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Cultural evolution of genetic heritability

Ryutaro Uchiyama[a], Rachel Spicer[b] and Michael Muthukrishna[b]

[a]NTU–Cambridge Centre for Lifelong Learning and Individualised Cognition, Nanyang Technological University, Singapore 639798 and [b]Department of Psychological and Behavioural Science, London School of Economics and Political Science, London WC2A 2AE, UK.
inbox@uchiyamaryutaro.com
https://www.uchiyamaryutaro.com;
r.a.spicer@lse.ac.uk
http://www.lse.ac.uk/PBS/People/Rachel-Spicer;
m.muthukrishna@lse.ac.uk
https://michael.muthukrishna.com/

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Abstract

Behavioral genetics and cultural evolution have both revolutionized our understanding of human behavior – largely independent of each other. Here, we reconcile these two fields under a dual inheritance framework, offering a more nuanced understanding of the interaction between genes and culture. Going beyond typical analyses of gene–environment interactions, we describe the cultural dynamics that shape these interactions by shaping the environment and population structure. A cultural evolutionary approach can explain, for example, how factors such as rates of innovation and diffusion, density of cultural subgroups, and tolerance for behavioral diversity impact heritability estimates, thus yielding predictions for different social contexts. Moreover, when cumulative culture functionally overlaps with genes, genetic effects become masked, unmasked, or even reversed, and the causal effects of an identified gene become confounded with features of the cultural environment. The manner of confounding is specific to a particular society at a particular time, but a WEIRD (western, educated, industrialized, rich, and democratic) sampling problem obscures this boundedness. Cultural evolutionary dynamics are typically missing from models of gene-to-phenotype causality, hindering generalizability of genetic effects across societies and across time. We lay out a reconciled framework and use it to predict the ways in which heritability should differ between societies, between socioeconomic levels, and other groupings within some societies but not others, and over the life course. An integrated cultural evolutionary behavioral genetic approach cuts through the nature–nurture debate and helps resolve controversies in topics such as IQ.

1. Introduction

Business is booming in behavioral genetics. We're in the midst of a genome-wide association gold rush (Visscher et al., 2017). The availability of powerful computers and sequenced DNA of millions of people has led to an industrious search for single-nucleotide polymorphisms (SNPs) that correlate with a variety of psychological and behavioral traits (Harden & Koellinger, 2020; Horwitz, Lam, Chen, Xia, & Liu, 2019; Mills & Tropf, 2020). These range from memory capacity (Papassotiropoulos et al., 2011), cognitive ability (Coleman et al., 2019), and educational attainment (Lee et al., 2018) to moral attitudes (Brandt & Wetherell, 2012), political orientation (Hatemi et al., 2011), temporal discounting (Sanchez-Roige et al., 2018), socioeconomic status (Hill et al., 2016), temperament (Zwir et al., 2020), and happiness (Wingo et al., 2017). The significance threshold for discovering correlations is high (a typical threshold being $p < 5 \times 10^{-8}$; Fadista, Manning, Florez, & Groop, 2016) and there are claims that the curse of reverse causality has been lifted. As Plomin and von Stumm (2018) put it, genome-wide polygenic scores "are an exception to the rule that correlations do not imply causation in the sense that there can be no backward causation … nothing in our brains, behavior or environment changes inherited differences in our DNA sequence."

The last two decades have also seen a parallel revolution in cultural psychology and cultural evolution that has identified significant cultural variation in our psychology and behavior (Gelfand, 2018; Henrich, 2016; Henrich, Heine, & Norenzayan, 2010; Muthukrishna & Henrich, 2019; Muthukrishna et al., 2020; Nisbett, 2003). These range from fairness and prosocial norms (Henrich et al., 2010; Schulz, Bahrami-Rad, Beauchamp, & Henrich, 2019) and attribution of blame (Barrett et al., 2016) to perceptual style (Kitayama, Duffy, Kawamura, & Larsen, 2003), susceptibility to visual illusions (Henrich et al., 2010), visual perception more broadly (Lupyan, Rahman, Boroditsky, & Clark, 2020), numeric chunking (Domahs, Moeller, Huber, Willmes, & Nuerk, 2010), interpretation of linear and logarithmic numeric

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scales (Dehaene, Izard, Spelke, & Pica, 2008), neural correlates of reading (Bolger, Perfetti, & Schneider, 2005; Tan, Laird, Li, & Fox, 2005), event segmentation (Swallow & Wang, 2020), memory (Amici et al., 2019; Guida et al., 2018; Wang, 2021), spatial cognition (Majid, Bowerman, Kita, Haun, & Levinson, 2004), motor development (Karasik, Tamis-LeMonda, Adolph, & Bornstein, 2015), folkbiology (Medin & Atran, 2004; Waxman, Medin, & Ross, 2007), and personality (Gurven, von Rueden, Massenkoff, Kaplan, & Vie, 2013; Smaldino, Lukaszewski, von Rueden, & Gurven, 2019). Cultural evolution is part of a broader theoretical framework – dual inheritance theory (Boyd & Richerson, 1985; Cavalli-Sforza & Feldman, 1981)[1] – that incorporates genes, environment, culture, and learning to offer an explanatory and predictive framework for human psychology and behavior (Muthukrishna & Henrich, 2019). This body of research suggests that humans not only have a genetic inheritance from their parents, as do all animals, but also a substantial cumulative cultural inheritance from their societies, well beyond any culture found in other primates, birds, and whales (Dean, Vale, Laland, Flynn, & Kendal, 2014; although also see Whiten, 2019). Genes, culture, and the environment have often coevolved, shaping our species (Henrich, 2016; Laland, 2018).

The revolutions in behavioral genetics and cultural evolution have occurred largely independently of each other. Some attempts have been made to integrate the fields (e.g., Cavalli-Sforza & Feldman, 1973; Cavalli-Sforza, Feldman, Chen, & Dornbusch, 1982; Creanza, Kolodny, & Feldman, 2017; Feldman & Ramachandran, 2018; Laland, Odling-Smee, & Myles, 2010), with these efforts typically being launched by researchers in cultural evolution. As a result, cultural evolution has incorporated some aspects of behavioral genetics. Behavioral genetics, in turn, has been largely agnostic with respect to cultural evolution, which is perhaps understandable given the focus, size, and historical trajectory of the field. However, given the extensiveness of the cultural and culturally shaped environment, cultural evolution offers an important but typically missing complement to otherwise insightful methodological and empirical analyses within behavioral genetics (e.g., Brumpton et al., 2020; Turkheimer, Pettersson, & Horn, 2014; Young, Benonisdottir, Przeworski, & Kong, 2019).

The effect of culture in behavioral genetics is typically incorporated into a broad environmental term that is partitioned into between- and within-family variance components. While behavioral genetic research implicitly or explicitly incorporates an understanding of the way in which genetic evolution shapes genotypes, the environment – cultural or otherwise – is assumed to be exogenous or at best shaped by genes (Plomin, DeFries, & Loehlin, 1977; Rutter, 2007). The environment is treated as given, while the genome is at least implicitly understood through the lens of selection and function. But just as genetic evolution offers a systematic framework for understanding how distributions of alleles change over time, cultural evolution offers a framework for understanding how distributions of cultural traits change over time; both evolving in response to ecological, demographic, and social factors. Thus, an understanding of cultural change can provide insights into the structure and dynamics of the environmental component of phenotypic variation as well as their interaction with genes. Statistical and theoretical models of gene–environment interactions and correlations are well-trod territory (Plomin et al., 1977; Purcell, 2002; Rutter, 2007), but these models do not capture the cultural evolutionary dynamics of environmental change or the coevolutionary dynamics of gene–culture interactions and correlations.

Humans have long been evolving in ways that deeply intertwine genes and culture: A prominent example is that we have jaws too weak and guts too short for a world without controlled fire and cooked food (Aiello & Wheeler, 1995; Wrangham, 2017); we lack genes for fire-making or cooking, instead relying on culture to compensate. This kind of coevolutionary history renders some aspects of gene–culture or gene–environment interactions puzzling when taken outside of a dual inheritance framework. Such insights help with interpretation of data. For example, it is well understood and also statistically obvious that reducing environmental variation will increase heritability scores (Stoolmiller, 1999). What's less obvious is the way in which culture can either mask or unmask genetic variation, and the way in which cultural diffusion and innovation can increase or decrease heritability. It's similarly not obvious how to define a single society for the purposes of measuring heritability, without being able to identify cultural cleavages that can lead to Scarr–Rowe type effects (see sect. 4.1; Tucker-Drob & Bates, 2016; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Our goal is to offer a path to reconciliation between behavioral genetics and cultural evolution with new or complementary interpretations for various puzzles, such as differences in heritability between and within populations, differences in heritability across development, and the Flynn effect. In doing so, we hope to instigate a discussion that nuances common interpretations of the nature and nurture of human behavior.

Ryutaro Uchiyama is a Research Fellow at the NTU–Cambridge Centre for Lifelong Learning and Individualised Cognition (CLIC), based in Nanyang Technological University, Singapore. He obtained his Ph.D. in 2021 from the London School of Economics, and is a cultural and biological psychologist who has been trained in neuroscience, evolutionary biology, cognitive science, and spatial design in the UK, US, Canada, and Japan. His research revolves around the interaction between cultural evolution and cognitive plasticity.

Rachel Spicer is a Postdoctoral Fellow in the Department of Psychological and Behavioural Science at the London School of Economics, where she serves as the Chief Data Scientist of the Database of Religious History. Her research focuses on applying data science techniques to investigate cultural evolution using the database. Previously, she undertook a Ph.D. in Biological Science at the University of Cambridge and the European Bioinformatics Institute (EMBL-EBI), where she used metascientific methods to conduct research on the reusability of metabolomics data.

Michael Muthukrishna is Associate Professor of Economic Psychology and STICERD Developmental Economics Group Affiliate at the London School of Economics, CIFAR Azrieli Global Scholar at the Canadian Institute for Advanced Research, and Technical Director of the Database of Religious History. His research focuses on (1) human evolution, in particular the culture–gene coevolution of the human brain and (2) cultural evolution, focusing on cooperation, corruption, innovation, and the navigation of cross-cultural differences. He develops formal theories using mathematical and computational modeling and tests their predictions using experimental and data science approaches from psychology and economics. In 2021, the Association of Psychological Science (APS) awarded him their APS Rising Star award.

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2. Interpreting heritability

Heritability is an important metric in behavioral genetics. In its standard formulation, heritability refers to the proportion of phenotypic variance for some trait that is explained by genetic variance. Much has been written about the misunderstandings and pitfalls that commonly occur when interpreting heritability (e.g., Haworth & Davis, 2014; Lewontin, 1974; Visscher, Hill, & Wray, 2008; Vitzthum, 2003), and we will not recapitulate these arguments here except when necessary. Suffice to say, heritability is sometimes incorrectly treated as an index of the genetic basis of a trait, and hence as a measure of the relative contribution of nature vis-à-vis nurture.

Cultural evolutionary theory can contribute to our understanding of the interpretation of heritability by describing the processes of cultural transmission and cultural change. In standard treatments of heritability, phenotypic variance is partitioned into a component explained by genetic variance and a component explained by environmental variance. We will further conceptually partition this environmental component into a subcomponent whose distribution is shaped by cultural transmission (the cultural environment or culture) and another whose distribution is not (the ecological environment or ecology). For the purposes of exposition, we will treat these as separable, though of course this dichotomization is merely an approximation as any sharp separation between the two is in reality implausible (Laland et al., 2015).

2.1 An example: Skin pigmentation and UV

Before we unfold the complexities of interpreting the cultural evolution of genetic heritability, let's begin with a simple illustration that introduces some basic concepts in both behavioral genetics and cultural evolution: the heritability of cancers associated with skin pigmentation. Genes affect the level of skin pigmentation and propensity for tanning instead of burning (Crawford et al., 2017). These are ancestral adaptations to levels of ultraviolet (UV) radiation at different latitudes (Barsh, 2003; Sturm & Duffy, 2012). Darker pigmentation protects against high levels of UV radiation, such as near the equator. Lighter pigmentation enables vitamin D synthesis in low levels of UV radiation, such as at northern latitudes (Jablonski & Chaplin, 2010, 2017). It is important to get the correct amount of UV radiation – too much causes skin cancer, but too little causes vitamin D deficiency, which is associated with other health problems (Edlich et al., 2009; Garland et al., 2006).

Worldwide migration has led to people with skin pigmentation mismatched to the level of UV radiation: Australians with European ancestry have higher rates of skin cancer compared to Australian Aboriginals and other non-European populations (Australian Institute of Health and Welfare, 2016), and conversely, Europeans with African and South Asian ancestry have higher rates of vitamin D deficiency and associated afflictions (Cashman et al., 2016; Spiro & Buttriss, 2014). A gene by environment interaction (G × E) approach could measure how these mismatches affect the heritability of skin cancer or vitamin D concentrations, but not how we should predict these heritability estimates to change over time through cultural evolution, specifically through diffusion and innovation. Through cultural evolution, non-genetic adaptations evolve to compensate for genetic mismatches: Fairer Australians wear sunscreen, a hat, and covered clothing (Montague, Borland, & Sinclair, 2001),[2] while darker Europeans consume vitamin D supplements and vitamin D-rich or fortified foods (Spiro & Buttriss, 2014).

In this example, the challenges to measuring and interpreting heritability and understanding genome-wide association study (GWAS) results are, perhaps, more obvious than for many psychological traits. The heritability of skin cancer, for example, should be highest when there is more diversity of skin pigmentation (genes), more homogeneity of cultural practices (culture), and high UV radiation (ecology)[3]; see Figure 1. While ecology and genes may change to some degree (e.g., smaller hole in the ozone layer and immigration, respectively), cultural change can be particularly fast and potent – greater uptake of anti-skin cancer practices and technologies or new medical interventions for treating cancer. In some cases, cultural evolution is broadly predictable because of directionality – few are working on ways to *increase* rates of skin cancer. Of course, there may be other forces that work in the opposite direction, such as a tan becoming associated with attractiveness.[4] Here, it is easier to see that heritability is a function not only of genes, traits, and ecology, but also of a cultural environment that is evolving according to dynamics that can be understood. The environment of the genome is, therefore, not an inert backdrop against which genes should be evaluated, but rather, a moving reference frame that rapidly evolves in relation to both genes and ecology.

This example helps us understand four key points. The first of these is well understood by behavioral geneticists and the second is sometimes noted, but the third and fourth points are typically absent from these discussions because of the disconnect between behavioral genetics and cultural evolution. First, there is no overarching, one-quantity heritability of a trait to be discovered. There is no fixed answer to the question, "What is the heritability of skin cancer?" Second, this answer will depend not only on ecology, but also on culture and specifically on cultural diffusion and innovation – both of which can rapidly change and therefore rapidly change heritability estimates. Third, the diffusion and innovation are broadly directional.[5] Cultural diffusion of sunscreen, clothing, shade and sunglasses, and cultural innovation toward more effective screening and treatment of melanomas all work to reduce heritability estimates. In these cases, we expect a reduction in heritability because of the masking effect of the cultural trait. Were any of these an example of culture unmasking genetic effects, such as tanning salons that induce differential risk according to the skin pigmentation level, we would have predicted an increase in heritability. Insofar as a preference for avoiding some outcome outweighs other forces that tend to bring it forth, there will be a directional trend over time, similarly to directional selection in genetic evolution (Byars, Ewbank, Govindaraju, & Stearns, 2010; Sanjak, Sidorenko, Robinson, Thornton, & Visscher, 2018). Fourth, we might also expect the cultural response to be stronger where the ecological and cultural selection pressures are stronger – skin cancer mitigation in Australia but vitamin D supplementation in Northern Europe. Heritability or changes in heritability of these cases have not been tested to our knowledge, but the predictions are clear.

Like heritability, the question "Which SNPs are associated with skin cancer?" is similarly culturally dependent. In societies where sunscreen use is common, we expect SNPs associated with skin pigmentation to be less predictive of skin cancer compared to societies where this is not the case. Similarly, we would expect SNPs associated with antioxidant metabolism (Oskina et al., 2014) to be less predictive of skin cancer in societies whose foods are rich in antioxidants – such as in traditional Mediterranean cuisine (Visioli & Galli, 2001).

That heritability is affected by the environment is widely understood (Charmantier & Garant, 2005; Feldman & Ramachandran,

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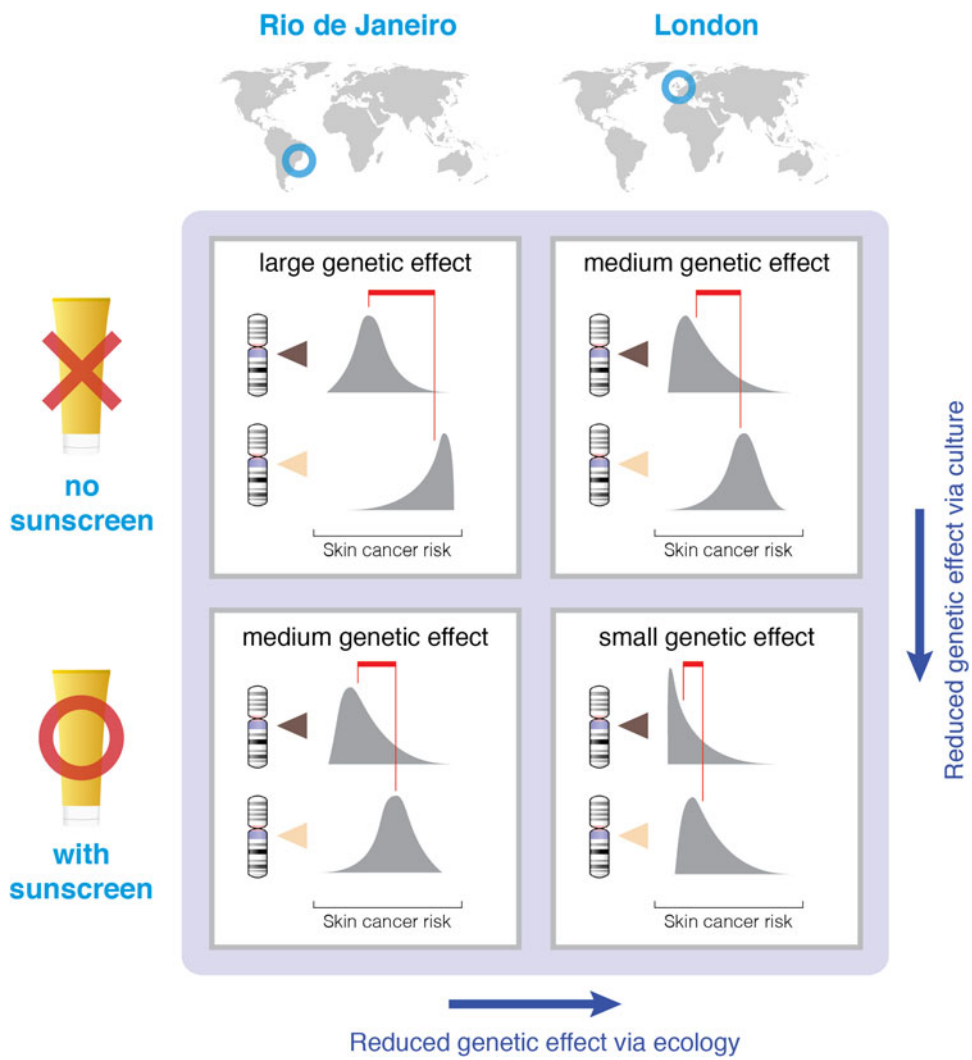

Figure 1. Illustration of the effect of sunscreen and geographic location on the effect size of a skin pigmentation gene with respect to skin cancer risk. The largest genetic effect should be found in societies that lack sunscreen and reside in locations with high levels of UV radiation (top-left square). Genetic effects should be reduced with either the introduction of sunscreen or residence in a lower-UV environment, both factors that mask the effect of skin pigmentation (bottom-left and top-right squares, respectively). The smallest effect should be found in societies that have both low UV and sunscreen (bottom-right square). Each cell represents a hypothetical scenario – if Rio and London did or did not have sunscreen. Chromosomes with dark indicators represent genes for strong pigmentation, and those with light indicators represent genes for light pigmentation. Gray distributions represent population distributions for skin cancer risk, and red lines point to the mean of each distribution.

2018; Hamer & Sirota, 2000; Haworth & Davis, 2014; Moore & Shenk, 2016; Tenesa & Haley, 2013; Turkheimer et al., 2014; Vitzthum, 2003). And researchers such as Lewontin and Feldman (Feldman & Lewontin, 1975; Lewontin, 1970; 1974) long ago described the fallacy of extrapolating heritability scores from one population to another. Their argument was made from the standpoint of gene–environment interactions: Genetic effects must be understood in the environmental conditions under which the genes are expressed. In this target article, we build on this rich body of research to launch a discussion of how the cultural environment changes over time and affects heritability – that is, the cultural evolution of genetic heritability (we schematically capture some of these key ideas in Fig. 2).

2.2 Cultural evolution shapes heritability

Assume that for a given society, we were able to collect comprehensive data on genetic effects across all relevant environmental variables that contribute to some trait. This would allow us to exhaustively map out the reaction norms (pattern of phenotypic expression across a range of environments) that specify expected phenotypic outcomes over the full range of extant genetic and environmental variation, thus setting up the conditions for a G × E analysis. But in a species like ours, extant environments are not necessarily a meaningful backdrop against which to judge genetic effects. Human environments have already been shaped over deep historical timescales by cumulative cultural evolution – functionally overlapping with genetic evolution (Richerson, Boyd, & Henrich, 2010) and can, therefore, obscure our interpretation of genetic evolution, unless properly accounted for. For humans, the environmental axes of a reaction norm analysis do not simply map out the space of environmental parameters that impact phenotypic outcomes; they rather map out the local and global peaks that have already been climbed by cultural evolution, which correspond to the many solutions to problems that have been discovered and refined over human history. Because cultural evolution enables faster adaptation compared

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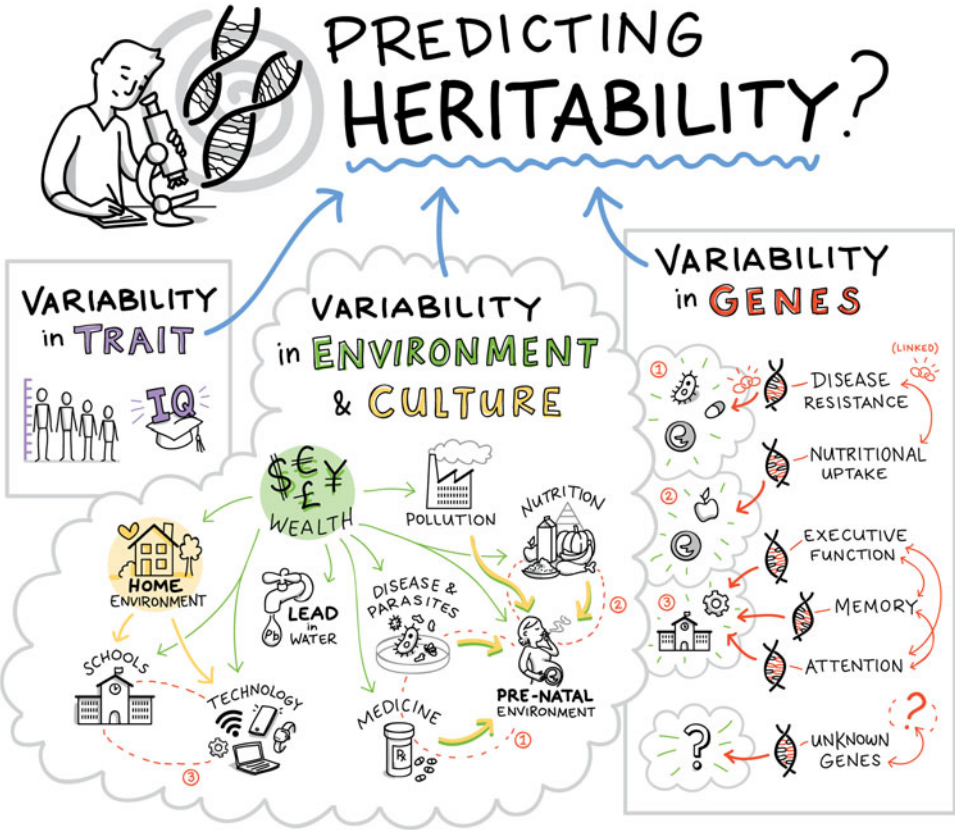

Figure 2. Genetic heritability is a function of variability in the phenotypic trait, variability in the environment, including the cultural environment, and variability in genes. Although heritability is often interpreted as a genetic effect, cultural evolution and diffusion can also systematically shape the variability of environmental variables, and thus heritability. Psychological and behavioral phenotypes are typically the outcome of a complex network of interactions that involve all these factors.

to genetic evolution, a substantial portion of our adaptations are built into our cultures rather than our genomes.

The human environment is deeply shaped by culture. As heritability is a function of both genetic and environmental variance, cultural evolution carries significant implications for the interpretation of heritability. This idea goes back to the beginnings of the cultural evolutionary paradigm. For example, two founders of the field of cultural evolution, Cavalli-Sforza and Feldman (1973), modeled the effect of parent-to-child ("vertical") cultural transmission on standard behavioral genetic estimates of genetic effects, showing that vertical transmission should inflate heritability estimates relative to a gene-only model. In contrast, here we focus on a different aspect of the interplay between culture and heritability, namely how oblique and horizontal transmission can impact heritability through processes of broad diffusion that systematically shape phenotypic distributions. We begin this discussion by describing the effects of cultural diffusion and innovation upon heritability, as well as the predictions we can make for differences in heritability between societies.

2.2.1 Cultural diffusion

In human societies, mechanisms such as conformist-biased learning (learning from the majority or plurality at a rate above population frequency; Boyd & Richerson, 1985; Cavalli-Sforza & Feldman, 1981; Muthukrishna, Morgan, & Henrich, 2016), payoff biases (e.g., learning from successful others; Boyd & Richerson, 1985; Cavalli-Sforza & Feldman, 1981), and norm enforcement (identifying norms and punishing norm violators; Chudek & Henrich, 2011) result in particular behaviors, beliefs, and norms disseminating widely across a society. To the degree that these cultural traits mask the effects of genes (such as in the skin cancer and vitamin D examples), heritability is reduced. In contrast, to the degree to which these cultural traits unmask or interact positively with genes without masking, heritability is increased.

One example that can illustrate both masking and unmasking through diffusion is genes that support the perception of lexical tone in tonal languages such as Cantonese and Yoruba (Dediu & Ladd, 2007; Wong et al., 2020). To the degree that tonal languages such as Cantonese or Yoruba diffuse in the population (and holding constant other genetic contributions), heritability of language ability would increase in a manner proportional to variation in these genes; to the degree that non-tonal languages such as Norwegian or Russian diffuse in the same population, heritability of language ability would decrease. As another example: It is known that fertility is predicted by genes (Zietsch, Kuja-Halkola, Walum, & Verweij, 2014), but Briley, Paige Harden, and Tucker-Drob (2015) report a large rise in the heritability of fertility in the United States over the mid-twentieth century, likely because of the increase in the variety of acceptable reproductive choices that was brought about by the diffusion of new social values. In this case, an increase in cultural variance unmasked the effect of genes associated with reproductive behaviors and preferences. Conversely, cultural or policy changes that

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reduce variation in reproductive practices – such as rigid child-bearing norms or a one-child policy – would be expected to mask the genetic effect.

The diffusion of cultural traits is not random and is, in fact, well studied within cultural evolution and elsewhere (Henrich, 2001; Muthukrishna & Henrich, 2016; Rogers, 2003). One mechanism for cultural diffusion that is important in industrialized societies is formal education. We use this example, because the effect of this particular cultural institution on heritability has been studied. Samuelsson et al. (2008) measured the heritability of reading and spelling test scores. Australian twins demonstrated a narrow-sense heritability of 0.84 in kindergarten and a similar score of 0.80 in Grade 1. In contrast, Swedish and Norwegian twins demonstrated a heritability of only 0.33 in kindergarten, rising to 0.79 in Grade 1. Heritability was at the same level in both the Australian and Scandinavian children in Grade 1, but not in kindergarten. Why? Cultural diffusion of literacy. Australian children begin receiving compulsory literacy instruction in kindergarten, while in Scandinavia the kindergarten curriculum emphasizes social, emotional, and esthetic development – literacy instruction only begins in Grade 1. Here, we see the effect of national curriculum policy differences affecting cultural diffusion of literacy and thus heritability, in a case where the cultural trait interacts with genes. Australian kindergarteners are exposed to standardized environmental input and much of the remaining variation in reading ability is explained by genetic differences, whereas for the Swedish and Norwegian kindergarteners, variation in the amount of reading instruction received at home is much larger than any genetic differences. In line with this interpretation, Samuelsson et al. (2008) show that the boost in heritability among the Scandinavian children was also accompanied by an almost equivalent decrease in phenotypic variance attributed to the common (home) environment, which would include home instruction.

If we were to assess the genetic basis of literacy skill in schoolchildren without accounting for the impact of their particular educational curricula on cultural diffusion and environmental variation, we would be subjecting ourselves to a selection bias, with no idea of the magnitude of this bias. This would distort our understanding of the generalizability of our finding to samples that have undergone different educational curricula, and even more so to those with different levels of educational attainment. Note that even the literacy instruction provided in the home environment is already shaped by cultural evolution, both in terms of the content being transmitted (reading and spelling), and the structures that are transmitting (family organization in Western countries; Henrich, 2020; Schulz et al., 2019). In societies that produce literate children, culture impacts the heritability of literacy from the moment that variation in this trait emerges in development, virtually sealing off the possibility of assessing "baseline" heritability without cultural interference, even at the very start of life (e.g., children born in literate societies are typically surrounded by writing and literate adults). Heritability is a composite measure that captures both genetic and cultural effects, and without knowledge of the cultural context, it is difficult or impossible to judge what is being measured. When we say that the heritability of reading among Scandinavian children jumps up to 0.79 when they enter Grade 1, this measurement reveals just as much if not more about the disseminative power of modern schooling than it does about the genetic basis of literacy.

Looking toward a broader social context, several insightful studies have shown that the heritability of educational attainment increases with equality of opportunity. We see evidence for this within countries over time (Heath et al., 1985), within and between countries over time (Engzell & Tropf, 2019; de Zeeuw, de Geus, & Boomsma, 2015), and within countries following a major policy change (Colodro-Conde, Rijsdijk, Tornero-Gómez, Sánchez-Romera, & Ordoñana, 2015; Rimfeld et al., 2018; Ujma et al., 2022). Although there is also some evidence going in the other direction (Silventoinen et al., 2020), the overall pattern appears to be that diffusion of educational opportunity results in genes explaining a relatively greater proportion of variance in educational attainment. These studies serve as robust demonstrations of how heritability can be boosted by changes in social context broadly, and diffusion of particular forms of environmental exposure more specifically. Although these patterns are broadly consistent with our thesis, both the consistent and inconsistent results may obscure cultural structuring, such as the hidden cluster problem and cultural Simpson's paradox, as we discuss in section 3.

2.2.2 Cultural innovation

The human capacity for cumulative cultural evolution (Dean et al., 2014; Henrich, 2004a) ensures that in our societies, the diffusion of extant cultural traits goes hand-in-hand with the continual generation of new cultural traits. Whether emerging through serendipitous discovery, iterated trial-and-error, or recombination of ideas, some portion of new technologies and behaviors manages to spread across a society, displacing other competing variants. The dynamics of innovation are a significant component of the cultural evolutionary framework (Henrich, 2004a; Muthukrishna & Henrich, 2016).

Cultural evolution predicts that societies will vary in their rate and type of cultural innovation (e.g., number of patents and incremental vs. revolutionary invention) because of differences in, for example, the size and interconnectedness of their social networks (sociality), effectiveness of cultural transmission (e.g., education), and tolerance for diversity; for a review see Muthukrishna and Henrich (2016). Tolerance for diversity can drive differences in heterogeneity. Some societies such as Pakistan and Indonesia maintain relatively low levels of cultural heterogeneity, whereas others such as Brazil and Australia maintain relatively high levels of heterogeneity. One metric that can serve as a proxy for this tolerance is *cultural tightness/looseness*, which represents the degree to which societies tolerate deviation from social norms, and is expected to correspond to their rate of innovation (Gelfand, 2018; Gelfand, Nishii, & Raver, 2006; Gelfand et al., 2011). Societies that are more tolerant of cultural variation allow for more individual-level exploration, and if the best of these cultural mutants can be selectively incorporated into the mainstream, those societies will tend to undergo higher rates of increase in cultural complexity (Henrich, 2004a). Cultural variation is the engine of cultural change. Aside from variables such as cultural looseness, policies such as social safety nets and forgiving bankruptcy laws can also create the incentive structure for promoting exploration and innovation (Muthukrishna & Henrich, 2016).

The initial spread of an innovation will be tied to an increase in environmental variance if that innovation is disrupting an earlier, relatively homogeneous state. To the degree that innovation is masking genes, heritability will begin to decrease. To the degree that innovation is unmasking genes, heritability will begin to increase.

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2.2.3 Predicting differences in heritability across societies

Over long historical durations, cultural evolution tends toward a compressive environmental effect, as ecological problems are solved and more favorable environmental conditions spread. But on shorter timescales, diffusion and innovation have opposing effects on environmental variance. Although indoor plumbing, antibiotics, and formal education have diffused to the point of being ubiquitous in the Western world, new innovations are also constantly emerging. During their early spread, such innovations increase environmental variance and between-group differences, thereby bringing forth new forms of diversity and inequality. But, as these new traits diffuse further, some of them become gradually established within the cultural corpus, reducing environmental variation through homogenization. The dynamics of diffusion and innovation thus differ in their effects, although the magnitude and direction of these effects also depend upon whether the trait in question masks or unmasks genetic variation. Moreover, across societies, the balance between diffusion and innovation differs as well.

If we imagine a society in which innovations are rapidly diffused across the whole society shortly after they emerge, the society will have a low level of cultural variation at most times. This is a society in which a high rate of diffusion quickly overpowers the environmental variance-increasing potential of cultural innovation. Now imagine a contrasting society in which innovations take a much longer time to diffuse. Perhaps, the society is more culturally clustered with less interaction between individuals in different subgroups, or perhaps there is a subset of individuals who are well-connected with the inventor and become early adopters but the novel trait diffuses only gradually beyond this inner circle. This would be a culturally unequal society that is characterized by high environmental variability at any given point in time. This kind of stunted diffusion may suggest cultural clustering, with relatively dense connections within subgroups and relatively sparse connections between subgroups. In such societies, even highly useful forms of cultural knowledge may not easily permeate social barriers, and the waves of environmental change are correspondingly "loose." These barriers are not necessarily ethnic boundaries: They could be organized around class, wealth, occupation, political alignment, religion, or incidental geographic layout. A society may be clustered for reasons that stem from within the society itself, or it could simply be a matter of nominal mismatch between the political boundaries that we use to refer to a society and the actual organization of cultural groups. Greater differential clustering can lead to a cultural Simpson's paradox (discussed in sect. 3.4).

We can derive testable hypotheses from these dynamics: Culturally homogeneous societies will have higher heritability for culturally transmissible phenotypic traits[6] compared to culturally diverse or clustered societies. We model this prediction in the Appendix. One way to measure cultural diversity would be to use cultural tightness/looseness as a proxy (Gelfand et al., 2006, 2011). One way to measure cultural clustering would be to use the cultural fixation index (CF_{ST}) as a measure of cultural distance between groups within a society (Muthukrishna et al., 2020) – we discuss this in more detail in section 3.2. Whatever the measure, homogeneity of cultural traits should be associated with higher heritability on average, but with respect to specific traits, the relationship between homogeneity and heritability will also depend upon the genetic masking or unmasking effect of the trait.

For cultural traits that neither mask nor unmask genetic effects, heritability will generally be higher in culturally homogeneous societies than in culturally diverse societies because of the reduced environmental variance that is a consequence of behavioral uniformity, as described above. For cultural traits that unmask genes, heritability will be higher overall compared to traits that neither mask nor unmask, and on average higher in homogeneous societies than in diverse societies. For cultural traits that mask genes, heritability will be lower overall compared to traits that neither mask nor unmask, but whether it is higher in homogeneous or diverse societies will depend upon which has the stronger effect: genetic masking or the reduction in environmental variance accompanying homogeneity. If the effect of genetic masking is stronger, heritability will be lower in culturally homogeneous societies than that in culturally diverse societies; if the reduction in environmental variance is stronger, heritability will be higher in homogeneous societies than that in diverse societies.

Thus, heritability estimates reflect cultural evolutionary dynamics. Researchers such as Harden (2021) have highlighted how heritability can be used as a tool for measuring useful characteristics across environments such as cross-national differences in social opportunity. These interpretations are consilient with our approach, but are limited by treating the environment as exogenous rather than modeling its dynamics and interactions. Such complexities in the role of the cultural environment have historically been obscured, because, in part, of a number of methodological and epistemological problems that encumber standard behavioral genetic analysis.

3. Problems that obscure the effect of culture

The effects of cultural evolution on our understanding of heritability are complex, but we hope that at least the significance of this relationship has now become more obvious. At the very least, we hope this target article will spark a vibrant discussion of the role of cultural evolution in behavioral genetics. There are several features of behavioral genetic methodology that tend to obscure the effect of culture and cultural evolution. Here, we will discuss three of these problems – the western, educated, industrialized, rich, and democratic (WEIRD) sampling problem, the hidden cluster problem, and the causal locus problem – and then describe a cultural Simpson's paradox that emerges at their junction. Various aspects of these problems have been discussed in prior literature, but we will focus on how the problems specifically interact with the detection and interpretation of effects stemming from cultural evolution. Clarity regarding these issues will be a first step toward integrating the cultural evolutionary framework with behavioral genetics.

3.1 WEIRD sampling problem

Behavioral genetics suffers from its own variant of the WEIRD people problem, which was originally raised in the field of experimental psychology (Apicella, Norenzayan, & Henrich, 2020; Henrich et al., 2010). The WEIRD people problem refers to the vast over-representation in the published studies of individuals from developed Western countries, who are similar in their cultural history, social values, and standards of living. Just as behavioral experimental samples are psychologically WEIRD, behavioral genetic samples are both genetically and culturally WEIRD. This results in a WEIRD sampling problem that limits the variation required to make sense of genetic effects. This problem involves both genetic and cultural restriction of range, and we will discuss each in turn.

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3.1.1. WEIRD genetics

A comprehensive meta-analysis that claims to contain essentially all twin studies published between 1958 and 2012 (Polderman et al., 2015) reveals that 94% of sampled twin pairs were from Western populations. The United States, United Kingdom, and Australia alone accounted for almost 60%, and Nordic countries accounted for another 25%. Of the non-Western countries (6%), two-thirds (4%) are from northeast Asia – specifically, China, Japan, South Korea, and Taiwan, countries that are not Western, but have most of the remaining letters of the WEIRD acronym. The remainder of the world, representing the vast majority of the human population, accounts for only 2% of the dataset.

GWASs too suffer from a myopic focus on WEIRD genomes (Need & Goldstein, 2009; Popejoy & Fullerton, 2016; Sirugo, Williams, & Tishkoff, 2019). As of 2017, 88% of samples in GWASs were of European ancestry (Mills & Rahal, 2019).[7] Paralleling the twin studies data, 72% of participants were recruited from just three countries – United States, United Kingdom, and Iceland – with nearly 20% of the remainder being recruited from Japan, China, and South Korea.

Polygenic scores do not translate well across ancestry groups (Bitarello & Mathieson, 2020; Curtis, 2018; Guo et al., 2021; Kim, Patel, Teng, Berens, & Lachance, 2018; Martin et al., 2017, 2019). For example, European ancestry-derived polygenic scores have only 42% of the effect size in African ancestry samples (Duncan et al., 2019). From a cultural evolutionary perspective, this is not unexpected given the cultural environment, coevolution between culture and genes, and cultural differences between populations.

Polygenic scores are also highly sensitive to inadequately controlled population stratification (Berg et al., 2019; Morris, Davies, Hemani, & Smith, 2020; Sohail et al., 2019). Even within a single ancestry group, the predictive accuracy of polygenic scores can be dependent on age, sex, and socioeconomic status (Mostafavi et al., 2020) – this too, from a cultural evolutionary perspective, is not unexpected given the cultural variation that exists within a population (Muthukrishna & Henrich, 2019; Muthukrishna et al., 2020). Similarly, the SNPs that contribute to the variance of a trait are different in different populations (Akiyama et al., 2019; Gurdasani et al., 2019; Pemberton et al., 2018; Rotimi et al., 2017) and it is difficult to disentangle the genetic, environmental, and cultural contribution to differing polygenic scores between populations (Rosenberg, Edge, Pritchard, & Feldman, 2019). Recent projects have aimed to capture a greater degree of global human genetic diversity (e.g., Simons Genome Diversity Project, Mallick et al., 2016; the exome analysis of Lek et al., 2016; and the GenomeAsia project, Wall et al., 2019), but we are far from proportionately representing the genetic diversity of the global population.

3.1.2. WEIRD culture

When we restrict the scope of genetic samples, the cultural environment against which genetic effects are evaluated also becomes skewed, and this greatly reduces the interpretability of genetic effects. Because of a combination of cultural-group dynamics (Chudek & Henrich, 2011; Henrich, 2004b; Richerson et al., 2016) and cumulative cultural evolution (Dean et al., 2014; Henrich, 2004a), the human species is characterized by large amounts of cultural and hence environmental variation between societies, which exceeds genetic variation by orders of magnitude (Bell, Richerson, & McElreath, 2009). The WEIRD countries that are over-represented in genetic samples are clustered together along multiple cultural dimensions (Hofstede, 2001; Inglehart & Welzel, 2005; Muthukrishna et al., 2020), and are perhaps an extreme unrepresentative outlier on many psychological and behavioral measures, with these countries registering the highest scores for traits such as individualism, analytical thinking, and prosociality toward strangers, and the lowest scores on opposite constructs such as collectivism, holistic thinking, and prosociality toward relatives but not strangers (Henrich, 2020; Henrich et al., 2010; Muthukrishna et al., 2020; Schulz et al., 2019).

WEIRD societies are also roughly aligned on basic environmental factors such as technology, civil infrastructure, healthcare, schooling, and transportation, with typically advanced levels in each of these. Within each of these countries as well, inequality is low compared to non-WEIRD countries, as can be inferred from the comparatively low Gini coefficients of Western countries, with the United States being somewhat of an unequal outlier (Selita & Kovas, 2019).

Just as this restricted cultural range limits our understanding of human psychology (Henrich et al., 2010), it also limits the inferences that can be made about human genetics. Behavioral geneticists understand that genes can have different effects under different environmental conditions (Assary, Vincent, Keers, & Pluess, 2018; Moffitt, Caspi, & Rutter, 2005), but there is less appreciation of how those environmental conditions and interactions change through cultural evolution. Cultural evolution can mask or unmask genetic effects, such that gene function becomes confounded with the effect of the cultural environment in a manner that is dependent on the specifics of the underlying dynamics. Cutting through this confound requires a theoretical understanding of the cultural dynamics as well as empirical data on genetic effects across a wide variety of cultural environments. The WEIRD sampling problem, therefore, harms accurate inference of genetic effects.

3.2 Hidden cluster problem

Despite this severe restriction of range among behavioral genetic samples, these samples may also paradoxically be too inclusive. This is because of the presence of cultural clusters that introduce population structure into genetic samples, but which remain hidden to standard behavioral genetic methods. A cultural evolutionary approach can help reveal environmental structure that does not necessarily map onto conventional demographic groupings.

3.2.1 Cultural clustering

Whether the method is a twin study or GWAS, behavioral genetic studies typically draw their data from databases such as twin registries or biobanks, whose coverage spans some circumscribed geographic range such as a country or a subnational region. Because these samples trace over political or administrative boundaries, they may not match up with the actual structure of environmental variation, which will often be dependent upon the organization of cultural clustering. Culture, generally, tends to agglomerate, such that groups of individuals who are bound together by dense links of cultural influence share a substantially greater number of features of their cultural environment than do individuals who belong to different groups (Richerson et al., 2016). As has been discussed extensively in the cultural evolutionary literature, this in-group similarity is generated and maintained by processes such as conformist learning (Boyd & Richerson, 1985), norm enforcement (Chudek & Henrich, 2011; Fehr &

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Fischbacher, 2004), symbolic markers of in-group membership (Boyd & Richerson, 1987), and cultural-group selection (Henrich, 2004b; Richerson et al., 2016). The structure of environmental variation is, thus, shaped by networks of cultural interaction, and the topology of these networks is assembled over time by cultural-group dynamics and cultural evolution.

A highly clustered population is one that can be easily partitioned into multiple subgroups whose members share among each other cultural traits that are substantially different from those found in other subgroups. When we know the underlying social network, network clustering algorithms (Emmons, Kobourov, Gallant, & Börner, 2016) can help identify these clusters. When we have measures of a range of cultural traits, we can see the effects of clustered cultural influence by measuring the cultural distance between subgroups, for example by using CF_{ST} (Muthukrishna et al., 2020). CF_{ST} applies the "fixation index" (Cavalli-Sforza, Menozzi, & Piazza, 1994) to the World Values Survey of cultural beliefs and behaviors (Inglehart et al., 2014), and can quantify the cultural differentiation between any two groups. When applied to subgroups within societies, this measure can tell us, for example, the degree to which Singaporean Catholics differ from Singaporean Protestants, thus identifying the degree to which a nation state or other political grouping contains large clustering (note that this is different than diversity – a country may be diverse, but uniformly so, showing no large clustering).

Cultural clusters are typically created by barriers that impede cultural interaction, such as topography (e.g., mountain ranges or bodies of water separating populations) or cultural conflict (e.g., conflicting religious beliefs). Mutually unintelligible languages are an example of a social barrier that impedes cultural and even genetic mixing. Thus, the density of languages within a population will also predict clustering. A topographically fractured country like Papua New Guinea with its 839 often mutually unintelligible languages is likely to be a much more clustered population than an equally populated but less multilingual society like Austria. Populations can also become clustered because of more extrinsic factors. Countries that grew through recent immigration, such as Canada, are likely to have more clustering than countries that are characterized by relative cultural homogeneity, such as Japan (Fearon, 2003). Countries whose borders are drawn arbitrarily with respect to the geographic arrangement of cultural groups, for example as a product of past colonial administration (many countries in Africa), are also likely to have high clustering (Michalopoulos & Papaioannou, 2020). Note the parallel between the present argument and assortative mating (Schwartz, 2013): whereas assortative mating results in phenotypic clustering because of reproduction, cultural transmission results in phenotypic clustering because of segregated learning.

Moreover, cultural clusters may cut through divisions of society that we don't always think of as being "cultural," such as stratification by social class or wealth. The strength of this stratification regulates the amount of within-stratum versus between-stratum cultural interaction. Although this picture may be complicated by factors such as prestige bias engendering asymmetric influence from upper to lower classes (Henrich & Gil-White, 2001), such forms of social hierarchy can create clusters. These examples show that cultural clustering is not necessarily bound by geographic contiguity. Although spatial proximity is a good predictor of cultural influence, cultural influence may also be decoupled from geography, particularly with modern forms of mass media and information technology (Anderson, 2006; Martin & Yurukoglu, 2017). In section 4.1, we discuss in more detail how cultural clustering may contribute to the phenomenon of differential heritability across socioeconomic levels.

In sum, the scope of a genetic sample is often defined by political and administrative boundaries, but these boundaries may be mismatched to the organization of cultural clusters within a society, and the extent of this mismatch will vary greatly across populations. So, in addition to measuring cultural variation (diversity) using indices such as cultural looseness, we need to also measure cultural clustering ("segregated diversity") to fully understand the environment. Once we are able to do this, the next intellectual step would be to achieve an understanding of the superimposition of cultural and genetic clusters as well as their interaction.

3.2.2 Contrasts between cultural and genetic clustering

Behavioral geneticists have devoted much effort to the study of genotypic clustering, usually referred to as population stratification or population structure. Populations can become stratified in the presence of genetic variation arising from systematic differences in subpopulation ancestry or from other forms of structured, non-random mating (Brumpton et al., 2020). This kind of genetic clustering can introduce spurious associations between genotypes and traits. As such, researchers continue to develop an array of methods for dealing with this problem (Hellwege et al., 2017; Price, Zaitlen, Reich, & Patterson, 2010).

Genetic clustering and cultural clustering are linked – a well-known example is the close alignment of genes and languages in historical populations (Cavalli-Sforza, 2001; Cavalli-Sforza, Piazza, Menozzi, & Mountain, 1988; Pagani et al., 2012; Tishkoff et al., 2009). This gene–language alignment occurs because languages, similar to genes, have traditionally been transmitted vertically – that is, from parents to children or within the family. This is because of critical periods for language learning that close as early as 6 months for phonology (Kuhl, Williams, Lacerda, Stevens, & Lindblom, 1992) and year 7 for grammar (Johnson & Newport, 1989), because of plasticity in the relevant cortical networks being progressively staunched by "molecular brakes" (Werker & Hensch, 2015). Early learning of this kind is primarily dispensed by parents or other immediate caretakers (Hewlett, Fouts, Boyette, & Hewlett, 2011; Kline, Boyd, & Henrich, 2013), and it is unsurprising that genes and languages should be largely aligned in reconstructions of population history.[8]

However, beyond early childhood, children become increasingly exposed to other children from different families as well as to non-kin adults, and the range of sources for cultural learning widens accordingly: a two-stage model of cultural learning (Kline et al., 2013). Because of transmission through these horizontal (i.e., age-peers) and oblique (i.e., non-parent adults) channels, cultural learning becomes untethered from strict vertical transmission and extends laterally across genetic lineages, similar in structure to horizontal gene transfer that is prevalent in bacteria and archaea (Soucy, Huang, & Gogarten, 2015). Similar to horizontal gene transfer, horizontal cultural transmission is rapid, because of it being unconstrained by the generational cycle of vertical transmission. Oblique and horizontal transmission play a large role in cultural diffusion both within and across extant small-scale societies (Henrich & Henrich, 2010; Hewlett et al., 2011; Kline et al., 2013), just as they do in large-scale industrialized societies where innovation, knowledge, and practices across domains such as technology, art, education, political systems, and supernatural beliefs commonly diffuse horizontally (Rogers,

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2003). Indeed, with only vertical transmission, culturally evolution as it is currently understood would not work (Enquist et al., 2010). The importance of oblique and horizontal transmission in human cultural evolution may have been amplified by adaptations such as the timing of weaning relative to brain maturation, which is substantially earlier in our species than would be expected on the basis of our close primate relatives, thereby expediting the human child's exposure to cultural models other than parents (Finlay & Uchiyama, 2020; Hawkes & Finlay, 2018).

Because, in part, of horizontal transmission, the diffusion of culture can occur much more rapidly than the diffusion of genes, or "demic" diffusion. In historical populations, the speed of demic diffusion has been limited by rates of reproduction and migration (Ammerman & Cavalli-Sforza, 1984; Fort, 2012). Horizontal transmission is the diffusion of ideas rather than people and has no such limits; cultural clusters are, therefore, more fluid and malleable than genetic clusters. For example, if archeological data show cultural commonality between two contemporaneous populations but genomic analyses suggest disjunction, we can often infer that the shared cultural traits between the two groups were because of cultural transmission rather than migration or admixture (e.g., Fu et al., 2016; Olalde et al., 2018).

Cultural clusters are not only more fluid than genetic clusters, but they also explain intergroup differences better than genes do. Cultural distances between neighboring countries are an order of magnitude larger than genetic distances (Bell et al., 2009). Among small-scale societies in Kenya, pastoral clans are differentiated by cultural traits considerably more than they are by genes, and cooperation among these clans is predicted by their cultural – but not geographic – distance to each other (Handley & Mathew, 2020). These findings are consistent with the predictions of *cultural-group selection* (Henrich, 2004b; Richerson et al., 2016), particularly in how competition between cultural trait-groups helps explain the evolution of human cooperation (Henrich & Muthukrishna, 2021). Cultural-group selection requires large differences between groups of cultural traits relative to differences within groups (Apicella, Marlowe, Fowler, & Christakis, 2012; Fehr & Fischbacher, 2003).

Reconciliation between cultural evolution and behavioral genetics requires an update in the way we think about culture. An ethnolinguistic conception of culture that revolves around vertical transmission roughly in alignment with genes is insufficient. For example, linguistic, ethnic, religious, or caste boundaries can create genetic population structure through endogamous marriage practices, whereby individuals marry others who are culturally similar. In the modern world, such assortative mating may be exaggerated as people become more mobile and better connected, making it easier to mate with partners who share cultural traits across many dimensions. Efficiency of assortment may be boosted by cultural matching through dating apps, stretching the tails of the genetic distribution. These are all examples of human culture but they represent only one particular aspect of it, namely cultural effects that channel mating and thereby regulate genetic clusters. This aspect of culture is what appears as signal in population genetic data and is thus often the focus of attention for geneticists insofar as culture is concerned. This ethnolinguistic conception of culture that reduces culture to its effects on genetic clustering is inadequate for comparing genetic effects to environmental effects, given the many non-reproductive cultural effects that shape human phenotypes and environments. Indeed, an understanding of cultural clustering may elucidate novel aspects of genetic population stratification.

Abdellaoui and colleagues (Abdellaoui, Verweij, & Nivard, 2021; Abdellaoui et al., 2019) offer evidence for geographic clustering of polygenic scores in the United Kingdom that is statistically independent of genetic ancestry, and explained instead by recent migration. For example, coal mining regions experienced "brain drain" and other forms of trait-conditional departure because of changes in the UK economy over the twentieth century. The outcome is detectable in genetic assortment at both the source and destination regions. This is an example of gene–environment correlation, where the environmental factor is a macroeconomic variable with a relatively clear geographic distribution. But this kind of genetic sorting is likely to occur also with respect to environmental factors that are shaped by cultural transmission yet not as readily localizable along geography or other conventional social scientific dimensions. Just as we can use genomic methods to reconstruct aspects of cultural history (e.g., Dai et al., 2020; Peter, Petkova, & Novembre, 2020; Petkova, Novembre, & Stephens, 2016), a research strategy that builds upon cultural transmission may help us reconstruct and even predict the emergence of genetic clusters that coalesce around – and perhaps feed back onto – cultural clustering of human behavior, psychology, and environments.

3.3. Causal locus problem

The hidden cluster problem described the implicit complexity that exists within social groupings, which can hinder robust inference unless parsed properly. Below, we discuss the causal locus problem, which refers to a source of complexity that exists within the space of functional organization in which genes and culture interact. Inference is hindered here when we view culture as an unstructured exogenous variable, when in fact it is a constructive system that accumulates functional adaptations in a directed manner over time.

3.3.1 Genes that break and genes that make

The more complex a system, the more ways it can fail. Take the history of lighting: Compared to the two ways in which a wood-fueled fire can be extinguished (smothering and exhaustion of fuel), there are seven known failure modes for a fluorescent bulb and more than 30 for the newer LED bulb (de Groot, Vos, Vogels, & van Driel, 2013). A faulty rubber O-ring caused the space shuttle Challenger to explode, and a severed fiber-optic cable knocked out internet access for a large swath of people across India and the Middle East. There is a fundamental asymmetry between the identification of elements that support a system and those that undermine it. A well-functioning system is the product of a design process that has solved many problems and closed many paths that do not work. For such a system, "something going wrong" can be caused by singular aberrations, but "going right" and "going well" are properties of the integrated system as a whole. The notions of success and failure in this context, therefore, point to very different things. For complex functional systems such as machines and organisms, it is easier to identify ways to break the system than ways to explain or improve it.

Gene function can be viewed through this lens. Organisms are the outcomes of complex, emergent interactions involving many genes and their surroundings (Davies, 2014), but there are many ways these interactions can go wrong. It is easier to identify deleterious genetic mutations than beneficial mutations, as deleterious mutations are more common. The space of failure is larger

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than the space of success, making genes that break more detectable than genes that make. For example, a single mutation can cause Mendelian disorders such as cystic fibrosis and Huntington's disease, but no single mutation creates genius. Over 1,000 genes have been linked to intelligence (Davies et al., 2018; Lee et al., 2018; Savage et al., 2018). Each gene only explains a miniscule fraction of variation in intelligence, and the causal mechanisms are unlikely to be straightforward (Gottlieb, 2003; Turkheimer, 2000). In contrast to these genes that make, the causal mechanisms behind single-gene mutations that cause intellectual disability – for example, *BCL11A* (Dias et al., 2016), *PHF8* (Bathelt, Astle, Barnes, Lucy Raymond, & Baker, 2016), *ZDHHC9* (Schirwani, Wakeling, Smith, Study, & Balasubramanian, 2018) – are relatively well understood.

This spectrum of localizability ranging from Mendelian to polygenic to "omnigenic" traits (Boyle, Li, & Pritchard, 2017) has been discussed extensively, but its interaction with cumulative culture has not sufficiently been appreciated. We have known for a long time that increasing nutrition (Lynn, 1990; Stoch, Smythe, Moodie, & Bradshaw, 1982), improving schooling (Ceci, 1991; Davis, 2014; Ritchie & Tucker-Drob, 2018), and removing parasites (Jardim-Botelho et al., 2008) have positive effects on IQ. None of this is surprising, but it means that in a society where parasite infection is kept under control, we would not notice that parasite status correlates with intelligence, because of a lack of sufficient variation in parasite load. For the same reason, a correlation between lead exposure and IQ (Needleman & Gatsonis, 1990; Wasserman et al., 1997) will not be revealed in a society where lead is not a problem. And by corollary, genes that provide protection against malnutrition, parasites, or pollution would only be positively associated with intelligence in environments where these insults occur. In environments where these insults have been removed, the same genes would not be associated with intelligence, and can even be deleterious, as in the well-known example of sickle cell trait (Elguero et al., 2015). Similarly, alleles that protect against parasite infection (Carter, 2013) or lead poisoning (Onalaja & Claudio, 2000) will be predictive of IQ only if the environmental risk factors are present in sufficient quantities.

3.3.2. Cumulative culture masking genes

Our living conditions have not always been the way they are today. Just 200 years ago, 89% of humanity lived in extreme poverty (Ravallion, 2016), 88% were illiterate (van Zanden, Baten, Mira d'Ercole, Rijpma, & Timmer, 2014), and 43% of children died before they were 5 years old (Gapminder, 2020). Conditions have rapidly improved: rates of extreme poverty are now 10%, illiteracy is down to 14%, and deaths before 5 years of age are now 4% (UNESCO Institute for Statistics, 2013; World Bank Group – International Development, Poverty, & Sustainability, 2020). Of course, even if most are now better off, our world still suffers from immense global inequality. Given this restriction in historical range combined with the WEIRD sampling problem, it is not clear how well the genetic effects that have been cataloged thus far can be generalized beyond the particular cultural and temporal contexts in which they were studied. And even if this generalizability issue is acknowledged in principle, the bounds on generalizability are unknown.

Genes can be functionally masked by cumulative cultural evolution, and we expect that this masking is extensive and systematic. Cultural masking may help explain the limited portability of polygenic scores across populations (Kim et al., 2018; Martin et al., 2019, 2017). There is nothing too mysterious about this phenomenon: It's what happens when there are multiple evolutionary systems operating within a shared space of biological function. To build upon an example discussed by Deacon (2003): Vitamin C is an essential nutrient and its acquisition is thereby an essential biological function. Endogenous synthesis of vitamin C requires a gene called *GLO*, and *GLO* is present across most of the animal kingdom. But, because vitamin C synthesis is metabolically costly, the gene is inactive in some species that have access to sufficient quantities of the nutrient in their diets (Drouin, Godin, & Page, 2011). These include taxa such as teleost fishes, guinea pigs, many bats, some passerine birds, and anthropoid primates, that is, monkeys and apes (Chatterjee, 1973). Anthropoids, for instance, occupy a frugivorous niche, and fruits often contain sufficient vitamin C. Here, gene function is offloaded onto environmental resources. In turn, this offloading has behavioral implications. If a species becomes dependent on its environment ("auxotrophic") for vitamin C, both its behavioral range and evolutionary trajectory become constrained by the availability of the nutrient. Humans are a nice example of this. As our species migrated across the planet, we found ourselves in environments where vitamin C was in short supply. A deficiency of vitamin C causes scurvy – the bane of seafarers until the trial-and-error discovery that certain food items such as sauerkraut and citrus could prevent ships from being packed with tired, bleeding, toothless, and eventually dead sailors (Lamb, May, & Harrison, 2017).

Other species have other ways of obtaining vitamin C from their environments, and once these alternative pathways are established, the function of *GLO* becomes masked. A functionally masked gene is removed from the selection pressures that brought it about in the first place, and can therefore be culled from the genome, as evolution tends to do to unused elements (Albalat & Cañestro, 2016; Wolf & Koonin, 2013). Masking does not necessarily need to be in the direction from culture to genes: We can think of genetic assimilation (Crispo, 2007; Waddington, 1953) as the same process working in the opposite direction, where a trait that is regularly acquired through learning gradually transfers its locus to the genome (see Baldwin effect; Morgan, Suchow, & Griffiths, 2020). Genetic assimilation can eliminate the cost of learning, but only by sacrificing flexibility. The masking of genes by cumulative culture can eliminate the metabolic cost of endogenous synthesis in the case of vitamin C, and perhaps other costs in other cases, but only by sacrificing reliability of outcome.

Culture and genes are tightly intertwined in this manner, but this kind of functional interaction occurs across various levels of biological organization: between genes within the same genome – *intragenomic* (Phillips, 2008), between nuclear and organellar (mitochondria and plastid) genomes – *cytonuclear* (Sloan et al., 2018), and between host and microbial symbiont genomes – *holobiontic* (Bordenstein & Theis, 2015). Mitochondria, for example, are believed to have undergone extensive reductive evolution, transferring nearly all of their genes to the nuclear genome (Sloan et al., 2018; Wolf & Koonin, 2013). Indeed, the residual mitochondrial and nuclear genomes collaboratively assemble "chimeric" proteins (Osada & Akashi, 2012). This kind of coevolution is not uncommon in the history of life (Jablonka & Lamb, 2005; Laland et al., 2015).

Similar to these cross-level interactions, culture and genes are interwoven in the construction of many behavioral traits, making separation effectively impossible. Because these two systems

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interact within a shared space of phenotypic variation, a focus on only one or the other leaves significant ambiguity in the causal underpinnings of these phenotypes. For example, is language primarily the result of culture or genes? Such a question can be answered only by recourse to both sides of our dual inheritance (Chater, Reali, & Christiansen, 2009; Christiansen & Chater, 2008; Deacon, 1997; Dediu, 2011; Dediu & Ladd, 2007; Wong et al., 2020). Epistemological biases in the interpretation of the causal loci of phenotypic traits can misinform policy decisions and impede progress in the research and development of beneficial interventions. Given the inherent ambiguity that arises because of gene–culture interaction, it is important to adopt a sufficiently integrative framework to interpret findings.

3.4 Cultural Simpson's paradox

The WEIRD sampling problem, the hidden cluster problem, and the causal locus problem are fairly general issues. But, when we consider them jointly, the three problems contribute to a more specific problem that is perhaps not so obvious without a cultural evolutionary perspective. In particular, cultural adaptations that mask genetic effects (causal locus problem) can confound the measurement of genetic effects when the researcher lacks information about the fine-grained distribution of cultural adaptations (hidden cluster problem) and simultaneously lacks access to a sufficiently broad range of samples that could otherwise cut through the cultural confound (WEIRD sampling problem). This set of problems can create a Simpson's paradox (Kievit, Frankenhuis, Waldorp, & Borsboom, 2013; Simpson, 1951): the association between two variables qualitatively changing after controlling for subgroup structuring.

As an example, consider UV once again. In section 2.1, we discussed how mismatch between genes (skin pigmentation) and ecology (UV levels) can be masked by the cultural diffusion of sunscreen, especially in regions with more exposure to sunlight. In other parts of the world, the more important health issue is the set of problems associated with underexposure to the sun, which causes vitamin D deficiency. Low vitamin D is associated with a broad range of risk factors, most significantly for bone integrity but also for muscle strength, autoimmune disease, cardiovascular disease, cancer (Holick, 2007), and perhaps COVID-19 (Meltzer et al., 2020; Rhodes, Subramanian, Laird, Griffin, & Kenny, 2021). Controlling for skin pigmentation, vitamin D deficiency is correlated with latitude: One US study compared a sample in Erie, Pennsylvania (42°N) to one in Bradenton, Florida (27°N) that was matched along many key variables, and found that the northern group had lower levels of serum vitamin D and were at much greater risk of vitamin D deficiency than the southern group (Leary, Zamfirova, Au, & McCracken, 2017). A study in France similarly found that people in more northern regions had lower levels of vitamin D and a much higher prevalence of vitamin D deficiency compared to people in more southern regions (Chapuy et al., 1997).

Despite this intuitive pattern within countries, several studies have shown that when we compare across countries within Europe, we see the opposite pattern where people in northern countries have *higher* levels of serum vitamin D than people in southern countries (Lips, 2001; Lips et al., 2001; Van der Wielen et al., 1995). This is the case even when data collection is conducted during the winter months when sunlight is scarce, and even when the data are processed by a central laboratory facility, avoiding confounding by variation in laboratory procedures. What's going on? This inverted pattern may be partly because of genetic factors, for example, people in southern European countries having more pigmented skin, but a substantial part of it is likely because of culture. As an explanation, various researchers have pointed to the high consumption of fatty fish and cod liver oil in Northern Europe, as well as greater sun-seeking behavior in these countries compared to Mediterranean Europe; policies for vitamin D fortification of foods may also give the northern countries an advantage (Brustad, Sandanger, Aksnes, & Lund, 2004; Lips, 2007; Mithal et al., 2009; Pilz et al., 2018). These are potent cultural adaptations: Brustad et al. (2004) fed participants with the traditional northern Norwegian fish dish *mølje* three times over a span of 2 days, and discovered that it had supplied 54 times the recommended daily dosage of vitamin D. Therefore, the relationship between latitude and vitamin D levels goes one way within a country, and the other way between the countries of Europe.

Another example of a cultural Simpson's paradox from Europe is excess mortality in winter months compared to non-winter months across countries: Excess winter mortality is highest in warmer countries such as Portugal and Malta, and lowest in colder countries such as Finland and Iceland (Fowler et al., 2015; Healy, 2003; McKee, 1989). Portugal has the highest excess winter mortality among the countries estimated by Healy (2003) at 28% while Finland has the lowest at 10%, despite a much sharper temperature differential between summer and winter months (as well as lower absolute temperature) in Helsinki and Tampere compared to Lisbon and Porto. This has been called the "paradox of excess winter mortality" (Healy, 2003). The likely cause is that houses in warmer climate regions tend to be poorly insulated, which causes lower indoor temperatures. Populations in these regions also do not wear appropriate clothing when outdoors in cold weather (Healy, 2003; The Eurowinter Group, 1997). In south Finland, 72% of people wore hats while outdoors at 7°C, but only 13% of people in Athens did (The Eurowinter Group, 1997). A number of studies show that *within* countries, regions with colder winters experience higher excess winter mortality (Aylin et al., 2001; Davie, Baker, Hales, & Carlin, 2007): Here too there appears to be an inversion when comparing the effect between countries and within countries.

If we had been Martian anthropologists who did not know that the populated landmass known as "Europe" can, in fact, be broken down into subunits called "countries," these examples would be standard examples of a Simpson's paradox (Kievit et al., 2013; Simpson, 1951). In these cases, the paradox occurs when we do not know how to partition the higher-order population (Europe) into lower-order units. Fortunately, we do know how to partition continents into countries, but in other cases, the relevant units may not always be as easily identifiable as countries or administrative regions.

Countries are territorially exclusive political entities, but we can also see them as cultural groups – clusters of cultural traits that can be identified using tools such as CF_{ST}. People within a cultural group share more cultural traits than do people between separate cultural groups. All else being equal, there is more mutual cultural influence among individuals within a country than there is among people between countries. Even when we take a powerful social organizing force like religion into account, co-residents of a country who belong to different religions are more psychologically similar to each other than to co-religionists who live in different countries, although religion nonetheless also explains a sizable amount of similarity (White, Muthukrishna, &

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Norenzayan, 2021). This suggests that ideas and practices spread more easily within countries than between countries, and that countries constitute cultural clusters that can be used to partition higher-order levels of organization, such as continents. Countries too may, in turn, be decomposed into relevant cultural clusters – more acutely in places such as Africa, where colonial borders were often drawn arbitrarily without respect for ethnolinguistic groups (Michalopoulos & Papaioannou, 2020), but clustering is prevalent across many countries as revealed by subnational CF_{ST} analyses (Muthukrishna et al., 2020). To avoid a cultural Simpson's paradox, we must be able to measure cultural clustering.

Hidden clustering creates inferential problems for behavioral genetics precisely because cultural clusters are the most salient unit of organization upon which cultural-group dynamics and cultural evolution act (Handley & Mathew, 2020). Cultural evolution is fast and potent but bound within cultural clusters to varying degrees. Because different clusters are each independently able to incorporate cultural adaptations, genetic effects can get differentially masked in a cluster-wise fashion. The extent of this masking varies across clusters, and those that are exposed to greater ecological challenges (e.g., higher latitudes) may build up stronger cultural adaptations and undergo deeper masking. When these cultural adaptations not only compensate for the ecological challenge, but also overcompensate, and do so proportionally to the magnitude of the ecological challenge, the outcome is an inversion of the natural relationship between ecologies and phenotypes – as we saw earlier in the inverted correlation between latitude and vitamin D (Brustad et al., 2004; Lips, 2007; Mithal et al., 2009; Pilz et al., 2018) and between latitude and winter mortality (Fowler et al., 2015; Healy, 2003). The cultural Simpson's paradox thus arises when cluster-wise cultural adaptation across an ecological gradient changes the slope of the ecological effect: attenuating, neutralizing, or inverting its directionality depending on the strength of the cultural adaptation relative to the ecological challenge.

Therefore, the relationship between genes, ecology, and phenotype will often be qualitatively different for humans than it will be for other animals. We should be careful of inferential errors that may derive from neglecting the cluster-wise cultural evolution of environments. Even if the gradient of cultural adaptation does not go as far as inverting the ecology–phenotype relationship and instead takes a more moderate form, it can nonetheless confound measurement of genetic or ecological effects. If the arrangement of the cultural clusters within a population is unknown, such gradients may be difficult to detect in the first place.

To summarize the problems that we have discussed thus far: The WEIRD sampling problem and the hidden cluster problem each obscure the variance structure of a sample – the former by concealing the range of total variation that exists outside of a restricted (possibly unrepresentative) segment, the latter by concealing the heterogeneity within that segment. The causal locus problem allows for differential masking among the heterogeneous subgroups, and poses a challenge to interpretation of gene function, for example in the form of a cultural Simpson's paradox. Collectively, these problems have long obscured the effect of culture within behavioral genetics. Cultural evolution can help us understand phenotypic distributions in human societies as well as more accurately represent the structure of our nature and nurture.

4. Behavioral genetic puzzles in light of cultural evolution

A dual inheritance and cultural evolutionary theoretical framework can help make sense of various puzzles in behavioral genetics. Here, we discuss three: differences in heritability across socioeconomic levels, differences in heritability across development, and the Flynn effect.

4.1 Heritability across socioeconomic levels

The heritability of IQ is higher among affluent, high socioeconomic status (SES) households than among poorer, low-SES households in some societies (sometimes referred to as the Scarr–Rowe effect; Rowe, Jacobson, & Van den Oord, 1999; Scarr-Salapatek, 1971), but the relationship between SES and heritability is mixed in other societies (Giangrande et al., 2019; Hanscombe et al., 2012; Nisbett et al., 2012; Platt, Keyes, McLaughlin, & Kaufman, 2019; van der Sluis, Willemsen, de Geus, Boomsma, & Posthuma, 2008; Turkheimer et al., 2003). A cultural evolutionary perspective can shed light on these findings.

4.1.1. Discrepancies in cultural transmission across societies

A meta-analysis (Tucker-Drob & Bates, 2016) found the gene × SES interaction on IQ in a subset of US samples, but not in samples from Europe and Australia. Pooling the US studies, the authors found an effect size that corresponds to a heritability estimate of 0.61 at 2 standard deviations above the mean SES but only 0.26 at 2 standard deviations below the mean. In Western Europe and Australia, heritability is more uniform. The cause of this interaction is still debated.

Several researchers (e.g., Bates, Lewis, & Weiss, 2013; Beam, Turkheimer, Dickens, & Davis, 2015; Tucker-Drob, Briley, & Paige Harden, 2013) have suggested that gene–environment correlation via phenotype-to-environment transmission, otherwise referred to as "reciprocal causation," is the most likely explanation. By this explanation, those with genes well suited to a task can better nurture their skills in a wealthier environment compared to in a poorer environment. That is, initially small differences in genetic potential become gradually amplified over time because of the iterative matching of environments to abilities: An increase in expressed ability brings forth new environmental conditions that enable further growth along that dimension (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr, 1992). Such processes can increase genetic heritability, but through reciprocal shaping between genetic potential and environment, rather than through innately specified ability levels. The reasoning is that high-SES households are able to provide environments that do this more effectively and are thereby able to let genetic potential be more reliably associated with the corresponding outcomes, lifting heritability as a result. While such reciprocal causation may indeed be occurring, reconciling this explanation with the findings from Europe and Australia seems more challenging or at least incomplete.

Heritability is a function of the variability in culture, which is shaped by cultural-group dynamics and cultural evolutionary forces. In the United States, the differences between, for example, school and home environments among high-SES households is likely to be small relative to differences between school and home environments among low-SES households, where factors such as school lotteries can dramatically affect the cultural input. In contrast, the cultural environment is less unequal in Western Europe and Australia, where, for example, high-quality schools are available across SES. Where these two explanations make different predictions is for poorer countries. The reciprocal causation explanation would predict low heritability in poorer

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countries. The cultural evolution of genetic heritability explanation would instead predict high heritability where there is equal access to similarly poor schools and household conditions, but low heritability if inequality is high.

Although environmental variability is commonly construed through the framework of economic inequality, we can also look at it through the lens of cultural variability. For example, even if a society maintains economic equality, it may still contain cultural subdivisions that lead to multiple clusters each with different traditions and behaviors. When this cultural clustering creates differences in the level of some given trait between clusters, the heritability of this trait decreases in accordance with the strength of the clustering.

We predict that networks of cultural transmission are more fractured (i.e., contain a higher density of cultural clusters) in low-SES households than they are in high-SES households comparing within a country, at least for (but not limited to) transmission of cultural information and influence that impacts traits in domains such as cognitive ability. We also predict that these cultural networks are more fractured in the United States compared to those in Australia and Europe, with the greatest fracturing expected in low-SES US communities. Moreover, we predict that these differences across groups will explain at least some portion of the interaction between SES and heritability in the measurement of IQ. These effects could be tested through causal identification techniques, including randomized controlled trials or natural experiments such as school lotteries.

A number of studies have examined unstandardized variance components to make sense of the forces that are driving the gene × SES interaction effects for cognitive ability, but the results have been mixed. In some studies, the interaction is apparently because of an increase in variance explained by the shared environment (the C component of the ACE model; A: additive genetic variance, C: shared environmental variance, E: nonshared environmental variance) in lower-SES households compared to higher-SES households (e.g., Hanscombe et al., 2012; Kremen et al., 2005), while other studies suggest that it is instead because of a decrease in variance explained additively by genes (the A component of the ACE model) (e.g., Bates et al., 2013; Kirkpatrick, McGue, & Iacono, 2015). The shared environment explanation is more consistent with cultural dynamics, although reality may be more complicated with other processes such as reciprocal causation (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr, 1992) contributing to the same effect through other pathways, such as via moderation of the additive genetic component. Moreover, we would predict that this environmental variance would be reduced if cultural opportunities and transmission networks among low-SES households became more broadly connected, supporting greater cultural homogeneity. This would be true, even if the level of poverty or other indicators of well-being remained just as low. That is, this effect is not about poverty or deprivation per se – heritability can be high even among lower-SES groups – but more about cultural clustering. Simply moving neighborhoods can vastly improve life outcomes for low-SES Americans (Chetty & Hendren, 2018a, 2018b), but we would not predict such large effects for high-SES Americans.

In general, we predict that Scarr–Rowe-like discrepancies in the heritability of IQ will be found when comparing groupings that vary in their degree of within-group cultural clustering (or homogeneity) whenever this clustering imposes barriers upon the cultural transmission of cognitive ability. The more clustered society will be associated with a lower heritability for the trait; this effect would be expected across many culturally transmissible traits beyond IQ as well. When disaggregated by cultural cluster (e.g., SES), we should expect lower heritability among the more culturally diverse and/or more culturally clustered subgroup.

4.1.2 Discrepancies in environmental variation between humans and other animals

A comparison of these SES effects on humans with similar experiments in rodents offers additional insight. Sauce et al. (2018) found that mice reared in an enriched environment exhibited lower heritability of a rodent analog of general intelligence (for standard rodent-learning tasks such as odor discrimination and navigation) than did mice reared in a control environment, with a heritability of 0.15 in the enriched group versus 0.55 in the controls. The directionality of this difference is opposite of what we have described earlier for the human literature, and the opposite of what the reciprocal causation explanation would suggest (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr, 1992). If we assume that this type of environmental enrichment paradigm can be mapped onto differences between high- and low-SES environments in humans, as has been argued (Hackman, Farah, & Meaney, 2010; Rosenzweig & Bennett, 1996), the reported discrepancy between rodent and human studies might appear surprising.

Non-genetic phenotypic variation in mice appears to be generated in part by initially small, random differences in experience being amplified over the course of development (Kempermann, 2019) – complex environments enable greater amplification of these initial differences and hence "individualization," whether the environmental complexity is of a physical (Freund et al., 2013) or social nature (Shemesh et al., 2013). In these studies, both genes (because of inbreeding) and the shared environment are controlled. It is, therefore, the nonshared environment, or individually unique forms of experience, that is driving this differentiation. The enriched environment enables a greater range of variation in experience and as a consequence, genetic effects are proportionally reduced.

Compare this to humans, where cultural transmission gradually homogenizes the environment within a cultural cluster (though not necessarily between cultural clusters), and to varying degrees across clusters as well. We predict that high-SES US households as well as households within Australia and European countries are less culturally fragmented compared to low-SES US households, leading to higher environmental similarity within Australians, Europeans, and high-SES Americans, respectively (but not necessarily high similarity between the groups). In other words, relatively enriched environments (high-SES or Australia/Europe) are also those that are more culturally homogeneous. This, in turn, may explain why enriched environments would be negatively associated with heritability in mice (Sauce et al., 2018) but positively associated with heritability in humans at least under certain conditions (Tucker-Drob & Bates, 2016). Our prediction is that the opposite effect between the two species arises because in humans, the amplification of initial differences in experience by complex environments (individualization) is overwhelmed by the homogenizing effect of cultural learning and group dynamics (e.g., through oblique transmission from favored models in the previous generation and horizontal transmission from peers). The more variation is generated by enriched environments and individualization, the more incentive there is to learn from successful individuals or from those who are likely to have learned from successful individuals themselves.

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Cultural diffusion systematically squashes the variation created by innovation (see the Appendix for a model of such dynamics).

A cultural evolutionary approach to heritability distinguishes between humans and other animals in ways that a standard behavioral genetics approach does not. Whereas behavioral genetics provides no clear reason to differentiate analysis of genetic effects in humans from genetic effects in nonhuman animals, cultural evolutionary theory explicitly formalizes the difference between humans and other animals: our extensive dual inheritance (Boyd & Richerson, 1985). The human-specific predictions that arise from a cultural evolutionary behavioral genetics offer clear tests for our hypotheses. The approach may, thus, also be validated by comparative behavioral genetic analyses between humans and nonhuman animals. This research direction may help make sense of some of the limitations of nonhuman animal models, in cases where cultural adaptations create unexpected gaps in generalizability despite sufficient phylogenetic conservation of relevant phenotypes.

4.2 Heritability across development

Cultural heterogeneity may also vary across development. Because culture typically acts as a phenotypic homogenizer within cultural clusters, we should be able to detect the influence of culture across development in the form of changes in genetic heritability. How we learn and who we learn from changes over the lifespan. One, especially, important transition is the shift from learning primarily from parents and other family members to learning from more distant models who are selected from a broader swath of society (Cavalli-Sforza & Feldman, 1981). In the first of these two phases, there is less choice in what to learn, and much of the acquired knowledge is passed down through the same route as genetic information – from parent to child – by *vertical transmission*. In the second phase, the child is more independent, and has the opportunity to update what they have learned from a broader range of models, using learning strategies to decide whom to learn from, by *oblique transmission*. This expansion in learning models is essential for cumulative cultural evolution (Enquist et al., 2010), and may be facilitated by adaptations such as early weaning (Finlay & Uchiyama, 2020; Hawkes & Finlay, 2018). Building on Cavalli-Sforza and Feldman (1973), who show that vertical transmission inflates heritability estimates by increasing similarity between caretakers and children, our argument focuses on how broad, community-level oblique and horizontal transmission can either inflate or deflate heritability by unmasking or masking genetic effects. Both processes are likely to be operating simultaneously, although if oblique transmission is broad enough, it may weaken the confounding effect of vertical transmission.

This transition from vertical to oblique learning moves the child from the idiosyncrasies of their parents and household to the larger environment they now have in common with other adolescents and young adults. When the child is primarily relying on vertical transmission, the characteristics of their household plays a larger role in explaining variation in cultural input, in which case we should expect a high proportion of phenotypic variance to be explained by the shared (home) environment in twin studies. When the child switches to oblique learning, they now share more common influences with other children. Insofar as these traits unmask genetic differences, this would be expected to reduce environmental variation across the population and increase heritability.

This reasoning implies that for phenotypic traits that are molded in real time by the current shared environment instead of by the persisting effects of earlier parental influence, heritability should increase at this later life stage. Indeed, this is precisely what Hatemi et al. (2009) find in the case of political orientation, or where one lies on a progressive–conservative spectrum, measured in a US sample by questionnaire (Fig. 3). Monozygotic (MZ) and dizygotic (DZ) twin pairs are both equally similar from middle childhood up to early adulthood, although the degree of twin similarity increases over time for both. Right around the age at which American children leave home, this pattern is broken, and the phenotypic correlations drop precipitously in DZ twins while remaining steady in MZ twins, and this discrepancy persists for the rest of the lifespan. The drop in DZ but not MZ correlation at this age suggests that the shared home environment exerts a convergent influence for both twin types early in life, but that once this influence is removed, genetic effects become unmasked and able to guide political attitudes independently from the shared environment. More phenotypic variance is explained by genes from this point onward, thus boosting heritability. In countries such as Italy and Croatia, where the mean age of leaving the parental household is past 30 (European Statistical Office, 2020), we would predict the developmental time course of heritability to reflect this later independence relative to American samples. Note that the present example has the same overall structure as the literacy example discussed earlier (Samuelsson et al., 2008), with heritability increasing as cultural influences from outside of the home environment kick in. Both examples indicate that heritability can be an index of shared life history and communal structure.

Although the use of shared household environment to analyze twin data is a standard methodological convention, the household is, in fact, just one among many groupings of cultural organization that generate environmental convergence (Harris, 1995). Households may be the most potent cultural grouping for some phenotypic traits, but other groupings may have significant impact as well for specific kinds of traits. These may include schools, peer-groups, sports teams, religious communities; society-wide groupings such as different mass media and popular culture; more diffuse groupings that are organized around particular sets of values such as political ideology or professional values; and possibly new kinds of groupings such as online communities.

Separating out the effect of household from the effect of genes is typically considered to be an explanatory goal, but there may be further phenotypic variance that could be meaningfully explained if we were able to match phenotypes to other relevant cultural groupings and therefore to other sources of cultural influence. Twins share "common environments" across multiple scales of social organization in this manner, but when phenotypic similarity is engendered by cultural groupings that extend beyond the household, the resulting correlations will usually be relegated to the broad category of "nonshared environment," unless membership, for example, happens to align with household structure (Plomin & Daniels, 1987; Plomin, Asbury, & Dunn, 2001). Although the nonshared environment is typically discussed as environmental exposure that is specific to the individual, it remains possible that there are multiple layers of communal structure embedded within this variance component.

This point becomes somewhat obvious when we consider systems outside of the European Marriage Pattern (Henrich, 2020), such as the Israeli kibbutzim (Lieblich, 2010), the "walking marriages" of the Mosuo of southwest China (Ji et al., 2013), or the communal child rearing of the Ache of eastern Paraguay or of

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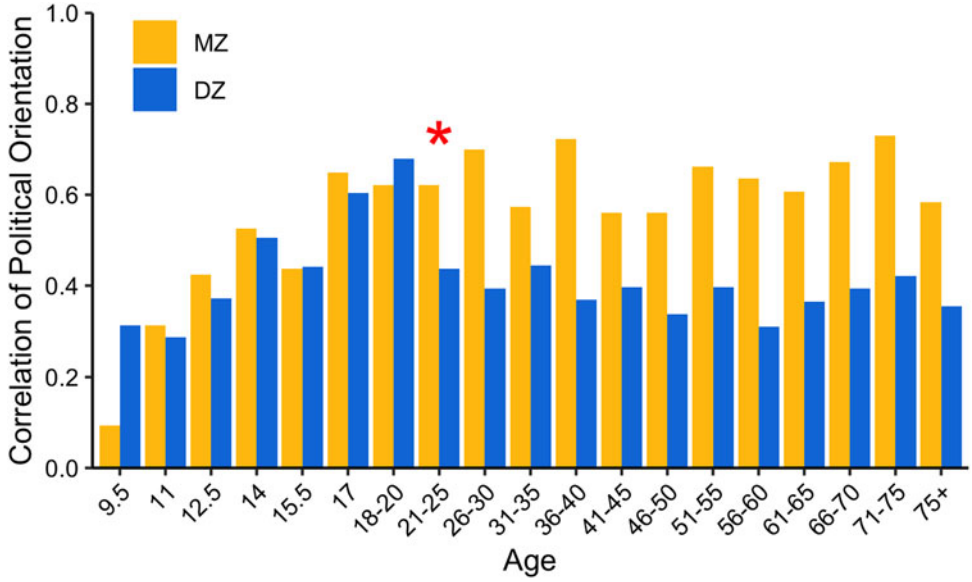

Figure 3. Twin concordances in political orientation. In middle to late childhood, within-twin correlations for reported political orientation are roughly the same between monozygotic (MZ) and dizygotic (DZ) twin pairs in a US sample. In the early 20s, shortly after many US youth leave home for the first time to attend university, the correlation drops for the DZ twins but not for the MZ twins (identified with a red asterisk). This shift corresponds to a sudden rise in heritability, as genetic similarity now predicts similarity in political orientation. When the effect of the home environment is weakened and replaced with more diverse cultural input, the effect of genes becomes unmasked and separates the phenotypic concordances between the two twin types. The horizontal axis indicates age and the vertical axis indicates percentage twin concordances in political orientation. Figure reproduced from Hatemi et al. (2009).

the Hiwi of Venezuela (Hill & Magdalena Hurtado, 2009). In such societies, similarity of developmental environments may not be adequately parsed at the unit of the household (Kağitçibaşi, 1984). The Western notion that socialization takes place primarily in the household may not apply even to Western societies (Harris, 1995). If this is the case, then the strategy of looking for phenotypic correlations within the household that are unexplained by genes may be appropriate for answering questions about the specific influence of the household, but is insufficient for answering questions about nature–nurture more broadly.

One potential way to overcome this limitation is to map out clusters of cultural similarity within a society using a tool like cultural F_{ST} (Muthukrishna et al., 2020), explained earlier, and match this finer-grained population structure with genetic data. If assignment to a cluster explains phenotypic similarity over and above that of the shared household, for instance, such methods could be leveraged to capture a broader picture of the environmental factors that guide phenotypic development. If we were able to track the changing organization of these cultural clusters over time, it would allow us to follow the dynamic trajectory of environmental structure and partition this out from measurements of genetic effects longitudinally. We expect social phenomena such as urbanization and demographic change to be associated with change in these cultural clusters over time.

General intelligence is another trait whose heritability is known to change over the course of development (Briley & Tucker-Drob, 2013; Haworth et al., 2010; for a number of other traits see Bergen, Gardner, & Kendler, 2007). This takes the form of a steady increase from childhood through adolescence all the way to early adulthood, after which it remains more or less steady over the lifespan. Although estimates vary, one meta-analysis (Haworth et al., 2010) put the heritability of general intelligence at 0.41 in childhood and 0.66 in adulthood. Explanations for this pattern typically invoke a combination of (1) gradual activation of relevant genes over the course of brain development and (2) active gene–environment correlation or "reciprocal causation" (Bouchard, 2013; Haworth et al., 2010; Plomin, DeFries, Knopik, & Neiderhiser, 2016; Tucker-Drob et al., 2013).

In contrast, a cultural evolutionary perspective would attribute the rise in the heritability of IQ to the developmental time course of cultural influence. One clear testable prediction is that in a society with different constraints on the development of cultural learning, as in the above example of political orientation, the developmental trajectory of heritability would also differ. A society that engages with communal childrearing immediately from infancy will impose a different set of developmental constraints (and opportunities) compared to a typical Western society. Even in Western societies, cultural development may look very different from now just a few decades into the future, if information technology continues to transform children's networks of social interaction and influence. A cultural evolutionary explanation can help make sense of the relevant data. For example, we predict that sharp changes in heritability will map onto sharp changes in an individual's cultural environment (e.g., the start of school, university, or military service). These milestones may shift because of policy changes, allowing for causal tests of this hypothesis. If, for example, children start higher education later, then large increases in heritability should also occur later.

Whether or not one agrees with our construal of these particular examples, it is logical that genetic effects for many traits become confounded with communal structure unless the developmental time course of cultural influence is properly accounted for. Cultural evolutionary theory predicts that over the life course, cultural influence begins from a primarily parental setting and progressively expands its scope into the greater community, diversifying the models from which an individual can learn (Fogarty, Creanza, & Feldman, 2019; Muthukrishna, Doebeli, Chudek, & Henrich, 2018). Insofar as this occurs, we predict a

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general increase in heritability over the lifespan for culturally malleable traits. The slope of this increase would depend upon cultural parameters and vary by society. Conversely, if there is a correlation between communal structure and the heritability of some trait, this may serve as an index of the cultural malleability of that trait. Our goal here is to bring attention to the conceptual and analytic power that cultural evolution can contribute to developmental behavioral genetics.

4.3 The Flynn effect

The Flynn effect describes the rise in IQ test scores over time (Flynn, 1984, 1987) – roughly 2–3 IQ points per decade on average around the world (Flynn, 2009; Pietschnig & Voracek, 2015; Trahan, Stuebing, Hiscock, & Fletcher, 2014). The rate of increase differs between countries, being largest in countries that have recently started modernizing, and smallest in countries that had attained modernization by the beginning of the twentieth century (for a review, see Nisbett et al., 2012). In some countries in Northern and Western Europe including Denmark, the Netherlands, and the United Kingdom, there is evidence that the Flynn effect has been slowing down and even reversing in recent decades (Dutton, van der Linden, & Lynn, 2016). This negative Flynn effect is even less well understood compared to the positive Flynn effect. Bratsberg and Rogeberg (2018) find that in Norway, the negative Flynn effect is found within families (between siblings), thereby making it unlikely to be explained by demographic changes or immigration, and instead supporting an environmental explanation.

There is no consensus regarding the cause of the Flynn effect, but given the recent and rapid increase, genetic explanations are unlikely. Various hypotheses include increases in test familiarity, improvements in education, sophistication of the technological and media environment, better nutrition, decreasing family size, and slowing life history, and increased out-breeding or "hybrid vigor" (Bratsberg & Rogeberg, 2018; Clark, Lawlor-Savage, & Goghari, 2016; Johnson, 2006; Joshi et al., 2015; Nisbett et al., 2012; Pietschnig & Voracek, 2015; Trahan et al., 2014; Woodley, 2012).

Flynn (2007) and Greenfield (1998, 2009) suggest that the effect is caused by a rapid worldwide increase of cultural practices, technologies, and environments that promote abstract cognitive processing as opposed to more traditional forms of concrete, pragmatic thinking. Some examples explored by these authors included urbanization, mass media, video games, education style, counterfactual thinking, and white-collar occupations. This account is mostly consistent with a cultural evolutionary explanation, which would suggest that intelligence is not just about hardware – genes, parasites, pathogens, pollution, and nutrition affecting health and brain development, but also software – the increasingly complex cultural package we acquire from our societies (Bloom, 2020; Gordon, 2018; Jones, 2020; Mesoudi, 2011). By this account, not only is the idea of a culture-free IQ test implausible, but so too is the idea of culture-free IQ (for discussion, see Muthukrishna & Henrich, 2016). Indeed, the largest Flynn effect can be seen on the supposedly culture-free Raven's matrices (Flynn, 2007; Nisbett et al., 2012), and on tests for fluid IQ rather than crystallized IQ (Pietschnig & Voracek, 2015). When it comes to heritability, subtests of IQ that are more culturally influenced are more heritable (Kan, Wicherts, Dolan, & van der Maas, 2013).

Beyond the diffusion of specific traits and abilities, a cultural evolutionary explanation also highlights how the Flynn effect is driven by the reorganization of cultural transmission pathways themselves. The introduction and improvement of formal schooling is one major instance of reorganization in cultural transmission that is also known to positively impact IQ (Brinch & Galloway, 2012; Ceci, 1991; Davis, 2014; Ritchie & Tucker-Drob, 2018). Greenfield (1998) describes how IQ scores in some rural US towns in the early twentieth century increased rapidly at the same time as a number of coordinated changes in infrastructure, including better access to urban areas and new, high-quality road systems. Such enhancements in social connectivity directly translate into cultural connectivity, allowing for the influx and diffusion of psychological and behavioral traits that are considered valuable within the broader society. In much of the modern world, the kind of abstract information-processing ability measured by IQ tests is considered valuable, as it is useful in various white-collar professions that are typical of WEIRD societies. The Flynn effect, therefore, captures the progressive enhancements in cultural connectivity that have been occurring around the world because of improvements in various domains of infrastructure and technology including transportation, urbanization, education, and media. Global IQ rises in response to both the invention of relevant cultural traits and the enrichment of cultural transmission networks that carry those traits.

One clear test of this cumulative culture explanation for the Flynn effect would be to randomly assign children to attend or avoid formal schooling. Such a test would be highly unethical and differences in school attendance and quality are typically associated with various kinds of deprivation. Where policy changes have changed years of schooling in ways that lend themselves to causal identification through a natural experiment, an increase in formal education has been shown to cause an increase in IQ (Brinch & Galloway, 2012; Ritchie & Tucker-Drob, 2018). A recent test by Davis, Stieglitz, Tayo, Kaplan, and Gurven (2020) offers even clearer data. Children, but not adult IQ performance is compared within age groups, because we assume children perform better as they get older. Exploiting a natural experiment where access to education was uncorrelated with SES, nutritional status, and acculturation, Davis and colleagues show that without access to education, there is no correlation between age and IQ test performance. With moderate access, we see a moderate slope and with more access, a slope similar to the West.

5. Cultural evolutionary behavioral genetics

Behavioral genetics offers a powerful empirical approach to understanding human behavior, but since the advent of whole-genome methods, its strategy appears to lean toward the notion that with enough data, ground truths about human nature and nurture, if only for some traits, will be revealed. Data alone is not enough (Muthukrishna & Henrich, 2019); the thrust of our theoretical case is that human psychology and behavior have a large cultural component that has been changing over history (Boyd, 2018; Boyd & Richerson, 1985; Chudek, Muthukrishna, & Henrich, 2015; Henrich, 2020, 2016; Laland, 2018; Muthukrishna, Henrich, & Slingerland, 2021; Nunn, 2020; Wilson, 2019). Most recently, our psychology has been shaped by the advent of writing, numeracy, different types of agriculture, the Industrial Revolution, the internet, and smart phones (Domahs et al., 2010; Firth et al., 2019; Ong, 1982; Talhelm et al., 2014; Uskul, Kitayama, & Nisbett, 2008; Wilmer, Sherman, & Chein, 2017). As new adaptive traits emerge

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(Muthukrishna & Henrich, 2016), initially those who possess these traits will have an advantage, as in the case of access to new food sources, better healthcare, more efficient technologies, or easier methods of learning. But eventually the traits will reach fixation in the population through the processes of cultural diffusion (Henrich & Broesch, 2011; Muthukrishna et al., 2016), at least until they are unseated by subsequent innovations (Kolodny, Creanza, & Feldman, 2015; Muthukrishna & Henrich, 2016). We predict that these cultural dynamics are reflected in heritability estimates.

As any geneticist knows, genetic heritability is a function of the variability in the environment, variability in genes, and variability in the phenotype. There is little to predict if the phenotype is homogeneous, as in the number of fingers or kidneys. There is little to predict *with* if the environment or genes are homogeneous. But what is factored into the environment includes not only the physical ecology, but also the cultural environment. While variance in genes and ecology may be relatively stable, the variance in the cultural environment is continually changing through the processes of cultural evolution. Genetic heritability estimates are highly affected by not only cultural diversity and cultural clustering, but also by factors that affect cultural transmission such as sociality (Muthukrishna, Shulman, Vasilescu, & Henrich, 2014), transmission fidelity (Morgan et al., 2015), tolerance for variation (Muthukrishna & Henrich, 2016), population structure (Henrich, 2004a), and social network topology (Derex & Mesoudi, 2020; Muthukrishna & Schaller, 2020). Under most empirical conditions, behavioral genetics underestimates the contribution of culture, including in estimates of heritability. We don't disagree with the findings in these fields or the data used, but instead argue that more nuance is required in how they are interpreted. Our dual inheritance demands that a genetic account of human psychology and behavior must also account for culture and cultural evolution.

5.1 Toward a dynamic model of environment

We are surrounded by the products of culture yet are generally unaware of the generative processes that bring such complex objects and conditions into existence. Cultural transmission spans broad networks of interconnected individuals, as well as deep timescales of inheritance. Each individual experiences just a snapshot, leaving the global mechanics opaque. Thus, each of us is left with an intuition that *our* world is largely *the* world, which perhaps explains why the extent of the WEIRD people problem went unappreciated for so long, even a decade after publication of Henrich et al. (2010) (Apicella et al., 2020; Barrett, 2020; Nielsen, Haun, Kärtner, and Legare, 2017; Pollet and Saxton, 2019; Tiokhin, Hackman, Munira, Jesmin, and Hruschka, 2019). From this limited vantage point, we evaluate questions such as the relative contributions of nature versus nurture. But our understanding of "nurture" remains fundamentally anchored in our restricted experience of being enculturated into a particular environment, which leads us to implicitly see environmental features shared by members of our community as factors to be held constant, while our variables of interest – be they the absence of a parent, a childhood illness, birth into nobility, or a polygenic score – become matched to outcomes in our predictive models. Such models may be informal or formal, either encountered in community gossip ("children raised by single parents usually become") or in scientific journals ("growth mindset interventions predict"; Sisk, Burgoyne, Sun, Butler, and Macnamara, 2018[9]). Our need for causal explanations (Gopnik et al., 2004; Penn & Povinelli, 2007) meets our tendency to essentialize people and groups, where genes offer a better essentialist vehicle than the environment (Dar-Nimrod & Heine, 2011; Heine, 2017).

The importance of limiting behavioral genetic findings to the reference population was famously argued for by Lewontin (1970), and remains a caveat for the analysis of genetic effects. But it is far less appreciated that the reason why a multitude of phenotypic factors can be successfully held constant (or controlled for) in the first place is, in large part, because of the convergent force of cultural learning. Lewontin (1970), in his counterargument to Jensen's (1969) controversial article that argued for the innateness of IQ, used inbred corn and a uniformly acting nutrient solution as his rhetorical props for explaining the environmental sensitivity of genetic effects. Domesticated crops experience a more homogeneous environment not by accident, but as a product of human cumulative culture. Lewontin's famous example is, thus, an unintentional illustration of how culture can generate at times extreme phenotypic convergence in significant features of the environment – either our own or of our domesticated flora and fauna.

We are all aware of gene–environment interactions (Hunter, 2005; Lewontin, 1970; Moffitt et al., 2005), but we still tend to focus on what is predictive in our statistical models, which are constructed in a particular population, environment, and period of time but whose apparent lessons are commonly generalized beyond these contexts (e.g., the effects of an educational intervention). These models typically do not capture how the relevant environments are distributed within and between populations or how (or why) one type of environment transitions into another – "environment" is simply given as an exogenous variable. The cultural evolutionary approach forces us to explicitly recognize that human environments do not just happen to fall into place; they are rather the outcome of a dynamic, adaptive process that responds to both environmental and genetic factors. The literature on gene–environment interaction already recognizes genes and environments as non-orthogonal, but dependencies between the two are likely to be tighter and more prevalent than would be expected in a culture-free framework. This dynamic view of the environment also suggests that the problem of limited portability of polygenic scores across populations (Kim et al., 2018; Martin et al., 2019, 2017) is also likely to be a problem across historical time in a single population, as the issue is not just about differences in nucleotides across groups but also about the buildup of the cultural environment.

If we are to accommodate culture, the environment can no longer be treated as a static projection plane over which active elements (i.e., genes and G × E interactions) drop their shadows. Instead, both genes and environment – the latter animated by cultural dynamics – are in motion with respect to each other (as an example, see language–brain coevolution; Christiansen, 1994; Christiansen & Chater, 2008; Deacon, 1997; and cultural niche construction; Laland & O'Brien, 2011; Laland, Odling-Smee, & Feldman, 2001). An environment can be used as a reference frame against which to judge the effect of genes, but this is done for pragmatic purposes and not because environments are intrinsically fixed (Haworth & Davis, 2014). We might take our cue from James Gibson's contribution to the study of vision, which he summarizes in the following manner:

> The standard approach to vision begins with the eye fixed and exposed to a momentary pattern of stimuli… The ecological approach to visual perception works from the opposite end. It begins with the flowing array of the observer who walks from one vista to another, moves around an object

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> of interest, and can approach it for scrutiny, thus extracting the invariants that underlie the changing perspective structure. (Gibson, 1979, p. 303)

Gibson recognized that environmental change is not noise, but rather the very medium through which the scientist obtains knowledge about visual function. Our argument presents an analogous approach to the study of genes.

5.2 Toward a culturally situated understanding of intelligence

The genetic underpinnings of intelligence have roots going back to the beginning of behavioral genetics (Galton, 1869, 1874) and have been fiercely debated since at least Jensen (1969) and Lewontin (1970). The topic remains contentious, but a dual inheritance perspective cuts through some of this debate. Here, we summarize some key points.

IQ appears heritable – often measured at around 0.4 in toddlers and increasing up to 0.7 or 0.8 in adults (Bergen et al., 2007; Bouchard, 2009). But as we and others have discussed, a high heritability score does not necessarily tell us whether a trait is primarily genetic; high heritability can also be an indicator of environmental homogeneity. Intelligence is a function of both our hardware (brain) and our software (culture) (Heyes, 2018; Hutchins, 1995; Vygotsky, 1980), and the software has been changing far more and far more rapidly than has the hardware (Uchiyama & Muthukrishna, in press). Genes certainly contribute to the size and organization of our brains – indeed, the cultural brain hypothesis predicts a strong selection pressure for larger brains (Muthukrishna et al., 2018), still evident in the rapid increase in emergency birth interventions as a function of head size (Lipschuetz et al., 2015). But those genes are explaining residual phenotypic variation only after accounting for environmental factors that also affect the quality of neural hardware, such as nutrition (Lynn, 1990; Stoch et al., 1982), parasites (Jardim-Botelho et al., 2008), air pollution (Zhang, Chen, & Zhang, 2018), and lead exposure (Needleman & Gatsonis, 1990; Wasserman et al., 1997). All are known to influence intelligence, but in societies that have been able to minimize variation in such factors through cultural diffusion, the environmental effect is also minimized. And it is not only such physical and physiological variables: changes in and diffusion of the cultural package delivered by schooling (Ceci, 1991; Davis, 2014; Davis et al., 2020; Ritchie & Tucker-Drob, 2018) and our ever more complex entertainment media (Greenfield, 2009; Johnson, 2006) also reduce the variation to be explained. All these processes unfold outside of any genetic changes to our neural hardware.

Recent, high-powered GWASs have found that genes associated with intelligence are expressed predominantly in the central nervous system (Davies et al., 2018; Savage et al., 2018; Sniekers et al., 2017), but these findings too only explain the residual variation that remains after cumulative culture has reduced variation across many other variables – such as pathogens, parasites, and nutrition – that would otherwise account for huge portions of variation in IQ test performance. The expression of "intelligence genes" may cluster inside the head (Davies et al., 2018; Savage et al., 2018; Sniekers et al., 2017), but this expression profile cannot be meaningfully evaluated without first considering the prior contributions of cumulative culture, which are invisible to standard methods in behavioral genetics. We predict that, in general, the set of identified genes that explain human traits such as intelligence is liable to change as a result of cumulative cultural evolution, both across cultures and across time within a single culture. Gene–phenotype mappings for culturally modifiable traits in humans are typically more transient than they are in other species, whose gene–phenotype mappings will typically (but not exclusively; see niche construction: Laland, Matthews, & Feldman, 2016) only change at the slower timescales of genetic evolution or of passive ecological change. Just as limited sampling across historical time has obscured this difference in the temporal horizon of genetic effects across humans and nonhuman animals, the WEIRD sampling problem has obscured differences between humans and nonhumans with respect to within-species generalizability as well.

Even if intelligence is highly heritable in humans, this does not indicate that its genomic substrate works independently of the environment. The cultural environment can amplify heritability, and the degree of this amplification covaries with the extent to which a society has been able to reduce variation in physical, physiological, and informational factors that impact the phenotype. Developed countries, almost by definition, have been most successful in reducing this variation. This flattening of variation is an outcome brought about by expanded networks of cultural influence and greater cultural connectivity, both in part because of technological innovation and in part because of culturally evolving social norms and institutions. High heritability of intelligence is, therefore, most likely to reflect the effect of the cultural environment in these societies. As we argue in our discussion of the Flynn effect, cultural traits associated with high IQ are themselves transmitted through these cultural networks.

If a society demonstrates high heritability of some trait together with a high relative level of the trait, this combination may give the strong impression of genetic advantage, but the argument that we have developed in this target article points to a different kind of explanation. Comparisons of mean IQ across countries, or "National IQ" (Lynn & Vanhanen, 2012), have been under scrutiny recently, but apart from the various methodological flaws that have been pointed out (e.g., Ebbesen, 2020), the non-obvious role of the cultural environment in shaping both the trait and its measurement should be seriously considered in this discussion as well. Epistemic issues such as the cultural Simpson's paradox should also be considered in cross-national analysis, as it can create associations between groups and traits that are a product of the cultural environment even when it appears otherwise. The examples that we use to illustrate the paradox – vitamin D deficiency and excess winter mortality (sect. 3.4) – are easy to grasp, but actual instantiations of the cultural Simpson's paradox are likely to be less straightforward.

The cultural evolutionary framework possesses unique strength in providing these kinds of explanatory strategies, as well as a general theoretical basis for understanding the formation and distribution of psychological traits such as intelligence. The approach that we develop here builds upon earlier, culturally situated approaches to intelligence (Berry, 1972; Cole, 1998; Greenfield, 1998; Nisbett, 2009; Vygotsky, 1980), but goes further in incorporating the population dynamics of cumulative culture, which offers greater clarity in how traits like IQ are shaped by non-genetic processes that span generational time.

6. Conclusion

> Genetics is indeed in a peculiarly favoured condition in that Providence has shielded the geneticist from many of the difficulties of a reliably controlled comparison. The different genotypes possible from the same mating have been beautifully randomised by the meiotic process. A more perfect control of conditions is scarcely possible, than that of different genotypes appearing in the same litter. (Fisher, 1952)

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Above, Sir Ronald Fisher exalts the inferential purity that is afforded by the powerful pairing of sexual recombination with simultaneous multiple birth, which conveniently flattens environmental variation. But of course, this purity becomes progressively degraded with age, as environmental effects channel offspring through different developmental trajectories. Even among inbred, genetically identical mice that cohabit an experimentally controlled space, self-organizing trajectories of environmental experience result in clear differentiation in phenotypes such as exploration, sociality, play behavior, and postnatal neurogenesis (Freund et al., 2013, 2015; Shemesh et al., 2013).

Humans' trajectories differentiate so much more. We inhabit almost every ecosystem on Earth, not by speciating as many animals do, but through cultural adaptation, opening different developmental pathways in different societies. But even within a single society, our massive specialization leads to high levels of differentiation. Our genetic variation explains some of this, but we are the least genetically diverse great ape – two groups of chimpanzees in the Congo are more genetically different from each other compared to two groups of humans plucked from Berlin and Beijing (Prado-Martinez et al., 2013). Most of our diversity is cultural rather than genetic (Bell et al., 2009; Muthukrishna et al., 2020); culture drives much of our within-species phenotypic variation. At best, genetic effects can only be specified within the ambit of a specific cultural context, but even the boundaries of that context cannot be specified without cultural evolutionary tools. And because culture is also evolving over time, these cultural contexts also require a timestamp. Heritability is not a property of a trait in itself, because in the absence of a reference culture it is necessarily unstable.

Cultural evolution yields cultural clusters. Within each society, environments are relatively homogeneous, and the homogeneity that we find within societies is coupled with pronounced heterogeneity between societies (Bell et al., 2009; Richerson et al., 2016). Extrapolating genetic effects beyond a species is obviously mistaken, but so too is extrapolating a genetic effect beyond a culture. But this is what researchers have been doing since Galton, and it is ingrained in both our methodology and our thinking, culminating in the recent triumphalist discourse surrounding behavioral genetics and GWASs.[10] The movement toward more diverse genomic data ought to make some of these problems more obvious, just as more diverse psychological data made the problems of WEIRD psychology more obvious. But here too, data alone will not solve the problem (Muthukrishna & Henrich, 2019; Muthukrishna et al., 2020). The question is not whether genes or culture contribute more to a behavioral trait, as behavioral traits can only be understood as emergent products of our dual inheritance, genetic and cultural. Nothing in behavioral genetics makes sense except in the light of cultural evolution.

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Notes

1. Research within this framework also falls under culture–gene coevolutionary theory and the extended evolutionary synthesis (Laland et al., 2015).
2. Australia's Slip! Slop! Slap! campaign encourages practices to reduce UV radiation exposure: "slip on a shirt, slop on sunscreen and slap on a hat." More recently, it has been followed by the SunSmart program, which expanded upon the original message to further decrease exposure: "seek shade or shelter, and slide on sunglasses."
3. An analogous case can be made for vitamin D deficiency, which should be highest under the same circumstances, except that instead of high UV, it should be low UV levels that most reveal the genetic effect.
4. Many potential forces may contribute to this: Prestige bias creates trends – historically, prior to the 1920s, tanning was associated with working in a field, but after Coco Chanel was sunburnt while holidaying in the French Riviera, her fans are said to have copied her tan; success bias as tanning is associated with more time for outdoor leisure; or simply some version of runaway cultural selection (Boyd & Richerson, 1985).
5. When the cultural forces are well understood, this directionality may be analyzable. And of course, not all cultural forces are adaptive. As in genetic evolution, some may be maladaptive (e.g., female genital mutilation; Efferson, Vogt, & Fehr, 2020; Howard & Gibson, 2017), mismatched (the Western diet causing disease; Cordain et al., 2005), neutral, or somewhere between these.
6. Culturally transmissible traits are those whose distribution can be shifted by cultural learning (e.g., many psychological and behavioral traits).
7. An improvement from 2009 when 96% of GWAS participants were of European ancestry (Need & Goldstein, 2009)!
8. Note that this is not the case for modern multicultural populations that can expose children to languages not spoken by their parents or family during this critical period.
9. As an aside, that growth mindset might only replicate among low-SES or at-risk students (if it replicates at all) fits with the general point that prediction is a function of variability. In this case, where there is a deficit, interventions may work, but where there is not, the potential gains are marginal or non-existent.
10. Some recent prominent examples that have entered the public discourse include Plomin (2019) and Murray (2020). Plomin states, "Polygenic scores are the ultimate psychological test because, for the first time, they can tell our genetic fortunes." Similarly, Murray declares: "By the end of the 2020s, it will be widely accepted that quantitative studies of social behavior that don't use polygenic scores usually aren't worth reading. More formally, it will be widely accepted that the predictive validity of polygenic scores gives us useful information about causes even though we still don't understand the causal pathways."

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Appendix A: Modeling the effect of cultural evolution on heritability

A.1 Variance partitioning model

Here, we describe a simple mathematical model that captures the relationship between cultural evolution and heritability. Cultural evolution is a process in which some cultural variants spread through a population more prolifically than others. This spread can be partly because of intrinsic differences in the trait (e.g., steel axes are better than stone axes) and partly because of social learning strategies such as the conformist bias, success bias, and prestige bias (for summary, see Chudek et al., 2015; Kendal et al., 2018). Such strategies vary in their rules for selecting what to learn or whom to learn from, but they all lead to the disproportionate adoption of particular cultural variants over others, and thus to a reduction in the population-level variability of behaviors. Individual incremental improvement, individual learning (Legare & Nielsen, 2015; Rogers, 1988), cultural transmission error, recombination of cultural traits, and other sources of innovation (Muthukrishna & Henrich, 2016) will continue to inject novel variants into the population, but the fact that a nearly unrestricted number of learners can inherit the behaviors and ideas of a few influential individuals makes it easy for cultural transmission to induce homogeneity. It is not only behavioral traits that become similar within a population through cultural transmission, but also environmental factors that shape behavioral traits, such as nutrition, sanitation, education, and media.

Heritability is defined as the proportion of phenotypic variance for some trait that is explained by genes. For broad-sense heritability, $H^2 = V_G/V_P$, where H^2 is the heritability, V_P is the variance in phenotype, and V_G is the phenotypic variance explained by all genetic factors including additive genetic variance, dominance interactions, and epistatic interactions. Because total phenotypic variance is made up of contributions from both genes and environment ($V_P = V_G + V_E$), a reduction in the environmental contribution necessarily increases heritability; cultural evolution has been a powerful force for behavioral homogenization and hence reduction in environmental variance over human history. Behavioral geneticists partition phenotypic variance into genetic and environmental components, but here we further partition the environmental component into environmental variation unaffected by cultural evolution (e.g., UV exposure because of geographic location) and environmental variation affected by cultural evolution (e.g., UV exposure because of sunscreen use). For convenience, we refer to the former as the ecological environmental variance component and the latter as the cultural environmental variance component, and represent this partition using the following notation:

$$V_E = V_e + V_c \tag{1}$$

where V_e and V_c denote the phenotypic variance explained by ecology and culture, respectively. In reality, the cultural environment and even individual cultural traits will typically have a high-dimensional variance structure, such as with the various components of a tool or a ritual that can be independently modified, but here we employ a unidimensional space of cultural variation. For simplicity, we model cultural environmental variation as a uniform continuous distribution that is bound by k_{min}, the most unfavorable environmental state (for some given phenotype) within the experienced range of environments, and k_{max}, the most favorable. We can use the theoretically expected variance of the continuous uniform distribution ($(1/12)(b-a)^2$, where a and b are the minimum and maximum values) to rewrite (1):

$$V_E = V_e + \frac{1}{12}(k_{max} - k_{min})^2 \tag{2}$$

We can then substitute (2) into the standard formula for broad-sense heritability:

$$H^2 = \frac{V_G}{V_G + V_e + (1/12)(k_{max} - k_{min})^2} \tag{3}$$

Heritability, thus, decreases when $(k_{max} - k_{min})$ is large and increases when it is small. The smaller the experienced range of aspects of the cultural environment that contribute to phenotypic variation, the more phenotypic variation there is left to be explained by genes and by consequence, the higher the heritability. The magnitude of this cultural effect depends upon (a) the ratio of V_c to V_e, which is the extent of cultural influence upon the environmental variance component as a whole, as well as (b) the ratio of genetic influence to total environmental influence (V_G to V_E). We illustrate the effect of each of these variance components on heritability in Figure 4. To keep the model simple, we omit the gene–environment interaction and gene–environment correlation terms.

This model shows how heritability can be shaped by a shifting cultural environment. Once again, this insight is in itself neither new nor surprising. But the model we describe here allows us to go further and consider the particular, systematic ways in which the cultural environment is expected to change. Although this model does not incorporate cultural dynamics as such, we can nonetheless map two broad cultural dynamics onto its framework: (1) new innovations elevating the upper bound of cultural complexity (Henrich, 2004a; Muthukrishna & Henrich, 2016) and (2) diffusion of these innovations (Comin & Hobijn, 2010; Henrich, 2001; Rogers, 2003) – new traits emerge and then spread to fixation in the population. Within this framework, we can think of increases in cultural complexity as pushing up k_{max}, the most favorable cultural conditions in a society. In turn, we can think of diffusion as pushing up k_{min}, the most unfavorable cultural conditions in a society. As an example, imagine k_{max} is the educational contribution of the best school in a society and k_{min} is the educational contribution of the worst school in a society. In some societies, educational innovations diffuse quickly, whereas in others, there is more lag between the discovery of a new technology or pedagogical technique and its widespread adoption. Some societies are highly equal ($k_{max} - k_{min}$ is small) and others are more unequal ($k_{max} - k_{min}$ is large). The magnitude of the lag between increasing k_{max} and k_{min} – for example, how quickly educational innovations in the best schools diffuse to other schools – impacts environmental heterogeneity and thus heritability: We illustrate this effect as different trajectories of cultural change in Figure 4c. Moreover, by further decomposing k_{max} and k_{min}into their governing cultural dynamics, we can predict the trajectories of heritability over time.

A.2 Cultural dynamics model

To capture the effect of a changing cultural environment on heritability estimates over time, we construct a simple model that incorporates a time axis and whose parameters can be adjusted to compare different cultural trajectories. This dynamic model builds upon the previous variance partitioning model, but extends it by representing k_{min} and k_{max} – the upper and lower bounds of the acquired range of cultural complexity – as sigmoidal trajectories (s-shaped curves), which are commonly used to model cultural diffusion (Henrich, 2001; Rogers, 2003). Here, we use the Gompertz function, which is a special case of the generalized logistic function that is commonly used in biology to model growth. In particular, we employ the following re-parameterization of the Gompertz that is given by Tjørve and Tjørve (2017):

$$k_{min} = k_0 \left(\frac{k_0}{A}\right)^{\exp(-e\beta_{k_{min}} t/A)-1} \tag{4}$$

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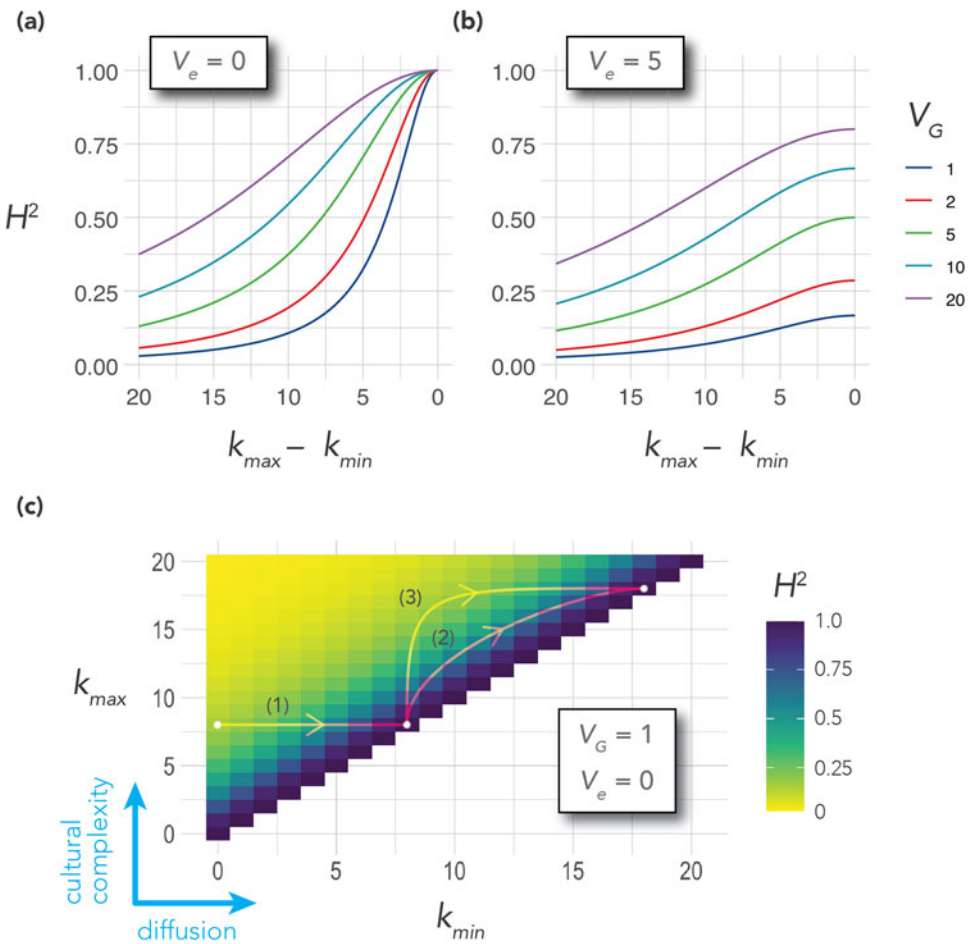

Figure 4. Visualizations of equation (3). Heritability curves as a function of cultural range ($k_{max} - k_{min}$) and of the amount of genetic variance (V_G). (a) Values are computed for $V_e = 0$ (the environment is entirely explained by cultural factors) and (b) for $V_e = 5$ (some of the environment is explained by non-cultural factors, such as climate). (c) An alternative visualization in which we look at the absolute values of k_{max} and k_{min} rather than just their difference, plotted for $V_e = 0$ and $V_G = 1$. An increase in k_{max} expands environmental variation and implies increasing maximum cultural complexity, whereas an increase in k_{min} reduces environmental variation and implies diffusion. Trajectory 1 represents a society's transition from a more unequal cultural environment to a more equal cultural environment, but with no increase in cultural complexity. Trajectories 2 and 3 represent a simultaneous increase in cultural complexity and diffusion of the newly established complex traits, where a rising k_{max} pulls k_{min} upward but with varying lags: For trajectory 2 there is little lag between increase in the complexity of the culture and its spread, whereas for trajectory 3 there is considerable lag, with a longer period of relative cultural inequality. Genetic heritability decreases with rising cultural complexity and increases with cultural equality (diffusion). For example, continued innovation will reduce heritability up to the point at which the society is maximally unequal, and then increase heritability once more as the cultural innovations spread to the entire population – that is, curves 2 and 3 are non-monotonic.

$$k_{\max} = k_0 \left(\frac{k_0}{A}\right)^{\exp(-e\beta_{k_{\max}} t/A)-1} \tag{5}$$

where t is the time; A is the upper asymptote of both k_{min} and k_{max}; k_0 is a parameter that fixes both k_{min} and k_{max} to a particular value at $t = 0$; and $\beta_{k_{min}}$ and $\beta_{k_{max}}$ are the maximum growth rates of k_{min} and k_{max}, respectively. At any given time point, $k_{min} \leq k_{max}$ by definition: this condition is satisfied for all $t \geq 0$ (but not for $t < 0$) when we set $\beta_{k_{max}}$ to be larger than $\beta_{k_{min}}$, because of both curves being fixed to the same value k_0 at $t = 0$. This model, therefore, requires that k_{min} and k_{max} only be evaluated over non-negative time points.

The parameters $\beta_{k_{min}}$ and $\beta_{k_{max}}$ determine the shapes of the diffusion and innovation curves, respectively. They indicate maximum (absolute) growth rates, which occur at the inflection points of k_{min} and k_{max}. In a Gompertz function, inflection occurs at $1/e = 36.79\%$ of the upper asymptote value A, regardless of the parameter values. Therefore, curves with a larger maximum growth rate are steeper and approach the asymptote more rapidly. By adjusting $\beta_{k_{min}}$ and $\beta_{k_{max}}$, we can model the variation in relative rates of cultural innovation and diffusion across different types of society, as well as the impact of these different cultural trajectories on heritability. Many factors can affect these rates of innovation and diffusion (De, Nau, & Gelfand, 2017; Gelfand, 2018; Kolodny et al., 2015; Muthukrishna & Henrich, 2016; Muthukrishna & Schaller, 2020; Rogers, 2003; Schaller & Muthukrishna, in press). For example, a "tight" society may have low rates of radical innovation, high rates of incremental innovation, and high rates of diffusion of these innovations, whereas a "loose" society may have high rates of radical innovation but lower rates of diffusion (Gelfand, 2018). Other societies may be high or low in both. In Figure 5, we illustrate a 2×2 of societies that vary in their innovation and diffusion rates.

Heritability is computed using the same procedure as the variance partitioning model, but dynamically. Following equation (2), we compute the cultural variance component V_c at time t by taking the variance of the uniform distribution over the interval $[k_{max}, k_{max}]$ at time t. Phenotypic variance explained by the environment (i.e., ecology and culture) at time t therefore takes the following form:

$$V_{E(t)} = V_e + \frac{1}{12}(k_{\max(t)} - k_{\min(t)})^2 \tag{6}$$

where V_e is again the ecological variance component, which we treat as static compared to the rapidly changing cultural environment. By selecting values for both V_e and V_G (the genetic variance component), we can thus compute

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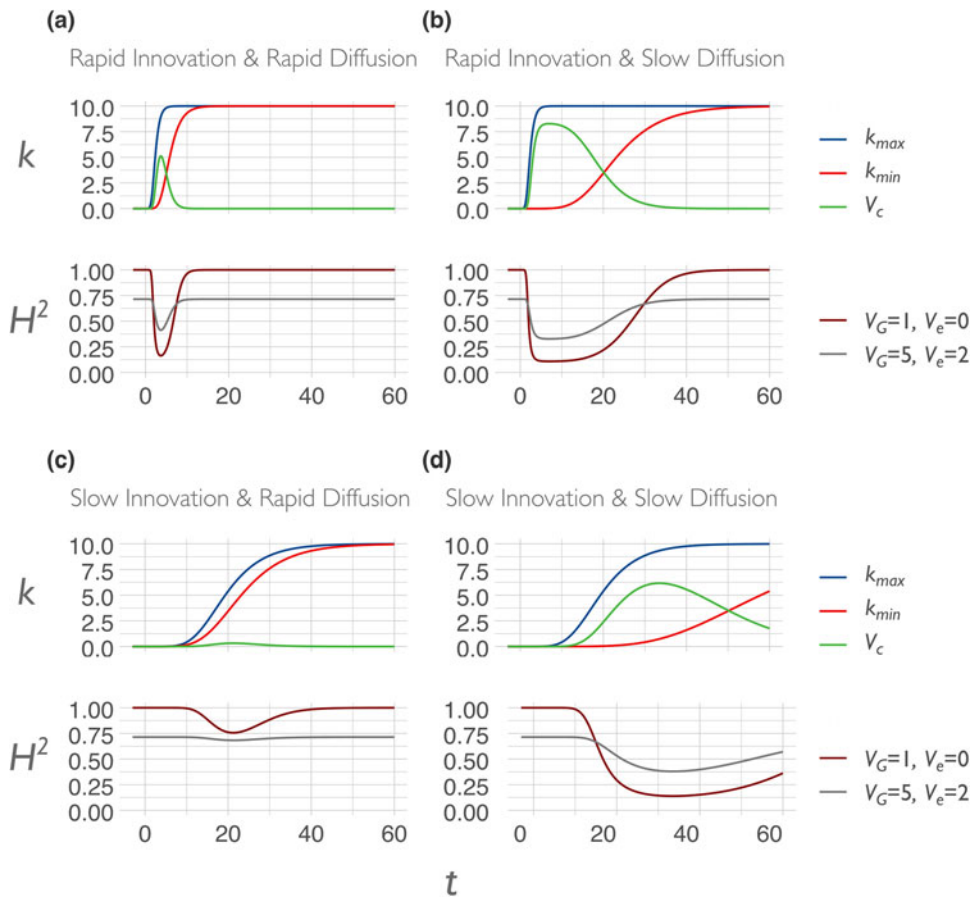

Figure 5. Output examples of the cultural dynamics model, for four prototypical societies. For each society, the upper panel displays the change over time in the lower ($k_{\min}$) and upper bounds ($k_{\max}$) of the society's cultural complexity as it pertains to some given phenotypic trait, together with the variance explained by the cultural environment V_c, which is the theoretically expected variance of the uniform distribution over the interval [$k_{\min}$, $k_{\max}$]. The lower panel displays the change in heritability over time under two different settings for genetic and ecological variance components. Global model parameters are set to $A = 10$ and $W_0 = 10^{-6}$. (a) A society that innovates rapidly while also diffusing these innovations across the population rapidly; maximum growth rates of $k_{\min}$ and $k_{\max}$ are $\beta_{k_{\min}} = 2$ and $\beta_{k_{\max}} = 5$, respectively. (b) A society that innovates rapidly but whose innovations are slow to diffuse; $\beta_{k_{\min}} = 0.5$ and $\beta_{k_{\max}} = 5$. (c) A society that innovates only gradually but whose innovations diffuse quickly; $\beta_{k_{\min}} = 0.5$ and $\beta_{k_{\max}} = 0.6$. (d) A society that innovates only gradually and whose innovations take even longer to diffuse; $\beta_{k_{\min}} = 0.2$ and $\beta_{k_{\max}} = 0.6$.

broad-sense heritability at time t in the same manner as (3):

$$H^2_{(t)} = \frac{V_G}{V_G + V_e + (1/12)(k_{\max(t)} - k_{\min(t)})^2} \tag{7}$$

These dynamics of changing environmental variance and heritability were only implicitly included in the variance partitioning model above (see Fig. 4c) but are represented here explicitly. The effects of innovation and diffusion on cultural variance and heritability for each society are illustrated in the lower panels of Figure 5. These results show, for example, that societies with both rapid innovation and rapid diffusion of these innovations should experience large transient changes in heritability (Fig. 5a), while a combination of slow innovation and rapid diffusion has little impact on heritability (Fig. 5c). Societies with rapid innovation but long lags in diffusion will experience large changes in heritability over longer timescales (Fig. 5b), as will societies with slow innovation and slow diffusion (Fig. 5d). In reality, there may be multiple relevant cultural innovations for some given phenotypic trait at any given point in time – under such a scenario, heritability would tend to fluctuate around some intermediate value rather than traverse the full range depicted in the lower panels of Figure 5. The output of this model predicts that on average, heritability of culturally transmissible traits should be higher in more homogeneous (tight or less clustered) societies than in less homogeneous (loose or more clustered) societies, as discussed in section 2.2.3.

Open Peer Commentary

The evolutionary dance between culture, genes, and everything in between

Abdel Abdellaoui

Department of Psychiatry, Amsterdam UMC University of Amsterdam, 1100 DD Amsterdam, The Netherlands.
a.abdellaoui@amsterdamumc.nl

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Abstract

Uchiyama et al. describe how a more complete measurement of the dynamic nature of culture could help us unmask the true richness of genetic effects on behaviour. I underscore this notion

here by reflecting on the role that the dynamic relationship between culture and DNA has played in our evolutionary history and will play in our evolutionary future.

As with everything else in the universe, the protein building blocks encoded in our DNA are not very meaningful on their own. They gain their meaning as part of the complex and highly interconnected machinery out of which our minds and behaviours emerge. Here, they transcend their being as an individual particle, and become part of elementary processes that are in constant motion and communication with each other and their surroundings. Out of these processes, multiple levels of higher order processes emerge that also find their meaning in their interconnected and dynamic nature. We, human beings, are such a higher order process and work in a similar fashion: We are individually meaningless and function only as interconnected groups out of which culture emerges, the highest order process known to us. Culture is the social environment where the stories of our lives unfold. Like all other underlying processes, culture is in constant development and engages in an ongoing evolutionary dance with all the underlying processes down to DNA itself.

Uchiyama et al. deliver an important message about the necessity for cultural context in interpreting and understanding the relationship between genes and behaviour. The nature and magnitude of genetic effects are always estimated against a cultural background. This cultural background is dynamic as it keeps changing in its content (cultural innovation) and its reach (cultural diffusion) across time and space. This makes any estimation of heritability or genetic effects only a snapshot of the dynamic relationship between DNA and behaviour. These snapshots have the potential to be informative about our biology and environment, but can be misleading if one is not aware of its dynamic nature. Uchiyama et al. emphasize the importance for genetics research to incorporate this dynamic nature, which could indeed impact our interpretations greatly. Cultural change does not only make certain genetic effects more or less visible for researchers, however, it also does so for nature itself, where it can make genetic effects more deleterious or more advantageous in an evolutionary context. This can turn culture into a, at times, potent natural selection pressure.

The close relationship between our cultural and genetic evolution has led to several revolutions in our evolutionary history which helped us take over this planet. One of the earlier major innovations occurred ~1.9 million years ago when *Homo erectus* started cooking its food, allowing the first human ancestor that made the cross to Eurasia to spend much less time chewing and digesting (Wrangham, 2009). This resulted in a major shift in the gene pool, leading to a smaller digestive tract, which may have freed up energy for a larger brain to develop, capable of more sophisticated cultural innovations. Another major innovation related to our food supply was the agricultural revolution ~12,000 years ago, which has left traces that we can detect in our genomic sequence data today, such as the ability to digest lactose into adulthood (Laland, Odling-Smee, & Myles, 2010). By domesticating plants and animals, our basic dietary needs were more easily met, which allowed us to live in much larger groups and freed up more time for cultural innovations. This gave rise to the first civilizations and more advanced modes of communication, such as writing, substantially expanding the reach of cultural diffusion. A more recent major innovation that greatly advanced our abilities for highly advanced innovations is the Scientific Revolution (~500 years ago), out of which several other major innovations were born soon thereafter: the Industrial Revolution (~300 years ago) and the Information Revolution (ongoing).

The evolution of our culture has brought us to extraordinary heights with respect to innovative potential and far-reaching cultural diffusion. We are the first life-form on earth able to read out our own DNA sequence and observe the results of our own evolutionary history on a molecular level. We are able to estimate genetic effects on behavioural, mental, and physical health outcomes, but have only done that for very specific cultural circumstances so far (Mills & Rahal, 2019). It is today much easier to reliably measure our DNA sequence than to capture the dynamic nature of culture in a research setting. By incorporating culture across time and space into our genetic association studies, as suggested by Uchiyama et al., we could estimate how different genes have different favourable or unfavourable effects across different cultural contexts, which may also increase our appreciation for the value of genetic variation. Just as you need different types of proteins to make cells, you need different types of humans for well-functioning cultures to arise. On a larger timescale, where culture acts as selection pressures, nature needs variation to pick parts from to build the machinery of the next generations. Possibly, this type of selection focuses largely on existing common variation rather than rare new mutations. Behaviour is influenced by many genetic variants with small effects, many of them common in the population (Abdellaoui & Verweij, 2021), creating a big space of variation for culture to choose from.

Some of our latest cultural innovations allow us to influence genes in our offspring, and thereby our evolution, much more directly. Genetic effect estimates are already being used to select embryos (Turley et al., 2021), and genetic engineering is developing at a rapid pace (Musunuru et al., 2021). Without a full appreciation for the value of genetic variation, these kinds of interventions will likely be used to decrease disease risk, which will reduce genetic variation. By decreasing genetic variation, we risk decreasing room for adaptation and making the evolutionary dance between genes and culture more rigid. A substantial amount of genetic effects associated with disease risk overlaps with dimensions of healthy variation – autism and bipolar disorder, for example, show positive genetic correlations with IQ and higher education, respectively (Abdellaoui & Verweij, 2021). What is considered healthy behaviour depends on social circumstances and norms, which vary greatly across time and space. Geneticists should aim to incorporate culture in all its richness in order to make us all better appreciate genetic variation in all its richness.

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Cultural evolution: The third component of mental illness heritability

Davide Amato[a,b]

[a]Department of Neuroscience, Medical University of South Carolina, Charleston, SC 29425, USA and [b]Department of Psychiatry and Psychotherapy, Charité – Universitätsmedizin Berlin, 10117 Berlin, Germany.
amatod@musc.edu

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Abstract

Uchiyama et al. provide a theoretical framework to explain the gap between reported gene–environment interactions and real-life epidemiological statistics. Through cultural evolution, informed behavioral approaches mitigate the impact of environmental risk on disease onset. Similarly, here we propose that fostering certain behavioral traits, transmitted culturally or through access to scientific knowledge, could confer resilience to mental illnesses such as schizophrenia.

Gene–environment interaction is a concept in genetic epidemiology postulating that exposure to certain environmental conditions elicits expression of a phenotype (i.e., a disease state) in persons with specific gene alleles. Indeed, most diseases, including mental disorders, stem from an interaction between genes and the environment, rather than one or more genes alone. Uchiyama et al. observe that the gene–environment interaction is not yet an optimal equation to reliably predict disease occurrence. They argue that gene and environment are impactfully regulated by cultural evolution, or the density of cultural subgroups within a population that, along with diverse culturally dependent behaviors, significantly affect heritability estimates. In simpler terms, if environmental factors including pollution, pathogen, and pesticide levels, or dietary choices, affect the expression of susceptibility genes (Hunter, 2005; Rojas-Rueda et al., 2021), then according to Uchiyama et al. different culturally driven approaches to the environment along with the use of advanced technologies that are protective against environmental risk factors can further regulate gene responses. As an example, application of sunscreen alleviates the risk conferred by ultraviolet (UV) exposure on development of skin cancer in genetically at-risk populations. Thus cultural evolution can mitigate the impact of environmental risk on disease prevalence.

The need to describe additional factors underlying disease onset is implicitly recognized in studies of mental disorders. For example, schizophrenia is a mental illness resulting from a highly heritable albeit poorly understood genetic component. In the case of schizophrenia genetic risk variants include both rare genes and multiple common risk alleles with small effect sizes (Schizophrenia Working Group of the Psychiatric Genomics, 2014). Despite this genetic contribution, the majority (∼90%) of individuals with schizophrenia have no parents with schizophrenia, and ∼40% of monozygotic twins of schizophrenia patients remain healthy (Akdeniz, Tost, & Meyer-Lindenberg, 2014a; Svrakic, Zorumski, Svrakic, Zwir, & Cloninger, 2013). This evidence illustrates the power of environmental factors, including prenatal maternal stress, poor nutrition, infection or birth complications, as well as adolescent and adulthood behaviors such as addictive drug consumption and social factors such as emigration and minority status (van Os, Kenis, & Rutten, 2010), in influencing gene expression leading to schizophrenia. These important risk factors share the potential to function as triggers to reveal the behavioral expression of mental illness, but they do not explain why only about 1% of the global population is consistently impacted by these non-genetic factors.

It is understood that reducing environmental variation increases the calculated heritability of disease (Stoolmiller, 1999) and that populations living in geographical areas with high cultural homogeneity will exhibit higher heritability outcomes compared with culturally diverse populations. Conversely, areas with diversified cultural landscapes are expected to be exposed to more diverse culturally informed behavioral models that, if rapidly diffused, may confer protection against vulnerable genes and environmental risk factors. Thus, while many factors may underlie geographical differences in the prevalence of schizophrenia (Charlson et al., 2018; He et al., 2020; Jablensky et al., 1992), including socioeconomics, population density, and healthcare practices, based on the argument elaborated here perhaps cultural evolution, as defined by Uchiyama et al. should also be considered as a contributing factor in the gene–environment equation.

Examples of behaviors that have been shown to vary by culture globally include infant–caregiver attachment styles (Agishtein & Brumbaugh, 2013) and maternal and/or infant healthcare (Beck et al., 2010; Lee, Talegawkar, Merialdi, & Caulfield, 2013; Wang et al., 2020; Yaya & Ghose, 2019). In these examples, increased social engagement within families and/or communities and improved maternal and infant health would be expected to protect against risk factors known to increase the incidence of schizophrenia such as social isolation (Michalska da Rocha, Rhodes, Vasilopoulou, & Hutton, 2018; Veling, Hoek, Selten, & Susser, 2011), birth complications, maternal stress (Davies et al., 2020; Lipner, Murphy, & Ellman, 2019), and nutrition in infants and young children (Wahlbeck, Forsen, Osmond, Barker, & Eriksson, 2001). Interestingly, this culturally driven biological protection may operate: (1) at the behavioral level (i.e., through healthier behavioral approaches to cope with environmental risks factors) and/or (2) through specific forms of neural plasticity. Moreover, protective behaviors established during childhood could alleviate the risk of detrimental behaviors including poor sleep practices or use of addictive substances, which may be linked to psychotic symptoms later in life (Evins, Green, Kane, & Murray, 2012; Waite, Sheaves, Isham, Reeve, & Freeman, 2020). Similarly, a healthier social structure within families and societies, and improved support for children and caregivers could reduce the incidence of psychological and physical traumas linked to development of psychotic symptoms (Popovic et al., 2019). Brain mechanisms through which culturally conditioned behaviors may modulate the expression of psychosis include changes in brain structure or compensatory alterations in molecular mechanisms of synaptic transmission. This hypothesis is bolstered by

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the fact that deficits in social interaction and communication are core pathologies in patients with schizophrenia (Fett et al., 2011; Green, 1996; Green, Horan, & Lee, 2015; Green, Waldron, & Coltheart, 2007; van Os et al., 2010) and that cortical and subcortical brain structure and function are altered by mild or chronic stress such as social deprivation or exclusion (Akdeniz et al., 2014b; Gianaros et al., 2007; Liston, McEwen, & Casey, 2009; Luethi, Meier, & Sandi, 2008).

Based on this hypothesis, application of healthier cultural models of communication and social engagement could deter the strengthening of neural pathways that permit psychosis (Canli & Lesch, 2007; Casey, Giedd, & Thomas, 2000; Chiao & Blizinsky, 2010; Cook & Black, 2012; Miller et al., 2009). Cultural factors, such as tolerance for diversity and diffusion of beneficial habits and scientific knowledge may significantly compensate for both genetic risk variants and exposure to environmental risks, conferring resilience through culturally informed behaviors. Consideration of cultural evolution theory could help to explain the consistent incidence of schizophrenia at ∼1%, resulting from the presence of both genetic and environmental risks *and the absence* of protective behaviors. It is tempting to hypothesize that globalization, and rapid knowledge transfer through modern internet and telecommunications platforms could aid in more efficient cultural evolution across geographical distance.

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Evolving the blank slate

Timothy C. Bates

Department of Psychology, University of Edinburgh, Edinburgh EH8 9JZ, UK.
tim.bates@ed.ac.uk

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Abstract

We support Uchiyama et al. in the value of genetics, sample diversification, and context measurement. Against the example of vitamins, we highlight the intransigence of many phenotypes. We caution that while culture can mask genetic differences, the dependence of behaviour on genetics is reinvented and unmasked by novel challenges across generations.

Most behaviour geneticists will react with pleasure and recognition of their programmatic approach captured in Uchiyama et al. Figure 2. As Uchiyama et al. note, behaviour genetics developed G × SES models (Purcell, 2002), integrating these with measured moderators (Tucker-Drob & Bates, 2016), testing these between cultures (Bates, Hansell, Martin, & Wright, 2016; Bates, Lewis, & Weiss, 2013), including non-WEIRD (western, educated, industrialized, rich, and democratic) cultures (Hur & Bates, 2019). It also developed methods and theory to visualise changes in heritability over time (e.g., Briley, Harden, Bates, & Tucker-Drob, 2015). This is exemplified in, for example, cross-cultural + longitudinal + genetically informed work on dyslexia (Samuelsson et al., 2008) and continues with non-transmitted gene measures revealing parental competence and social-network niche construction as envisaged in the target (Bates et al., 2018). In this sense, Uchiyama et al.'s superbly curated encyclopaedia of cutting-edge behaviour genetic, evolutionary and cultural research shows Uchiyama et al.'s future is "already here, just not evenly distributed." Wider use of this approach would yield significant benefits (Sherlock & Zietsch, 2018) (*note*: since the 70s, Eaves and colleagues presaged and implemented much of this programme from cultural transmission and G × E to inspiring non-transmitted genetic models) (Eaves, 2017; Eaves, Pourcain, Smith, York, & Evans, 2014; Heath et al., 1985). Similarly integrative figures exist in evolution and in culture research of course, for example, Lumsden and Wilson (2005).

Applications and tests of bio-psycho-sociocultural models require samples that are unnecessarily scarce. For instance, there is only one test of G × SES for intelligence in Africa (Hur & Bates, 2019). We agree with Uchiyama et al. that many, many more, not fewer, genetically informative twin and molecular studies in more diverse global samples are urgently needed, if the target article's ambitions are to be achieved. Uchiyama et al. thus, highlight exciting opportunities in macro, micro, and occulted cultural clusterings combined with genetics. Moving beyond postcode and parental education, and doing this globally with measured genetics needs investment but would repay it richly. The 11-year-old in a poor, neighbourhood but who edits Wikipedia using creative commons MIT chemistry lectures, and who collaborates on zero-to-one endeavours with friends on discord is invisible under typical SES metrics. So too the canalisation or invariance of potential and capability under challenging environments is too seldom examined.

Finally, I applaud the authors for their focus on intelligence. Birthed in equal parts in education, intervention, genetics, I/O psychology, sociology, and, in part because of controversy, in methodological rigour and innovation, intelligence research reflects much of what is desired by the target article. Perhaps more than at any time in the past 40 years, however, research in this area is under pressure and this is likely a tragic cultural error. Intelligence (alongside equally under-researched traits of goal-focussed ambition and creative zeal) is essential to the origin of the wonders of invention inspiring Uchiyama et al. – from vitamin-D supplements to co-opting our cellular machinery using consciously designed mRNA, masking genetics in precisely the ways the target article highlights.

Three questions for the research programme: For how long could a population thrive if furnished with all of today's inventions and institutions, but shorn of ability-associated genetic polymorphisms? Genetic causes of ability are likely crucial to "keeping the mask up" and emphasising this seems valuable. Second, does the theory reserve sufficient space for the intransigence of phenotypes? Vitamins provide clean examples of efficient masking of genetics by invention. However, precisely because of their genetic complexity, many more phenotypes have resisted reengineering, reflected in unyielding mental diseases such as schizophrenia or depression, and, glaringly, the resolute unwillingness of IQ to yield up durable and deep increases above the effects of school exposure (Ritchie, Bates, Der, Starr, & Deary, 2013), or, even more glaring, the absence of any heritability suppressing large effect vitamin-C style intervention craved in education (e.g., Li & Bates, 2019, 2020). As Ceci (1996) noted, by realising potential, education *per se* opposes rather than advances egalitarian goals. Does the theory speak to this?

Studies corroborate the ability of environment to amplify, reduce, or even reverse G × E effects on intelligence (Tucker-Drob & Bates, 2016). What they have not shown is similar sign changes on genetic main effects. It is now possible to directly test this with measured variants. So a third question: Regarding sign-invariance of genetic effects rather than effect size changes, what fraction of DNA variants associated with traits such as cognition or reading skill do Uchiyama et al. predict will reverse their effects under conditions which raise mean educational outcomes? This has not been seen to date (Samuelsson et al., 2008), with results supporting conventional mostly additive models in genetics (Hill, Goddard, & Visscher, 2008).

This brings us to a final point. It often seems that rather than the Elephant that is culture being ignored, it is the less visible DNA-based machinery maintaining the galactically complex machinery of the body and brain that is ignored. Moreover is often implied that because genetic variance is always above zero, malleable, and dynamic, then perhaps genes place no limits on the phenotype. Can the authors speak to how they articulate the difference between the complex learning machinery that is

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the "blank slate" provided by genetics and what is written on it? Asked by a friend how he should educate his child, the originator of the blank slate idea, Locke, recognised that because knowledge was so much more taught rather than discovered by children, that precisely what was being taught was crucial to their later attainment and character. But it is less clear that this follows for the "slate" itself, as opposed to what is stored on it. A useful direction, then, would be to incorporate a well-specified distinction between changes of state in the sense of what is stored in the system, from changes to the nature of the system in the sense that vitamin-C changes the nature of the biological system, or an engineered molecule may create a *de-novo* capacity: for instance, allowing the immune system to destroy a cancer cell. Can the theory capture such distinctions? For example, school changes knowledge about the topics taught (storage), but much of the capacity to operate on that knowledge seems orthogonal to this (Engelhardt, Briley, Mann, Harden, & Tucker-Drob, 2015; Ritchie, Bates, & Deary, 2015). Widespread understanding and acceptance that genetics is at least as diverse and impactful as culture in individual differences is, perhaps, the challenge of this decade and certainly crucial to understanding many practical issues in education and society. The target article is a step towards meeting this challenge and we commend it.

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(Super-)cultural clustering explains gender differences too

Lynda G. Boothroyd[a] and Catharine P. Cross[b]

[a]Department of Psychology, Durham University, Durham DH1 1HQ, UK and [b]School of Psychology & Neuroscience, University of St Andrews, St Andrews, Fife KY16 9AJ, UK.
l.g.boothroyd@dur.ac.uk, cpc2@st-andrews.ac.uk
http://www.boothlab.org

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Abstract

The target paper shows how cultural adaptations to ecological problems can underpin "paradoxical" patterns of phenotypic variation. We argue: (1) Gendered social learning is a cultural adaptation to an ecological problem. (2) In evolutionarily novel environments, this adaptation generates arbitrary-gendered outcomes, leading to the paradoxical case of larger sex differences in more gender equal societies.

Some cross-cultural datasets show that countries with the highest levels of legal and political gender equality also show the largest sex differences in diverse measures such as personality traits, mate choice preferences, and subject or career choice (e.g., Falk and Hermle, 2018). This "paradoxical" finding has been taken as evidence that legal gender equality "frees" women and men to express different, evolved, traits (Schmitt, 2015). We argue, instead, that this pattern is similar to the complex relationship between vitamin D, skin cancer risk, and latitude discussed in the target article: It all makes sense if you consider cultural evolution and its power to shape the phenotypic landscape more broadly.

Humans are a cooperatively breeding species inhabiting diverse ecological niches (Sear, 2016). For instance, forager groups differ in the proportion of both direct care (Kramer & Veile, 2018) and calories provided by fathers (Marlowe, 2001). They also vary in reliance on forms of subsistence which are less compatible with care for very young infants (e.g., open sea hunting; Marlowe, 2007). Furthermore, it is likely that humans have adopted diverse

mating systems across our evolutionary history. As such, the ability to acquire gendered behaviour flexibly is likely essential.

Tight hormonal control of specific sex-typed behaviours is absent in humans. Instead, hormones such as testosterone (among other behavioural effects) appear to bias the process by which behaviour is acquired, by facilitating a tendency to copy the behaviour of own-gender groups (Hines, 2020). It's important to note that this apparent "own-gender" bias can itself be understood as a more simple "copy self-similar others" bias (Meltzoff, 2007; Wood, Kendal, & Flynn, 2013), where children select those "others" from among the available categories within their culture. It may also be reinforced by a tendency to copy one's playmates in societies where children are segregated by sex and/or gender (Wood et al., 2013).

Where the cultural learning of locally adaptive gendered behaviour guides individuals into one of a few niches, patterns of sex differences might be relatively easy to predict. Research on spatial cognition, for instance, has demonstrated that patterns of geographical mobility in hunting and trading may be responsible for adult sex differences in spatial cognition in some cultural contexts (Twe and Himba: Vashro, Padilla, and Cashdan, 2016) but not others (Tsimane: Trumble, Gaulin, Dunbar, Kaplan, and Gurven, 2016). When populations undergo changes, however, predicting the effects of these changes will be complex. While the introduction of schooling and concomitant reduced differences in mobility patterns among Twa children *reduced* sex differences in spatial cognition (Davis, Stack, & Cashdan, 2021), long-term settlement in the Agta *increased* sex differences in child domestic labour (Page et al., 2021). Given that both of these changes could be thought of as making a population more "WEIRD" (western, educated, industrialized, rich, and democratic), we see here that predicting the effects of a construct such as "development" or "gender equality" on sex differences is complex.

Populations with high gender equality indices would mostly be characterised as "WEIRD" societies in the target article. They represent very different ecologies from those we mentioned above. Consider industrialised labour markets; not only are gender roles less constrained by physiology than in foraging societies, but also the number of roles and behavioural and social niches an individual can occupy is vast (Smaldino, Lukaszewski, von Rueden, & Gurven, 2019). The search for locally adaptive behaviour is less constrained and therefore more complex. Where decisions are difficult, social learning will play more of a role (Morgan, Rendell, Ehn, Hoppitt, & Laland, 2012) and choices may be arbitrary. We, therefore, expect individuals to be more influenced by factors such as gender in industrialised societies than in foraging and subsistence societies, even when those industrialised societies are relatively gender egalitarian (see Anker [1998] for discussion of gendered labour in Nordic countries). The dramatic shift in computing from being a female profession to a male-dominated one in the United States and United Kingdom (Abbate, 2017) illustrates that the association of gender with behaviour in such societies is malleable and can be arbitrary.

The target paper also argues that typical units of analysis for group comparison effects, such as nation states, or racial groups, are unlikely to capture the actual cultural clustering in a population. This is likewise true for studies of variation in sex differences. However, in addition to the possibility that lower-level clusters are critical, we note that industrialised countries exist within higher-level clusters created by shared media culture (TV, advertising, social media, etc.). For instance, the outputs of film industries in Hollywood and Bollywood (which are highly gendered in both similar and different ways, e.g., Ghaznavi, Grasso, and Taylor, 2017) are both consumed across large, sometimes overlapping, swathes of the global population.

Our proposition here is in some ways similar to Wood & Eagly's (2012) argument that a society's division of labour drives stereotypes and thereby children's learning. While the actual distribution of adult roles might be observable in small groups, however, children in industrialised nations likely have their perceptions of appropriate behaviours shaped more by visual media than by the social roles of the adults around them. We have already demonstrated that, during market integration, globalised media alter preference for sexually relevant traits, reducing idealised female body and waist size (Boothroyd et al., 2020; Swami et al., 2010) even where ecological pressures such as nutritional stress should direct preferences in the opposite direction (Jucker et al., 2017). Given that high gender equality indices are associated with economic development, access to visual media (particularly advertising) might be at its most potent in shaping gendered preferences (Fisher & Jenson, 2017) when structural inequality is relatively small.

In other words, we support the claim in the target article that cultural clusters "explain intergroup differences better than genes do." By considering gender as a "self-organizing trajectory of environmental experience" like those described in the target article, we believe this insight can make sense of the "paradoxical" relationship between gender equality indices and sex differences in behaviour and personality.

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Cultural evolutionary theory is not enough: Ambiguous culture, neglect of structure, and the absence of theory in behavior genetics

Callie H. Burt

Department of Criminal Justice & Criminology, Georgia State University, Andrew Young School of Policy Studies & Center for Research on Interpersonal Violence (CRIV), Atlanta, GA 30303, USA.
cburt@gsu.edu
www.callieburt.org

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Abstract

Uchiyama et al. propose a unified model linking cultural evolutionary theory to behavior genetics (BG) to enhance generalizability, enrich explanation, and predict how social factors shape heritability estimates. A consideration of culture evolution is beneficial but insufficient for purpose. I submit that their proposed model is underdeveloped and their emphasis on heritability estimates misguided. I discuss their ambiguous conception of culture, neglect of social structure, and the lack of a general theory in BG.

In their expansive article, Uchiyama et al. address the neglect of cultural evolutionary dynamics in contemporary behavior genetics (BG) models by unification under a dual inheritance (genetics and cultural) model. I concur with their arguments about the need to incorporate cultural evolutionary theory into BG; however, I submit that their proposed model is underdeveloped and their focus on heritability estimates is misguided. I address four issues here: (1) the ambiguity in the conception of culture in the proposed dual inheritance model, (2) the model's neglect of social structure, (3) the lack of a theory of human development and behavior in BG, and (4) the questionable focus on heritability and cross-population comparisons thereof.

Uchiyama et al. correctly, in our view, fault BG approaches for their failure to adequately incorporate cultural evolutionary dynamics. To remedy that deficit, Uchiyama et al. proffer an approach that merges cultural evolutionary theory with BG. Unfortunately, the scope and adequacy of their model is limited by the ambiguity of their key concepts. What Uchiyama et al. mean by "culture" and related concepts – "cultural traits," "cultural opportunity," "cultural clusters," and "cultural inequality" – are not defined. Throughout, Uchiyama et al. seem to conceive of culture as tantamount to social environments or group dynamics, writ large. However, humans are not only cultural; we are hierarchical. The social environments in which we are born and develop include both structural and cultural forces, shaping experiences, constraints, and opportunities.

Although not defined by Uchiyama et al., culture is commonly conceptualized as shared information (beliefs, values, and skills), habits, and styles that exist in human minds and shape collective worldviews or cognitive landscapes (Hannerz, 1969; Wilson, 2010). Social structure refers to the organized hierarchy of society, including social roles and positions and the social machinery organizing and perpetuating these social positions (Sampson, 2008; Wilson, 2010). Individuals in the same society who share a culture may nonetheless face distinct experiences given their different positions in the social hierarchy – stratified along SES, sex/gender, racial/ethnic, age, and other social positions. A wealth of research highlights the importance of both social structure and culture in shaping behavioral differences, including rich depictions of culture as adaptation to structure (Anderson, 1999; Burt, 2018; Massey & Denton, 1993; Patterson & Fosse, 2015; Wilson, 2010).

In Uchiyama et al.'s model, different social structural positions and constraints are either ignored or conceived as cultural differences. This both distorts our understanding of culture and impairs our ability to adequately explain human behavioral differences within and across contexts. To fully appreciate and explain the environmental influences on divergent social outcomes of human groups, we must take into account both structure and culture as well as their interplay (Burt, 2018; Carter, 2003; Patterson & Fosse, 2015).

The more significant problem is, however, the deficit of an overarching theory in BG – specifically, a theory of human development and differences. Although Uchiyama et al. suggest that BG explicitly or implicitly adopts evolutionary theory, I disagree. Frequently, BG scholarship seems unconcerned with, even contradictory to, evolutionary theory. The fact that humans did not evolve for educational attainment, wealth, income, happiness, or even longevity – phenotypes commonly studied in BG – but to leave descendants is invariably neglected. Considerations of the goal of evolutionary processes – enhanced survival and fitness in environments that are constantly changing – is often entirely absent from contemporary BG models focusing on phenotypes that capture mainstream WEIRD-cultural notions of "social success" or high status (viz., educational attainment, IQ, risk behavior, and income). (WEIRD = western, educated, industrialized, rich, and democratic.) We are, thus, left with contradictions: BG theorizes "education-related genetics" as reflecting the

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"winners of the genetic lottery," even as BG scholarship reveals that present educational attainment and income are associated with lower not higher fertility (Belsky et al., 2016, 2018). In short, while incorporating cultural evolutionary theory is beneficial, it is not enough. BG has and continues to produce mounds of estimates and evidence, but without a guiding theory it is unable to organize and explain this evidence, including heritability estimates.

Finally, I will note I am perplexed by Uchiyama et al.'s focus on heritability estimates, in general, and their argument for comparing heritability estimates across populations, in particular, as a way of "[cutting] through the nature–nurture debate and [helping] resolve controversies" (abstract). Heritability estimates do not overcome the nature-nurture debate; they perpetuate it. Furthermore, as Uchiyama et al. recognize on occasion, comparing heritability estimates across contexts is foolhardy because heritability estimates are a function of genetic and environmental – cultural, structural, and physical – variation. However, by conceiving of culture as equivalent to the environment, Uchiyama et al. argue that controlling for culture (via proxies of culture such as "cultural looseness/tightness" or the CF_{ST}) will provide a reference that can shed light on differences in heritability across contexts. Yet, even if we could adequately control for cultural variation – and I think unlikely given that culture is multifaceted and some cultural influences are trait-specific – we would still have uncontrolled structural and physical environmental influences, all of which are constantly changing and interacting. Controlling for culture is not enough because the environment is much more comprehensive. Any results are likely to be partial, at best, and likely misleading. Reconciling BG with cultural evolutionary theory does not make heritability estimates comparable across populations.

In the end, this inability to compare heritability estimates across populations is no great loss. Trying to refine, as we and others have argued, a conceptually and methodologically problematic and ultimately not very useful (outside of controlled breeding) heritability estimate is a wasteful distraction (Burt & Simons, 2014; Turkheimer, 2011). As Turkheimer (2011) averred more than a decade ago: "In the real world of humans, in a given context everything is heritable to some extent and environmental to some other extent, but the magnitudes of the proportions are variable from situation to situation, and have nothing whatsoever to do with the causal properties of genes and environments for the trait in question, unless one is interested in the pointless null hypothesis that one of the components is zero" (p. 598). Science is about causal explanations, and heritability is not about either. The time for heritability estimates is past, with or without cultural evolutionary theory.

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Culture and causal inference: The impact of cultural differences on the generalisability of findings from Mendelian randomisation studies

Amy Campbell[a,b], Marcus R. Munafò[a,b], Hannah M. Sallis[a,b,c], Rebecca M. Pearson[d,e] and Daniel Smith[a,f]

[a]MRC Integrative Epidemiology Unit, University of Bristol, Bristol BS8 2BN, UK; [b]School of Psychological Science, University of Bristol, Bristol BS8 1TU, UK; [c]Centre for Academic Mental Health, Population Health Sciences, Bristol Medical School, University of Bristol, Bristol BS8 2BN, UK; [d]Bristol Medical School, University of Bristol, Bristol BS8 2BN, UK; [e]Faculty of Health, Psychology & Social Care, Manchester Metropolitan University, Manchester M15 6GX, UK and [f]Population Health Sciences, Bristol Medical School, University of Bristol, Bristol BS8 2BN, UK.

amy.campbell@bristol.ac.uk
marcus.munafo@bristol.ac.uk
hannah.sallis@bristol.ac.uk
r.pearson@mmu.ac.uk
dan.smith@bristol.ac.uk

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Abstract

Cultural effects can influence the results of causal genetic analyses, such as Mendelian randomisation, but the potential influences of culture on genotype–phenotype associations are not currently well understood. Different genetic variants could be associated with different phenotypes in different populations, or culture could confound or influence the direction of the association between genotypes and phenotypes in different populations.

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Uchiyama and colleagues present a comprehensive overview of how cultural evolution can influence heritability estimates. We expand on this and discuss how cultural differences can influence causal analyses, such as Mendelian randomisation (MR). MR uses genetic variants associated with an exposure as proxies for that exposure when testing exposure–outcome associations (Smith & Ebrahim, 2003). Human genotypes are fixed at conception and, according to Mendel's laws, should be randomly and independently assorted within families. Therefore, in principle and under certain assumptions, MR allows researchers to draw causal conclusions by overcoming some limitations associated with observational epidemiology – in particular, confounding, including reverse causation (Smith & Ebrahim, 2002). However, if the potential effects of culture on MR results are not adequately considered, MR assumptions and the generalisability of findings could be undermined.

MR studies of the relationship between educational attainment and body mass index (BMI) (as a marker of obesity) across high- and low-income countries illustrate this. MR studies using samples from high-income countries have found evidence for a causal effect of lower educational attainment and higher BMI (Sanderson, Davey Smith, Windmeijer, & Bowden, 2019). However, there is observational evidence for the opposite association in low-income countries (Cohen, Rai, Rehkopf, & Abrams, 2013). No MR studies have been conducted in this setting, but it is warranted given the possibility that different causal pathways may operate. Cross-cultural variability could mean that different genetic variants are associated with different phenotypes in different populations, and/or that the causal pathways between genotypes and phenotypes operate through different mechanisms.

Are different genetic variants associated with different phenotypes in different populations?

MR assumes that the genetic instrument used is robustly associated with the exposure. Polygenic risk scores (PRSs) derived from genome-wide association study (GWAS) findings are generally used as genetic instruments. Approximately 80% of existing GWASs have used samples of European ancestry (Martin et al., 2021) typically drawn from WEIRD (western, educated, industrialised, rich, and democratic) populations (Henrich, Heine, & Norenzayan, 2010). WEIRD populations differ from many other populations, so conclusions may not be generalisable. The predictive power of PRS is reduced in non-European populations (Scutari, Mackay, & Balding, 2016), which could reflect differences in allele frequency and population substructure, or differences in how phenotypes manifest in different populations (e.g., Abdellaoui & Verweij, 2021). This limits the generalisability of MR studies using European samples and the potential for using PRS derived from European populations as genetic instruments in studies sampling from other populations.

Is the pathway between the genotype and phenotype partially confounded?

MR also assumes that the association between the genetic instrument and outcome is independent of confounders. However, cultural effects can influence genetic features of populations and introduce confounding through population stratification. For example, educational attainment is influenced by both cultural (Bowles, Gintis, & Groves, 2009) and genetic (Morris, Davies, Hemani, & Davey Smith, 2020) factors and assortative mating may occur based on educational attainment (Morris et al., 2020). This means that individuals with similar educational attainment phenotypes are more likely to produce offspring together. Because of the genetic influence on educational attainment, if assortative mating occurs based on this phenotype a pair of individuals who produce offspring is likely to be more genetically similar than a random pair of individuals. Assortative mating can influence the genetic features of a population, such as allele frequency (Yengo et al., 2018) and population stratification (Sebro & Risch, 2012), which can confound the genotype–phenotype association. This is not limited to assortative mating – phenomena such as migration can also influence population genetics through culture (Rogers & Jorde, 1987). As culture influences behaviour, and behaviour influences populations' genetic features, it becomes increasingly difficult to make an estimate of a phenotype–genotype association that is not biased by cultural effects. One method often used to reduce this bias is to adjust analyses for the first 10–20 principal components of genetic architecture. However, even after accounting for 100 principal components, bias because of confounding may still be present (Abdellaoui et al., 2019), although this bias is likely to be small (Morris et al., 2020).

Is the causal pathway between the genotype and phenotype direct?

The final assumption of MR is that the genetic instrument influences the outcome solely via the exposure. If the genotype–phenotype association is mediated by another variable the interpretation of the causal pathway may be complex. For example, the effect of educational attainment and BMI may be opposite in high- versus low-income countries because the effect operates via access to resources, and the impact of access to resources may differ in each setting. Income and educational attainment increase in line with one another and as income increases, so does access to resources (Psacharopoulos & Patrinos, 2018). In high-income countries, a healthier diet composed of lean meat and fresh fruit and vegetables is generally more costly than an unhealthier diet consisting of processed meats, refined grains, and added sugars and fats. Conversely, in many low-income countries, foods with high levels of added sugars and fats are more costly, and the most affordable foods are the ones with the lowest nutrient density, such as corn (Headey & Alderman, 2019). In high-income countries, people with lower educational attainment and income are generally priced out of the "healthy" food market, which may result in higher BMI. In low-income countries, lower educational attainment may lead to decreased likelihood of buying foods high in nutrient density, leading to lower BMI. If causal pathways between genotype and phenotype are indirect and differ between populations (as with educational attainment, access to resources and diet in higher- vs. lower-income countries), interpretation of MR results may differ across these contexts and may not be generalisable.

Conclusion

MR represents an exciting opportunity to use genetic data to understand causal relationships between phenotypes. However, when interpreting results from MR studies researchers should carefully consider how cultural contexts can influence these results and their generalisability. We should also endeavour to extend the current evidence beyond samples of European ancestry to understand the full range of human genetic and cultural diversity, and how they interact.

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Changes in heritability: Unpredictable and of limited use

Stephen M. Downes[a] and Jonathan Michael Kaplan[b]

[a]Department of Philosophy, University of Utah, Salt Lake City, UT 84112, USA and [b]Philosophy Program, Oregon State University, Corvallis, OR 97331, USA.
s.downes@utah.edu; jonathan.kaplan@oregonstate.edu
https://philosophy.utah.edu/profile.php?unid=u0031428
https://liberalarts.oregonstate.edu/users/jonathan-kaplan

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Abstract

We argue that heritability estimates cannot be used to make informed judgments about the populations from which they are drawn. Furthermore, predicting changes in heritability from population changes is likely impossible, and of limited value. We add that the attempt to separate human environments into cultural and non-cultural components does not advance our understanding of the environmental multiplier effect.

While we agree with Uchiyama et al. that there are many complex interactions between cultural variation and genetic variation, and that heritability estimates are influenced by culture, we take issue with some of the ways in which the authors approach the establishment of this claim. We view the authors' central claims as consistent with work by Lewontin, Turkheimer, Dickinson, and Flynn and others who emphasize the role of environment and gene–environment interaction in the development of traits. Like these researchers, Uchiyama et al. highlight the failure of heritability estimates to provide a meaningful entry into understanding the causal role(s) played by genes in development.

We, however, have two concerns. First, we can see no way to predict how the heritability of a trait will respond to changes in the environment, independently of knowing an implausible amount about the development of the trait in question (so much so that the heritability of the trait would no longer be of any use). Depending on how development responds to environmental change, the same kind of environmental change might cause the heritability of a trait to increase, decrease, or to stay the same, and there is no way to know independently of having gained an understanding of the development of the trait across those environments, as each situation crucially depends on what the relevant genes do in those different environments. Sauce et al.'s (2018) work, discussed by Uchiyama et al., on the heritability of cognitive traits in mice is relevant here. As Uchiyama et al. note, Sauce et al. show that enriched environments *reduce* heritability of cognitive traits in mice, whereas in children in the United States we see an increase in heritability in enriched environments (see also Beam and Turkheimer, 2013). In some ways, this should not be a surprise – if we thought that cognitive ability in mice worked like maze-running ability in Cooper & Zubek's (1958) rats, we would expect high heritability in the "normal" environment, and low in the enriched one (see Fig. 1B).

In humans, heritability can depend, as Uchiyama et al. argue, on how culture features in different situations and for different traits. However, given what we say above, Uchiyama et al. should, perhaps, not agree with Harden's (2021) suggestion that one can use the heritability of a trait in humans to measure the degree and equality of "opportunity" in a culture. Increasing the degree and equality of "opportunity" *can* increase heritability, but sometimes it won't, and whether it will or not depends on the details of the trait's development across different environments given the genetic variation in that population.

This target article is further illustrated by realized ability to see well: In cultures with lots of opportunity, reasonably equally distributed, everyone gets checked by optometrists, and gets glasses if they need them, masking the effect of poor eyesight (in just the way that "sunscreen" can mask the effects of light skin in high ultraviolet [UV] regions – see sect. 2.1, paras 2, 4, and 5); in a culture with less opportunity, the effects of bad eyesight are not masked. The former will have lower heritability of realized poor eyesight than the latter. The insight that different environments can produce different interaction effects is one of the central claims in Lewontin's "Analysis of Variance and Analysis of Causes" (1974). Figures 1A and 1B illustrate different partial reaction norms corresponding to these alternate relations between environment and heritability (interpreted in this case as being about the average effect of an allele on the trait in question).

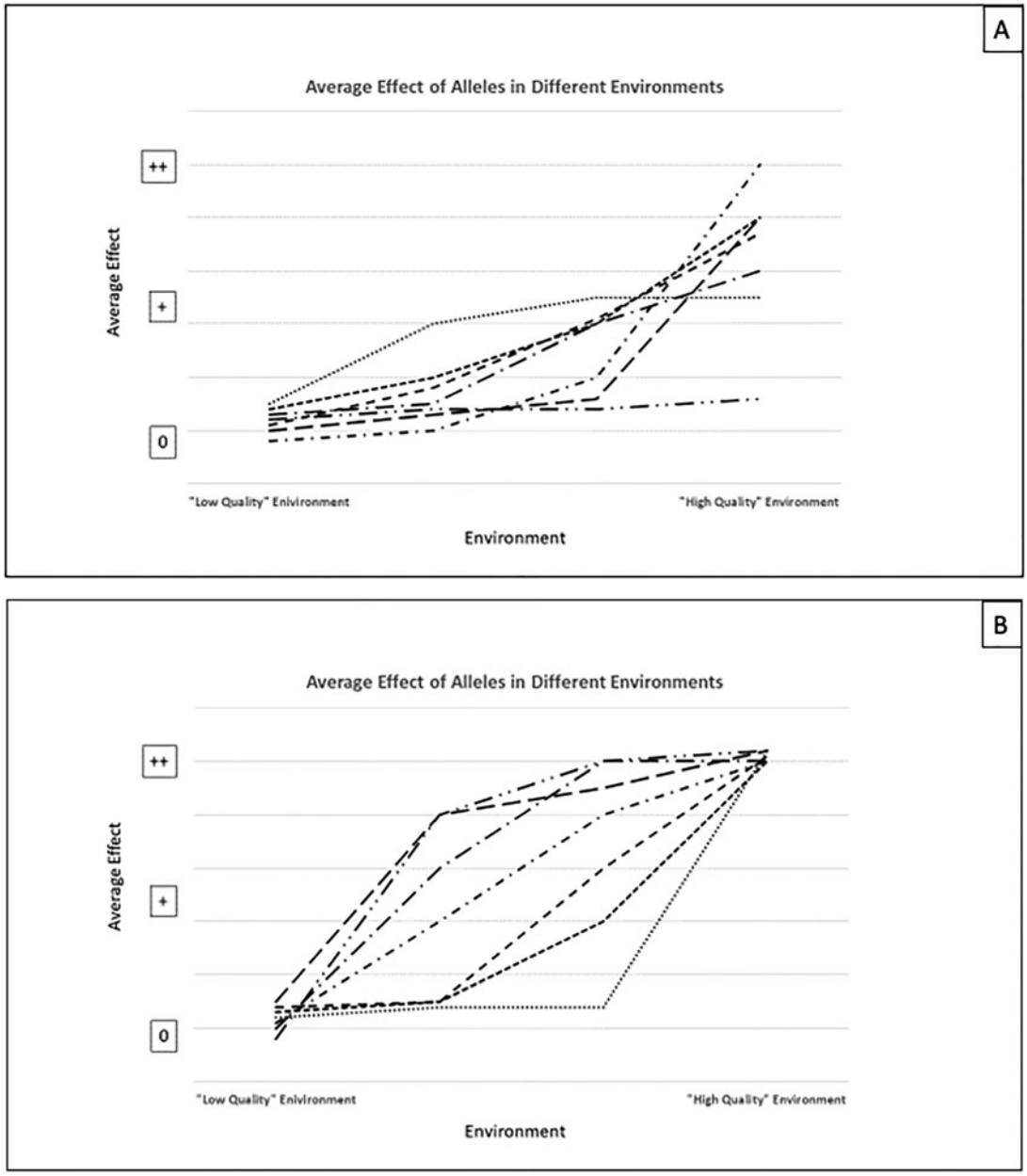

Figure 1 (Downes and Kaplan). When Uchiyama et al. suggest, first, that populations in higher quality environments (for the development of a particular trait) will tend to have higher heritability, and second, that reductions in environmental variation will increase heritability, they likely have a picture like (A) in mind. However, there is nothing implausible about (B) (adapted very loosely from Cooper & Zubek's [1958] study on maze-running ability in rats). In such a case, higher quality environments would have lower heritability, and some ways of eliminating environmental variation would actually tend to decrease heritability.

In short, without knowledge of the relevant reaction norm, one simply cannot predict how a trait will respond to changes in the environment, or how heritability estimates will change in response to environmental changes. Uchiyama et al.'s own discussion of the Flynn effect, and the current lack of any consensus about its cause, highlights this. As Lewontin forcefully noted, the only way to find out how much say IQ test-taking performance will change in response to an intervention is to *try* it, because merely knowing the heritability of the trait tells you nothing of any import (1992, p. 35). On the contrary, if one already has access to the information provided by the relevant norms of reaction (you know how organisms with particular genotypes will, in fact, respond to the proposed changes in the environment), the need for or usefulness of heritability estimates falls away, and one can instead work directly with the anticipated changes.

Our second concern is about the idea that we can separate out different parts of an organism's environment. For example, we don't see a good way to separate environments into "culture" and "non-culture" and worry that Uchiyama et al. invoke some hidden assumptions here. Lewontin's work has also been influential on this issue. In "The Organism as the Subject and Object of Evolution" (1983), he introduced the idea that organisms "construct" their own environments by determining what is relevant to them, providing the founding idea for the niche construction approach in evolutionary biology (see e.g., Laland, Oddling-Smee, and Feldman, 2000). The key takeaway here is

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that even in the apparently trivial case of temperature as a human environmental factor, there is no good way to separate the "actual" temperature outside from the experience of the temperature, as mediated by culture. More generally, there is no "natural" environment that humans face independently of the social/cultural context in which they live. We are always enmeshed in a complex culture which determines our experiences of the world. This thinking also leads us to skepticism about the authors' idea that we can rank environments by how "favorable" they are for phenotypic development, even in cases in which we can hold genotypes fixed, such as in pure strains of mice (see e.g., Crabbe, Wahlsten, and Dudek, 1999). As a result, Uchiyama et al.'s claim "For simplicity, we model cultural environmental variation as a uniform continuous distribution that is bound by k_{min}, the most unfavorable environmental state (for some given phenotype) within the experienced range of environments, and k_{max}, the most favorable" (Appendix, sect. A.1, para. 2) is far too optimistic (see e.g., Turkheimer, 2004).

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Unpackaging cultural variability in behavioral phenotypes

Ronald Fischer[a,b]

[a]Cognitive Neuroscience and Neuroinformatics Unit, D'Or Institute for Research and Education (IDOR), Rio de Janeiro, 22281-100, Brazil and [b]School of Psychology, Victoria University of Wellington, Wellington 6021, New Zealand.
Ronald.Fischer@vuw.ac.nz;
https://en.idor.org/post_scientists/ronald-fischer-phd/

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Abstract

We need better understanding of functional differences of behavioral phenotypes across cultures because cultural evolution (e.g., temporal changes in innovation within populations) is less important than culturally molded phenotypes (e.g., differences across populations) for understanding gene effects. Furthermore, changes in one behavioral domain likely have complex downstream effects in other domains, requiring careful parsing of phenotypic variability and functions.

I agree with Uchiyama et al. that cultural dynamics need greater attention, but I argue that an important avenue is to aim for a better understanding of cultural variability and implications for behavioral phenotypes. Cultural evolution research has made great progress in identifying mechanisms of cultural inheritance but has been less successful in understanding relatively stable cultural differences in human populations embedded in specific ecologies. More broadly, variability in phenotypes is important to parse because population-level differences might be an expression of culturally shaped behavioral responses based on a common genetic background, differential expression of individual and population-level genetic differences leading to culturally shaped phenotypes or more complex gene–culture interactions. Any observation of behavioral phenotypes within their specific ecological environment needs careful in-situ analysis of the underlying mechanistic causes (Tinbergen, 1963), measurement equivalence (Boer, Hanke, & He, 2018; Fischer & Karl, 2019; Fischer & Poortinga, 2018), and a rigorous exploration of gene expression (the issue of behavioral plasticity).

To use the skin cancer example, sun tan sends different social status signals depending on the economic context. Social status is tightly linked to mating success and offspring survival (Hrdy, 2000; von Rueden & Jaeggi, 2016). In agricultural settings, darker skin relative to other community members signals low status because of sun exposure while laboring in fields; whereas lighter skin signals higher social status. Skin cancer typically manifests relatively late in life after offspring have reached reproductive age (Fontanillas et al., 2021). All else being equal, individuals with higher social status and lower sun exposure will have higher reproductive success and lower risk of skin cancer. Given low life expectancy until quite recently (World Bank, 2021), low status individuals may not reach the age when cancer manifests or other health conditions have a larger impact on lifespan compared to skin cancer.

In postindustrial settings, most individuals work in service jobs and cannot afford being exposed to sun, whereas high social status individuals can afford leisurely sun exposure, tan signals social status, and increases reproductive success. Skin cancer may reduce the lifespan of high social status individuals only, which evolutionary speaking is offset by increased reproductive success. In addition to the conceptual implications, empirically the estimation of heritability estimates would be most accurate among high status individuals in tertiary sector economies, given the higher base rates.

Switching to cognitive ability studies, both cognitive ability and academic achievement motivation (as a relevant correlate with likely different genetic architecture) differ substantively within and across cultures (Chen et al., 2013, 2015), with substantive variation because of socioeconomic and cultural value differences across schools and societies (Dekker & Fischer, 2008; OECD,

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2010). Obtaining good grades in specific subjects will have different life consequences depending on cultural pressures (e.g., desirable professions, socialization goals; Bond & Lun, 2014; Chen & Stevenson, 1995), job market conditions (e.g., salaries and vacancies; Sortheix, Parker, Lechner, & Schwartz, 2019; Vecchione et al., 2016), and personal ambitions. Schools (and schooling systems) both within and across cultures employ diverse teaching methods, making some gene effects more or less likely to be expressed depending on what abilities or skills are salient at a particular point in time. In line with this reasoning, Samuelsson et al. (2008) showed that heritability estimates for reading and spelling scores increased from kindergarten to first grade among Scandinavian children, but not in US and Australian children (but note differences in spelling difficulty between English vs. other Germanic languages; Hambleton & Zenisky, 2010). In Scandinavia, social skills are emphasized in kindergarten whereas testing relevant material is only introduced at grade 1. An emphasis on social skills is related to communitarian and egalitarian values in Scandinavian societies, as a result of long-term ecological adaptations (van de Vliert, 2009). These patterns are parsimoniously explained via shifts in the contribution of relevant genes to cognitive (information processing demands, memory, attention, etc.) versus social-behavioral phenotypes (impulse control, social skills, etc.) that are differentially matched across locations.

More broadly, the concept of intelligence is culturally molded (Friedman et al., 2006; Serpell, 1979; Smith, Fischer, Vignoles, & Bond, 2013; Sternberg, 1985). Depending on the test and context (e.g., group vs. individual testing, familiarity with tester and testing context), obtaining high scores may require both specific cognitive and social skills during the testing situation, which has obvious implications for gene expression. Any measured phenotype is embedded within specific cultural testing environments and reflects the outcome of both cognitive and behavioral processes, which in turn are influenced by different gene systems (Chen, Moyzis, Lei, Chen, & Dong, 2015). Crucially, the interpretation of phenotypic test scores across groups depends on adequate levels of equivalence, including examination of functional equivalence (Fischer & Poortinga, 2018).

A final point about the cultural variability of phenotypes is related to the central assumption that cultural evolution is directional and increases adaptation. This can be questioned by evidence that cultural innovation is neutral (e.g., does not enhance individual or group fitness) or maladaptive for either the individual or community (e.g., shifts in health behaviors and reproductive patterns in industrial and postindustrial societies; cargo cults; social hierarchy systems). Cultural innovations such as introducing safe drinking water may lead to greater birthrates and malnutrition because of shifting behavioral strategies (Gibson & Mace, 2006), hence cultural innovations in one domain might be associated with complex adaptations across the behavioral repertoire of a group, with possible non-adaptive downstream effects (Laland, 2017; Laland et al., 2015). I already touched upon sun tans: In industrial societies cultural innovations such as tanning studies increase social status and mating chances because of social signaling, with the negative effects of skin cancer possibly being offset by late onset, often well past prime reproductive age (Fontanillas et al., 2021). We need more focused examinations of long-term cultural differences, specifically an understanding of the functionality of behavioral phenotypes within their relevant ecological environments and then link these directly to relevant (poly)genic candidate models. In summary, I agree that cultural variability matters, but understanding the interaction between culture and genetics requires more careful attention to the phenotypic expressions before firm conclusions on cultural evolution effects can be made.

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Cultural dynamics add multiple layers of complexity to behavioural genetics

Laurel Fogarty[a] and Nicole Creanza[b]

[a]Department of Human Behavior, Ecology, and Culture, Max Planck Institute for Evolutionary Anthropology, 04103 Leipzig, Germany and [b]Department of Biological Sciences, Vanderbilt University, Nashville, TN 37212, USA.
laurel_fogarty@eva.mpg.de;
https://www.eva.mpg.de/ecology/staff/laurel-fogarty/index.html
nicole.creanza@vanderbilt.edu; http://nicolecreanza.com

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Abstract

As emphasized in early cultural evolutionary theory, understanding heritability of human traits – especially, behavioural traits – is difficult. The target article describes important ways that culture can enhance, or obscure, signatures of heritability in genetic studies. Here, we discuss the utility of calculating heritability for behavioural traits influenced by cultural evolution and point to conceptual and technical complications to consider in future models.

The target article takes a nuanced look at a fundamentally important problem of the genomics era: How the cultural environment and gene–culture coevolution make it difficult to understand the genetic underpinnings of human behaviour.

However, heritability is a murky concept for human traits, particularly behavioural traits. This is the case for many reasons, not least the complicating effects of culture. Here, we consider, first, whether a conclusion of this paper is not that we need to consider culture when we calculate heritability, but rather that attempting to calculate heritability for culturally complex human traits might add little to our understanding of human evolution and behaviour. And second, that heritability, influenced by culture, is conceptually and technically more complicated than the models in the appendix suggest.

In the modelling section (Appendix 7), genotype-by-environment (G × E) interactions are excluded for simplicity. However, as discussed at length elsewhere (Feldman & Lewontin, 1975; Feldman & Ramachandran, 2018; Lewontin, 1974), it is difficult or impossible to ignore G × E interactions for most complex human behavioural traits, and there is no simple way to understand the relative importance of genes and environment in the determination of phenotype when the two interact. Furthermore, as shown in foundational papers on cultural evolution (e.g., Cavalli-Sforza & Feldman, 1973a, 1973b), cultural transmission can distort the signatures of genetic heritability and deepen the impact of G × E interactions, further obscuring the relationship between heritability and family phenotypic resemblances. The importance of cultural inheritance and genotype-by-culture interactions, and the extent to which these affect or even invalidate inferences derived from calculations of heritability in the case of human behavioural traits, is difficult to overstate.

The example given in the modelling section – the trait of melanin production in response to UV exposure – illustrates these issues. The authors point out that this trait has genetic, environmental, and cultural components, but we must also consider that these components interact. Sunscreen absence or presence might have a different effect in different environments but also, for example, the cultural trait of sunscreen use might be most likely to spread to those who have a genetic lack of melanin and high-UV exposure, and populations lacking sunscreen might implement other cultural interventions, such as hats or clothing. This means that the expression of the trait of melanin production is jointly governed by genetics, cultural context, the physical environment, and the numerous complex interactions between those components. As the authors note, the heritability of such a trait is not fully captured by equation (3) without terms accounting for those interactions. Quantitative genetic models were often developed for situations with relatively controlled environments, such as artificial breeding programmes, and G × E interactions could often be safely ignored in these contexts (Falconer & Mackay, 1996). For phenotypes influenced by culture, this is rarely the case, requiring justification beyond simplification of the model.

It may be helpful to ask: How can we use the measures of heritability derived in the target article? Heritability is a trait-specific, population-specific measure and is not, alone, globally informative. Narrow-sense heritability is often used, for example, to assess the response of a trait to selection in a given population in a given environment (e.g., Lande, 1979). The models in the target article, then, aim to assess the effect of a relevant cultural influence on trait heritability and to improve our understanding of the evolution of these traits. We suggest that there are some technical details that require cautious consideration, implementation, and interpretation to approach this aim. First, the relationship between heritability and the response to selection (and other theoretical aspects of phenotypic evolution) depends on the assumption of a Gaussian phenotypic distribution (see e.g., Karlin, 1988; de Villemereuil, Schielzeth, Nakagawa, & Morrissey, 2016). This assumption can be violated by this model because the cultural phenotypic contribution is modelled as a bounded uniform distribution, and the sum of the Gaussian genotypic and environmental contributions and the uniform cultural contribution need not be normal. Second, the use of phenotypic evolution models, for example, for retrospective selection studies, relies on the assumption that phenotypic and genotypic variances remain constant over time (Schluter, 1984; but see e.g., Turelli, 1988). Models involving dynamically changing phenotypic variances involve complex departures from standard theory (e.g., Gilpin & Feldman, 2019; Karlin, 1988) and the ways in which these variances are free to change in real systems are not straightforward (Arnold, Bürger, Hohenlohe, Ajie, & Jones, 2008). Adding phenotypic variance fluctuations over time in this model must, thus, also be justified by showing why the assumptions that warrant holding phenotypic variances constant in the absence of culture (e.g., logarithmic metric scales) do not apply to those changes caused by culture.

Finally, it is important to note that the heritable component of a trait with culturally evolving influences is not just genetic, it is also

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cultural. Therefore, the measure of interest for practical purposes should include the heritable cultural component in the numerator in equation (3), alongside the heritable genetic components. The ability of a trait to respond to selection is determined by how much variation in that trait is amenable to selection – in other words, as pointed out by Danchin and Wagner (2010), the important value to consider is not just the genetic variance but the heritable variance in total. Separating the environmental component of heritability into cultural and ecological components is an important step in the complicated process of modelling cultural influences on heritability. However, cultural traits can alter selection pressures on genetic traits (Feldman & Zhivotovsky, 1992; Laland, Kumm, & Feldman, 1995), influence mating patterns causing hidden population structure (Creanza & Feldman, 2014), be transmitted beyond the family unit via oblique or horizontal transmission, and alter the parameters of their own evolution in multiple ways (e.g., Creanza, Fogarty, & Feldman, 2012; Fogarty, Creanza, & Feldman, 2013, 2019) all of which could have profound effects on how we understand, calculate, and use heritability.

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Heritability is a poor, if not unhelpful, measure of complex human behavioral processes

Agustín Fuentes[a] and Kevin Bird[b]

[a]Department of Anthropology, Princeton University, Princeton, NJ 08540, USA and [b]Department of Horticulture, Michigan State University, East Lansing, MI 48824, USA.
afuentes2@princeton.edu; birdkevi@msu.edu
https://anthropology.princeton.edu/people/faculty/agustin-fuentes; https://www.canr.msu.edu/people/kevin_bird

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Abstract

Heritability is not a measure of the relative contribution of nature *vis-à-vis* nurture, nor is it the phenotypic variance explained by or because of genetic variance. Heritability is a correlative value. The evolutionary and developmental processes associated with human culture challenge the use of "heritability" for understanding human behavior.

Much of what are referred to as "traits" in behavioral genetics and cultural evolution are not bounded entities or structurally and functionally individual units of transmission or specific targets of selection. Human culture, as an evolutionary and developmental process, calls into question the very meaning and significance of "heritability" for understanding why and how humans do what they do (Fuentes 2016; Jansson, Aguilar, Acerbi, & Enquist, 2021; Lewens, 2015; Rosenberg, Edge, Pritchard, & Feldman, 2019; Stotz, 2010; Uller & Laland, 2019).

Uchiyama et al.'s goal of offering a more nuanced understanding of the interaction between "genes" and "culture" is laudable and partially achieved. As is hinted at throughout the article, the focus on explaining the relative genetic or cultural contribution to variation in traits may be misrepresenting the actual dynamics, developmental, social/experiential, and physiological processes interacting to produce the measured target. This is particularly salient in the case of human behavioral-cognitive-developmental-culturally mediated/structured parameters, such as IQ. Figure 2 of the target article ("Predicting Heritability?") identifies three key contributing processes: variability in the trait, variability in environment and culture, and variability in genes. Uchiyama et al. acknowledge that "Psychological and behavioral phenotypes

are typically the outcome of a complex network of interactions that involve all these factors" (Figure 2 legend). but still graphically represent each of the three as if they are processually independent as analytical units. This is despite much in the article arguing against such simplistic representation. This kind of "particulate and separate" – with a nod to "we know in reality it is not this simple" – approach is exactly the problem at hand, and leads to substantive errors of interpretation and understanding.

The continued misuse of "heritability" facilitates such errors. Uchiyama et al. note heritability is not a "measure of the relative contribution of nature vis-à-vis nurture" (sect. 2, para. 1), but their description of it as phenotypic variance *explained by* or *because of* genetic variance is also misleading because it implies causality. Falconer (1967) notes heritability is the ratio of additive genetic variance and phenotypic variance and that it is the regression of breeding value on phenotypic value (Falconer, 1967, p. 165). Kempthorne (1978) argues that an analysis of variance (e.g., heritability estimates from twin studies) can tell us nothing about causation and we should abandon phrasing like *X*% of variance is "because of" *Y* and use the more accurate *X*% of variance is "associated linearly" with *Y* (Kempthorne, 1978). Both descriptions make clear that heritability is a correlative value. The extent to which a linear association can be interpreted as a causal relationship depends on properly controlling for confounding variables. In plant breeding, this is a reasonable inference because of the ability to randomize environments with respect to genotype through experimental design and replication of genotypes (Kempthorne, 1978). In human genetics, this is not possible and several processes complicate causal interpretation of heritability estimates (Briley et al., 2019).

Genome-wide association studies (GWASs) and polygenic scores for human behavioral traits do not rectify the heritability problem. GWAS loci are fundamentally correlative and although the environment has no direct influence on nucleotide sequence, the processes that lead to a correlation between a genetic variant and a trait are not necessarily because of genetic causation. It is difficult to interpret polygenic scores as unconditionally providing strong information about genetic causation when estimates frequently contain effects of non-transmitted parental alleles (Kong et al., 2018), and effect sizes vary across age, sex, and socioeconomic status (Mostafavi et al., 2020). GWAS samples show inherent bias based on selective participation (Tyrrell et al., 2021), and disentangling the genetic, environmental, and cultural contribution to differing polygenic scores between populations is difficult or impossible (Rosenberg et al., 2019). Mathieson (2021) extends the omnigenic model, arguing that polygenic scores show differential prediction because the effects of peripheral genes (those that affect traits indirectly through interactions with other genes) differ across populations. Mathieson further argues that peripheral environmental factors (those that indirectly affect traits through interaction with other environmental factors) differ in their effect across populations. Culture provides an explanation for how this may occur because cultural factors structure and constrain the relationships between traits and environments. Uchiyama et al. use language to the effect that culture "masks" or "unmasks" genes. This implies there is a "true" genetic architecture to the trait. However, Mathieson's argument that the roles of peripheral genes and environments vary across contexts would mean that the biological and environmental underpinnings of fundamentally social traits, like education or IQ, change depending on culture, space, and time. In other words, the nature of the traits themselves is radically dynamic. We see this also in plant breeding where, for example, yield of corn plants is related to multiple factors such as nitrogen uptake, resistance to drought, leaf angle, plant architecture, and so on, but the contribution of these other traits to yield varies greatly by location and year (Tucker et al., 2020).

Culture systematically structures the types of shared and unshared environments experienced by individuals, the interactions between those shared and unshared environments, the interactions and correlations between genes and environments that can exist, and even genetic associations with a trait. Culture is a permeating confound that produces contingent, often non-causal, linear relationships between genetic relatedness and phenotypic similarity. Culture will determine the kinds of factors within a target population that influence the correlation between a genetic variant and a phenotype. When linear associations between genetic similarity and phenotypic similarity are this deeply confounded, the very meaning and significance of heritability estimates are called into question.

If, as Uchiyama et al. state, "Heritability is not a property of a trait in itself, because in the absence of a reference culture it is necessarily unstable" (sect. 6, para. 2) and "Nothing in behavioral genetics makes sense except in the light of cultural evolution" (sect. 6, para. 3), it stands to reason that heritability is a poor, if not unhelpful, measure for effective evaluation of the dynamics of complex human behavioral processes. One could read the core of the argument of this manuscript as tentatively making this case, and we suggest that it can, and should, be made more conclusively.

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The many geographical layers of culture

Friedrich M. Götz[a], Tobias Ebert[b] and Peter J. Rentfrow[c]

[a]Department of Psychology, University of British Columbia, Vancouver, British Columbia, V6T1Z4, Canada; [b]Mannheim Centre for European Social Research, University of Mannheim, 68159 Mannheim, Germany and [c]Department of Psychology, University of Cambridge, Cambridge CB2 3EB, UK.
friedrich.goetz@ubc.ca; https://psych.ubc.ca/profile/friedrich-gotz/
tobias.ebert@uni-mannheim.de;
https://www.mzes.uni-mannheim.de/d7/en/profiles/tobias-ebert
pjr39@cam.ac.uk; https://www.psd.psychol.cam.ac.uk

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Abstract

Uchiyama et al. present a dual inheritance framework for conceptualizing how behavioural genetics and cultural evolution interact and affect heritability. We posit that to achieve a holistic and nuanced representation of the cultural environment and evolution against which genetic effects should be evaluated, it is imperative to consider the multiple geographic cultural layers impacting individuals and genetic heritability.

We applaud Uchiyama and colleagues for their thoughtful and important contribution which marks a major step towards a culture-attentive, dynamic understanding of the genetic heritability of psychological and behavioural traits. In our commentary, we argue that an even richer and more comprehensive account of relevant sources of cultural influence can be accomplished by more explicitly considering geographical cultural layers within societies.

Like the authors and others before them (Cohen, 2009; Muthukrishna et al., 2020), we are convinced that few societies are culturally homogeneous and firmly espouse the notion of culture as a construct that exists in many shapes and forms (e.g., religion and social class). Here, we seek to direct attention towards the multiple geographical layers of culture. The national level might be the most obvious and accessible geographical cultural layer. However, to capture the cultural space in which everyday life experiences occur, it could be valuable to consider more granular levels, such as regions (Ebert et al., 2021; Rentfrow & Jokela, 2016), cities (Park & Peterson, 2010), or neighbourhoods (Jokela, Bleidorn, Lamb, Gosling, & Rentfrow, 2015).

Thanks to the advent of big data, researchers now have the means to make these previously hidden cultural layers visible (Obschonka, 2017; Rentfrow, 2020). Indeed, under the banner of geographical psychology, ample research has demonstrated pronounced intranational variation along cultural constructs, such as tightness–looseness (Chua, Huang, & Jin, 2019; Harrington & Gelfand, 2014), collectivism (Talhelm et al., 2014; Vandello & Cohen, 1999), and personality (Götz, Ebert, & Rentfrow, 2018; Rentfrow, Gosling, & Potter, 2008; Rentfrow, Jokela, & Lamb, 2015). Variation in such psychological constructs could affect variation in the frequency and type of cultural innovation that occurs (Harrington & Gelfand, 2014; Lee, 2017; Obschonka, Schmitt-Rodermund, Silbereisen, Gosling, & Potter, 2013). For example, regions high in cultural looseness and openness have been shown to have higher rates of inventions (Chua et al., 2019), entrepreneurship (Obschonka et al., 2013, 2015), creative capital (Jackson, Gelfand, De, & Fox, 2019), and patent production (Fritsch, Obschonka, & Wyrwich, 2019; Harrington & Gelfand, 2014).

Importantly, subnational geographical cultural units are not only smaller than countries, but also more culturally nimble. That is, although cultural changes in the country-level typically unfold over decades and often centuries (Grossmann & Varnum, 2015; Inglehart & Baker, 2000), regions or cities may experience considerable cultural shifts within shorter periods of time. For example, regional variation in the legalization of same sex marriage in the United States led to swift and substantial state-wide differences in implicit and explicit antigay bias (Ofosu, Chambers, Chen, & Hehman, 2019). Likewise, Götz et al. (2021) showed that changing amenities in cities (measured by housing prices) lead to swift and substantial changes in city-level openness.

To sum up, we readily acknowledge the importance of countries as salient and consequential containers of culture, and of households as the most nuclear cultural entity discussed by Uchiyama and colleagues. Nonetheless, we posit that to achieve a holistic and nuanced representation of the cultural environment and evolution against which genetic effects should be evaluated, it is imperative to consider the multiple geographic cultural layers impacting individuals and genetic heritability.

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The dubious precision and utility of heritability estimates

Steven J. Heine[a] and Ilan Dar-Nimrod[b]

[a]Department of Psychology, University of British Columbia, Vancouver, V6T 1Z4 BC, Canada and [b]School of Psychology, University of Sydney, Sydney 2006, Australia.
heine@psych.ubc.ca, ilan.dar-nimrod@sydney.edu.au
https://heinelab.psych.ubc.ca/person/steven-heine/
https://www.sydney.edu.au/science/about/our-people/academic-staff/ilan-dar-nimrod.html

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Abstract

Uchiyama et al. question heritability estimates in a convincing manner. We offer additional arguments to further bolster their claims, highlighting methodological issues in heritability coefficients' derivation, their misuse in various contexts, and their potential contributions to exacerbating common erroneous intuitions that have been shown to lead to deleterious social phenomena. We conclude that science should move away from using them.

We are largely in agreement with the convincing, important arguments of Uchiyama et al. As they cogently explain, culture plays an oversize role in human phenotypes, and this has been largely neglected in behavioral genetics research to date. It is not surprising that it has taken a while for researchers to appreciate the key ways in which culture is implicated in our phenotypes, as culture so often remains invisible to observers. People rarely notice culture until they encounter exotic others who are doing things differently, and still this often just leads people to appreciate how culture shapes other people, as opposed to themselves (Causadias, Vitriol, & Atkin, 2018). This culture-blindness has affected commonplace approaches in behavior genetics, but also has been exacerbated by its predominant methodological approaches and the conclusions they introduce. For example, both comparisons of monozygotic and dizygotic twins, and comparisons of adopted versus biological siblings, are conducted almost entirely within cultures because of the availability of such samples. These foundational methods of behavioral genetics have thus precluded the ability to detect the influence of culture in heritability, leading to a systemic neglect of culture in that field.

We argue that when one considers the arguments of Uchiyama et al., the accuracy of heritability estimates becomes deeply questionable, as a major contributor to the variability explained is not only neglected, but is also often erroneously attributed to a different source – genes. For example, consider the heritability of self-esteem (see Dar-Nimrod and Heine, 2011a). Self-esteem's heritability was estimated to be around 0.5, with shared environmental effects explaining a negligible amount of the variance (e.g., Roy, Neale, and Kendler, 1995). These analyses neglected the substantial cultural variability in the construct (e.g., a meta-analysis estimated the magnitude of cultural differences in self-esteem between Westerners and East Asians to be $d = 0.91$: Heine and Hamamura, 2007). The neglect of this large cultural component of self-esteem surely must contribute to an overestimation of the heritability.

Heritability estimates are often inappropriately used to establish a purported hierarchy of the degree to which genetics affects different phenotypes; for example, the estimated heritability of schizophrenia has been found to be higher than it is for depression (e.g., Wray and Gottesman, 2012). However, Uchiyama et al.'s arguments suggest that these contrasting heritability values may reflect instead that the uniformity of a culture contributes more to schizophrenia risk than it does for depression, undermining the utility of these comparisons across traits.

Another way that heritability estimates are commonly used is to provide an estimate of the ceiling of genetic variability that polygenic scores can be used to account for, often terming the residual unexplained variability that is attributed to the genes as "missing heritability." For example, a paper may conclude that because a given trait is 40% heritable, and that polygenetic scores can predict 20% of variability in that trait, then this means that the polygenic estimate is accounting for half of genetic variability (e.g., Derringer et al., 2010; Schunkert et al., 2011). However, this would also seem to be a dubious conclusion given that Uchiyama et al. make the case that the absolute value of heritability for a given sample can never be known until cultural factors are fully accounted for. Moreover, it is important to note that people do not just belong to a single monolithic cultural group, but rather belong to many overlapping subcultures that include their religion, social class, ethnic group, geographic region, and so on (e.g., Cohen, 2009). Hence, it will rarely be straightforward to identify all of a given individual's cultural influences, and thus heritability estimates will remain imprecise.

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Heritability estimates may not only be inaccurate, as discussed above, but may also introduce societal costs. People have essentialist intuitions that are particularly accessible whenever genetic contributions to phenotypes are discussed (Dar-Nimrod & Heine, 2011a, 2011b; Heine, 2017). Essentialist intuitions lead people to assume that the ultimate cause of any natural phenomenon is the result of some invisible forces that lie deep within it (Gelman, 2003); we also seem to treat many social categories like we treat natural categories (Rothbart & Taylor, 1992). Hence, people are tempted to assume that whenever you see differences between two groups, the most likely cause of those differences lies inside them, such as in their respective genomes. In contrast, however it requires more cognitive effort to consider other potential causes, such as in their cultures, their life histories, or their social environments. These intuitions can become especially problematic when we reflect on the heritability of desirable traits, which are often undergirding discussions of racism, sexism, and eugenics (e.g., Heine, Dar-Nimrod, Cheung, and Proulx, 2017). The unwarranted air of scientific confidence in the precision of heritability estimates may have contributed to these deleterious intuitions, as research indicates that those intuitions are magnified by common media portrayals of scientific findings (Dar-Nimrod et al., 2021). We are hopeful that Uchiyama et al.'s clear-eyed questioning of heritability estimates may also contribute to dampening people's reflexive urges to turn to simple genetic essentialist accounts and the harms associated with them.

The combination of the questionable practices at the basis of the derivation of heritability estimates, their misuse in some contexts (e.g., the missing heritability), and their potential contributions to sustaining erroneous, deleterious intuitions, leads us to call for discarding this metric under most circumstances. We acknowledge the potential utility for it when one restricts their heritability estimates to a specific homogeneous population from which a large enough sample has been assessed (with potential use for medical research), but we contend that, as it is used today, it brings about more damage and less precision to the scientific endeavor it was meant to serve. With growing technological capabilities and better ability to derive useful metrics, such as polygenetic scores and penetrance, we will benefit from moving away from the mirage of accurate heritability estimates.

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From the trajectory of heritability to the heritability of trajectories

Rogier A. Kievit[a], Jessica A. Logan[b] and Sara A. Hart[c,d]

[a]Cognitive Neuroscience Department, Donders Institute for Brain, Cognition and Behavior, Radboud University Medical Center, 6525 EN Nijmegen, The Netherlands; [b]Department of Educational Studies, The Ohio State University, Columbus, OH 43210, USA; [c]Department of Psychology, Florida State University, Tallahassee, FL 32308, USA and [d]Department of Psychology, Florida Center for Reading Research, Florida State University, Tallahassee, FL 32310, USA.
rogier.kievit@radboudumc.nl; logan.251@osu.edu; sahart@fsu.edu

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Abstract

Although compelling and insightful, the proposal by Uchiyama et al. largely neglects within-person change over time, arguably the central topic of interest within their framework. Longitudinal behavioural genetics modelling suggests that the heritability of trajectories is low, in contrast to high and increasing cross-sectional heritability across development. Better understanding of the mechanisms of trajectories remains a crucial outstanding challenge.

In their target article, Uchiyama et al. argue for a nuanced, integrative perspective on understanding phenotypic variation as a function of cultural and genetics dynamics. Although compelling and insightful, arguably the most important source of dynamics is neglected: within-person change over time (Molenaar, 2004). Within-person changes such as slopes across waves capture how traits develop and grow over time. They have unique variance components and contributing factors distinct from baseline individual differences. In section 4.2, Uchiyama et al. describe the role of cultural heterogeneity on heritability across development, describing the behavioural genetics literature that exists on the "development" of traits – We scare quote development because the overwhelming majority of behavioural genetics work cited by Uchiyama et al. uses cross-sectional methods. However, many of their proposed mechanisms, such as gene–environment

interactions or the impact of (changing) cultural innovation operate within individuals over time. Ignoring within-person change is a crucial omission, as only under extreme and implausible circumstances (i.e., ergodicity, Molenaar, 2004) will cross-sectional inferences generalize to (longitudinal) processes within individuals. Although comparatively rare, longitudinal behaviour genetics studies of phenotypic change do exist, commonly studied by combining latent growth curve models with quantitative genetics decomposition, and suggest two key messages germane to the target article.

First, the heritability of within-person change is generally *lower* than that of baseline or static scores (e.g., Hart et al., 2013; Hatoum, Rhee, Corley, Hewitt, & Friedman, 2018; Lyons et al., 2017; Reynolds, Finkel, Gatz, & Pedersen, 2002). In other words, the process of learning, growing, and changing across the lifespan has a stronger environmental component than any one single measurement occasion. At the same time, age-specific (i.e., cross-sectional) heritability estimates of the same skills (e.g., reading) may *increase* across the lifespan – A distinct instance of Simpson's paradox that further complicates our mechanistic understanding of phenotypic processes.

Second, to the extent that within-person changes (slopes) are heritable, empirical evidence suggests that the genetics influences on slopes are partially or wholly *distinct* from the genetics influences on baseline scores, as well as partially or wholly distinct between constructs (e.g., Finkel, Davis, Turkheimer, & Dickens, 2015; Logan et al., 2013). This suggests there is meaningful variance on the within-person slope that is distinct from that of baseline scores: The genetics mechanisms affecting the baseline phenotype are not the same as those governing within person changes over time. In addition, the decreased heritability on the slope is not always associated with increased nonshared environmental influences. Together, these findings demonstrate that the lower heritability estimates of slopes compared to intercepts cannot be explained away by the methodological challenges of measuring and estimating change over time, and as such reflect meaningful patterns that need to be reconciled in frameworks that hope to integrate both sources of phenotypic variation.

We believe these two key messages have at least three implications for the target article. First, we should be transparent about the source and locus of "phenotypic variation." Phenotypes vary between people, but also within people over time (called slopes or trajectories). When relying on cross-sectional evidence, we are drawing conclusions about how *heritability* changes across development. In other words, the target article studies the *trajectory of heritability*. This is distinct from the heritability of how a phenotype changes over time: the *heritability of trajectories*. When positing explanatory mechanisms, we must scrutinize whether the empirical evidence we rely on is suitable to provide evidence for the mechanism at hand. Within-person changes are governed by genetics influences and cultural exposure which differ across the lifespan *within the same person*. Differences in trajectories (and baseline-trajectory correlation) are arguably a primary source of increases, and decreases, in phenotypic variance and thus can affect heritability estimates across the lifespan. For such reasons, Finkel and Pedersen (2004, p. 331) conclude "cross-sectional investigations of genetic and environmental influences on aging that can estimate the contributions to the mean level of performance are failing to capture the dynamic process of aging." In short, by studying cross-sectional snapshots as a proxy for development, we may draw incorrect inferences about the most intrinsically developmental component: change itself.

Second, the extent to which genetics and cultural influences differ when modelled using cross-sectional versus longitudinal methods may provide a unique source of insight. For instance, the target article discusses the greater risk of skin cancer for European ancestry Australians. However, here too within-person processes are crucial. Among genetically similar individuals, the elevated risk of skin cancer is present almost exclusively in those *born* in Australia (Olsen et al., 2020). Genetically comparable individuals who move to Australia later in life are at much less elevated risk, suggesting that skin cancer risk is mostly because of early life exposure to the sun, further narrowing down the mechanisms at play. In other words, although cross-sectional data can provide a start of our investigations, it is only by taking a truly developmental perspective that we can hope to truly triangulate mechanisms.

Third and finally, large longitudinal datasets are the most promising source of unique and novel insight. This, combined with the existing dearth of longitudinal sources of information, should encourage funders and scholars alike to better resource larger, collaborative studies that better capture the richness of phenotypic processes, with a special focus on longitudinal, within-person measurements.

Uchiyama et al. make a compelling case that aggregate findings do not generalize to subgroups. The same is true about phenotypic change: Trajectories of heritability are not necessarily informative about heritability of trajectories, yet it is at the level of within-person processes that individual lives unfold. We think the cultural evolutionary behavioural genetics approach would be enriched by making within-person change the core topic of interest. By studying the heritability of trajectories instead of the trajectory of heritability, we may finally, in the words of Molenaar (2004) "bring the person back, this time forever."

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Going beyond heritability: Mechanisms of gene–culture coevolution

Shinobu Kitayama and Qinggang Yu

Department of Psychology, University of Michigan, Ann Arbor, MI 48109, USA.
kitayama@umich.edu; https://kitayama.psych.lsa.umich.edu/wp/
qinggang@umich.edu;
https://lsa.umich.edu/psych/people/graduate-students/qinggang.html

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Abstract

The target article offers an important cautionary note on the interpretation of the heritability index. However, it does not directly address how culture and genes might interact. Here, we suggest that one allele of the dopamine D4 receptor gene promotes the acquisition of cultural values and practices and likely has coevolved with the human culture over the last 50,000 years.

The Uchiyama et al.'s article takes a cultural evolutionary approach to argue that genetic effects on behavioral traits depend on culture. In particular, the authors show how the heritability index might take different values, depending on cultural contexts. This point offers an invaluable cautionary note on how to interpret the heritability index. However, beyond this, the authors' effort does not directly address the original question that motivated it, namely, how culture and genes have coevolved and have interacted. As the authors note, "culture and genes are interwoven in the construction of many behavioral traits, making separation effectively impossible" (sect. 3.3.2, para. 5). Unfortunately, much of the current knowledge on gene–culture coevolution pertains only to genetic evolution that took place far before culture as we know it today emerged (e.g., how the invention of cooking shortened the human guts). Some isolated examples, such as the effect of herding culture on the evolution of lactose tolerance (Tishkoff et al., 2007) and that of rice farming on the evolution of "Asian flush" (Peng et al., 2010), are arguably more recent. However, beyond the consumption of milk and alcohol, little else is known about the coevolutionary dynamic for cultural traits.

Here, we seek to readdress this blind spot of the field by focusing on one gene. For a while, it has been known that one varying length polymorphism of the dopamine D4 receptor gene (*DRD4*) likely coevolved with human cultural evolution over the last 50,000 years, which took place in the Eurasian continent (a similar process must have occurred in Africa, but today, little is known). Crucially, the population-level frequency of a key allele of *DRD4* (called the 7- or 2-repeat allele, or 7/2-R allele for short) increases systematically as a function of distance from Africa (Chen et al., 1999; Matthews & Butler, 2011). This 7/2-R allele is known as a plasticity allele because carriers of this allele are strongly influenced by the quality of parenting (Belsky & Pluess, 2009). Recent evidence shows that this plasticity effect is likely to result from the function of this allele to upregulate the fidelity of computing reward contingencies (Glazer et al., 2020).

Although much of the *DRD4* literature focused on the genetic modulation of parenting quality effects, we wanted to see if we could extend this evidence to cultural influences. Because reinforcement-mediated learning processes constitute a powerful mechanism of cultural learning, the carriers of the 7/2-R allele might acquire, internalize, and thus "carry" mainstream cultural traits. In our study, we have focused on a contrast between European Americans (who tend to be independent) and East Asians (who tend to be interdependent) (Markus & Kitayama, 1991). For example, compared to European Americans, East Asians feel happier when connected with others (Kitayama et al., 2006) and take another's perspectives more readily (Wu & Keysar, 2007).

In one earlier study, we assessed these cultural traits with validated self-report measures and found European Americans are relatively more independent, and East Asians, relatively more interdependent. Importantly, however, this cultural difference was significantly more pronounced among the carriers of the 7/2-R allele than for non-carriers (Kitayama et al., 2014). Indeed, the non-carriers showed no such cultural difference. In more recent study, we tested cultural differences in brain structures that would support cultural traits among carriers and non-carriers. We had earlier observed that independence (vs. interdependence) positively predicts the gray matter (GM) volume of the orbitofrontal cortex (OFC) (Kitayama et al., 2017). As may be expected, the OFC GM volume was larger for European Americans than that for East Asians. Again, however, this cultural difference was evident only among the carriers (Yu et al., 2018). Similar evidence exists for the temporal–parietal junction; its GM volume increased by interdependence (vs. independence). As may be expected, it was greater for East Asians than that for European Americans as long as they carried the crucial 7/2-R allele (Kitayama et al., 2020; see Kitayama & Yu, 2020, for a review).

We may argue that the 7/2-R allele of *DRD4* emerged over the last 50,000 years to "turbo-charge" the acquisition of culturally sanctioned behaviors suitable for survival (e.g., interdependence in East Asian regions that was suitable for rice-farming) (Talhelm et al., 2014). Bear in mind, however, that this "turbo-charging" could backfire because culture sometimes does require changes and innovations. For this reason, we suspect this 7/2-R allele might account only for 30–40% of the population.

The study summarized above has begun to clarify how culture and genes might have coevolved. By the time humans spread out of Africa approximately 50,000–60,000 years ago, humans have been fully equipped with massive genetic networks underlying component processes involved in reinforcement learning (e.g., detection of reward cues and computation of reward contingencies). We suspect that the 7/2-R allele served as a hub of these existing gene networks to amplify the fidelity of reward processing, which, in turn, helped the carriers acquire the most mainstream cultural traditions (as long as they are properly socialized). They may, thus, have become carriers in double, that is, carriers of both the 7/2-R allele and the mainstream culture of different ethnic groups. Meanwhile, non-carriers might

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well have remained agnostic to the viability of the mainstream culture, which might have enabled them to innovate and change that culture when such changes were called for.

In sum, Uchiyama et al. show that the heritability index of various traits takes different values depending on the cultural environment. This is a valuable contribution. However, this analysis stops short of directly addressing the dynamic interaction between culture and genes. We have offered the hypothesis that even though cultural traits are entirely contingent on ecological (i.e., environmental) factors, they may still be modulated by *DRD4*. This gene likely helped the human species acquire and sustain cultural traditions over the last 50,000 years. Future study may perform the heritability analysis separately for the carriers and non-carriers of the 7/2-R allele of *DRD4*. In combination, a day may come when we can better understand how genes and culture might have coevolved to produce contemporary cultural and individual variations.

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Differential application of cultural practices at the family and individual levels may alter heritability estimates

Oren Kolodny[a], Marcus W. Feldman[b], Arnon Lotem[c,d] and Yoav Ram[c,d]

[a]Department of Ecology, Evolution & Behavior, The A. Silberman Institute of Life Sciences, The Hebrew University of Jerusalem, Jerusalem 9190401, Israel; [b]Department of Biology, Stanford University, Stanford, CA 94305, USA; [c]Faculty of Life Sciences, School of Zoology, Tel Aviv University, Tel Aviv 6997801, Israel and [d]Sagol School of Neuroscience, Tel Aviv University, Tel Aviv 6997801, Israel.
oren.kolodny@mail.huji.ac.il; mfeldman@stanford.edu; lotem@tauex.tau.ac.il; yoav@yoavram.com
sites.google.com/view/oren-kolodny-homepage; profiles.stanford.edu/marcus-feldman; arnonlotem.weebly.com; www.yoavram.com

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Abstract

Uchiyama et al. emphasize that culture evolves directionally and differentially as a function of selective pressures in different populations. Extending these principles to the level of families, lineages, and individuals exposes additional challenges to estimating heritability. Cultural traits expressed *differentially* as a function of the genetics whose influence they mask or unmask render inseparable the influences of culture and genetics.

Uchiyama et al. propose a thought-provoking synthesis between the fields of behavioral genetics and cultural evolution. We suggest that the principles laid out by Uchiyama et al. can be extended to the levels of the family, the lineage, and even the individual. These extensions expose further challenges in assessment and interpretation of heritability even when cultural clusters are well defined.

Uchiyama et al. point out, in their third and fourth key points, that cultural effects on phenotypes can be directional and predictable, and can become stronger as ecological and cultural selection becomes stronger. These points are well demonstrated, respectively, by the case of sunscreen use and the expression of skin cancer, and by the increased use of sunscreen by Australians of European ancestry. They further highlight that cultural clustering, and particularly hidden clustering, may hinder proper interpretation of heritability scores. We propose that further challenges stem from the same fundamental principle that Uchiyama et al. propose when considered at the level of the lineage, family, or individual.

Even within a tight mono-cultural society, cultural vertical transmission is strong for many traits (Chen, Cavalli-Sforza, & Feldman, 1982), and different families may vary in their choice of available cultural tools or knowledge. This between-family variation may influence the expression of related phenotypes in a consistent and directional manner, masking or unmasking genetic effects (Feldman et al., 2013). For example, a lineage with a history of skin cancer may use sunscreen and other protection more than the

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average family, thus reducing the risk of cancer via this cultural practice. That is, when genetic variation has an observable phenotypic impact, families may *differentially* implement cultural practices that mask or unmask this genetic variation as a function of the genetic variants that each family carries. This effect can be exacerbated by the emergence of personal genomics services and precision medicine. Because familial structure is correlated with genetics, the challenges extend beyond the consideration of hidden cultural clusters (as discussed by Uchiyama et al.). In particular, differential practice of cultural behaviors that are *designed* to alter genetically influenced phenotypes *as a function of those phenotypes' expression* is expected to render the effects of genetics and culture inseparable. Teasing apart the influence of these factors then becomes impossible without manipulation experiments, which are unethical and unfeasible in humans.

Additional challenges arise when considering phenotypes expressed gradually or repeatedly during the lifetime of an individual. In such cases, if cultural practices designed to enhance or inhibit the phenotype are available, different *individuals* are likely to use them differentially and in a manner that is highly directional, possibly masking or unmasking genetic effects and deflating or inflating heritability scores. For example, if a certain individual exhibits symptoms that place her at high risk for autoimmune disorders if excessively exposed to UV, she may remain indoors or use protective measures above and beyond the average use in her cultural cluster, thereby reducing both heritability scores and phenotypic variation. In contrast, gifted young athletes may invest in their training well above the average and thus perform better than would be expected based on their genetic makeup. Such individual-based effects can accordingly increase or decrease heritability scores.

Finally, we would like to point out that the masking or unmasking of genetic effects on the phenotype by cultural practices may usefully be viewed as a form of differential niche construction (Odling-Smee, Laland, & Feldman, 2013). In this view, different cultural clusters may alter the selective pressures that they experience as a function of the perceived severity of these pressures (Ihara & Feldman, 2004). The same extension that we presented above holds here: Differential application of cultural practices at the family or individual level, when matched with the selection differential they would have experienced otherwise, can offset the selection differential and diminish its effect.

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Interpreting and reinterpreting heritability estimates in educational behavior genetics

Sally A. Larsen

Department of Psychology, University of New England, Armidale, NSW 2351, Australia.
slarsen3@une.edu.au

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Abstract

Interpreting heritability estimates through the lens of cultural evolution presents two broad and interlinking problems for educational behavior genetics. First, the problem of interpreting high heritability of educational phenotypes as indicators of the genetic basis of traits, when these findings also reflect cultural homogeneity. Second, the problem of extrapolating from genetic research findings in education to policy and practice recommendations.

"That is not what I meant at all; That is not it, at all."
T. S. Eliot (1915)

Interpreting the findings of behavior genetics studies is fundamentally a quest for meaning. This is certainly true in a field like education where the purpose of research is ultimately to improve the provision of education, and by extension, improve the outcomes attained by students. The bold concluding claim of Uchiyama et al., "Nothing in behavioral genetics makes sense except in the light of cultural evolution" (sect. 6, para. 3), should prompt us to reexamine the meaning of decades of educational behavior genetics studies. If heritability estimates are consistently high for educationally relevant traits what should this information mean? And, how should it be applied to the real world of students, teachers, schools, and education systems?

Uchiyama and colleagues identify education as a prime example of a culturally transmitted phenomenon. Indeed, access to universal education is so embedded in western societies (the locus of much behavior genetics research) that it is difficult to imagine a world in which it did not exist. It may be self-evident to point out that educational contexts are not static: They continuously evolve both within and between systems, influenced by local and global cultural and policy shifts (e.g., Addey, 2017; Vadeboncoeur, 1997). For example, development of theories of learning, constantly changing administrative structures, variable recommendations on instructional practices, and frequent reform movements are features of many educational systems (Rose, 2006; Stanovich & Stanovich, 2003; Woods, 2021). For readers who come to the behavior genetics literature from the field of education research, discussion of heritability estimates may seem to put an overabundance of emphasis on quantifying the genetic influence on phenotypes at the expense of examining the cultural variation within and between school systems, or over time.

In behavior genetics, educational cultural contexts are encompassed by the terms *shared* and *nonshared* environment. The

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complexity and richness of culture is, perhaps necessarily, stripped out in the textbook definitions for *shared environment*, "all nongenetic influences that make family members similar to one another," and *nonshared environment*, "all nongenetic influences that are independent for family members, including error of measurement" (Plomin, DeFries, Knopik, & Neiderhiser, 2013, p. 96). In behavior genetics, the environment is represented as the reverse of the genetic coin: whatever remains in the *absence* of genetic effects. While this dichotomy is a simplification of a more complex reality, the emphasis on genes versus environments, rather than genes situated within environments, tends to obscure both cultural evolution within contexts, and differences between educational systems that may affect educational provision and outcomes.

The fact that heritability estimates are population statistics is well understood and not trivialized among behavior geneticists. Nonetheless, the central importance of interpreting heritability as a population statistic (e.g., Smith, 2011) is often glossed over in discussions about "genes for" traits like reading and mathematics ability in educational studies. Inferences are made that by measuring the heritability of traits we have somehow found out something definitive about the "genetics" of that trait. The central contention of Uchiyama and colleagues is that such overarching conclusions are not supportable given the presence of cultural homogeneity within twin samples, and continually evolving educational cultures.

Uchiyama and colleagues make a compelling case that high heritability estimates of educational phenotypes may not indicate optimal educational environments, as is often claimed (e.g., Kovas, Tikhomirova, Selita, Tosto, and Malykh, 2016). High heritability may instead reveal something about the cultural homogeneity of environments from which twin samples are drawn. Heritability may be equally high in poor education systems as in excellent education systems, so long as the system is relatively homogeneous, and all students obtain the same poor or excellent education. For example, heritability estimates for reading skills are not remarkably divergent in educational systems where overall student attainment is relatively high (e.g., Hong Kong Chinese; Chow, Ho, Wong, Waye, and Bishop, 2011) compared with systems where average attainment is consistently relatively lower (e.g., Australia, see Byrne, Olson, & Samuelsson, 2019; Thomson, Hillman, Schmid, Rodrigues, & Fullarton, 2017). This point alone is a problem for the context-free interpretations of behavior genetics findings in education and should prompt some hesitation about the broad extrapolations that are made about the genetic basis of academic abilities.

The definitional and interpretational difficulties described above lead next to questions around the applicability of behavior genetics research to educational policy and practice. There is disagreement about the extent to which behavior genetics research can and should be extrapolated to educational policy and practice recommendations (Asbury & Wai, 2020; Byrne et al., 2020; Panofsky, 2015). Nonetheless such recommendations are made. One of the recurrent themes of educational behavior genetics is the idea that understanding the genetic etiology of traits will lead to improved interventions (Shero et al., 2021), redesign of school systems (Asbury & Plomin, 2014), and personalized education (Kovas et al., 2016). Such claims, however, elide the fuzziness of interpretation inherent in behavior genetics. Turkheimer (2015) argued that the "layer of theory between data and their interpretation is thicker and more opaque [in behavior genetics] than in more established areas of science" (p. s32). That is, interpretations of behavior genetics research rely on an accepted theoretical understanding of heritability estimates (e.g., Harden, 2021), which underlies suggested applications in the real world. However, if heritability estimates in educational studies are, (a) inflated to an unknown extent (e.g., Coventry & Keller, 2005; Keller & Coventry, 2005), arguably by sample-specific cultural homogeneity, and, (b) can be confounded by cultural evolution, then the suggested applications to policy and practice could easily be wrong. The value of Uchiyama and colleagues to the field of educational behavior genetics is in their articulation that accepted explanations of heritability estimates are indeed contestable theoretical positions and can (should?) be interpreted differently via the lens of cultural homogeneity and evolution.

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There is no such thing as culture-free intelligence

Gary Lupyan

Department of Psychology, University of Wisconsin–Madison, Madison, WI 53706, USA.
lupyan@wisc.edu; http://sapir.psych.wisc.edu

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Abstract

Cognitive scientists and psychometricians are unaccustomed to thinking about culture, often treating their measures – memory, vocabulary, intelligence – as natural kinds. Relying on these measures, behavioral geneticists likewise seem to not wonder about their origin and cultural provenance. I argue that complex human traits – the sort we are most interested in measuring – are cultural products. We can measure them and their heritability, but to conclude that what we have measured is unbound to a time and place is hubris.

Uchiyama et al. conclude that "Nothing in behavioral genetics makes sense except in the light of cultural evolution" (sect. 6, para. 3). This claim may seem like an exaggeration, but like Uchiyama et al., I think human genetics cannot be considered without recourse to culture because *so many* of our behaviors – indeed most of what makes humans unique – do not exist outside of culture. Absent cultural support, not only do we lack skills like reading, arithmetic, and rugby, but also species-typical behaviors like a compositional and productive communication system (i.e., natural language). Absent culture, we can't even feed ourselves! Our digestive system has adapted to cooked food (Wrangham, 2009) and how many of us would invent *de novo* control of fire and rediscover cooking? With such behaviors being at the mercy of cultural learning, can we really talk about heritability of a trait without understanding its cultural underpinnings? I will argue that we cannot.

A common way of thinking about environmental effects on heritability is in terms of environmental deprivation that masks genetic potential. For example, a person's height may be stunted by malnutrition preventing them from reaching their "genetic potential" and reducing measured heritability (Perkins, Subramanian, Davey Smith, & Özaltin, 2016). The intuition is that the person's true potential is being masked. If only they had received the normal amount of nutrition, they would grow to the height their genes prescribe.

But just on the other side of this equation is the *unmasking* of traits by culture. And here, our intuitions begin to break down. Consider De Moor et al.'s (2007) calculation of a 0.66 heritability of being a UK high school or university athlete. This seems impressive, but what would the calculated heritability be if the study took place in the United Kingdom 500 years ago? Or at a contemporary time, but in a place with no school sports? If the answer is that question doesn't even make sense because there would be no outcome to observe, that is precisely the point. Cultural institutions have created a domain – competitive sports – whose heritability we can measure. But that measurement is necessarily restricted to a particular time and place.

Perhaps, no other human trait has been the subject of more attempts at heritability estimates than intelligence, with estimates running as high as 0.8 (Plomin & Deary, 2015) leading to the puzzle of "missing heritability" (Feldman & Ramachandran, 2018). Similarly to Feldman and Ramachandran, Uchiyama et al. argue that the resolution of the puzzle lies in integrating culture. It is worth elaborating on some of the problems with attempts to view intelligence as a culture-free trait.

While the goal of conventional tests is to measure knowledge or ability, the goal of IQ tests is to measure intelligence itself. This is justified by the observation that when people are tested on a variety of cognitive measures: vocabulary, analogies, figuring out what rule a sequences of shapes follow, remembering numbers, the scores are positively correlated. Some people excel more on some tasks than others, but in general, doing well on one means doing well on the others. This so-called positive manifold is reasoned to have a common cause which is *g* (Ritchie, 2015). Estimates of heritability of intelligence are estimates of the heritability of *g*. The problem is that measures of intelligence (IQ tests) are ineluctably cultural products. Heritability of *g* is therefore necessarily linked to the culture.

There are several objections to the claim that IQ cannot be considered independently of culture. First, it may be argued that although some IQ subtests such as vocabulary are culturally loaded, others such as fluid reasoning are not (Jensen, 1980). A problem with this contention is that more culturally loaded tests show *greater* heritability than putatively culture-free tasks (Kan, Wicherts, Dolan, & van der Maas, 2013). The claim that assessments of fluid reasoning are culture free because they are nonverbal is also naïve to the reliance of these tests on culturally learned patterns and symbols (Richardson, 2002; Roebuck & Lupyan, under review; Rosselli & Ardila, 2003).

Second, it may be argued that although any specific IQ test is a product of a specific time and place, the quantity it measures generalizes beyond cultures and time periods. We can make this claim more vivid using a thought experiment. Take 100 modern Americans whose measured adult IQ scores span a wide range and transport them (as infants) to various times and places. Allow them to grow up and be enculturated at their destinations, and then test them *on that culture's version of an intelligence test* (one having similarly high predictive validity in life outcomes as our IQ tests). If IQ tests measure *g*, then the person scoring in the top 1% would come out on top regardless of the time and place they were transported to thanks to the preservation of their intelligence-coding genes. The person at the 30th percentile would likewise stay around there. The rank correlation would be preserved. Perhaps it would. But this assumption has zero empirical support and there are good reasons to doubt it.

Consider: for most of human existence, someone with poor wayfinding abilities would be at an extreme disadvantage and likely considered rather unintelligent. Now, they can just use their phones. Variation in wayfinding has become masked and is certainly not something we include in IQ testing. If we did, it might decrease the strength of the positive manifold (Hegarty, Montello, Richardson, Ishikawa, & Lovelace, 2006). Conversely,

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individual differences in logical reasoning, reading, and the ability to sit still for long periods would be of little consequence in times past, but have now been unmasked by the demands of modern culture. The former two skills are explicitly measured by intelligence testing; the latter is an implicit prerequisite (DeDeo, 2018; Stephenson, 2012). Is it not hubris to think that figuring out sequences of shapes – a mainstay of modern IQ tests – is a proper measure of "general intelligence" while wayfinding is a mere specialized skill? It is time to recognize that culture is not a peripheral appendage on the leash of our genes (*pace* Wilson, 2004), but is the vehicle that makes possible many of our most important behaviors.

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Cultural evolution and behavior genetic modeling: The long view of time

Kristian E. Markon[a], Robert F. Krueger[b] and Susan C. South[c]

[a]Department of Psychological and Brain Sciences, University of Iowa, Iowa City, IA 52242, USA; [b]Department of Psychology, University of Minnesota, Minneapolis, MN 55455, USA and [c]Department of Psychological Sciences, Purdue University, West Lafayette, IN 47907, USA.
Kristian-markon@uiowa.edu; krueg038@umn.edu; ssouth@purdue.edu

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Abstract

We advocate for an integrative long-term perspective on time, noting that culture changes on timescales amenable to behavioral genetic study with appropriate design and modeling extensions. We note the need for replications of behavioral genetic studies to examine model invariance across long-term timescales, which would afford examination of specified as well as unspecified cultural moderators of behavioral genetic effects.

Uchiyama et al. compellingly demonstrate the importance of cultural context in interpreting estimates of genetic effect. As they show, genetic pathways depend on cultural processes that evolve and change, providing a broad, pervasive system of environmental influences transcending individuals and their families.

The need for an integrative longitudinal framework

Although Uchiyama et al. insightfully draw attention to this, it is important to note the literature they discuss provides little in the way of formally integrating long-term societal change with shorter-term developmental change and genetic modeling. The authors discuss cross-sectional comparisons between culturally distinct geographic areas, longitudinal accounts of developmental change, longitudinal accounts of long-term societal changes in behavior (the Flynn effect), and long-term accounts of changes in heritability (fertility and educational attainment), but without an overarching modeling framework that integrates these effects simultaneously.

For example, in discussing UV exposure, although the authors note that "cultural change can be particularly fast and potent" (sect. 2.1, para. 3), and "the environment of the genome is…a moving reference frame that rapidly evolves in relation to both genes and ecology" (sect. 2.1, para. 3), their empirical discussion is focused on geographical differences in vitamin D in relation to UV, and not changes *per se* tied to cultural evolution. Other cited research on differences in heritability as a function of cultural variables (e.g., Engzell & Tropf, 2019) is similarly cross-sectional. The limitations of making dynamic inferences based on cross-sectional data are well known, and have been raised in the context of the studies discussed (Morris, 2020). In short, although these studies work in promising directions, existing literature and the discussion provided by Uchiyama et al. lack a unified modeling treatment, one that integrates two very different timescales of human experience.

Challenges in the modeling of culture, genes, and environment, and potential directions

We see direct parallels between the phenomena discussed by Uchiyama et al. and age-period-cohort (APC) models in the epidemiological literature (Fosse & Winship, 2019). APC models decompose variance into portions associated with age (development), as well as temporal period (the shared experiences of those living at a particular moment in time, regardless of age or cohort) or cohort (the shared experiences of those developing together during a particular period). Although APC models traditionally do not concern themselves with genetics, they do jointly account for developmental and contextual effects across different timescales.

Extending APC frameworks to include genetic and environmental effects would be relatively straightforward. Multicohort twin and family studies have been conducted; molecular genetic information could also be included in APC designs, such as through polygenic scores or specific alleles (as in Mendelian randomization paradigms). Doing so would provide a formal paradigm for joint modeling of developmental and cultural processes, guiding attention to important theoretical and methodological issues in the study of culture and genetics.

Research on APC models highlights the intertwined nature of developmental and cultural effects and potential challenges in modeling them jointly. Work in this area has demonstrated how effects that appear conceptually distinct can be difficult to distinguish when specified in models (Fosse & Winship, 2019). Changes in genetic effects modeled in terms of cohorts (e.g., Briley, Harden, & Tucker-Drob, 2015; Rosenquist et al., 2015; Sanz-de-Galdeano, Terskaya, & Upegui, 2020), for example, might equivalently be framed in terms of periods (e.g., war, socioeconomic conditions, or policy eras) which are often implicitly the focus of explanation anyway. Other research suggests that apparently simple sociological concepts might require relatively complex model features to capture when considered simultaneously against the backdrop of development (Fosse & Winship, 2019).

Many authors have noted that age and period are theoretical proxies for other, unspecified factors of interest, such as specific cultural or environmental agents impacting everyone living at a particular point in time, or neurodevelopmental processes that unfold at specific points in the lifespan. Focusing on these specified causal factors, rather than making assumptions about unspecified factors in the form of age, period, or other (e.g., geographic) proxies, provides numerous theoretical and modeling benefits (Fosse & Winship, 2019). However, doing so also highlights the importance of how cultural and environmental variables are defined and measured. How are cultural variables different from other measured environmental variables in biometric frameworks, if at all? Is it the scope of an exposure in time or space (the Great Recession, for instance, vs. family cohesiveness)? Transmission across generations? How do you operationalize and validate such measures in a way that provides rigorous tests of a theory?

Although it is true that cultural evolution "can be particularly fast and potent" when compared to genetic evolution, it can also be relatively slow compared to the timeframe typically employed in isolated behavioral genetic studies. Even the effects of relatively discrete, "potent" once-in-a-generation events (e.g., the onset of world war) might require data from multiple generations to discern, and are difficult to capture via serendipity in study design.

In general, these issues, taken together with issues highlighted by Uchiyama et al., point to the need for replication of behavior genetic findings across long time spans, and to do so in a formal, comprehensive longitudinal modeling framework. Doing so allows systematic examination of how parameters of population and molecular behavior genetic models might or might not be invariant across different periods, and to examine potential measured causal or mechanistic variables that might be affecting the non-invariance of those model components. Such designs will be critical in moving past cross-sectional studies to more causally informative, comprehensive accounts of the dynamic interplay between genes, development, and culture as envisioned by Uchiyama et al.

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Developmental noise is an overlooked contributor to innate variation in psychological traits

Kevin J. Mitchell

Trinity College Dublin, Smurfit Institute of Genetics, Dublin 2, Ireland.
Kevin.Mitchell@tcd.ie
www.kjmitchell.com

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Abstract

Stochastic developmental variation is an additional important source of variance – beyond genes and environment – that should be included in considering how our innate psychological predispositions may interact with environment and experience, in a culture-dependent manner, to ultimately shape patterns of human behaviour.

The target article presents a very welcome and much-needed overview of the importance of cultural context in the interpretation of heritability. The authors discuss a range of complex interactions that can occur between cultural and genetic effects, illustrating how already complicated gene–environment correlations and interactions can vary at a higher level as a function of cultural factors or secular trends.

However, the framing with genes and environment as the only sources of variance ignores an extremely important third component of variance, which is stochastic developmental variation (Vogt, 2015). Genetic effects on our psychological traits are mainly developmental in origin, but genetic differences are not the only source of variance in developmental outcomes (Mitchell, 2018). The genome does not specify a precise phenotype – there is not enough information in the 3 billion letters of our DNA to encode the position of every cell or the connections of every neuron. Rather, the genome encodes a set of biochemical rules and cellular processes through which some particular outcome from a range of possible outcomes is realized (Mitchell, 2007).

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These processes of development are intrinsically noisy at a molecular and cellular level (Raj & van Oudenaarden, 2008), creating substantial phenotypic variation even from identical starting genotypes (Kan, Ploeger, Raijmakers, Dolan, & van der Maas, 2010). The importance of chance as a contributor to individual differences was recognized already by Sewell Wright in a famous 1920 paper (Wright, 1920) and is ubiquitously observed for all kinds of morphological and behavioural traits across diverse species (Honegger & de Bivort, 2018; Vogt, 2015). For brain development in particular, the contingencies and nonlinearities of developmental trajectories mean that such noise can manifest not just as quantitative, but sometimes as qualitative variation in the outcome (Honegger & de Bivort, 2018; Linneweber et al., 2020; Mitchell, 2018).

The implication is that individual differences in many traits are more (sometimes much more) innate than the limits of the heritability of the trait might suggest. In other words, not all of the innate sources of variation are genetic in origin, and not all of the non-genetic components of variance are actually "environmental." Indeed, a sizeable proportion of the confusingly named "nonshared environmental" component of variance may have nothing to do with factors outside the organism at all, but may be attributable instead to inherently stochastic developmental variation (Barlow, 2019; Kan *et al.*, 2010; Mitchell, 2018). This may be especially true for psychological traits, where heritability tends to be modest, but systematic environmental factors that might explain the rest of the variance have remained elusive (Mitchell, 2018). Proposals that idiosyncratic experiences should somehow have more of an effect than systematic ones (Harris, 1998) provide no convincing evidence that this is the case, nor any persuasive arguments for why it might be so.

This does not overturn any of the important points that the authors make but does suggest an important reframing. Rather than thinking solely of genetic versus environmental sources of variance, and the interaction between them, we can think of the interplay between innate predispositions – which reflect both genetic and developmental variation – and experience. Culture can have a huge influence on this interplay, especially on how much scope it gives for individual differences in psychology to be expressed or even amplified through experience.

However, if such predispositions do not solely reflect genetic influences then the implications of such effects for heritability become less obvious. If genetic variance predominates at early stages, then heritability may increase across the lifespan, as is observed for cognitive ability. On the other hand, if the influence of stochastic developmental variance (included in the nonshared environment term) is larger, then heritability may decrease with age, as observed for example for many personality traits (Briley & Tucker-Drob, 2017). In both cases, innate differences may be amplified, as observed in mice (Freund *et al.*, 2013).

An already complicated picture of interactions and meta-interactions thus becomes even more so. In addition, there may be further interactions at play, as the degree of developmental variability is often itself a genetic trait. This has been observed in various experimental systems, which have found that variability of a trait can be affected by genetic variation and even selected for, with no concomitant effect on the phenotypic mean (e.g., Ayroles et al., 2015). More generally, the developmental programme has evolved to robustly produce an outcome within a viable range (Wagner, 2007). However, that robustness depends on all of the elements of the genetic programme and the multifarious feedforward and feedback interactions between them. Increasing genetic variation is, therefore, expected to not just affect various specific phenotypes, but also to degrade the general robustness of the overall programme and thus increase the variability of outcomes from some genotypes more than others.

This is illustrated by the special case of increased variance in many traits in males compared to females, observed across diverse phenotypes in many different species (Lehre, Lehre, Laake, & Danbolt, 2009). A proposed explanation is that hemizygosity of the X chromosome in males reduces overall robustness of the programmes of development and physiology and thus increases variance in males. Strong support for this hypothesis comes from the evidence that the direction of this effect is reversed in species, including birds for example, where females are the heterogametic sex and show increased phenotypic variance (Reinhold & Engqvist, 2013). Sex is, thus, another factor that may affect patterns of variation of human traits through this kind of general influence on developmental variability. In addition, of course, cultural factors differ hugely between the sexes, which may differentially influence how innate predispositions are expressed by males and females.

One final complication is that environmental conditions may either buffer or further challenge the developmental programme, reducing or exposing variability, as demonstrated in classic experiments (Waddington, 1957; Wagner, 2007). Overall then, the already complex interactions very thoroughly discussed by the authors should be expanded to include the often overlooked but hugely important third component of variance: Noise inherent in the developmental processes by which genotypes become realized as specific phenotypes.

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Genetic solutions to cultural problems?

Lesley Newson and Peter J. Richerson

Department of Environmental Science and Policy, University of California Davis, Davis, CA 95618, USA.
lgnewson@ucdavis.edu; pjricherson@ucdavis.edu

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Abstract

In theory, observed correlations between genetic information and behaviour might be useful to members of the WEIRD (western, educated, industrialized, rich, and democratic) populations. Guiding young people to choose educational opportunities that best match their abilities would benefit both the individual and society. In practice, however, such choices are far more profoundly limited by the culture people have inherited than their genes.

Uchiyama et al. have provided a valuable explanation of some of the limitations in trying to draw useful conclusions from observations of correlations between sequences in genomes and the behaviours and life course of the individuals who possess those genomes.

The behavioural genetic findings and techniques exist, however, and it's not surprising that the scientists involved are keen to see the fruits of their investigations put to good use. Their findings may not be applicable to all populations, but it's tempting to hope that they could be applied in the WEIRD (western, educated, industrialized, rich, and democratic) populations that best match those in which the research was performed.

One of the roles that culture plays in human lives is the coordination of effort. Throughout human evolution, the most successful groups were those that evolved rules, customs, and beliefs which encouraged people of different ages and abilities to work effectively together to further their own interests and those of the group as a whole. Our ancestors accomplished this without knowledge of genes, but in small-scale societies elders could observe youngsters as they grew up, see which skills they found easiest, and encourage them to develop their talents in ways that would be useful to the group. Such minute observations aren't possible in the educational institutions attended by children in WEIRD populations. So, could information from genetic testing fill in the gaps? If teachers were able to guide pupils towards the educational and life choices which they will find most fulfilling, their efforts would be applied more effectively and society would be better coordinated.

This is a nice idea in theory but it's unlikely to be to be very helpful in practice.

It's not the lack of genetic information that prevents children in WEIRD populations being helped to make appropriate educational and life choices. It's more to do with the cultures of WEIRD populations and the many ways they have evolved that hamper the effective coordination of effort.

For example, American culture includes the dogma that free choice and hard work are highly valued. Children are often told that they can choose from a wide range of possible futures and urged to have faith that, if they work hard enough, they will realize their dreams. In fact, children's choices and their success are severely limited not just by the genes they inherit from their parents but also by the culture that they inherit from the family and community they grow up in and via the media they are exposed to. It is their culture that provides the expectations, values, goals, opportunities, rewards, and so on that motivate humans.

If American culture truly valued hard work, wouldn't the members of society who do essential work that is difficult and unpleasant, such as the production and processing of food, receive better compensation than those who, for example, perform research and teach at a university?

Perhaps, few Americans actually believe the dogma about free choice and hard work, but its frequent repetition still serves to inhibit the development of programmes aimed at reducing inequality.

It's not just poverty that limits freedom of choice for young members of WEIRD populations. Imagine, for example, the opportunities available to a female as she graduates from high school. One choice she might reasonably consider is becoming pregnant. At this age, her body is in peak condition for maternity and it's likely that her own parents and those of the potential father of her baby are still young and fit enough to help her care and provide for her baby. In some communities within WEIRD populations a young woman's ambition to be a mother could be realized, but in others women are expected to want to go to university, establish a career, and own a home before even thinking of motherhood. Their parents are too busy with their own careers to take on any grandparental duties. The work of raising the next generation is considered to be of relatively low value in the cultures of WEIRD populations. Not surprisingly, the fertility rate of these populations is below replacement.

For the last few generations, cultural change has been very rapid in human populations and our species has become very successful in the sense that the human population has grown rapidly. But it's now more than apparent that in the course of modernization populations evolve a number of cultural maladaptations that prevent the effective organization of labour and use and distribution of resources.

Modern societies are currently neither demographically or environmentally sustainable. Many of them suffer other kinds of social dysfunction. The classical eugenicists looked to genetic evolution as a source of human problems and solutions. They were looking under the wrong rock. Humans are far more variable culturally than genetically and, even though both genes and culture are hard to change, culture is by far the more tractable. Our ancestors have been finding cultural solutions to their problems for many thousands of generations and our descendants will carry on the tradition. Uchiyama et al.'s useful dive into the relationship between genetic and cultural inheritance and evolution shouldn't encourage us to forget the practical bottom line.

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The implications of the cultural evolution of heritability for evolutionary psychology

Timothy P. Racine

Department of Psychology, Simon Fraser University, Burnaby, BC V5A 1S6, Canada.
tracine@sfu.ca
https://www.sfu.ca/psychology/about/people/profiles/tracine.html

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Abstract

Uchiyama et al. provide a compelling analysis of cultural influences on estimates of the genetic contribution to psychological and behavioral traits. Their focus is on the relevance of their arguments for behavioral genetics and their work resonates with other contemporary approaches that emphasize extra-genetic influences on phenotype. I extend their analysis to consider its relevance for evolutionary psychology.

Uchiyama and colleagues provide a timely and compelling analysis of cultural influences on estimates of the genetic contribution to psychological and behavioral traits. Their focus is on the relevance of their arguments for the practice of behavioral genetics. Their target article also resonates with current emphases in both standard and extended evolutionary approaches briefly mentioned by the authors that are used in psychology and allied fields which, for example, distinguish the ecological from the cultural environment, highlight niche construction, afford a constitutive role for culture, and emphasize other "extra-genetic" forms of stable influence on a phenotype, such as epigenetic inheritance (see e.g., Jablonka & Lamb, 2014). As such, Uchiyama et al.'s analysis can be extended to evolutionary psychology (EP), including the programmatic sense of EP associated with the theoretical and empirical work of Buss, Cosmides, Tooby, and Pinker (e.g., Buss, 1995; Pinker, 2002; Tooby & Cosmides, 1992).

Although behavioral genetics and EP have different goals and criticisms of the former might appear to have little relevance for the latter, the target article will likely be seen by some of EP's critics to call into question several of the assumptions of the approach. For example, in contrast to Uchiyama et al., EP is well known for: (1) arguing that the speed of evolutionary change is slow because the speed of genetic change is slow; (2) emphasizing adaptation to the ancestral environment; (3) relying on mismatch arguments between ancestral and present environments; (4) locating human nature in the ancestral environment; (5) dichotomizing culture into a transmitted versus evoked form; and, (6) making strong commitments to psychological domain specificity. Although many of these concerns are valid, they also have the potential to repeat certain misconceptions about EP.

EP is mostly unconcerned with variation, genetic or otherwise; its goal is to describe a universal human nature that, as such, is genetically fixed (e.g., Tooby & Cosmides, 1990). One might think of the basic human body plan as analogous to what EP has in mind. They claim there is a basic human psychology plan that is universal, genetically fixed, and in the language of behavioral genetics not heritable; universal phenotypes have zero heritability by definition. EP focuses on adaptations that would have promoted ancestral survival and reproduction. From this point of view, psychological adaptations are complex and analogous to the evolution of a phenotype like the human eye with a deep evolutionary history that precedes humans. As such, psychological adaptations shared with other species are still seen as part of our uniquely human nature. Complex adaptations like these seem necessarily laid down in the distant past with the pace of genetic change being too slow to be greatly affected by adaptation to the Holocene. Thus, although it is well known that genetic change continues to occur and that, for example, genes for lactase persistence have been selected in several human populations with the advent of animal husbandry, these sort of single-gene changes seem highly unlikely to radically transform any evolved human capacities. This is the central point of EP, and it is one that is not always given its fair due. If one conceives of, for example, the capacity for color perception or language competence as analogous to other psychological capacities such as the adaptations for social exchange singled by Tooby and Cosmides, or so-called core knowledge competencies like theory of mind, folk physics, numerosity, and so on, this is the basic idea of what EP is arguing. Of course, theoretical and empirical research in evolutionary developmental biology that has emerged in parallel to EP suggests that the causal story involves more than mutation-driven selection acting over evolutionary time (see e.g., Hall, 2012), and how such capacities develop in real time is also complex (Witherington & Lickliter, 2016), but the notion that humans might have evolved a basic human psychology that is analogous to a basic human physiology has certainly been a fruitful endeavor despite its detractors (Buss, 2020).

The work of Uchiyama et al. appears to add additional challenges to EP that turn on the ability to logically separate the effects of genes and cultures. As such, it calls into question some of the ontological distinctions between biology and culture or genes and environments that are made in EP and in the field of evolutionary studies more generally. It also speaks to the wider WEIRD (western, educated, industrialized, rich, and democratic) problem that has bedeviled the field. However, it is worth repeating the central point that despite the potential lack of universality of certain psychological competencies, a chief example used by Uchiyama et al. is the evolution of skin pigmentation, which is a paradigm case of the evolution of a simple phenotype. Although it is instructive that even in such a simple case it is easy to overlook possible cultural factors, it is not as easy to imagine that such issues necessarily obtain when addressing more complex phenotypes. It does speak to the need of more complex models though and the importance of pluralism in evolutionary explanation; it also suggests that explaining all aspects of human psychology could not be tantamount to theorizing their underlying ancestral contexts. To be fair though, EP takes pains to distance itself from such reductionism and determinism. Whether they are fully successful continues to be openly debated both by its advocates and detractors.

In conclusion, the target article adds to a growing body of literature that emphasizes the importance of considering the cultural dimension of inheritance and the necessity for a more nuanced and dynamic conception of the evolutionary process. However, it also has the potential to re-invite familiar criticisms of EP. This is not to say that EP, or any theory, is beyond criticism, but to paraphrase a point recently made by Apicella, Norenzayan, and Henrich (2020, p. 319) in their 10-year retrospective on the WEIRD problem discussed in the target article, a cultural analysis of heritability should not be taken to be a

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"knock-down critique of evolutionary psychology." However, Uchiyama et al. could add to potential calls for the construction of a more nuanced EP.

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Models of gene–culture evolution are incomplete without incorporating epigenetic effects

Gillian Ragsdale[a] and Robert Andrew Foley[b]

[a]The Ronin Institute for Independent Scholarship, Montclair, NJ, USA and [b]Department of Archeology, University of Cambridge, Cambridge CB2 3DZ, UK.
gill.ragsdale@ronininstitute.org
raf10@cam.ac.uk
http://ronininstitute.org/

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Abstract

Epigenetics impacts gene–culture coevolution by amplifying phenotypic variation, including clustering, and bridging the difference in timescales between genetic and cultural evolution. The dual inheritance model described by Uchiyama et al. could be modified to provide greater explanatory power by incorporating epigenetic effects.

The case for the complex and context-dependent nature of gene–culture interactions is well made by Uchiyama et al. However, the dual inheritance model they describe could be extended to provide greater explanatory power by incorporating epigenetic effects. Epigenetic gene regulation can amplify phenotypic variation (mimicking greater gene variation) and accelerate both cultural and genetic evolution.

"Epigenetics" refers to changes in gene expression brought about by chemical modifications that do not change the DNA sequence itself. The processes themselves appear to operate mainly during development rather than adulthood – preparing the individual mammal for the specific situation into which it has been born. Epigenetically regulated genes are disproportionately found in the brain, suggesting selection for influencing behaviour and hence, culture (Keverne, 2014). Epigenetics explains how long-term changes in brain chemistry are programmed by short-term experiences, especially during developmental windows in early life.

These epigenetic tags, such as DNA methylation, are reversible but can potentially persist across two generations if they occur in a woman pregnant with a daughter who carries all the eggs for her lifetime while still in the womb herself. This timescale is much more comparable with cultural change than classical selection for true allelic variation, and so an invaluable bridge between the two. To date, a major obstacle to even modelling gene–culture coevolution has been the different timescales, because culture can change very rapidly compared to gene frequencies.

The authors refer to prosocial norms as an example of a cultural psychological trait that has been modelled as culturally evolving and the associated trait of "social trust" can be provided here as an example of how epigenetics can inform models of gene–culture coevolution. Norms of social trust are also transmitted from birth both vertically and horizontally. A cultural spectrum influenced by norms of social trust runs, for example, from more collective cultures to more individualistic cultures.

Trusting behaviour is regulated by a hub of neurotransmitters and hormones including oxytocin and serotonin (Riedl & Javor, 2012). There are a number of genes that influence the levels of these chemicals such as the oxytocin receptor gene, OXTR, and the serotonin transporter gene, SERT. Both of these genes are polymorphic, with alleles associated with different levels of influence on trusting behaviour (Feldman, Monakhov, Pratt, & Ebstein, 2016; Iurescia, Seriipa, & Rinaldi, 2016). At least some of the alleles are also epigenetically regulated (Iurescia et al., 2016; Kumsta et al., 2013).

In mammalian history, a stressful world would typically have been one with a harsh environment resulting in strong competition for resources in terms of food, mates, and territory. In such an environment, positive trust and prosocial behaviour may have deadly consequences and come at too high a cost. However, the exact costs and benefits of trust may vary locally and over shorter timescales, and classic selection would be too insensitive. The advantage of epigenetic processes are that they enable gene expression to respond developmentally to these changing trade-offs caused by rapid environmental (*sensu lato*) signals.

The serotonin transporter gene illustrates how much variation in adaptation is possible given a gene that is both polymorphic and epigenetically regulated. The short allele of the SERT gene, "S," is less expressed than the long allele, "L." Caspi et al.'s (2003) study found that this common polymorphism of the serotonin receptor gene affects the probability of depressive episodes as the number of stressful life events accumulates (i.e., there is a gene–environment interaction mediating the risk of depression). S carriers were vulnerable to this effect because the S allele is epigenetically regulated – it is methylated in response to stress in early life. Methylation of the serotonin transporter gene is associated with altered emotional processing mediated by altered brain

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activity in regions including the amygdala and anterior insula (Frodl et al., 2015). The result is an increased response to fear which promotes mistrust. S carriers are less trusting, and more discriminatory to outgroups – especially under stress.

Rather than viewing the S allele as a risk factor, however, it is better modelled as facilitating a sensitivity that also has positive consequences. For example, S carriers are also more sensitive to social signalling generally – and derive more benefit from social support (Way & Lieberman, 2010). Social support is an effective buffer against the increased sensitivity to stress experienced by S carriers. Without variable levels of stress in their lives, the different effects of S and L alleles in the carriers would be undetectable. Clearly, an awareness of the different epigenetic regulation of S and L carriers is necessary to fully evaluate heritability estimates of associated traits such as social trust.

Global population differences in S and L allele frequencies are well-documented (Minkov, Blagoev, & Bond, 2015). S frequencies are consistently higher in East Asian populations than that in North European (70–80% S carriers vs. 40–45%). This prompts the questions: Have population differences in S and L allele frequencies come about by neutral processes or selection? Collectivism in Asia is associated with high S allele frequency, and there is some evidence that the increased social support in collectivist cultures might buffer against the increased risk of stress-vulnerability and depression (Way & Lieberman, 2010).

Socially sensitive OXTR allele frequencies also vary East to West in a similar way (Luo & Han, 2014), further supporting a gene–culture coevolution model involving social trust. An intervention in the United States providing social support to at-risk families has been found to be more effective with S carriers who derive more benefit from social support – and in this case there is also less OXTR methylation (Beach et al., 2018).

So, there appears to be a network of genes that are epigenetically sensitive to stress and possibly social stress in particular. Indeed, it is likely that the detection of the influence of single alleles is made possible because the phenotypic measure actually reflects selection at multiple related loci.

The suggestion that epigenetic regulation of socially sensitive alleles of genes such as SERT and OXTR can influence group level behaviour raises the possibility of epigenetically mediated gene–culture coevolution whereby ecological stress and/or social stress impacts cultural traits such as social trust. This is just one example illustrating how epigenetic effects may impact gene–culture coevolution and suggest the need for a triple inheritance model rather than a dual one, with further implications for sampling and estimating heritability.

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Measuring heritability: Why bother?

David M. Shuker[a] and Thomas E. Dickins[b]

[a]School of Biology, University of St Andrews, St Andrews KY16 9TH, UK and [b]Department of Psychology, Faculty of Science & Technology, Middlesex University, London NW4 4BT, UK.
david.shuker@st-andrews.ac.uk
T.Dickins@mdx.ac.uk
https://insects.st-andrews.ac.uk
http://tomdickins.net/

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Abstract

Uchiyama et al. rightly consider how cultural variation may influence estimates of heritability by contributing to environmental sources of variation. We disagree, however, with the idea that *generalisable* estimates of heritability are ever a plausible aim. Heritability estimates are always context-specific, and to suggest otherwise is to misunderstand what heritability can and cannot tell us.

Uchiyama et al. address the role cultural variation may play in influencing estimates of the quantitative genetic parameter of heritability, h^2. They argue, rightly in our view, that cultural differences among human groups can contribute an important component of environmental variation in human behaviour. Indeed, given the preponderance of social learning in human populations, it is right to consider how this source of variance may influence our estimates of heritability, by shaping the relationship between genetic and non-genetic sources of variation in behaviour and other traits.

Where we depart from Uchiyama et al.'s view is in terms of their emphasis on the generalisability of heritability estimates. Heritability estimates are *always* context specific (Falconer & Mackay, 1996; Visscher, Hill, & Wray, 2008). Even if one is able to identify, and then control for, components of environmental variance (as those studying quantitative genetic parameters in the wild often do: Charmantier, Garant, and Kruuk, 2014), one

is still left with a context-specific estimate. Importantly, this context specificity is not just in terms of the role of gene-by-environment (G × E) interactions – emphasised in the context of culture by Uchiyama et al. – but also in terms of *all* the components of variance that go into the heritability calculation, including other sources of environmental variance, and population genetic parameters such as the frequencies of causal alleles segregating in the population. This means that it is a mistake, and a misunderstanding, to expect a *generalisable* estimate of heritability for any given trait, an aim stated more than once by Uchiyama et al.

The interpretation of heritability is crucial. Uchiyama et al. provide many reasons for not over-interpreting heritability, but these are at odds with imagining that there could be such a thing as a "true" heritability. Heritability only speaks to the sources of variance expressed by a trait in a given sample. In terms of environmental sources of variation, if these are large then additive genetic effects may be swamped, but it does not necessarily mean that they are absent. Behavioural traits are often thought to be contaminated with large sources of environmental variation for example (for discussion see Dochtermann, Schwab, Anderson Berdal, Dalos, & Royauté, 2019; Stirling, Réale, & Roff, 2002). Similarly, if environmental sources of variation are small, then heritabilities may be high, but again this tells us rather little about the additive genetic variance itself. It is worse than that though, as additive and residual sources of variance may be non-independent (Hansen, Pélabon, & Houle, 2011; Houle, 1992). One alternative is the coefficient of additive genetic variation, CV_A, which may be more comparable across contexts (Houle, 1992).

As such, differences in heritability can be because of both differences in environmental and additive genetic components (bundling away non-additive effects for brevity), but of course the same is true for similarities: Heritabilities may be alike, but for different causal reasons. Uchiyama et al. talk about techniques such as polygenic scores in terms of unpicking the quantitative genetic basis of traits, but as they note recent research has shown that the causal variants identified by polygenic score methods do not replicate well across populations (including for intensively studied human populations and traits such as height, which are highly heritable across populations: Mathieson, 2021). This means that even if there was a "true" heritability, repeatable across populations (which there isn't), and we could unpick the cultural influences, it would not necessarily mean that we were looking at the same underlying genetics. And if we are not looking at the same underlying genetics, then what is the purpose of trying to generalise heritability estimates? It is after all uncontroversial that most traits exhibit heritable variation (Lynch & Walsh, 1998; but see Blows & Hoffmann, 2005 for complications).

Heritability is a useful statistic, particularly coming into its own in comparative studies across traits and organisms (Mousseau & Roff, 1987; Weigensberg & Roff, 1996). Hundreds of studies across animals have told us that morphological, life-history, and behavioural traits typically vary in their h^2 estimates, going from higher to lower respectively. Moreover, within a species, variation in heritabilities with age, for example, can give us hypotheses about (a) how selection acts at different ages, or (b) how developmental processes, and the genes and environments they influence and call upon, change over the lifetime (Wilson, Kruuk, & Coltman, 2005). But there is no generalisable, canonical h^2 waiting to be discovered. Developmental processes, via the moment-to-moment interactions of organisms in their environments, do not call on genes in such a way that could generate such a canonical measure; put simply, the whole genome is not scrutinised moment-to-moment by an organism, in its environment. Instead, if one wants such an over-arching genetic perspective, then the molecular basis of traits of interests needs to be considered more directly, one that embraces changes in gene expression, within- and across-tissues, across time, as the organism lives its way through its social, cultural, and other environments.

Why the emphasis on h^2? Given our clarification that a given h^2 estimate says rather little without understanding the underlying sources of variance, and indeed given much of Uchiyama et al.'s discussion, why view h^2 as potentially generalisable at all? We are not sure. Heritability is a fundamentally flawed way of arguing that some traits are more or less "genetic" in origin than others. *All phenotypes*, including culturally inherited behaviours and artefacts, have a genetic component to them, because the bodies and brains that produce those phenotypes are built by genes living in environments. Heritability does not speak to that aspect of the genetic basis of traits though, it only speaks to the variance in those traits. So, what are we trying to generalise?

For humans, culture is in the environmental mix in terms of sources of phenotypic variance. As such, controlling for cultural exposure may help reveal patterns in variation in h^2 that can lead to interesting hypotheses and further tests. But to over-emphasise heritability, and to imagine that it can be meaningfully generalised, is to misunderstand what it can, and cannot, tell us about the evolution of humans and other organisms.

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A cultural evolutionary behavior genetics will need a more sophisticated conceptualization of cultural traits

Moin Syed and Phuong Linh L. Nguyen

Department of Psychology, University of Minnesota, Minneapolis, MN 55455, USA.
moin@umn.edu; https://cla.umn.edu/about/directory/profile/moin
nguy4006@umn.edu; https://nguyenllpsych.github.io/

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Abstract

A framework that brings together cultural perspectives and behavior genetics has long been needed. To be successful, however, we need sophistication in the conceptualization of culture. Here, we highlight three imperatives to this end: the need for a clear definition of cultural traits, inclusion of the role of societal power, and recognizing the distinction between traits and characteristic adaptations.

Uchiyama et al. provide a much-needed framework for integrating cultural perspectives into behavior genetics research. Although we agree with the broad strokes of their arguments, we found some of the critical details lacking. In this commentary, we highlight how inattention to the definition of *cultural traits*, *societal power*, and distinction between *traits* and *characteristic adaptations*, can derail the proposed framework and potentially lead to a murky literature that does more harm than good.

Remarkably, given the centrality of cultural traits to their arguments, Uchiyama et al. never provide a definition of the concept. From the text, we infer that they use cultural traits equivalently to *cultural syndromes* (Triandis, 1996), which are culturally shared patterns of individual differences in beliefs, attitudes, and norms (e.g., individualism/collectivism and tightness/looseness). Syndromes are trait descriptions of the supra-individual level, most commonly nations, and are taken as indicative of the national psychology. Accordingly, an index of the syndrome is a proxy for the psychologies of the individuals therein. This *ecological fallacy* is common in cultural psychological work that focuses on syndromes, clearly evident in the research that takes a dichotomous view of cultures as individualistic or collectivistic and applies that to understand individual psychological phenomena (e.g., Kitayama & Park, 2021). Some argue that the ecological fallacy is irrelevant because inferences are restricted to the national versus individual level. However, measures of syndromes are based on aggregate individual-level reports (Brewer & Venaik, 2014), using highly select (Hofstede, 1980) or small (Gelfand et al., 2011) samples that are not representative of the populations. Uchiyama et al. magnify the problem by suggesting that cultural tightness/looseness (a proxy for individuals' beliefs) can itself serve as a proxy for *tolerance for diversity*, which, in turn, is one possible factor that may contribute to cultural heterogeneity and rate of innovation. This line of argument, thus, goes through several layers of abstraction and assumptions. The flaws of using proxies are evident, but their convenience perpetuates their use. If we take the authors' goal seriously – the importance of understanding cultural and environmental characteristics beyond simple national boundaries – then abandoning convenient practices in favor of rigorous practices is a must. This is a larger problem for behavior genetics research, which tends to favor complex designs over well-measured phenotypes (Tam et al., 2019). Using a proxy measure (tightness/looseness) as a proxy for another concept (tolerance for diversity) will yield poor answers to otherwise good questions.

Moreover, there is no clear criteria that demarcates a set of traits as constituting a syndrome, and thus they can be any random assortment of traits that are bundled together and labeled. Indeed, Muthukrishna et al. (2020) did exactly this by unsystematically aggregating responses from the World Values Survey to create the cultural fixation index (CF_{ST}), promoted in the target article. Similarly, the WEIRD (western, educated, industrialized, rich, and democratic) acronym (Henrich, Heine, & Norenzayan, 2010) was developed through a non-systematic process that omits highly relevant dimensions of variation (e.g., race and religion). So, what makes a set of traits *cultural*? Following the authors' arguments on the pervasive influence of cultures, wouldn't all traits be cultural traits? How would we decide the degree to which a trait is cultural?

Additionally, on whose authority do a set of traits become cultural? Any conceptualization of culture, related to traits or otherwise, is necessarily incomplete without a serious consideration of societal power structures, which is jarringly absent from Uchiyama et al. This is not necessary only for the conceptualization of cultural traits, but for their whole set of arguments. How is innovation promoted or constrained? How are cultural clusters formed? Why is the environment more variable for some groups than others? The authors seem to suggest these conditions just emerge as part of a natural process rather than being intentional acts by those in power. Any model of cultural genetics must take societal power into consideration. The authors make brief references to colonial influences on the clustering of cultural groups, but this requires more explicit attention, given how it is a powerful driving force to forming the clusters central to the proposed model.

Finally, the conceptualization of cultural traits raises questions not only about how traits are bundled together into syndromes, but also whether what is bundled are traits at all. Personality psychology has coalesced around a distinction between traits and characteristics adaptations (McAdams & Pals, 2006). Although traits are relatively stable patterns of individual differences in attitudes, behaviors, and cognition that exist in all societies (e.g., extraversion and sensation-seeking), characteristic adaptations are goals, interpretations, and strategies grounded in culture (DeYoung, 2015). Given the loose definitions and lack of psychometric work, it is not clear whether cultural traits consist of traits, characteristic adaptations, or a mix of both.

The distinction between traits and characteristic adaptations is of central importance not only to the conceptualization of cultural traits, but also to the target phenotype when trying to understand what heritability tells us about cultural evolutionary processes. Although the majority of behavior genetic research has focused on traits, the evidence suggests that characteristic adaptations show lower heritability and greater shared environment compared to traits (Nguyen, Syed, & McGue, 2021). Using an example from Uchiyama et al., political ideology (trait) has higher heritability and lower shared environment than party affiliation (characteristic adaptation; Alford, Funk, & and Hibbing, 2005). Failing to appreciate these taxonomic distinctions in individual differences could lead to erroneous conclusions within a cultural evolutionary behavior genetic framework. This problem can be difficult to

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detect, though, because a construct might be conceptualized as a characteristic adaptation but measured as a trait (or vice-versa), and characteristic adaptation-like items in aggregate might also create a trait measure. The lack of attention to this distinction highlights the potential problems of using heritability estimates of phenotypes to infer cultural evolutionary processes.

To reiterate, we are broadly supportive of the framework outlined by Uchiyama et al., and anticipate it will generate greater sophistication in work bringing together cultural evolution and behavior genetics. It will only be successful, however, by incorporating a more sophisticated understanding of cultural traits.

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This time I mean it: The nature–nurture debate is over

Eric Turkheimer

Department of Psychology, University of Virginia, Charlottesville, VA 22904-4400, USA.
ent3c@virginia.edu; https://uva.theopenscholar.com/eric-turkheimer

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Abstract

The target article is skeptical of the heritability concept while maintaining an old-fashioned point of view about it. As a descriptive statistic, it is to be expected that heritability goes up and down in different circumstances, but the relationship between heritability coefficients and the biological processes that underlie them is difficult to specify, and may be impossible in humans.

Twenty years ago, I wrote a paper (Turkheimer, 2000) that began with the sentence, "The nature–nurture debate is over." That assertion has sometimes been misinterpreted to mean that one side had been victorious, but that is not what I meant. It's true that I predicated the idea on the results of the twin studies of the era, which showed over and over again that identical twins are more similar than fraternal twins for pretty much everything, from height to how much TV they watch. My conclusion, however, was not that the causally vague genetic "influence" implied by $r_{MZ} > r_{DZ}$ actually demonstrated that television watching is in some important sense "genetic." Instead, my point was that nature has certain methodological advantages over nurture. Genes are relatively discrete and developmentally stable, whereas the environment is amorphous and ever-changing. Genes are not more influential; rather the effects of genes are easier to detect and quantify than the effects of the environment.

This entire field of study was undertaken by Francis Galton, who gave the nature–nurture debate its unfortunate name. The discussion, then as now, was conducted in terms of a number called a heritability coefficient. The quantitative notion of heritability is based on ideas developed by R. A. Fisher, who derived ratios of genotypic over phenotypic variances that expressed, in some eternally fraught sense, the extent to which phenotypic differences could be "attributed" to genotypic differences. Galton and Fisher interpreted their primitive statistics, as more modern scientists interpret their structural equation models, as methods for keeping score in the nature–nurture tournament. The modern twin study era was conducted in this spirit. Summaries of behavior genetics by the twin researchers of the time (e.g., Bouchard, 2004), fairly bristle with certainty that the estimation of big heritability coefficients proves that genetic differences are the dominant force shaping human differences.

The target article, with its interest in non-genetic forms of generational transmission, allows itself some skepticism about the old heritability concept, but remains committed to an old-fashioned interpretation of it. The authors refer to heritability as both a number (once, unfortunately, as a "score"), and as a biological process in which differences in phenotype are passed from one generation to the next with differing degrees of determination. There is nothing wrong with documenting the vicissitudes of heritability coefficients, but noting developmental and socioeconomic trends in heritability coefficients is one thing, and understanding the causal interplay of biological and cultural processes in the determination of phenotypic differences is another. Although I am skeptical whether the developmental processes underlying human heritability coefficients can ever be worked out causally or quantitatively, I do think that the rich appreciation of genetically and culturally informed human complexity has led somewhere very important.

In the years since Bouchard (2004), behavior genetics has been revolutionized by the onset of the human genome project. The roller-coaster of scientific events is by now familiar: linkage

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analysis didn't find any genes of large effect; candidate gene studies didn't find any genes of medium effect; genome-wide association study (GWAS) didn't find any genes of tiny effect; GWAS was able to estimate human heritabilities without twins, but they were much smaller than twin heritabilities; GWAS was used to sum tiny DNA effects into polygenic scores (PGSs), which were modestly correlated with behavioral phenotypes; those PGSs are able to make genetic discriminations within families in ways that twins are not; in particular they are able to separate genetic effects on parents transmitted environmentally to offspring from genetic effects originating directly in the offspring genome; and all of these analyses are confounded in complex ways by genetic, ethnic, and phenotypic clustering, that is, culture.

But in the newest genomic studies of human behavior, something remarkable has happened. Chastened, perhaps, by the absence of any actionable genes, by the diminishing heritabilities, by the elusiveness of meaningful biology, by the less than practical performance of PGSs, yet spurred on by the deep fractal complexity of modern genomics, the most recent behavior genetic papers include no nature–nurture content whatsoever. Consider, for instance, the recent GWAS of sexual orientation (Ganna et al., 2019). A few single-nucleotide polymorphism (SNP) hits were identified, some small heritability was quantified, and on a charitable reading some interesting biological and behavioral pathways were suggested. All of which is fine, and may or may not lead to insight in the long run. But in the meantime, the paper contains absolutely zero Bouchardian litigation of nature–nurture issues as regards sexual orientation.

When I first wrote in these pages (Turkheimer & Gottesman, 1991), we were responding to a target article by Plomin and Bergeman (1991) that, ironically, had a great deal in common with the current one. Where the current article notes that the transmission of differences across generations often occurs along cultural rather than strictly genetic pathways, Plomin and Bergeman (1991) argued that ostensibly environmental modes of transmission often encompass genetic variance. From the point of view of a social scientist trying to sort it all out, it is the same conclusion viewed through the nature–nurture looking glass. In response, Gottesman and I concluded,

> Our concern is about where all this will lead. Behavior is influenced by genotype and environment. The environment provided by a parent is influenced by the parent's (not to mention the child's) genotype, and the parent's rearing environment, which had its own tangle of reciprocal genetic and environmental influences. Everything is intercorrelated; everything interacts. Where does this leave the columns of "model-fitting heritabilities," meticulously computed to two decimal places and starred for statistical significance on the basis of path models that cannot hope to keep pace with the reciprocal causal structures described in the target article?

We now know where it led: To the end of nature–nurture as a serious question to be debated by genetically informed social scientists. Good riddance.

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Hidden clusters beyond ethnic boundaries

Alejandro Peréz Velilla, Cody J. Moser and Paul E. Smaldino

Department of Cognitive and Information Sciences, University of California, Merced, CA 95343, USA.
aperezvelilla@ucmerced.edu; cmoser2@ucmerced.edu
psmaldino@ucmerced.edu; https://smaldino.com/wp/

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Abstract

Hidden cluster problems can manifest when broad ethnic categories are used as proxies for cultural traits, especially when traits are assumed to encode cultural distances between groups. We suggest a granular understanding of cultural trait distributions within and between ethnic categories is fundamental to the interpretation of heritability estimates as well as general behavioral outcomes.

The target article argues that accounting for human social categories is essential for understanding aggregate measures of ostensibly non-social phenomena, such as the heritability of intelligence. Uchiyama et al. rightly highlight what they call the "hidden cluster problem," (sect. 3), in which geopolitical or ethnolinguistic boundaries often used by geneticists to account for culture may fail to accurately represent important cultural and even environmental clustering. We agree that this is a problem, and further propose that the hidden cluster problem creates challenges not only for behavioral genetics, but also for social scientists who want to better understand the full spectrum of influences to cultural and behavioral traits. We draw particular attention to the classic anthropological work of Barth (1969) in outlining the importance of developing more granular understandings of human cultural trait distributions.

As the human behavioral sciences have expanded over the last several decades, special attention to sampling and methodological issues, such as the WEIRD (western, educated, industrialized, rich, and democratic) sampling and causal locus problems discussed by Uchiyama et al., have been extensively explored. In contrast, the hidden cluster problem has received less attention outside of the social sciences despite recurrent examples of cultural clustering in social-scientific and ethnographic accounts (Colleran, 2020; Henrich, Heine, & Norenzayan, 2010; Richerson & Boyd, 2008; Schulz, Bahrami-Rad, Beauchamp, & Henrich, 2019). Hidden clusters represent a particularly pernicious problem because the categories that individuals use to socially identify themselves may not necessarily map onto well-defined cultural trait distributions. Consider, for

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example, caste and ethnicity in India and differentiation *within* these categories: Biswas and Pandey (1996) found that traditionally defined categories of identity in India did not map well onto self-perceived economic condition or social status once one accounts for economic mobility (also see Schooler, 2010). This means that using these endemic social categories with the aim of controlling for cultural differences between individuals does not ensure that the cultural differences represented by those categories are the ones most relevant for studying the problem at hand.

Hidden clusters can confound behavioral analysis when self-ascribed ethnicity is used as a proxy for culture, especially when there is scarce information about the distribution of cultural traits within and across ethnic groups in a society. In the case of estimating heritability, this can potentially blind us to the extent of cultural heterogeneity in the social environment, as outlined in the target article. Scholars going back as far as Barth (1969) have warned that self-ascribed ethnicity should not be used as a proxy for substantive cultural trait distributions between ethnic groups. Rather, because ethnicities emerge in the interactions between groups and cluster around particular cultural dimensions that give rise to demarcation, they do not reflect the entirety of the cultural trait distribution of a group, only the part of it that is relevant to the boundaries of group membership. Knowing these boundaries means knowing across which cultural dimensions groups differentiate from one another, which relates directly to the degree of heterogeneity of the social environment and its effect on heritability estimates.

Consider a society where two ethnic groups are differentiated by their cooking practices, but share other cultural traits, such as kinship norms, in common. These self-ascribed ethnic categories, demarcated by differences in cooking practices, tell us nothing about the clustered nature of kinship norms across ethnic groups. If (for the purposes of this example) kinship norms are causally intertwined with political preferences, and we want to study the genetic heritability of political preferences, researchers may be presented with hidden environmental homogeneity, which would increase the measured genetic heritability of political preferences, leading to the erroneous conclusion that one is accounting for cultural differences in the behavioral dimensions of interest. Using self-ascribed ethnicity as a proxy for culture opens us up to the risk of ignoring hidden clusters, leading us to overestimate the heritability of political preferences because of the residual variation explained by genes in our example.

In its moment (and beyond), the Barthian notion of ethnic identity was important for proposing ethnic ascription as an indicator of group-level interactions at work. In the study of cultural evolution, this way of thinking about identity has been generalized beyond ethnicity, particularly in contemporary notions of social identity and its relation to behavioral clustering (McElreath, Boyd, & Richerson, 2003; Smaldino, 2019; Smaldino & Turner, 2021). Social identities emerge in an evolving ecology of groups, and thus are driven by potential patterns of coexistence, cooperation, competition, domination, dependence, hierarchization, and so on. In human societies, where social identities can be nested and multidimensional (in great part owing to the possibility of multiple group membership), understanding which cultural clusters correlate with which facets of social identity is necessary in order to construct a sufficiently clear view of a society's cultural trait landscape. For example, the social identity dimension of social class can be defined, at least in part, by the bounded set of cultural traits that correlate with socioeconomic status (Bourdieu, 1987). Certain traits, in this example, may be shared by all high socioeconomic status members of a society, regardless of ethnicity (even though ethnicity is often non-independent of socioeconomic status across groups). From this view, it becomes clear that, if the aim is to explain where a particular behavior comes from and the extent to which it is genetically heritable, the goal of understanding a society's cultural dynamics with fine granularity in regards to its unique traits – and even more critically, the extent to which these traits overlap with other cultural groups – must be incorporated into the research process.

Understanding the emergence of cultural clusters is a work in progress, with both theory and methodology still under construction. That said, turning a blind eye to cultural clustering in societies of interest is a potential problem for any science of human behavior that seeks to account for the effects of cultural differences. Accounting for the existence of hidden cultural clusters should be a default aim for *all* behavioral sciences, including but not limited to behavioral genetics.

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Culture is reducing genetic heritability and superseding genetic adaptation

Timothy M. Waring[a], Zachary T. Wood[b] and Mona J. Xue[c]

[a]School of Economics, Mitchell Center for Sustainability Solutions, University of Maine, Orono, ME 04469, USA; [b]School of Biology and Ecology and Maine Center for Genetics in the Environment, University of Maine, Orono, ME 04469, USA and [c]Department of Human Evolutionary Biology, Harvard University, Peabody Museum, Cambridge, MA 02138, USA.
timothy.waring@maine.edu; https://timwaring.info/
zachary.t.wood@maine.edu; https://ztwood.weebly.com/
monajixue@gmail.com

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Abstract

Uchiyama et al. reveal how group-structured cultural variation influences measurements of trait heritability. We argue that understanding culture's influence on phenotypic heritability can clarify the impact of culture on genetic inheritance, which has implications for long-term gene–culture coevolution. Their analysis may provide guidance for testing our hypothesis that cultural adaptation is superseding genetic adaptation in the long term.

Uchiyama et al. have made an important contribution to dual inheritance theory. To date, dual inheritance theory has focused mainly on the coevolution of a pair of genetic and cultural traits (e.g., Gerbault et al., 2011) or on the genetic evolution of cultural evolution itself (e.g., Henrich & McElreath, 2003). The question of how genetic evolution might itself evolve culturally has received less rigorous inquiry. Uchiyama et al. broach this issue, showing how cultural adaptation (especially group level cultural adaptation) can change genetic heritability by intervening between genes and their effects on survival and reproductive outcomes. In a recent article, two of us explore this same dynamic from the perspective of long-term gene–culture coevolution (Waring & Wood, 2021). Here, we consider the implications of the insights of Uchiyama et al. on long-term gene–culture coevolution.

The authors argue that cultural contributions to phenotypes can modify genetic heritability, an important and often overlooked point. Phenotypic variation in humans is the result of genetic, environmental, and cultural factors, and their interactions. Genetic heritability can be given as:

$$\frac{V_G}{V_G + V_c + V_e}$$

where V_G, V_c, and V_e denote phenotypic variation with a genetic, cultural, and environmental basis, respectively. Our capitalization is consistent with Uchiyama et al., but our formulation differs in order to highlight the idea that cultural effects on phenotype are not limited to acting through environmental variation.

The influence of culture on genetic heritability is complex, and the result of indirect feedbacks by which one V component affects another. Culture may generate phenotypic variation directly (increasing V_c), affect environmental (or ecological) variation indirectly (changing V_e), and mask or unmask genetic variation (decrease or increase V_G). For example, medicine can reduce the effect of diseases (an environmental variable) on health, reducing the role of immunity genes in determining phenotypic outcomes, but making health contingent on the health system one is part of. In this example, the novel cultural adaptation decreases V_e and V_G and increases V_c. These changes would decrease genetic heritability if changes in V_G and V_c outweigh those in V_e. Therefore, the overall impact depends on the relative phenotypic contribution of each type of variation.

Uchiyama et al. appear to assume that cultural evolution tends to decrease cultural and environmental variation within groups, citing mechanisms such as conformist learning (Henrich & Boyd, 1998), prestige-biased learning (Henrich & Gil-White, 2001), and success-biased learning (Baldini, 2012). However, structured group-level cultural traits complicate the argument (Smaldino, 2014). Because of specialization and divisions of labor, social learning mechanisms often generate adaptive cultural complexity within a society, rather than merely homogenize it. For traits that mask genetic effects, increases in cultural variation, V_c, cause decreases in genetic heritability. Thus, there may be more scenarios which decrease genetic heritability than previously thought.

Cultural influences on genetic heritability have major consequences for human evolution well beyond those discussed by Uchiyama et al. A key point they omit is how these cultural influences would alter genetic adaptation and evolution. Increased heritability strengthens the evolutionary response of functional genes to selection, while decreased heritability weakens this response (Lush, 1943). Laland (1992) has shown how the transmission of an adaptive behavior via social learning can preempt adaptation by natural selection on genes. This effect should be the expected result. Therefore, students of human evolution should ask whether there is any average long-term trend in culturally mediated changes to heritability.

We hypothesize a general directionality to the role of culture in determining phenotypic variation in the long term: Culturally determined phenotypic variation is increasing (V_c is growing), and cultural evolution is simultaneously decreasing genetically determined phenotypic variation by breaking the link between genotype and phenotype (V_G is shrinking). For example, educational attainment depends on both genetic and cultural factors, and generally comes at a reproductive cost. But, as Hong (2020) shows, educational attainment is likely to continue to increase even while the genetic component declines.

We think that V_c has increased, not for all traits at all times, but as part of a long-term average trend across human societies over the course of evolution. Evidence for this comes from the broad and striking increase in the emergence, diversification, and refinement of cultural systems and technology that improve human fitness outcomes in food production, collective defense, health, and so on. Such complex group-level cultural adaptations increase cultural variation and, thus, V_c, in the human species. When V_c increases, then genetic heritability decreases. Thus, we suspect, along with Uchiyama et al. and others, that cultural adaption has already been replacing genetic adaptation in humans (e.g., Mathew & Perreault, 2015). However, we also hypothesize that this trend, highlighted by decreasing genetic heritability, will continue in the long term.

Together, cultural preemption (or masking) and increased V_c are expected to reduce genetic heritability, and this has dramatic implications for the future of human evolution (see Waring & Wood, 2021). As the response of any trait to selection depends in part on that trait's heritability, culturally mediated reductions in genetic heritability could weaken the role of genetic evolution in shaping human fitness and adaptation. At the same time, reductions in genetic heritability and genetic adaptation in humans could pave the way for a more predominant role of culture in human evolution, creating an accelerating positive feedback (Crespi, 2004). This trend is not just a curious possibility but represents a dominant and growing mode in human gene–culture coevolution over long time scales (Waring & Wood, 2021).

We feel that Uchiyama et al. help geneticists and social scientists better understand how genes and cultures interact to shape human heritability. But, we believe the most important implication of their work is the emergence of culture as our primary system of evolutionary adaptation, a long-term pattern which uniquely defines the human species in the past, present, and future.

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Understanding cultural clusters: An ethnographic perspective

Polly Wiessner[a,b]

[a]School of Human Evolution and Social Change, Arizona State University, Tempe, AZ 85287, USA and [b]The University of Utah, Salt Lake City, UT 84112, USA.
wiessner@soft-link.com
https://shesc.asu.edu/people/pauline-wiessner

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Abstract

The cultural evolutionary approach to the dynamics of cumulative culture is insufficient for understanding how culture affects heritability estimates; it ignores the agency of individuals and internal complexity of social groups that drive cultural evolution. Both environmental and social selection need consideration. The WEIRD (western, educated, industrialized, rich, and democratic) problem has never plagued anthropology: A wealth of ethnography is available for the problem at hand.

The authors have tackled the important question of how and why genetic effects are often obscured by effects of cumulative culture, applying a dual inheritance framework. They attribute this deficit in part to the WEIRD (western, educated, industrialized, rich, and democratic) sampling problem where western subjects are considered to be representative of our species. They then seek the cultural "generative processes that bring such complex objects and conditions into existence" (sect. 5.1, para. 1) proposing that answers can be reached through a cultural evolutionary framework. Fortunately, the WEIRD problem has never plagued anthropologists whose goal has always been to document cultural systems so that other societies do not appear weird. What can we learn from the vast body of ethnography collected over centuries to further our understanding of the dynamics of cultural clusters?

The authors take a diffusionist perspective with the unit of selection being cultural group selection. Innovations are proposed to spread by conformist biases, learning from successful others, and punishing norm violators to produce cultural clusters. However, this approach sidesteps the ethnographically documented agency of individuals and internal complexity of social groups which so drives cultural evolution. At least three forces of selection acting upon agents need to be considered to understand how cumulative culture and cultural clusters are generated and evolve. The first is adaptation to the natural or culturally altered environment. The second is social selection (Hrdy, 2016; Nesse, 2007; West-Eberhard, 1979), a frontline selection pressure that partially determines with whom one marries, cooperates in child rearing, and exchanges, as well as how many supporters and opponents one has over the life cycle. It has everything to do with shaping our psychology and masking or unmasking estimates of genetic heritability. Take the skin cancer example. The use of ochre and other means for sun protection appears to over 100,000 years old (Lüpke et al., 2020). Today, the Himba women in Namibia use fat and ochre to maintain sexually attractive skin; men do not. The heritability of cancer for one gender only will be masked (Summers et al., nd).

Social selection appears to be responsible for our better angels and devils. Boehm (2012) and Wrangham (2019) have proposed that it was the key process for our self-domestication. Nesse (2016) has argued that only social selection can explain our extreme pro-sociality. Moreover, social selection is a strong force behind the universal human obsession with reputation. It can lead to oppression, exploitation, and manipulation, creating segments within societies. Knowing who benefits from innovations is essential for understanding the internal configurations of cultural clusters. Finally, there is cultural group selection for which evidence remains debated (commentary, Richerson et al., 2016). The impermeability of barriers created by mutually unintelligible languages or cultural clusters that would facilitate cultural group selection is often overestimated. As Barth (1998) has proposed, ethnic distinctions do not depend on the absence of social interaction and acceptance, but on the contrary, are often the very foundations on which embracing social systems are built. Open boundaries are evidenced by the fact that many people in traditional and modern societies speak three to four languages, a trend pronounced in South America and West Africa (Lüpke et al., 2020).

Cumulative culture comes in single tools or traits, complex subsistence strategies, and entire cultural institutions. The level of selection operant on different features of cumulative culture profoundly structures their import for obscuring genetic heritage. Many traits that distinguish social clusters are functionally equivalent and of purely symbolic value; they are products of social selection to define clusters of people who share obligations to one another. Examples from Highland Papua New Guinea abound as linguistic and dialect groups distinguish themselves by signature styles of body decoration and ceremonial dress. Some groups choose other means: The Etoro, Onabasulu, and Kakuli of the Bosavi area differentiate by customs in male initiations. Growth and maturation are contingent on insemination with the semen of elders; how semen is transmitted through

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different forms of intercourse is believed to produce culturally distinct beings (Kelly, 1974). Such traits marking identity stand fast at boundaries regardless of rates of interaction.

Traits that mark identity obscure many essential commonalities across cultures. Complex cultural packages that confer strong selective advantages spread rapidly across boundaries allowing people from distinct linguistic and cultural groups to share many behaviors and adaptations. I will give three examples. (1) Sweet potatoes, introduced to Highland Papua New Guinea cultures some 400 years ago, released constraints on agricultural production and spread widely in response to environmental pressures long before first contact with Europeans (Ballard, Brown, Bourke, & Harwood, 2005). Utilization of the new crop created homogeneity in subsistence practices and pig husbandry across vast areas, similarities which could be obscured by linguistic differences and expressions of cultural identity. (2) Among the Enga, bachelors' cults to discipline and educate cohorts of youths arose before European contact in some clans of central Enga under conditions of intense competition in trade, ceremonial exchange, and warfare. Big-men along trade routes identified successful clans from whom to purchase the transformative rites to improve clan fortune, while bachelors raised the wealth and went on journeys to do so (Wiessner & Tumu, 1998). Within two to three generations, bachelors' cults were adopted by 500 or more clans in the five dialect groups of Enga, fostering group loyalty and masking individualistic agendas. Meanwhile influential big-men exerted pressures to proclaim their enterprising sons as marriageable years before others to jump start their polygynous careers, creating reproductive inequalities. (3) Intraclan institutions applying restorative justice were adopted across linguistic groups in most Highland societies where intergroup competition was fierce. Social selection drove their development to bring potentially productive transgressors back into community, compensate for harm done, restore cooperation, and avoid the grudging conformity that ensues from punishment. Restorative measures fostered tolerance, openness, and innovation (Wiessner, 2020).

The many selection pressures that operate on agents who steer the course of cultural evolution must be considered to understand how cultural heterogeneity and homogeneity are generated and whether their content is significant for masking or unmasking genetic inheritance. For this purpose, the ethnographic record is most valuable.

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Cultural evolution may influence heritability by shaping assortative mating

Tian Chen Zeng and Joseph Henrich

Department of Human Evolutionary Biology, Harvard University, Cambridge, MA 02138, USA.
tianchen_zeng@g.harvard.edu
joseph.henrich@gmail.com

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Abstract

Uchiyama et al. productively discuss how culture can influence genetic heritability and, by modifying environmental conditions, limit the generalizability of genome-wide association studies (GWASs). Here, we supplement their account by highlighting how recent changes in culture and institutions in industrialized, westernized societies – such as increased female workforce participation – may have increased *assortative mating*. This alters the distribution of genotypes themselves, increasing heritability and phenotypic variance, and may be detectable using the latest methods.

Nearly 50 years ago, mathematical concepts from population genetics were first applied to understand how cultural evolution could shape genetic heritability (Cavalli-Sforza & Feldman, 1973). Building on those efforts and much subsequent work, Uchiyama et al., productively fuse modern *dual-inheritance theory* (Henrich & McElreath, 2007; Laland, Brown, & Brown, 2011) – which can account for how cultural evolution has shaped our environments in specific ways – with prevailing ideas from behavioral genetics, including approaches to studying how the effects of large numbers of genetic variants additively combine to shape heritable phenotypes in genome-wide association studies (GWASs). Stated simply, any dataset of phenotypes is shaped by two landscapes of variation, one genetic and another cultural, that were generated by different evolutionary processes and transmission rules.

The authors' ideas imply that much of the *exposome* (Wild, 2012) is shaped by transmissible cultural traits and therefore possesses its own population history, structure, and dynamics. Because the environment influences genetic effects, either in aggregate (e.g., Amin et al., 2017) or for single genes (Gauderman et al., 2017; e.g.,

Rask-Andersen, Karlsson, Ek, & Johansson, 2017), the tendency of GWASs to focus on populations that have evolved culturally in ways that minimize certain kinds of variation – through mechanisms such as universal schooling, social safety nets, and parasite-free environments – limits the scope of their inferences and biases variant discovery toward those that impact the phenotype in this limited environmental range.

One important mechanism by which culture evolution can drive up genetic heritability, which the authors only allude to, involves covariation between genes and sociocultural environments caused by the sorting of genotypes into specific environments ("reciprocal causation," Dickens & and Flynn, 2001). Recent cultural and institutional changes in western, educated, industrialized, rich, and democratic (WEIRD) societies are especially likely to have strengthened such sorting. In fact, purely cultural changes (no natural selection), by increasing social sorting, may even alter genotype distributions themselves through *assortative mating* – which occurs whenever spouses resemble each other phenotypically and genetically.

Assortative mating increases the additive genotypic variance directly (recall, $h^2 = V_g/V_{total}$). Additionally, culturally induced assortative mating can – if it occurs *across* traits (e.g., if tall men marry educated women) – create a genetic correlation between two different traits (Keller et al., 2013). If a genetic correlation was induced by assortment, it would have arisen purely from population-level phenomena and not from physiological or developmental processes. Such effects are notable for two reasons: (1) through assortative mating, cultural change can systematically alter, sometimes quite rapidly, a statistic-like genetic variance or genetic correlation without influencing natural selection; (2) Uchiyama et al. largely focus on the denominator of the expression for heritability, that is, the cultural contribution to the total phenotypic variance ($V_{culture}$ in $V_{total} = V_E + V_{culture}$), and less on what we highlight here – heritability's numerator.

How assortative mating increases genotypic variance is well-understood (Peyrot, Robinson, Penninx, & Wray, 2016). Intuitively, more people would have extreme genotypic values for height if the tall mated with tall and the short with short. However, why should assortative mating have increased in recent cohorts? First, a growing proportion of those populations typically sampled in genetic studies participate intensively in institutions that have an explicit sorting function – such as schools, universities, specialized occupations, and labor markets; this is accelerated by the dismantling of social barriers (e.g., during the entry of women into the workforce; Breen & Andersen, 2012; Greenwood, Guner, Kocharkov, & Santos, 2014). Relative to traditional forms of communality such as religious institutions or neighborhoods, such institutions also increasingly shape our social lives – and our mating opportunities. Second, with social liberalization, WEIRD people have an increasingly homogeneous exposure to the set of social niches and behavioral choices offered by those aforementioned institutions, in addition to those offered by consumption, lifestyle, and entertainment markets, increasing the scope for *self*-selection into specific social environments. Third, major shifts in norms, technology, and economic behaviors lead to increased urbanization and increased geographic and relational mobility (Ancestry.com, 2020; Maas & Zijdeman, 2010). It also changes in how people meet their mates, with social contexts possessing low relational mobility such as church or neighborhood declining in importance relative to high relational-mobility social environments such as bars, the workplace, or dating apps (Rosenfeld, Thomas, & Hausen, 2019). This grows the pool of potential mates, increasing the efficiency of assortative mating.

These effects have not been directly demonstrated, but a growing body of evidence indicates that the requisite conditions exist. Some social groups in modern industrialized societies, such as occupational groups, are behaviorally differentiated at both the phenotypic and genotypic levels. For example, individuals employed in STEM have increased autism spectrum quotients (Daysal, Elder, Hellerstein, Imberman, & Orsini, 2021; Ruzich et al., 2015), and the incidence of autism among newborns is elevated in regions with high-occupational participation in STEM, suggesting some genetic sorting (Roelfsema et al., 2012). In an Icelandic dataset, high polygenic scores for schizophrenia and bipolar disorder strongly predict participation in creative industries and membership in artistic societies (Power et al., 2015); these correlations were replicated in Sweden (MacCabe et al., 2018). High polygenic scores for these two disorders also contribute to increased time in the educational system (Bansal et al., 2018; Demange et al., 2021); and relatives of tenured university academics suffer elevated rates of these disorders (Parnas, Sandsten, Vestergaard, & Nordgaard, 2019). If mating within such behaviorally differentiated groups has increased over time because of the sociocultural and institutional changes we highlighted previously, assortative mating would strengthen, which indeed has happened for educational and occupational specializations (Eika, Mogstad, & Zafar, 2019). High rates of within-trait and cross-trait assortative mating for psychiatric diagnoses at the phenotypic level were found in a dataset drawn from the Swedish population register (Nordsletten et al., 2016).

Together, these theoretical insights and empirical facts suggest that culture can, and likely has, influenced genetic heritability through multiple pathways, including – in addition to the effects suggested by Uchiyama et al. – assortative mating. The target article presents a window into fascinating processes entangling genes and culture that deserve to be studied empirically with the newest methods. Such a program would represent the flowering of a dual-inheritance theory that has been fortified by modern data and research designs.

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Authors' Response

Integrating cultural evolution and behavioral genetics

Ryutaro Uchiyama[a], Rachel Spicer[b] and Michael Muthukrishna[b]

[a]NTU–Cambridge Centre for Lifelong Learning and Individualised Cognition, Nanyang Technological University, Singapore 639798 and [b]Department of Psychological and Behavioural Science, London School of Economics and Political Science, London, WC2A 2AE, UK
ryutaro.uchiyama@ntu.edu.sg;
https://www.uchiyamaryutaro.com
r.a.spicer@lse.ac.uk
https://www.lse.ac.uk/PBS/People/Rachel-Spicer
m.muthukrishna@lse.ac.uk
https://michael.muthukrishna.com

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Abstract

The 29 commentaries amplified our key arguments; offered extensions, implications, and applications of the framework; and pushed back and clarified. To help forge the path forward for cultural evolutionary behavioral genetics, we (1) focus on conceptual disagreements and misconceptions about the concepts of heritability and culture; (2) further discuss points raised about the intertwined relationship between culture and genes; and (3) address extensions to the proposed framework, particularly as it relates to cultural clusters, development, and power. These commentaries, and the deep engagement they represent, reinforce the importance of integrating cultural evolution and behavioral genetics.

R1. Introduction

Our hope in writing our target article was to start a conversation between cultural evolution and behavioral genetics. These two disciplines occupy the same space in attempting to explain variation in human behavior but haven't sufficiently engaged with one another. And yet, a richer understanding of the role of culture and genes on behavior has implications for the broader human sciences, as well as public discourse. The commentaries we received reinforced the importance of this discussion and we hope the discussion continues beyond the pages of *Behavioral and Brain Sciences*.

A truly interdisciplinary group of scholars responded. Not just researchers in behavioral genetics and cultural evolution, but also evolutionary biology more broadly, anthropology, psychology, psychiatry, education research, and philosophy. We are overwhelmed by the response, and we thank the authors of these commentaries for refocusing, challenging, amplifying, and expanding the arguments in our target article.

The goal of our reply is to push forward cultural evolutionary behavioral genetics. To do this, we focus on the challenges and extensions to our proposed framework. Our reply is organized as follows: First, we deal with the discrepancies and misconceptions about key concepts, particularly related to heritability and culture. Next, we address commentaries related to how culture and genes interact to produce behavior. Finally, we respond to the many proposals, questions, and critiques that serve to expand the scope of our proposed cultural evolutionary behavioral genetic framework.

R2. Conceptual clarifications

The first challenge for interdisciplinary work is developing a common language. We agonized over phrasings and definitions, recognizing that our readers represent such different disciplines with unfamiliar jargon and even common words for different concepts, and yet the curse of knowledge left some key terms undefined. The few discrepancies and misconceptions revealed by the commentaries, particularly around the concepts of heritability, culture, and related terms, reveals that there's more work to be done in developing common language and common understanding. Let's begin with heritability.

R2.1. Heritability

Shuker & Dickins emphasize that there is no general heritability for a trait nor is heritability a measure of the genetic basis of traits.

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They frame their commentary as an argument against the way in which they read heritability as being discussed in our target article, which we must admit was surprising to us. We completely agree with both emphases. Indeed, the context-dependence of heritability was the crux and starting point for our framework. We described various processes that empirically and hypothetically shape or should shape heritability estimates across social and cultural environments. In the Appendix, we presented a set of mathematical models that show how changes in cultural variance alone (i.e., even in the absence of culture–gene interactions, which would provide further modulation of heritability) could influence the heritability of traits in systematic and predictable ways. So yes, we fully agree that there is no general heritability nor is heritability an indication of the genetic basis of a trait.

Indeed, amplifying our argument, **Heine & Dar-Nimrod** list various ways in which the target article undermines some uses of the heritability concept, including expectations about its stability and generalizability. And furthermore, **Shuker & Dickins** mirror several of the arguments we make in the target article, though we sometimes go further. For example, they note that, "within a species, variation in heritabilities with age, for example, can give us hypotheses about (a) how selection acts at different ages, or (b) how developmental processes, and the genes and environments they influence and call upon, change over the lifetime." As we describe in the target article, it is not only hypotheses about selection and development, genes and environment that can be recovered from variation in heritabilities, but in the human species, also specific hypotheses about cultural transmission and cultural clustering.

Where we do disagree with **Shuker & Dickins** is where we make a more radical argument regarding how to think about the concept of heritability from a dual inheritance perspective. For example, on phenotypic transmission they state that "controlling for cultural exposure may help reveal patterns in variation in heritability that can lead to interesting hypotheses and further tests." This view is accurate but remains grounded in the conventional framework of behavioral genetics. In contrast, as our target article argues, culture is not something to be simply controlled for as a grouping variable. Cultural transmission perpetually reorganizes environmental distributions, and this dynamic character of the cultural environment is what often makes heritability non-generalizable. But this also means that if we can model cultural transmission, then we can track the cultural component of the heritability statistic, rendering it useful under certain conditions. An example from the target article is how cross-cultural variation in curricula influences the heritability of literacy in kindergarteners (sect. 2.2.1). It is currently rare for cultural transmission to be measured alongside genetic effects, but as the simultaneous estimation of both becomes regular practice, the utility of heritability may become more apparent. That is to say, the non-generalizability of heritability is not an Achilles heel but rather a resource that can be refined through careful analysis of cultural transmission, allowing us to understand the interplay between cultural dynamics and genetic effects.

Heritability appears intractable from a conventional behavioral genetics perspective because of the narrow attention on genetic variation, even though it is clear from the statistical formula that environmental variation is just as relevant. That is, heritability is often thought of as being about genetic variation but could equally be thought of as a measure of cultural variation. This point is echoed by **Turkheimer** who reemphasizes arguments he first made two decades ago: that high heritability indicates that genetic effects are easier to detect and quantify, not that genes are more influential (Turkheimer, 2000). Indeed, as **Larsen** notes, within education research, high heritability is often interpreted to indicate an optimal education environment.

Researcher priors affect whether they think the heritability statistic is useful. If one believes in either the general stability of environments, convergence over time, or commonality between cultural clusters, one might reify the heritability statistic; if one believes in either the general instability of environments or unpredictability and intractable dynamics within environments, one might dismiss the heritability statistic. The approach we advocate is to spend as much effort in understanding how the environment, particularly culture, varies and changes, as one does in measuring the additive effects of single-nucleotide polymorphisms (SNPs) correlated with outcomes or on clever designs to estimate heritability in one time and place (but often without stating or measuring the temporal, geographic, and cultural-group bounds of the finding).

The cultural evolutionary approach described in the target article offers tools and methods for capturing cultural distributions, and importantly, changes over time. This allows us to move beyond the relatively simple (e.g., one-dimensional monotonic) models of environmental variation that are common in gene–environment interaction analyses. Such an approach can help advance our understanding of human behavior across many domains. Two commentaries offer clear examples. **Larsen** considers the application of the framework to the educational context and **Amato** considers the application to psychopathology. As Amato argues, a cultural evolutionary framework may explain the global incidence of schizophrenia and could be used to guide interventions to instill resilience. We are intrigued by this proposal and look forward to further research in this area.

A few commentaries go further, calling for heritability as a concept to be retired. **Heine & Dar-Nimrod** amplify many arguments developed in the target article but argue that these ultimately discredit the heritability statistic as being in any way useful. They further argue that due to the essentializing tendencies of human psychology when it comes to questions about nature and nurture, heritability will inevitably be interpreted as being about genes by most people, and genes will ultimately be viewed as primary explanations for human phenotypes (see Heine [2017] for a book-length exposition of this argument). Given its lack of utility and ultimate misuse, they argue that it would be better to discard the concept of heritability altogether. **Downes & Kaplan** express a similar sentiment: that heritability as a concept should be discarded due to the purportedly intractable nature of environmental complexity. We address their critique in more detail in R3.3.

Turkheimer and **Bates** each describe how the standard view of heritability has already undergone considerable refinement among a new generation of scientists who work on the genetics of human behavior. Turkheimer summarizes the historical progression of nature–nurture debates within behavioral genetics, arguing that in the past behavioral geneticists assumed high heritability demonstrated the overarching influence of genetics on phenotypes, but today no longer debate nature versus nurture. There is no dichotomy; nature and nurture are interwoven. We fully agree. The goal of our target article was not to litigate between nature and nurture (which we agree is nonsensical beyond highly specific time, geographic, population, and cultural-group bounds). Instead, it was to offer a framework for understanding this interweaving between culture and genes.

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Bates reviews the overlap between the framework and the forefront of behavioral genetics, with several provocative clarifying questions. These questions are instructive, so we address them in more detail in R3.1

Overall, we agree with these commentaries – that our target article undermines some interpretations of heritability – but calls for the concept to be retired seem premature. Behind many of these calls is an intuition or assumption that environmental distributions and their dynamics cannot be measured or theorized. We argue this is incorrect. Even models and measurement aside, developing an intuition for how environmental distributions can, for example, become compressed by cultural diffusion or broadened by cultural innovation in specific terms, can reduce the reflexive association between heritability and genetic causation and reframe the questions we ask in understanding the role of culture and genes in creating behavior. Such shifts in intuition have occurred in other domains. For example, learning about visual processing as a reconstructive rather than camera-like process shifts our intuitions about seeing. Similarly, learning about supply and demand, market frictions, or comparative advantage shifts our intuitions about markets. Here, learning more about cultural evolution should shift our intuitions about the role of genes. Thus, what is required is a more mature understanding of human environments and how they are shaped by the forces of cultural transmission. Our target article is an initial attempt to shift these intuitions and move beyond disputes about whether or not environments are stable enough to support the validity of heritability estimates and toward an investigation of how environmental stability and instability manifest.

Fogarty & Creanza amplify our arguments but conclude that calculating heritability for behavior adds little to our understanding of human evolution and behavior. However, the bulk of their commentary focuses on how one might model the effect of culture. We discuss this in more detail in R3.3.

Fuentes & Bird too question the utility of heritability but make a further related argument about the ostensible misuse of terminology, including in our target article. For example, they critique the use of phrases such as "phenotypic variance *explained by* the environment [or genes]" or "phenotypic effects *due to* the environment [or genes]" (emphases ours) because they seem to imply a causal relationship. We are sympathetic to this argument but are personally not convinced that these phrases imply causality (at least among researchers). "Explained variance" and "explained sum of squares" are commonplace in statistics, without any causal implication. As for "due to," we do agree that this phrase implies causality, but we also use this phrase intentionally only when explicitly proposing a causal explanation and never in the context of a statement about correlated variance per se.

Fuentes & Bird also critique our use of the terms "masking" and "unmasking" whereby genetic effects become amplified or attenuated by culturally transmitted traits. Citing Mathieson's (2021) excellent review of the omnigenic model for the effect of genes – which, for the record, we suspect is ultimately correct – they suggest that usage of these terms "implies that there is a 'true' genetic architecture to the trait." However, in our view, an omnigenic model and Mathieson's (2021) explanation for cross-population differences are isomorphic to many of the arguments and framework developed in our target article. Here, masking and unmasking are why some SNPs may be identified in one population and others in another, most obvious in the sickle cell trait and malaria example. We also disagree that the terms "masking" and "unmasking" require fixed or true genetic architecture. To this point, **Lupyan**; **Kolodny, Feldman, Lotem, & Ram** (**Kolodny et al.**); and **Waring, Wood, & Xue** (**Waring et al.**) offer elaborations on our argument for how cultural masking/unmasking occur, without implying or presupposing a fixed or true genetic architecture.

R2.2. Culture

Burt and **Syed & Nguyen** point out our lack of a formal definition of culture or cultural traits, despite culture being the central theme of the target article. Burt offers a characterization of culture by enumerating a diverse set of elements such as beliefs, values, skills, habits, and styles. In a similar manner, Syed & Nguyen map cultural traits onto the notion of "cultural syndromes" (Triandis, 1996) which consist of beliefs, attitudes, and norms. These proposals represent an itemizing or enumerative approach to the conceptualization of culture, which has a long but contentious history (Bennett, 2015). It is not hard to see how disagreement could arise around such *extensional* definitions – any list of items is necessarily contestable. Note how the notion of "syndrome" is itself a canonical example of an enumerative approach, whose weaknesses and pre-theoretical status have been pointed out repeatedly within psychopathology (Fried, 2021; Lilienfeld & Treadway, 2016). **Downes & Kaplan** may also be seeing the problem through this same lens when they express that they "don't see a good way to separate environments into 'culture' and 'non-culture'."

Our use of the term "culture" follows the standard definition within the field of cultural evolution, as described by two of its founders in various publications, for example: "Culture is information capable of affecting individuals' behavior which they acquire from other members of their species through teaching, imitation, and other forms of social transmission. By information we mean any kind of mental state, conscious or not, that is acquired or modified by social learning and affects behavior" (Richerson & Boyd, 2005). Here culture is conceptualized not as a collection of enumerable traits, but rather by its mode of acquisition and the effect of this acquisition on behavior. In their enumerative definitions, both **Burt** and **Syed & Nguyen** add the qualification that when something is cultural, it is "shared" (Burt) or "culturally shared" (Syed & Nguyen). However, a trait, behavior, or mental state could be shared among people due to shared genes, individual trial-and-error learning, or social transmission. In the cultural evolutionary framework, it is only the last of these that makes something cultural. This perspective also implies that many traits are only partly cultural, insofar as modes of transmission can be mixed. Culture is thus a graded rather than categorical attribute: a point that cannot be captured by an extensional definition.

Syed & Nguyen's criticism of the cultural fixation index (CF_{ST}) and WEIRD (western, educated, industrialized, rich, and democratic) acronym is similarly motivated. As Muthukrishna et al. (2020) describe, CF_{ST} is a theoretically-defensible measure of cultural distance that describes between-group differentiation caused by cultural selection, migration, and social-learning mechanisms deviating from random social influence in a well-mixed population (just as the fixation index [F_{ST}] describes deviation from random mating over a well-mixed population). They offer an aggregate measure but advocate subsetting questions where there is theoretical justification for doing so. CF_{ST} is robust to the choice of cultural traits as long as these are sufficiently broad, because cultural traits cluster within cultural groups through social learning and institutions (the authors conduct

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several robustness tests to confirm this, showing that even 50% or more randomly removed data or questions result in the same CF_{ST}). Therefore, through selective subsetting on the part of the researcher, CF_{ST} is able to accommodate enumerative definitions of culture. However, the measure works just as well without committing to any particular definition. This is because it aggregates a broad array of attitudes, values, ideas, and beliefs, all of which we hypothesize are at least partially acquired or shaped by cultural transmission. Because human psychology and behavior are extensively influenced by cultural transmission in this manner, even an unsystematic aggregation of responses (as Syed & Nguyen put it) is able to capture systematic variation that arises from cultural clustering and segregated transmission. With respect to the WEIRD backronym: its components were never meant to be taken comprehensively or even literally but were instead a consciousness-raising device (Apicella, Norenzayan, & Henrich, 2020). To treat its constituent parts as an enumeration for the purposes of measurement would be like measuring the Big Bang by how much "big" and how much "bang." Indeed, as suggested in Muthukrishna et al. (2020), CF_{ST} can be used to develop a more nuanced proxy for a WEIRD scale.

R3. On the relationship between culture and genes

With conceptual concerns and misconceptions out of the way, we turn to the commentaries that focused on different aspects of the interplay between culture and genes. We begin with commentaries that focus on the role of genes, then those that focus on culture–gene interactions. Finally, we focus on commentaries that assume that culture can only impact genetic effects through culture–gene interactions.

R3.1. Genes

Here we address commentaries that focus on the role of genes, in particular the commentary by **Bates**.

R3.1.1. Collective cleverness is more important than genes for genius

Bates poses three questions and an additional point that are useful for both vetting our proposed framework and revealing where our target article diverges from cutting-edge behavioral genetics. The first of these is: "for how long could a population thrive if furnished with all of today's inventions and institutions, but shorn of ability-associated genetic polymorphisms?" This question is an interesting inversion of the "lost European explorer experiment," a didactic scenario often invoked by Boyd and Henrich in the cultural evolutionary literature (Boyd, Richerson, & Henrich, 2011; Henrich, 2016). The scenario describes historical cases in which teams of well-equipped European explorers are forced to sustain themselves in an unfamiliar ecological environment. From Burke and Wills in the Australian outback to Franklin in the Arctic, these lost European explorers typically fail despite having every advantage except for the cultural knowledge possessed by the local population (e.g., Henrich, 2016). These anecdotes are used as illustrations of the broader literature on how human ecological adaptation is to a large extent dependent on cumulative culture with genetic adaptation playing an unexpectedly small role. To put it another way, when most animals encounter a new environment, they are forced to genetically adapt – powerful muscles to outrun local predators, fur and fat to keep from freezing, proteins to make plants less poisonous. Our species has some local genetic adaptations (for a review, see Fan, Hansen, Lo, & Tishkoff, 2016; on adaptation to UV radiation, see Jablonski & Chaplin, 2017; on malaria, see Kwiatkowski, 2005; on altitude, see Yi et al., 2010), but has largely *culturally* adapted to live in almost every ecosystem on Earth (Barsbai, Lukas, & Pondorfer, 2021; Henrich, 2016).

Bates's counterfactual genetic-mirror of the lost European explorers is a provocative thought experiment. But specifically, what is meant by ability-linked polymorphisms is important. Obviously, to some degree the social and institutional infrastructure that supports modern industrialized societies is dependent upon the genes that make us human, no doubt many of which coevolved with culture. Attempts to acculturate other great apes have failed; we are unable to bequeath our civilization to any other primate. The commentator probably has in mind the polymorphisms associated with high intelligence, educational attainment, and so on, within the variation present in genome-wide association study (GWAS): What would happen if these specific polymorphisms disappeared? It is of course difficult to predict what the consequence of such a sudden dramatic shift in the genetic makeup of a population might be, but if culture and institutions are retained, a collapse and halting of future progress is not a foregone conclusion.

As background, the cultural brain hypothesis (Muthukrishna, Doebeli, Chudek, & Henrich, 2018) suggests there is a selection pressure toward larger brains that can store, manage, organize, and use more information to keep up with a growing corpus of cumulative culture. Even today, larger heads are linked to a greater likelihood of an emergency birth intervention – an emergency cesarean or an emergency instrumental birth (Lipschuetz et al., 2015) – consistent with both ongoing selection pressures and sufficient variation within the population. Thus, we are in no way denying that there are genetic differences between people. The question is to what degree is, for example, innovation dependent on these large-brained individuals? Here the model is also informative: The switch toward greater reliance on socially transmitted information – culture – can lead to a *decline* in brain size (here a proxy for ability-associated polymorphisms) with innovation continuing to increase. Why? Social transmission offers a more efficient way to arrive at the same adaptive outcome than learning by oneself. Humans are like a child in class who cheats on an exam instead of studying by themselves. But who are the clever students that they're relying on? Not geniuses, but on the endowed cultural package of thousands of years of accumulated knowledge of past generations. A low-ability individual or even population can do quite well given modern technology and infrastructure. Empirically, there is some evidence of a decline in brain size over the last 10,000–20,000 years (Ruff, Trinkaus, & Holliday, 1997) in support of the model, although the explanations for this finding are debated. More recently, there is evidence for genetic selection against educational attainment coinciding with a Flynn effect rise in IQ test performance (Beauchamp, 2016). But what of innovation and future progress? Here, the field of cultural evolution might diverge considerably from Bates's assumption about where all that knowledge and progress comes from, and the role and reason for genius (individuals with cognitive performance several sigma higher than the mean).

As argued in depth in Muthukrishna and Henrich (2016), innovation and progress are not driven by heroic geniuses and then passed on to the masses any more than your thoughts hinge on a particular neuron. Rather, innovation is dependent upon our societies and social networks. Increasing innovation isn't driven by a sudden increase in genetic geniuses, but by features such as

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population size, interconnectedness, the ability to transmit information, and tolerance for diversity (but also see the paradox of diversity; Schimmelpfennig, Razek, Schnell, & Muthukrishna, 2022). That is, innovation is a population-level process, creating more geniuses culturally rather than genetically by making each of us more intelligent through cumulative cultural evolution. None of this is to deny that people differ in their cognitive abilities, including because of genes, but only that geniuses aren't created by genetic differences alone – genes are not sufficient and may not be necessary either. A question sometimes posed is: Where have all the geniuses gone? The answer is that thanks to the spread of education, opportunity, and increasing cultural complexity, there are too many today for any to stand out. But there are still many lost Einsteins not because of inequality of ability, but because of inequality of opportunity (Bell, Chetty, Jaravel, Petkova, & Van Reenen, 2019).

R3.1.2. The apparent immutability of cognitive phenotypes

We reemphasize that we are not arguing that genes are unimportant, only that the scope and speed of cognitive change is dominated by cultural change, which has historically had a far greater impact on human behavior and, we argue, still has far greater scope and speed for future behavior. This naturally leads to **Bates**'s second question which refers to what he describes as "the intransigence of phenotypes." Beyond relatively simple examples like vitamins masking genetic effects, Bates asks whether our theory works for more complex behavioral phenotypes like mental illness and education, which seem unyielding to interventions.

Many mental illnesses can have multiple alternative explanations: "genes that break" (sect. 3.3.1), environmental factors, stochastic developmental variation (SDV), and deviation from healthy variation. **Abdellaoui** and **Zeng & Henrich** offer examples of polygenic scores associated with autism, bipolar disorder, and schizophrenia that are linked to positive outcomes, while **Amato** offers a sketch of how the proposed framework can help elucidate the genetic basis of psychiatric illnesses like schizophrenia. Although the cross-cultural variation in the outcomes associated with mental illness based on the local context (e.g., Larøi et al., 2014; Luhrmann, Padmavati, Tharoor, & Osei, 2015) hint at the possibility of interventions, we are not expecting yet-to-be-discovered interventions that will resolve these illnesses.

In contrast, the often cited failure of educational interventions is more interesting and seems to contradict the overall effect of education on intelligence cited by **Bates** (Ritchie & Tucker-Drob, 2018). We would argue that this apparent contradiction exists primarily due to the ubiquity of education and the marginal ability to make large educational changes.

The peculiar Western-style formal educational institution we call "school" has spread to most corners of the globe, at least to some degree. This has been a boon for human development, but a challenge for the scientific study of exactly how education has rewired our brains, and consequently, our societies. Two centuries ago, only 12% of the world could read and write. Today, only 14% are unable to read and write. And that's thanks to school. But with everyone, everywhere, exposed to school, we no longer know what people without any schooling look like. The variation in schooling we see is an extreme and clear example of the narrowing illustrated in Figure R1.

Schooling delivers not only what we learn, but also how we learn – teaching delayed gratification, sitting and studying for exams in a distant future; self-control in focusing on a single task for long periods; abstract, logical reasoning necessary for a variety of modern tasks; and mental models of cultural technologies like numbers and mathematics. Evidence suggests that when children develop reading and math skills, they learn to derive meaning from symbols, such as letters and numbers (Blair, Gamson, Thorne, & Baker, 2005). By "learning to learn" more effectively, via the written word, diagrams, or graphics, children may improve abstract problem-solving skills (Adams, 1994; Reis & Castro-Caldas, 1997). School-derived skills may also enhance knowledge-seeking desires and behavior, in addition to domain-specific competence, and by affecting skills such as analytic perception, epistemic norms (what constitutes a good argument), and by facilitating a transition from concrete to formal operational thinking (Blair et al., 2005; Cain & Oakhill, 2009; Oakhill & Cain, 2012).

But causally quantifying the effects of schooling is challenging because in almost all contexts, a lack or poorer quality of education is thoroughly confounded with poverty, pollution, disease, war, or other insults. But even in these contexts, interventions can bring children closer to parity with those with fewer insults. For example, Chetty et al. (2011) show that when low-socioeconomic status (SES) children are randomly assigned to higher quality classrooms from K-3, they are more likely to attend college, save for retirement, and eventually live in better neighborhoods. Depending on the degree of deprivation, we should be surprised that educational interventions have even the small and temporary results that they do. Such interventions are often on people who have experienced adverse prenatal and early childhood environments and who aside from the intervention lack the invisible cultural pillars that support education, such as educated parents, educated adults in the community, and a value placed on learning. Indeed, when interventions are earlier, such as prebirth, the effects are larger (e.g., micronutrients to malnourished mothers; Prado et al., 2017). Thus, Ritchie and Tucker-Drob (2018) are likely underestimating the effect of education on intelligence and skepticism about potential interventions and educational innovations are likely a product of the challenges of radical reform restricted by path dependence. As an example, although Programme for International Student Assessment (PISA) scores indicate that some national curricula perform better in subjects such as math (Singapore and Shanghai are prominent examples), importing these curricula to even developed countries like the United Kingdom and United States are stymied by the challenges of teacher retraining, expected examinations, student prior preparation, and so on.

High heritability estimates, as high as 80% for cognitive ability among high-SES groups (Hanscombe et al., 2012); or even effectively 100% for core executive function (Engelhardt, Briley, Mann, Harden, & Tucker-Drob, 2015; Friedman et al., 2008), have led to conclusions, mistaken in our opinion, that these aspects of our psychology are highly genetically determined. This work fails to account for the tremendous variability in culture and education across the globe and over time and the potential change the future holds. Where interventions take place, they fail to account for the broader cultural infrastructure, such as the presence of successful, educated adults in families and the broader community. More causal research, perhaps natural experiments in the few locations that have yet to receive education, may help resolve these debates and shed light on the true plasticity of intelligence.

In a related third question, **Bates** asks us what fraction of DNA variants associated with traits such as cognition or reading skill we believe will reverse their effects under conditions that raise mean educational outcomes, given that such reversals in genetic main effects have not yet been found. It would be presumptuous

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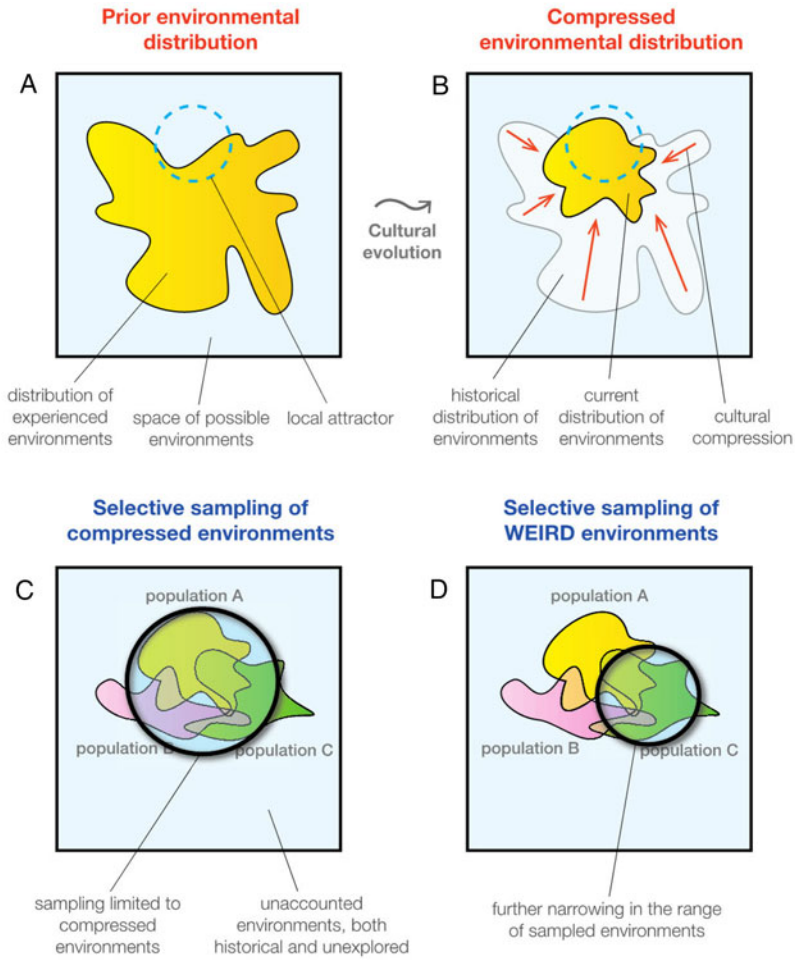

Figure R1 (Uchiyama et al.). Restricted sampling of environments due to historical trajectories of cultural evolution and selective (WEIRD) sampling. (a) The yellow region represents the distribution of experienced environments of a hypothetical society at a past time point. The unoccupied light blue area represents the unexplored regions of the space of possible, viable environments. The blue dashed circle represents a set of environmental states that are better adapted to ecological challenges and functions as a local attractor on an adaptive landscape. (b) The environmental distribution at a later time point. Through cultural evolutionary dynamics such as conformist transmission and selective imitation, the society has converged around the local attractor. (c) Even if researchers were able to obtain samples from all extant populations, their observations would be limited to a particular subspace of possible environments that is contingent upon cultural history. Because genetic effects can only be evaluated with respect to particular environments, genes may have vastly different effect sizes or functions outside of this observable range. (d) In practice, researchers conduct the majority of their analyses within a handful of societies that represent a small fraction of global genetic and cultural variation. This limitation further narrows down the range of observed environments and thus impedes generalizability of genetic effects.

of us to estimate the proportion of DNA variants that could plausibly undergo such a reversal. However, we would predict that such variants do exist and that most of these reversals are currently hidden due to severely restricted sampling from the range of possible environments (Fig. R1) and the restricted time range – data for GWAS at best represent the last two decades. Moreover, we may expect to see shifts in what traits are valuable in the future, especially if it is true that genetic variants are additive in their effects (Crow, 2010; Hill, Goddard, & Visscher, 2008; Hivert et al., 2021). For example, once upon a time, traits associated with semantic memory were highly associated with educational outcomes and lifetime earnings – think knowledge-based careers such as law and medicine that required storage of information in memory. But increasingly, the ability to multitask or to seek out relevant information in a noisy informational environment may be more predictive of educational and professional success. As our economies and technologies change, the content of school curricula and the character of work also changes, shifting genetic effects associated with these domains. The same can be said for autistic traits: The social challenges that characterize this phenotype may have conferred grave disadvantages in traditional societies, but today, many individuals embodying such traits are able to thrive in novel behavioral niches like the tech industry. If we were able to sample across these gradients of social, economic, and technological variation, we might be able to obtain a better picture of how genetic effects change – at times possibly even reversing their sign.

R3.1.3. Culture and genes are interwoven in human cognition

In a final point, **Bates** appeals for a principled distinction between the learning machinery provided by genes – what he refers to as the "blank slate," invoking Locke – and the specific content that is learned by this system. In making this point, he references a study that finds a common latent factor of executive function that is 100% heritable in a sample of American children (Engelhardt et al., 2015) and another that finds a direct effect of education on domain-specific cognitive skills but not through general intelligence as a mediating factor (Ritchie, Bates, & Deary, 2015). We addressed this point in part above, but would add that it is an epistemological fallacy to identify these findings with insights about the structure of cognition and learning. The metaphor of a blank slate or *tabula rasa* does indeed carve up the sphere of mental activity into substrate and information, but this is a poor model for understanding the computational mechanisms that support learning. We are not blank slates, but culture is as biological as genes. In nervous systems, unlike digital computers, there is no clear distinction between activity and structure. For example, rather than encode information in designated memory stores, the mammalian brain sculpts mnemonic representations out of the same circuits that are used for the analysis of sensory input (Hasson, Chen, & Honey, 2015; Lee, Nader, & Schiller, 2017; Postle, 2006). Moreover, in humans, the input itself adapts to the processing demands of the brain through cumulative cultural evolution (Christiansen & Chater, 2008; Dehaene & Cohen, 2007; Uchiyama & Muthukrishna, in press), rendering

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the input or content non-independent from the properties of the computational machinery. Psychometric findings may appear to suggest the idea of a separation between the mechanics and content of cognition, but there is little evidence for such a structural distinction in the contemporary cognitive and neural sciences. It would be a mistake to interpret these psychometric findings as an ontology of brain function.

From literacy and numerical ability to conceptual categories and social cognition, human cognitive function is thoroughly shaped by cumulative culture. We easily lose sight of this fact when comparisons between individuals are typically conducted within a single culture – indeed, a culture in which we all are endowed by the accumulated skills of generations past through education and in which almost all consume some similar cultural input. Cognitive machinery is thus installed through cultural transmission – the effect of culture is as biological as the effect of genes. And indeed, even if a gene is linked to some cognitive ability, it remains ambiguous whether the gene is associated with the ability itself (e.g., the ease with which the ability is acquired even in the absence of cultural input), whether the gene is associated through aspects of the process of cultural transmission of that ability, or with some mix of both. **Kitayama & Yu** offer the example of the dopamine D4 receptor gene *DRD4*, which appears to be associated with enculturation itself (Kitayama et al., 2014). The commentators speculate that this gene may have played a significant role in human evolution. However, evolutionary scenarios involving *DRD4* would need to look outside of the gene itself and conceptualize the coevolution of the gene and the cumulative culture that makes its effect meaningful. Such an analysis would also lend itself well to interactive processes that are hypothesized to drive phenotypic development – for example, how does *DRD4* modulate the processes of "reciprocal causation" (Bronfenbrenner & Ceci, 1994; Dickens & Flynn, 2001; Scarr, 1992) that we had discussed in the target article? Kitayama & Yu focus on static relationships between variation in the *DRD4* genotype and local cultural traits such as independence/interdependence, but longitudinal interactions between gene and environment may prove informative (see also **Kievit, Logan, & Hart** [**Kievit et al.**]).

We nonetheless agree with **Kitayama & Yu** that the implications of culture-gene coevolution for behavioral genetics will only be fully fleshed out through more comprehensive analyses that encompass genetic evolution – a point also expanded on by **Waring et al.**. In our target article, we made the prediction that all else being equal, societies with greater cultural homogeneity will exhibit higher heritability of culturally transmissible traits, due to there being less variance in the cultural environment to explain phenotypic variation. If the 7/2-R allele of *DRD4* is associated with higher fidelity of cultural transmission, as Kitayama & Yu argue, then we would predict societies with higher frequencies of this allele to exhibit higher heritability of the class of traits whose transmission fidelity is supposed to be increased by this genotype (e.g., independence/interdependence).

This deep intertwining of culture and genes also provides a window onto the issue raised by **Racine**, namely the implications of our framework for evolutionary psychology – in the programmatic sense of the term associated with the work of researchers such as Buss (1995) and Tooby and Cosmides (1992). We suggest that the most promising way forward is a *coevolutionary* psychology (Henrich & Muthukrishna, 2021) that places sufficient weighting on both genetic and cultural transmission, and is able to study evolutionary trajectories that arise through the interaction of the two. If culture can mask and unmask genetic effects, as we suggest in the target article, there is little meaning to focusing on just one at the expense of the other, and this dynamic should be taken into account when assessing cognitive abilities in humans, including general intelligence as discussed by **Lupyan**. Like the argument presented by **Bates**, evolutionary psychology has often made an implicit theoretical distinction between the genetically specified neural hardware and the culturally acquired informational software, but this separation breaks down at a functional level in the case of human cumulative culture. A coevolutionary psychology would place theoretical priority on neither genetic nor cultural evolution, but rather on their interaction and coupled dynamics. This would likely require refinement in methods for understanding not only when apparent genetic differences are better explained by cultural transmission, but also perhaps when apparent differences due to cultural transmission are better explained by differential gene expression due to ecological differences – an analytical balance advocated for by **Fischer**.

R3.2. Culture–gene interaction

Waring et al. hypothesize that the balance between cultural and genetic transmission may itself be shifting over time, with culture playing a progressively more dominant role relative to genes in influencing the distribution of human phenotypes. They call this long-term trend "cultural pre-emption" (Waring & Wood, 2021). Waring et al. argue that phenotypic variance explained by culture (V_c in the Appendix model) has been increasing over human history due to the continual emergence of complex cultural-group-level adaptations across domains such as food production, medical treatment, and defense, and predict a continued decrease in heritability of relevant traits into the foreseeable future. **Abdellaoui** pushes this scenario further by raising the possibility of forthcoming cultural technologies such as polygenic embryo selection (Turley et al., 2021) playing an outsized role in the decrease of phenotypic variance explained by genes (V_G), a societal shift that may contribute to the trend of declining heritability predicted by Waring et al.

Waring et al. and **Abdellaoui**'s mechanisms seem plausible to us as specific instantiations of forces that change cultural and genetic variance. From the cultural dynamics angle, whether the long-term trend skews toward an increase or decrease in heritability depends upon which of the following dominates: the processes of increasing cultural complexity and genetic masking discussed by Waring et al. and by ourselves in the target article, or the process of increasing cultural connectivity and progressively far-reaching diffusion that we also discuss in the target article. The former is expected to decrease heritability, while the latter is expected to increase it.

Focusing on the genetic rather than environmental component of phenotypic variation, **Zeng & Henrich** provide an overview of how modern culture and gradual changes in social organization within developed countries have also been shaping genetic variation through assortative mating. They argue that assortative mating has been increasing and thus increasing phenotypically consequential genetic variation over recent history, a dynamic that is expected to increase heritability if the environmental component of phenotypic variance were held constant. This contrasts with the processes described by **Waring et al.** and **Abdellaoui**, which predict reduced heritability over time. Of course, both heritability-increasing and heritability-decreasing processes

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could be operating simultaneously. Assortative mating alone could drive an increase in both the genetic and cultural components of unstandardized phenotypic variance, if the increasing assortativity in the sexual domain described by the Zeng & Henrich is accompanied by increasing assortativity in the cultural domain through cultural clustering (see target article sect. 3.2). The long-term trend of genetic effects will depend upon the balance between these various processes. Assortative mating has implications for not only heritability but also analytical methods like Mendelian randomization, as **Campbell, Munafò, Sallis, Pearson, & Smith** (**Campbell et al.**) point out. Therefore, the interpretation of Mendelian randomization and related methods over longer time horizons may depend upon the dynamics of assortative mating described by Zeng & Henrich.

R3.3. Cultural evolutionary dynamics outside of culture–gene interaction

Fogarty & Creanza question how useful calculating heritability is for culturally complex traits like many behavioral traits with several critiques of the simple model presented in the paper. These are important considerations. First, they point out that although we exclude gene–environment interactions for simplicity, these interactions are critical in assessing the influence of culture inheritance on genetic effects. We completely agree and point readers to the cited foundational work of Lewontin, Feldman, and others that discuss this at length. Our goal here was to demonstrate in the simplest possible way that even without these interactions (which are familiar to behavioral geneticists) heritability is nonetheless thoroughly confounded by culture. Indeed, this confounding occurs at multiple levels as **Kolodny et al.** describe (see our reply in R4.1).

Next, **Fogarty & Creanza** address technical details in building models that assess the effect of culture on heritability for the purposes of understanding the evolution of traits. Regarding the assumption of a Gaussian phenotypic distribution, we assume the commentators have in mind future models building on this framework that attempt to understand the evolution of a trait. In the model presented in our paper, we don't model the phenotypic distribution, only its variance. Thus, a uniform distribution, with a lower bound of 0 is appropriate for the points being made. This simple model is agnostic to the underlying phenotypic distributions but modeling the evolution of the trait would remove this abstraction. The commentators also point out that genetic evolutionary models assume constant phenotypic variance, which is also observed empirically (e.g., Arnold, Bürger, Hohenlohe, Ajie, & Jones, 2008). In contrast, we do assume fluctuation in cultural phenotypes and even convergence contra the genetic evolutionary models, but there are reasons to believe this assumption can be violated. Cultural evolution does not require discrete replicators or memes, which may maintain variance, and thus the assumptions made for genetic evolutionary models cannot be assumed to also apply to cultural evolutionary models. Cumulative, adaptive evolution has been theoretically and empirically studied for continuous traits within cultural evolution (for a clear discussion, see Henrich, Boyd, & Richerson [2008]). Cultural evolution allows for blending, which means variances can fluctuate. But of course, all evolutionary models require some variance. In cultural evolution, the Jenkin (1867) swamping critique is overcome through forces such as mistakes during cultural transmission, serendipity, and recombination (for further discussion, see Muthukrishna and Henrich, 2016). And empirically, fluctuating cultural variances is consistent with changes over time (Jackson, Gelfand, De, & Fox, 2019) in tightness–looseness and the effect of ecological and material threats of different kinds (Jackson, Gelfand, & Ember, 2020).

Finally, the suggestion for including culture in the numerator is similar to the point about including interactions. This too is a better reflection of reality for all the reasons mentioned in the target article and by **Fogarty & Creanza** as well as by **Zeng & Henrich**. As Fogarty & Creanza argue, and we agree, this further complicates modeling and measuring the role of genes in explaining human behavior. Many of these points are only obvious in light of cultural evolution and we welcome future integrative work in this area.

Downes & Kaplan also comment on the importance of incorporating gene–environment interactions, which we agree with as mentioned above. Where we diverge from their interpretation is on the reason why environmental enrichment (often indexed by SES in humans) and the heritability of cognitive ability commonly exhibit a positive correlation in humans (Bates, Lewis, & Weiss, 2013; Rowe, Jacobson, & Van den Oord, 1999; Scarr-Salapatek, 1971; Tucker-Drob & Bates, 2016; Tucker-Drob, Briley, & Harden, 2013) but are negatively correlated in the mouse study of Sauce et al. (2018) – discussed in section 4.1.2 of the target article. These commentators draw from the study of Cooper and Zubek (1958) to argue that this apparent cross-species reversal can be attributed to a nonlinear reaction norm, where the between-allele variability in genetic effects (for a given trait, e.g., maze-running ability) increases with moderate degrees of environmental enrichment, but then decreases again (with a higher average effect) with further enrichment. Downes & Kaplan appear to be proposing that the Scarr-Rowe Effect is only an intermediate outcome on the spectrum of environmental enrichment, and that if it were possible to induce even greater enrichment in already high-SES groups (or perhaps to sample from only the top sliver of SES), we should see a reduction in the variability of genetic effects as in their Figure R1b. This would result in a reduction in the heritability of traits that are affected by enrichment, just as we see in the mice of Sauce et al. (2018). The described scenario is possible and an open empirical question, but without stronger theoretical justification, we see little reason to expect it to be true.

Downes & Kaplan interpret the target article exclusively through the lens of gene–environment interaction. For instance, they write,

> we can see no way to predict how the heritability of a trait will respond to changes in the environment, independently of knowing an implausible amount about the development of the trait in question… Depending on how development responds to environmental change, the same kind of environmental change might cause the heritability of a trait to increase, decrease, or to stay the same

This narrow focus on gene–environment interaction as the primary cause of the manifestation of phenotypes is useful for explanatory purposes, because of how widespread this perspective is. In contrast, we reemphasize that the target article focused primarily on cultural dynamics that shape environmental variation through cultural connectivity, and how this is expected to influence heritability. In particular, section 2.2.3 described how processes like cultural diffusion and innovation could decrease, increase, or leave unchanged the heritability of a phenotype depending on whether relevant cultural traits mask, unmask, or are neutral with respect to relevant genes. **Downes & Kaplan**

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are thus entirely correct in their recognition of multivalent outcomes. However, the cultural dynamics that we describe are able to shape heritability separately from such gene–environment or gene–culture interactions, by acting upon cultural transmission networks that regulate the distribution of environmental exposure. All else being equal, societies with rapid diffusion will tend to have higher heritability due to greater environmental homogeneity, and societies with rapid innovation will tend to have lower heritability due to reduced environmental homogeneity (or greater heterogeneity). Unless cultural dynamics are systematically confounded with the directionality of culture–gene interactions (masking/unmasking), the approach described in the target article will be able to statistically predict trends and patterns in the heritability of traits. This framework does not require an implausible amount of knowledge about the development of a trait in order to predict its change in heritability over time, or to predict its relative heritability compared to societies with different cultural dynamics. However, the accuracy of these predictions can only be verified by future empirical work.

R4. Extensions to the framework

Several commentaries raised issues that help expand the scope of the framework laid out in the target article. We first discuss extensions to the notion of cultural clusters (R4.1), then about development (R4.2) and finally power (R4.3).

R4.1. Cultural clusters

A key construct that we discussed in our target article was the notion of *cultural clusters*. When cultural transmission within a society is fractured into subgroups that are more connected within themselves than they are to other subgroups, then the society has high cultural clustering. Obvious sources of cultural clusters are regions within a country or linguistic groups within a population, but our discussion touched upon how dimensions of social organization such as socioeconomic status and social class can also be seen through the lens of differential cultural transmission, rather than being limited to their standard conceptualization in the social sciences. Several commentators homed in on this construct and proposed various ways to expand its range of conceptual utility.

Peréz Velilla, Moser, & Smaldino (**Peréz Velilla et al.**) argue that the presence of hidden clusters has not received sufficient attention in the social sciences in general. They describe epistemological problems that arise from the conflation of conventional group identities with the actual structure of trait distributions and cultural transmission. Detailed ethnographic studies like Moya and Boyd (2015) and Tucker et al. (2021) support their argument, demonstrating that group boundaries are much more porous, contingent, and multilayered than a narrow focus on ethnicity would suggest. This argument is also made by **Wiessner**, who further highlights the importance of consulting the ethnographic record to find internal cultural clustering and other within-group forces.

Approaching the same problem from a different angle, **Götz, Ebert, & Rentfrow** (**Götz et al.**) describe research on the hidden geographic clustering of psychological traits, which can be made visible with big data approaches. Although country-level comparisons have long been the mainstay of cross-cultural psychological research (e.g., Hofstede, 2001), the commentators highlight the value of studying psychological variation among subnational units such as regions or cities. They claim that cities, for example, are more prone to rapid cultural change than are countries. We look forward to the deeper confluence of cultural evolution, geography, and urban science in the future.

Boothroyd & Cross invoke cultural clustering as an explanatory factor for gender differences. These commentators discuss how cultural traits are transmitted within genders and how this dynamic can produce differential effects depending upon ecological and economic context. For example, modern WEIRD societies tend to mask the effect of sexually dimorphic anatomical and physiological traits in people's choices of what kind of social and behavioral niches to occupy (e.g., most jobs do not rely on physical strength), while also offering a greater diversity of such niches (Smaldino, Lukaszewski, von Rueden, & Gurven, 2019) compared to, for example, plow-based agricultural societies (Alesina, Giuliano, & Nunn, 2013). The commentators argue that under these WEIRD societal parameters, choices about which niches to occupy (e.g., choice of career or lifestyle) becomes less constrained, and thus a more arbitrary and complex decision. This choice complexity in turn engenders a reliance on within-gender cultural transmission, and gives rise to the well-known paradoxical finding of larger sex differences in psychology and behavior among more gender-equal societies (e.g., Falk & Hermle, 2018). A common interpretation of this paradox is that sexually dimorphic traits selected by genetic evolution are more strongly expressed in developed, gender-equal societies (Lippa, 2010; Schmitt et al., 2017) – an explanatory approach that has advantages over classical theories that emphasize the socialization of gender roles. The hypothesis explored in the commentary of Boothroyd & Cross offers an alternative explanation for how gender phenotypes may be influenced by cultural dynamics that systematically respond to societal organization.

Although we did not consider the cultural clustering of gender in the target article, we did mention various levels of organization at which clustering of cultural transmission may occur, including socioeconomic status, religious and political affiliation, and exposure to mass media or online communities. **Kolodny et al.** argue that hidden clustering of cultural traits can also occur at the level of families, family lineages, or individuals, in a manner that is responsive to genetic traits. According to these commentators, our discussions of the Causal Locus Problem (sect. 3.3) and the Cultural Simpson's Paradox (sect. 3.4) in the target article are therefore pertinent to the interpretation of cultural effects at these more fine-grained levels of organization, a perspective that goes back to Cavalli-Sforza and Feldman (1973). This commentary mirrors some of the arguments made by **Fogarty & Creanza**, who call for a more substantial incorporation of genotype–environment interactions into the target article's theory and model.

R4.2. Development

Although the target article briefly discussed the interaction of cultural transmission with development, the full depths of this rich topic were left unexplored. Fortunately, several commentators extended the discussion into various aspects of human development. **Kievit et al.** rightly point out that the developmental examples included in the target article revolve around cross-sectional methods, and that the conclusions we derive from these studies may be restricted by this methodological scope. Most of the progress in the psychological sciences has come from studying constructs primarily at the population level, using psychology's

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standard statistical armamentarium. This is largely true for the study of psychological development as well, but the commentators argue that alternative methods for studying detailed longitudinal change *within* individuals are critical for understanding development in general, and for understanding constructs like heritability in particular.

One major strength of a cultural evolutionary approach to behavioral genetics is its capacity to represent environments with greater complexity and in a theory-driven manner, compared to more common approaches such as reaction norms or even "exposome" analyses (Niedzwiecki et al., 2019; Wild, 2012), essentially high-dimensional reaction norms. In particular, a cultural evolutionary approach is able to model the population distributions of environmental exposures as well as their dynamics – either across a single lifespan or across cross-generational timespans – by viewing them through the networks of cultural transmission that organize environmental exposure in humans. Because local network topologies (e.g., number of connections, centrality) vary between individuals within societies, and global topological properties (e.g., degree distribution, clustering/modularity) vary between societies, a cultural evolutionary analysis takes it as a starting point that environments are person-specific constructs that cannot be captured by any assumption of within-group homogeneity. We agree.

The person-specific or "intraindividual" (Molenaar & Campbell, 2009; Molenaar, Huizenga, & Nesselroade, 2003) approach advocated by **Kievit et al.** demonstrates the inadequacy of statistical methods such as standard factor analysis for many developmental phenomena in the face of within-person variation over time. Cultural evolution can complement this analytic strategy, by offering a richer framework for representing environments as dynamic and person-specific constructs. For example, Smaldino et al. (2019) propose a model that explains cross-cultural variation in the factor structure of personality – a Big Five in WEIRD societies, but fewer factors in small-scale societies (Gurven, von Rueden, Massenkoff, Kaplan, & Lero Vie, 2013; Lukaszewski, Gurven, von Rueden, & Schmitt, 2017) – as an outcome of variation in the diversity of social and ecological niches across societies. Although the model focuses on between-society rather than within-society variation, there is nothing in the model that limits its implications to differences between separate societies. Real societies clearly contain meaningful within-society variation in niche complexity, and Smaldino et al.'s (2019) model can therefore offer predictions for the person-specific environmental factors that causally contribute to both between- and within-society heterogeneity in the factor structure of personality and other traits. A confluence with cultural evolutionary understanding of environmental structure and its causal role in the dynamics that underlie intraindividual (developmental) heterogeneity may lead to a comprehensive framework for human development.

A complementary commentary by **Markon, Krueger, & South** (**Markon et al.**) uses the example of age-period-cohort (APC) models to stress the need for longitudinal behavioral genetics research, further highlighting limitations of cross-sections in making dynamic inferences. APC models are epidemiological models that are used to study how development (age), specific events in particular years (period), for example, wars or pandemics, and shared experiences of those developing within the same cohort, results in changes in outcome, for example, tuberculosis mortality (Fosse & Winship, 2019). These models have been applied to cultural traits such as alcohol consumption (Livingston et al., 2016) and religion (Schwadel, 2011). The commentators suggest that APC models could be extended to include genetic and environmental effects. Cultural transmission poses an additional level of complexity to be incorporated into APC models due to the many mechanisms of social learning.

Adding additional complexity, **Mitchell** calls for SDV, to be added to our framework. SDV is random noise that causes different phenotypes to be produced from the same genotype, in the same environment (Vogt, 2015). Along with genetic and environmental variation, SDV is an additional source of variation on development outcomes. The current exclusion of SDV in behavioral genetic research has been criticized, and it has been proposed that the nonshared environment be split into an external and internal nonshared environment, where the latter is the portion explained by SDV (Tikhodeyev & Shcherbakova, 2019) – a possibly useful distinction, but at the expense of overstretching the meaning of "environment."

As **Mitchell** notes, potentially interesting scenarios come to the fore when we consider how SDV might be exposed or masked by the cultural environment. For example, handedness, and perhaps to some extent sexual orientation, are traits that are partly shaped by SDV (Mitchell, 2018). We know that different cultures allow these traits to be exposed in different ways, thus modifying their phenotypic consequences. Such examples suggest that culture can interact directly with SDV independently of its interaction with genotypes. These examples also suggest that there may be forms of latent phenotypic variation – either stochastic or genotypic – that are masked in our cultural milieu but would be revealed under different conditions. As **Lupyan** argues, greater sensitivity to the role of cultural contingency on gene expression is necessary for a deeper understanding of traits like intelligence. **Newson & Richerson** highlight how this cultural masking and unmasking guides the direction of whole societies, by shaping choices in domains such as career preferences and childbearing.

Ragsdale & Foley argue that our framework should be expanded to include epigenetics. Epigenetics and SDV are interlinked as SDV is moderated by epigenetic processes. Epigenetics maybe a useful mediator between genetics and culture, but we disagree with Ragsdale & Foley's example of the candidate gene, serotonin receptor gene (SERT or 5-HTT). Associations between SERT and depression have repeatedly failed to replicate (Border et al., 2019; Culverhouse et al., 2018). Better examples are the influence of cultural factors such as diet and smoking on epigenetic profiles (Jablonka, 2016). Differences in methylation between population subgroups reflect both genetic differences and differences in cultural practices (Galanter et al., 2017).

R4.3. Power

Burt and **Syed & Nguyen** argue for the need to include the role of "societal power structures" (Syed & Nguyen) and "social structures" (Burt) in our discussions about culture. These commentators use these terms in the sense of power hierarchies that impose asymmetrical constraints on cultural dynamics, advantaging some subset of the population at the expense of others in the process of cultural transmission. We agree that power structures play a substantial role in cultural transmission, for example, through privileged access to more adaptive cultural innovations and practices or asymmetric influence in social transmission. Indeed, such asymmetries can persist over generations. We support the inclusion of these factors but are unsure how they might be included – there is a need for greater theoretical expansion of these forces within

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cultural evolution. Part of the challenge is the lack of clear definitions of power.

Syed & Nguyen express that in the target article we "seem to suggest that these conditions just emerge as part of a natural process rather than being intentional acts by those in power" but provide no rationale for why they believe individual intentions to be the appropriate analytic unit for a scientific understanding of societal structure, how people acquire power, where the range of intentional actions come from, the motivating incentives and norms, the origins, and dynamics. For example, how people acquire wealth or political power has changed substantially over the centuries as has the behavior and "intentional acts" of wealthy and powerful people and this requires explanation. Also unexplained are why "intentional acts" are mutually exclusive with explanations that suggest underlying cultural changes that "emerge as part of a natural process," that is, a population- or systems-level perspective. It is difficult to imagine how an account based on individual intentions can explain, for example, how relatively egalitarian hunter-gatherer bands transition into feudal societies with stark power hierarchies. Similarly, it would be a mistake to assume that intentions are sufficient for understanding how power operates within our contemporary world. Cultural evolution offers conceptual and modeling tools for understanding how human group organizations emerge, and how these are sustained and amplified over time. These group dynamics in turn allow one to engage with the topic of power structures without the need to postulate individual actions as primary, ultimate explanations. While explanations of power based on individual intentions may be rhetorically effective, integration into our proposed framework would require an ultimate-level approach that generates hypotheses about causal structure and consequent testable predictions. For examples, see Henrich (2020), Henrich, Chudek, and Boyd (2015), Henrich and Muthukrishna (2021), Muthukrishna, Henrich, and Slingerland (2021), Norenzayan et al. (2016), and Schulz, Bahrami-Rad, Beauchamp, and Henrich (2019).

R5. Closing remarks

The commentaries that were submitted in response to the target article not only cover a wide range of disciplines, but also a wide range of topics from conceptual interpretation and philosophical argumentation to empirical examples and evolutionary scenarios. In some cases, the commentaries amplified our arguments, taking them further; in others they directly expanded the scope of our discussion; and in others still they pointed out valuable sources of misunderstandings or discrepancies with respect to our arguments that hopefully have enabled us to build bridges between divergent viewpoints. One point that stands out is the enormous variability in the extent to which the notion of a culturally evolved and evolving environment was intuitive for our commentators that reflected relevant disciplinary backgrounds. In responding to these commentaries, we hoped to facilitate a valuable conversation in our reply. The outcomes, conclusions, and remaining questions that have emerged further underscore the need for greater convergence between the fields of cultural evolution and behavioral genetics in the study of how we become us.

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