Archaeological Dialogues (2020), **27**, 1–16
doi:10.1017/S1380203820000021

CAMBRIDGE UNIVERSITY PRESS

DISCUSSION

On the biodeterministic imagination

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Abstract
Biological determinism continues to rest on belief rather than evidence. The racial genetics of David Reich and his immediate predecessors exemplify science applied as racist ideology which obscures evidence for social criticism and moral accountability for inequity.

Keywords: Genetics; naturalism; racism; Reich; biocultural; epistemology

Introduction

> *The ancient Greeks also had slaves. They were not Negroes but white men who had been taken captive in war. There could be no talk of racial differences. And yet Aristotle, one of the great Greek philosophers, declared slaves inferior beings who were justly subdued and deprived of their liberty. It is clear that he was enmeshed in a traditional prejudice from which, despite his extraordinary intellect, he could not free himself.*
> (Albert Einstein, 'The Negro Question', *Pageant*, 1946, in Jerome and Taylor, 2005, 87–88).

A near fetish of DNA has arisen in Western science and society. Appearing as the mark of objective and universal fact, the excessive authority ascribed to genetic explanation is undergirded by a philosophy of *nature* which precedes Enlightenment science, certainly back to Aristotle's fallacious use of it to argue that slavery is 'just' (in his *Politics*). It resides in Christian notions of a grand scheme of nature deliberately synthesized in European Enlightenment science as it transitioned to a secular use of nature (Marks 2018).

The concept of nature is used in many ways (Blakey 1991), among which is the ideological externalization of cause to it and away from moral human decision making (apology). In evolutionary schemata, 'nature' may provide the most plausible evidential framework available regarding the causes of hominin interspecific variation over deep time. When, however, evolutionary theory is loaded with the a priori intuition of *ranking* made sensible by common social and economic inequities in the lives of scholars, evidence of expanding biological variation in the material natural world is converted into a view of nature as testament to universal inequality, thus disavowing current societal injustice as choice. The resulting idea, that biological variation determines human social ranks and conditions, is the active ingredient of racism. The biodeterministic imagination ties historical and societal differences to any correlated biological traits which can be found to justify the status quo.

This article is about the dangers of archaeological collaboration, or intuitive agreement, with biological determinists. Whether considering human evolution, ecological theories of 'prehistory', biohistory or the tracking of major human migrations and inter-group relationships or disease etiologies with genetic information, it will be important to recognize genetics' intrinsic subjectivity and political implications. Also, as geneticists, including David Reich, are soliciting archaeological

and palaeo-anthropological samples from archaeologists around the world, these should be aware of such geneticists' unacknowledged technical limitations and the political leanings evident in Reich's interpretations of human DNA.

Obstinate

As the Cold War wound down, the more equitable worlds we had come to imagine by the Second World War either transformed or waned. The political, economic and scientific pendulum swung back again in the United States. The biodeterminists, long engrained if recently dislodged, were intrepid. The Western populace, led by elite schools and past intuition, continued to produce and consume racial IQ studies, the new sociobiology, biocriminology and soft-line natural histories in charming media and authoritative curricula. These continued to compete with the simultaneous advance of social-science exposés of the artificiality, rather than naturalness, of class and race inequities due to power relations created by greed and maintained, as the wealthy only can, with popular complicity in capitalist democracies.

The adamant persistence of biological determinism (the reduction of all phenomena to biological, genetic or natural causes) is based on its consistent ideological service to apology (justifying the status quo of social inequalities). Its storyline rarely avails practical information. Biological determinism is a logical component of the naturalism which generally defines European Enlightenment interpretations of life. While often viewed as an objective basis of self-critical and secular scientific thought, naturalism (more philosophy than thing) and the institution of science (which evaluates and writes it) owes much to the Christian roots of the Enlightenment (see Jennings 2010; Keel 2018; Marks 2018). As a form of *externalization* (cause externalized from human decision making), natural causes effectively supplanted acts of God as scientists came to repopulate the rooms of the monastery. There, religious or secular, a world view was constructed defining acceptable paths for the negotiation of inegalitarian social life, much of which would be seen as beyond one's control.

For reasons requiring a more thoroughgoing analysis than is possible here, such ideology would long represent a negotiation of rising post-agricultural systems of inequity against a human intolerance of unfairness. Human sociality is a reciprocal system (Blakey 1994b; Montagu 1955). Audrey Smedley (2007) shows one consequence of this in Thomas Jefferson's 'paradox', which personifies the American world view to come: he uses Enlightenment science to resolve the contradiction between his belief that 'All men are created equal' and his right to enslave other human beings to reproduce his profit and comfort. This secular apology came on the heels of a full realization: 'The Almighty has no attribute which can take side with us [enslavers]' (Jefferson 1785, 163). Only if people were naturally unequal might slavery be just, as Aristotle argued with cherry-picked data in his *Politics*, lest the republic and its masters be deemed 'unjust' during Alexander's imperialism. Jefferson and his heirs were determined to know themselves as 'naturally' just while hoarding the fruits of other's lands and labours. Such apologetic conclusions often trumped the contrary evidence of deliberate human economic exploitation of otherwise equal persons.

African diasporic intellectuals for whom the lie was obvious were the first to point out that the American and French academies disregarded the evidence of equally capable black people belaboured by exploitative 'circumstances' (Douglass 1950) and anthropology's convenient absence of 'positivism' when it came to assertions of a natural white-supremacist order (Firmin 2000). All fell on the deaf ears of European and white American anthropologists, retarding science for as much as a century (Fluehr-Lobban 2000). Franz Boas (1911; 1940) would later make a similar case, most consistently for American Jews. The foothold social science had established after the devastating stupidity of Nazi eugenics and the value of an epistemological turn toward 'environment' (further stimulated by the civil rights movement's criticism of social inequality) would be undercut by the

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

lingering biodeterministic imagination and its structural reinforcement in the authority still awarded to natural science. I will show that the convenient belief in apologetic naturalism continues to compel the otherwise inexplicable return to eugenical claims by David Reich and his immediate predecessors.

As the science of race waned, genetic or DNA variation continued to be elevated as the key to knowing what we *really* are (for which 'it's in our DNA' has achieved broad metaphorical use). In essence, while race once was the surrogate for genomes, genomes became the surrogate for race in societies infused by the biodeteriministic imagination. In this world of surrogates and dog whistles, an advancing formal societal recognition of the morality of anti-racism would take to new terms that simultaneously protected white privilege. The popular myth of the level playing field made black 'reverse-racism' claims seem logical (despite the fact that virtually all institutional racism was white supremacy) in opposition to affirmative-action correctives. The silencing and delegitimation of the discussion of racism made its very recognition facile as 'playing the race card' (see Haney Lopez 2009). Thus racism was rhetorically redressed by whites by simply denying its existence, fallaciously leading to accusations of racism against those who report it, and therefore evading redress of many of the material and intellectual privileges of white supremacy.

The sciences of such alleged post-racial societies, certainly in the US, participated in those logics which, absent acknowledgement of white privilege, implied by default that hierarchy (still obviously remaining before our eyes) is 'natural' (Blakey 1994a). White Americans, while ostensibly shifting to the new ethical high ground of anti-racism, remained in the way of progress in psychological fragility, intolerant of exposure (see DiAngelo 2011). It is this racist anti-racism to which geneticist David Reich reacts today, attempting to extricate genetics from the arena of its contradictions, but in precisely the wrong way.

For this problem to continue there must be an effective amnesia. Now 75 years after the Nazis, naturalism still adheres, too deeply embedded, too ideological, too dear to the defence of the status quo to be scratched below its surface. What did we forget? Nature, as an idea manipulated by human hands, authorized Jim Crow, immigration restriction, apartheid and the Holocaust while furthering a common, unempathetic world view that inequalities of our world were not inequities. Phase by phase, the class apologetics of worthy elite familial blood lines (Isenberg 2016), elaborated by an initial group affiliation with supernatural Christian election or Christendom, would be followed by 19th-century anthropologies of white supremacy (Jennings 2010; Keel 2018) which made these Godly entitlements 'natural', to be further institutionalized as the eugenical synthesis of polygenesis in motion as social Darwinisms (Blakey 1987; 1996; Gould 1996; Marks 2018). Humans, whether as different static species or evolving races were rightly (naturally) ruled by a white elite. Eventually, even non-North Western European whites were incorporated into racial ranking in intermediate tiers. Morally exposed by its mortal effects on these marginalized *whites* during the Second World War (Cesaire 2000), anti-colonial movements, and civil rights protest, the false technical assumptions of biological determinism were exposed (see Montagu 1951, UNESCO Statement on Race) and the naturalness of race categories was debunked (Livingstone 1962; see understandingrace.org; Blakey 1999).

There was scientific experimentation, as the use of systematic evidence is a scientific innovation to be valued best as a questionable hedge against baseless belief. Were the Holocaust's six million eugenic killings not a sufficient test, not only of the inhumane methods of applied genetics, but of the *technical irrelevance of genetic variation for purposes of human betterment*? They proved that genes have nothing to do with it. Was Germany or the world improved by the intensive genetic engineering the Nazis *completed*?

Indeed, the tautological fallacy of the fact that the genetically inferior people can only be identified as the oppressed group (obviously not the rulers) is problem quite enough. The fact that Albert Einstein (now *the* symbol of intelligence) *fled* Germany as a result of its racial hygiene with others of his kind to help improve America and elevate their lowly eugenical status within it (from Grant 1916 to Sacks 1994) has nothing to do with improved breeding, but represents a clear social

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

process. The experiment failed. What a tragedy, indeed, to lose sight of that fact. Nor should one overlook that in some minds, to 'Make America great again' is to view the end of the days of eugenically sanctioned racial segregation as a mistake. Locking the borders on the brown, Mexican and Muslim 'horde' (as US eugenics banned swarthy Eastern and Southern Europeans in the 1920s), and gating wealth with black and brown incarcerations that criminalize the consequences of inequity, show that some public policy initiatives in the 21st century persistently mirror the eugenical imagination. Naturalism provides unempathetic and amoral cover for the inequities of capital's accumulation of worker's surplus value, to blame the 'undeserving other' for the frustrations of 'hard-working [white] Americans', and for the enormous and otherwise starkly unfair taking of the 1 to 5 percent of the world's wealthiest citizens.

There are antithetical historical-materialist analyses of the human condition that lay responsibility at our own feet. Marxist political economy informs us that human biology and health are also social phenomena, moulded by, and reflections of, our social histories and environment (Doyal 1979; Levins and Lewontin 1987). The resources upon which the organism depends and the power, or lack thereof, to access resources have defined the poignant element of the human environment ever since agriculture put our societies in control of our needs. It is an environment of our own making. Africana scholars argued that 'Man ... achieves by making his own history' (Firmin 2000) and that social inequities create the conditions that vary our biologies (Douglass 1950; Du Bois 1906) over a century ago, as a rising tide of biocultural anthropology (Goodman and Leatherman 1998; Zuckerman and Martin 2016) shows today.

These two ideas continue to clash. The brand long favoured by elite and white power, the nature politic, is most securely established. It is established not only as a scientific paradigm in the Kuhnian (1970) sense of assumptions maintained among lineages of scientists' gated conversations, but also among race- and class-segmented publics where the nature politic is marketed and an elite only talk and listen to themselves.

The non-racial (or implied racial) biodeterminism of our current era exfoliates the now tainted trappings of its marked racist past to claim a new skin for the reproduction of old, admonished, purposes. Below, I offer three examples of the biodeterministic imagination of the past 40 years (biological history, medical genetics and behavioural genetics of IQ). These efficient examples demonstrate the elements of belief and denial intrinsic to such scientific results. Lessons from these examples prepare the intuitive basis for my critique of Reich.

Sanguine

'Bio-history' was embraced by historians in the 1980s to provide natural explanations of American history. In so doing, humanists employed the authority of natural science. It resembles an expected pattern of soft scientists attaching themselves to natural-science authority, which Allen (1975) showed occurred in the 1930s, just as biologists have expressed 'physics envy' pursuing the authority of universal truth (Gould 1996; epitomized by Lumsden and Wilson 1981)[1] – nearer my God to thee.

Several influential authors in this vein extrapolated from the real regional differences in infectious-disease immunities to construct nature as a teleological hand of God in service to whites. It is a fundamental fact that people from the Old World had developed immunities to many of the epidemic diseases such as measles and chickenpox to which they were commonly exposed. The peoples of the Americas and the Pacific, not previously exposed to these diseases, only developed immunities to them after suffering the devastating epidemics of first European contact. Native peoples of the Americas were devastated time and again upon first encounters with Europeans, but ultimately acclimatized (not by natural selection) to the same level of resistance as Europeans, though at obvious great cost. Several authors will overly attribute both the success and the causes (essentially the inevitability) of European conquest to these and other natural

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differences. Diamond (1997), physiologist-cum-historian, saw complementary advantages of European 'guns, germs, and steel' in his narrative of 'fate' behind the late, unchosen, colonial European hoarding of others' wealth. That teleological view, from a retrospective of 11,000 years of 'different rates' of continental development, substitutes for the immoral choices of the last 500 (ibid., 16). The nuanced line between apologetic interpretation and other descriptions is thus easily overstepped.

Kiple and King (1981; Kiple 1984; 1988) extrapolated wildly. Their most telling analysis attributes even slavery itself to 'racial factors' (Kiple and King 1981, 154) in immunities of 'the black', thus naturally equipped for enslaved work because of their lower mortality from Old World diseases. Note the illogical stretch from capacity to cause (I have fingers, so I write). African immunities, however (both acquired and due to their unacknowledged advanced use of inoculation; see Cobb 1981; Koo 2007), would make of Africans more immediately durable labourers than were indigenous Americans. Native slavery would become pervasive, nonetheless, in Latin America. More importantly, Europeans too carried Old World immunities. How was it inevitable to enslave Africans for the same acquired traits that Europeans possessed? Imagine racial biology as the cause.

Their ancillary analysis of racism is equally misguided. They might have argued that Europeans chose to apply their Christian chauvinism and increasingly drew upon their physiognomic distinctions from others to dehumanize them for brutal exploitation. My analysis shows something like that. But Kiple and King (1981, 23–207) attribute racism to another natural cause: the purported genetic susceptibility of 'the black' to tuberculosis, cholera and other infectious diseases (including syphilis) which brought racial stigmata and, with this, prejudices against them.[2] These are diseases of poverty, and syphilis was first spread by European colonials to non-European women whom they raped and who had not previously known the infection (see Khudabux 1991; Null et al. 2009). These are results of European imperialism and racial slavery, not the reverse. Naturalizing slavery is to externalize it from the processes of human decision making and immorality, with a dispassionate gaze resembling objectivity.

Alfred Crosby embraced the miracle of natural ecological theory most tightly. His Phi Beta Kappa Award-winning book *Ecological Imperialism* (1986) explained the conquest of the Americas as a simple matter of natural organic competition. Whether the superiorities of Europeans are genetic or acquired seems unimportant to his grand scheme of nature. The growth and spread of European populations in 'the temperate zones' are sufficient evidence of their organic competitiveness in his book. The natural explanation of European competitive superiority is enabled by Crosby's use of portmanteau organisms: the crops, livestock, even weeds that were attached to the European human organism. To these he adds Africans, the collateral of colonization, like the spread of Spanish moss, a fertile packing material. All now being neatly represented as European humans and their portmanteau, these naturally outcompeted American and South Pacific organisms as Europeans naturally terraformed the temperate Americas, South Africa and New Zealand to make them into little Europas. To top off the evocation of inevitability, Crosby seeds his biological history with biblical quotations to achieve a story of 'God on our side, to nature on our side, and back again' (Blakey 1989, 421). Perhaps some found this compelling, touched still by an ancient Enlightenment world view of white election over the non-Adamic other. It is but apologetic natural philosophy painted onto history.

The biodeterministic imagination is in this instance as blinding as is race. Consider that five million of the six million people who migrated from the Old World to the New between 1492 and 1776 were African (Dodson 2001, 119). Africans were deliberately made part of European enterprises, indeed, but only in the imagined category of 'portmanteau' organisms do they become vestigial parts of European biology, less even than Aristotle's metaphorical 'hands' on a body ruled by Greeks as 'heads'. Perhaps even less than as cattle (sharing the Latin root for chattel), as blacks were more generally imagined in their time, despite their continuous human resistance and intelligent contributions (such as agricultural knowledge) to the building not only of the

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Americas, but also of the European Industrial Revolution (Inikori 2002), later defining modern American culture and civil rights. Only a corrupted imagination, then and now, would invent these black and brown human persons as the weeds they themselves plucked and the animals they tended. Even 'barnyard fowl' recognized these as 'men', Frederick Douglass (1950) retorted when the first craniometric biodeterminists defended slavery by claiming, scientifically, that they were not, and as such were unharmed by whites' enslavement of them.

Medical genetics

Hypertension A love of natural, Darwinian, explanation is clearer still in the popular clinical medical mythology of the etiology of African American hypertension. Wilson and Grim (1991) proposed an evolutionary hypothesis for high rates of essential hypertension among African Americans, previously published in Kiple (1988) (Wilson 1988). They argued that high disease rates resulted from a force of selection unique to the transatlantic Middle Passage, which *would have* selected for a gene responsible for increased sodium retention among retching, sweating human cargoes.

The theory is a good example of what other scientists, especially those who follow the Boasian habit of demanding data for Darwinian conclusions, call a 'just-so story'. It is a story with natural-historical props and nothing more. The heat, perspiration and dehydration of the Middle Passage were undoubtedly real. A gene for variation in the efficiency of sodium loss, however, has never been observed, much less its variation for those before or after the passage. In other words, while this hypothesis carries the scientific authority of being an evolutionary explanation, it remains unadorned by scientific evidence a quarter-century after its proposal, despite criticism of its plausibility (Armelagos 2005; Curtin 1992). Yet Jared Diamond asserts it uncritically in his larger narrative (1997) and I cannot count the number of times a physician has offered the Middle Passage explanation as proven fact.

The massive Detroit studies of Earnest Harburg and his associates (1978a; 1978b) looked for an association between blood pressures and statistical models of inheritance but found instead that the social and economic factors used as controls to refine genetic observations were in fact the sole independent variables determining blood pressure differences between groups. Divided into high and low socio-economic status groups (differing income, job status, wealth, etc.) generally corresponding to inversely high and low social instability (crime, family disruption, etc.) correlated with the major differences in blood pressure throughout Detroit and its suburban communities.

An interesting finding, first considered as evidence of a genetic etiology of hypertension, was the correlation of higher pigmentation (darker skin colour) with higher blood pressures among African American Detroiters. Was this evidence of African genes for high blood pressure?

Of stunning importance for me, if not for Dr Harburg (Harburg, Gleiberman and Harburg 1982), for white Detroiters the correlation of skin colour and blood pressure was the opposite of that shown for blacks. Whites with the lightest skin (Irish descent populations, more representative of inner-city whites than the Mediterranean Greek, Italian and Middle Eastern suburbanites with higher incomes) had the highest blood pressures. The world distribution of skin pigmentation is continuous (from darkest to lightest people). Were skin colour (as African genetic admixture) the independent variable, both correlations would have been in the same direction, like a slash (/) on a line graph, yet Detroit's graph was shaped like an X, with the biological (skin) correlation for blacks' blood pressures running opposite to that for whites.

This study accidentally bumped into a brilliant demonstration of the central role of social and economic factors in blood pressure differences, and the role of social history in human biology. The socio-economic correlates of skin colour among African Americans are different from those of European Americans. While low pigmentation was correlated with higher average education in blacks (and therefore roughly with income), low pigmentation among whites was associated with

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

different socio-economic histories of their pigment-differentiated groups. African American ethnic history and biography are replete with colour-caste privileges (Russell, Wilson and Hall 1992) of the white supremacy with which they contend daily. Privileges were extended with greater likelihood to the yellower people descended from the plantocracy, who were more often than their darker cousins awarded the better jobs by white people within the structure of resource allocation of a society whose white supremacy is institutional, interpersonal and internalized. Blacks' own aesthetic around hair and facial features has been notoriously contaminated by these things. African American high blood pressures are, therefore, not a natural matter of fact, but evidence of the societal discrimination to which black Americans and/or the poor are chronically exposed from cradle to grave.

Earnest Harburg, on the other hand, launched a project in the South Pacific to explore his imagined normal relationship (the white pattern) there based on the hypothesis that the neurotransmitter, melatonin, of the pineal gland could elevate blood pressure (Harburg, Gleiberman and Harburg 1982). But this melanin-producing gland has nothing to do with skin pigment variation. A loyal adherent to nature, indeed.

Note that genetics is usually irrelevant to drug treatments for hypertension and the vast majority of other disorders. Treatment affects physiology, which is evolved to respond in large part to environmental differences experienced by genetically similar organisms. Such fluctuations (like high and low blood pressures and immune resistance) are its adaptive purpose (understood in neo-Darwinian terms), which in our inequitable socially taxing society often leads to pathological disorders, challenging human structural (homeostatic) limits. Genes locking one into one side or another of such fluctuation is expensive and wasteful, and defeats the purpose of physiological adaptation, as would being born sweating or dry, vasodilated or shunted, happy or sad, rather than respond with these as needed for environmental acclimation. Pharmaceuticals effect such plastic physiological states as are underfunctioning to restore them within effective limits.

BiDil BiDil, a drug for heart failure, was the first medication marketed to only one racial group. Its flimsy research protocol, which tested *only* blacks, found just enough effect to win FDA approval for marketing the drug to blacks 'for the specific indication of congestive heart failure in African Americans' (Brody and Hunt 2006, 556). Consider the biased assumption that an effective drug in blacks is somehow unlikely to be effective among others who, if white, would constitute a standard human test group. According to Kahn (2004, 33), 'The role of the federal legal and regulatory system in producing BiDil as an ethnic drug is especially important because it lends the imprimatur of the state to the use of race as a biological category'. The fact that the drug was effective in blacks in no way demonstrates racial variation in its effectiveness. Nor does this demonstrate genetic variation at all. The search for real genetic (base-pair) effects, futile as it might be, is curtailed by the marketed belief in an imagined racial basis for drug effectiveness.

Brody and Hunt (2006, 559) take the problem to its economic entanglement: understanding that the 'great majority of clinical trials of drugs in the United States are now funded by the pharmaceutical industry'. They showed that researchers recognized that 'race is a poor scientific prop upon which to base the efficacy of a drug', but used it anyway because 'there is every incentive for the [pharmaceutical] company to decline to undertake … research' concerning 'the identification of a specific genetic trait, correlated with positive therapeutic response', if what they might find would contract their sales market. Remember this when considering Reich's prostate cancer analysis.

Furthermore, 'BiDil offers a good example of how sociocultural factors in disease causation may be overlooked as a result of an overly simplistic assumption of a racial and hence presumed genetic difference' (ibid., 556):

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> There is a danger that the apparent success of BiDil will lead to a further de-emphasis of research into these social and environmental contributors to disease, while all the research funding is devoted to possible genetic bases. We already have seen a major shift in research funding in the United States as a result of the heavy influence of the pharmaceutical industry. A possibly highly effective nondrug treatment for a life-threatening disease is today less likely to receive research support than a slightly effective drug therapy for a minor lifestyle condition where a lucrative market exists. The BiDil experience is likely to cause this disparity in research funding to grow (ibid., 559).

The human genome

The Human Genome Project was the big genetics project of the 21st century, with an NIH (Francis Collins) and a private (J. Craig Venter) branch. A third component called the Human Genome Diversity Project established in 1991 and led by Luca Cavalli-Sforza was financially abandoned because its re-racialized assumptions (the existence of static non-European 'tribes' as an alternative language of race; see Pierre 2013) were brought under heightened scrutiny (I participated in the Wenner-Gren Foundation-sponsored meetings with NSF in 1994 that led to reducing the funding of the Diversity Project). It did not convince agencies that its approaches to sampling human genetic variation passed ethical standards or were technically useful. The Genographic Project, backed by IBM and the National Geographic Society, nonetheless replaced it for the commercial farming of the genomes of the Third World (see Marks 2018, 97–99).

Human biologists in need of funding did as most people seeking support will do: they exaggerated the value of their work. The HGP, unlike its Diversity Project, successfully convinced NIH and the public that all manner of social problems and diseases would likely be better understood and solved by a billion-dollar investment in genetics.

The Human Genome Project, public and private, did the important job of describing the human genome in molecular terms. The public promise and excitement about the Human Genome Project, however, concerned imagined results which that project and many others before it have *not* produced. The idea is that DNA will show us the *real* causes of our disorders – organic, psychological and behavioural. Certainly, knowing the landscape of base-pair sequences is the better way to explore any such effects of genes on disease. The answers we are getting, however, rarely show more than minute genetic difference, usually of no known significance, between different social and economic groups along which lines the big differences in psychological, chronic and infectious diseases are shown to occur. We are encouraged to have faith that studies failing to show a genetic etiology still mean that a positive finding is just around the corner. Old racial and natural intuitions bolster the sense that such hopes are reasonable.

Increasingly, physicians use genetics to test the varieties of cancer within us to devise more precise treatments. This, I think, is a good thing. What remains a separate issue is the search for intraspecific human variation (racial or otherwise) in the causes of most diseases or the recent research shift to identifying racially varied efficacies of different drug treatments for disease. Such studies pay geneticists but usually show correlations that are so thin as to rarely exceed the fallacy of BiDil.

The complexity shown by genetic mapping has also led to a new emphasis on epigenetics, which, if not carefully nuanced, might transform the societal conditions which interact with human biology into an aspect of genetics. Lord help us if geneticists become the hard-science interpreters of environmental effects (their interactions at the cellular level) as though the physiological effects of gene–environment interaction in individuals are the ultimate causes of different disease rates, rather than focus on differing societal circumstances produced by political and economic organisation that vary different social groups' exposures to pathological risk.

Another new development, which actually began in my shop (see Mack and Blakey 2004), albeit by a renegade researcher (see Nelson 2016), is the commercial use of molecular genetics

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

to trace genealogy. Direct-to-consumer (DTC) ancestry companies like AfricanAncestry.com or 23 and Me sell the identification of a living person's cultural origins. Their methodological reliability is rarely tested beyond internal consistency between statistical results and the company's own algorithmic biases. This circular reasoning of reliability is similar to *The bell curve's* demonstrably unbiased ability to measure what it measures (see below) or Lewis *et al.*'s (2011) attempt to re-establish Morton's craniometric 'objectivity' by showing that his measurements were repeatable, despite profoundly subjective results based on the necessarily biased interpretations required to give measurements meaning. A DTC company's assertions of reliability are irrelevant to whether or not the methods accurately answer the consumer's question about his or her origins in the past 500 years (Nelson 2016; Reardon 2017). When most people ask about reliable or unbiased knowledge, they want to know whether or not their questions are reasonably answered, which scientists may conveniently misinterpret as whether or not their methods were diligently applied.

When tested by others using likely comparable methods (Abel and Sandoval-Velasco 2016) and with persons of known African ancestry (Ely *et al.* 2006; 2007), the mtDNA matches ranged from 10 to 14 per cent reliable, to an arguable 50 per cent unreliability given common genetic overlap with widely dispersed African populations. This is different from selling the public 'some' information. How reliable are genetic estimates of ancient migrations without this level of verification? As the original inspiration for these services (Nelson 2016), the African Burial Ground Project acknowledged that many subjective factors (which SNPs, how many SNPs and which comparative databases were used) completely alter the ancient origins identified (Mack and Blakey 2004). Genetics is one line of evidence, no better than artefacts. One should scrutinize what all genetics, not just racial genetics, has actually demonstrated, not its promises.

Haughty

IQ studies have traditionally been deeply biased by the unvalidated assumptions of their statistical models of inheritance. In the first decades of the 20th century, Charles Spearman's *g* (for general intelligence or native intelligence) was a statistical artefact which falsely stood for objective intelligence (Spearman 1904; in Gould 1996, 287–288):

> a person's performances on various mental tests tend to be positively correlated – that is, if you do well on one kind of test, you tend to do well on others. This result is scarcely surprising, and is subject to either purely genetic (the innate thing in the head that boosts all scores) or purely environmental interpretation (good books and good childhood nutrition to enhance all performances). Therefore, the positive correlations [constituting the mysterious *g* that stands as inherited intelligence for biodeterminists] say nothing in themselves about causes (Gould 1996, 371).

Sir Godfrey Thomson (1939, 299) found that an equally objective resetting of the statistic reified as a thing called 'general intelligence' or even mental 'energy' converted it into diverse intelligences rather than Spearman's 'monarchic' reduction to a single 'engine' of intelligence. This example of Gould's evinces the differences between what I call objectivity #1 (observation and measurement) and false objectivity #2 (scientific neutrality capable of universal truth). Diverse interpretations of an albeit singularly measured, imagined and reified 'thing' (in this case the statistical artefact deemed *g*) do not speak for it as a single, neutral fact. Interpretations determine meaning, not measurement, and interpretation is always subjective.

I now turn to the highly publicized and best-selling *The bell curve* of Herrnstein and Murray (1994; Blakey 1996a), to apply Stephen Jay Gould's ample evisceration of it. *The bell curve* sought to demonstrate that the genetics presumably attached to an individual's IQ score accounted for the

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financial successes of a 'cognitive elite' on the one hand and for poverty and crime on the other. It recommended ending Head Start, a Great Society programme of early supplementary education for poorer children. Gould (1996), and the consistently critical commentary of biological anthropologists in *Current anthropology* (1996; including me, in full disclosure), found its authors to be insultingly disingenuous. For Gould the book's disingenuousness was in its content:

> denying that race is an important subject of the book at all; instead ... [blaming] the press for unfairly fanning these particular flames, while the whole book is as much about racial differences, perhaps more so, as about individuals; or Murray's denial cloaked in an arrogantly false modesty 'Here is what we hope will be our contribution to the discussion [of race]. We put it in italics; if we could we would put it in neon lights: *The answer doesn't much matter*' (Gould 1996, 370)

and in its argument, claiming that its IQ test results are unbiased:

> Lack of S-[statistical] bias means that the same score, when achieved by members of different groups, predicts the same consequence – that is, a black person and a white person with an identical IQ score of 100 will have the same probabilities for doing anything that IQ is supposed to predict ... But V-bias, the source of public concern, embodies an entirely different issue that, unfortunately, uses the same word. The public wants to know whether blacks average 85 and whites 100 because society treats blacks unfairly – that is, whether lower black scores record bias in the social sense (Gould 1996, 374).

Furthermore, the study's R2 correlations (including those between IQ and criminality independently of socio-economic status) were weak (the vast majority below 0.1) and hidden in the appendices (Gould 1996, 370–78).

In the end, the truth will out. One lay reader (Leon Wiesletier) said in the *New republic* (31 October 1994), 'Murray, too, is hiding the hardness of his politics behind the hardness of his science. And his science, for all I know, is soft'. The authors finally clarify the meaning of their deliberately arcane science in what Gould lays bare:

> The penultimate chapter presents an apocalyptic vision of a society with a growing underclass permanently mired in the inevitable sloth of their low IQs. They will take over our city centers, keep having illegitimate babies (for many are too stupid to practice birth control), commit more crimes, and ultimately require a kind of custodial state, more to keep them in check (and out of our high IQ neighborhoods) than with any hope for an amelioration that low IQ makes impossible in any case. Herrnstein and Murray actually write (p. 526): 'In short, by custodial state, we have in mind a high-tech and more lavish version of the Indian reservation for some substantial minority of the nation's population, while the rest of America tries to go about its business' (Gould 1996, 377).

Occupiers, still.

It was all the talk of Washington back then that the timing of *The bell curve*'s release to correspond to that of a proposed Republican contract with America was no coincidence. Those policies, partly implemented through diverse laws, were for reduced school spending, increased prison building, reduced environmental regulation of industries and an attenuation of the welfare system.

While all programmes related to affirmative action were roundly discouraged by *The bell curve*, one example best reveals its mean spirit, and bears repeating. The Head Start programme gave added educational time, tools and environment to poor pre-school and elementary-school children who were usually black and brown. Despite accelerated performance toward the white

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

middle class while in the programme, Head Start kids' scores rapidly declined once they were reintroduced to the common conditions of their community's public schools (Herrnstein and Murray 1994).

The idea that those results demonstrate that performance is tied to a genetic tether, rather than a need to elevate all the conditions of these children's later years, is to subordinate reason to an unbelievably stupefying level of self-deception (of which we will see more). The expectation that the individual and his genes are the prevailing cause of these children's loss of achievement seems as realistic as the inevitable emergence of Lord Greystoke as King of the Jungle.

Reich: prostate cancer and IQ return to race

In 2018 a *New York times* opinion by Harvard geneticist David Reich advocated that the new method (to observe actual genes in relation to behaviours) was ready to be imported from its current arena of individual variation back to 'race', from which, I have argued, the naked truth had chased the well-dressed lie years ago.

Reich attempted to shed biodeterminism's tainted past by emphasizing the need to take a more humane approach to racialized behavioural genetics than before. He sought to harness the authority of objectivity by stressing that it was not science but its misuse that was the problem:

> The concern is that such research, no matter how well intentioned, is located on a slippery slope that leads to the kinds of pseudoscientific arguments about biological difference that were used in the past to try to justify the slave trade, the eugenics movement and Nazis' murder of six million Jews (Reich 2018a).

This scientist, however, walks the same road paved with good intentions as past scientists, and operates within the terms of the same epistemic institution in which they operated (as science, not pseudoscience), coming, in his case, from an interpretive vantage of similar privilege to that of previous biodeterministic scientists (in the US, Jews have become white and disproportionately elite).

Reich first dismisses any necessary critical gaze upon his work by casting the problems of scientific racism as non-science (pseudoscience), thus naively protecting the institution of science as mythically neutral. All science is and must be subject to the common sense (varying with societal experience and political interests) and cultural assumptions which give its numbers meaning to its human producers and consumers. Only outright liars (like Sir Cyril Burt) might be deemed pseudoscientists. Polygeny and eugenics constituted the sincere but fallacious reasoning of the mainstream of human biologists on both sides of the Atlantic for 150 years. Post-Second World War transformations of the political climate allowed a critical lens to reveal the technical and moral fallacies of an intensively and globally tested racial determinism. Thus the academy turned its attention in new non-racial biological and sociological research directions, contesting still-entrenched biodeterministic belief. The current institutionalisation of that historic test and turn is now deemed 'orthodoxy' (dogma) by Reich, when nothing could be farther from the truth of science history, though a truth inconvenient for his proposed return to the disproven beliefs he wants, with professional benefit, to pursue:

> It is true that race is a social construct. It is also true, as Dr. Lewontin wrote, that human populations are remarkably similar to each other from a genetic point of view. But over the years this consensus has morphed, seemingly without questioning, into an orthodoxy. The orthodoxy maintains that the average genetic differences among people grouped according to today's racial terms are so trivial when it comes to meaningful biological traits that those differences can be ignored (Reich 2018a).

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

Well, that's quite true. Science has repeatedly shown, but under enormous questioning, that these anti-racist facts are left standing against the grain of dominant naturalizing scientific values refined a century ago by the Harvard–Washington (Smithsonian) 'Axis' (Spencer 1979) and Cold Spring Harbor (Allen 1975), whose eugenics were enthusiastically supported by the moneyed elite (Ludmerer 1972).

In the Western world in which white and elite privilege continues to live, the big genetics of Reich's professional life remain keeper of the flame of a hopeful imagination of those wishing to assume that their unwillingness to share the commonly produced wealth is 'natural' (acceptable). Reich's 'sympathy for the concern' that his data will be 'misused' by the lay public seems pompous to me when I recognize that the problem is actually the exaggerated facts he and his colleagues create again and again to appear as differences 'no longer possible to ignore'. No human biologist has said that average difference cannot be found among groups of all kinds. The point is that these are artefacts of non-racial evolutionary processes and rarely demonstrative of biologically or behaviourally significant effects. So we move on.

He gives the example of his study of seven 'risk factors' related to prostate cancer at a genomic location 'with about 2.8 percent more African ancestry than the average … African Americans who happen to have entirely European ancestry in this small section of their genomes had about the same risk for prostate cancer as random Europeans'. We should ask, what socially relevant phenotypic traits also correlate with this measure of 'African' or 'European' ancestry that would effect different social histories in these individuals? Is this like Harburg and his associates' observation of a pigment correlation with blood pressure, only to find that pigment relates to stressful conditions and income? As Reich said, however, 'we found exactly what we [geneticists] were looking for', not what those interested in the social etiology of disease would look for. Such possibilities must be tested, if the genetic causative argument is to depart from the theatre of just-so stories where wishful thinking may be narrated into fact.

Reich and his colleagues were actually unable to identify a causative gene (Freedman *et al.* 2006). They attempt to circumvent that failing by substituting disease and trait correlations with African admixture under the dubious assumption of the existence of European alleles and African alleles in the absolute. Neither their study nor another upon which the significance of the 8q24 locus rests (Amundadottir *et al.* 2006) had evidence of the genes' effect: 'the contribution of the -8 allele [at the 8q24 locus] to risk is insignificant' in their African American sample and it had no greater effect than thousands of traits that just happen to be more frequent in African Americans. Reich's study concluded only that they were in the vicinity of 'a major, still-unidentified risk gene for prostate cancer at 8q24, motivating intense work to find it' (Freedman *et al.* 2006, 14068). Was their answer still right around the corner from where Ales Hrdlička left it in 1921 when amassing countless anthropometric measurements for the day when we would know 'what is right and what is not' (Hrldička 1921; in Blakey 1987, 14)? The failed non-racialized genetics, now obscured by substituting the facile intuition of racial determinism, might only have become explanatory if he were to do the work of actually demonstrating the causative physiology (not correlation) and epidemiological significance of his 7 SNPS. He did not.

I would look for the possibilities that these genes are but an artefact of social histories, allelic correlates of racialized physical features, perhaps, that expose to differences in the socio-economic and toxic environments in which cancers emerge. The study of physiological effects of racism (social analysis) is different from biodeterministic studies of race (racial biological analysis). Such study is warranted before any biodeterministic conclusions can be made.

Avaricious

Reich (2018a) argues that his desire to return to the racial analysis of *intelligence* is different from those of colleagues whose open claims of white supremacy and genetic claims for black inferiority brought sanction upon them:[3]

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

> What makes Dr. Watson's and Mr. Wade's [racist] statements so insidious is that they start with the accurate observation that many academics are implausibly denying the possibility of average genetic differences among human populations [geographical races, actually], and then end with a claim – backed by no evidence – that they know what those differences are and that they correspond to racist stereotypes. They use the reluctance of the academic community to openly discuss these fraught issues to provide rhetorical cover for hateful ideas and old racist canards.

So those in the scientific community who have moved beyond racial explanation after nearly two centuries of testing are to be blamed for Watson's racist comments because they are through talking about the little 'average genetic differences' between them, thus giving cover for hateful ideas? What about, let us move on, does he not understand? His complaint does resonate, however, with a real problem mentioned early in this paper. The silencing and evasion of both the discussion and redress of racism are real in white America (although African Americans frequently discuss it and ever attempt to marginalize it in their lives). If consistent with other white Americans, Reich's colleagues have three options: (1) discuss the harshness of racism from which they benefit in white privilege, (2) assert the validity of racial biology as a determinant of the inequalities they see around them, or (3) evade, with emotional fragility, any public mention of the problem of race and racism. Reich, in fact, takes up the same mission he claims, among others, to be racist (option 2), only attempting to absolve them of the problem of not being 'backed by … evidence'.

Reich's comments embodied the recent objections to 'political correctness' (option 3) of his white peers involved in the same kinds of 'private conversation' about race and intelligence on which Herrnstein commented in *Atlantic Monthly* as early as 1971, against precepts of the Great Society (or the possibility of option 1). Here, they are stuck, unable to acknowledge the unfairness of the fact that the wealth into which they were born is not equally distributed among other equally hard-working families, and that their legator's past and their current class advantages and racism (see, for example, Turner, Fix and Struyk 1991; Pager, Western and Bonikowski 2009, experimentally proving massive hiring discrimination) are largely responsible. Racist explanations remain a desirable alternative to questioning their moral right to hoard but are publicly silenced by a disingenuous civil discourse (the myth of the level playing field) which only evades the problem. While Reich's company rightfully deem this fatuous, their alternative is a charitable scientific re-examination of race and IQ to part the haze, if only to scrape for the smallest effects geneticists imagine must be plausible. More a return to honest racism than moving on. Whatever happened to option 1?

If not disingenuous, Reich's comments reflect that he and his colleagues feel cornered – by facts, I think. In that corner, he sets the stage for his next offering, a racial basis for differences in intelligence. Rather than recommend another – say, biocultural – research direction and policy correctives to obvious societal inequities likely to cause health and educational differences (option 1), Reich uses his best, though flimsy, case to call upon us to empathize with a consensual attitude in his community that it is reasonable for them to consider the 'possibility' (rightly put by a black anthropologist colleague, Joseph Jones) of his genetically superior intelligence. And thus he exhibits a white incapacity to empathize with the other's *certainty* of complete humanity. They are not likely to be interested in his stupid question. They already know the evident problems around them which Reich's community belligerently refuses to acknowledge by leveraging the biodeterministic assumptions of their search for causes externalized from the history of their own greed. But then, given his frame of reference to white conversations, he is not speaking to 'the other' at all, is he? The Harvard Faculty Club, perhaps? His race-adherent colleagues at the Smithsonian Institution, per chance?[4]

As Reich (2018a) reaches from these premises, nonetheless, to a racial basis for differences in intelligence, it becomes clear that he believes he would find there exactly what he is looking for:

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

> Is performance on an intelligence test or the number of years of school a person attends shaped by the way a person is brought up? Of course. But [here it comes] does it measure *something* having to do with some aspect of behaviour or cognition? Almost certainly. And since all traits influenced by genetics are expected to differ across populations (because the frequencies of genetic variations are rarely exactly the same across populations), the genetic influences on behaviour will differ across populations, too.

This sounds quite reasonable, except that the first (almost certain) assumption has failed every test for a century and a half, from brain size, to brain complexity, to Spearman's *g*. Gould (1996) gives adequate discussion of the necessary ambiguity of what we call 'intelligence' in his critique. Only by belief against the empirical evidence (as Firmin warned the Gobinists) does this immaterial '*something* . . . [a]lmost certainly' exist.

Reich points us to two recent studies in his first op-ed (Kong *et al.* 2017; Okbay, Beauchamp and Benjamin 2016, 540) which, ironically, reveal the power of imagination on the cutting edge of the molecular genetics of intelligence. Okbay and associates found positive correlations between, on the one hand, SNPs physically located near to others associated with aspects of neural development and, on the other, years of educational attainment, significantly reduced rates of Alzheimer's disease, slightly increased schizophrenia rates, significantly higher head volume, size of brain regions, significantly smaller body mass index and slightly greater standing height. Kong *et al.*'s (2017) study also showed lower tobacco smoking. Consider that, with the exception of schizophrenia rates, every factor is correlated with high income, including a lean body build (low BMI), higher stature and the larger head associated with it (we have seen this correlation proven unrelated to intelligence before as failed craniometric evidence of biological differences in racial intelligence).

Kong's researchers importantly discovered that these correlations in a large Icelandic database are prominently due to another factor I associate with high socio-economic status: later birth of first child and lower total births than those with these qualities (Kong *et al.* 2017). These socio-economic correlations will inform my debate with Reich. Reich (2018a), however, took comfort in the fact that these collateral differences in the presumed effects of so-called EDU-genes 'showed that these [74] genetic variations also nudge people who carry them to delay having children. So these variations may be explaining longer times at school by affecting a behaviour that has nothing to do with intelligence'.

How far-fetched is that? A presumptive connection between age of bearing a child and *nudging genes* should immediately raise a red flag, because of the idea that behaviours as flexible and tied to social ecologies as marital age would not plausibly be caused by specific genes. Later marriage, producing lower birth rates, is associated with urbanism, industrialism, elevated class and economic crisis in many societies where earlier marriages and higher birth rates had been common in these same people's rural agrarian or low-income pasts (dare I say, my patrilineal great-grandparents had 12 children, my grandparents had two to four, and I have one). Indeed, what Kong and his colleagues had identified was a small group of SNPs in an extensive new Icelandic database which *correlated* (remember this) with individuals who tended to have later and fewer births as well as more years of education than others in Iceland's population. Why would they think that these genes represented a natural phenomenon of intelligence rather than a social-historical corollary?

The first assumptive perspective is that Iceland is imagined to be a genetically homogeneous population (although they looked for and found genetic variation in it) due to its racial conformity (Scandinavian) as a kind of surrogate for a socially neutral and universal standard for people. They could not be whiter.

Iceland also has socio-economic diversity owing to its feudal, colonial and capitalistic social and economic history (Magnusson 2010). Like the Americas, Iceland was populated with inequalities.

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

A limited group of Danish families were deeded land, as were the English gentry of Virginia (Isenberg 2016), establishing themselves as the wealthy. As fishing and other industries developed and participated in Atlantic and global economies, these and other specific families accrued capital. As relative elites, these families, like all elite families, would have been relatively endogamous, marrying amongst themselves as a consolidation of wealth. They may even have derived from long-standing elite families in Denmark deriving disproportionately from a common region of the country and its genomes.

Given that most genetic variation is between *families* (Lewontin's essential point in the 1972 article that Reich acknowledges), some genes, however inconsequential in themselves, are not only likely, but sure, to exist in greater quantity among the families of the Icelandic elite than among others. Familial variation is, therefore, far more likely than racial differences to be 'no longer possible to ignore', but he does so. Like all elites, they will marry later and have fewer children than the average Icelandic worker. They will have been afforded the most years of education, and will therefore tend to be better nourished, taller and with above average head size; less obese; and with lower rates of Alzheimer's disease. I suspect that these genes, deceptively named EDU genes, are simply a vestigial signature of some of the wealthiest families in Iceland – correlated with wealth, of course, but with no observed causal relationship with any social behaviour. As Franz Boas remarked in 'Changes in bodily form of descendants of immigrants' (1911) a hundred years ago, the biodeterminists must 'prove' their story against mine, or it is left as a 'just-so' story, compelling only to believers.

Indeed, Kong's study recognizes that their conclusions have wandered into territory long troubling to the eugenics movement: the wealthy with the highest educations and IQ scores marry and bear children late and thus have fewer offspring than the masses. If their genes determine intelligence, IQ scores should be in rapid long-term decline. After laying out the detailed assumptions of their models of selection which attach IQ scores to the EDU genes of late- and low-reproducing parents, Kong *et al.* write,

> by extrapolation, the decline of [these genes] would lead to a decline of 0.038 x (30/3.74) = 0.30 IQ points per decade. This would be a very substantial effect if the trend persists for centuries. By contrast, a meta-analysis estimated that IQ scores have increased by 13.8 points between 1932–1978, a rate of 3.0 points per decade ... This rate is 10 times the estimated effect due to the decline of the genetic component, and, more importantly, in the opposite direction. Many commenters ... consider the [Flynn] effect to be due to changes in the socioeconomic and technological environment faced by successive generations of humans ... Assuming that a similar magnitude of the Flynn effect [of increased resource equity] is found in the Icelandic population, it is clear that such environmentally induced increases of IQ scores more than compensate for, and indeed mask, any potential decline in the genetic propensity for IQ (Kong *et al.* 2017, E730).

Genes are shown, again, to be irrelevant to human variation in intelligence, reified as IQ. What a dark comedy: the moment they finally thought they had closed in on 'exactly what they were looking for', it dissipates like dust (yet again). Reich acknowledges this negative 'selection', seemingly more alarmed by the danger of not tracking the decline of so-called EDU genes than by the danger to his biodeterministic theory in what Kong *et al.* reveal as the meaninglessness of any such genes in society.

Conclusion

Repeatedly barking up the wrong tree must work for those who refuse to acknowledge and make corrections for option 1. Racism is the use of biology to deny whites' moral responsibility for taking. Biodeterminism is its active ingredient, and one that works for classism and

https://doi.org/10.1017/S1380203820000021 Published online by Cambridge University Press

sexism nearly as well. The scientific imagination has been institutionalized to validate such moral cover with neutral-sounding nature narratives. As archaeologists, or as scientists and humanists more generally, we must think out of the box of the Enlightenment's colonial assumptions (Blakey 1998), requiring careful study of the political histories of our fields and the theories we borrow and use. Rarely part of standard curricula, for what may be obvious reasons, I have found the problem of white supremacy to be pervasive in the arts and sciences. Only with a sophisticated understanding can one rationally challenge racist colleagues and replace their imaginations with self-critical and progressive inquiry on the field of evidence.

> Many a sincere person will answer me: 'Our attitude toward Negroes is the result of unfavorable experiences which we have had by living side by side with Negroes in this country. They are not our equals in intelligence, sense of responsibility, reliability.' I am firmly convinced that whoever believes this suffers from a fatal misconception. Your ancestors dragged these black people from their homes by force; and in the white man's quest for wealth and an easy life they have been ruthlessly suppressed and exploited, degraded into slavery. The modern prejudice against Negroes is the result of the desire to maintain this unworthy condition … (Albert Einstein, 'The negro question', *Pageant*, 1946, in Jerome and Taylor 2005, 86–87).

Notes

1 Lumsden and Wilson used the calculus to demonstrate their best case of an epigenetic primordial (essentially racial) basis for food preferences, a few years before today's explosive cosmopolitan culinary choices would make this laughable.

2 Olaudah Equiano's (1998) first accurate accounting of African societies swept into the American slave trade emphasizes the people's fastidious cleanliness, pointing to the source of southerner's descriptions of them as 'filthy' (Kiple and King 1981) as derivative of their imagination, the conditions they imposed on Africans, or both.

3 Single-word headings are taken from Carolus Linnaeus's first scientific descriptions of different races in *Systema naturae* (1758), in which he writes Eurocentric stereotypes, convenient to moralizing their exploitative interactions with others, onto nature.

4 The Smithsonian's Douglas Owsley led the racial interpretation of Kennewick Man (the Ancient One) as Caucasoid or Eurasian (Owsley and Jantz 2014) in order to have his way around NAGPRA law to its analysis. It would take 20 years for Danish geneticists to prove what Native Americans (and any working from evolutionary rather than racial taxonomic assumptions) already knew: the Ancient One is most closely related to Native Americans (Rasmussen *et al.* 2015).

Archaeological Dialogues (2020), **27**, 16–19
doi:10.1017/S1380203820000033

Imagined biodeterminism?

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Michael Blakey's paper is an important reminder of the egregious paths down which the 'biodeterministic imagination' can lead. This kind of biologically deterministic thinking is a major concern amongst archaeologists digesting recent studies of archaeogenetics (Booth 2019; Frieman and Hofmann 2019; Hakenbeck 2019). Sudden progress in methods of sampling, sequencing and analysis of DNA extracted from ancient human remains has meant there has been a recent glut of papers which use ancient human DNA to investigate past population histories. Some of these articles find evidence for major population movements associated with changes to