races,” where culture and ethnicity replace race (Balibar 1990). In the first study on anti-Black discrimination in Germany called “Afrozensus,” 99.1% of 2452 respondents indicated that “people ask me where I really come from and how long I have lived in Germany” and 78.6% state: “I am told to go back to where I came from” (Aikins et al. 2021, p. 213). Critical stances against racism require a nuanced understanding of racism that is not solely based on traditional racist terminology.

Recent survey studies by the newly established “National Discrimination and Racism Monitor” document that racist beliefs in biologicistic categorizations, cultural hierarchies, and in the legitimization of social inequalities are found in 33–50% of the German population (DeZIM 2022). Accordingly, racism is present in essentially all German social systems, including health care, education, housing, and employment, that need to be further investigated as potential sources of population disparities in health and well-being (Aikins et al. 2021). Racism is not isolated to right-wing organizations; it is everywhere. We will return to sociogenomics research, exploring how these social structures may *get under the skin*, in Sect. 2. But first, we turn to lessons from human genomics that address the falsehood of genetic myths still held by so many.

2.3 Human Genetic Variation is Inherently Biosocial

Genomics has provided powerful tools to display how humans are similar to each other. “Instead of definable boundaries, genetic gradients run between human groups. Among the 3.2 billion base pairs in the human genome, there is no fixed difference that separates, for example, Africans from non-Africans” (Fischer et al. 2019)². Put differently, “we’re all related to each other to varying extents, in a complex web of

² Quote from the website of the Jena Declaration: <https://www.uni-jena.de/en/190910-je-en>.

genealogical relations that form an unimaginably complicated family tree” (Coop 2022, p. 3).

Thus, we can use genomic data to map our degree of relatedness and trace patterns of migration. For example, ancient DNA technology has shown that humans have always migrated across geographically widespread regions (Liu et al. 2021; Skoglund and Mathieson 2018). When people migrate, their language and rituals often accompany them, i.e., cultures migrate. Therefore, genes and cultures correlate (Abdellaoui et al. 2022).

Gene–culture correlation can seriously confound associations of genetic variants with complex human traits in genomic studies (for an illustrative example see Hamer and Sirota 2000). If groups differ, even a little bit, in the frequency of a genetic variant owing to **population stratification** (i.e., patterns of migration and the lack of prolonged interbreeding between subpopulations) and also differ in a **phenotype** for entirely cultural/environmental reasons, this can induce a spurious association and scientifically incorrect conclusions. Therefore, it is imperative for genomic studies of complex human traits to account for population stratification, which they typically attempt to do by restricting analyses to more or less homogenous populations and by including “principal components of **ancestry**” as covariates (Price et al. 2006).

Homogenous populations in genomics are often defined in terms of so-called genetic ancestry, e.g., “European genetic ancestry” or “African genetic ancestry.” The usage of genetic ancestry labels in genomic science has received a great deal of criticism. The justified fear is that a genetic science that partitions humans into “genetic ancestry groups” using continental geographic labels will reinforce false beliefs in the existence of biologically distinct racial groups (Fujimura and Rajagopalan 2011; Olson et al. 2005). As racial assignments “incorporate information (alongside beliefs) about individuals’ phenotypic characteristics or geographic origins, they can be said to be informed by (or correlated with) biology” (Morning 2014, p. 191). Although the biological concept of race has been scientifically delegitimized, there is risk of confusing biological and social concepts related to race, ethnicity, migration, population, and ancestry (for guidance see Bartram et al. 2023; Kattmann 2017; Lipphardt 2019). Geneticists therefore increasingly emphasize that genetic ancestry is not considered a unidimensional score linked to continental geographic categories but a multidimensional view of who your ancestors were and where they moved to (Lewis et al. 2022).

Scientifically, terms such as “European genetic ancestry” are imprecise descriptions of genetic similarity to some predefined set of reference groups (Coop 2022). Rather than reflecting some kind of stable biological boundary between human groups, the number and regional specificity of the reference groups are dependent on data availability. As more people participate and more DNA data become available, DNA-based estimates of ancestry will change. Genetic ancestry labels and the definition of what constitutes a population reflect the social context of how samples were chosen and described by researchers.

Even restricting analyses to samples with apparently high genetic similarity, such as “people whose four grandparents all come from the UK,” and including **principal components of genetic similarity**, has been found to not fully account for population stratification (Leslie et al. 2015; Olalde et al. 2018). Therefore, researchers

are increasingly using within-family designs to study genetic effects (Howe et al. 2021). For example, examining how siblings raised in the same family differ from each other is a scientifically and conceptually powerful approach to thinking about how genetic differences may influence complex human traits. In contrast, looking at DNA from different people living in different environments is useful for studying our relatedness and genetic similarity across histories of migration, mating, and mortality.

In the next section, we review sociogenomics research that has used DNA-based measures to study how economic and racialized inequality may get *under the skin*. Sociogenomics is a new scientific field that integrates genetic and epigenetic measures and theory into the social sciences to study emergent social, behavioral, and health outcomes.

3 Genes and Social Inequality Matter to Human Development

3.1 Genes Contribute to Complex Human Traits

Although all humans are 99.9% genetically the same, most parents of two or more children will have observed that the remaining 0.1% make a difference not only to how we look but also to who we are. Twin and family studies have empirically established that genes contribute to our appearance, behaviors, health, personality, and cognition through processes starting very early in ontogeny (Turkheimer 2000; Polderman et al. 2015).

After some unsuccessful candidate gene approaches, genomic studies today are commonly based on **genome-wide association studies (GWAS)** of hundreds of thousands or millions of people (for a review on computational methods see Abdellaoui et al. 2023). For example, owing to GWAS we now know common genetic regions associated with human height (Yengo et al. 2022), markers of chronic inflammation (Ligthart et al. 2018), body mass index (Pulit et al. 2019), and depression (Howard et al. 2019). Similarly, common genetic regions have been identified that correlate with highly social outcomes, such as educational attainment (Okbay et al. 2022), income (Hill et al. 2019), and risk-taking behaviors (Karlsson Linnér et al. 2019).

Results from GWAS can be used to compute polygenic indices (PGIs). PGIs are DNA-based correlates of individual differences in human traits. Compared with candidate gene studies, PGIs that summarize thousands of genetic variants, each with miniscule effect sizes identified in very large GWAS, have resulted in reproducible associations of non-negligible size (Dick et al. 2015; van de Weijer et al. 2022). For example, the most recent PGI of educational attainment accounts for up to 16% of the variance in educational attainment in separate target samples (Okbay et al. 2022). (Note that the utility of PGIs to the social sciences is an area of active debate, e.g., Meyer et al. 2023).

The biological, developmental, and environmental mechanisms involved in associating these PGIs with phenotypes are not well understood. Importantly, they are likely to differ vastly between **direct genetic effects** that are largely expressed

through *under the skin* processes and indirect and **transactional genetic effects**, which rely on developmental and social experiences (Plomin et al. 1977; Scarr and McCartney 1983).

Most phenotypic cues that we rely on to categorize others into racial groups, such as skin tone and hair texture, are influenced by direct genetic effects. The association of genotype to phenotype can be described with reference to biological components that exist within an individual's body (e.g., molecules, cells, organs). Humans are not biologically determined to belong to a certain racial group or identity, but phenotypic information is typically used to assign context-specific racial categorizations.

In contrast, most traits of interest to developmental scientists, sociologists, and epidemiologists, such as cognition, personality, education, and all-cause morbidity and mortality, are influenced by transactional genetic effects. The association between genotype and phenotype (e.g., education) depends on the behavior of an individual in interactions among people occurring inside and outside the skin (e.g., learning), which are affected by *outside the skin* social inequality (e.g., disparate school resources and safety). Their development cannot be described with reference to biological components that exist exclusively within an individual's body.

Compared with direct genetic effects, transactional genetic effects are highly dependent on environmental context. Yet, even direct genetic effects are not immutable to environmental contexts (e.g., a person's skin tone often darkens in response to sun light). To some extent, all genes dynamically respond to the environment, our internal states, the time of day, and other genes, in a probabilistic manner (Dawkins 1982; Raffington et al. 2022).

Moreover, because genes and environments correlate, gene–phenotype associations can be confounded by environments. A stringent test of genetic measures is to examine whether they can statistically account for differences within families (siblings or parent–offspring trios). Which genetic variants people inherit from their parents (and which variants they do not inherit), and how they differ genetically from their full biological siblings, is the outcome of chance. Therefore, PGI data, when combined with family structure data, allow us to root analyses in at least one variable that can be reasonably treated as exogenous.

Studies have shown that effect sizes of PGI with respective outcomes are often substantially reduced in within-family studies, commonly by 30–50%, indicating that part of the association of PGIs with outcomes is due to environmental and familial confounding, among other factors (Howe et al. 2021; Kong et al. 2018). Between-family confounding appears to be more prevalent for PGIs of transactional and highly social outcomes, such as education, compared with more proximal measures, such as inflammatory markers (Howe et al. 2021; Okbay et al. 2022). This finding is consistent with the notion that those complex human outcomes rely more heavily on interactions with the environment.

Because genetic effects are context-specific observations within a studied group of people, the estimated effects associated with a certain trait are expected to differ across populations. In genomics, those studied people have been overwhelmingly socially positioned white people in Western, educated, industrialized, rich and democratic (WEIRD) countries (Martin et al. 2019). Thus, current PGI studies do

not inform our understanding of racialized population disparities in any human trait or outcome.

3.2 A Biosocial Perspective on Genetics and Racism

To integrate how genetics and racism may influence human development, we return to our biosocial perspective in Fig. 1. The yellow arrow depicts how *outside the skin* macro-environmental factors, such as the political and socioeconomic structure that constitute dimensions of social inequality (e.g., systemic racism, classism, etc.) and associated differences in the built environment (e.g., neighborhood safety, school resources), become measurable *under the skin* by influencing our proximate environments, behaviors, health, and interpersonal relationships (e.g., air pollution, family stress).

Environmental interventions and genetic studies provide causal evidence for the importance of environmental influences on the development of cognitive, socioeconomic, and health traits (Campbell et al. 2014; Duncan et al. 2017; Engelhardt et al. 2019; Wertz et al. 2019). Further, studies of humans and other animals have identified several biological pathways through which social status and socially induced stress drives disease, including dysregulation of immune and metabolic systems (Snyder-Mackler et al. 2020). Moreover, these studies indicate that early life adversity has especially pertinent effects on individual differences in lifelong health and well-being (Hayward and Gorman 2004; Heckman 2006).

Developmental cohort studies in the USA have documented that children who identify as Black/African-American and Latinx/Hispanic are at a substantially higher risk of living in families and neighborhoods that are socioeconomically disadvantaged compared with white children and adolescents (James et al. 2021; Raffington et al. 2023c). Thus, racial and ethnic social identities correlate with socioeconomic resources. Many families of color have faced a lack of adequate access to health care, nutrition, educational opportunities, access to greenspace, clean air, and rest across the lifespan (Krieger 2020). Socioeconomic disadvantage has been found to statistically account for substantial but often not all of the racial/ethnic disparities in psychological and health outcomes (Raffington et al. 2023b). This shows that other environmental factors related to racism and white privilege, which the developmental sciences have largely neglected, affect human development (Williams 2018).

In reverse, the green arrow depicts that under-the-skin genetic differences affect not only our biological attributes (e.g., neuroanatomy, physical appearance), but also our behaviors, health, and interpersonal relationships. Critically, the developmental pathways through which direct genetic effects become associated with physical attributes, such as pigmentation gene variants involved in melanin production, are qualitatively different from the pathways through which indirect and transactional genetic effects become associated with cognition, mental health, and educational attainment. For example, children born with genetic propensities to greatly enjoy reading are more likely to read a lot and to evoke cognitively stimulating experiences from their parents (Kirkpatrick et al. 2011; Tucker-Drob and Harden 2012).

Transactional genetic effects, which involve human behavior and interactions between people in society, are expected to depend on environmental inequalities

tied to systemic racism. In this way, racism extends all the way down to become measurable in our brains, hormones, inflammatory markers, and epigenome (Goosby et al. 2018; Gravlee 2009; Krieger 2021). The social becomes biological.

Although the yellow arrow travels from *outside* to *under the skin*, the green arrow is truncated and does not end in *outside-the-skin* social systems. Genes contribute to our appearance, behaviors, and personality through complex pathways that are dependent on the environment (Lewontin 1974; Scarr and McCartney 1983). Alongside environmental chance that determines whether we are born into a poor or rich family, random genetic inheritance has been proposed to be another important source of individual differences that needs to be addressed through equity-focused social policy (Harden 2021). But, our genes do not cause systemic racism and other forms of “-isms” (e.g., classism, sexism). Our societies impose a hierarchy of value on different appearances, behaviors, and personality attributes. In turn, these social biases and structures drive population disparities in health and well-being. Genetic differences are not the cause, reason, or primary mechanism of racialized differences in complex human outcomes such as education or all-cause mortality. Racism is.

3.3 The Expression of Genetic Variation is Affected by Socioeconomic and Racialized Inequality

Recent PGI research has probed the theory that social inequality influences the expression of genetic effects. Studies have found that education-PGI became more predictive of attained education following the fall of the Soviet Union in Estonia (Rimfeld et al. 2018) and Hungary (Ujma et al. 2020). This is consistent with the theory that increasing educational opportunities across society can, in some cases, make genetic variation more visible. Further, socioeconomically advantaged schools in the USA buffer students with lower education-PGI from dropping out of math, whereas parents who provide their children with more cognitive stimulation foster their academic performance, controlling for direct genetic inheritance (Armstrong-Carter et al. 2019; Harden et al. 2020; Wertz et al. 2019). However, other studies have not found interactions between education-PGI and socioeconomic inequality, suggesting instead that they might explain largely separate aspects of school performance amongst white participants (Isungset et al. 2022; Judd et al. 2022).

There is also evidence that socioeconomic privilege can buffer against genetic risk for negative health outcomes. PGIs of body mass are less predictive of body size in Germans who report higher educational attainment and income (Frank et al. 2019). These results correspond to quasi-experimental results in the UK showing that a policy to increase the age of compulsory schooling preferentially benefits the health of people with higher PGI of body mass (Barcellos et al. 2018). Similarly, a PGI of smoking was found to be less predictive of smoking behavior amongst Black/African-Americans living in neighborhoods characterized by greater social cohesion, whereas it was amplified among individuals who had experienced an increased number of traumatic life events (Meyers et al. 2013). Collectively, these findings are consistent with hypotheses that (a) genes matter to complex human traits, (b) social inequality affects the phenotypic expression of genetic variation,

and/or (c) that different genetically influenced characteristics matter in different social contexts.

The interaction between social inequality and genetic variation is regulated by epigenetic mechanisms. Nascent epigenetic research has explored whether socioeconomic and racialized inequality are related to differential patterns of gene expression, as indicated by analysis of **DNA methylation** (DNAm; see also **Epigenetic Profile Scores** in the glossary). For example, socioeconomic inequality has consistently been found to correlate with epigenetic profile scores developed to quantify aging-related health, disease, and mortality (Raffington et al. 2023a; Raffington and Belsky 2022; Willems et al. 2023). These measures also indicate race and ethnic differences in the epigenetic regulation of aging that are associated with racialized health disparities in the USA (Crimmins et al. 2021; Graf et al. 2022). Differences in epigenetic profile scores of aging-related health between high and low socioeconomic status groups and between white and marginalized racial/ethnic groups are consistent with PGI findings that social privilege can buffer against genetic risk for negative health outcomes.

Moreover, these epigenetic profile scores appear to be able to record the emergence of socioeconomic and racialized health inequalities as early as childhood (Niccodemi et al. 2022; Raffington et al. 2023b, c; Schmitz and Duque 2022). In our own research, we computed epigenetic profile scores developed in studies of adult aging, disease, cognition, and mortality, and calculated these same profiles in over $n = 3200$ 8- to-18-year-old children and adolescents from two sociodemographically diverse US cohorts that combine twin and longitudinal study designs, the Texas Twins Project and the Future Families and Child Well-Being Study (Raffington et al. 2023b).

We found that children and adolescents growing up in low socioeconomic status families and in neighborhoods with concentrated disadvantage, and from marginalized racial and ethnic groups, already show the molecular signatures of faster biological aging, worse cognitive health, and higher adult BMI, as measured by their epigenetic profiles (Raffington et al. 2023b, c, d). This work is important because it implies that the molecular effects of socioeconomic and racialized inequality arise early in the life course, decades before racial health inequities become visible.

In sum, sociogenomics research is utilizing novel DNA-based methods, such as polygenic indices and epigenetic profile scores, to examine human development within socially stratified environments across the life course. This work has highlighted that the dynamic, bidirectional interactions between biological phenomena and social relationships and contexts truly occur over the entire life course (Harris and McDade 2018). To not only not repeat history but to use genetic science in ways that reduce inequalities in health and well-being (Harden 2021), we address some of the goals and challenges for the implementation of race-critical biosocial research in the concluding section.

4 A Roadmap Toward Race-Critical Biosocial Science

Figure 2 summarizes steps that may strengthen (1) race-critical biosocial research and (2) a race-critical scientific system.

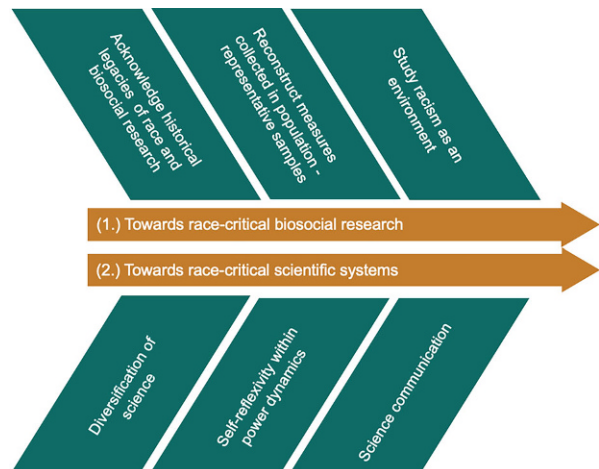
4.1 Toward Race-Critical Biosocial Research

We highlight three key components of conducting race-critical biosocial research: (1a) acknowledging historical legacies of race, (1b) reconstructing measures and concepts in population-representative samples, and (1c) studying racism as an environment. In order to counteract racist beliefs and research practices, researchers need to reflect on how the concepts used in their studies have historically been defined and utilized to legitimize discrimination (Bauche 2021; Burmeister 2021; Plümecke 2013). For example, reconstructing population and ancestry labels is gaining momentum in genomic science, where some geneticists have emphasized the need to embrace a “multidimensional, continuous view of ancestry and move away from continental ancestry categories” (Lewis et al. 2022, p. 250; Committee on the Use of Race, Ethnicity, and Ancestry as Population Descriptors in Genomics Research et al. 2023).

Similarly, the visualization of research results can serve to reinforce or counteract persistent biases. Principal component analyses of ancestry are methodologically important to genomics research, yet the scaling of corresponding charts can create an impression of large differences between populations that neglects the true continuity of human genetic variation. Importantly, colonized and racialized communities remain underrepresented in genomic sciences, but their knowledge production and tools are essential to race-critical biosocial research (Amarante et al. 2022; Harding 2016).

In a next step, measures collected in population-representative samples can be used to study racism as an environment in which human development unfolds, leading to disparities in health, education, and employment (Krieger 2021). The German

Fig. 2 A roadmap toward race-critical biosocial science



discourse on social inequality is marked by avoidance of race and racism. Correspondingly, there remains a dearth of datasets that can be leveraged to study racism. Researchers have used a variety of proxy variables to study racial discrimination in Germany, such as citizenship, country of birth, or migration background (Ahyoud et al. 2018; Bauman et al. 2018). However, these proxies are insufficient to measure socially constructed race, processes of racialization, and the effects of racism on human development. Again, marginalized communities, who have been affected by colonialism and the Holocaust, need to be centered in future efforts to conduct research on racism in Germany.

4.2 Toward a Race-Critical Scientific System

Second, we highlight three key components in constructing race-critical scientific systems: (2a) diversification, (2b) self-reflexivity of power dynamics, and (2c) science communication. German scientific systems do not adequately represent the German population (Ahyoud et al. 2018). Yet, diversification of the scientific workforce has been shown to foster innovation that serves society at large (Etzkowitz and Leydesdorff 2000; Mowery and Sampat 2006). Thus, addressing the lack of diversity among researchers through structural policies is crucial (Tilghman et al. 2021). This also includes integrating and strengthening communities-led research into science institutions. Moreover, collaborating across disciplines such as genomics, biology, sociology, anthropology, history, and philosophy can help to ensure that biosocial research is conducted in a way that contextualizes human biology within social and historical contexts and dismantles racist priors.

Further, self-reflexivity of power dynamics can be understood as a collective undertaking practiced by the scientific community that, in principle, is never complete, as it builds on the accomplishments of past reflexivity (Emirbayer and Desmond 2012). This can be practiced through self-reflection, by seeking feedback from colleagues or peers, and by creating space for community-led research. Researchers recognize their own social position in racialized systems and how this may shape the way in which data are collected, analyzed, and interpreted. This may lead to a redefinition of the aim and method of human group categorizations, particularly at the intersection of race and genetics. Applying **quantitative critical approaches** can serve as a useful tool to guide the critical evaluation of research concepts, analyses, and visualizations (Garcia et al. 2018)³. Further, researchers can incorporate principles from feminist and decolonial science to ensure that racism is not reproduced and that existing power dynamics are restructured (Harding 2015, 2016).

Finally, biosocial researchers can use a variety of science communication tools to reduce the risk of the bio-deterministic misinterpretation of their results. For example, genomic studies can emphasize the unpredictability and importance of environmental factors in phenotype–genotype predictions using plain language and visual displays (Harden 2023). Transparency in communicating the limitations and uncertainties of research findings and methods can ensure that the interpretation and understanding of the results are accurate and nuanced.

³ <https://sites.lsa.umich.edu/pvincentruz/quantcrit-resources/>.

Plain-language FAQs and education programs can improve the accuracy of scientific news coverage and public accessibility (Ganna et al. 2019). This, in turn, could facilitate the development and implementation of policies and interventions aimed at eliminating inequalities. Moreover, biosocial researchers can engage in scientific outreach. A unique randomized controlled school experiment in the USA suggests that when students learn about race and contemporary genomics—in contrast to Mendelian genetics as is common in German biology curricula—their genetic essentialist beliefs about race might decline significantly (Donovan et al. 2023).

5 Conclusion

Historically, racialized pseudo-science disguised as genetic research was used to justify the political exploitation, oppression, and genocide conducted by colonial and Nazi regimes. Thus, we anticipate that the integration of genetic measures into German social science cohorts is likely to be met with justified reluctance. Recent survey studies have revealed that a large proportion of the German population still believes that race is a biological rather than a social construct. To mitigate the risk of misapplication of sociogenomics in Germany, we proposed a roadmap for race-critical biosocial science that breaks with the historically informed avoidance of race to reconstruct race-critical concepts, datasets, and scientific systems. Although race is not defined by biological boundaries, racism has biosocial effects on people's health and well-being through their lived experiences. Indeed, novel sociogenomics tools have been used to study how social inequality affects the phenotypic expression of genetically influenced characteristics. A biosocial perspective integrates how genes get *out of the skin* and how racism gets *under the skin*.

Glossary

Ancestry	Ancestry refers to information about the people that an individual is biologically descended from, including their genetic relationships. Genetic information can be combined with historical information to infer where an individual's (distant) ancestors lived/.
Bio-deterministic	Bio-determinism or "genetic determinism" is the belief that human behavior is determined by "innate" physiology—typically a person's genetic makeup.
Black	A term referring to racialized people of African descent. Black is often associated with naming and analyzing anti-Black racism. It is also used as a socio-political and cultural identity of individuals and communities who experience anti-Black racism.
Direct genetic effects	Direct genetic effects refer to effects that largely transpire through inside the skin processes, where the influence of genotype on phenotype can be satisfactorily explained via biological processes that are minimally contingent upon the environment.
DNA methylation	DNA methylation (DNAm) describes the presence or absence of a methyl group attachment to a CpG site on the genome. DNAm allows different cells in the body with the same DNA to develop into different tissues. It is critical for development and changes dynamically with age and environmental inputs.
Epigenetic Profile Scores	Epigenetics are the biological mechanisms through which the expression of genes is regulated. This includes DNA methylation, histone modification, and noncoding RNA action. One class of epigenetic profile scores is computed on the basis of genome-wide DNA methylation. Prominent examples include

epigenetic profile scores of biological aging, also known as “epigenetic clocks”. Here, we focus on DNAm, acknowledging that epigenetic mechanisms function synergistically. DNAm describes the presence or absence of a methyl group attachment to a CpG site on the genome. DNAm allows different cells in the body with the same DNA to develop into different tissues. Thus, it is critical for development and changes dynamically with development (Fraga and Esteller 2007; Wilson and Jones 1983).

Akin to deriving polygenic indices from GWAS, researchers have used statistical methods to mine DNAm datasets and derive epigenetic profile scores of smoking, body mass index, inflammation, educational attainment, cognition, and aggressive behaviors, among others (Ligthart et al. 2016; McCartney et al. 2022; Rutledge et al. 2022; Wahl et al. 2017). The majority of epigenetic social science studies have focused on epigenetic profile scores of health and aging, sometimes referred to as “epigenetic clocks” (*for review see* Raffington and Belsky 2022). DNAm and other epigenetic modifications are considered hallmarks of aging and theorized to be key transducers of the biological embedding of social adversity (López-Otín et al. 2013). There have been several generations of epigenetic profile scores of aging-related health and these measures differ in their sensitivity to social determinants of health (Raffington and Belsky 2022).

Compared with PGIs, the development of epigenetic profile scores tends to require smaller discovery samples, result in more portable measures across populations, and have larger effect sizes in separate target samples (Hamilton et al. 2019). This may partially be due to the fact that epigenetic profile scores are capturing environmental exposures, genetic variation, and developmental idiosyncrasy (Raffington et al. 2023b).

Genome-wide association studies (GWAS)	Genome-wide association studies associate differences in single-nucleotide polymorphisms (SNPs) with differences in a phenotype. In most cases, the phenotype of interest is systematically regressed onto a single SNP and a standard set of covariates, such as age, sex, birth cohort, and the interactions among them. This model is estimated for each SNP in the dataset, often resulting in a wide variety of SNPs related to the outcome.
Phenotype	A phenotype is a trait or characteristic (i.e., depressive symptoms) that is neither a genetic variable (i.e., SNPs) nor an environmental variable (i.e., living conditions), although it is the outcome of gene–environment interplay.
Physical anthropology	Physical anthropology categorized people into races based on physical traits, including skin tone, eye color, hair texture, and skull shape, that were thought to reflect diverging genetics and evolutionary histories.
Polygenic indices	Polygenic indices are calculated as a weighted sum of a person’s alleles, where weights correspond to effect estimates reported in the GWAS summary statistics. They are not measures of something “innate” about a person.
Population	A group of individuals who share certain characteristics and who are often similar in their common ancestry, geographic location, or other demographic factors. Different scientific fields have different ways of defining populations.
Population stratification	Population stratification describes that populations of more distal ancestry differ in which genetic variants are present, how common or rare those variants are, and how those variants correlate with each other across the genome (i.e., patterns of linkage disequilibrium). The genome-wide differences that result in population stratification arise because of migration and the resulting lack of prolonged interbreeding between subpopulations. This causes allele frequencies across the genome to diverge between groups, mostly due to random fluctuations called “genetic drift” and to a lesser extent due to natural selection and/or nonrandom mating. Owing to population stratification, the results of GWAS conducted in one population are not portable to another population.
Principal components of genetic similarity	Principal components of genetic similarity refer to linear combinations of genotypes of SNPs, where each SNP has a “loading” giving its contribution to the principal component. They are often used as statistical representations of

	genetic similarity among people due to population stratification and/or cryptic relatedness. These principal components are included in genetically informative studies to reduce the bias of confounding.
Quantitative critical approaches	Quantitative critical (QuantCrit) research is an emerging field rooted in critical theory that recognizes the presence of structural racism and sexism within various societal systems, such as the economy, politics, and education. In QuantCrit research, the goal is to challenge narratives that portray marginalized groups as deficient and actively work to dismantle oppressive systems through anti-racist and anti-sexist efforts (Garcia et al. 2018).
Race-critical	A race-critical approach is based on perspective, position, and practice that is directed toward anti-racism. It analyzes and criticizes racialization processes, racist domination, and inequality, as well as their ideological legitimization, and strives for social change.
Race evasiveness	Race evasiveness serves as a strategy to disengage from conversations about race and racism. It expands the framework of color blindness by acknowledging the intentionality in avoiding substantive discussion or acknowledgment of race and its connection to white supremacy, power, and/or privilege.
Racism	Social and institutional racism includes the generational legacy of state-sanctioned social power, resources, representation, and favoritism lived by privileged people, e.g., white privilege, which comes at the cost of marginalized racial and ethnic groups.
Social inequality	Social inequality describes unequal challenges and opportunities for different social statuses, which occur at the intersection of socially constructed dimensions of race, ethnicity, skin tone, gender, wealth, education, physical ability, sexuality, nationality, and age, among others.
Sociogenomics	Sociogenomics, also called social and behavioral genomics, integrates genetic and epigenetic measures and theory into the social sciences to study emergent social, behavioral, and health outcomes.
Transactional genetic effects	Transactional genetic effects refer to effects that originate from a person's own genome, but require interaction with the environment, thereby transpiring through processes inside and outside the skin. Most traits of interest to psychologists and sociologists, such as mental health, physical health, personality, cognitive skills, and education depend on transactional gene–environment effects.
White	White describes a racial group that was derived from pseudoscientific racism. It was used as a political tool benefitting white people politically and economically.

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