Chapter 4

A Biosocial Return to Race? Racial Differences in DOHaD and Environmental Epigenetics

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4.1 Introduction

The growth of research within the Development Origins of Health and Disease (DOHaD) and related environmental epigenetics fields has catalysed a shift in the understanding of how genes and environments shape phenotypes. The attention to embryonic and fetal development as critical periods with important long-term health effects has led to a focus on the gestational environment and maternal experiences like nutrition and stress, as *intergenerational* determinants of health [1–3]. This emerging science has inspired claims that social exposures, including race-related inequalities, can drive physiological, developmental, and epigenetic processes operating in utero and during early postnatal life, becoming 'embodied' as relatively durable, albeit in principle modifiable, biological differences [4–6].

By eschewing fixed genetic differences, 'biosocial' perspectives on race have brought with them a renewed hope for a focus on the social, historical, and political bases of contemporary health disparities [7]. This emerging understanding of the role of environment-driven phenotypic and epigenetic plasticity is often viewed as aligning with progressive policy goals because it demonstrates newly appreciated pathways by which major health differentials might be reversed by timely intervention. This is reflected, for instance, in the emphasis on the 'first 1000 days' in global health initiatives [8, 9] and a vibrant area of economics that harnesses DOHaD frameworks to promote investments in maternal and child health [10, 11].

However, despite the promise of DOHaD and environmental epigenetics to set out modifiable and plastic models of biological inheritance, social scientists have illustrated how enduring forms of 'environmental determinism' [12] may become intertwined with local conceptions of racial difference. As one example, one thread of research has argued that environmental exposure to poverty (which is highly racialised in many contexts) could impair early brain development and determine children's lifelong potential [13]. Indeed, a growing number of scholars, in studies from microbiomics to brain development, have raised critiques of what could be characterised as a postgenomic reinstantia-

An extended version of this chapter has originally appeared as an article in the American Journal of Human Biology. Ref: Meloni, M., Moll, T., Issaka, A., & Kuzawa, C. W. (2022). A biosocial return to race? A cautionary view for the postgenomic era. American Journal of Human Biology, 34(7), e23742. https://onlinelibrary.wiley.com/doi/full/10.1002/ajhb.23742 This article was originally published under a CC-BY License (https://creativecommons.org/licenses/by/4.0/). It is re-printed here, with a modified title, under the same license.

https://doi.org/10.1017/9781009201704.006 Published online by Cambridge University Press

tion of race [14–18].[1] As sociologist Dorothy Roberts has warned, 'When scientists write that epigenetic effects of racial discrimination are durable across generations, it sounds perilously close to biological theories of race' [18, p. 143].

As epigenetic and DOHaD analyses of racial/ethnic health disparities expand significantly in scope and impact, we echo others in urging caution in the collection and interpretation of these new data. We recognise that much of this growing work has gravitated to biosocial understandings of health disparities in part because these understandings both avoid reductionist genetic explanations and offer new explanations that can hopefully be harnessed to foster positive social change, such as making links between current health differentials and past injustices [20–22].

Our cautionary view stems from two arguments that we will lay out in the following: in the first, we seek to undermine the assumption that environmentally driven effects are always inherently progressive. Not unlike gene-centric models of race, environmentally driven models are similarly capable of being abused and used to promote racial hierarchies, as evidenced by work on race in Latin America [23]. Here, we explore a lengthy history of proto-racism that traces presumed inherent group differences to environments, not genes. In the second, we explore the results of a review of current literature on racial health disparities in DOHaD and environmental epigenetics. This review demonstrates the enduring problems of reductionism and typological thinking in contemporary research. We believe that ongoing interdisciplinary work between social and biological scientists is key to correcting these creeping trends and strengthening this research in service of the goals of social justice. Towards these ends, we suggest various tools, such as community participation in all stages of research and moderation in reporting results, that can help avoid a potential reification of racial typologies in DOHaD research.

4.2 On the Long History of Biological Determinism and Racialisation

4.2.1 Genetic Determinism and Its Counterparts

For many contemporary researchers who grapple with debates about biological race, the modern concept that humans can be arranged into hierarchical typologies is often a starting point for discussion [24, 25]. In the eighteenth century, the Linnaean system of classifying living things, including humans [26], became the template for later anthropological work that assumed that humans could be ordered into distinct, indelible types that varied in level of sophistication as a matter of inborn potential. Modern racial science, grounded in assumptions of permanent psychophysical differences, experienced new legitimation in simplified understandings of Mendelism and early twentieth-century anthropology and eugenics. The crux of the argument was that genetic differences, assumed to determine phenotypes in a direct fashion, rendered environmental exposures or habits insignificant when considering racial characteristics: human types, now conceptualised as clustering of genes within geographically bounded groups, were viewed as fundamentally unchangeable at least within certain geographic clusters [27].

[1] Postgenomics is an increasingly common umbrella term that covers all research on the complex molecular architecture that connects genomic sequences to the phenotype, inclusive of a new set of approaches dubbed the '-omics' (e.g. epigenomics, microbiomics, and transcriptomics [19][20]).

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Genetic determinism – 'the idea that genes alone have the power to shape both bodies and behaviors' – enjoyed a remarkable albeit controversial success during the twentieth century [28]. The assumption that diverse groups of people can be characterised and essentialised based on presumed, immutable genetic characteristics has been evoked to naturalise the social, political, and historical underpinnings of inequality. As obvious examples of these dangers, during the twentieth century, research on human genetics and hard hereditarianism helped justify scourges like forced sterilisations in the USA and the Holocaust in Nazi Germany. More recently, widely discussed and controversial books have argued for a genetic basis to intelligence and a need to temper public investments in education [29], joining a long tradition of hard hereditarians that considered public welfare a wasted or misguided form of sentimentalism – a classical eugenic trope since the time of Galton (1822–1911).

As a response to the twentieth-century abuses of genetic determinism, the idea that human differences are tied to environmental influences and nurture has maintained an allure of progressivism [30], especially in the social sciences and humanities [31]. This is particularly obvious in Northern Europe and North America where most of the eugenics movement drew from theories and practices of genetic determinism. This means that the historical prominence of environments as determinants of racial typologies remains hidden. Focusing on a longer history, *spanning two millennia rather than three centuries*, demonstrates the potential for hierarchy and discrimination to be grounded in, and justified by, patterns of human difference tracing *to shared environments and experiences* (food, climate, and habits) rather than genetic or innate factors.

This ancient proto-racism reflected a persistent tendency at least since Graeco-Roman antiquity (where the most ancient evidence can be found) to refer to a range of sciences, prominently including humoralist medicine and geography, to express prejudices and a hierarchy of values among different populations, often in the context of imperial or military arguments [32]. We argue that a re-emergence of conceptualisations of body and race as open and malleable rather than fixed could lead to the subtle but gradual replication of biological race in contemporary postgenomic and biosocial developments, at a time when biology is moving away from the presumed centrality of DNA sequences as masters of phenotypic development. In what follows, we provide a concise summary of a lengthy history of pre-modern essentialism, in which intrinsic group-level human differences were viewed as an output of environmental mechanisms.

4.2.2 The Power of the Environment Before the Gene

Although this was not the only way to construct racial hierarchies in pre-modern times, the tendency to view people as deeply shaped by the places where they lived or the food they ate was a powerful intellectual device to assert the superiority of certain human groups [32, 33]. Often combined with a strong moralistic flavour, arguments about racial differences acquired through the embodiment of different environments were used to condemn whole human groups to inferiority because of the unfavourable places where they were born or, more subtly, by claiming that their placement in particularly unfavourable settings was a sign of their subordinate nature [34]. Nations were viewed as fit or unfit to rule not because of innate deficiencies but because of the power of the outside, such as the persisting effects of climate or habits on their bodies and minds. This framework has shaped pre-modern ideas of racial inferiority for centuries, connecting, with different nuances, Greek and Roman views of the East, to Columbus' interpretation of the tropics as inhabited by people unfit to 'exercise power' [35].

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In Classical Antiquity, grouping physical and moral traits of different populations and relating them to various environments – the geography of the places they lived, the climate in those areas, or the food they ate – was a common tool in developing the tropes and hierarchies of differing populations [32, 33, 37]. This is clearly seen in Hippocrates' *Airs, Waters and Places* (fifth century BCE), a medical treatise written as a guide for travelling doctors, in which Asians are described as 'more gentle and affectionate' than Greeks as they live in a land where the weather is uniform and everything grows 'more beautifully'. *Airs, Waters and Places*, while often overlooked in histories of proto-racism, was a widely influential text and translated for centuries through Pagan Antiquity, Latin and Oriental Christendom, and the Muslim world. It is considered a foundational text for theories of health, ecology, and geography of disease, and further one of the first scientific texts to establish 'the greatest and most marked differences' between Europeans and Asians.

A generation after Hippocrates, Aristotle built on these ideas to justify political differences within a wider imperial framework. People of Asia were now described as 'intelligent and skilled but cowardly. Thus, they are in a perpetual state of subjection and *enslavement*' (350 BCE: *Politics,* 7.5.6.1327b our emphasis; translation in [37, p. 44]. Filled with references to eugenic topics, the seventh book of the *Politics* (available in the West since 1260) went on to decisively influence early modern debates in the Spanish, French, and British colonies. There, the Greek/Asian dichotomy was replaced by one between temperate and tropical weather, leading to a climatological distinction between master races and naturally born slaves sealed by the authority of Aristotelian natural philosophy [38].

The Roman world continued and expanded the climatological tradition. Roman military treatises developed similar theories to maintain, for instance, that Orientals were naturally prone to slavery (from the ancient Roman historian Livy 36.17), to distinguish between the environment and hence 'innate' characters of different troops to favour a more rational process of recruiting militias (late-Roman writer Vegetius), or to avoid the risk of dangerous environmental influences in foreign areas [32]. We also see the appearance of a certain asymmetry in how negative and positive environmental effects are perceived as impacting populations, which foreshadow later doctrines of racial purity: with Roman historians like Tacitus or Livy, men transplanted from Rome into 'inferior' locales 'acquire the degenerate characteristics of the alien environment' but the reverse is only rarely mentioned [33, 37, p. 33].

While the Middle Ages are often overlooked in histories of racialisation based on environments, this period's influence on the mental cartography of early modern European colonialism and political theory was immense [39, 40]. From the twelfth century onwards, the Middle Ages saw an increasing tendency to essentialise biological differences in humoral composition based on emerging ideas of human nature, heredity, or religious affiliation. Not only were people seen as a mirror of where they lived, but human groups who differed by 'blood' were often thought to inherit the same traits if living 'under the same sky'. However, factors that could potentially alter the *innate but changeable* complexion of human groups were incorporated into medical and geographical treatises of the time. Hippocratic-Galenic ideas of environmental effects on *humours* led to concerns about the 'transplantation' of human groups into new soils and under new stars that 'would affect not only themselves but their descendants' [41], deeply shaping anxieties surrounding the first colonial expansions and lasting well into the European Renaissance and Elizabethan England [42]. Hence, colonies became places where the coloniser could be 're-raced' [42, p. 19], spurring anxieties around the potential degeneration of a nobler European 'stock' under new environmental conditions [14].

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As colonial expansion continued, and migration became increasingly common, racial science began to intermingle fixed and malleable characteristics strategically. For instance, this included the growing colonial anxiety that white settlers could degenerate in hot climates. Some historians have argued that it was precisely this fear of changeability under new environmental conditions, and the tendency of these ideas to potentially hinder colonial projects, that incentivised their replacement by notions of race as innate and immune to such environmental effects [43]. Lamarckian thinking, melded with rising social stratification and inequality, fuelled concerns about decay and degeneration in the European metropoles [44]. At the very foundation of the Enlightenment in the eighteenth century, we find a strong presence of environmental and climatological explanations of race differences. It would therefore be somewhat artificial to view the history of racism as the sole brainchild of the Enlightenment, overlooking the reality that Enlightenment intellectuals themselves wittingly inherited their ideas from Greek and Roman sources.

4.2.3 Historical Lessons for Current Work in DOHaD and Epigenetics

Our historical review highlights that the emphasis on immutable characteristics as essential to differentiating and hierarchising populations is a relatively recent phenomenon, and one influenced by the much lengthier prior history of differentiating groups based on shared environments. Without flattening different historical contexts into a simplified continuity or denying the distinct implications of environmental determinism with regard to contemporary political, legal, and economic formations, we suggest that it is possible to highlight a number of recurring characteristics in models of environmentally patterned human difference. Firstly, there is a predominance of *typological* models based on the causal power of the environment where common biological essences are viewed as being directly established by environmental effects and ignoring within-group variability. Secondly, *binary thinking* manifests in several ways. Environments were divided into categories of normal (that of the observer) and abnormal/pathological (that of the colonial subject or 'other'), and 'exposures' were similarly viewed as having effects that were either present or absent, ignoring the possibility of a spectrum of phenotypic outcomes. Thirdly, there was a tendency to establish an asymmetry between *negative* and *positive* environmental effects, with the former more common and used to characterise the developmental trajectory of non-Western or subordinated groups. Fourthly, this work often assumed that environmental and social disturbances were transferred directly to individual bodies, which are portrayed as passive recipients of external forces: damaged environments (or non-European ones) were viewed as becoming *ipso facto* damaged bodies, thus eliding a wider focus on underlying causes. Finally, it was common to argue that environmental factors can *cause loops* that are difficult to break, with whole groups being stuck in social or cultural inertia because of acquired environmental insults.

Of course, even when based on environmental models, contemporary expressions of environmentally or socially patterned race and biology do not extrapolate seamlessly from these recurring patterns and historical examples. Our point is simply that environments are neither an innocent nor an inherently more progressive factor in explaining racial health disparities. Clearly, current postgenomic work around race and embodiment has overwhelmingly good intentions – of clarifying pathways, reducing societal impacts, and addressing the unequal distribution of ill health. However, some of the

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conventions of biomedical research may create openings to unwittingly recapitulate typological and essentialised thinking [18, 45]. We thus set out to investigate the literature and findings in DOHaD and environmental epigenetics that address the role of race/ethnicity in human health.

4.3 Current Work in DOHaD, Environmental Epigenetics, and Race

How common are essentialised and typological notions of environment-driven race and human difference in the DOHaD and environmental epigenetics literature? While an important catalyst for studies of developmental plasticity, DOHaD remains a niche in a wider trend exploring relationships between epigenetic changes, particularly DNA methylation (DNAm), and racial/ethnic differences. Within this broader field, do we see an emphasis on environmental determinism, a focus on negative environments understood as leading to permanent scarring, or perspectives that foster binary interpretations of exposures and outcomes?

Before we look specifically at research that addresses race and ethnic health disparities, there are some common practices within research design in the field generally that are worth noting for their potential to contribute to a reductionist portrait. For one, some DOHaD or epigenetic studies use observational and population-based case-control designs; these have a high potential for confounding because key influences on health, such as environmental stressors, diet, or activity levels, tend to cluster as a result of influences like socio-economic status, ethnicity, class, or gender [46]. Other studies have harnessed natural or quasi-experimental designs, using 'exogenous' stressors such as a war-imposed famine, terrorist attack, global pandemic, or earthquake (for instance, [47]) to evaluate the impacts of maternal exposure during pregnancy. Because this work approximates a randomised exposure, it achieves a stronger basis for causal inference; however, it does so at the expense of studying severe shocks and stressors, which are not effective targets for intervention. (See Pentecost et al. in this volume on the move in DOHaD to preconception intervention trials.) Such 'shock' focused research is not capable of assessing more subtle exposures that reflect typical lived experience, let alone potentially beneficial or favourable exposures. On a similar note, experimental animal model research, which represents the 'gold standard' of causal evidence in this field, often imposes extreme prenatal nutritional stress on species with far less maternal capacity for fetal nutritional buffering than humans [3, 5]. In addition to using models of severe stress, relatively little DOHaD work to date has been explicitly designed to clarify the potential reversibility of early life effects (see Lloyd et al. in this volume). This creates a default assumption that any effects induced by these (again, severe) exposures are also permanent. This simplified picture of permanent scarring may be further reinforced by the common convention in biomedical research of reporting relationships in a binary fashion, as being present or absent, depending on whether a threshold for statistical significance has been reached [48] (see also Sigurdadottir and Ayis in this volume).

As many of these observations apply to population-based health research more generally, we sought to offer a specific analysis of the use of the race concept in DOHaD and environmental epigenetics. We conducted a scoping review of studies

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within the fields of DOHaD and environmental epigenetics that address racial health inequities. We limited our review to empirical human studies that focused on race and ethnicity in health and related to epigenetics within a DOHaD framework. We reviewed 49 studies in total as they met all inclusion and exclusion criteria (see [49] for a full description of our methodology).

Given the largely biomedical nature of the reviewed literature, an emphasis on pathology is predominant, and exposures are generally understood exclusively in the negative, that is, as a source of risk for chronic disease and mortality and dysfunction of biological processes. Populations emerging from often self-reported categories are reframed as aligning through differences in methylation level, for instance, from our sample: 'African American adults', 'African American children', 'black women', 'black ethnicity', 'Hispanic ethnicity', and 'Native Hawaiians'. All these groups are defined as at-risk populations mostly via reference to abnormal methylation levels, even in instances when the data do not fit with this account (e.g. higher global methylation levels, suggesting reduced cancer risk in African American children). Intra-group variability in biological responses to environmental exposures is rarely given credence, and often differences – such as immigration status or the wide array of meanings, countries, and backgrounds coalescing under 'Hispanic' in the USA – are flattened into typological race categories.

Many social scientists have urged researchers to reframe their discussions and suggestions for policy towards structural factors – namely enduring systems of racism, widespread income inequality, and the historical legacies of colonialism (see Kenney and Müller, Keaney et al., and Karpin in this volume). In our review, we found that only three articles (6 per cent) mention or recognise the importance of wider socio-structural factors as 'drivers of racial health differences' [50]. Similarly, reversibility is explicitly mentioned by 14 articles (28 per cent), but most discussions of this are brief and often limited to the conclusion.

Only a limited number of studies are self-reflective about the uncritical usage of racial categories (e.g., [50]). A few go in the opposite direction and suggest that methylation markers differ significantly by race [51], and one claims that it is possible to separate distinct populations (Caucasian American, African American, and Han Chinese American) by using differences in methylation [52]. One study is explicit about the importance of having one basal methylome map for each population and the potential value of epigenetic marks as distinct criteria for racial classification beyond and sometimes in contrast to genetic findings ([53], see [49] for further details).

A final significant finding in our sample is the application of epigenetic clock studies, which use methylation to gauge the pace of biological ageing, to explain racial or ethnic differences in health outcomes. In a highly cited article in our sample, the authors remain cautious about the mechanisms by which 'race/ethnicity and sex affect molecular markers of aging' [53]. At the same time, the study uses several conventions that reify typological thinking around human population variation. As one example, the authors describe differences across these groups in largely typological terms, without devoting space to intra-population heterogeneities (e.g. 'African Americans have been shown to have longer telomere lengths than Caucasians'; 'Hispanics have a consistently lower IEAA (i.e. intrinsic epigenetic *age* acceleration) compared to Caucasians'; 'Tsimane have a lower intrinsic aging rate than Caucasians') [53, p. 170].

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4.4 Fostering a Balanced Approach in Postgenomic Treatments of Race

Echoing a growing number of scholars [12–18], we believe it is important to interrogate practices within DOHaD and environmental epigenetics that run the risk of reinstantiating new forms of biological race. For one, we believe it important to remain vigilant against 'damage-centred research', a term coined by Indigenous academic Eve Tuck [54] to describe research that catalogues harms endured in a marginalised community with the intention of producing change, yet in practice rarely alters the social, material, or political causes of those harms and leaves populations labelled as 'damaged'. Yet, we do not want to convey only criticism: these fields are stimulating crucial new understandings of the social and historical pathways underlying health inequalities, and many communities are leveraging this research to advance agendas of social justice and community resilience ([22, 55]; Keaney et al. in this volume). In the spirit of moving beyond critique, we end with recommendations for ways that researchers can help ensure that their work benefits communities while avoiding any unintended stigma or repetition of the simplifications and pitfalls of the past.

As noted in many of our above points, there are practices across biomedical research that may contribute to reductionist, simplified, and potentially stigmatising portraits of marginalised communities. The predominant focus in DOHaD research on documenting exposure–disease relationships that are characterised in such a *de facto* binary fashion (present or absent) can reinforce the idea that populations faced with early life adversity and stress *necessarily* carry negative biological baggage because of those experiences. These binary assessments can often also fail to find evidence of an effect simply due to a small sample size or, conversely, can find evidence that biologically trivial effects are significant if sample sizes are large enough [48]. Publication bias, as Non [56] points out, also contributes to foregrounding research that shows dramatic methylation differences, but that may not translate into phenotypic differences.

This convention in reporting and discussing findings leads to a form of binary thinking in which effects are either present or not, and the magnitude of effect, or biological importance in a typical human population, often receives comparably little attention. Thus, we support the efforts in fields like statistics and epidemiology to do away with this focus on binary or 'bright line' assessments of the significance of findings [57]. Furthermore, we identify practices that could prevent stigmatising groups: (1) moving away from interpretations of data that reinforce simplified cause-effect models, (2) avoiding characterisation of outcomes as present or absent, and (3) avoiding the generalisation of pathologies to entire groups without considering the magnitude, heterogeneity, or reversibility of these effects.

Non's [56] recent review also points to other conventional practices in biomedical research that may contribute, for instance, to sampling biases favouring white populations (which perhaps feeds into the use of white populations as the norm from which other groups are seen as differing as we have detailed in our sample, see above). This type of practice, which we have documented in our sample (see for wider materials [49]), runs the risk of ascribing abnormality to marginalised communities ([45]; see also [58]). Researchers should consider the implication of their samples and the implicit racial 'narratives' (see Kenney and Müller's chapter on narrative choreographies) that may emerge as a result.

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Finally, we both echo the calls of communities involved in DOHaD research to study resilience and amelioration from early life adversity and reiterate alongside other chapters within this volume (Tu'akoi et al., Bourke, and Lovett) that future research needs greater collaboration with communities on DOHaD research. In the first, our review demonstrated an overwhelming, though not surprisingly, focus on pathology and ill heath arising from early life events. But DOHaD research cannot be limited to this. Future work should also explore the development of resilience from early adversity and the capacity for reversibility or amelioration of early life effects in response to later favourable experiences or other interventions. When reversibility is not explored, the default of permanence may often be assumed, thus increasing the potential for stigmatisation.

In the second, our point regarding reversibility has in fact been made by many communities that are the subject of DOHaD research, demonstrating the emergence of 'bottom-up' demand for research into practices that build resilience [55]. This demand points to the need for researchers to conduct future work in ways that are aligned with the interests of affected communities, including requests for reparation. This will require meaningful engagement with participants across the research cycle. (See Tu'akoi et al. and Saulnier et al. in this volume.)

Collaborative and interdisciplinary endeavours will continue to prove essential to any future efforts to improve the production, interpretation, and consumption of epigenetic and DOHaD knowledge. This volume is a testament to the growing embrace, challenges, and value of interdisciplinary work in DOHaD. If we can apply the metaphors from this field to its development, early exposure to cross-disciplinary collaboration – from inception, funding, and through the research cycle – should also foster introspection and a stronger mature science.

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