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REVIEW

Obsolete Science and Egalitarian Meta-political Activism in Contemporary Gene-Culture Coevolution: A Response to Lala and Feldman (2024)

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Abstract

We counter two broad claims offered by Lala and Feldman (L&F) in support of what they call *Contemporary Gene-Culture Coevolution* (CGCC): (1) *Cultural transmission pathways confound and inflate heritability estimates for a range of traits*: This is tackled via a meta-analysis of behavior-genetic studies that estimate vertical cultural transmission effects on trait variance for a wide variety of phenotypes. We find that this pathway plays only a very small role (relative to genetic effects) in conditioning phenotypic variance. (2) *GCC only works narrowly on functionally well-circumscribed genes; in the case of complex polygenic traits drift mechanics prevent the emergence of population differences as there is an extremely low probability of coherence involving drift direction across many genes favoring (in terms of associated trait means) one population over another; purely cultural forms of evolution are therefore sufficient to account for cross-cultural variability in the means of certain traits*: We conduct a cross-cultural sociogenetic analysis of the causes of variation in life history characteristics and find indications that culture-gene coevolution (but not purely cultural evolution) best describes the distribution of these traits. Drift is also ruled out as a cause of the underlying genetic variegation among our units of analysis. Multiple other contrary lines of evidence are also reviewed in light of the *biocultural dynamics* research program, which we posit as an alternative to CGCC. We also highlight meta-political activism latent in L&F's selection and interpretation of evidence, concluding that this risks anti-scientific censorship and fails to address the political issues that concern them.

Keywords Contemporary gene-culture coevolution · Biocultural dynamics · Meta-political activism

Lala and Feldman (2024)

In a Perspective published in the *Proceedings of the National Academy of Sciences,* Kevin Lala[1] and Marcus Feldman (Lala & Feldman, 2024) (henceforth L&F), two giants of 20th (and 21 st) century evolutionary biology, attempt to defend "quantitative studies of cultural evolution [CE] and gene-culture coevolution [GCC]" (L&F, p. 1) from accusations of "racism" and "reductionist and determinist claims" (L&F, p.1). Their particular formulation of CE and GCC, which they term *contemporary gene-culture coevolution* (CGCC), therefore, when properly understood, acts as a bulwark against, and antidote to, research into "'race and intelligence quotient (IQ)' and 'human sociobiology'" (L&F, p.1). These scientific misunderstandings are problematic insofar as they are alleged to have facilitated the spread of what L&F claim are racist and other potentially socially harmful views (such as eugenicism).

In this response we argue that CGCC is radically out of step with recent germane scientific developments, and, insofar as it makes testable predictions, can be easily falsified. We make the case that the preponderance of contemporary developments in the fields of interest to L&F support what we have termed the *biocultural dynamics* research program (Woodley of Menie et al., 2023), which is a broadly synthetic approach to understanding the distal causes of historical and

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[1] To avoid confusion, Lala and Laland are the same person, and citations will reflect whichever variant of the name was in use at the time (Laland, 2020, p. 654).

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cultural variation in traits, assimilating insights from cliodynamics, social biogeography, and archaeogenetics, as well as information from "more traditional" GCC and CE models. To that end, we rebut via review of pertinent literature and with novel empirical analyses, two core claims from L&F in defence of their position. In so doing we demonstrate that these (and various other) findings are consistent with predictions stemming from biocultural dynamics models. A case is then made that L&F's arguments at their core constitute a form of egalitarian meta-political[2] activism, specifically designed to foreclose both intentionally and unintentionally politically problematic applications of GCC to socially sensitive questions.

In the discussion, additional recent and significant theoretical developments that are highly germane to CE and GCC and which go uncommented upon by L&F are described. The article concludes by noting that L&F's closing plea for more scientific research into CGCC is incompatible with their meta-political objectives, which necessarily entail scientific censorship. This in turn, we argue, cannot mitigate the harms caused by public misunderstandings of complex biosocial phenomena, such as race.

Contemporary Gene-Culture Coevolution

L&F's paper is divided into eight sections. Most of these deal with what they believe to be misunderstandings of the nature of social race and its correlates and of heritability. The theoretical and empirical bases of CGCC are not really explored in detail until the section entitled *Heritability and Human Inheritance*. Much of the preamble involves characterization of the opposing allegedly reductionist and determinist position, and its perceived flaws. L&F return to discussion of their model in the final section entitled *The Richer Explanation*, but only after further digressions into the purported erroneous ideas surrounding issues of, e.g., race differences in disease risk and IQ and educational attainment.

Core Claim 1

CGCC appears to be premised on two core empirical claims, the first of which is explicated on page 5, wherein L&F argue that vertically transmitted forms of environmentality can significantly confound genetic estimates of trait heritability—specifically they note that:

> Cavalli-Sforza and Feldman... first established that vertical (i.e., parent-to-offspring) cultural transmission can inflate genetic heritability estimates for complex traits.... Further analyses established that—whether the heritability is estimated from parent–offspring or twin correlations, or on a liability scale, in the presence of vertical cultural transmission—the true heritability (i.e., that based on genetic variation) may depart substantially from such estimates... When cultural inheritance is incorporated into statistical models for the determination of phenotypes, and the models are fitted to human familial datasets, sharply lower estimates of the genetic contribution to these phenotypes are obtained. (p.5).

According to L&F, cultural inheritance is itself just one of a broader, complex set of inherited environmental or extragenetic influences that can co-contribute (along with genes) to phenotypic variance between individuals and (both racialized and non-racialized) population groups, with there being (at least) three distinct additional extragenetic variance components in play. These include: (1) *epigenetic inheritance,* meaning patterns of transmitted molecular markers that can be acquired in response to certain environmental exposures, and which modulate patterns of gene expression; (2) *somatic inheritance,* for example, symbiotic gut biota assemblages passing from mother to infant; and (3) *ecological inheritance,* meaning those aspects of the physical environment that persist across generations and can either beneficially or adversely affect development, such as in the case of pollution.

Core Claim 2

The second core claim concerns the limits of GCC and other forms of selection in shaping cultural variation among human populations. CGCC assumes that processes such as niche construction, encompassing the ways in which members of a species manipulate the parameters of their niche, can facilitate purely cultural adaptation, in addition to affecting the distribution of alleles among populations via their effects upon patterns of selective pressures acting on specific traits given sufficient time and opportunity. L&F give genes associated with lactase persistence and parallel cultural adaptations as examples of this, noting that:

> The spread of lactase persistence was shown to depend on the accuracy of cultural transmission... However, cultural adaptation to the absence of lactase persistence may also occur, such as the use of milk fermentation by Central Asian herders..., illustrating the variety of outcomes possible under GCC. (p.2)

L&F observe that many human genes show signals of having undergone recent selection, which they interpret as evidence of widespread GCC. Despite this, CGCC is

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[2] Meta-politics denotes "the ultimate founding ideas, myths, and values behind all concrete forms of political practice" (Bosteels, 2010, p. 880). On this basis, we claim that L&F are engaging in *meta-political activism* as they are imbuing certain scientific arguments with moral significance (both positive and negative, depending on the specific claim) in a conscious effort to shape political discourse.

incompatible with certain other forms of GCC that have been theorized in the past, such as Lumsden and Wilson's (1981) model of culture-gene amplification, where relatively small genetic differences among populations can potentiate cultural differences via positive frequency-dependent copying. CGCC is also incompatible with models of genetically biased cultural transmission, in which the probability of cultural transmission is influenced by genetic differences that either increase or decrease the ability of a population to acquire certain cultural patterns. Former *New York Times* journalist Nicholas Wade's (2014) book *Troublesome Inheritance* is given as an example of a work advancing these problematic forms of GCC. These supposedly faulty theories are challenged by noting that:

> More recent research has established that human decisions concerning which cultural variants are adopted are not tightly constrained by genes but may change and diversify rapidly, according to a variety of well-documented general learning heuristics, including copying successful or prestigious individuals, or conforming to the majority view... Cultural transmission allows individuals to adjust to relatively quickly changing aspects of their environments, which would be impossible under tight genetic constraint... and leaves Lumsden and Wilson's primary conclusions untenable... A major conclusion of contemporary GCC is that the causal interactions between genes and culture are bidirectional, and that the properties of culture do not reduce to genetic causes. (p.3)

Even though "many genes" (L&F, p.7) in the human genome have apparently been subjected to recent selection, possibly facilitated by widespread GCC, CGCC seemingly works in a narrow-spectrum capacity, acting only on genes exhibiting well-circumscribed functional associations (such as those related to lactase persistence). This fact presumably makes GCC theorizing in these cases feasible, as the straightforwardness of their phenotypic associations de-burdens, by virtue of reducing the number of assumptions, hypotheses invoking GCC as a causal explanation. On the other hand, GCC explanations for population disparities in levels of complex polygenic traits, such as those connected to learning, cognition, and behavior, are allegedly implausible owing to the purportedly predominant role played by drift in genetically structuring such traits. In relation to this, L&F specifically note that:

> [I]f, as the data suggest, intelligence is affected by many genes of small effect, it becomes implausible that IQ differences between socially defined races arose through a process of random genetic drift; this is relevant because analyses of genetic variation show that recent human evolution has been dominated by drift rather than selection... The probability that a long sequence of random changes would all go in the same direction, leading to increases in the intelligence of one population and not others, approaches zero (p.7).

CGCC attempts primarily to explain the association between functionally well-circumscribed genes and the distribution of cultural traits. At the core of this model is the assertion that there are highly promiscuous interactions between different variance components of the inheritance system, many of which are extragenetic in character and irreducible with respect to genetic causation (Claim 1). Thus, models of genetically biased cultural transmission and gene-culture amplification, both of which posit constraints on cultural affinity and flexibility, are allegedly excessively genetically reductionist and genetically determinist, and are at odds with the putative strongly interactionist character of inheritance in GCC. Complex polygenic traits, comprised of large numbers of variants with small effects, owe their genetic architecture to drift dynamics, which due to the large numbers of involved genetic variants and the stochastic and therefore uncorrelated nature of frequency shifts in these between populations, should not favor one population over another in terms of broad genetic propensity toward higher or lower levels of, e.g., cognitive ability. The loose-knit association between genetic and cultural transmission (as per Claim 1) means therefore that cultural differences among populations are just that – cultural, and constrained by purely cultural forms of evolution (as per Claim 2). Directly related to this claim is the argument that social race in CGCC is distinct from population clustering, the former being arbitrary with respect to the latter. Nevertheless, it *can* be biologically actualized in the sense that extragenetic sources of inheritance *racialize* environments via inequitable patterns of niche construction (meaning the creation of persistent cultural transmission patterns that privilege one social racial group at the expense of another), which make the associations between social race and seemingly heritable traits, such as cognitive ability and health, irreducibly holistic through complex patterns of interactions involving different components of heredity. This core claim of CGCC stands in stark contrast to those latent within allegedly reductionist and determinist racial hereditarian models advanced by Arthur Jensen, William Shockley, and others.

Empirical Evidence Contra CGCC's Core Claims

Here we critically and empirically examine the two core claims latent in CGCC. The test of its second core claim is foregrounded with respect to a broad-based discussion of the major supporting lines of evidence associated with the biocultural dynamics research program.

A Meta-analytic Test of Claim 1

As noted, L&F argue strongly that vertically transmitted culture is a major factor confounding, specifically by inflating, the heritabilities of traits like general cognitive ability (GCA; also known as general intelligence or *g*). In evidencing this, they refer to the extensive past theoretical work of one of the coauthors (Feldman) and Luigi L. Cavalli-Sforza conducted on this topic in the 1970s and 80s (e.g., Cavalli-Sforza & Feldman, 1973, 1976, 1978, 1981; Feldman & Cavalli-Sforza, 1976, 1977), noting that:

> [Feldman and Cavalli-Sforza's] first models showed that parent-to-offspring cultural transmission could produce the appearance of high heritability even though the transmission was not genetic, disproving Jensen's claim that high heritability implied traits would be difficult to alter.... Later, they constructed dynamic models incorporating both genetic and cultural inheritance... and used a variety of methods that extended mathematical evolutionary theory... (p. 2).

On the basis of this L&F argue that:

> [G]ene-culture coevolutionary modeling reduces estimates of genetic heritability for IQ from standard values of 0.5 to 0.8 when vertical cultural transmission is ignored to 0.29 to 0.42 when it is incorporated; the estimates depend on how cultural transmission is modelled... (p. 5).

The source of L&F's 0.29 and 0.42 values is a 1982 paper by Rao and colleagues, which found, based on a crude path analytic model employing pooled correlations derived from different combinations of twins, siblings, parents, and adopted siblings, sourced from several different studies, that a so-called cultural inheritance factor accounted for 42.4% of the variance in IQ under the assumption of phenotypic homogamy, and accounted for 29.2% of the variance under a general model run under a mixed homogamy assumption instead, but only in the child sample in the case of the latter. The adult sample value was much lower in this instance (9.6% of the variance explained). Another older study, that of Rice and colleagues (Rice et al., 1980) using a similar approach to estimate so-called cultural inheritance effects to that used subsequently by Rao and colleagues (Rao et al., 1982), found that their b^2 parameter, this corresponding to their cultural inheritance variance component from their best-fitting model, accounted for 28.9% of the variance in IQ.

The key problem with both Rao and colleagues' (Rao et al., 1982) and Rice and colleagues' (Rice et al., 1980) findings is that they *do not* estimate the subcomponent of shared environment that actually captures purported cultural inheritance. In the case of Rice and colleagues (Rice et al., 1980) this is made clear on page 77, where these researchers state that "[i]n the BETA model … T is partitioned as T = A + B, where A denotes additive genetic factors and B denotes heritable cultural factors."

The equation given in their publication just substitutes "heritable cultural factors" for the more traditional shared environmentality variance component (c^2). According to Boyd and Richerson (1985), cultural transmission pathways include: (1) *Vertical transmission, from parents to offspring*[3]; (2) oblique transmission, from nonparental adults to youngsters; and (3) horizontal transmission, from peers to peers.

Early researchers such as Rao and Rice instead seem to naively treat the shared environmental effect as though it were capturing *only* "cultural inheritance" pathways.

A body of much better powered and more methodologically sophisticated research into the specific influences of vertical transmission effects on various traits has been assembled in more recent years, with these studies overwhelmingly showing small to null effects of this variance component when it is isolated *properly*. This, needless to say, strongly challenges L&F's claims of potent vertical culture confounding effects in behavior-genetic measurement models of (e.g., IQ) heritability. To demonstrate this, we conduct the first meta-analysis of such studies in order to determine the precise magnitude of the vertical transmission pathway variance component. Another researcher has conducted a comprehensive qualitative review of this (not especially extensive[4]) literature, which currently spans 14 studies published between 1986 and 2020, all of which we use in the present meta-analysis. Supplemental Boolean searches using Google Scholar for *vertical transmission* or *vertical cultural* and various phenotypes coupled with backwards searches of the reference lists of other papers, netted us an additional four studies, two of which examined vertical cultural effects on smoking proclivity, one of which examined these in relation to antisocial personality disorder, and one that examined these in relation to educational attainment.

Effect sizes were obtained at both the level of the individual phenotypes and were also aggregated at the study level, in order to estimate funnel asymmetry and trim-and-fill (the latter estimates the number of missing effect sizes needed to make a funnel distribution symmetric). Both the Rice and colleagues (Rice et al., 1980) and Rao and colleagues (Rao

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[3] This being the pathway of interest in Rice et al. (1980) and Rao et al. (1982).

[4] This is in large part due to the demands of these studies, which require extended family relationships in addition to twins or adopted children to properly estimate large numbers of behavior-genetic variance components.

et al., 1982) studies were included in our meta-analysis, despite their flaws. This bought the study number to 19.

Meta-analysis Results

A total of 66 effects of (in some cases purported, but in most cases actual) vertical cultural transmission and their corresponding sample sizes were collected from the 19 studies. A multilevel meta-analysis was performed, with study ID as a random effect, using the *rma.mv* function found in the *metafor* package (Viechtbauer, 2015). The analysis revealed a significant albeit small effect of vertical cultural transmission ($zr = 0.154$, $se = 0.043$, $p = 0.0004$; $r = 0.152$, $R^2 = 0.023$) accompanied by high levels of heterogeneity ($Q = 12{,}740.773$, $p < 0.0001$). Small in this context corresponds to r values that range in magnitude from 0.100 to 0.290, as in the current case (Cohen, 1988).

A subsequent moderation analysis was performed with year, phenotypic category (contrast 1: cognitive vs. attitudinal/health vs. personality; contrast 2: attitudinal/health vs. personality), and inclusion/exclusion in the extended studies of spouses, siblings, or offspring. The last moderator set tests for the influence of method variance across studies on the stability of the estimates. The model revealed that year ($b = 0.004$, $p < 0.0001$) and the second contrast for phenotypic classification reached statistical significance ($b = 0.042$, $p < 0.0001$). In terms of the latter, this result indicated greater vertical cultural transmission effects for attitudinal/health traits than for personality traits. A secondary meta-analysis based on effect sizes aggregated into point-means per study supported this findings, as represented in Fig. 1, wherein the effect of vertical cultural transmission reached statistical significance but remained small in magnitude ($zr = 0.161$, $se = 0.039$, $p < 0.0001$; $r = 0.159$, $R^2 = 0.025$), accompanied by high heterogeneity levels ($Q = 2854.019$, $p < 0.0001$).

A regression test for funnel plot asymmetry was performed using the *regtest* function ($t = -0.906$, $p = 0.3768$). A trim-and-fill analysis suggested the absence of five studies on the right side of the funnel plot's center line, as indicated in Fig. 2. The recomputed effect for vertical cultural transmission slightly increased in magnitude, but remained small ($zr = 0.213$, $se = 0.038$, $p < 0.0001$; $r = 0.210$, $R^2 = 0.044$) and was also associated with high levels of heterogeneity ($Q = 4043.372$, $p < 0.0001$). Overall, these results indicate that vertical cultural transmission explains between 2.5 and 4.4% of the variance in human attitudinal, cognitive, and personality traits. This effect is inflated by the inclusion of Rice and colleagues (Rice et al., 1980) and Rao and colleagues (Rao et al., 1982), which are clear visual outliers. Consistent with this, the removal of these two studies reduces the aggregate effect size ($zr = 0.115$, $se = 0.025$, $p < 0.0001$; $r = 0.115$, $R^2 = 0.013$). Rerunning the trim-and-fill test indicated no *missing* studies after removing these outliers, these results are featured in Fig. 3.

Meta-analysis Conclusions

Contrary to the claims of L&F, vertical cultural effects account for very little variance across a range of different traits when estimated properly. Only in the case of the contrast involving social attitudes and certain health traits

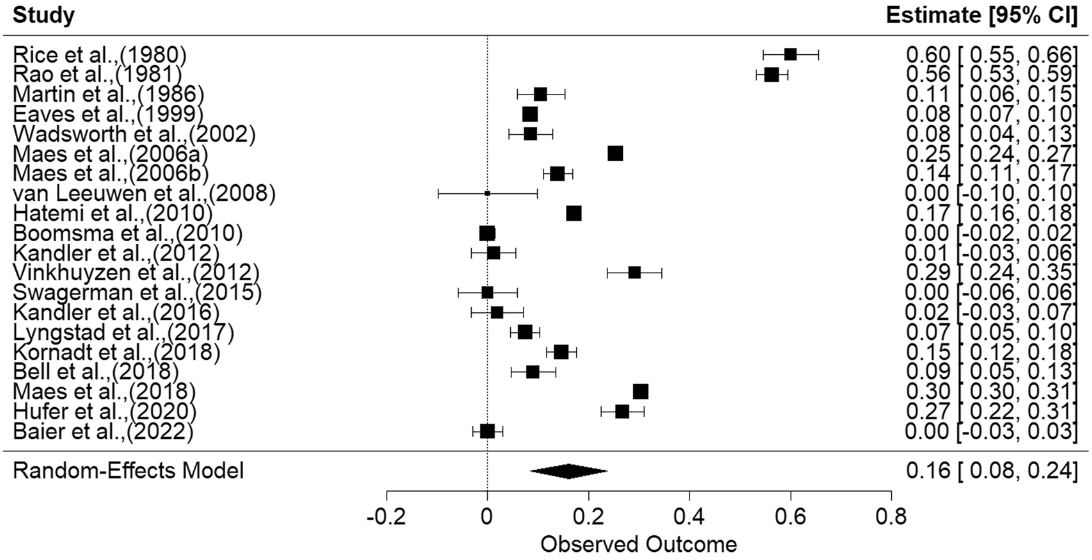

Fig. 1 Forest plot of vertical cultural transmission aggregated study-level effects. Citations are included in the references

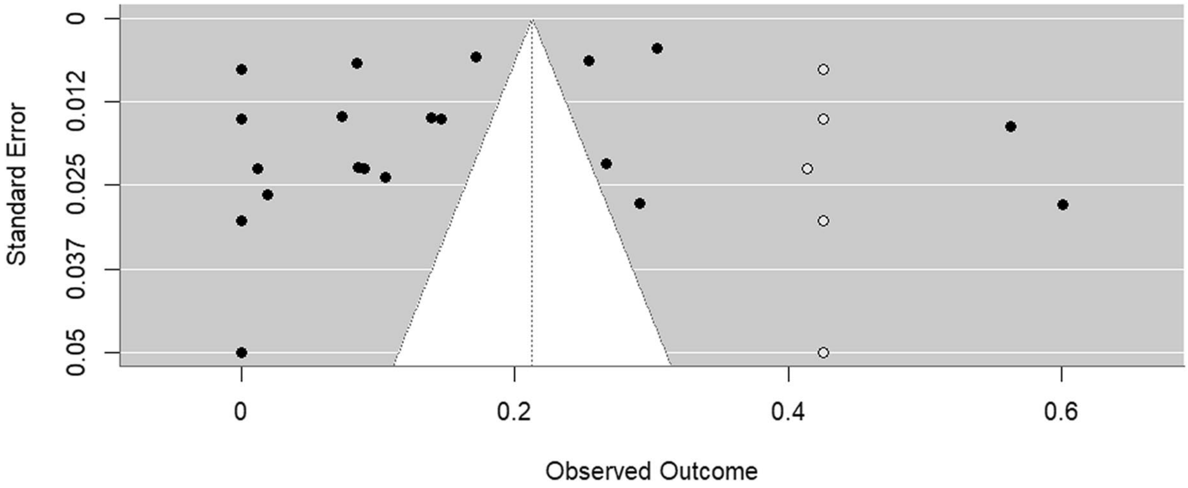

Fig. 2 Funnel plot of vertical cultural transmission aggregated effects. Trim-and-fill imputed *missing* data points are unfilled circles

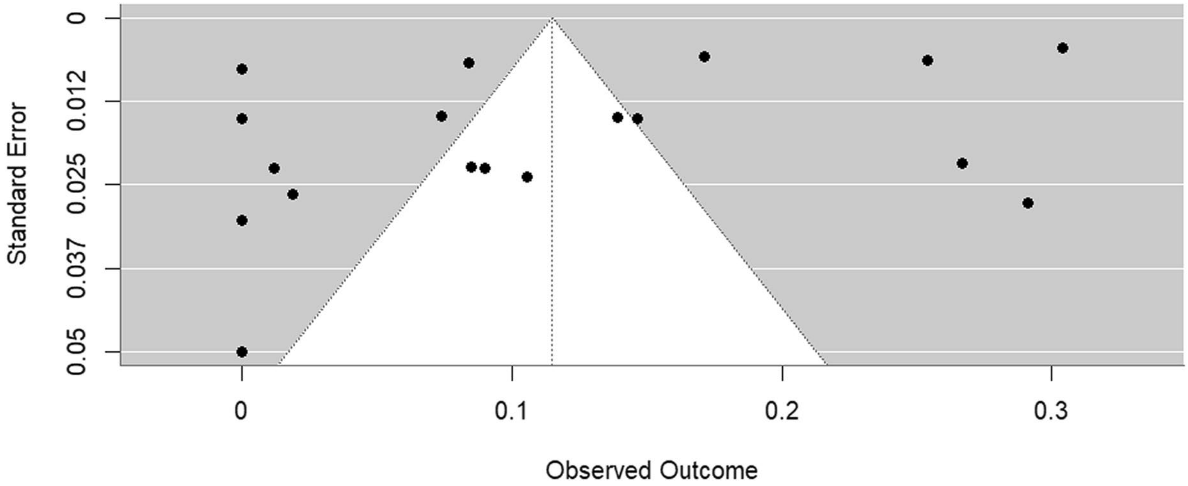

Fig. 3 Funnel plot of aggregated vertical cultural transmission effects after removing Rice and colleagues (Rice et al., 1980) and Rao and colleagues (Rao et al., 1982). Trim-and-fill did not suggest any missing studies

do these vertical effects seem to have a significantly larger impact relative to personality traits, a finding that is unsurprising given the generally low heritability of social attitudes in particular. Rice and colleagues' (Rice et al., 1980) and Rao and colleagues' (Rao et al., 1982) seemingly larger estimates of the influence of *cultural inheritance* on IQ (.289 and.260 respectively) do not replicate in studies employing better methods and common (as opposed to pooled) samples. For example, van Leeuwen and colleagues (van Leeuwen et al., 2008) found that 100% of the resemblance between parents and their offspring in IQ could be accounted for via shared genetic factors, leaving no room for cultural inheritance. Vinkhuyzen and colleagues (Vinkhuyzen et al., 2012) find that vertical cultural transmission accounts for a mere 8% of the variance in IQ, but this was non-significant given their sample size. These newer findings therefore fail to support claims of widespread confounding of heritability estimates for IQ with cultural inheritance, as argued by L&F. The existence of five "missing" studies was implied by trim-and-fill to the right of the center line, suggesting that studies reporting larger cultural transmission/inheritance effects are underrepresented among these data. However, the removal of the Rice and colleagues (Rice et al., 1980) and Rao and colleagues (Rao et al., 1982) data points reduced this estimate to zero. Finally, there appear to be signs of a reverse *decline effect* in this literature, with more recently published effect sizes being slightly larger relative to older ones. This could possibly implicate a secular trend of some sort increasing the potency of this transmission pathway over time, at least for certain traits.

It is important to stress that the relative magnitudes of behavior-genetic variance components estimated using individual-level data are not necessarily informative about the role that these sources of differentiation may play in conditioning social racial or other population differences. Given the very wide range of ancestral and recent historical environmental factors to which different groups have been exposed, which would include slavery in the case of African Americans, it is possible that cultural, or maybe more specifically epigenetic, inheritance effects could be greatly amplified as a source of persistent differences at these higher levels of biosocial aggregation. Models that posit a role for factors such as vertical cultural and epigenetic confounding in global admixture analyses seem to us therefore to be more plausible (e.g., Schraiber & Edge, 2024, pp. 5–6). As to the possibility that purported cultural inheritance effects may function as a *major* confounding factor in conventional biometric estimates of heritability, the data from modern methodologically sophisticated and high-powered research simply do not support anything more than a very marginal role for these effects in the case of most phenotypes.

Moreover, these results are consistent with Richerson and Boyd's (2004) review of the literature on vertical cultural transmission, who note:

> Results from several independent studies suggest that cultural transmission within the family is not very important; the similarity between parents and offspring is mainly due to genes. If these results stand up and generalize to other sorts of characters, then it would tell us that parents are less important in cultural transmission than many people suppose. (p. 36)

Evidence from Biocultural Dynamics

The theoretical and empirical framework of biocultural dynamics integrates several levels of ecological and evolutionary principles when examining human variation between populations (Figueredo et al.,2017; Woodley of Menie et al., 2023). Such an approach integrates a taxonomy of ecological dimensions comprised of: (1) Physical ecology, capturing variation in altitude, latitude, precipitation, and temperature; (2) Community ecology, capturing heterospecific interactions such as mutualism, commensalism, amensalism, competition, parasitism, and predation; (3) Social ecology, capturing the emergence and persistence of interactions and relationships among conspecifics—in human populations it includes dynamics such as competition and cooperation across levels of sociopolitical aggregation; current theoretical proposals further disaggregate this ecology by incorporating Bronfenbrenner's bioecological model, starting with the *microsystem* that includes individuals (ego) alongside their immediate and extended kin, the mesosystem composed of interactions and relationships among local communities, and the macrosystem corresponding to infra-national and cross-national variation (Hertler et al., 2018); (4) Cultural ecology, capturing differences in cultural variants and institutions above and beyond the social interactions found at the level of social ecology; whereas cooperation within the microsystem and mesosystem is predominantly sustained via kin selection/inclusive fitness payoffs, indirect reciprocity, and reciprocal altruism, cooperation within sizable communities depends on strong reciprocity and ultrasocial institutions that prescribe cooperation between genetically distantly related individuals and proscribe social parasitism; and (5) Cognitive ecology, capturing variation in cognitive capital between and within populations.

The methods of biocultural dynamics can be applied both *diachronically,* meaning longitudinally over historical time, and *synchronically,* meaning cross-sectionally at one historical period. Diachronically, Woodley of Menie and colleagues (Woodley of Menie et al., 2017), building on earlier work (Woodley &Figueredo, 2013), applied a variety of novel quantitative methods, including longitudinal multilevel modeling, to trace the recent evolution of different components of human cognitive ability over the past few centuries, using various different temporal intervals of up to 400 years and applying a mix of psychometric, anthropometric, and lexicographic methods. They found that selection favoring the development of various environmentally specialized abilities was seemingly promoted by a regime of greater individual selection as compared to group selection, whereas selection favoring heritable general intelligence was seemingly promoted by a regime of greater group selection as compared to individual selection. Figueredo and colleagues (Figueredo et al., 2019a, 2019b) afterwards applied similar methods to the study of both differentiation and integration of biodemographic slow life history traits, meaning the systematic weakening or strengthening of correlations among the different indicators of life history strategy, in the populations of both Gallic and Britannic Empires over the past two centuries. Applying the relevant theories from quantitative theoretical ecology, these studies demonstrated that the restriction of niche breadth in the Gallic biocultural group led to the progressive *integration* of life history traits whereas the expansion of niche breadth in the Britannic biocultural group led to the progressive *differentiation* of life history traits over much of the Late Modern Era, presumably to adapt to more biogeographically varied environments.

Synchronically, Figueredo and colleagues (Figueredo 2020b, 2021) conducted cross-cultural analyses using a sample of national polities for which the majority of the populations were indigenous, meaning that they had not experienced any massive invasions or colonizations from other biogeographical regions within recent history, using AD 1500 as the cutoff for this criterion. This sampling was done to restrict the selection of biocultural groups to those that could be reasonably expected to have become adapted to their current ecologies. Zoogeographic regions (Wallace, 1876; Holt et al., 2013) explained the preponderance of the cross-national variance in both biodemographically-assessed slow life history strategies (78.6%) and psychometrically-assessed (71.4%) GCA.

Another finding of note is that, although the outdated system of geographically defined races (with three major groupings of Mongoloid, Caucasoid, and Negroid) has often been used as to proxy biogeography of origin in some evolutionary writing, zoogeographical regions accounted for significantly (16.5%) more variance in biodemographic life history than this outmoded scheme of geographical racial classification. This finding indicates that the program of biocultural dynamics is not just a smokescreen and euphemism for scientific racism, but is a much more explanatory and better-grounded framework for evolutionary theory and research into the causes of human biocultural diversity.

Biocultural dynamics shares its goal of reaching a theoretical and empirical consilience between ecology and evolution with previous cross-national examinations. For example, Newson and Richerson (2009) argued that environmental hypotheses describe the influence of synchronic contemporary social and economic factors on human behaviors. For the authors, evolutionary hypotheses concern temporally distant causes of human change, such as environmental variables shaping patterns of natural selection. It follows that these distal environmental factors alter a population's genetic landscape, generating a lag between a generation's genes and their ancestral environmental conditions (Newson & Richerson, 2009). As described by Odling-Smee colleagues (Odling-Smee et al., 2003), inceptive cultural niche construction is capable of either modifying a community's selective pressures and leading to further cultural evolution (route 1) or, under circumstances where a cultural response is absent or weak, the culturally modified environment alters local selective pressures operating on gene frequencies (route 2). The authors further explain that:

> Route 2 applies whenever human cultural processes fail to express a sufficiently effective response (via route 1) to an environmental change that has previously been induced by cultural niche construction. In such cases, and to the extent that cultural processes cease to buffer culturally induced environmental changes, the latter are likely to give rise to culturally modified natural selection pressures. There may then be changes in allelic frequencies in human populations. (p.339)

The following subsections provide a brief summary of several significant publications under the framework of biocultural dynamics.

Archaeogenetic Evidence for Selective Sweeps and Against Genetic Drift

A considerable amount of compelling evidence in the form of archaeogenetic and cross-population data on the deep-historical trajectories of variants associated with GCA, in addition to a variety of behavioral and anthropometric traits, has now been published. Collectively the research work indicates that the relevant genetic variants are overwhelmingly selectively non-neutral, and have undergone significant changes in frequencies over time. In some cases, neutral models have been directly tested and falsified.

The earliest study to investigate archaeogenetic patterns in relation to genetic variants associated with educational attainment (EA) and GCA is that of Woodley of Menie, Yonuskunju, Balan, and Piffer (Woodley et al., 2017). This study estimated ascertainment-bias corrected polygenic scores (PGSs) comprised of variants that had been identified in previous genome wide association studies (GWASs) as predictors of both EA and GCA, using the genomes of 99 Eurasian individuals who lived between 4.56 and 1.21 Kyr before present, specifically before AD 1950. These were compared with a sample 503 ancestrally matched genomes sourced from the 1000 Genomes project. Significant and positive mean differences were noted when the modern genomes were compared to the ancient ones in terms of the levels of three different polygenic scores, using both conditional and unconditional tests (Fisher's exact test and the G-test). These PGSs also outperformed pseudo-PGSs drawn from 6740 minor-allele frequency (MAF) matched and non-overlapping variants using a Monte Carlo model. It was found that:

> … the POLYCOG outperformed the random polygenic scores (producing lower OR values) in the majority of instances (9 SNPs = 76.4 % [572 out of 749 draws]; 130 SNPs = 84.6% [44 out of 52 draws]; 11 SNPs = 77.8% [477 out of 613 draws]) (p. 275).

This result is consistent with the action of a soft selection sweep as the increase occurs in sets of alleles that are functionally linked to a selectively relevant outcome, not in randomly selected, but MAF-matched, alleles. Among a subsample of 66 of the ancient genomes, there was also a statistically significant positive correlation between PGS levels and sample recency.

These findings have now been extensively replicated. Kuijpers and colleagues (Kuijpers et al., 2022) generated a suite of PGSs for heritable traits, which included BMI, lipoprotein concentrations, cardiovascular disease, and GCA (among other traits) using a sample of 872 ancient European genomes, the earliest of which were over 25,000 years old, covering the Upper Paleolithic, with the most recent being from the post-Neolithic era, ranging from 5000 BC to more recent times. These ancient European samples were combined with a sample of 250 modern Western European genomes sourced from the 1000 Genomes database.

The authors note that "[p]rior to the Neolithic revolution no significant changes in [PGS] are observed; however, after the start of the Neolithic period a significant increase in coronary artery disease [PGS] can be seen as well as a decrease in HDL cholesterol [PGS]" (Kuijpers et al., 2022, p. 5). In contrast to these "[s]ignificant decreases in [PGS] can be seen prior to the Neolithic revolution for all cognition-related traits except fluid intelligence, followed by significant increases in [PGS] over time. This pattern for cognition-related traits is reversed for unipolar depression" (Kuijpers et al., 2022, p. 6).

To test for a selection signal in these trends, mean Fst scores were estimated for each trait in order to determine whether they differed relative to the distributions of 10,000 LD and MAF matched mean Fst scores, a method very similar to the one developed by Woodley of Menie and colleagues (Woodley of Menie et al., 2017). It was found that:

> … traits like educational attainment in years, intelligence, BMI, HDL, LDL, and skin tanning ease *show significant selective pressure* between pre-Neolithic and Neolithic samples. During the Neolithic to post-Neolithic period, HDL *still shows strong selective pressure* in contract to the other metabolic traits. Lastly, between the post-Neolithic and modern samples, BMI, educational attainment in years, fluid intelligence, skin color, skin tanning ease, and standing height *show significant levels of selection* (p. 7, italics added for emphasis).

In an even more recent study, Piffer and Kirkegaard (2024a) employed an even larger sample of ancient and Holocene genomes (2,625) in order to expand on the findings of Kuijpers and colleagues (Kuijpers et al., 2022), in terms of trait coverage, quality of imputation, and spatiotemporal resolution. They excluded the medical phenotypes employed by Kuijpers and colleagues (Kuijpers et al., 2022), and instead incorporated PGSs for mental-health-salient traits, including autistic-like personality, neuroticism, and schizophrenia. In addition to these, PGSs for anthropometric traits such as height and intracranial volume were estimated along with two PGSs for EA, one for GCA, and one for occupational status. These authors note in relation to their cognitive PGSs that:

> [W]e identified the most substantial discrepancies across phenotypes between the Upper Paleolithic and Neolithic periods, likely attributable to the profound cultural and lifestyle shifts occurring between these two epochs [with] the Neolithic Revolution, marking the transition from hunter-gatherer ways of life to agriculture-centered settlements, initiat[ing] a surge in population density, escalated social complexity, and necessitate[ing] enhanced planning and organization. Consequently, cognition underwent selection to navigate the intricacies of labor division, specialization, and the hurdles of social competition and collaboration. A significant 'leap' in the PGSs for EA and IQ was also observed between the Bronze Age and the Iron Age, hinting at a period of selection favoring sophisticated cognitive abilities (p. 43).

PGSs for behavioral phenotypes showed mixed temporal trends, with declines noted in the case of neuroticism and schizophrenia, possibly consistent with the action of a selection sweep promoting cognitive variants, as negative associations between GCA and these phenotypes have been extensively documented in clinical data. Positive associations have also been noted between autistic-like personality and GCA, which may account for the convergent positive temporal trajectory associated with the PGS for the former. The temporal trends for intracranial volume- and height-related PGSs were positive, but no temporal correlations were noted between the former and the cognitive-ability PGSs. Also "[c]ontrary to the cold winters theory, the study found no significant correlation between latitude and intelligence [PGSs]" (p. 30). This adds to the mounting evidence that cognitive ability has been under recent and relatively strong GCC pressures, which intensified with the Neolithic Revolution in response to significant increases in sedentarism, inter-group competition, standing population densities, and socio-cultural complexity (as theorized in Woodley of Menie et al., 2017). Consistent with this, Piffer & Kirkegaard (2024a) note that the last 10,000 years saw an approximately 1.2 sigma increase in the levels of the cognitive PGSs.

In another recent paper, Piffer & Kirkegaard (2024b) estimated height PGSs using both multi-population between- and within-family whole-GWAS data sourced from 51 populations. Joint effects of both genetic and environmental influences on height were identified, which potentially solves Deaton's (2007) African paradox—this being the contrast between greater stature and poorer-quality nutrition in African populations—as the polygenic propensity toward attaining greater stature is markedly increased in African populations relative to others. Critically, Piffer & Kirkegaard (2024b) were able to rule out drift as an explanation for the differences involving the first set of PGSs, noting that:

> … tests of divergent selection based on the QST (i.e., standardized measure of the genetic differentiation of a quantitative trait among populations) and FST (neutral marker loci) measures exceeded neutral expectations, reaching statistical significance ($p <.01$) with the MIX-Height PGS but not with the SIB-Height PGS. This

> result indicates potential selective pressures on body height-related genetic variants across populations (p. 265).

These studies demonstrate very clearly that, *contra* L&F, selection acting on traits with a complex genetic basis involves soft selective sweeps acting simultaneously on thousands of variants of small effect, drawing them into correlation with one another.

This survey of the literature is not intended to be comprehensive. Many more studies have been published examining selection sweeps using molecular data. The findings presented here do however serve to illustrate the fact that an impressive amount of archaeogenetic research has revealed strong indications of the action of cultural transitions in initiating, and moderating the intensity of, selection sweeps, especially those acting on cognitive, behavioral, and health-related genotypes.

Evidence from Neolithic and Bronze Age Migrations and Expansions

Narasimhan and colleagues (Narasimhan et al., 2019) suggested the presence of seven clines in Eurasia before and after the arrival of farming: (1) Western European Hunter-Gatherers; (2) Eastern European Hunter-Gatherers; (3) West Siberian Hunter-Gatherers; (4) East Siberian Hunter-Gatherers; (5) Anatolian Farmers; (6) Iranian Farmers; and (7) South Asian Hunter-Gatherers. Regarding the process of the spread of farmers into Central and Western Europe, the archaeological and archaeogenetic evidence suggests considerable regional variation in cultural and genetic admixture between Mesolithic Hunter-Gatherers and Neolithic farmers (Narasimhan et al., 2019). For example, Central European populations close to the Danube between 4000 and 3000 BC exhibit a preponderant Anatolian ancestry with a minor contribution of Western European Hunter-Gatherers (Narasimhan et al., 2019). In contrast, the proportion of Western European Hunter-Gatherers in Iberia in 3000 BC is considerably more extensive, although the percentage of Anatolian ancestry remains predominant (Narasimhan et al., 2019).

Lethal aggression was present within and among Neolithic communities (cf. Roksandic, 2004, for archaeological evidence recovered from Mesolithic sites). Keeley (1997) argued that the interactions between Neolithic Farmers and Foragers were not entirely peaceful or even tolerant: for example, he observed that Linear Pottery Culture frontier sites often featured ditches and palisades. Moreover, Keeley identified buffer zones between foragers and farmers. Eventually, the descendant communities of Mesolithic foragers in Central Europe were either displaced or assimilated by Neolithic biocultural groups. Consequently, the admixture profiles uncovered in Neolithic populations demonstrate not only a cultural shift but also a partial genetic replacement. Thus, subsequent archaeogenetic studies need to examine the presence of genetic signals evidencing differences in cultural complexity between Mesolithic foragers and Neolithic farmers.

A similar replacement occurred as part of the Chalcolithic and Bronze Age transition, a historical period characterized by migrations of pastoralist societies from the Pontic-Caspian Region, which further altered the cultural and genetic landscape of Eurasia. According to Heyd (2021), the extent of replacement varied as a function of the corresponding migration routes. For example, the expansion of the Yamnaya horizon, with the principal migration bulk traversing south of the Carpathians in close proximity to the Danube, occurred as a mild replacement (Heyd, 2021). In contrast, local societies inhabiting environments north of the Alps and the Carpathians experienced a primary cultural and genetic replacement by descendants of pastoralist societies such as the Corded Ware Horizon (Heyd, 2021). Previous studies provide further evidence of this replacement. Haak and collaborators (Haak et al., 2015) performed admixture analyses based on the proportion of Early Neolithic, Wester European Hunter-Gatherer, and Yamnaya ancestral component scores. Consistent with previous archaeological publications, Starcevo, Linear Pottery, Gamba, and Stuttgart samples evidenced a predominant Early Neolithic ancestry (Haak et al., 2015). In contrast, samples associated with Karsford and Corded Ware horizon displayed a sizable proportion of Yamnaya ancestry (Haak et al., 2015). Archaeological evidence also suggests that descendants of Neolithic farmers experienced intense competition with pastoralist societies. For example, Schroeder and colleagues (2019) suspected that Corded Ware raiders were responsible for killing several Globular Amphorae individuals. The repercussions of these and other biocultural replacements are evident in the genetic profiles of contemporary populations.

Dutton's Rule

Further evidence of genetic multilevel selection as reflected in historical migrations comes from the work of Cabeza de Baca and colleagues (Cabeza de Baca et al., 2020). A world-spanning review of molecular-genetic evidence was conducted to test the validity of *Dutton's Rule* in the dynamics of cross-ethnic hybridizations, which is the general principle that the higher-status group will contribute the preponderance of Y-chromosomal haplogroup DNA (which is inherited exclusively through the patriline) and the lower-status group will contribute the preponderance of the Mitochondrial haplogroup DNA (which is inherited exclusively through the matriline). This principle was upheld in all ten such historical cases for which molecular-genetic data were available: (1) Early Modern African slavery in

North American and Southwest Asia; (2) Neolithic Bantu expansion from West Africa across much of the rest of Sub-Saharan Africa; (3) Late Medieval Islamic Imperial expansion from the Arabian Peninsula across North Africa; (4) Neolithic Southwest Asian expansion into Europe; (5) Medieval Anglo-Saxon migration from Continental Europe into Britannia; (6) Hellenistic Era Ashkenazi and Sephardic migration from the Levant into Europe; (7) Bronze Age Indo-European migration from Central Asia into the Indian Subcontinent; (8) Bronze Age Indo-European migration from Central Asia into Iran; (9) Han Dynasty expansion from northern China into southern China; and (10) Medieval Mongolian Imperial expansion from East Asia into Central Asia. The global applicability of Dutton's Rule strongly indicates that well-documented historical migrations altered the genetic landscape of numerous human societies in a manner consistent with expectations logically derived from multilevel selection theory. There were no exceptions found to this principle in the societies reviewed.

An Empirical Test of Claim 2

Building on the foregoing, the following subsections present a novel social biogeographical analysis of the distal causes of cross-national variation in life history traits, showcasing the full range of theoretical and methodological perspectives available to those working within biocultural dynamics. In so doing, we conduct a direct test of claim 2 of CGCC.

Methods

Measures and Statistical Analysis

Country-level data on the genetic frequencies of 5-HTTLPR (a gene involved in the transport of serotonin with the short allele correlated with risk-taking behavior) and DRD4 (a dopaminergic receptor gene associated with impulsivity), both of which have been implicated in human life history, were obtained from Minkov and Bond (2015). Biodemographic data on life expectancy, total fertility rate, and infant mortality rate at the national level were collected from the World Bank (World Bank, 2012a, b, c). Data on ancestral sociopolitical centralization were collected from Giuliano and Nunn (2018). These researchers developed a database based on Murdock's Ethnographic Atlas, the World Ethnographic Sample, and cross-cultural repositories assembled by other sources. According to these researchers, they connected the various sociopolitical, cultural, and ecological indicators with the distribution of current populations as specified by the *Ethnologue*. Data on the Human Development Index (HDI) for the year 2017 were obtained from the *UN Human Development Report* and accessed via the online platform *Our World in Data* (https://ourworldindata.org/grapher/human-development-index?time=2017). The HDI is comprised of four indicators: (1) life expectancy at birth; (2) mean years of schooling; (3) expected years of schooling; and (4) Gross National Income per capita.

Additionally, cross-national data on state functioning indicators were collected from Rindermann and colleagues (Rindermann et al., 2015). These included government effectiveness, innovation, and national competitiveness. A unit-weighted factor was computed after standardizing these indicators and estimating an average across the corresponding *z*-scores (Gorsuch, 1983).

The present study employed a phylogenetic tree incorporating national populations as its nodes and derived using a matrix of genetic distances across nations. These values were reported initially in Spolaore & Wacziarg's (2018) supplementary material as Fst estimates. The *upgma* function associated with the *phangorn* package (Schliep, 2011) was used to generate the phylogeny. Phylogenetic independent contrasts (PIC) were generated with the *pic* function associated with the *ape* package (Paradis et al., 2004) for ancestral sociopolitical centralization and HDI, and the genetic slow life history factor and the state functioning factor. A unit-weighted biodemographic slow life history factor was estimated based on the PIC for life expectancy, the PIC for the reverse-scored total fertility rate, the PIC for the reverse-scored infant mortality rate, and subsequently correlated with the PIC of the genetic slow life history factor (this being the average of the frequencies of the two variants reported in Minkov & Bond, 2015).

Model comparison across selection regimes was performed on the ancestral sociopolitical centralization and HDI values, the genetic slow life history factor scores, and the state functioning factor scores. The examined models included the following (based on descriptions provided in the *geiger* package; Pennell et al., 2014): (1) Brownian motion, which is a random-walk, cumulative model of trait change; (2) Ornstein–Uhlenbeck, which indicates (potentially adaptive) evolution towards a central value; (3) Lambda, which relates covariation of trait levels to degree of shared ancestry; (4) Early burst, which concerns exponential trait increase and/or decrease in evolutionary rates; (5) Kappa, which concerns a punctuational model (i.e., periods of rapid trait change punctuated by periods of stasis); (6) Delta, which is a time-sensitive model, contrasting early relative to later evolutionary changes; and (7) White noise, which can indicate a non-phylogenetic mode of trait change, or the presence of severe measurement error.

The PICs of the variables were used to estimate six competing path models with the *lavaan* package (Rosseel, 2012). As several alternative models had the same number of degrees of freedom (although they examined different theoretical predictions), it was deemed inappropriate to use likelihood ratio tests to determine the corresponding model adequacy. Consequently, a model comparison was conducted based on *CFI*, *TLI*, *AIC*, *BIC*, *SABIC*, *ΔSABIC*, and *SABIC* weights.

Rather than rejecting a-priori the influence of cross-national differences in genetic frequencies, as per L&F, the present study developed rival models concerning the role of genetic slow life history factor scores and sociopolitical variables. We classified these analyses into three main sets: (1) *Genetically driven*, giving priority to the genetic slow life history factor in the sequence, represented in Fig. 4a and b; (2) *Socioculturally driven*, giving priority to the sociopolitical and development indicators represented in Fig. 4c and d; and (3) *Genetically mediated*, including the genetic slow life history factor as a mediator between ancestral sociopolitical centralization and contemporary variables of development and state functioning, represented in Fig. 4e and f.

Results

Measurement Models

Phylogenetic comparative examinations determined that the unit-weighted genetic slow life history factor scores displayed a sizable phylogenetic signal (Pagel's $\lambda = 0.771$) that was significantly different from both zero ($p < 0.0001$) and one ($p < 0.0001$); these results strongly suggest that the factor scores covary in part due to shared ancestry; however, they do not follow a pattern consistent with Brownian Motion. Similarly, ancestral sociopolitical centralization exhibited a large phylogenetic signal (Pagel's $\lambda = 0.917$), with a value significantly different from zero ($p = 0.0038$) and one ($p = 0.0302$). The HDI values were also phylogenetically conserved (Pagel's $\lambda = 0.897$) and were significantly different from zero ($p < 0.0001$) and one ($p = 0.0023$). Lastly, the state functioning unit-weighted factor scores were found to exhibit a small-to-medium phylogenetic signal (Pagel's $\lambda = 0.398$), with this value remaining similar to zero ($p = 0.0505$) but significantly different from one ($p < 0.0001$). Figure 5 displays an ancestral character reconstruction based on maximum likelihood with the unit-weighted slow life history factor scores featured on the left side of the diagram and the ancestral sociopolitical centralization factor on the right side.

Bivariate correlations among the PIC for the biodemographic slow life history factor indicated significant part-whole correlations with the PIC for life expectancy at birth, the PIC for reverse scored total fertility rate, and the PIC for reverse scored infant mortality rate, supporting the structural validity of this latent dimension. Additionally, the analyses also explored the associations among the PIC for the genetic slow life history factor, the PIC for life expectancy at birth, the PIC for reverse scored total fertility rate, the PIC for reverse scored infant mortality rate,

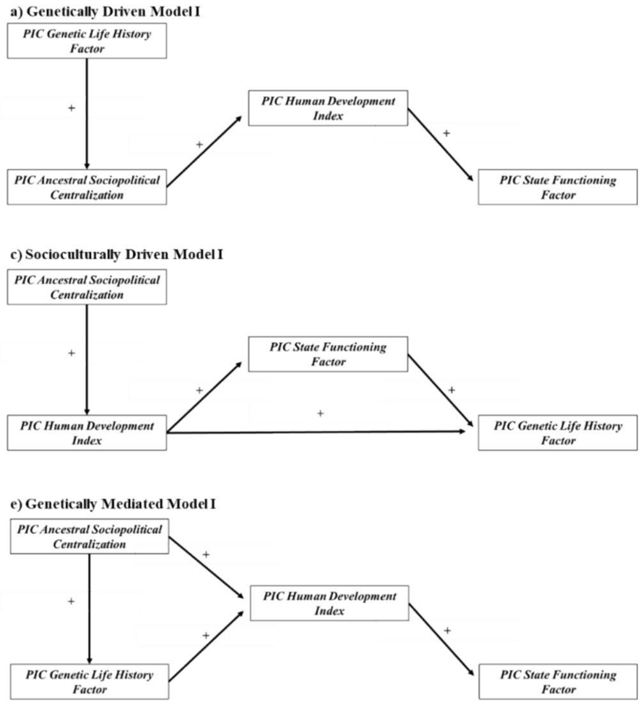

Fig. 4 Six alternative path analytical models examining the interplay among the PIC for ancestral sociopolitical centralization, the PIC for the genetic slow life history factor, the PIC for the HDI, and the PIC for the state functioning factor

and the PIC for the biodemographic slow life history factor. Significant positive associations were found between the PIC for the genetic slow life history factor, the PIC for the respective life history indicators, and the PIC for the biodemographic slow life history factor. These results are further described in Table 1. One notable finding here is that the genetic slow life history factor exhibits a high magnitude ($r \geq 0.5$, Cohen, 1988) correlation with biodemographic measures of the same ($r = 0.659$, $p < 0.0001$), indicating that this genetic index has strong predictive validity.

The model comparison for the ancestral sociopolitical centralization scores indicated that the Ornstein–Uhlenbeck model exhibited the best statistical fit, whereas White noise was associated with the worst. These results indicate that the evolution of ancestral sociopolitical centralization varies toward a central value, suggesting the action of adaptive optimization (Butler & King, 2004). A model comparison across selection regimes on the unit-weighted genetic slow life history factor scores indicated that Lambda exhibited the best statistical fit. Thus, across nations, the underlying phylogenetic structure conditions the similarities in genetic slow life history factor scores due to shared ancestry. With regard to the HDI, it was found that Lambda exhibited the best statistical fit, whereas White noise exhibited the worst. Consequently, shared (phylogenetically structured) ancestry across the populations of different nations is a contributing factor to the observed similarities in the HDI. Lastly, the model comparison for the unit-weighted state functioning factor scores suggested that Lambda was the best fitting model. These results are further described in Table 2.

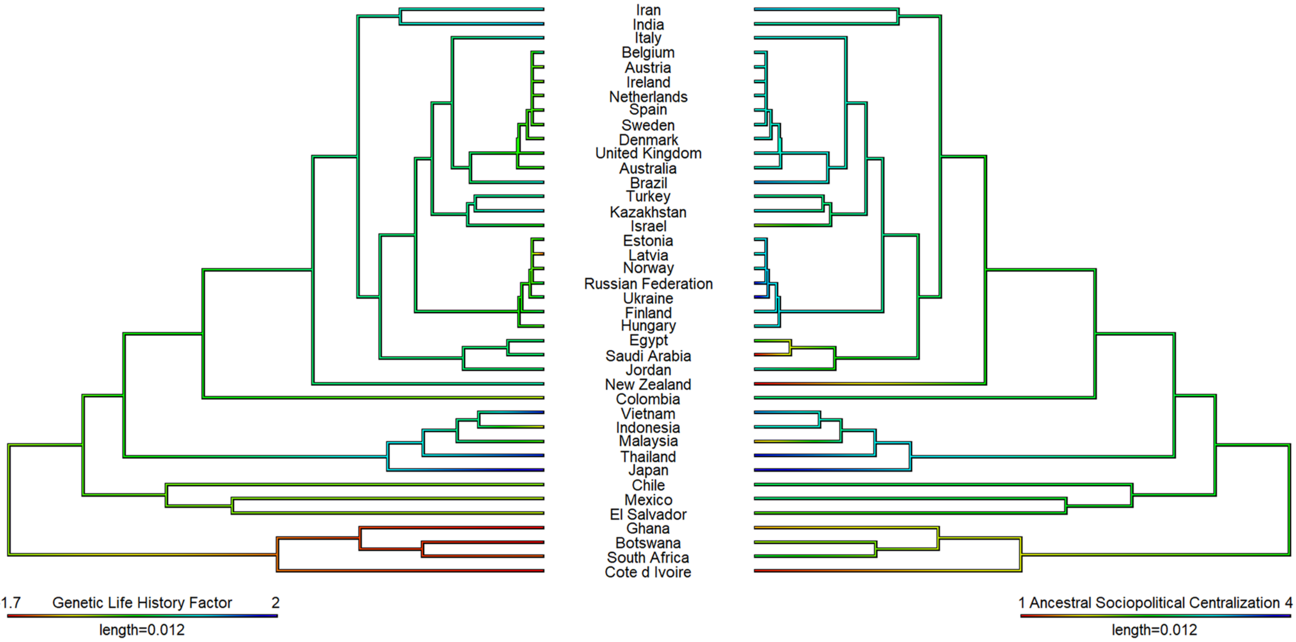

Fig. 5 Maximum likelihood reconstruction of the genetic slow life history factor and ancestral sociopolitical centralization

Table 1 Bivariate correlations among the PIC for the genetic slow life history factor, the PIC for life expectancy at birth, the PIC for the reverse scored total fertility rate, the PIC for the reverse scored infant mortality rate, and the PIC for a biodemographic slow life history factor

	1	*2*	*3*	*4*	*5*
PIC genetic slow life history factor (1)	1.000				
PIC life expectancy at birth (2)	0.682	1.000			
PIC rev total fertility rate (3)	0.397	0.587	1.000		
PIC rev infant mortality rate (4)	0.722	0.940	0.699	1.000	
PIC biodemographic slow life history factor (5)	0.659	0.926	0.837	0.967	1.000

Note. All correlations are statistically significant at $p < 0.05$

As L&F's position is that for complex traits, such as life history, drift should be the major factor acting on its associated variants, we furthermore examined whether the genetic slow life history factor score varied due to drift. Observed evolutionary rates for the genetic slow life history factor score were computed with the *calc_rate* function. Simulated scores were produced using the *fastBM* function, and these values were subsequently used to estimate simulated evolutionary rates. The analysis revealed a statistically significant difference between the simulated rates and the observed rates for the genetic slow life history factor ($p < 0.0001$), indicating that the observed variance in this latent dimension was *not* attributable to drift.

Structural Models

Model comparison revealed that the sixth model exhibited the best statistical fit given baseline appraisals (*CFI* = 1.000, *TLI* = 1.000, *RMSEA* = 0.000) and when compared to alternative models (*ΔSABIC* = 0.000, *SABIC weight* = 0.849), with the genetic slow life history factor mediating the influence of ancestral sociopolitical centrality on HDI and the state functioning factor as indicated in Fig. 6. In turn, the third model exhibited the weakest statistical fit given baseline appraisals (*CFI* = 0.675, *TLI* = 0.351, *RMSEA* = 0.443) as well as when compared to alternative models (*ΔSABIC* = 24.053, *SABIC weight* = 0.000). Table 3 presents the results of these model comparisons in further detail.

The main objective of this analysis is to challenge L&F's second clam by demonstrating how testing multiple working hypotheses regarding the sources of variation across human populations can be conducted. Our best fitting model is consistent with GCC, as the ancestral sociopolitical centralization variable, which captures potential cultural evolutionary trends, is predictive of variance in the genetic slow life history factor, suggesting that changes in ancestral sociopolitical centralization have *favored* higher frequencies of alleles associated with slow life history via GCC. Our finding is further evidence that such selection does not necessarily act on single genes in isolation, but acts simultaneously on multiple alleles, the genetic slow life history factor essentially functioning as a "mini" PGS comprised of two variants whose frequencies are highly intercorrelated across populations (Minkov & Bond, 2015), and which are likely to be correlated with large numbers of other functionally related variants that are unmodelled. Increases in the frequencies of these variants may in turn have facilitated increases in HDI via the construction of environments in which wealth can increase via comparative advantage. The provisioning of stable environments may also have permitted education and concomitant acquisition of cognitive capital, in addition

Table 2 Model comparison across several selection regimes with respect to the observed unit-weighted genetic slow life history factor scores. Best fitting models in bold

Model	ASCC AICc	ASCC ΔAICc	ASCC AICc weight	GLH AICc	GLH ΔAICc	GLH AICc weight	HDI AICc	HDI ΔAICc	HDI AICc weight	SF AICc	SF ΔAICc	SF AICc weight
Brownian motion	88.748	6.985	0.020	109.748	17.086	0.000	−75.925	7.287	0.013	226.287	29.399	0.000
Ornstein–Uhlenbeck	81.763	0.000	**0.653**	101.931	9.269	0.009	−81.886	1.325	0.248	201.998	5.110	0.051
Lambda	86.050	4.287	0.077	92.662	0.000	**0.944**	−83.211	0.000	**0.482**	196.888	0.000	**0.662**
Early burst	90.748	8.985	0.007	111.748	19.086	0.000	−73.925	9.287	0.005	228.287	31.399	0.000
Kappa	87.282	5.519	0.041	98.831	6.169	0.043	−80.969	2.242	0.157	210.523	13.635	0.001
Delta	84.141	2.377	0.199	104.705	12.044	0.002	−79.982	3.229	0.096	217.051	20.163	0.000
White noise	92.439	10.676	0.003	106.670	14.008	0.001	−60.244	22.967	0.000	198.564	1.675	0.286

Note. *ASCC*, ancestral sociopolitical centralization; *GLH*, genetic slow life history factor; *HDI*, human development index; *SF*, state functioning

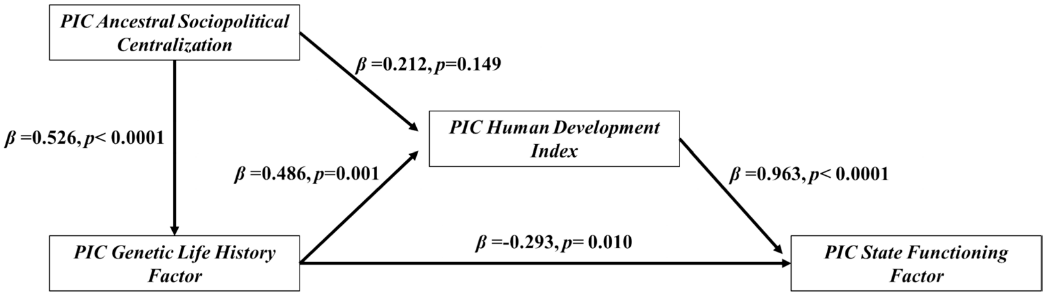

Fig. 6 Path analysis with phylogenetic independent contrasts examining the influence of ancestral sociopolitical centralization on the genetic slow life history factor and the state functioning factor, and HDI

Table 3 Model comparison featuring various statistical fit indices for six alternative path analytical models. Best fitting model in bold

Model	RMESA	AIC	BIC	SABIC	ΔSABIC	SABIC weight	CFI	TLI
1	0.340	280.890	290.871	272.103	14.658	0.001	0.808	0.617
2	0.329	276.774	288.419	266.523	9.078	0.009	0.881	0.642
3	0.443	290.285	300.266	281.498	24.053	0.000	0.675	0.351
4	0.222	272.193	283.838	261.942	4.497	0.090	0.946	0.837
5	0.251	273.277	284.921	263.025	5.580	0.052	0.930	0.791
6	**0.000**	**269.161**	**282.469**	**257.445**	**0.000**	**0.849**	**1.000**	**1.000**

to the creation of food surpluses, allowing for levels of nutrition to rise. Boosted HDI in turn engendered more efficient state functioning. The direct effect of the genetic slow life history factor on state functioning is negative in direction, however. This possibly reflects the action of the increased numbers of fast life history individuals that can be accommodated via slow life history inceptive niche construction raising environmental carrying capacity, and therefore standing genetic diversity.

Egalitarian Meta-political Activism

While the empirical and theoretical shortcomings of CGCC are serious, a potentially far greater problem for this research program concerns its meta-political goals. L&F's framing of CGCC seems to be purely oppositional in nature. Much of their Perspective is devoted to characterizing a very loose knit but nonetheless expansive rival paradigm, which incorporates the eugenics of Shockley, the racial hereditarianism of Jensen, the sociobiology of Lumsden and Wilson, and even extends out to encompass the massive modularity of the Santa Barbara School of evolutionary psychology. The last of these specifically is criticized in relation to L&F's broader dismissal of the work of Lumsden and Wilson (1981), where they note:

> These claims, which were subject to strong criticism at the time, are inconsistent with extensive data in humans documenting both widespread cultural inheritance... and extensive interaction between cognitive domains within the human brain..., leading to a shift away from claims of "massive modularity" (p. 3).

The fact that Jensen's work vitalizing Spearman's concept of the *g* factor, the positive manifold among performance on many different, and seemingly discrete, measures of cognitive ability (Warne & Burningham, 2019), stands at odds with claims of massive modularity (for more on this tension see Woodley of Menie & Sarraf, 2021), goes wholly unremarked upon. L&F seem to want to simply force these (in some cases, starkly) divergent theoretical perspectives into a broad associative rubric of reductionism and genetic determinism.

Beyond the purported theoretical and empirical deficiencies of these sorts of ideas, there is the allegation that such thinking has extra-scientific costs, specifically that it is dangerous. In critiquing the theory of *genetic nurture*, a *mainstream sociogenomic* theory which maintains that phenotypes develop in response to the distribution of genotypes among conspecifics, influencing trait development through indirect genetic effects (see Kong et al., 2018), L&F note that:

> This "genetification" of culture, sometimes labeled "genetic nurturing"..., has a *sinister side*. If all facets of an individual's life were determined by genetics, rather than social experience and access to resources, *then inequities in power and wealth could be (falsely)*

> *cast as natural and inevitable*... Economist Gregory Clark's... suggestion that inheritance of social status in England over a 400-y period is genetic is a case in point. (p.8, italics for emphasis)

L&F further note in relation to Clark's (2023) work that:

> [w]hile [Clark's] study does not mention race, *its messages are disturbingly evocative of Jensen's claims of 50 B.P. and will perhaps inevitably be interpreted similarly*. (p.8, italics for emphasis)

The term racism and cognates appear 21 times in the main text of L&F's paper, the word even being included in the title. Racism per se is never defined concretely anywhere in the text of their article. This strategic vagueness permits the casting of shade over any idea or concept that does not sit well with the egalitarian ideological views of the authors. That these authors *are* committed egalitarians is clear from a plain reading of their essay. They proclaim that thinking of race and racial differences in any reductively biological sense, or even entertaining ideas that could *lead* to such reductively biological thinking, is dangerous as "inequities in power and wealth could be (falsely) cast as natural and inevitable" (p.8), and yet they prevaricate on the issue of the actual harms stemming from a *mere* belief in "natural inequality," alluding simply to "racism and hate crimes" being "on the rise" (p.1), the only reference given here being to a book written by a left-wing journalist. Cavalier appeals to authority take the place of reasoning when it comes to asserting expert consensus on the apparent lack of genetic differences between socially defined racial groups. Yet no attention is paid to major surveys that find that this consensus is not as firm as is often asserted, such as that of Nelson and colleagues (Nelson et al., 2018), who note that:

> Through a qualitative content analysis of free-text comments from 515 survey respondents, we identified key themes pertaining to multiple meanings of race, the use of race as a proxy for genetic ancestry, and the relevance of race and ancestry to health. Our findings suggest that for many genetics professionals the questions of what race is and what race means *remain both professionally and personally contentious*… While there may be consensus in the scientific community that socially defined races are not discrete taxonomic, biological, or genetic groups, *disagreements remain about whether and to what extent race is a useful proxy for genetic or other biological differences between individuals*. (p. 222, italics for emphasis)

Similarly, no less an august personage than Harvard University's Professor David Reich had this to say in the *New York Times* in 2018:

> …over the years this consensus [that race is a purely social construct] 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 any meaningful biological traits that those differences can be ignored… I have deep sympathy for the concern that genetic discoveries could be misused to justify racism. But as a geneticist I also know that it is simply no longer possible to ignore average genetic differences among 'races.' Groundbreaking advances in DNA sequencing technology have been made over the last two decades… *With the help of these tools, we are learning that while race may be a social construct, differences in genetic ancestry that happen to correlate to many of today's racial constructs are real. Recent genetic studies have demonstrated differences across populations not just in the genetic determinants of simple traits such as skin color, but also in more complex traits like bodily dimensions and susceptibility to diseases.* I am worried that well-meaning people who deny the possibility of substantial biological differences among human populations are digging themselves into an indefensible position… that will not survive the onslaught of science. (Online article, no page numbers given, italics added for emphasis)

In their zeal to decry the biologization of race, L&F commit other errors. For example, on page 6, L&F claim that "the overlap between races and genetic ancestry is at best partial." Yet, fully in line with Reich's (2018) article, studies find exceptionally strong correlations (> 0.9) between self-identified race and biogeographic ancestry (e.g., Tang et al., 2005). This does not of course militate against the idea that race exhibits socially constructed features, or that perceptions of these purely social features can be manipulated such that they are no longer salient to coalition membership (as has been demonstrated experimentally; see the meta-analysis of Woodley of Menie et al., 2020). It does however indicate that so-called races are *more* than merely a social reality (Edwards, 2003). Accepting that these are in some useful sense biologically real does not, needless to say, entail resurrection of naïve folk conceptions of race, or the geographical race concept, the notion that human races correspond to subspecies. More nuanced forms of population thinking applied to race and related concepts have been proposed (e.g., Piggliucci & Kaplan, 2003, who suggest that human populations might constitute *ecotypes*). In another instance, L&F assert that:

> Within evolutionary biology the term "race" has a precise scientific meaning, broadly equivalent to "subspecies," but human populations are not distinct enough to qualify as races. Subdivision is commonly quantified using a statistic called FST, which relates the genetic variation within and between populations. FST ranges from 0 (no population subdivision) to 1 (complete subdivision), with an FST exceeding 0.25 potentially indicating the existence of genetic subgroups... An authoritative estimate from the 1000 Genomes Project... of the global human FST was 0.052 to 0.083, revealing that the human population exhibits relatively little genetic subdivision. (p.3)

Fst (also termed the fixation index) *is not and has never been* used as a basis for ascertaining the existence of subspecies, a strictly *taxonomic category*. Traditionally visual sorting approaches by phenotype, such as the 75% rule, have been used to taxonomically validate subspecies. This rule is based on the idea that a prospective subspecies is taxonomically valid only when ≥ 75% of those organisms comprising it fall outside 99% of the range of variation for a given trait, or set of traits, when compared to a second grouping in the same species (Patten & Unitt, 2002).

Fst therefore has nothing to do with the identification of subspecies, it does however describe the degree to which loci are differentiated at the level of population structure as opposed to at the level of individuals and functions analogously to group and individual level partitioning in ANOVA type models. Low values indicate no differentiation (such as in the case of a panmictic population), and high values indicate the presence of great differentiation, with such populations being organized into discrete subgroups. When Sewall Wright (1978) developed this metric, he proposed that values between 0.15 and 0.25 be used as the basis for determining the presence of *moderately great* levels of population subdivision. Contrary to what is claimed by L&F an Fst > 0.25 *is not and never has been* a criterion for accepting the existence of "population subgroups" which they seem to imply would count as subspecies. This peculiar claim originated in a paper by Alan Templeton published in 1998 (Templeton, 1998), having since gone on to become something of a scientific urban legend. Wright (who believed that humans were polytypic purely on the basis of visual classification[5]) notes also that "[d]ifferentiation is, however, *by no means negligible if [Fst] is as small as 0.05 or even less*" (p.85, italics for emphasis). This is because small numbers of highly polymorphic loci varying at only a few sites in the genome can generate substantive differentiation, even when Fst is low (see Coyne, 2010, Ch. 8). While Fst cannot be used to establish the presence of taxonomic subspecies, it is nonetheless notable that there are several species whose Fst values have been found to be *lower* than or within the range of values reported for humans by L&F (0.052 to 0.083), but which are nevertheless considered to be taxonomically polytypic (again based on visual classification). Examples include the Canadian Lynx (*Lynx canadensis*, autosomal Fst = 0.033, Schwartz et al., 2002), K = 3 subspecies (according to at least some taxonomists, see Wozencraft, 2005), and the African Buffalo (*Syncerus caffer*, autosomal Fst = 0.059, Van Hooft et al., 2000), K = 5 subspecies. Trying to argue against the idea that humans are polytypic on the basis of their modest Fst value is flawed and can easily backfire, as illustrated by the aforementioned examples. To reiterate, Fst is not used as grounds for identifying subspecies. That errors of this sort permeate L&F's essay does not help their case in any way.

More apparent instances of L&F's motivated reasoning can be seen in their treatment of cross-population variation in national cognitive abilities (NCA), as exemplified in the following statement:

> [It has been] show[n] that recently published claims about average IQ differences between nations are seriously flawed, being reliant on data that fall well below academic standards, yet these data are still regularly used by credulous scholars. (p.7)

According to Warne (2023), critics (such as the one referenced by L&F) have argued that as several samples in NCA databases were gathered from children, it is methodologically incorrect to draw inferences concerning the IQ of adults in those populations. This argument stems from critics' obliviousness to fundamental psychometric distinctions between absolute ontogenetic changes in cognitive ability levels and IQ as a measure indexing *relative* ability level among peers; hence, IQ scores remain age-neutral and suitable for comparisons between age cohorts (Rindermann, 2018; Warne, 2020, 2023). Additionally, longitudinal analysis provides empirical support for the stability of IQ rank orders across the life course, which is to say that those with IQs of, e.g. 110 at age 8 are likely to have IQs very close to 110 at age 55 (Eichelberger et al., 2023). Critics have also averred that as some of the samples in NCA databases are small in size, this introduces unacceptable levels of error into such estimates, compromising their usefulness. However, in mathematically examining this question Warne found that samples with at least 96 individuals (assuming a 95% CI) generated values of ± 3 IQ points. Larger samples ($n = 216$ and $n = 856$) were associated with narrower ranges

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[5] "There is also no question, however, that populations that have long inhabited separated parts of the world should, in general, be considered to be of different subspecies by the usual criterion that most individuals of such populations can be allocated correctly by inspection. It does not require a trained anthropologist to classify an array of Englishmen, West Africans, and Chinese with 100% accuracy by features, skin color, and type of hair in spite of so much variability within each of these groups that every individual can easily be distinguished from every other" (Wright, 1978, p. 439).

(± 2 and ± 1 IQ points, respectively; Warne, 2023). Of the more than 130 countries examined in Lynn and Becker's IQ database, only five nations fell outside of the range of ± 3 points in IQ (Warne, 2023).

Moreover, several publications on human behavioral phenotypes such as mating strategies, attachment styles, and personality traits (Buss, 1989; Jonason et al., 2020; Schmitt, 2003, 2005) have not been the target of similar criticisms despite similarly being dependent on small-to-medium size samples. It is quite frequent for researchers involved in these lines of work to examine the latter traits by aggregating individual-level data as means, medians, variances, or *D*-coefficients, and correlating these values with subnational or national level information. Moreover, the results of student assessment studies involving large numbers of countries (such as the PISA tests administered to the OECD countries) are routinely used in cross-cultural social science with little objection, despite the fact that outcomes in these assessments highly correlate with NCA estimates (see Warne, 2023, Table 2).

Despite the basically identical methodological approaches of research on NCA and other phenotypes such as those named above, *isolated demands for rigor* apparently stemming from ideologically motivated reasoning have been applied uniquely to NCA analyses. Additionally, current examinations of so-called non-WEIRD samples, as in the case of experimental economic studies (Henrich, 2005), also explore cross-population differences in behavioral traits by aggregating participant data at the level of sociocultural communities. It is also interesting to note that these cross-population differences are investigated using precisely the kind of cross-national study designs that L&F criticize, but this line of research does not seem to attract controversy.

Concerning potential outliers in NCA databases, removing the problematic values, using transformation procedures (such as winsorizing), or updating the estimates based on more recent data effectively deals with this methodological challenge. Moreover, a recent meta-meta-analysis examination (Peñaherrera-Aguirre & Woodley of Menie, in press) of effect sizes estimated between NCA and numerous ecological, sociopolitical, environmental, and other variables provides strong evidence of predictive and convergent validities within the extremely large database of studies using NCA estimates.

Ultimately, L&F give the distinct impression that *any* explanation for social-racial or other population differences that posits any causal factor outside the category of extragenetic influence, is simply wrong by fiat. The following text exemplifies this motivated reasoning:

> Such "racial" differences can persist over time, but not because of genetic transmission, which is now recognized to be just one component of human inheritance. Rather, humans have constructed "inequitable niches"... which persist through the legacies of inherited norms and institutions, inherited wealth and power, inherited values and traditions, and inherited environments that vary in their amenities and opportunities. These inequitable niches explain why Ashkenazi Jews score highly for IQ, why Jamaicans excel at sprinting, and why African Americans are more likely to die of heart disease. That these and other discrepancies between socially defined races can be attributed to genetic differences, may be intuitive, but it is wrong. (p.8)

From all that they write on the matter, it would seem that L&F consider belief in environmentalist theories such as these to be an unalloyed good. The vast suffering that radical egalitarians inflicted in the course of trying (and failing) to purportedly perfect human populations via extreme forms of totalitarian social engineering, sometimes involving the violent suppression of genetic science, is never commented upon anywhere in L&F's essay. Lysenkoism in Stalin's USSR is a textbook example of this. Yet it is no less reasonable to assume that the sort of environmental absolutism that they propound *could* (and in actuality *did*) facilitate horrific political abuses (such as the Killing Fields of Cambodia, the Great Leap Forward, and the Holodomor, as well as many terrorist atrocities committed in Western countries in the 1970s and 80s in the name of Marxism, etc.) than to assume that thinking of a genetically determinist or reductionist sort will normalize inequality and potentiate the far right and racist violence (for related arguments see Pinker, 2002; Woodley of Menie, Sarraf, & Peñaherrera-Aguirre, 2023; Woodley of Menie et al., 2025). To be clear, we are not claiming that L&F are proponents of Lysenkoism, or totalitarian leftism. But that they are blind to the possibility that their thinking *could be misused* as justification for objectively harmful policies or behaviors (such as terrorism of a leftist sort) nevertheless belies their bias, and reveals their work to be, at its core, an instance of *egalitarian meta-political activism* (for more on the defining characteristics of this, see Woodley of Menie et al., 2023). In opposition to this sort of activism, we champion the Mertonian norms of science, specifically *universalism*, *communality*, *disinterestedness*, and *organized skepticism* (Woodley of Menie et al., 2025).

L&F further add to the mix the charge of eugenicism, this apparently being yet another element, or rather potential consequence, of the reductionist and determinist paradigm against which they position CGCC. In this regard they note that:

> The intellectual distance between claims of genetic bases for educational attainment, occupational status, and economic success, and eugenic ideas of class and racial differences *has never been great*. As a result, it

> behooves scholars to be ready with responses to claims that might initially seem innocuous but may actually turn out to be pernicious. (p.8, italics for emphasis)

Owing to this apparently small difference, even those with the most unimpeachable of leftist bona fides, such as Robert Plomin and K. Paige Harden, are objects of suspicion to L&F:

> Psychology professor Robert Plomin in his (Plomin 2018) book states that parent–offspring correlation in occupational status and income are "*chiefly caused genetically*" (p. 101), that the ability of "*the educational attainment polygenic score … to predict intelligence and reading comes from generalist genes*," and that "*the most systematic and objective predictor of occupational status and income*" is "*inherited DNA differences*" (pp. 100–101). These claims are reiterated by Harden (Harden 2021), who states that "*genes cause differences in educational attainment*" (p. 125) and that genetic differences between people "*cause social inequalities … and fertility outcomes like age at first birth*" (p. 129). (p. 8, italics in original)

This is especially fascinating, as it strikingly highlights a conflict between those pursuing different egalitarian meta-political gambits noted in Woodley of Menie and colleagues (Woodley of Menie et al., 2023). Some activists (especially those associated with the Hastings Center) seem to want to merely gatekeep behavior genetics and sociogenomics so as to prevent what are supposed to be racist uses of the data of those fields. They seem to either accept or be indifferent to certain facts that even the most liberal of sociogenomics researchers (such as Harden) find completely uncontroversial—such as the aforesaid claim that "genes cause differences in educational attainment" (Harden, 2021, p. 125). Others, of an apparently more radical sort, such as L&F, take exception even to these findings, stating that "it behooves scholars to be ready with responses to claims that might initially seem innocuous but may actually turn out to be pernicious." (p.8). It can only be presumed therefore, that the latter sort of egalitarian would, if given the opportunity, likely move to censor and "cancel" the former sort.

Discussion

We have demonstrated that the two core claims of CGCC are incorrect. By contrast, successful empirical tests of biocultural dynamics hypotheses include but are not limited to the following, which will be listed here without recapitulating the voluminous evidence that we have already presented and detailed above: (1) Finding that the changing balance between the pressures of individual and group selection predicts the varying trajectories of evolution for the general and specialized components of cognitive ability over the past 400 years; (2) Finding that the outcomes of historical competition among rival empires predict the integration and differentiation of biodemographic life history traits over the past 200 years; (3) Finding that zoogeographical regions predict most of the variance in both life history and cognitive ability among both national and subnational polities; (4) Finding robust effects in archaeogenetic studies of cultural transitions in initiating and moderating the intensity of selective sweeps acting on cognitive and behavioral genotypes; (5) Finding that archaeological and archaeogenetic evidence supports the existence of multiple successive large-scale population migrations and expansions during the Neolithic and Bronze Age, leading to varying biodemographic outcomes ranging from near-total population replacements to major biocultural admixtures; (6) Finding that multiple instances worldwide support the general principle in the dynamics of cross-ethnic hybridizations that higher-status groups contribute the preponderance of Y-DNA and lower-status groups contribute the preponderance of Mt-DNA. In directly testing the second of L&F's claims, we (1) found that a molecular-genetic index of behavioral life history predicts biodemographic life history; (2) found that ancestral sociopolitical complexity predicts a genetic factor score for slow life history; (3) found that the genetic factor score for slow life history predicts HDI as well as a state functioning factor; and (4) found that HDI also predicts the state functioning factor. The last three demonstrate recent GCC, with the model specifying the relevant causal pathways outperforming those that make alternative predictions concerning causal associations among these variables.

Aside from its *methodological* failings, there are also serious *theoretical* limitations of the CGCC approach. L&F's conceptualization of CGCC fails to incorporate certain very important innovations in evolutionary theory introduced since the turn of the current century. For example, the seminal work of West-Eberhard (2003) on the dynamic relations between developmental plasticity and evolutionary processes goes completely ignored. We provide a necessarily very brief synopsis of her book, which is over 800 pages in length, followed by a statement of its relevance to the problem of GCC.

According to West-Eberhard's theory, there typically exists a reservoir of genetic variation in any population that is not currently of any adaptive significance, although populations not possessing such a reservoir run a high risk of extinction. Any novel environmental or genetic (meaning mutational) input may act differentially upon these variable genotypes to produce different epigenetic variants in response to these novel inputs by means of *gene-environment (G*E)* or *gene–gene (G*G)* interactions. These G*E or

G*G interactions cause pre-existing developmentally plastic phenotypes in the population to undergo the processes of *phenotypic accommodation*, which are developmental reorganizations producing novel phenotypic variants in response to environmental changes (including any new genetic mutations that might arise).

The novel phenotypic variants produced may then be subject to positive or negative selection, based upon the relative fitness of each epigenetic response in the context of the new environment. This positive or negative selection among the novel phenotypic variants indirectly generates selection upon the genotypes involved in the G*E or G*G interactions that epigenetically produced these variants, a process called *genetic accommodation.* Genetic accommodation either upregulates (raises) or downregulates (lowers) the quantitative thresholds at which genotypes react to scaling magnitudes of the new environmental or genetic input by epigenetically expressing the novel phenotypic variants, a process called *threshold selection.*

Threshold selection may act to genetically fix a trait in the population by either *raising* or *lowering* the quantitative threshold for expression respectively *above* or *below* the limits of the stochastically probable (or ecologically natural) range of environmental variability normally experienced by the population. Threshold selection acting to raise the threshold for expression of the novel phenotypic trait *beyond* the limits of the stochastically probable (or ecologically natural) range of environmental variability is a special case of genetic accommodation, previously called *genetic assimilation* by Waddington (1942, 1953). When thresholds for epigenetic trait expression fall *within* the limits of the stochastically probable (or ecologically natural) range of environmental variability, threshold selection produces *polyphenism* in the population, based upon the developmental plasticity entailed in such an intermediate threshold.

Thus, the very stark dichotomy introduced by L&F to describe the processes of GCC, wherein environmental effects are implicitly given primacy over genetic effects, falls far short of the richness and nuance of more contemporary ideas on the matter. According to West-Eberhard (2003), phenotypic accommodation frequently leads in the process of evolutionary change, but these adaptations cannot be made permanently fixed within the population without subsequent genetic accommodation to establish the novel epigenetic effect as the species-typical norm of reaction. This all implies that genetic selection, and not just cultural selection, is absolutely essential to the process of GCC, as originally envisioned in the seminal work of Lumsden and Wilson (1981).

More recently, this process of genetic selection for the epigenetic effects evoked by the adaptive landscapes presented by environments has been dubbed *epigenetic selection* (Figueredo et al., 2020a) for greater simplicity of reference in place of extended theoretical expositions like the present one.

Conclusion

Finally, the point in drawing attention to the more explicitly meta-political aspects of L&F's work, is not to simply demonstrate how they compound error, but to highlight a serious contradiction between their stated goals, and the likely effects of their activism.

L&F conclude their essay with an appeal for more research within the spirit of CGCC:

> By helping to partition variation more appropriately, and by explicitly recognizing the multiplicity of interacting causal paths, analyses that incorporate CE and GCC can complement other excellent resources... as important tools to this end. (p. 8)

Yet their hermeneutic of suspicion "behooves scholars to be ready with responses to claims that might initially seem innocuous but may actually turn out to be pernicious" (p.8). Logically, given this, research of the sort that they seek to encourage must necessarily be constrained by its social consequences—even in instances where these are not *known* beforehand. So presumably, researchers could (as we have done) conduct formal tests of CGCC in the spirit of "helping to partition variation more appropriately," but which falsify an important claim (e.g., that drift prevents the emergence of *coherent* patterns of genetic variation among populations in terms of the frequencies of certain trait-determinative alleles), and which therefore *must* be countered on extra-scientific, specifically meta-political, grounds (e.g., such research could occasion racist or other violence and cannot therefore be tolerated as it is pernicious, etc.) This deep fact-value conflation (Cofnas, 2016) therefore exposes the core motives at play in L&F's Perspective, that is the (eventual) prevention of *all* research into behavior genetics and genomics, and related fields for fear that these findings might lead to the spread of eugenic and racist thinking, *not* because they are false, but because they might be true. Such beliefs are simply inconsistent with Mertonian norms of good scientific practice; furthermore, regimes of de facto scientific censorship such as that seemingly tacitly favored by L&F, never in reality lead to reductions in public misunderstandings of scientific facts (as Reich notes, in practice, politically "correct" misunderstandings are simply replaced with another older set of misunderstandings). Such actions furthermore deflect attention away from the very real causes of violence motivated by political extremism, such as irresponsible media coverage of, e.g., mass shootings (see Jetter & Walker, 2022; for very detailed discussion of this and related issues, see Woodley of Menie et al., 2025).

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*Indicates that the study was included in the meta-analysis

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