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Developmental Plasticity, Epigenetics, and Race: Historical Lessons and Contemporary Considerations

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Abstract

Recent studies demonstrating epigenetic and developmental sensitivity to early environments, as exemplified by fields like the Developmental Origins of Health and Disease (DOHaD) and environmental epigenetics, are bringing new data and models to debates on race, genetics and society. In this article, the authors first survey the historical prominence of models of environmental determinism in early formulations of racial thinking to illustrate how notions akin to biological plasticity have been used to naturalize racial hierarchy and inequality in the past. They next discuss how empirical work in DOHaD and environmental epigenetics, with its primary focus on documenting the durable impacts of early stress runs the risk of reifying perceived biological differences at the population level, not via hard-wired genes but the lingering impact of environmental exposures at critical windows of development. Specifically, they feel that common conventions in these fields tend to reinforce binary interpretations of the causes and impacts of environmental exposures that map onto ethnicity or socially defined race. This may lead to simplified causal models in which exposures are viewed as having effects that are either present or absent, and with effects impacting entire demographic groups in a typological and essentialized way. Finally, after reviewing recent trends in DOHaD research, the authors conclude with a series of suggestions that they feel will help researchers harness these new fields and methods to benefit disadvantaged groups while avoiding the dissemination of new forms of stigma or prejudice.

Developmental plasticity, epigenetics and race: Historical lessons and contemporary considerations

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1. Introduction: Race, Genetics and Society

In race-stratified societies like the United States, disease susceptibility is often strongly predicted by one's racial identity. As one well-documented example, rates of hypertension are 30-40% higher among African Americans than in other US demographic groups (Benjamin et al., 2018), and there are similar disparities in conditions like diabetes, low birth weight and renal failure (Matoba & Collins, 2017). Among some medical and public health practitioners, it is often assumed that these biological differences trace in part to

population distributions of genetic variants (Collins et al., 2003). Perhaps the best-known precedent is the example of sickle cell allele, which provides protection against *Plasmodium falciparum* malaria and was under strong selection in world regions with a high ancestral burden of this disease, including among West African populations that were an important source of the US slave trade. More recent work suggests that a high burden of treponematosis in West Africa similarly selected for a variant of the *APOL1* gene that now heightens risk for renal disease among some individuals with West African ancestry (Limou et al., 2014; Ko et al., 2013).

Despite these few examples, extensive research has failed to identify consistent genetic contributors to most race-related health inequalities, including conditions like hypertension, diabetes and low birth weight (Cooper et al., 2003). Critics of the genetic race concept have traditionally emphasized that a large majority of genetic variation is shared across all continental regions (for instance: Serre and Paabo, 2004; Lewontin, 1972), while racial group membership is defined based upon cultural, historical and political criteria specific to each society rather than to ancestry alone (Goldberg, 2016). Social epidemiologists and the environmental justice movement have shown for decades that factors that vary in relation to social-racial categories, including socioeconomic status (SES), discrimination, neighbourhood-level segregation or the maldistribution of public benefits (such as access to care) are strong predictors of disease risk (Williams 1999; Bullard, 2008), and that statistical adjustment for such factors often attenuates or fully accounts for race-related health inequalities (Kaufman et al., 1997). These findings have led to a general consensus among social scientists that race is a social construct can profoundly shape patterns of health and disease (Gravlee, 2009; Krieger, 2011; Hicks et al., 2014).

In recent years, studies demonstrating epigenetic and developmental sensitivity to early environments, as exemplified by fields like the Developmental Origins of Health and

Disease (DOHaD) and environmental epigenetics, are bringing new data and models to bear on these debates and "long-theorized process of embodiment", (Evans et al., 2021; Krieger, 2011)

The DOHaD field explores the environmental sensitivity of prenatal and early postnatal development on long-term biological development and disease risk, including via epigenetic changes that influence gene regulation (Gluckman et al., 2010). This emerging science has inspired claims that social exposures, including race-related inequalities, can drive physiological, developmental and epigenetic processes, becoming "embodied" as relatively durable biological differences (Author 3, 2009). Because embryonic and foetal development are recognized as critical periods with important long-term health effects, this has led to a focus on the gestational environment, and maternal experiences like nutrition and stress, as intergenerational determinants of health (Author 3, 2005). This emerging understanding of the role of environment-driven phenotypic and epigenetic plasticity to health outcomes is often viewed as naturally 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 (Black et al, 2013; Martorell, 2017) and a vibrant area of economics that harnesses principles of developmental plasticity to promote investments in maternal and child health (Almond and Currie, 2011).

In contrast to this, models that trace health inequalities to underlying genetic effects are often viewed as inherently fatalistic because they essentialize diverse demographic groups on the basis of presumed, immutable (hard) genetic characteristics and have been evoked to naturalize structural inequality (Lee et al., 2001; Montoya 2007). As obvious examples of these dangers, during the 20th century research in human genetics and hard hereditarianism helped justify scourges like forced sterilizations in the US and the holocaust in Nazi

Germany. More recently, widely discussed and controversial books (Herrnstein and Murray, 2010; Wade, 2014; Murray 2020) have argued for a genetic basis to intelligence and a need to temper public investments in education, joining a long tradition of hard-hereditarians that considered public welfare as a wasted or misguided form of sentimentalism(Galton, 1890).

Undoubtedly, emerging biosocial research programs that study the environmental malleability of epigenetic and developmental biology offer great hope for clarifying the pathways — and thus potential bases for reversing — social-structural health inequalities. At the same time, however, tracing group biological differences to the impacts of environments and experiences (food, climate, habits) was a prominent part of the racializing discourse in the intellectual western traditions since the beginning of Greek humoralist medicine (Isaac, 2006; Eliav-Feldon et al. 2009; Bethencourt, 2013).

With this article, we have three aims. We first survey the historical prominence of environmental determinism in early formulations of racial thinking, which underscores how certain premodern forms of what we would today call biological plasticity and biosocial thinking have dovetailed with practices, views and institutions of racial prejudice and stigmatization in the past. This body of work suggests that, if we disentangle scientific racism from modernistic ideas, and particularly Victorian fixed typologies and post-Mendelian views of innate traits, many more potential and dynamic combinations of racial stigmatization, bodies and environments appear. In the light of this longer history, we suggest to read racism in science through *a more agile conceptual instrument* (Heng, 2011). What race *is* and how certain races cohere in particular bodies changes over time and between contexts, shaped by politics and history. Race mixes up biology, bodies, and ancestry with cultural phenomena, a malleability that Peter Wade (2002) describes as “strategic equivocation” between nature and culture. The lessons from our historical evidence — that there are many templates for biological racism, or better racisms in the plural (Bethencourt, 2013) — concurs with the

social science argument that racism is a “moving target” and a “scavenger ideology” (Solomos, 1996; M’Charek 2013).

We next explore how empirical work in DOHaD and environmental epigenetics, with its primary focus on documenting the durable impacts of early stress , runs the risk of reifying biological differences at the population level not via hard-wired genes but the lingering impact of environmental exposures at critical windows of development. Specifically, we feel that common conventions in these fields tend to reinforce binary (present or absent) and typological (affecting homogenously entire groups regardless of random effects and individual variation) interpretations of the causes and impacts of environmental exposures. Given that these differential exposures often map onto ethnicity or socially-defined concepts of race, we ask whether this literature may inadvertently replicate, in novel experience-focused guises, a form of typological thinking that was common in the pre-genetic era when the biological individuality was subsumed under its group characteristics (Mayr, 1982). Third, we review recent trends in the scholarship focused on DOHaD, epigenetics and race that suggests that some of these effects may already be evident in the framing and interpretation of research aimed at unpacking the causes of race/ethnicity-based health inequalities. Although race is not always the explicit goal of this literature, the concept keeps returning, to stabilize acquired biological differences between populations that blur the boundary between plasticity and fixedness.

We conclude with a series of suggestions that we feel will help researchers harness these new fields and methods to benefit disadvantaged groups while avoiding the dissemination of new forms of stigma or prejudice for disadvantaged groups.

2. Biological plasticity in the origins of scientific racism

2.1. The malleable and the fixed in early conceptions of race

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 (Odom 1967, Stocking, 1987). In the eighteenth century, the Linnaean system of classifying living things, including humans (1735; Sloan, 1995), 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. This racism grounded in assumptions of permanent psychophysical differences found further legitimation in simplified understandings of Mendelism and early 20th century anthropology and eugenics. The crux of the argument was that genetic differences made environmental exposures or habits insignificant when considering racial characteristics: human types were viewed as fundamentally unchangeable (Weiss, 2010). This narrative is not incorrect, but it is certainly partial. A more global and longer history of racializing tropes (Wade, 2002; Heng, 2011) disrupts several neat distinctions between the innate and the acquired, bodies and surroundings, the 'biological' and the 'cultural', and gives more prominence to the role of what we would now describe as developmental or phenotypic plasticity and environmental effects in shaping stable racial types.

To be clear: Biological 'plasticity' and 'racism' *are* modern concepts. However, ideas and practices based on the more general notion that bodies, races and mentalities bear the mark of specific areas, are affected by environmental factors or weakened by 'indulgent' or 'morally corrupt' habits, and that such traits are heritable, is one of the oldest ways of defining and ranking human groups (Author 1, 2019) [see Table 1]. This ancient proto-racism reflected a tendency since Graeco-Roman antiquity to refer to a range of sciences, including medicine and geography, to express prejudices and a hierarchy of values among different populations (Isaac, 2006; Kennedy et al., 2013). As we trace racism back to this form of

premodern environmental determinism we are mindful of the need to avoid the trap of anachronistically projecting contemporary concerns onto the past (Jardine, 2000). Nor we want to deny specificity to modern racism and its violent connection to state power or racial wars (Goldberg, 2016). We have a more limited, but we believe important heuristic task: We hope to show that the association of racism with genetic determinism and related ideas of innateness and hard-wiring is a very recent historical product [table 1]. This has to be situated in a longer and complex view where the making of racial typologies was not necessarily "wedded to notions of fixity and indelibility" (Wade, 2002;). By inserting premodern racializing tropes into the conversation (Heng, 2011) we also hope to illustrate how post-Enlightenment racism drew not just on ideas of fixity but on a more subtle view of biological fluidity and environmental effects that remains a shadowy but constantly present pattern in the construction of racism.

2.2. A longer family album for 'biological' racism[i]

Although this was not the only way to construct racial hierarchies in premodern times (e.g. Goldenberg, 2003), the tendency to view people as deeply shaped by the places where they lived or the food they ate has been a powerful device to assert the superiority of certain human groups (Isaac, 2006, 2017). In this framework, *environments were understood as being inherited* in ways analogous to the role of "blood" in later times (see Wood, 2007). 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 (Livingstone, 1991). Nations were viewed as fit or unfit to rule not because of innate deficiencies but because of the persisting effects of climate or habits, on

their bodies and minds. This framework has shaped premodern 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" (Wey-Gómez, 2008). In a foundational passage of *Airs Waters and Places* (5th c BCE) Hippocrates describes Asians as "more gentle and affectionate" than Greeks as they live in a land where the weather is uniform and everything grows "more beautifully". In contrast, in the seasonally-changing weather of the Mediterranean

> the frequent shocks to the mind impart wildness**,** destroying tameness and gentleness. For this reason, I think, *Europeans are also more courageous than Asiatics*. For uniformity engenders slackness, while variation fosters endurance in both body and soul; rest and slackness are food for cowardice, endurance and exertion for bravery. *AWP* 23.25-26: our italics)

Such comments may be viewed in retrospect as little more than an ethnographic curiosity. However, a generation after Hippocrates, Aristotle, the tutor of Alexander the Great, capitalized on a variant of the Hippocratic trope – opposing here temperate and hot weather - with the intent of justifying 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*." (*Politics*, 7.1327b23–33, our emphasis; translation in Kennedy et al., 2013: 44). Political and military treatises in Rome developed similar theories alternatively to maintain that Orientals were naturally prone to slavery (Livy 36.17). In medieval times, Saracens were typically identified in line with the above medical trope as "cunning but physically weak, more likely to conquer through underhanded use of poison than by force" (Bartlett, 2001).

The embodiment and transmission of climatic or geographic factors could also be heavily imbued with ideas of virtue and nobility or inferiority and servitude (Kennedy et

al., 2013). In the pseudo-Aristotelian *Problemata* (III C BCE) after a connection is made between extreme climates and brutality of character, we read that "the Ethiopians and the Egyptians" are "bandy-legged," possibly because "their bodies become distorted by heat, like logs of wood when they become dry. "The condition of their hair," the author claims, in an obvious moralistic use of geography, "supports this theory; for it is curlier than that of other nations, and curliness is as it were crookedness of the hair" (book IV: "Problems connected with the effect of locality on temperament"; see Foster, 1927: 902). In The Middle Ages, books on the nature of places (for instance, by Albert the Great) skilfully melded naturalistic and moralistic explanations for the characters of different groups. "Everything generated in a place," argues Albertus, "derives its natural properties from that place." (cited in Author 1, 2019).

The notion that "both the physique and the characteristics of the inhabitants" were "assimilated to the nature of the land" (AWP, 8) deeply shaped anxieties of the first colonial expansions and lasted well into the European Renaissance and Elizabethan England (Floyd-Wilson, 2003). A mixture of fluidity and essentialism, humoralist views of race understood bodies and their surroundings as a fundamental unity and were hence troubled by the "transplantation" of human groups into new soils, and under new stars (Hannaford, 1996; Feerick, 2010). Hence, colonies became at the same time places for re-racing lowborn groups (ibid.) or a site of intense anxieties of degeneration of a nobler European race under new environmental conditions (food, waters, stars: Earle 2014; Baedke and Delgado 2019).

The post-Enlightenment emergence of fixed racial typologies still drew heavily on models of environmental imprints on racial traits (Schuller, 2018). Racial plasticity was, however, used to claim that the potential for change was *not available to everyone* and that certain effects of environmental imprints were too engrained to be amenable to change (Huntington 1920 cited in Campbell & Livingstone, 1983: 277). Once again, this differential

conceptualization of biological permeability – which was alternatively viewed as a burden from a phylogenetic past or an acquired capacity for social progress – speaks to the open ended nature of ideas of biological plasticity when connected to race ideology (Bowler, 1984; Author 1, 2016).

3. Epigenetics, DOHaD and contemporary understandings of biological race: perils and potentials

Our brief historical review underscores how intrinsic differences between groups were historically traced to the durable effects of environments and experiences, with the melding of scientific racism and genetics a comparably recent phenomenon [table 1]. Taking this longer historical view illustrates how biological racism does not equate strictly with innate factors, and that the congenital was seen as malleable in premodern views. It reminds us that the power of the environment to *impress directly* on bodies was easily harnessed to justify the establishment of hierarchies among human groups, with a complex negotiation about which effects were more durable and which populations could overcome the power of initial impressions. This work also evidences a *typological* emphasis – at least from Hippocrates' and Aristotle's views of 'Asians' - on considering *all individuals of a certain group* as impacted homogenously by environmental exposures with little or no space for individual trajectories or random effects. It is important to remember here that for a generation of geneticists and evolutionary theorists who contributed to the so-called Modern Synthesis of genetics and Darwinism after WWII, there was a close conceptual link between an environmentally-based paradigm of organismic change (and hence racial traits) and a typological view that leaves little room for chance and individual variation (Kronfeldner, 2006). As authors as different as Medawar, Mayr and Lewontin have claimed, an evolutionary model of change based on *direct environmental effects* – not unlike to what we see today in DOHaD and environmental epigenetics - can lead to the assumption that in a

given environment all individuals follow a similar developmental trajectory and acquire the same structures and adaptations (and possibly pass them on to succeeding generations). In this view, change is assumed to be uniform in the whole population rather than statistically distributed across individuals (Medawar, 1953; Lewontin, 1983). Thus, *binary thinking* (exposed or not), *essentialism* and *typology* (populations are reified into a common biological type and inter-individual variability is seen as a shadow of a deeper biological essence) are hence integrated in this 'environmentalist' framework (Mayr, 1982).

Of course, contemporary expressions of a fluid conceptualization of race and biology do not extrapolate seamlessly from these historical examples, given that current thinking reflects a unique confluence of political, economic, and scientific realities. Most obviously, work in fields like DOHaD and environmental epigenetics has the explicit goal of clarifying the causes, and reducing the societal impacts, of preventable disease. Because they trace health differentials to environments, they move blame from genes – which are not obvious targets for intervention – to experiences and environments, which are. When current health differentials can be understood as partially tracing to past injustices, this also helps connect a group's historically marginalized status to the biological and health inequalities that they experience today.

We agree with the spirit of this project and have made contributions to these arguments ourselves. However, we hope our historical review makes clear that tracing human biological difference to experience and environment, is not necessarily less "racializable" than other frameworks, despite the good intentions of researchers. This includes reifying simplified causal models in which exposures are viewed as having effects that are either present or absent, and with effects impacting entire demographic groups.

3.1 The design and reporting of empirical findings in DOHaD and environmental epigenetics

Diseases like obesity, diabetes or hypertension have been extensively studied and are understood as having complex, multi-factorial etiologies. Since fields like DOHaD and environmental epigenetics are relative newcomers to these already sprawling literatures, most work has aimed at demonstrating that such effects plausibly exist, with comparatively little space devoted to discussing the biological magnitude of the effects or their potential reversibility in response to favourable experiences or other forms of interventions. Indeed, a recent NIH-funded research network acknowledged the paucity of evidence-based interventions available to reverse or compensate for long-term health risks studied by the DOHaD field (Reiss et al., 2019).

The most detailed mechanistic insights into the pathways that underlie findings in the DOHaD literature, and the most causally convincing evidence, often come from animal model studies. Animal experiments often involve imposing large, and at times supraphysiologic, exposures that increase the likelihood of seeing changes in the outcome but tend to be more severe than typical gradients of experience in human populations. For instance, it is not uncommon for studies exploring the long-term impacts of prenatal undernutrition to restrict diet by one-third to one-half of the intake fed to control animals (Beauchamp et al., 2015; Zelko et al., 2019). On top of this, the most common subjects of such experiments, mice and rats, have reproductive biologies that are markedly different from that of humans, which further limits the applicability of these findings. As an illustration, a mouse litter accounts for roughly 35% of the mother's weight, which for a human would be the equivalent of giving birth to a 40-50 lb newborn. The higher relative metabolic allocation to reproduction in mice limits the potential for the mother's body to buffer the impacts of nutritional shortfall on the next generation in this species (Author 3, 2020). Indeed, there is evidence that the effect of pregnancy nutritional stress on offspring outcomes scales with body size and life history of the species, with by far the smallest effects among those species

investigated—reflecting more effective buffering—in the large-bodied and long-lived human (Author 3, 2011).

Similar issues apply to studies of these processes in human populations. Most DOHaD-oriented research is conducted using data generated by retrospective (Almond, 2006) or longitudinal cohort studies (Fraser et al., 2011; Adair et al., 2011). Longitudinal cohorts have the benefit of tracking individuals as they age, and directly measure many aspects of experience and biological state repeatedly through time. Such studies provide a powerful means of exploring pathways, while the sheer breadth of data collected allows future, unanticipated questions to be addressed. Their downside is that they tend to be observational, meaning that individuals are passively observed without experimental intervention. This leads to extensive potential for confounding because key exposures and influences on health, such as environmental stressors, diet, or activity levels, tend to cluster together as a result of influences like socioeconomic status, race, class, or gender (Hernan, 2018). Scientists pursuing DOHaD-inspired questions have approximated randomized experimental treatment by harnessing natural or quasi-experimental designs, such as by evaluating the impacts of maternal exposure during pregnancy to "exogenous" stressors like a terrorist attack, a global pandemic, or earthquake (LaPlante et al., 2008; Torche, 2011). Because this work approximates a randomized exposure, it achieves a stronger basis for causal inference, but it does so at the expense of studying unusually severe shocks and stressors. As with animal model experiments, this focuses attention on the effects of unusual exposures that are not generally targets for policy or intervention, and may be less able to address any beneficial effects of enrichment or favourable exposures that are less amenable to study as an exogenous shock.

In addition to these common features of DOHaD studies, which run the risk of painting a simplified picture of permanent scarring, these practices are further reinforced by

the common scientific convention of reporting relationships in a binary way, as being 'present' or 'absent', depending on whether a threshold for statistical significance has been reached. In fields like statistics and epidemiology, there has been a strong push to do away with this focus on binary or "bright line" assessments of the significance of findings (Cummins and Marks, 2020), which can fail to find evidence of an effect simply due to small sample size, or conversely, can find evidence that biologically-trivial effects are significant if sample sizes are large enough (Wasserstein and Lazar, 2016).

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. These issues may be further exacerbated by the common publication biases in most fields that tend to reward publication of findings that are viewed as interesting, while discouraging efforts to write up and publish negative findings (Easterbrook et al., 1991).

The predominant focus in DOHaD research on documenting exposure-disease relationships that are characterized in a *de facto* binary fashion can reinforce the idea that populations faced with early life adversity and stress tend to carry persisting negative biological baggage as a result of those experiences. These arguments apply to the fields of DOHaD and epigenetics in general. However, we feel that they have particular salience when applied to address race-based health inequity, because they have the potential to slot back into historic norms of viewing race as an acquired but now congenital category that individuals are born with, and that characterizes the health and societal potential of entire populations. Here we see this potential for binary thinking to be extended – with effects not only present or absent, but with this label characterizing a demographic group, such as a member of a self-identified or societally-imposed race, *as a whole*.

3.2 A critical appraisal of the status of race in contemporary DOHaD research

Surveying the subset of DOHaD and epigenetic research that involves discussion of biological race reveals that some of these concerns are beginning to materialize. To gain insights into these issues, we surveyed four key journals in this field — the *American Journal of Human Biology*, the *Journal of the Developmental Origins of Health and Disease*, *Social Science & Medicine* and *Environmental Epigenetics* — for articles since 2010 that connect fetal exposure to adult health outcomes, and that brought race into their cases, context, or analysis[ii]. Together, this included 66 articles within these journals. Cohorts analyzed included racial/ethnic groups as different as Indigenous Australians, black US women, South Asian neonates, Yup'ik Alaskan women, 6-8-year old Maya children, Mediterranean/Muslim mothers in the Netherlands, and First Nations communities in Canada. To be clear, this is not meant to be a systematic review, but as an exploration of how racial categories are evoked and interpretated in this contemporary work.

The first notable feature of this work is the great variability in terms and categories employed, reflecting the historical and political nature of these definitions (Lee et al., 2001; Yudell, 2016). As one example, a Brazilian study used the categories of black, white, brown (*pardo*), yellow, and indigenous, which participants self-reported (Mueller et al., 2015). In contrast, a British study divided between South Asian and white Dutch, depending on whether all four grandparents were of those racial/ethnic categories (Karamali et al., 2015). A South African study (Slemming et al., 2017) used *apartheid*-era classifications, using white, black, "mixed/coloured," and Indian. However, even within the same context, ethnographic evidence shows that racial categories are unstable and mercurial. In Natali Valdez's (2019) research in a UK prenatal randomized control trial, she described the racial "improvisation" in the assigning of specific ethnic codes to participants, and with codes systems changing across years. In practice, assigning racial codes to participants was an unstable and contingent

negotiation between the data collection staffers and participants (Valdez, 2019). This heterogeneity in terms and usages underscores the central point that membership in racial categories does not have a clear genetic basis, but instead traces to historical, social and political forces.

In our literature review, most articles merely mentioned race/ethnicity as a confounder, with little to no explanation of how race was defined or why it was relevant (Washburn et al., 2010; Nye et al., 2016; Workman and Kelly, 2017). This echoes findings by previous analyses of race in published scientific work (Lee et al, 2009; Shim, 2005). Shim (2005) argues that this "standard operating procedure" to control for race is both a by-product of scientific infrastructures (funding requirements, such as for the US National Institutes of Health), an acknowledgement of race in shaping health inequalities, the need for replicability and reproducibility of research findings, and simply research habits. The use of race, Shim finds, acts as a cultural short-hand for a range of genetic, environmental, and cultural factors ("lifestyles", diets, exercise) that the epidemiologists she interviewed believe race encompassed. Without explanation for the relevance, usefulness, and meaning of race in these articles, researchers are in effect reproducing race as a meaningful biological category (Shim, 2005).

A second subset of articles provide the framing that genes *and* environment represent causal explanations for race inequalities in health pointing out that genetics, on its own, cannot fully explain these persisting gaps. Here, research is framed as shoring up the associations between, for instance, the "vulnerabilities of the African-American kidney" (Lampl et al., 2012), metabolic disorders (Wells, 2010), and methylation changes (Workalemahu, 2021), through the "environment." At times the "environment" is evoked to fortify the meaningfulness of fixed racial categories where genetics has fallen short. In one journal article, the authors start the article, "The trajectory for a person's health may be set

before they are born" (Harville et al., 2020: 188) and then describe how "experiences" and "prenatal and perinatal" environments "shape health. This kind of description reinforces the notion of phenotypes as inherited, if not through genetic explanations then through environmental ones. In such an encompassing explanation, there is often less space devoted to individual variation, agency, change, or resilience.

By saying that genetic links to racial health inequalities have come up short and then adding "environments" as inherited and effectively inborn, this work reproduces a variant of biological determinism, or one part of the "double-helix of racial inequality" (Byrd and Hughey, 2015). These tropes are appearing not only in DOHaD or epigenetic research, but also in other research spheres. In analysis of the neuroscience of poverty, Victoria Pitts-Taylor (2019) describes how scientific literature measures and classifies, and ultimately can reify, the "neurobiologically poor." This, she argues, creates the potential for "bio-social determinism," whereby biology and the social factors that in turn shape biology can become more or less fixed for certain populations, and obscures the political economy of poverty (2019:672-3). Pitts-Taylor's work demonstrates that determinism and the "fixing" – by this she means the classifying and reifying - of certain populations as damaged remains, regardless of whether the causality comes from genetic features or environmental features. That is, rather than contributing to an undoing of biological determinism, adding "the environment" reinforces it.

She further points to the habit of research foregrounding the body and biological at the expense of the social, political, and historical. An example here is when scholars reference racial or ethnic health inequalities (or the more neutral 'disparities') without acknowledging the historical and political contexts that shape unequal health outcomes — such as colonialism, enduring racial discrimination, or xenophobia (Gurven, 2012; Pearce et al., 2014). This critique is what sociologist and bioethicist Dorothy Roberts (2019) has

warned against: that while the new "biosocial" sciences offer the potential to document the harms of unequal structural violence, these fields thus far often obscure the political relationships that cause harm. This instead perpetuates the notion of inequality "as a product of flaws in peoples' bodies" (2016: 127). Backgrounding social, political, and historical context not only perpetuates the idea of flawed biology, but has the further implication of "reframing responsibilities in individualizing ways" (Pentecost & Ross, 2019:755), lead to often critiqued habits of "blame the mother" (Sharp, Lawlor & Richardson, 2014). These scholars emphasize the importance of ensuring that the political-economic and historical reasons for racial inequalities in health outcomes remain at the forefront of research.

All the recent empirical work that we review is well intentioned and not doubt makes novel and valuable additions to their fields. Furthermore, a few articles have also challenged some of the established assumptions on long-term adverse health effects of some in utero experience (for instance with ritual fasting: Savitri et al., 2020) while others have been explicit about the importance of including distal, structural determinants that lie outside health intervention (Slemming et al., 2017). Others explored explicitly the health impacts of racial discrimination (Dixon et al 2012; Thayer & Kuzawa 2015).

Our concern remains what new stereotypes, stigmas and norms regarding the biology of race could these new ideas foster in the minds of researchers, policy makers and the public. Some scholars have noted that the racialization of certain genetic diseases – sickle-cell anemia or cystic fibrosis – can lead to misdiagnoses (Yudell et al., 2016), a potential problem that may remain in the age of biosocial medicine. No less insidious is the potential for pathologizing certain populations, and essentializing certain races and ethnicities as enduringly at-risk for metabolic illnesses, cardiovascular disease, and learning disabilities emerging from environmental factors. The dangers of this approach are illustrated by work linking measures like birth weight or placental thickness with later cognitive function (Pearce

et al., 2014) or IQ scores (Misra et al., 2012). We feel that in the context of racially-defined cohorts extreme care must be taken to avoid co-option of research findings in service of eugenic arguments used to justify defunding of public investment in education or other social entitlements (e.g. Murray and Hernstein 1994; Wade 2014), this time in developmental rather than genetic form. In this light, the persisting use of a deterministic language of e.g. 'programming' or 'scarring' in the DOHaD literature also remains a reason for concern (for instance: Almond and Currie, 2011; McEniry, 2013; Escher, 2018; Roseboom et al., 2021).

The continued cataloguing of harms measured in the bodies of marginalized communities led Indigenous academic Eve Tuck (2009) to call for a suspension of what she calls "damage-centered research." By this, Tuck refers to research that documents pain and injury, without correspondingly inquiring about resilience, variation and strengths. In this regard, our review found an overwhelmingly negative skew. With few exceptions — some more "neutral" research (Helfrecht et al., 2018); or reviews of interventions to build resiliency (Phillips-Beck et al., 2019) — research exclusively examined negative effects of exposures, harms, and traumas. The overwhelmingly negative skew of DOHaD research (and epigenetics more broadly) in our review (for instance: Schrock et al 2017; Steine et al 2020; McCabe et al., 2020; Mah et al., 2020) can also easily be characterized as "damage-centered". Despite good intentions however, as Tuck notices, more often than not, cataloguing harms results in no changes to the material and political underpinnings of harms, and instead merely leaves populations with the label and self-perception of "damaged," perpetuating a modern variant of inborn hierarchy albeit framed in compassionate terms.

4. Fostering a balanced approach to study design, interpretation and dissemination

Fields like DOHaD and the study of environmental epigenetics are providing new tools to help clarify how structural inequalities can manifest biologically as health inequalities. Like many, we feel that these fields hold promise to help redirect attention in health inequalities research away from genes, which have generally not born fruit, to the effects of specific pathways that link health and wellbeing to modifiable features of environments and experiences. Our goal in writing this review is to highlight the need for care as we undertake this enterprise. Practices common to fields that focus on the long-term impacts of early environments reinforce the idea that stressors lead inevitably to "programming" of later disease, or potentially severe "scarring". These effects are then discussed as being present or absent in a binary fashion, and as characterizing entire demographic groups without consideration of intragroup heterogeneities. This illustrates how one form of biological essentialism tracing to presumed genetic influences can, if care is not taken, be replaced by a an environmentally-grounded one, a model that we have shown has important historical precedents.

While social criticism of DOHaD has thus far largely focused on the structural imbalance in gender discourses (Sharp et al., 2018), we believe it is important to encourage a specific focus on 'DOHaD and race' to mitigate some of the unintended consequences that emerging research may have. In our critical review, we certainly do not intend to convey that DOHaD is solely a negative field, and indeed, as we have emphasized, the field has helped stimulate crucial new understandings of the social pathways underlying health inequalities. We do feel, however, that care is needed to ensure that this work does not end up causing unintended harm to the communities that it seeks to benefit.. First, at a foundational level, it is important to be mindful of the common practice of describing effects in binary terms based upon a threshold of statistical significance, and instead candidly discuss the biological importance of any such effect and their underlying contingencies. Here we think it important

to acknowledge the ways in which animal model findings are good precedents for understanding human effects, while also discussing the ways that findings may be less generalizable. Although animal models give us powerful insights into biological pathways likely operative in our species, there are often reasons that the quantitative nature of an effect, such as the size of an exposure that generates a response, or the magnitude of that biological response, may be less applicable to humans (Author 3, 2011).

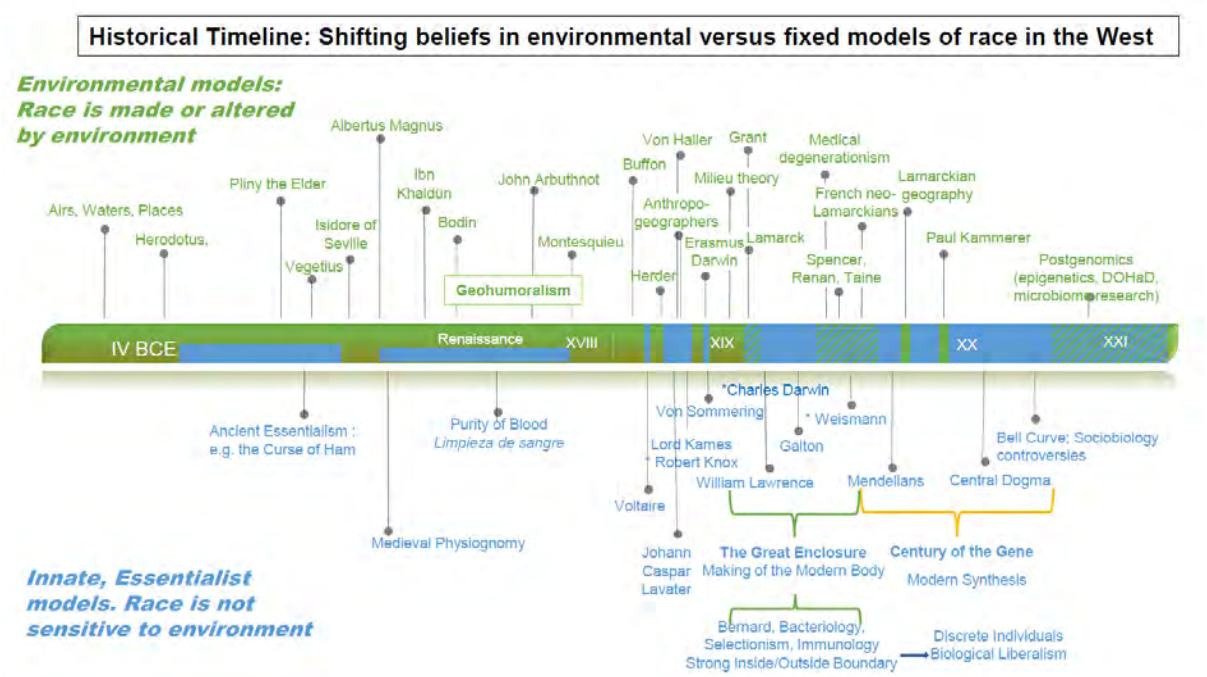
As a second point, it would be helpful for future work to explore not just the development of resilience from early adversity (Vassoler et al., 2013) but also the reversibility of early life effects in response to later favourable experiences or other interventions. When reversibility is not explored, the default of permanence may often be assumed, thus increasing risks of stigmatisation. It is significant that recent epigenetic research has increasingly highlighted the benefits of environmental enrichment (Gapp et al., 2016), and some of these findings have also influenced the DOHaD-related literature (Taouk and Schulkin, 2016). Building on this and similar work (for instance in early education) is certainly of vital importance moving forward.

Our final and perhaps broadest point is the need to be explicit about foregrounding the social, political and historical underpinnings of inequalities, and to not let these details get muddled in the drive to describe biological mechanisms (Roberts, 2019). It is important to emphasize that some of the articles in our review directly grappled with these issues. Important examples – for instance in a special issue on Indigenous health in the *Journal of Developmental Origins of Health and Disease* - foregrounded the colonial legacies in present-day health outcomes, questioned the meaningfulness of racial/demographic categories based on colonial legacies, and emphasized variability of biological effects within Indigenous populations (Bombay et al., 2019; Salmon et al., 2019, see also: Hicks et al., 2014). These articles are notable in their attempt to explain why race, ethnicity, and

Indigeneity remain relevant markers of health and, furthermore, foreground colonialism and racism as social, political, and economic contexts. Similarly, other researchers in the field are highlighting the need for "larger, more diverse and representative population-based samples" in epigenetics (and DOHaD) research, particularly to avoid the possibility that "race/ethnicity, gender, and socioeconomic status" will be described only "as individual-level characteristics" rather than at the wider scale of socio-structural effects (Evans et al., 2021). Bringing together different scales of analysis, from the micro-cellular to the socio-structural, will be essential if we hope to connect the dots linking individual experiences of environments with the broader societal structures that shape those environments.

Collaborative and interdisciplinary endeavours are essential to this goal given the entangled, bio-social nature of epigenetic knowledge (Müller et al., 2017). We are reminded that post-WWII genetics was able to disentangle itself from some of its darkest racist applications only thanks to intense exchanges and collaborations with anthropologists, sociologists, and historians which lead to a more liberal and humanistic view of race (Smocovitis, 2012). If we can apply the metaphors from this field to its own development, early exposures to cross-disciplinary collaborations should help foster critical introspection and a stronger mature science. We hope that this article, and the meeting of disciplines represented by us as authors, help to nurture this project.

Table 1-



Legend: *While Charles Darwin's selectionism complicates this dichotomy and his model of pangenesis included environmental origins of variation, the overall impact of Darwinian selectionism aligns more with the innate model.

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[i] The debate between 'racialism' and 'racism' escapes the goal of our article and is somehow inapplicable in the clean form that philosopher would want to past historical documents (Appiah, 1993). This is why, we will use for simplicity racism for the overall article.

[ii] We did a PubMed using key terms "DOHaD," "fetal programming," and "intrauterine environment." These results since 2010 were cross-referenced to avoid overlaps. Searches then filtered to assure articles focused on racial/ethnic health disparities/inequalities or made reference to a particular racialized group.