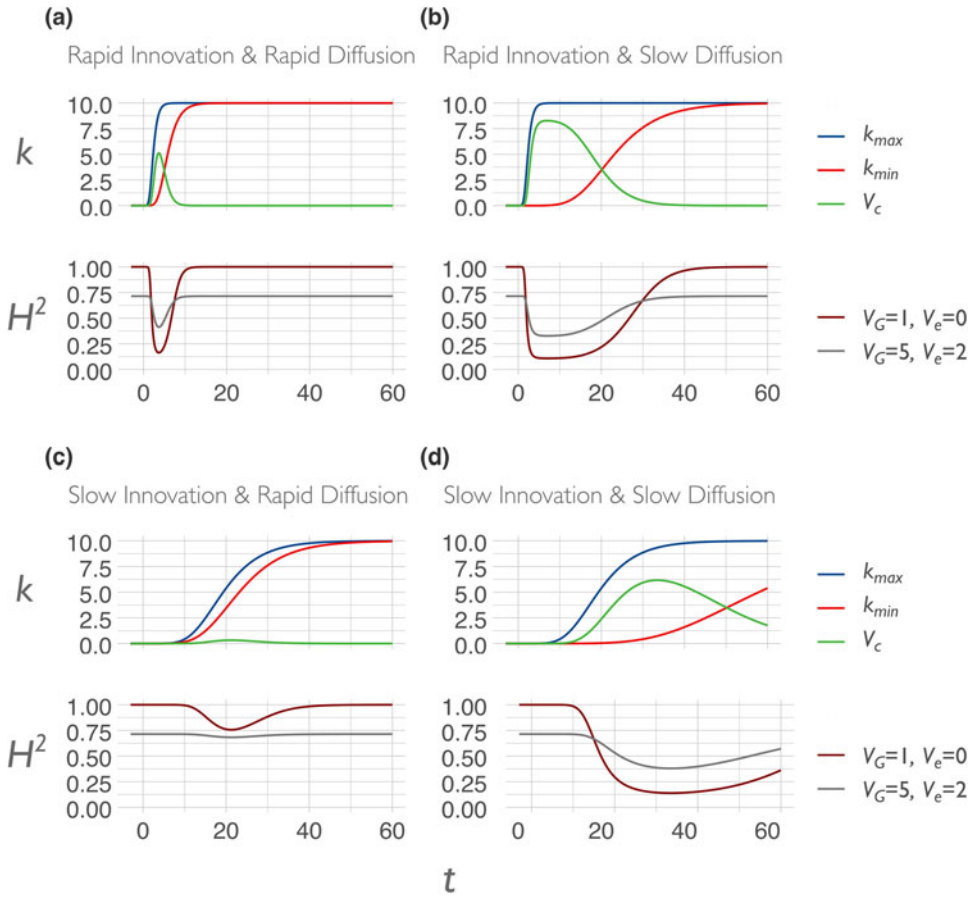

Figure 5. Output examples of the cultural dynamics model, for four prototypical societies. For each society, the upper panel displays the change over time in the lower (k_{min}) and upper bounds (k_{max}) of the society's cultural complexity as it pertains to some given phenotypic trait, together with 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):

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

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

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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

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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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References

Abdellaoui, A., & Verweij, K. J. (2021). Dissecting polygenic signals from genome-wide association studies on human behaviour. *Nature Human Behaviour*, *5*(6), 686–694.

Laland, K. N., Odling-Smee, J., & Myles, S. (2010). How culture shaped the human genome: Bringing genetics and the human sciences together. *Nature Reviews Genetics*, *11*(2), 137–148.

Mills, M., & Rahal, C. (2019). A scientometric review of genome-wide association studies. *Communications Biology*, *2*(1), 1–11.

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Musunuru, K., Chadwick, A. C., Mizoguchi, T., Garcia, S. P., DeNizio, J. E., Reiss, C. W., … Kathiresan, S. (2021). In vivo CRISPR base editing of PCSK9 durably lowers cholesterol in primates. *Nature*, *593*(7859), 429–434.
Turley, P., Meyer, M. N., Wang, N., Cesarini, D., Hammonds, E., Martin, A. R., … Visscher, P. M. (2021). Problems with using polygenic scores to select embryos. *New England Journal of Medicine*, *385*(1), 78–86.
Wrangham, R. (2009). *Catching fire: How cooking made us human*. Basic Books.

Cultural evolution: The third component of mental illness heritability

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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

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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References

Agishtein, P., & Brumbaugh, C. (2013). Cultural variation in adult attachment: The impact of ethnicity, collectivism, and country of origin. *Journal of Social, Evolutionary, and Cultural Psychology*, *7*(4), 384–405. doi: http://dx.doi.org/10.1037/h0099181.

Akdeniz, C., Tost, H., & Meyer-Lindenberg, A. (2014a). The neurobiology of social environmental risk for schizophrenia: An evolving research field. *Social Psychiatry and Psychiatric Epidemiology*, *49*(4), 507–517. doi: 10.1007/s00127-014-0858-4

Akdeniz, C., Tost, H., Streit, F., Haddad, L., Wust, S., Schafer, A., … Meyer-Lindenberg, A. (2014b). Neuroimaging evidence for a role of neural social stress processing in ethnic minority-associated environmental risk. *JAMA Psychiatry*, *71*(6), 672–680. doi: 10.1001/jamapsychiatry.2014.35

Beck, S., Wojdyla, D., Say, L., Betran, A. P., Merialdi, M., Requejo, J. H., … Van Look, P. F. (2010). The worldwide incidence of preterm birth: A systematic review of maternal mortality and morbidity. *Bulletin of the World Health Organization*, *88*(1), 31–38. doi: 10.2471/BLT.08.062554

Canli, T., & Lesch, K. P. (2007). Long story short: The serotonin transporter in emotion regulation and social cognition. *Nature Neuroscience*, *10*(9), 1103–1109. doi: 10.1038/nn1964

Casey, B. J., Giedd, J. N., & Thomas, K. M. (2000). Structural and functional brain development and its relation to cognitive development. *Biological Psychology*, *54*(1–3), 241–257. doi: 10.1016/s0301-0511(00)00058-2

Charlson, F. J., Ferrari, A. J., Santomauro, D. F., Diminic, S., Stockings, E., Scott, J. G., … Whiteford, H. A. (2018). Global epidemiology and burden of schizophrenia: Findings from the global burden of disease study 2016. *Schizophrenia Bulletin*, *44*(6), 1195–1203. doi: 10.1093/schbul/sby058

Chiao, J. Y., & Blizinsky, K. D. (2010). Culture-gene coevolution of individualism-collectivism and the serotonin transporter gene. *Proceedings. Biological Sciences*, *277*(1681), 529–537. doi: 10.1098/rspb.2009.1650

Cook, J. L., & Black, J. (2012). The influence of social interaction on cognitive training for schizophrenia. *Frontiers in Neuroscience*, *6*, 140. doi: 10.3389/fnins.2012.00140

Davies, C., Segre, G., Estrade, A., Radua, J., De Micheli, A., Provenzani, U., … Fusar-Poli, P. (2020). Prenatal and perinatal risk and protective factors for psychosis: A systematic review and meta-analysis. *The Lancet. Psychiatry*, *7*(5), 399–410. doi: 10.1016/S2215-0366(20)30057-2

Evins, A. E., Green, A. I., Kane, J. M., & Murray, R. M. (2012). The effect of marijuana use on the risk for schizophrenia. *Journal of Clinical Psychiatry*, *73*(11), 1463–1468. doi: 10.4088/JCP.12012co1c

Fett, A. K., Viechtbauer, W., Dominguez, M. D., Penn, D. L., van Os, J., & Krabbendam, L. (2011). The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, *35*(3), 573–588. doi: 10.1016/j.neubiorev.2010.07.001

Gianaros, P. J., Jennings, J. R., Sheu, L. K., Greer, P. J., Kuller, L. H., & Matthews, K. A. (2007). Prospective reports of chronic life stress predict decreased grey matter volume in the hippocampus. *Neuroimage*, *35*(2), 795–803. doi: 10.1016/j.neuroimage.2006.10.045

Green, M. F. (1996). What are the functional consequences of neurocognitive deficits in schizophrenia? *American Journal of Psychiatry*, *153*(3), 321–330. doi: 10.1176/ajp.153.3.321

Green, M. F., Horan, W. P., & Lee, J. (2015). Social cognition in schizophrenia. *Nature Reviews Neuroscience*, *16*(10), 620–631. doi: 10.1038/nrn4005

Green, M. J., Waldron, J. H., & Coltheart, M. (2007). Emotional context processing is impaired in schizophrenia. *Cognitive Neuropsychiatry*, *12*(3), 259–280. doi: 10.1080/13546800601051847

He, H., Liu, Q., Li, N., Guo, L., Gao, F., Bai, L., … Lyu, J. (2020). Trends in the incidence and DALYs of schizophrenia at the global, regional and national levels: Results from the global burden of disease study 2017. *Epidemiology and Psychiatric Sciences*, *29*, e91. doi: 10.1017/S2045796019000891

Hunter, D. J. (2005). Gene–environment interactions in human diseases. *Nature Reviews Genetics*, *6*(4), 287–298. doi: 10.1038/nrg1578

Jablensky, A., Sartorius, N., Ernberg, G., Anker, M., Korten, A., Cooper, J. E., … Bertelsen, A. (1992). Schizophrenia: Manifestations, incidence and course in different cultures. A world health organization ten-country study. *Psychological Medicine. Monograph Supplement*, *20*, 1–97. doi: 10.1017/s0264180100000904

Lee, S. E., Talegawkar, S. A., Merialdi, M., & Caulfield, L. E. (2013). Dietary intakes of women during pregnancy in low- and middle-income countries. *Public Health Nutrition*, *16*(8), 1340–1353. doi: 10.1017/S1368980012004417

Lipner, E., Murphy, S. K., & Ellman, L. M. (2019). Prenatal maternal stress and the cascade of risk to schizophrenia spectrum disorders in offspring. *Current Psychiatry Reports*, *21*(10), 99. doi: 10.1007/s11920-019-1085-1

Liston, C., McEwen, B. S., & Casey, B. J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences of the United States of America*, *106*(3), 912–917. doi: 10.1073/pnas.0807041106

Luethi, M., Meier, B., & Sandi, C. (2008). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience*, *2*, 5. doi: 10.3389/neuro.08.005.2008

Michalska da Rocha, B., Rhodes, S., Vasilopoulou, E., & Hutton, P. (2018). Loneliness in psychosis: A meta-analytical review. *Schizophrenia Bulletin*, *44*(1), 114–125. doi: 10.1093/schbul/sbx036

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., … Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences of the United States of America*, *106*(34), 14716–14721. doi: 10.1073/pnas.0902971106

Popovic, D., Schmitt, A., Kaurani, L., Senner, F., Papiol, S., Malchow, B., … Falkai, P. (2019). Childhood trauma in schizophrenia: Current findings and research perspectives. *Frontiers in Neuroscience*, *13*, 274. doi: 10.3389/fnins.2019.00274

Rojas-Rueda, D., Morales-Zamora, E., Alsufyani, W. A., Herbst, C. H., AlBalawi, S. M., Alsukait, R., & Alomran, M. (2021). Environmental risk factors and health: An umbrella review of meta-analyses. *International Journal of Environmental Research and Public Health*, *18*(2), 1–38. doi: 10.3390/ijerph18020704

Schizophrenia Working Group of the Psychiatric Genomics, C. (2014). Biological insights from 108 schizophrenia-associated genetic loci. *Nature*, *511*(7510), 421–427. doi: 10.1038/nature13595

Stoolmiller, M. (1999). Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. *Psychological Bulletin*, *125*(4), 392–409. doi: 10.1037/0033-2909.125.4.392

Svrakic, D. M., Zorumski, C. F., Svrakic, N. M., Zwir, I., & Cloninger, C. R. (2013). Risk architecture of schizophrenia: The role of epigenetics. *Current Opinion in Psychiatry*, *26*(2), 188–195. doi: 10.1097/YCO.0b013e32835d8329

van Os, J., Kenis, G., & Rutten, B. P. (2010). The environment and schizophrenia. *Nature*, *468*(7321), 203–212. doi: 10.1038/nature09563

Veling, W., Hoek, H. W., Selten, J. P., & Susser, E. (2011). Age at migration and future risk of psychotic disorders among immigrants in the Netherlands: A 7-year incidence study. *American Journal of Psychiatry*, *168*(12), 1278–1285. doi: 10.1176/appi.ajp.2011.11010110

Wahlbeck, K., Forsen, T., Osmond, C., Barker, D. J., & Eriksson, J. G. (2001). Association of schizophrenia with low maternal body mass index, small size at birth, and thinness during childhood. *Archives of General Psychiatry*, *58*(1), 48–52. doi: 10.1001/archpsyc.58.1.48

Waite, F., Sheaves, B., Isham, L., Reeve, S., & Freeman, D. (2020). Sleep and schizophrenia: From epiphenomenon to treatable causal target. *Schizophrenia Research*, *221*, 44–56. doi: 10.1016/j.schres.2019.11.014

Wang, D., Wang, M., Darling, A. M., Perumal, N., Liu, E., Danaei, G., & Fawzi, W. W. (2020). Gestational weight gain in low-income and middle-income countries: A modelling analysis using nationally representative data. *BMJ Global Health*, *5*(11), 1–9. doi: 10.1136/bmjgh-2020-003423

Yaya, S., & Ghose, B. (2019). Global inequality in maternal health care service utilization: Implications for sustainable development goals. *Health Equity*, *3*(1), 145–154. doi: 10.1089/heq.2018.0082

Evolving the blank slate

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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

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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References

Bates, T. C., Hansell, N. K., Martin, N. G., & Wright, M. J. (2016). When does socioeconomic status (SES) moderate the heritability of IQ? No evidence for G × SES interaction for IQ in a representative sample of 1176 Australian adolescent twin pairs. *Intelligence*, *56*, 10–15. doi:10.1016/j.intell.2016.02.003.

Bates, T. C., Lewis, G. J., & Weiss, A. (2013). Childhood socioeconomic status amplifies genetic effects on adult intelligence. *Psychological Science*, *24*(10), 2111–2116. doi:10.1177/0956797613488394

Bates, T. C., Maher, B. S., Medland, S. E., McAloney, K., Wright, M. J., Hansell, N. K., … Gillespie, N. A. (2018). The nature of nurture: Using a virtual-parent design to test parenting effects on children's educational attainment in genotyped families. *Twin Research and Human Genetics*, *21*(2), 73–83. doi:10.1017/thg.2018.11

Briley, D. A., Harden, K. P., Bates, T. C., & Tucker-Drob, E. M. (2015). Nonparametric estimates of gene × environment interaction using local structural equation modeling. *Behavior Genetics*, *45*(5), 581–596. doi:10.1007/s10519-015-9732-8

Ceci, S. J. (1996). *On intelligence – More or less: A bio-ecological treatise on intellectual development*. Prentice Hall.

Eaves, L. (2017). Genotype × environment interaction in psychiatric genetics: Deep truth or thin Ice? *Twin Research and Human Genetics*, *20*(3), 187–196. doi:10.1017/thg.2017.19

Eaves, L. J., Pourcain, B. S., Smith, G. D., York, T. P., & Evans, D. M. (2014). Resolving the effects of maternal and offspring genotype on dyadic outcomes in genome wide complex trait analysis ("M-GCTA"). *Behavior Genetics*, *44*(5), 445–455. doi:10.1007/s10519-014-9666-6

Engelhardt, L. E., Briley, D. A., Mann, F. D., Harden, K. P., & Tucker-Drob, E. M. (2015). Genes unite executive functions in childhood. *Psychological Science*, *26*(8), 1151–1163. doi:10.1177/0956797615577209

Heath, A. C., Berg, K., Eaves, L. J., Solaas, M. H., Corey, L. A., Sundet, J., … Nance, W. E. (1985). Education policy and the heritability of educational attainment. *Nature*, *314*(6013), 734–736. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/4039415.

Hill, W. G., Goddard, M. E., & Visscher, P. M. (2008). Data and theory point to mainly additive genetic variance for complex traits. *PLoS Genetics*, *4*(2), e1000008. doi:10.1371/journal.pgen.1000008

Hur, Y. M., & Bates, T. (2019). Genetic and environmental influences on cognitive abilities in extreme poverty. *Twin Research and Human Genetics*, *22*(5), 297–301. doi:10.1017/thg.2019.92

Li, Y., & Bates, T. C. (2019). You can't change your basic ability, but you work at things, and that's how we get hard things done: Testing the role of growth mindset on response to setbacks, educational attainment, and cognitive ability. *Journal of Experimental Psychology-General*, *148*(9), 1640–1655. doi:10.1037/xge0000669

Li, Y., & Bates, T. C. (2020). Testing the association of growth mindset and grades across a challenging transition: Is growth mindset associated with grades? *Intelligence*, *81*, 101471. doi:10.1016/j.intell.2020.101471

Lumsden, C. J., & Wilson, E. O. (2005). *Genes, mind, and culture – The coevolutionary process: 25th anniversary edition*. World Scientific.

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research*, *5*(6), 554–571. doi:10.1375/136905202762342026

Ritchie, S. J., Bates, T. C., & Deary, I. J. (2015). Is education associated with improvements in general cognitive ability, or in specific skills? *Developmental Psychology*, *51*(5), 573–582. doi:10.1037/a0038981

Ritchie, S. J., Bates, T. C., Der, G., Starr, J. M., & Deary, I. J. (2013). Education is associated with higher later life IQ scores, but not with faster cognitive processing speed. *Psychology and Aging*, *28*(2), 515–521. doi:10.1037/a0030820

Samuelsson, S., Byrne, B., Olson, R. K., Hulslander, J., Wadsworth, S., Corley, R., … Defries, J. C. (2008). Response to early literacy instruction in the United States, Australia, and Scandinavia: A behavioral-genetic analysis. *Learning and Individual Differences*, *18*(3), 289–295. doi:10.1016/j.lindif.2008.03.004

Sherlock, J. M., & Zietsch, B. P. (2018). Longitudinal relationships between parents' and children's behavior need not implicate the influence of parental behavior and may reflect genetics: Comment on Waldinger and Schulz (2016). *Psychological Science*, *29*(1), 154–157. doi:10.1177/0956797617717041

Tucker-Drob, E. M., & Bates, T. C. (2016). Large cross-national differences in gene × socioeconomic status interaction on intelligence. *Psychological Science*, *27*(2), 138–149. doi:10.1177/0956797615612727

(Super-)cultural clustering explains gender differences too

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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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References

Abbate, J. (2017). *Recoding gender: Women's changing participation in computing*. MIT Press.

Anker, R. (1998). *Gender and jobs: Sex segregation of occupations in the world*. International Labour Office.

Boothroyd, L. G., Jucker, J.-L., Thornborrow, T., Barton, R. A., Burt, D. M., Evans, E. H., … Tovée, M. J. (2020). Television consumption drives perceptions of female body attractiveness in a population undergoing technological transition. *Journal of Personality and Social Psychology*, *119*(4), 839–860. https://doi.org/10.1037/pspi0000224.

Davis, H. E., Stack, J., & Cashdan, E. (2021). Cultural change reduces gender differences in mobility and spatial ability among seminomadic pastoralist-forager children in northern Namibia. *Human Nature*, *32*(1), 178–206. https://doi.org/10.1007/s12110-021-09388-7.

Falk, A., & Hermle, J. (2018). Relationship of gender differences in preferences to economic development and gender equality. *Science (New York, N.Y.)*, *362*(6412), eaas9899. https://doi.org/10.1126/science.aas9899.

Fisher, S., & Jenson, J. (2017). Producing alternative gender orders: A critical look at girls and gaming. *Learning, Media and Technology*, *42*(1), 87–99. https://doi.org/10.1080/17439884.2016.1132729.

Ghaznavi, J., Grasso, K. L., & Taylor, L. D. (2017). Increasingly violent but still sexy: A decade of central female characters in top-grossing Hollywood and Bollywood film promotional material. *International Journal of Communication*, *11*, 23–47.

Hines, M. (2020). Human gender development. *Neuroscience & Biobehavioral Reviews*, *118*, 89–96. https://doi.org/10.1016/j.neubiorev.2020.07.018.

Jucker, J.-L., Thornborrow, T., Beierholm, U., Burt, D. M., Barton, R. A., Evans, E. H., … Boothroyd, L. G. (2017). Nutritional status and the influence of TV consumption on female body size ideals in populations recently exposed to the media. *Scientific Reports*, *7*(1), 8438. https://doi.org/10.1038/s41598-017-08653-z.

Kramer, K. L., & Veile, A. (2018). Infant allocare in traditional societies. *Physiology & Behavior*, *193*, 117–126. https://doi.org/10.1016/j.physbeh.2018.02.054.

Marlowe, F. (2001). Male contribution to diet and female reproductive success among foragers. *Current Anthropology*, *42*(5), 755–760. https://doi.org/10.1086/323820.

Marlowe, F. W. (2007). Hunting and gathering: The human sexual division of foraging labor. *Cross-Cultural Research*, *41*(2), 170–195. https://doi.org/10.1177/1069397106297529.

https://doi.org/10.1017/S0140525X21000893 Published online by Cambridge University Press

Meltzoff, A. N. (2007). "Like me": A foundation for social cognition. *Developmental Science*, *10*(1), 126–134. https://doi.org/10.1111/j.1467-7687.2007.00574.x.
Morgan, T. J. H., Rendell, L. E., Ehn, M., Hoppitt, W., & Laland, K. N. (2012). The evolutionary basis of human social learning. *Proceedings of the Royal Society B: Biological Sciences*, *279*(1729), 653–662. https://doi.org/10.1098/rspb.2011.1172.
Page, A., Hassan, A., Viguier, S., Dyble, M., Smith, D., & Migliano, A. (2021). *Livelihood diversity and childcare in a foraging population, the Agta of Palanan, Philippines*. Presented at the European Human Behaviour and Evolution Association, Krakow, Poland.
Schmitt, D. P. (2015). The evolution of culturally-variable sex differences: Men and women are not always different, but when they are…It appears not to result from patriarchy or sex role socialization. In T. K. Shackelford & R. D. Hansen (Eds.), *The evolution of sexuality* (pp. 221–256). Springer International. https://doi.org/10.1007/978-3-319-09384-0_11.
Sear, R. (2016). Beyond the nuclear family: An evolutionary perspective on parenting. *Current Opinion in Psychology*, *7*, 98–103. https://doi.org/10.1016/j.copsyc.2015.08.013.
Smaldino, P. E., Lukaszewski, A., von Rueden, C., & Gurven, M. (2019). Niche diversity can explain cross-cultural differences in personality structure. *Nature Human Behaviour*, *3*(12), 1276–1283. https://doi.org/10.1038/s41562-019-0730-3.
Swami, V., Frederick, D. A., Aavik, T., Alcalay, L., Allik, J., Anderson, D., … Zivcic-Becirevic, I. (2010). The attractive female body weight and female body dissatisfaction in 26 countries across 10 world regions: Results of the international body project I. *Personality and Social Psychology Bulletin*, *36*(3), 309–325. https://doi.org/10.1177/0146167209359702.
Trumble, B. C., Gaulin, S. J. C., Dunbar, M. D., Kaplan, H., & Gurven, M. (2016). No sex or age difference in dead-reckoning ability among Tsimane forager-horticulturalists. *Human Nature*, *27*(1), 51–67. https://doi.org/10.1007/s12110-015-9246-3.
Vashro, L., Padilla, L., & Cashdan, E. (2016). Sex differences in mobility and spatial cognition: A test of the fertility and parental care hypothesis in Northwestern Namibia. *Human Nature*, *27*(1), 16–34. https://doi.org/10.1007/s12110-015-9247-2.
Wood, L. A., Kendal, R. L., & Flynn, E. G. (2013). Whom do children copy? Model-based biases in social learning. *Developmental Review*, *33*(4), 341–356. https://doi.org/10.1016/j.dr.2013.08.002.
Wood, W., & Eagly, A. H. (2012). Biosocial construction of sex differences and similarities in behavior. In *Advances in experimental social psychology* (Vol. 46, pp. 55–123). Elsevier. https://doi.org/10.1016/B978-0-12-394281-4.00002-7.

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

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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

"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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References

Anderson, E. (1999). *Code of the street: Decency, violence, and the moral life of the inner city*. W.W. Norton.

Belsky, D. W., Domingue, B. W., Wedow, R., Arseneault, L., Boardman, J. D., Caspi, A., … Herd, P. (2018). Genetic analysis of social-class mobility in five longitudinal studies. *Proceedings of the National Academy of Sciences*, *115*(31), E7275–E7284.

Belsky, D. W., Moffitt, T. E., Corcoran, D. L., Domingue, B., Harrington, H., Hogan, S., … Williams, B. S. (2016). The genetics of success: How single-nucleotide polymorphisms associated with educational attainment relate to life-course development. *Psychological Science*, *27*(7), 957–972.

Burt, C. H. (2018). Racial discrimination and cultural adaptations: An evolutionary developmental approach. *Advances in Criminological Theory: Building a Black Criminology*, *24*, 207–252.

Burt, C. H., & Simons, R. L. (2014). Pulling back the curtain on heritability studies: Biosocial criminology in the postgenomic era. *Criminology; An interdisciplinary Journal*, *52*(2), 223–262.

Carter, P. L. (2003). "Black" cultural capital, status positioning, and schooling conflicts for low-income African American youth. *Social Problems*, *50*(1), 136–155.

Hannerz, U. (1969). *Soulside: Inquiries into ghetto culture and community*. Columbia University Press.

Massey, D., & Denton, N. A. (1993). *American Apartheid: Segregation and the making of the underclass*. Harvard University Press.

Patterson, O., & Fosse, E. (Eds.). (2015). *The cultural matrix: Understanding black youth*. Harvard University Press.

Sampson, R. J. (2008). Moving to inequality: Neighborhood effects and experiments meet social structure. *American Journal of Sociology*, *114*(1), 189–231.

Turkheimer, E. (2011). Commentary: Variation and causation in the environment and genome. *International Journal of Epidemiology*, *40*(3), 598–601.

Wilson, W. J. (2010). Why both social structure and culture matter in a holistic analysis of inner-city poverty. *The Annals of the American Academy of Political and Social Science*, *629*(1), 200–219.

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

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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.

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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References

Abdellaoui, A., & Verweij, K. J. (2021). Dissecting polygenic signals from genome-wide association studies on human behaviour. *Nature Human Behaviour*, *5*, 686–694. doi: https://doi.org/10.1038/s41562-021-01110-y.

Abdellaoui, A., Hugh-Jones, D., Yengo, L., Kemper, K. E., Nivard, M. G., Veul, L., & (2019). Genetic correlates of social stratification in Great Britain. *Nature Human Behaviour*, *3*(5), 1332–1342. doi: 10.1038/s41562-019-0757-5.

Bowles, S., Gintis, H., & Groves, M. O. (2009). *Unequal chances: Family background and economic success*. Princeton University Press.

Cohen, A. K., Rai, M., Rehkopf, D. H., & Abrams, B. (2013). Educational attainment and obesity: A systematic review. *Obesity Review*, *14*(12), 989–1005. doi: 10.1111/obr.12062.

Headey, D. D., & Alderman, H. A. (2019). The relative caloric prices of healthy and unhealthy foods differ systematically across income levels and continents. *The Journal of Nutrition*, *149*(11), 2020–2033. doi: 10.1093/jn/nxz158.

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world? *Behavioral and Brain Sciences*, *33*(2–3), 61–83. doi: 10.1017/S0140525X0999152X.

Martin, A. R., Kanai, M., Kamatani, Y., Okada, Y., Neale, B. M., & Daly, M. J. (2021). Clinical use of current polygenic risk scores may exacerbate health disparities. *Nature Genetics*, *54*, 584–591. doi: 10.1038/s41588-019-0379-x

Morris, T. T., Davies, N. M., Hemani, G., & Davey Smith, G. (2020). Population phenomena inflate genetic associations of complex social traits. *Science Advances*, *6*. doi: 10.1126/sciadv.aay0328

Psacharopoulos, G., & Patrinos, H. A. (2018). Returns to investment in education: A decennial review of the global literature. *Education Economics*, *28*(5), 445–458. doi: https://doi.org/10.1080/09645292.2018.1484426.

Rogers, A. R., & Jorde, L. B. (1987). The effect of non-random migration on genetic differences between populations. *Annals of Human Genetics*, *51*(2), 169-176. doi: 10.1111/j.1469-1809.1987.tb01059.x.

Sanderson, E., Davey Smith, G., Windmeijer, F., & Bowden, J. (2019). An examination of multivariable Mendelian randomization in the single-sample and two-sample summary data settings. *International Journal of Epidemiology*, *49*(3), 713–727. doi: 10.1093/ije/dyaa101.

Scutari, M., Mackay, I., & Balding, D. (2016). Using genetic distance to infer the accuracy of genomic prediction. *PLoS Genetics*, *12*(9). doi: 10.1371/journal.pgen.1006288.

Sebro, R., & Risch, N. J. (2012). A brief note on the resemblance between relatives in the presence of population stratification. *Heredity*, *108*, 563–568. doi: https://doi.org/10.1038/hdy.2011.124.

Smith, G. D., & Ebrahim, S. (2002). Data dredging, bias, or confounding. *British Medical Journal*, *326*(7378), 1437–1438. doi: 10.1136/bmj.325.7378.1437.

Smith, G. D., & Ebrahim, S. (2003). "Mendelian randomization": Can genetic epidemiology contribute to understanding environmental determinants of disease? *International Journal of Epidemiology*, *32*(1), 1–22. doi: https://doi.org/10.1093/ije/dyg070.

Yengo, L., Robinson, M. R., Keller, M. C., Kemper, K. E., Yang, Y., Trzaskowski, M., … Visscher, P. M. (2018). Imprint of assortative mating on the human genome. *Nature Human Behaviour*, *2*, 948–954. doi: https://doi.org/10.1038/s41562-018-0476-3.

Changes in heritability: Unpredictable and of limited use

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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).

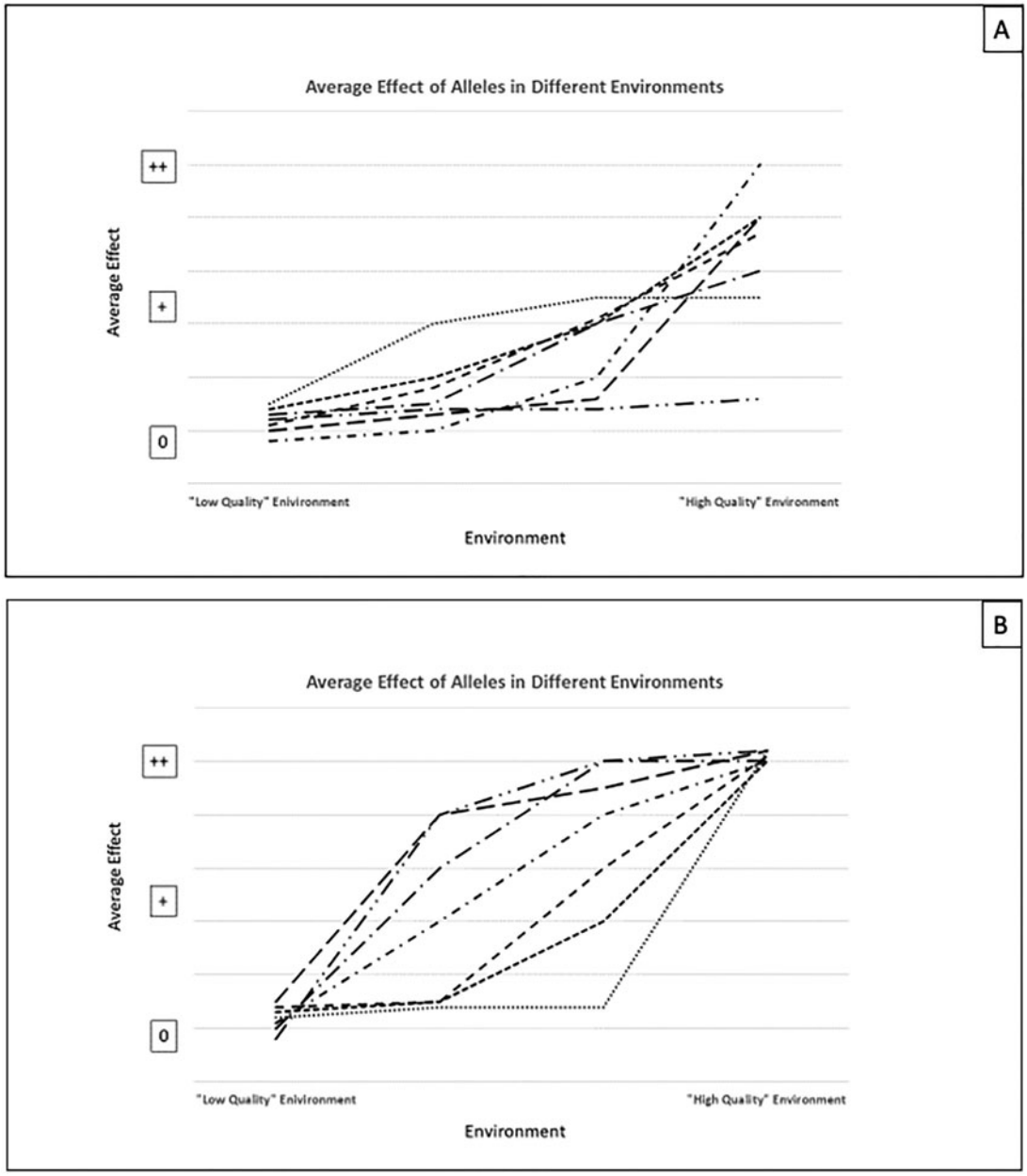

Figure 1 (Downes and Kaplan). When Uchiyama et al. suggest, first, that populations in higher quality environments (for the development of a particular trait) will tend to have higher heritability, and second, that reductions in environmental variation will increase heritability, they likely have a picture 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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References

Beam, C. R., & Turkheimer, E. (2013). Phenotype–environment correlations in longitudinal twin models. *Development and Psychopathology*, *25*(1), 7–16. https://doi.org/10.1017/S0954579412000867.

Cooper, R. M., & Zubek, J. P. (1958). Effects of enriched and restricted early environments on the learning ability of bright and dull rats. *Canadian Journal of Psychology/Revue Canadienne de Psychologie*, *12*(3), 159–164. https://doi.org/10.1037/h0083747.

Crabbe, J. C., Wahlsten, D., & Dudek, B. C. (1999). Genetics of mouse behavior: Interactions with lab environment. *Science (New York, N.Y.)*, *284*, 1670–1672.

Harden, K. P. (2021). "Reports of my death were greatly exaggerated": Behavior genetics in the Postgenomic era. *Annual Review of Psychology*, *72*(1), 37–60. https://doi.org/10.1146/annurev-psych-052220-103822.

Laland, K. N., Oddling-Smee, J., & Feldman, M. W. (2000). Niche construction, biological evolution, and cultural change. *Behavioral and Brain Sciences*, *23*, 131–175.

Lewontin, R. C. (1974). Analysis of variance and analysis of causes. *American Journal of Human Genetics*, *26*, 400–411.

Lewontin, R. C. (1983). The organism as the subject and object of evolution. *Scientia; Rivista di Scienza*, *118*, 65–95.

Lewontin, R. C. (1992). *Biology as ideology: The doctrine of DNA* (1st U.S. ed). Harper Perennial.

Sauce, B., Bendrath, S., Herzfeld, M., Siegel, D., Style, C., Rab, S., … Matzel, L. D. (2018). The impact of environmental interventions among mouse siblings on the heritability and malleability of general cognitive ability. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1756), 20170289. https://doi.org/10.1098/rstb.2017.0289.

Turkheimer, E. (2004). Spinach and Ice cream: Why social science is so difficult. In L. F. DiLalla (Ed.), *Behavior genetics principles: Perspectives in development, personality, and psychopathology* (pp. 161–189). American Psychological Association. https://doi.org/10.1037/10684-011.

Unpackaging cultural variability in behavioral phenotypes

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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,

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.

Conflict of interest. None.

References

Boer, D., Hanke, K., & He, J. (2018). On detecting systematic measurement error in cross-cultural research: A review and critical reflection on equivalence and invariance tests. *Journal of Cross-Cultural Psychology*, *49*(5), 713–734. https://doi.org/10.1177/0022022117749042.

Bond, M. H., & Lun, V. M.-C. (2014). Citizen-making: The role of national goals for socializing children. *Social Science Research*, *44*, 75–85. https://doi.org/10.1016/j.ssresearch.2013.11.002.

Chen, C., Chen, C., Moyzis, R. K., He, Q., Lei, X., Li, J., … Dong, Q. (2013). Genotypes over-represented among college students are linked to better cognitive abilities and socioemotional adjustment. *Culture and Brain*, *1*(1), 47–63. https://doi.org/10.1007/s40167-013-0003-3.

Chen, C., Moyzis, R. K., Lei, X., Chen, C., & Dong, Q. (2015). The encultured genome: Molecular evidence for recent divergent evolution in human neurotransmitter genes. In J. Y. Chiao (Ed.), *The Oxford handbook of cultural neuroscience* (pp. 315–336). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780199357376.013.22.

Chen, C., & Stevenson, H. W. (1995). Motivation and mathematics achievement: A comparative study of Asian-American, Caucasian-American, and East Asian high school students. *Child Development*, *66*(4), 1215–1234. https://doi.org/10.1111/j.1467-8624.1995.tb00932.x.

Dekker, S., & Fischer, R. (2008). Cultural differences in academic motivation goals: A meta-analysis across 13 societies. *The Journal of Educational Research*, *102*(2), 99–110. https://doi.org/10.3200/JOER.102.2.99-110.

Fischer, R., & Karl, J. A. (2019). A primer to (cross-cultural) multi-group invariance testing possibilities in R. *Frontiers in Psychology*, *10*, 1–18. https://doi.org/10.3389/fpsyg.2019.01507.

Fischer, R., & Poortinga, Y. H. (2018). Addressing methodological challenges in culture-comparative research. *Journal of Cross-Cultural Psychology*, *49*(5), 691–712. https://doi.org/10.1177/0022022117738086.

Fontanillas, P., Alipanahi, B., Furlotte, N. A., Johnson, M., Wilson, C. H., Pitts, S. J., … Auton, A. (2021). Disease risk scores for skin cancers. *Nature Communications*, *12*(1), 160. https://doi.org/10.1038/s41467-020-20246-5.

Friedman, N. P., Miyake, A., Corley, R. P., Young, S. E., Defries, J. C., & Hewitt, J. K. (2006). Not all executive functions are related to intelligence. *Psychological Science*, *17*(2), 172–179. https://doi.org/10.1111/j.1467-9280.2006.01681.x.

Gibson, M. A., & Mace, R. (2006). An energy-saving development initiative increases birth rate and childhood malnutrition in rural Ethiopia. *PLoS Medicine*, *3*(4), e87. https://doi.org/10.1371/journal.pmed.0030087.

Hambleton, R. K., & Zenisky, A. L. (2010). Translating and adapting tests for cross-cultural assessments. In D. Matsumoto & F. J. R. van de Vijver (Eds.), *Cross-cultural research methods in psychology* (pp. 46–70). Cambridge University Press. https://doi.org/10.1017/CBO9780511779381.004.

Hrdy, S. B. (2000). *Mother nature: Maternal instincts and how they shape the human species* (1st ed). Ballantine Books.

Laland, K. N. (2017). *Darwin's unfinished symphony: How culture made the human mind*. Princeton University Press.

Laland, K. N., Uller, T., Feldman, M. W., Sterelny, K., Müller, G. B., Moczek, A., … Odling-Smee, J. (2015). The extended evolutionary synthesis: Its structure, assumptions and predictions. *Proceedings of the Royal Society B: Biological Sciences*, *282* (1813), 20151019. https://doi.org/10.1098/rspb.2015.1019.

OECD (Ed.). (2010). *Pisa 2009 results. 2: Overcoming social background: Equity in learning opportunities and outcomes*. OECD.

Samuelsson, S., Byrne, B., Olson, R. K., Hulslander, J., Wadsworth, S., Corley, R., … DeFries, J. C. (2008). Response to early literacy instruction in the United States, Australia, and Scandinavia: A behavioral-genetic analysis. *Learning and Individual Differences*, *18*(3), 289–295. https://doi.org/10.1016/j.lindif.2008.03.004.

Serpell, R. (1979). How specific are perceptual skills? A cross-cultural study of pattern reproduction. *British Journal of Psychology*, *70*(3), 365–380. https://doi.org/10.1111/j.2044-8295.1979.tb01706.x.

Smith, P. B., Fischer, R., Vignoles, V. L., & Bond, M. H. (2013). *Understanding social psychology across cultures: Engaging with others in a changing world* (2nd ed.). Sage.

Sortheix, F. M., Parker, P. D., Lechner, C. M., & Schwartz, S. H. (2019). Changes in young Europeans' values during the global financial crisis. *Social Psychological and Personality Science*, *10*(1), 15–25. https://doi.org/10.1177/1948550617732610.

Sternberg, R. J. (1985). *Beyond IQ: A triarchic theory of human intelligence*. Cambridge University Press.

Tinbergen, N. (1963). On aims and methods of ethology. *Zeitschrift für Tierpsychologie*, *20*(4), 410–433. https://doi.org/10.1111/j.1439-0310.1963.tb01161.x.

van de Vliert, E. (2009). *Climate, affluence, and culture*. Cambridge University Press.

https://doi.org/10.1017/S0140525X21000893 Published online by Cambridge University Press

Vecchione, M., Schwartz, S., Alessandri, G., Döring, A. K., Castellani, V., & Caprara, M. G. (2016). Stability and change of basic personal values in early adulthood: An 8-year longitudinal study. *Journal of Research in Personality*, *63*, 111–122. https://doi.org/10.1016/j.jrp.2016.06.002.

von Rueden, C. R., & Jaeggi, A. V. (2016). Men's status and reproductive success in 33 nonindustrial societies: Effects of subsistence, marriage system, and reproductive strategy. *Proceedings of the National Academy of Sciences*, *113*(39), 10824–10829. https://doi.org/10.1073/pnas.1606800113.

World Bank. (2021). *DataBank*. https://databank.worldbank.org/home.aspx.

Cultural dynamics add multiple layers of complexity to behavioural genetics

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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 ($G \times E$) 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 $G \times E$ 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 $G \times E$ 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 $G \times E$ 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

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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References

Arnold, S. J., Bürger, R., Hohenlohe, P. A., Ajie, B. C., & Jones, A. G. (2008). Understanding the evolution and stability of the G-matrix. *Evolution; International Journal of Organic Evolution*, *62*(10), 2451–2461. https://doi.org/10.1111/j.1558-5646.2008.00472.x.

Cavalli-Sforza, L. L., & Feldman, M. W. (1973a). Cultural versus biological inheritance: Phenotypic transmission from parents to children (A theory of the effect of parental phenotypes on children's phenotypes). *American Journal of Human Genetics*, *25*(6), 618. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1762580/.

Cavalli-Sforza, L. L., & Feldman, M. W. (1973b). Models for cultural inheritance I. Group mean and within group variation. *Theoretical Population Biology*, *4*, 42–55.

Creanza, N., & Feldman, M. W. (2014). Complexity in models of cultural niche construction with selection and homophily. Proceedings of the National Academy of Sciences *of the United States of America*, *111*(3), 10830–10837.

Creanza, N., Fogarty, L., & Feldman, M. W. (2012). Models of cultural niche construction with selection and assortative mating. *PLoS ONE*, *7*(8), e42744. https://doi.org/10.1371/journal.pone.0042744.

Danchin, É., & Wagner, R. H. (2010). Inclusive heritability: Combining genetic and non-genetic information to study animal behavior and culture. *Oikos*, *119*(2), 210–218. https://doi.org/10.1111/j.1600-0706.2009.17640.x.

Falconer, D. S., & Mackay, T. F. C. (1996). *Introduction to quantitative genetics* (4th ed.). Fongman.

Feldman, M. W., & Lewontin, R. C (1975). The heritability hang-up. *Science (New York, N.Y.)*, *190*(4220), 1163–1168.

Feldman, M. W., & Ramachandran, S. (2018). Missing compared to what? Revisiting heritability, genes and culture. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1743). https://doi.org/10.1098/rstb.2017.0064.

Feldman, M. W., & Zhivotovsky, L. A. (1992). Gene–culture coevolution: Toward a general theory of vertical transmission. *Proceedings of the National Academy of Sciences of the United States of America*, *89*(24), 11935–11938. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=50672&tool=pmcentrez&rendertype=abstract.

Fogarty, L., Creanza, N., & Feldman, M. W. (2013). The role of cultural transmission in human demographic change: An age-structured model. *Theoretical Population Biology*, *88*, 68–77. https://doi.org/10.1016/j.tpb.2013.06.006.

Fogarty, L., Creanza, N., & Feldman, M. W. (2019). The life history of learning: Demographic structure changes cultural outcomes. *PLoS Computational Biology*, *15*(4), e1006821. https://doi.org/10.1371/journal.pcbi.1006821.

Gilpin, W., & Feldman, M. W. (2019). Cryptic selection forces and dynamic heritability in generalized phenotypic evolution. *Theoretical Population Biology*, *125*, 20–29. https://doi.org/10.1016/j.tpb.2018.11.002.

Karlin, S. (1988). Non-Gaussian phenotypic models of quantitative traits. In E. J. Eisen, M. M. Goodman, G. Namkoong, & B. S. Weir, (Eds.), *The Second International Conference on Quantitative Genetics* (pp. 123–144). Sinauer.

Laland, K. N., Kumm, J., & Feldman, M. W. (1995). Gene–culture coevolutionary theory: A test case. *Current Anthropology*, *36*(1), 131–156. Retrieved from http://www.sciencedirect.com/science/article/pii/0169534796100525.

Lande, R. (1979). Quantitative genetic analysis of multivariate evolution, applied to brain: body size allometry. *Evolution*, *33*(1), 402–416.

Lewontin, R. C. (1974). The analysis of variance and the analysis of causes. *American Journal of Human Genetics*, *26*, 400–411. https://doi.org/10.1093/ije/dyl062.

Schluter, D. (1984). Morphological and phylogenetic relations among the Darwin's finches. *Evolution*, *38*(5), 921–930. https://doi.org/10.1111/j.1558-5646.1984.tb00363.x.

Turelli, M. (1988). Phenotypic evolution, constant covariances, and the maintenance of additive variance, *42*(6), 1342–1347.

de Villemereuil, P., Schielzeth, H., Nakagawa, S., & Morrissey, M. (2016). General methods for evolutionary quantitative genetic inference from generalized mixed models. *Genetics*, *204*(3), 1281–1294. https://doi.org/10.1534/genetics.115.186536.

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

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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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References

Briley, D. A., Livengood, J., Derringer, J., Tucker-Drob, E. M., Fraley, R. C., & Roberts, B. W. (2019). Interpreting behavior genetic models: Seven developmental processes to understand. *Behavior Genetics*, *49*(2), 196–210.

Falconer, D. S. (1967). *Introduction to quantitative genetics*. Oliver and Boys.

Fuentes, A. (2016). The extended evolutionary synthesis, ethnography, and the human niche: Toward an integrated anthropology. *Current Anthropology*, *57*(Suppl 13), 13–26. https://doi.org/10.1086/685684.

Jansson, F., Aguilar, E., Acerbi, A., & Enquist, M. (2021). Modelling cultural systems and selective filters. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *376*, 20200045. https://doi.org/10.1098/rstb.2020.0045.

Kempthorne, O. (1978). A biometrics invited paper: Logical, epistemological and statistical aspects of nature-nurture data interpretation. *Biometrics*, *34*(1), 1–23.

Kong, A., Thorleifsson, G., Frigge, M. L., Vilhjalmsson, B. J., Young, A. I., Thorgeirsson, T. E., … Stefansson, K. (2018). The nature of nurture: Effects of parental genotypes. *Science (New York, N.Y.)*, *359*(6374), 424–428.

Lewens T. (2015). *Cultural evolution: Conceptual challenges*. Oxford University Press.

Mathieson, I. (2021). The omnigenic model and polygenic prediction of complex traits. *The American Journal of Human Genetics*, *108*(9), P1558–1563. https://doi.org/10.1016/j.ajhg.2021.07.003.

Mostafavi, H., Harpak, A., Agarwal, I., Conley, D., Pritchard, J. K., & Przeworski, M. (2020). Variable prediction accuracy of polygenic scores within an ancestry group. *eLife*, *9*, e48376.

Rosenberg, N. A., Edge, M. D., Pritchard, J. K., & Feldman, M. W. (2019). Interpreting polygenic scores, polygenic adaptation, and human phenotypic differences. *Evolution, Medicine, and Public Health*, *2019*(1), 26–34.

Stotz, K. (2010). Human nature and cognitive–developmental niche construction. *Phenomenology and Cognitive Science 9* 483–501.

Tucker, S. L., Dohleman, F. G., Grapov, D., Flagel, L., Yang, S., Wegener, K. M., … Rice, E. A. (2020). Evaluating maize phenotypic variance, heritability, and yield relationships at multiple biological scales across agronomically relevant environments. *Plant, Cell & Environment*, *43*(4), 880–902.

Tyrrell, J., Zheng, J., Beaumont, R., Hinton, K., Richardson, T. G., Wood, A. R., … Tilling, K. (2021). Genetic predictors of participation in optional components of UK biobank. *Nature Communications*, *12*(1), 1–13.

Uller, T., & Laland, K. N. (2019). *Evolutionary causation: Biological and philosophical reflections*. MIT Press.

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The many geographical layers of culture

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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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References

Chua, R. Y. J., Huang, K. G., & Jin, M. (2019). Mapping cultural tightness and its links to innovation, urbanization, and happiness across 31 provinces in China. *Proceedings of the National Academy of Sciences*, *116*(14), 6720–6725. https://doi.org/10.1073/pnas.1815723116.

Cohen, A. B. (2009). Many forms of culture. *American Psychologist*, *64*(3), 194–204. https://doi.org/10.1037/a0015308.

Ebert, T., Gebauer, J. E., Brenner, T., Bleidorn, W., Gosling, S. D., Potter, J., & Rentfrow, P. J. (2021). Are regional differences in psychological characteristics and their correlates robust? Applying spatial-analysis techniques to examine regional variation in personality. *Perspectives on Psychological Science*, 174569162199832. https://doi.org/10.1177/1745691621998326.

Fritsch, M., Obschonka, M., & Wyrwich, M. (2019). Historical roots of entrepreneurship-facilitating culture and innovation activity: An analysis for German regions. *Regional Studies*, *53*(9), 1296–1307. https://doi.org/10.1080/00343404.2019.1580357.

Götz, F. M., Ebert, T., Gosling, S. D., Obschonka, M., Potter, J., & Rentfrow, P. J. (2021). Local housing market dynamics predict rapid shifts in cultural openness: A 9-year study across 199 cities. *American Psychologist*, *76*(6), 947–961.

Götz, F. M., Ebert, T., & Rentfrow, P. J. (2018). Regional cultures and the psychological geography of Switzerland: Person–environment–fit in personality predicts subjective wellbeing. *Frontiers in Psychology*, *9*, 1–16. https://doi.org/10.3389/fpsyg.2018.00517.

Grossmann, I., & Varnum, M. E. W. (2015). Social structure, infectious diseases, disasters, secularism, and cultural change in America. *Psychological Science*, *26*(3), 311–324. https://doi.org/10.1177/0956797614563765.

Harrington, J. R., & Gelfand, M. J. (2014). Tightness-looseness across the 50 United States. *Proceedings of the National Academy of Sciences*, *111*(22), 7990–7995. https://doi.org/10.1073/pnas.1317937111.

Inglehart, R., & Baker, W. E. (2000). Modernization, cultural change, and the persistence of traditional values. *American Sociological Review*, *65*(1), 19. https://doi.org/10.2307/2657288.

Jackson, J. C., Gelfand, M., De, S., & Fox, A. (2019). The loosening of American culture over 200 years is associated with a creativity–order trade-off. *Nature Human Behaviour*, *3*(3), 244–250. https://doi.org/10.1038/s41562-018-0516-z.

Jokela, M., Bleidorn, W., Lamb, M. E., Gosling, S. D., & Rentfrow, P. J. (2015). Geographically varying associations between personality and life satisfaction in the London metropolitan area. *Proceedings of the National Academy of Sciences*, *112*(3), 725–730. https://doi.org/10.1073/pnas.1415800112.

Lee, N. (2017). Psychology and the geography of innovation. *Economic Geography*, *93*(2), 106–130. https://doi.org/10.1080/00130095.2016.1249845.

Muthukrishna, M., Bell, A. V., Henrich, J., Curtin, C. M., Gedranovich, A., McInerney, J., & Thue, B. (2020). Beyond Western, educated, industrial, rich, and democratic (WEIRD) psychology: Measuring and mapping scales of cultural and psychological distance. *Psychological Science*, *31*(6), 678–701. https://doi.org/10.1177/0956797620916782.

Obschonka, M. (2017). The quest for the entrepreneurial culture: Psychological big data in entrepreneurship research. *Current Opinion in Behavioral Sciences*, *18*, 69–74. https://doi.org/10.1016/j.cobeha.2017.07.014.

Obschonka, M., Schmitt-Rodermund, E., Silbereisen, R. K., Gosling, S. D., & Potter, J. (2013). The regional distribution and correlates of an entrepreneurship-prone personality profile in the United States, Germany, and the United Kingdom: A socioecological perspective. *Journal of Personality and Social Psychology*, *105*(1), 104–122. https://doi.org/10.1037/a0032275.

Obschonka, M., Stuetzer, M., Gosling, S. D., Rentfrow, P. J., Lamb, M. E., Potter, J., & Audretsch, D. B. (2015). Entrepreneurial regions: Do macro-psychological cultural characteristics of regions help solve the "Knowledge paradox" of economics? *PLoS ONE*, *10*(6), e0129332. https://doi.org/10.1371/journal.pone.0129332.

Ofosu, E. K., Chambers, M. K., Chen, J. M., & Hehman, E. (2019). Same-sex marriage legalization associated with reduced implicit and explicit antigay bias. *Proceedings of the National Academy of Sciences*, *116*(18), 8846–8851. https://doi.org/10.1073/pnas.1806000116.

Park, N., & Peterson, C. (2010). Does it matter where we live? The urban psychology of character strengths. *American Psychologist*, *65*(6), 535–547. https://doi.org/10.1037/a0019621.

Rentfrow, P. J. (2020). Geographical psychology. *Current Opinion in Psychology*, *32*, 165–170. https://doi.org/10.1016/j.copsyc.2019.09.009.

Rentfrow, P. J., Gosling, S. D., & Potter, J. (2008). A theory of the emergence, persistence, and expression of geographic variation in psychological characteristics. *Perspectives on Psychological Science*, *3*(5), 339–369. https://doi.org/10.1111/j.1745-6924.2008.00084.x.

Rentfrow, P. J., & Jokela, M. (2016). Geographical psychology: The spatial organization of psychological phenomena. *Current Directions in Psychological Science*, *25*(6), 393–398. https://doi.org/10.1177/0963721416658446.

Rentfrow, P. J., Jokela, M., & Lamb, M. E. (2015). Regional personality differences in Great Britain. *PLoS ONE*, *10*(3), e0122245. https://doi.org/10.1371/journal.pone.0122245.

Talhelm, T., Zhang, X., Oishi, S., Shimin, C., Duan, D., Lan, X., & Kitayama, S. (2014). Large-scale psychological differences within China explained by rice versus wheat agriculture. *Science*, *344*(6184), 603–608. https://doi.org/10.1126/science.1246850.

Vandello, J. A., & Cohen, D. (1999). Patterns of individualism and collectivism across the United States. *Journal of Personality and Social Psychology*, *77*(2), 279–292. https://doi.org/10.1037/0022-3514.77.2.279.

The dubious precision and utility of heritability estimates

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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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References

Causadias, J. M., Vitriol, J. A., & Atkin, A. L. (2018). Do we overemphasize the role of culture in the behavior of racial/ethnic minorities? Evidence of a cultural (mis)attribution bias in American psychology. *American Psychologist*, *73*, 243–255.

Cohen, A. B. (2009). Many forms of culture. *American Psychologist*, *64*, 194–204.

Dar-Nimrod, I., & Heine, S. J. (2011a). Some thoughts on essence placeholders, interactionism, and heritability: Reply to Haslam (2011) and Turkheimer (2011). *Psychological Bulletin*, *137*, 829–833.

Dar-Nimrod, I., & Heine, S. J. (2011b). Genetic essentialism: On the deceptive determinism of DNA. *Psychological Bulletin*, *137*, 800–818.

Dar-Nimrod, I., Kuntzman, R., MacNevin, G., Lynch, K., Woods, M., & Morandini, J. (2021). Genetic essentialism: The mediating role of essentialist biases on the relationship between genetic knowledge and the interpretations of genetic information. *European Journal of Medical Genetics*, *64*, 104119.

Derringer, J., Krueger, R. F., Dick, D. M., Saccone, S., Grucza, R. A., Agrawal, A., … Gene Environment Association Studies (GENEVA) Consortium (2010). Predicting sensation seeking from dopamine genes: A candidate-system approach. *Psychological Science*, *21*, 1282–1290.

Gelman, S. A. (2003). *The essential child: Origins of essentialism in everyday thought*. Oxford University Press.

Heine, S. J. (2017). *DNA is not destiny: The remarkable, completely misunderstood relationship between you and your genes*. Norton.

Heine, S. J., Dar-Nimrod, I., Cheung, B. Y., & Proulx, T. (2017). Essentially biased: Why people are fatalistic about genes. In J. Olson (Ed.), *Advances in experimental social psychology* (Vol. *55*, pp. 137–192). Academic Press.

Heine, S. J., & Hamamura, T. (2007). In search of East Asian self-enhancement. *Personality and Social Psychology Review*, *11*, 4–27.

Rothbart, M., & Taylor, M. (1992). Category labels and social reality: Do we view social categories as natural kinds? In G. Semin & K. Fiedler (Eds.), *Language, interaction and social cognition* (pp. 11–36). Sage.

Roy, M., Neale, M. C., & Kendler, K. S. (1995). The genetic epidemiology of self-esteem. *The British Journal of Psychiatry*, *166*, 813–820.

Schunkert, H., Konig, I. R., Kathiresan, S., Reilly, M. P., Assimes, T. L., Holm, H., … Samani, N. J. (2011). Large-scale association analysis identifies 13 new susceptibility loci for coronary artery disease. *Nature Genetics*, *43*, 333–338.

Wray, N. R., & Gottesman, I. I. (2012). Using summary data from the Danish national twin registers to estimate heritabilities for schizophrenia, bipolar disorder, and major depressive disorder. *Frontiers in Genetics*, *3*, 118.

From the trajectory of heritability to the heritability of trajectories

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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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References

Finkel, D., Davis, D. W., Turkheimer, E., & Dickens, W. T. (2015). Applying biometric growth curve models to developmental synchronies in cognitive development: The Louisville twin study. *Behavior Genetics*, *45*(6), 600–609. doi: 10.1007/s10519-015-9747-1

Finkel, D., & Pedersen, N. L. (2004). Processing speed and longitudinal trajectories of change for cognitive abilities: The Swedish adoption/twin study of aging. *Aging Neuropsychology and Cognition*, *11*(2–3), 325–345. doi: 10.1080/13825580490511152

Hart, S. A., Logan, J. A., Soden-Hensler, B., Kershaw, S., Taylor, J., & Schatschneider, C. (2013). Exploring how nature and nurture affect the development of reading: An analysis of the Florida twin project on reading. *Developmental Psychology*, *49*(10), 1971. doi: 10.1037/a0031348

Hatoum, A. S., Rhee, S. H., Corley, R. P., Hewitt, J. K., & Friedman, N. P. (2018). Etiology of stability and growth of internalizing and externalizing behavior problems across childhood and adolescence. *Behavior Genetics*, *48*(4), 298–314. doi: 10.1007/s10519-018-9900-8

Logan, J. A., Hart, S. A., Cutting, L., Deater-Deckard, K., Schatschneider, C., & Petrill, S. (2013). Reading development in young children: Genetic and environmental influences. *Child Development*, *84*(6), 2131–2144. doi: 10.1111/cdev.12104

Lyons, M. J., Panizzon, M. S., Liu, W., McKenzie, R., Bluestone, N. J., Grant, M. D., … Xian, H. (2017). A longitudinal twin study of general cognitive ability over four decades. *Developmental Psychology*, *53*(6), 1170. doi: 10.1037/dev0000303

Molenaar, P. C. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement*, *2*(4), 201–218. doi: 10.1207/s15366359mea0204_1

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Olsen, C. M., Pandeya, N., Law, M. H., MacGregor, S., Iles, M. M., & Thompson, B. S., … QSkin Study. (2020). Does polygenic risk influence associations between sun exposure and melanoma? A prospective cohort analysis. *British Journal of Dermatology*, *183*(2), 303–310. doi: 10.1111/bjd.18703

Reynolds, C. A., Finkel, D., Gatz, M., & Pedersen, N. L. (2002). Sources of influence on rate of cognitive change over time in Swedish twins: An application of latent growth models. *Experimental Aging Research*, *28*(4), 407–433. doi: 10.1080/03610730290103104

Going beyond heritability: Mechanisms of gene–culture coevolution

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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

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.

References

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*(6), 885–908. https://doi.org/10.1037/a0017376.

Chen, C., Burton, M., Greenberger, E., & Dmitrieva, J. (1999). Population migration and the variation of dopamine D4 receptor (DRD4) allele frequencies around the globe. *Evolution and Human Behavior*, *20*(5), 309–324. https://doi.org/10.1016/S1090-5138(99)00015-X.

Glazer, J., King, A., Yoon, C., Liberzon, I., & Kitayama, S. (2020). *DRD*4 polymorphisms modulate reward positivity and P3a in a gambling task: Exploring a genetic basis for cultural learning. *Psychophysiology*, *57*(10), e13623. https://doi.org/10.1111/psyp.13623.

Kitayama, S., King, A., Yoon, C., Tompson, S., Huff, S., & Liberzon, I. (2014). The dopamine D4 receptor gene (*DRD*4) moderates cultural difference in independent versus interdependent social orientation. *Psychological Science*, *25*(6), 1169–1177. https://doi.org/10.1177/0956797614528338.

Kitayama, S., Mesquita, B., & Karasawa, M. (2006). Cultural affordances and emotional experience: Socially engaging and disengaging emotions in Japan and the United States. *Journal of Personality and Social Psychology*, *91*(5), 890–903. https://doi.org/10.1037/0022-3514.91.5.890.

Kitayama, S., Yanagisawa, K., Ito, A., Ueda, R., Uchida, Y., & Abe, N. (2017). Reduced orbitofrontal cortical volume is associated with interdependent self-construal. *Proceedings of the National Academy of Sciences*, *114*(30), 7969–7974. https://doi.org/10.1073/pnas.1704831114.

Kitayama, S., & Yu, Q. (2020). Mutual constitution of culture and the mind: Insights from cultural neuroscience. In L. J. Kirmayer, C. M. Worthman, S. Kitayama, R. Lemelson, & C. Cummings (Eds.), *Culture, mind, and brain: Emerging concepts, models, and applications* (pp. 88–119). Cambridge University Press.

Kitayama, S., Yu, Q., King, A. P., Yoon, C., & Liberzon, I. (2020). The gray matter volume of the temporoparietal junction varies across cultures: A moderating role of the dopamine D4 receptor gene (DRD4). *Social Cognitive and Affective Neuroscience*, *15*(2), 193–202. https://doi.org/10.1093/scan/nsaa032.

Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, *98*(2), 224.

Matthews, L. J., & Butler, P. M. (2011). Novelty-seeking DRD4 polymorphisms are associated with human migration distance out-of-Africa after controlling for neutral population gene structure. *American Journal of Physical Anthropology*, *145*(3), 382–389. https://doi.org/10.1002/ajpa.21507.

Peng, Y., Shi, H., Qi, X., Xiao, C., Zhong, H., Ma, R. Z., & Su, B. (2010). The ADH1B Arg47His polymorphism in East Asian populations and expansion of rice domestication in history. *BMC Evolutionary Biology*, *10*(1), 15. https://doi.org/10.1186/1471-2148-10-15.

Talhelm, T., Zhang, X., Oishi, S., Shimin, C., Duan, D., Lan, X., & Kitayama, S. (2014). Large-scale psychological differences within China explained by rice versus wheat agriculture. *Science (New York, N.Y.)*, *344*(6184), 603–608. https://doi.org/10.1126/science.1246850.

Tishkoff, S. A., Reed, F. A., Ranciaro, A., Voight, B. F., Babbitt, C. C., Silverman, J. S., … Deloukas, P. (2007). Convergent adaptation of human lactase persistence in Africa and Europe. *Nature Genetics*, *39*(1), 31–40. https://doi.org/10.1038/ng1946.

Wu, S., & Keysar, B. (2007). The effect of culture on perspective taking. *Psychological Science*, *18*(7), 600–606. https://doi.org/10.1111/j.1467-9280.2007.01946.x.

Yu, Q., Abe, N., King, A., Yoon, C., Liberzon, I., & Kitayama, S. (2018). Cultural variation in the gray matter volume of the prefrontal cortex is moderated by the dopamine D4 receptor gene (DRD4). *Cerebral Cortex*, *29*(9), 3922–3931. https://doi.org/10.1093/cercor/bhy271.

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

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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

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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References

Chen, K.-H., Cavalli-Sforza, L. L., & Feldman, M. W. (1982). A study of cultural transmission in Taiwan. *Human Ecology*, *10*(3), 365–382.

Feldman, M. W., Christiansen, F. B., & Otto, S. P. (2013). Gene-culture co-evolution: Teaching, learning, and correlations between relatives. *Israel Journal of Ecology and Evolution*, *59*, 72–91.

Ihara, Y., & Feldman, M. W. (2004). Cultural niche construction and the evolution of small family size. *Theoretical Population Biology*, *65*, 105–111.

Odling-Smee, F. J., Laland, K. N., & Feldman, M. (2013). *W. Niche construction: The neglected process in evolution*. Princeton University Press.

Interpreting and reinterpreting heritability estimates in educational behavior genetics

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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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References

Addey, C. (2017). Golden relics & historical standards: How the OECD is expanding global education governance through PISA for development, *Critical Studies in Education*, *58*(3), 311–325. https://doi.org/10.1080/17508487.2017.1352006.

Asbury, K., & Plomin, R. (2014). *G is for genes: The impact of genetics on education and achievement*. Wiley Blackwell.

Asbury, K., & Wai, J. (2020). Viewing education policy through a genetic lens. *Journal of School Choice*, *14*(2), 301–315. https://doi.org/10.1080/15582159.2019.1705008.

Byrne, B., Little, C. W., Olson, R. K., Larsen, S. A., Coventry, W. L., & Weymouth, R. (2020). Comment on Asbury and WAI (2019), "viewing education policy through a genetic lens". *Journal of School Choice*, *14*(3), 501–515. https://doi.org/10.1080/15582159.2020.1779577.

Byrne, B., Olson, R. K., & Samuelsson, S. (2019). Behavior-genetic studies of academic performance in school students: A commentary for professionals in psychology and education. In D. Kilpatrick, R. Joshi & R. Wagner (eds), *Reading development and difficulties*, (pp. 213–232). Springer. https://doi.org/10.1007/978-3-030-26550-2_9.

Chow, B. W.-Y., Ho, C. S.-H., Wong, S. W.-L., Waye, M. M. Y., & Bishop, D. V. M. (2011). Genetic and environmental influences on Chinese language and reading abilities. *PLoS ONE*, *6*(2), e16640. https://doi.org/10.1371/journal.pone.0016640.

Coventry W. L., & Keller M. C. (2005). The extent of parameter bias in the classical twin design: A comparison of parameter estimates from the extended twin-family and classical twin designs. *Twin Research and Human Genetics*, *8*(3), 214–223. https://doi.org/10.1375/twin.8.3.214.

Harden, K. P. (2021). "Reports of my death were greatly exaggerated": Behavior genetics in the postgenomic era. *Annual Review of Psychology*, *72*, 37–60. https://doi.org/10.1146/annurev-psych-052220-103822.

Keller M. C., & Coventry W. L. (2005). Quantifying and addressing parameter indeterminacy in the classical twin design. *Twin Research and Human Genetics*, *8*(3), 201–213. https://doi.org/10.1375/twin.8.3.201.

Kovas, Y., Tikhomirova, T. N., Selita, F., Tosto, M. G., & Malykh, S. B. (2016). How genetics can help education. In Y. Kovas, S. B. Malykh, & D. Gaysina (Eds.), *Behavioural genetics for education* (pp. 1–23). Palgrave Macmillan.

Panofsky, A. (2015). What does behavioral genetics offer for improving education? *The Genetics of Intelligence: Ethics and the Conduct of Trustworthy Research*, (Hastings Center Report, 45, no. 5), S43–S49. https://doi.org/10.1002/hast.498.

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (6th ed.). Worth.

Rose, J. (2006). *Independent review of the teaching of early reading: Final report*. (Department for Education and Skills Report). Retrieved from https://dera.ioe.ac.uk/5551/.

Shero, J., van Dijk, W., Edwards, A., Schatschneider, C., Solari, E. J., & Hart, S. A. (2021). The practical utility of genetic screening in school settings. *Npj Science of Learning*, *6*(1), 1–10. https://doi.org/10.1038/s41539-021-00090-y.

Smith, G. D. (2011). Epidemiology, epigenetics and the "gloomy prospect": Embracing randomness in population health research and practice. *International Journal of Epidemiology*, *40*(3), 537–562. https://doi.org/10.1093/ije/dyr117.

Stanovich, P. J., & Stanovich, K. E. (2003). *Using research and reason in education*. (National Institute for Literacy Report). Retrieved from https://lincs.ed.gov/publications/pdf/Stanovich_Color.pdf.

Thomson, S., Hillman, K., Schmid, M., Rodrigues, S., & Fullarton, J. (2017). *Reporting Australia's Results: PIRLS* 2016. Camberwell, VIC: Australian Council for Educational Research. Retrieved from https://research.acer.edu.au/cgi/viewcontent.cgi?article=1001&context=pirls.

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Turkheimer, E. (2015), Genetic prediction. *Hastings Center Report*, *45*, S32–S38. https://doi.org/10.1002/hast.496.

Vadeboncoeur, J. A. (1997). Child development and the purpose of education: A historical context for constructivism in teacher education. In V. Richardson (Ed.), *Constructivist teacher education: Building New understandings* (pp. 15–37). The Falmer Press.

Woods, A. (2021). Are we there yet? Research with and for teachers and children and the possibilities of schooling in a complex world. *The Australian Educational Researcher*, *48*(1), 23–44. https://doi.org/10.1007/s13384-021-00431-1.

There is no such thing as culture-free intelligence

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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,

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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References

De Moor, M. H. M., Spector, T. D., Cherkas, L. F., Falchi, M., Hottenga, J. J., Boomsma, D. I., & De Geus, E. J. C. (2007). Genome-wide linkage scan for athlete status in 700 British female DZ twin pairs. *Twin Research and Human Genetics*, *10*(6), 812–820. https://doi.org/10.1375/twin.10.6.812.

DeDeo, S. (2018). IQ cults, nonlinearity, and reality: A bird-watcher's parable. *Axiom of Chance*. https://simondedeo.com/?p=337.

Feldman, M. W., & Ramachandran, S. (2018). Missing compared to what? Revisiting heritability, genes and culture. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1743), 20170064. https://doi.org/10.1098/rstb.2017.0064.

Hegarty, M., Montello, D. R., Richardson, A. E., Ishikawa, T., & Lovelace, K. (2006). Spatial abilities at different scales: Individual differences in aptitude-test performance and spatial-layout learning. *Intelligence*, *34*(2), 151–176. https://doi.org/10.1016/j.intell.2005.09.005.

Jensen, A. R. (1980). *Bias in mental testing*. Free Press.

Kan, K.-J., Wicherts, J. M., Dolan, C. V., & van der Maas, H. L. J. (2013). On the nature and nurture of intelligence and specific cognitive abilities: The more heritable, the more culture dependent. *Psychological Science*, *24*(12), 2420–2428. https://doi.org/10.1177/0956797613493292.

Perkins, J. M., Subramanian, S. V., Davey Smith, G., & Özaltin, E. (2016). Adult height, nutrition, and population health. *Nutrition Reviews*, *74*(3), 149–165. https://doi.org/10.1093/nutrit/nuv105.

Plomin, R., & Deary, I. J. (2015). Genetics and intelligence differences: Five special findings. *Molecular Psychiatry*, *20*(1), 98–108. https://doi.org/10.1038/mp.2014.105.

Richardson, K. (2002). What IQ tests test. *Theory & Psychology*, *12*(3), 283–314. https://doi.org/10.1177/0959354302012003012.

Ritchie, S. (2015). *Intelligence: All that matters* (1st ed.). John Murray.

Roebuck, H., & Lupyan, G. (under review). *Re-thinking non-verbal intelligence: The role of language*.

Rosselli, M., & Ardila, A. (2003). The impact of culture and education on non-verbal neuropsychological measurements: A critical review. *Brain and Cognition*, *52*(3), 326–333. https://doi.org/10.1016/s0278-2626(03)00170-2.

Stephenson, N. (2012). Arsebestos. In *Some remarks: Essays and other writing* (pp. 4–15). William Morrow.

Wilson, E. O. (2004). *On human nature: Twenty-fifth anniversary edition, with a new preface* (2nd ed.). Harvard University Press.

Wrangham, R. (2009). *Catching fire: How cooking made us human*. Basic Books.

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

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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.

References

Briley, D. A., Harden, K. P., & Tucker-Drob, E. M. (2015). Genotype × cohort interaction on completed fertility and age at first birth. *Behavior Genetics*, *45*(1), 71–83. https://doi.org/10.1007/s10519-014-9693-3

Engzell, P., & Tropf, F. C. (2019). Heritability of education rises with intergenerational mobility. *Proceedings of the National Academy of Sciences*, *116*(51), 25386–25388. https://doi.org/10.1073/pnas.1912998116

Fosse, E., & Winship, C. (2019). Analyzing age-period-cohort data: A review and critique. *Annual Review of Sociology*, *45*(1), 467–492. https://doi.org/10.1146/annurev-soc-073018-022616

Morris, D. (2020). Too early to declare a general law of social mobility and heritability for education. *Proceedings of the National Academy of Sciences*, *117*(46), 28564–28565. https://doi.org/10.1073/pnas.2011334117

Rosenquist, J. N., Lehrer, S. F., O'Malley, A. J., Zaslavsky, A. M., Smoller, J. W., & Christakis, N. A. (2015). Cohort of birth modifies the association between FTO genotype and BMI. *Proceedings of the National Academy of Sciences*, *112*(2), 354–359. https://doi.org/10.1073/pnas.1411893111

Sanz-de-Galdeano, A., Terskaya, A., & Upegui, A. (2020). Association of a genetic risk score with BMI along the life-cycle: Evidence from several US cohorts. *PLoS ONE*, *15*(9), e0239067. https://doi.org/10.1371/journal.pone.0239067

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

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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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References

Ayroles, J. F., Buchanan, S. M., O'Leary, C., Skutt-Kakaria, K., Grenier, J. K., Clark, A. G., … de Bivort, B. L. (2015). Behavioral idiosyncrasy reveals genetic control of phenotypic variability. *Proceedings of the National Academy of Sciences of the United States of America*, *112*(21), 6706–6711. doi: 10.1073/pnas.1503830112

Barlow, F. K. (2019). Nature vs. nurture is nonsense: On the necessity of an integrated genetic, social, developmental, and personality psychology. *Australian Journal of Psychology*, *71*, 68–79. https://doi.org/10.1111/ajpy.12240.

Briley, D. A., & Tucker-Drob, E. M. (2017). Comparing the developmental genetics of cognition and personality over the life span. *Journal of Personality*, *85*(1), 51–64. doi: 10.1111/jopy.12186

Freund, J., Brandmaier, A. M., Lewejohann, L., Kirste, I., Kritzler, M., Kruger, A., … Kempermann, G. (2013). Emergence of individuality in genetically identical mice. *Science (New York, N.Y.)*, *340*(6133), 756–759. https://doi.org/10.1126/science.1235294.

Harris, J. R. (1998). *The nurture assumption: Why children turn out the way they do*. Free Press.

Honegger, K., & de Bivort, B. (2018). Stochasticity, individuality and behavior. *Current Biology*, *28*(1), R8–R12. doi: 10.1016/j.cub.2017.11.058

Kan, K.-J., Ploeger, A., Raijmakers, M. E. J., Dolan, C. V., & van der Maas, H. L. J. (2010). Nonlinear epigenetic variance: Review and simulations. *Developmental Science*, *13*(1), 11–27. doi: 10.1111/j.1467-7687.2009.00858.x

Lehre, A. C., Lehre, K. P., Laake, P., & Danbolt, N. C. (2009). Greater intrasex phenotype variability in males than in females is a fundamental aspect of the gender differences in humans. *Developmental Psychobiology*, *51*(2), 198–206. doi: 10.1002/dev.20358

Linneweber, G. A., Andriatsilavo, M., Dutta, S. B., Bengochea, M., Hellbruegge, L., Liu, G., … Hassan, B. A. (2020). A neurodevelopmental origin of behavioral individuality in the drosophila visual system. *Science (New York, N.Y.)*, *367*(6482), 1112–1119. doi: 10.1126/science.aaw7182

Mitchell, K. J. (2007). The genetics of brain wiring: From molecule to mind. *PLoS Biology*, *5*(4), e113.

Mitchell, K. J. (2018). *Innate – How the wiring of our brains shapes who we are*. Princeton University Press.

Raj, A., & van Oudenaarden, A. (2008). Nature, nurture, or chance: Stochastic gene expression and its consequences. *Cell*, *135*(2), 216–226. doi: 10.1016/j.cell.2008.09.050

Reinhold, K., & Engqvist, L. (2013). The variability is in the sex chromosomes. *Evolution*, *67*(12), 3662–3668. doi: 10.1111/evo.12224

Vogt, G. (2015). Stochastic developmental variation, an epigenetic source of phenotypic diversity with far-reaching biological consequences. *Journal of Biosciences*, *40*(2015), 159–204.

Waddington, C. (1957). *The strategy of the genes*. Routledge.

Wagner, A. (2007). *Robustness and evolvability in living systems*. Princeton University Press.

Wright, S. (1920). The relative importance of heredity and environment in determining the piebald pattern of Guinea-pigs. *Proceedings of the National Academy of Sciences of the United States of America*, *6*(6), 320–332. doi: 10.1073/pnas.6.6.320

Genetic solutions to cultural problems?

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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

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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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References

Apicella, C., Norenzayan, A., & Henrich, J. (2020). Beyond WEIRD: A review of the last decade and a look ahead to the global laboratory of the future. *Evolution and Human Behavior*, *41*(5), 319–329. https://doi.org/10.1016/j.evolhumbehav.2020.07.015.

Buss, D. M. (1995). Evolutionary psychology: A new paradigm for psychological science. *Psychological Inquiry*, *6*, 1–30. https://doi.org/10.1207/s15327965pli0601_1.

Buss, D. M. (2020). Evolutionary psychology is a scientific revolution. *Evolutionary Behavioral Sciences*, *14*(4), 316–323. https://doi.org/10.1037/ebs0000210.

Jablonka, E., & Lamb, M. J. (2014). *Evolution in four dimensions: Genetic, epigenetic, behavioral, and symbolic variation in the history of life* (2nd ed.). MIT Press.

Hall, B. K. (2012). Evolutionary developmental biology (evo-devo): Past, present, and future. *Evolution: Education and Outreach*, *5*, 184–193. https://doi.org/10.1007/s12052-012-0418-x.

Pinker, S. (2002). *The blank slate*: *The modern denial of human nature*. Penguin.

Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality*, *58*(1), 17–67. https://doi.org/10.1111/j.1467-6494.1990.tb00907.x.

Tooby, J., & Cosmides, L. (1992). The psychological foundations of culture. In J. Barkow, L. Cosmides, & J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 19–136). Oxford University Press.

Witherington, D. E., & Lickliter, R. (2016). Integrating development and evolution in psychological science: Evolutionary developmental psychology, developmental systems, and explanatory pluralism. *Human Development*, *59*(4), 200–234. https://doi.org/10.2307/26765140.

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

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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

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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References

Beach, S. R. H., Lei, M. K., Brody, G. H., & Philibert, R. A. (2018). Prevention of early substance use mediates, and variation at *SLC6A4* moderates, SAAF intervention effects on *OXTR* methylation. *Prevention Science*, *19*, 90–100.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., … Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science (New York, N.Y.)*, *301*(5631), 386–389.

Feldman, R., Monakhov, M., Pratt, & Ebstein, R. P. (2016). Oxytocin pathway genes: Evolutionary ancient system impacting on human affiliation, sociality, and psychopathology. *Biological Psychiatry*, *79*, 174–184.

Frodl, T., Szyf, M., Carballedo, A., Ly, V., Dymov, S., Varisheva, F., … Booij, L. (2015). DNA Methylation of the serotonin transporter gene (SLC6A4) is associated with brain function involved in processing emotional stimuli. *Journal of Psychiatry Neuroscience*, *40*(5), 296–305. doi: 10.1503/jpn.140180

Iurescia, S., Seriipa, D., & Rinaldi, M. (2016). Looking beyond the 5-HTTLPR polymorphism: Genetic and epigenetic layers of regulation affecting the serotonin transporter gene expression. *Molecular Neurobiology*, *54*, 8336–8403.

Keverne, E. B. (2014). Significance of epigenetics for understanding brain development, brain evolution and behaviour. *Neuroscience*, *264*, 207–217.

Kumsta, R., Hummel, E., Chen, F. S., & Heinrichs, M. (2013). Epigenetic regulation of the oxytocin receptor gene: Implications for behavioural neuroscience. *Frontiers in Neuroendocrine Science*, *7*: 83. doi: 10.3389/fnins.2013.00083

Luo, S., & Han, S. (2014). The association between an oxytocin receptor gene polymorphism and cultural orientations. *Culture and Brain*, *2*, 89. doi: DOI.org/10.1007/s40167-014-0017-5

Minkov, M., Blagoev, V., & Bond, M. H. (2015). Improving research in the emerging field of cross-cultural sociogenetics: The case of serotonin, *Journal of Cross-Cultural Psychology*, *46*(3), 336–354.

Riedl, R., & Javor, A. (2012). The biology of trust: Integrating evidence from genetics, endocrinology, and functional brain imaging. *Journal of Neuroscience, Psychology and Economics*, *5*(2), 63–91.

Way, B. M., & Lieberman, M. D. (2010). Is there a genetic contribution to cultural differences? Collectivism, individualism and genetic markers of social sensitivity. *Social Cognitive and Affective Neuroscience*, *5*, 203–211.

Measuring heritability: Why bother?

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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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References

Blows, M. W., & Hoffmann, A. A. (2005). A reassessment of genetic limits to evolutionary change. *Ecology*, *86*, 1371–1384.

Charmantier, A., Garant, D., & Kruuk, L. E. (Eds.). (2014). *Quantitative genetics in the wild*. OUP.

Dochtermann, N. A., Schwab, T., Anderson Berdal, M., Dalos, J., & Royauté, R. (2019). The heritability of behavior: A meta-analysis. *Journal of Heredity*, *110*, 403–410.

Falconer, D. S., & Mackay, T. F. C. (1996). *Introduction to quantitative genetics* (4th Ed.). Longmans Green.

Hansen, T. F., Pélabon, C., & Houle, D. (2011). Heritability is not evolvability. *Evolutionary Biology*, *38*, 258–277.

Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics*, *130*, 195–204.

Lynch, M., & Walsh, B. (1998). *Genetics and analysis of quantitative traits*. OUP.

Mathieson, I. (2021). The omnigenic model and polygenic prediction of complex traits. *The American Journal of Human Genetics*, *108*, 1558–1563.

Mousseau, T. A., & Roff, D. A. (1987). Natural selection and the heritability of fitness components. *Heredity*, *59*, 181–197.

Stirling, D. G., Réale, D., & Roff, D. A. (2002). Selection, structure and the heritability of behaviour. *Journal of Evolutionary Biology*, *15*, 277–289.

Visscher, P. M., Hill, W. G., & Wray, N. R. (2008). Heritability in the genomics era – Concepts and misconceptions. *Nature Reviews Genetics*, *9*, 255–266.

Weigensberg, I., & Roff, D. A. (1996). Natural heritabilities: Can they be reliably estimated in the laboratory? *Evolution*, *50*, 2149–2157.

Wilson, A. J., Kruuk, L. E., & Coltman, D. W. (2005). Ontogenetic patterns in heritable variation for body size: Using random regression models in a wild ungulate population. *The American Naturalist*, *166*, E177–E192.

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

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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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References

Alford, J. R., Funk, C. L., & Hibbing, J. R. (2005). Are political orientations genetically transmitted?. *American Political Science Review*, *99*(2), 153–167. doi:10.1017/S0003055405051579

Brewer, P., & Venaik, S. (2014). The ecological fallacy in national culture research. *Organization Studies 35*(7), 1063–1086. doi:10.1177/0170840613517602

DeYoung, C. G. (2015). Cybernetic Big Five theory. *Journal of Research in Personality*, *56*, 33–58. doi:10.1016/j.jrp.2014.07.004

Gelfand, M. J., Raver, J. L., Nishii, L., Leslie, L. M., Lun, J., Lim, B. C., … Yamaguchi, S. (2011). Differences between tight and loose cultures: A 33-nation study. *Science*, *332* (6033), 1100–1104. doi:10.1126/science.1197754

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world? *Behavioral and Brain Sciences*, *33*(2–3), 61–83. doi:10.1017/S0140525X0999152X

Hofstede, G. (1980). *Culture's consequences: International differences in work-related values*. Sage.

Kitayama, S., & Park, J. (2021). Is conscientiousness always associated with better health? A U.S.–Japan cross-cultural examination of biological health risk. *Personality and Social Psychology Bulletin*, *47*(3), 486–498. doi:10.1177/0146167220929824

McAdams, D. P., & Pals, J. L. (2006). A new big five: Fundamental principles for an integrative science of personality. *American Psychologist*, *61*(3), 204–217. doi:10.1037/0003-066X.61.3.204

Muthukrishna, M., Bell, A. V., Henrich, J., Curtin, C. M., Gedranovich, A., McInerney, J., & Thue, B. (2020). Beyond western, educated, industrial, rich, and democratic (WEIRD) psychology: Measuring and mapping scales of cultural and psychological distance. *Psychological Science*, *31*(6), 678–701. doi:10.1177/0956797620916782

Nguyen, P. L. L., Syed, M., & McGue, M. (2021). Behavior genetics research on personality: Moving beyond traits to examine characteristic adaptations. *Social and Personality Psychology Compass*, *15*, e12628. doi:10.1111/spc3.12628.

Tam, V., Patel, N., Turcotte, M., Bossé, Y., Paré, G., & Meyre, D. (2019). Benefits and limitations of genome-wide association studies. *Nature Reviews Genetics*, *20*(8), 467–484. doi:10.1038/s41576-019-0127-1

Triandis, H. C. (1996). The psychological measurement of cultural syndromes. *American Psychologist*, *51*(4), 407–415. doi:10.1037/0003-066X.51.4.407

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

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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

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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References

Bouchard, Jr, T. J. (2004). Genetic influence on human psychological traits: A survey. *Current Directions in Psychological Science*, *13*(4), 148–151.

Ganna, A., Verweij, K. J., Nivard, M. G., Maier, R., Wedow, R., Busch, A. S., … Zietsch, B. P. (2019). Large-scale GWAS reveals insights into the genetic architecture of same-sex sexual behavior. *Science*, *365*, eaat7693. doi: 10.1126/science.aat7693.

Plomin, R., & Bergeman, C. S. (1991). The nature of nurture: Genetic influence on "environmental" measures. *Behavioral and Brain Sciences*, *14*(3), 373–427.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*(5), 160–164.

Turkheimer, E., & Gottesman, I. I. (1991). Is $H^2 = 0$ a null hypothesis anymore?. *Behavioral and Brain Sciences*, *14*(3), 410–411.

Hidden clusters beyond ethnic boundaries

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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

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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References

Barth, F. (1969). *Ethnic groups and boundaries: The social organization of culture difference*. Little, Brown.

Biswas, U. N., & Pandey, J. (1996). Mobility and perception of socioeconomic status among tribal and caste group. *Journal of Cross-Cultural Psychology*, *27*(2), 200–215.

Bourdieu, P. (1987). *Distinction: A social critique of the judgement of taste*. Harvard University Press.

Colleran, H. (2020). Market integration reduces kin density in women's ego-networks in rural Poland. *Nature Communications*, *11*, 266.

Henrich, J., Heine, S., & Norenzayan, A. (2010). Most people are not WEIRD. *Nature 466*, 29. https://doi.org/10.1038/466029a.

McElreath, R., Boyd, R., & Richerson, P. (2003). Shared norms and the evolution of ethnic markers. *Current Anthropology*, *44*(1), 122–130.

Richerson, P. J., & Boyd, R. (2008). *Not by genes alone: How culture transformed human evolution*. University of Chicago Press.

Schooler, C. (2010). Culture and social structure: The relevance of social structure to cultural psychology. In D. Cohen (Ed.), *Handbook of cultural psychology* (pp. 370–388). Guilford.

Schulz, J. F., Bahrami-Rad, D., Beauchamp, J. P., & Henrich, J. (2019). The church, intensive kinship, and global psychological variation. *Science*, *366*, eaau5141.

Smaldino, P. E. (2019). Social identity and cooperation in cultural evolution. *Behavioural Processes*, *161*, 108–116.

Smaldino, P. E., & Turner, M. A. (2021). Covert signaling is an adaptive communication strategy in diverse populations. *Psychological Review*, *129*(4), 812–829. https://doi.org/10.1037/rev0000344.

Culture is reducing genetic heritability and superseding genetic adaptation

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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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References

Baldini, R. (2012). Success-biased social learning: Cultural and evolutionary dynamics. *Theoretical Population Biology*, *82*(3), 222–228.

Crespi, B. J. (2004). Vicious circles: Positive feedback in major evolutionary and ecological transitions. *Trends in Ecology & Evolution*, *19*(12), 627–633. https://doi.org/10.1016/j.tree.2004.10.001.

Gerbault, P., Liebert, A., Itan, Y., Powell, A., Currat, M., Burger, J., … Thomas, M. G. (2011). Evolution of lactase persistence: An example of human niche construction. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *366*(1566), 863–877. https://doi.org/10.1098/rstb.2010.0268.

Henrich, J., & Boyd, R. (1998). The evolution of conformist transmission and the emergence of between-group differences. *Evolution and Human Behavior*, *19*(4), 215–241.

Henrich, J., & Gil-White, F. J. (2001). The evolution of prestige – Freely conferred deference as a mechanism for enhancing the benefits of cultural transmission. *Evolution and Human Behavior*, *22*(3), 165–196.

Henrich, J., & McElreath, R. (2003). The evolution of cultural evolution. *Evolutionary Anthropology: Issues, News, and Reviews*, *12*(3), 123–135. https://doi.org/10.1002/evan.10110.

Hong, Z. (2020). Modelling the on-going natural selection of educational attainment in contemporary societies. *Journal of Theoretical Biology*, *493*, 110210. https://doi.org/10.1016/j.jtbi.2020.110210.

Laland, K. N. (1992). A theoretical investigation of the role of social transmission in evolution. *Ethology and Sociobiology*, *13*(2), 87–113. https://doi.org/10.1016/0162-3095(92)90020-5.

Lush, J. L. (1943). Animal breeding plans. *Animal Breeding Plans, Edn 2.*

Mathew, S., & Perreault, C. (2015). Behavioural variation in 172 small-scale societies indicates that social learning is the main mode of human adaptation. *Proceedings of the Royal Society B: Biological Sciences*, *282*(1810), 20150061. https://doi.org/10.1098/rspb.2015.0061.

Smaldino, P. E. (2014). The cultural evolution of emergent group-level traits. *Behavioral and Brain Sciences*, *37*(03), 243–254. https://doi.org/10.1017/S0140525X13001544.

Waring, T. M., & Wood, Z. T. (2021). Long-term gene–culture coevolution and the human evolutionary transition. *Proceedings of the Royal Society B: Biological Sciences*, *288*(1952), 20210538. https://doi.org/10.1098/rspb.2021.0538.

Understanding cultural clusters: An ethnographic perspective

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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

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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References

Ballard, C., Brown, P., Bourke, R. M., & Harwood, T. (Eds.). (2005). *The sweet potato in Oceania: A reappraisal* (No. 19). University of Sydney.

Barth, F. (1998). *Ethnic groups and boundaries: The social organization of culture difference*. Waveland Press.

Boehm, C. (2012). *Moral origins: The evolution of virtue, altruism, and shame*. Soft Skull Press.

Hrdy, S. B. (2016). Development plus social selection in the emergence of "emotionally modern" humans. In C. L. Meehan & A. N. Crittenden (Eds.), *Childhood: Origins, Evolution, and Implications* (pp. 11–44). School for Advanced Research Press.

Kelly, R. C. (1974). *Etoro social structure: A study in structural contradiction*. University of Michigan.

Lüpke F, Stenzel K, Cabalzar FD, Chacon T, da Cruz A, Franchetto B, … Storto L. (2020). Comparing rural multilingualism in Lowland South America and Western Africa. *Anthropological Linguistics*. https://tuhat.helsinki.fi/ws/files/160501626/Comparing_multilingualism_SA_WA_FINAL2_FL.pdf.

Nesse, R. M. (2007). Runaway social selection for displays of partner value and altruism. *Biological Theory*, *2*(2), 143–155.

Nesse, R. M. (2016). Social selection is a powerful explanation for prosociality. *Brain*, *39*, e47.

Richerson, P., Baldini, R., Bell, A. V., Demps, K., Frost, K., Hillis, V., … Zefferman, M. (2016). Cultural group selection plays an essential role in explaining human cooperation: A sketch of the evidence. *Behavioral and Brain Sciences*, *39*, e58.

Summers, B., Lategan R., Rifkin, F., d'Errico, F., & Garcia-Moreno, R. (nd.) Sun protection from ochre compounds used traditionally by southern African indigenous populations. *Sciences*, *6*(33), 7577–7566. https://www.researchgate.net/profile/Riaan-Rifkin/publication/264897272_Sun_protection_from_ochre_used_traditionally_by_southern_African_indigenous_populations/links/53f4e22c0cf22be01c3ef196/Sun-protection-from-ochre-used-traditionally-by-southern-African-indigenous-populations.pdf.

West-Eberhard, M. J. (1979). Sexual selection, social competition, and evolution. *Proceedings of the American Philosophical Society*, *123*(4), 222–234.

Wiessner, P. (2020). The role of third parties in norm enforcement in customary courts among the Enga of Papua New Guinea. *Proceedings of the National Academy of Sciences*, *117*(51), 32320–32328.

Wiessner, P., & Tumu, A. (1998). *Historical vines*: Enga networks of exchange, ritual and warfare. Smithsonian Inst Press.

Wrangham, R. W. (2019). Hypotheses for the evolution of reduced reactive aggression in the context of human self-domestication. *Frontiers in Psychology*, *10*, 1914.

Cultural evolution may influence heritability by shaping assortative mating

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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.,

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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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References

Amin, V., Böckerman, P., Viinikainen, J., Smart, M. C., Bao, Y., Kumari, M., … Pehkonen, J. (2017). Gene–environment interactions between education and body mass: Evidence from the UK and Finland. *Social Science & Medicine*, *195*, 12–16.

Ancestry.com. (September 7, 2020). Does Distance Make the Heart Grow Fonder? Ancestry Blog. blogs.ancestry.com/cm/does-distance-make-the-heart-grow-fonder/. Archived at https://archive.li/nMtBQ.

Bansal, V., Mitjans, M., Burik, C. A., Linner, R. K., Okbay, A., Rietveld, C. A., … Koellinger, P. D. (2018). Genome-wide association study results for educational attainment aid in identifying genetic heterogeneity of schizophrenia. *Nature Communications*, *9*(1), 1–12.

Breen, R., & Andersen, S. H. (2012). Educational assortative mating and income inequality in Denmark. *Demography*, *49*(3), 867–887.

Cavalli-Sforza, L. L., & Feldman, M. W. (1973). Cultural versus biological inheritance: Phenotypic transmission from parents to children. (A theory of the effect of parental phenotypes on children's phenotypes). *American Journal of Human Genetics*, *25*(6), 618.

Daysal, N. M., Elder, T. E., Hellerstein, J. K., Imberman, S. A., & Orsini, C. (2021). Parental Skills, Assortative Mating, and the Incidence of Autism Spectrum Disorder.

Demange, P. A., Malanchini, M., Mallard, T. T., Biroli, P., Cox, S. R., Grotzinger, A. D., … Nivard, M. G. (2021). Investigating the genetic architecture of noncognitive skills using GWAS-by-subtraction. *Nature Genetics*, *53*(1), 35–44.

Dickens, W. T., & Flynn, J. R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review*, *108*(2), 346.

Eika, L., Mogstad, M., & Zafar, B. (2019). Educational assortative mating and household income inequality. *Journal of Political Economy*, *127*(6), 2795–2835.

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Gauderman, W. J., Mukherjee, B., Aschard, H., Hsu, L., Lewinger, J. P., Patel, C. J., … Chatterjee, N. (2017). Update on the state of the science for analytical methods for gene–environment interactions. *American Journal of Epidemiology*, *186*(7), 762–770.
Greenwood, J., Guner, N., Kocharkov, G., & Santos, C. (2014). Marry your like: Assortative mating and income inequality. *American Economic Review*, *104*(5), 348–353.
Henrich, J., & McElreath, R. (2007). Dual-inheritance theory: The evolution of human cultural capacities and cultural evolution. In R. Dunbar & L. Barrett (Eds.), *Oxford handbook of evolutionary psychology* (pp. 555–570). Oxford University Press.
Keller, M. C., Garver-Apgar, C. E., Wright, M. J., Martin, N. G., Corley, R. P., Stallings, M. C., … Zietsch, B. P. (2013). The genetic correlation between height and IQ: Shared genes or assortative mating?. *PLoS Genetics*, *9*(4), e1003451.
Laland, K. N., Brown, G. R., & Brown, G. (2011). *Sense and nonsense: Evolutionary perspectives on human behaviour*. Oxford University Press.
Maas, I., & Zijdeman, R. L. (2010). Beyond the local marriage market: The influence of modernization on geographical heterogamy. *Demographic Research*, *23*, 933–962.
MacCabe, J. H., Sariaslan, A., Almqvist, C., Lichtenstein, P., Larsson, H., & Kyaga, S. (2018). Artistic creativity and risk for schizophrenia, bipolar disorder and unipolar depression: A Swedish population-based case–control study and sib-pair analysis. *The British Journal of Psychiatry*, *212*(6), 370–376.
Nordsletten, A. E., Larsson, H., Crowley, J. J., Almqvist, C., Lichtenstein, P., & Mataix-Cols, D. (2016). Patterns of nonrandom mating within and across 11 major psychiatric disorders. *JAMA Psychiatry*, *73*(4), 354–361.
Parnas, J., Sandsten, K. E., Vestergaard, C. H., & Nordgaard, J. (2019). Schizophrenia and bipolar illness in the relatives of university scientists: An epidemiological report on the creativity-psychopathology relationship. *Frontiers in Psychiatry*, *10*, 175.
Peyrot, W. J., Robinson, M. R., Penninx, B. W., & Wray, N. R. (2016). Exploring boundaries for the genetic consequences of assortative mating for psychiatric traits. *JAMA Psychiatry*, *73*(11), 1189–1195.
Power, R. A., Steinberg, S., Bjornsdottir, G., Rietveld, C. A., Abdellaoui, A., Nivard, M. M., … Stefansson, K. (2015). Polygenic risk scores for schizophrenia and bipolar disorder predict creativity. *Nature Neuroscience*, *18*(7), 953–955.
Rask-Andersen, M., Karlsson, T., Ek, W. E., & Johansson, Å. (2017). Gene–environment interaction study for BMI reveals interactions between genetic factors and physical activity, alcohol consumption and socioeconomic status. *PLoS Genetics*, *13*(9), e1006977.
Roelfsema, M. T., Hoekstra, R. A., Allison, C., Wheelwright, S., Brayne, C., Matthews, F. E., & Baron-Cohen, S. (2012). Are autism spectrum conditions more prevalent in an information-technology region? A school-based study of three regions in the Netherlands. *Journal of Autism and Developmental Disorders*, *42*(5), 734–739.
Rosenfeld, M. J., Thomas, R. J., & Hausen, S. (2019). Disintermediating your friends: How online dating in the United States displaces other ways of meeting. *Proceedings of the National Academy of Sciences*, *116*(36), 17753–17758.
Ruzich, E., Allison, C., Chakrabarti, B., Smith, P., Musto, H., Ring, H., & Baron-Cohen, S. (2015). Sex and STEM occupation predict autism-spectrum quotient (AQ) scores in half a million people. *PLoS ONE*, *10*(10), e0141229.
Wild, C. P. (2012). The exposome: From concept to utility. *International Journal of Epidemiology*, *41*(1), 24–32.

Authors' Response

Integrating cultural evolution and behavioral genetics

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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.

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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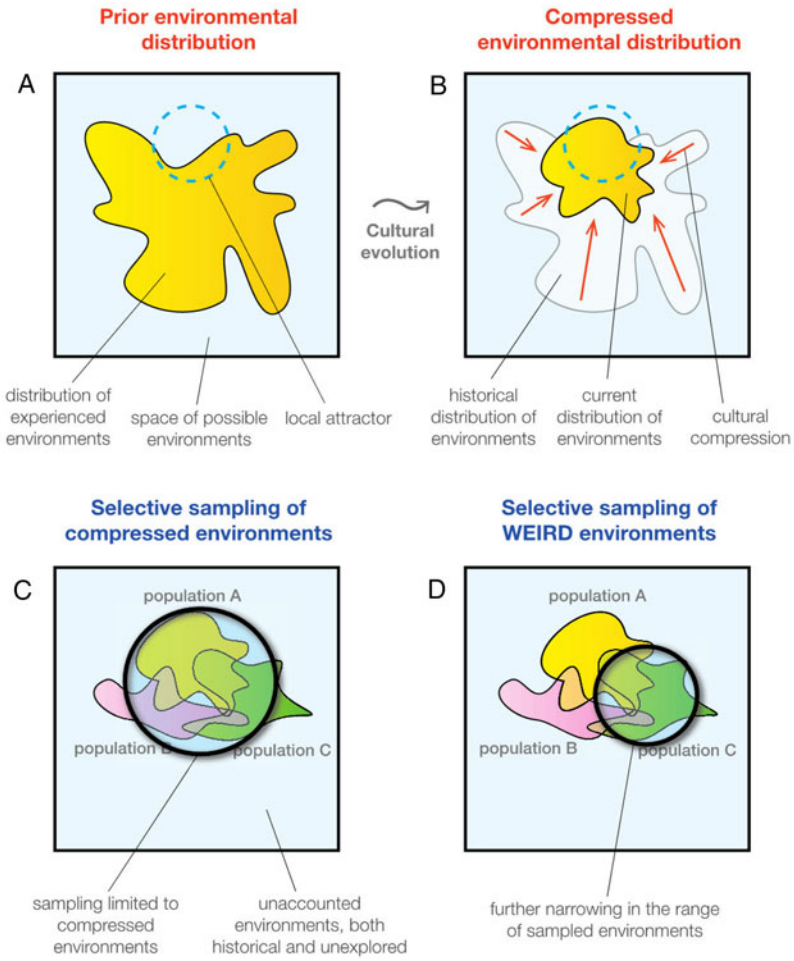

Figure R1 (Uchiyama et al.). Restricted sampling of environments due to historical trajectories of cultural evolution and selective (WEIRD) sampling. (a) The yellow region represents the distribution of experienced environments of a hypothetical society at a past time point. The unoccupied light blue 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.

References

Adams, M. J. (1994). *Beginning to read: Thinking and learning about print*. MIT press.

Alesina, A., Giuliano, P., & Nunn, N. (2013). On the origins of gender roles: Women and the plough. *The Quarterly Journal of Economics*, *128*(2), 469–530. https://doi.org/10/gctrzs.

Apicella, C. L., Norenzayan, A., & Henrich, J. (2020). Beyond WEIRD: A review of the last decade and a look ahead to the global laboratory of the future. *Evolution and Human Behavior*, *41*(5), 319–329. https://doi.org/10/ghndmz.

Arnold, S. J., Bürger, R., Hohenlohe, P. A., Ajie, B. C., & Jones, A. G. (2008). Understanding the evolution and stability of the G-Matrix. *Evolution*, *62*(10), 2451–2461. https://doi.org/10.1111/j.1558-5646.2008.00472.x.

Barsbai, T., Lukas, D., & Pondorfer, A. (2021). Local convergence of behavior across species. *Science (New York, N.Y.)*, *371*(6526), 292–295. https://doi.org/10/ghxt79.

Bates, T. C., Lewis, G. J., & Weiss, A. (2013). Childhood socioeconomic status amplifies genetic effects on adult intelligence. *Psychological Science*, *24*(10), 2111–2116. https://doi.org/10.1177/0956797613488394.

Beauchamp, J. P. (2016). Genetic evidence for natural selection in humans in the contemporary United States. *Proceedings of the National Academy of Sciences*, *113* (28), 7774–7779. https://doi.org/10.1073/pnas.1600398113.

Bell, A., Chetty, R., Jaravel, X., Petkova, N., & Van Reenen, J. (2019). Who becomes an inventor in America? The importance of exposure to innovation. *The Quarterly Journal of Economics*, *134*(2), 647–713. https://doi.org/10.1093/qje/qjy028.

Bennett, T. (2015). Cultural studies and the culture concept. *Cultural Studies*, *29*(4), 546–568. https://doi.org/10.1080/09502386.2014.1000605.

Blair, C., Gamson, D., Thorne, S., & Baker, D. (2005). Rising mean IQ: Cognitive demand of mathematics education for young children, population exposure to formal schooling, and the neurobiology of the prefrontal cortex. *Intelligence*, *33*(1), 93–106.

Border, R., Johnson, E. C., Evans, L. M., Smolen, A., Berley, N., Sullivan, P. F., & Keller, M. C. (2019). No support for historical candidate gene or candidate gene-by-interaction hypotheses for major depression across multiple large samples. *American Journal of Psychiatry*, *176*(5), 376–387. https://doi.org/10/gfwnhp.

Boyd, R., Richerson, P. J., & Henrich, J. (2011). The cultural niche: Why social learning is essential for human adaptation. *Proceedings of the National Academy of Sciences*, *108* (Supplement 2), 10918–10925. https://doi.org/10.1073/pnas.1100290108.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, *101*(4), 568–586.

Buss, D. M. (1995). Evolutionary psychology: A new paradigm for psychological science. *Psychological Inquiry*, *6*(1), 1–30. https://doi.org/10.1207/s15327965pli0601_1.

Cain, K., & Oakhill, J. (2009). Reading comprehension development from 8 to 14 years: The contribution of component skills and processes. In R. K. Wagner, C. Schatschneider, & C. Phythian-Sence (Eds.), *Beyond decoding: The behavioral and biological foundations of reading comprehension* (pp. 143–175). Guilford Press.

Cavalli-Sforza, L. L., & Feldman, M. W. (1973). Cultural versus biological inheritance: Phenotypic transmission from parents to children. (A theory of the effect of parental phenotypes on children's phenotypes). *American Journal of Human Genetics*, *25*(6), 618–637.

Chetty, R., Friedman, J. N., Hilger, N., Saez, E., Schanzenbach, D. W., & Yagan, D. (2011). How does your kindergarten classroom affect your earnings? Evidence from Project STAR. *The Quarterly Journal of Economics*, *126*(4), 1593–1660.

Christiansen, M. H., & Chater, N. (2008). Language as shaped by the brain. *Behavioral and Brain Sciences*, *31*(5), 498–508. https://doi.org/10.1017/S0140525X08004998.

Cooper, R. M., & Zubek, J. P. (1958). Effects of enriched and restricted early environments on the learning ability of bright and dull rats. *Canadian Journal of Psychology/Revue Canadienne de Psychologie*, *12*(3), 159–164. https://doi.org/10.1037/h0083747.

Crow, J. F. (2010). On epistasis: Why it is unimportant in polygenic directional selection. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *365*(1544), 1241–1244. https://doi.org/10.1098/rstb.2009.0275.

Culverhouse, R. C., Saccone, N. L., Horton, A. C., Ma, Y., Anstey, K. J., Banaschewski, T., … Bierut, L. J. (2018). Collaborative meta-analysis finds no evidence of a strong interaction between stress and 5-HTTLPR genotype contributing to the development of depression. *Molecular Psychiatry*, *23*(1), 133–142. https://doi.org/10/f9zprm.

Dehaene, S., & Cohen, L. (2007). Cultural recycling of cortical maps. *Neuron*, *56*(2), 384–398. https://doi.org/10.1016/j.neuron.2007.10.004.

Dickens, W. T., & Flynn, J. R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review*, *108*(2), 346.

Engelhardt, L. E., Briley, D. A., Mann, F. D., Harden, K. P., & Tucker-Drob, E. M. (2015). Genes unite executive functions in childhood. *Psychological Science*, *26*(8), 1151–1163. https://doi.org/10.1177/0956797615577209.

Falk, A., & Hermle, J. (2018). Relationship of gender differences in preferences to economic development and gender equality. *Science (New York, N.Y.)*, *362*(6412), eaas9899. https://doi.org/10.1126/science.aas9899.

Fan, S., Hansen, M. E. B., Lo, Y., & Tishkoff, S. A. (2016). Going global by adapting local: A review of recent human adaptation. *Science (New York, N.Y.)*, *354*(6308), 54–59. https://doi.org/10.1126/science.aaf5098.

Fosse, E., & Winship, C. (2019). Analyzing age-period-cohort data: A review and critique. *Annual Review of Sociology*, *45*(1), 467–492. https://doi.org/10/gf6rv7.

Fried, E. I. (2021). *Studying mental health disorders as systems, not syndromes*. PsyArXiv. doi.org/10.31234/osf.io/k4mhv

Friedman, N. P., Miyake, A., Young, S. E., DeFries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual differences in executive functions are almost entirely genetic in

https://doi.org/10.1017/S0140525X21000893 Published online by Cambridge University Press

origin. *Journal of Experimental Psychology: General*, *137*(2), 201–225. https://doi.org/10.1037/0096-3445.137.2.201.

Galanter, J. M., Gignoux, C. R., Oh, S. S., Torgerson, D., Pino-Yanes, M., Thakur, N., … Zaitlen, N. (2017). Differential methylation between ethnic sub-groups reflects the effect of genetic ancestry and environmental exposures. *ELife*, *6*, e20532. https://doi.org/10/gmh22v.

Gurven, M., von Rueden, C., Massenkoff, M., Kaplan, H., & Lero Vie, M. (2013). How universal is the Big Five? Testing the five-factor model of personality variation among forager–farmers in the Bolivian Amazon. *Journal of Personality and Social Psychology*, *104*(2), 354–370. https://doi.org/10.1037/a0030841.

Hanscombe, K. B., Trzaskowski, M., Haworth, C. M. A., Davis, O. S. P., Dale, P. S., & Plomin, R. (2012). Socioeconomic status (SES) and children's intelligence (IQ): In a UK-representative sample SES moderates the environmental, not genetic, effect on IQ. *PLoS ONE*, *7*(2), e30320. https://doi.org/10.1371/journal.pone.0030320.

Hasson, U., Chen, J., & Honey, C. J. (2015). Hierarchical process memory: Memory as an integral component of information processing. *Trends in Cognitive Sciences*, *19*(6), 304–313. https://doi.org/10.1016/j.tics.2015.04.006.

Heine, S. J. (2017). *DNA is not destiny: The remarkable, completely misunderstood relationship between you and your genes*. Norton.

Henrich, J. (2016). *The secret of our success: How culture is driving human evolution, domesticating our species, and making us smarter*. Princeton University Press.

Henrich, J. (2020). *The WEIRDest people in the world: How the West became psychologically peculiar and particularly prosperous*. Farrar, Straus and Giroux.

Henrich, J., Boyd, R., & Richerson, P. J. (2008). Five misunderstandings about cultural evolution. *Human Nature*, *19*(2), 119–137. https://doi.org/10/ffwks9.

Henrich, J., Chudek, M., & Boyd, R. (2015). The Big Man Mechanism: How prestige fosters cooperation and creates prosocial leaders. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1683), 20150013. https://doi.org/10/gfzktb.

Henrich, J., & Muthukrishna, M. (2021). The origins and psychology of human cooperation. *Annual Review of Psychology*, *72*(1), 207–240. https://doi.org/10.1146/annurev-psych-081920-042106.

Hill, W. G., Goddard, M. E., & Visscher, P. M. (2008). Data and theory point to mainly additive genetic variance for complex traits. *PLoS Genetics*, *4*(2), e1000008. https://doi.org/10.1371/journal.pgen.1000008.

Hivert, V., Sidorenko, J., Rohart, F., Goddard, M. E., Yang, J., Wray, N. R., … Visscher, P. M. (2021). Estimation of non-additive genetic variance in human complex traits from a large sample of unrelated individuals. *The American Journal of Human Genetics*, *108*(5), 786–798. https://doi.org/10.1016/j.ajhg.2021.02.014.

Hofstede, G. (2001). *Culture's consequences: Comparing values, behaviors, institutions and organizations across nations*. Sage publications.

Jablonka, E. (2016). Cultural epigenetics. *The Sociological Review Monographs*, *64*(1), 42–60. https://doi.org/10/ggxwsq.

Jablonski, N. G., & Chaplin, G. (2017). The colours of humanity: The evolution of pigmentation in the human lineage. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *372*(1724), 20160349. https://doi.org/10.1098/rstb.2016.0349.

Jackson, J. C., Gelfand, M., De, S., & Fox, A. (2019). The loosening of American culture over 200 years is associated with a creativity–order trade-off. *Nature Human Behaviour*, *3*(3), 244–250. https://doi.org/10.1038/s41562-018-0516-z.

Jackson, J. C., Gelfand, M., & Ember, C. R. (2020). A global analysis of cultural tightness in non-industrial societies. *Proceedings of the Royal Society B: Biological Sciences*, *287*(1930), 20201036. https://doi.org/10.1098/rspb.2020.1036.

Jenkin, F. (1867). The origin of species. *North British Review*, *46*, 277–318.

Kitayama, S., King, A., Yoon, C., Tompson, S., Huff, S., & Liberzon, I. (2014). The dopamine D4 receptor gene (DRD4) moderates cultural difference in independent versus interdependent social orientation. *Psychological Science*, *25*(6), 1169–1177. https://doi.org/10.1177/0956797614528338.

Kwiatkowski, D. P. (2005). How malaria has affected the human genome and what human genetics can teach us about malaria. *The American Journal of Human Genetics*, *77*(2), 171–192. https://doi.org/10.1086/432519.

Larøi, F., Luhrmann, T. M., Bell, V., Christian, W. A., Deshpande, S., Fernyhough, C., … Woods, A. (2014). Culture and hallucinations: Overview and future directions. *Schizophrenia Bulletin*, *40*(Suppl_4), S213–S220. https://doi.org/10.1093/schbul/sbu012.

Lee, J. L. C., Nader, K., & Schiller, D. (2017). An update on memory reconsolidation updating. *Trends in Cognitive Sciences*, *21*(7), 531–545. https://doi.org/10.1016/j.tics.2017.04.006.

Lilienfeld, S. O., & Treadway, M. T. (2016). Clashing diagnostic approaches: DSM-ICD versus RDoC. *Annual Review of Clinical Psychology*, *12*(1), 435–463. https://doi.org/10.1146/annurev-clinpsy-021815-093122.

Lippa, R. A. (2010). Gender differences in personality and interests: When, where, and why?: Gender differences in personality and interests. *Social and Personality Psychology Compass*, *4*(11), 1098–1110. https://doi.org/10.1111/j.1751-9004.2010.00320.x.

Lipschuetz, M., Cohen, S. M., Ein-Mor, E., Sapir, H., Hochner-Celnikier, D., Porat, S., … Yagel, S. (2015). A large head circumference is more strongly associated with unplanned cesarean or instrumental delivery and neonatal complications than high birthweight. *American Journal of Obstetrics and Gynecology*, *213*(6), 833.e1-833.e12. https://doi.org/10.1016/j.ajog.2015.07.045.

Livingston, M., Raninen, J., Slade, T., Swift, W., Lloyd, B., & Dietze, P. (2016). Understanding trends in Australian alcohol consumption – An age–period–cohort model. *Addiction*, *111*(9), 1590–1598. https://doi.org/10/bmwr.

Luhrmann, T. M., Padmavati, R., Tharoor, H., & Osei, A. (2015). Differences in voice-hearing experiences of people with psychosis in the USA, India and Ghana: Interview-based study. *British Journal of Psychiatry*, *206*(1), 41–44. https://doi.org/10.1192/bjp.bp.113.139048.

Lukaszewski, A. W., Gurven, M., von Rueden, C. R., & Schmitt, D. P. (2017). What explains personality covariation? A test of the socioecological complexity hypothesis. *Social Psychological and Personality Science*, *8*(8), 943–952. https://doi.org/10.1177/1948550617697175.

Mathieson, I. (2021). The omnigenic model and polygenic prediction of complex traits. *The American Journal of Human Genetics*, *108*, 1558–1563. https://doi.org/10.1016/j.ajhg.2021.07.003.

Mitchell, K. J. (2018). *Innate: How the wiring of our brains shapes who we are*. Princeton University Press.

Molenaar, P. C. M., & Campbell, C. G. (2009). The new person-specific paradigm in psychology. *Current Directions in Psychological Science*, *18*(2), 112–117. https://doi.org/10.1111/j.1467-8721.2009.01619.x.

Molenaar, P. C. M., Huizenga, H. M., & Nesselroade, J. R. (2003). The relationship between the structure of interindividual and intraindividual variability: A theoretical and empirical vindication of developmental systems theory. In U. M. Staudinger & U. Lindenberger (Eds.), *Understanding human development* (pp. 339–360). Springer US. https://doi.org/10.1007/978-1-4615-0357-6_15.

Moya, C., & Boyd, R. (2015). Different selection pressures give rise to distinct ethnic phenomena: A functionalist framework with illustrations from the Peruvian Altiplano. *Human Nature*, *26*(1), 1–27. https://doi.org/10.1007/s12110-015-9224-9.

Muthukrishna, M., Bell, A. V., Henrich, J., Curtin, C. M., Gedranovich, A., McInerney, J., & Thue, B. (2020). Beyond Western, educated, industrial, rich, and democratic (WEIRD) psychology: Measuring and mapping scales of cultural and psychological distance. *Psychological Science*, *31*(6), 678–701. https://doi.org/10/ggxkjb.

Muthukrishna, M., Doebeli, M., Chudek, M., & Henrich, J. (2018). The cultural brain hypothesis: How culture drives brain expansion, sociality, and life history. *PLoS Computational Biology*, *14*(11), e1006504. https://doi.org/10.1371/journal.pcbi.1006504.

Muthukrishna, M., & Henrich, J. (2016). Innovation in the collective brain. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *371*(1690), 20150192. https://doi.org/10/gfzkmd.

Muthukrishna, M., Henrich, J., & Slingerland, E. (2021). Psychology as a historical science. *Annual Review of Psychology*, *72*(1), 717–749. https://doi.org/10/ghrnb6.

Niedzwiecki, M. M., Walker, D. I., Vermeulen, R., Chadeau-Hyam, M., Jones, D. P., & Miller, G. W. (2019). The exposome: Molecules to populations. *Annual Review of Pharmacology and Toxicology*, *59*(1), 107–127. https://doi.org/10.1146/annurev-pharmtox-010818-021315.

Norenzayan, A., Shariff, A. F., Gervais, W. M., Willard, A. K., McNamara, R. A., Slingerland, E., & Henrich, J. (2016). The cultural evolution of prosocial religions. *Behavioral and Brain Sciences*, *39*, e1. https://doi.org/10.1017/S0140525X14001356.

Oakhill, J. V. & Cain, K. (2012). The precursors of reading ability in young readers: Evidence from a four-year longitudinal study. *Scientific Studies of Reading*, *16*(2), 91–121.

Postle, B. R. (2006). Working memory as an emergent property of the mind and brain. *Neuroscience*, *139*(1), 23–38. https://doi.org/10.1016/j.neuroscience.2005.06.005.

Prado, E. L., Sebayang, S. K., Apriatni, M., Adawiyah, S. R., Hidayati, N., Islamiyah, A., … Shankar, A. H. (2017). Maternal multiple micronutrient supplementation and other biomedical and socioenvironmental influences on children's cognition at age 9–12 years in Indonesia: Follow-up of the SUMMIT randomised trial. *The Lancet Global Health*, *5*(2), e217–e228. https://doi.org/10/gdxwk6.

Reis, A., & Castro-Caldas, A. (1997). Illiteracy: A cause for biased cognitive development. *Journal of the International Neuropsychological Society*, *3*(5), 444–450.

Richerson, P. J., & Boyd, R. (2005). *Not by genes alone: How culture transformed human evolution*. University of Chicago Press.

Ritchie, S. J., Bates, T. C., & Deary, I. J. (2015). Is education associated with improvements in general cognitive ability, or in specific skills? *Developmental Psychology*, *51*(5), 573–582. https://doi.org/10.1037/a0038981.

Ritchie, S. J., & Tucker-Drob, E. M. (2018). How much does education improve intelligence? A meta-analysis. *Psychological Science*, *29*(8), 1358–1369.

Rowe, D. C., Jacobson, K. C., & Van den Oord, E. J. C. G. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as moderator. *Child Development*, *70*(5), 1151–1162. https://doi.org/10.1111/1467-8624.00084.

Ruff, C. B., Trinkaus, E., & Holliday, T. W. (1997). Body mass and encephalization in Pleistocene Homo. *Nature*, *387*(6629), 173–176. https://doi.org/10.1038/387173a0.

Sauce, B., Bendrath, S., Herzfeld, M., Siegel, D., Style, C., Rab, S., … Matzel, L. D. (2018). The impact of environmental interventions among mouse siblings on the heritability and malleability of general cognitive ability. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1756), 20170289. https://doi.org/10.1098/rstb.2017.0289.

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development*, *63*(1), 1–19.

Scarr-Salapatek, S. (1971). Race, social class, and IQ. *Science (New York, N.Y.)*, *174*(4016), 1285–1295. https://doi.org/10.1126/science.174.4016.1285.

Schimmelpfennig, R., Razek, L., Schnell, E., & Muthukrishna, M. (2022). Paradox of diversity in the collective brain. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *377*(1843), 20200316. https://doi.org/10.1098/rstb.2020.0316.

Schmitt, D. P., Long, A. E., McPhearson, A., O'Brien, K., Remmert, B., & Shah, S. H. (2017). Personality and gender differences in global perspective. *International Journal of Psychology*, *52*, 45–56. https://doi.org/10.1002/ijop.12265.

Schulz, J. F., Bahrami-Rad, D., Beauchamp, J. P., & Henrich, J. (2019). The Church, intensive kinship, and global psychological variation. *Science (New York, N.Y.)*, *366*(6466), eaau5141. https://doi.org/10/ggckxh.

Schwadel, P. (2011). Age, period, and cohort effects on religious activities and beliefs. *Social Science Research*, *40*(1), 181–192. https://doi.org/10/brb64c.

Smaldino, P. E., Lukaszewski, A., von Rueden, C., & Gurven, M. (2019). Niche diversity can explain cross-cultural differences in personality structure. *Nature Human Behaviour*, *3*(12), 1276–1283.

Tikhodeyev, O. N., & Shcherbakova, O. V. (2019). The problem of non-shared environment in behavioral genetics. *Behavior Genetics*, *49*(3), 259–269. https://doi.org/10.1007/s10519-019-09950-1.

Tooby, J. & Cosmides, L. (1992). The psychological foundations of culture. In *The adapted mind: Evolutionary psychology and the generation of culture* (p. 60). Oxford University Press.

Triandis, H. C. (1996). The psychological measurement of cultural syndromes. *American Psychologist*, *51*(4), 407–415.

Tucker, B., Ringen, E. J., Tsiazonera, Tombo, J., Hajasoa, P., Gérard, S., … Garçon, A. H. (2021). Ethnic markers without ethnic conflict: Why do interdependent Masikoro, Mikea, and Vezo of Madagascar signal their ethnic differences? *Human Nature*, *32*(3), 529–556. https://doi.org/10.1007/s12110-021-09412-w.

Tucker-Drob, E. M., & Bates, T. C. (2016). Large cross-national differences in gene × socioeconomic status interaction on intelligence. *Psychological Science*, *27*(2), 138–149. https://doi.org/10.1177/0956797615612727.

Tucker-Drob, E. M., Briley, D. A., & Harden, K. P. (2013). Genetic and environmental influences on cognition across development and context. *Current Directions in Psychological Science*, *22*(5), 349–355. https://doi.org/10.1177/0963721413485087.

Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, *9*(5), 160–164. https://doi.org/10.1111/1467-8721.00084.

Turley, P., Meyer, M. N., Wang, N., Cesarini, D., Hammonds, E., Martin, A. R., … Visscher, P. M. (2021). Problems with using polygenic scores to select embryos. *New England Journal of Medicine*, *385*(1), 78–86. https://doi.org/10.1056/NEJMsr2105065.

Uchiyama, R., & Muthukrishna, M. (in press). Cultural evolutionary neuroscience. In *Oxford handbook of cultural neuroscience and global mental health*. Oxford University Press. https://doi.org/10.31234/osf.io/3pj8a.

Vogt, G. (2015). Stochastic developmental variation, an epigenetic source of phenotypic diversity with far-reaching biological consequences. *Journal of Biosciences*, *40*(1), 159–204. https://doi.org/10/f64xns.

Waring, T. M., & Wood, Z. T. (2021). Long-term gene–culture coevolution and the human evolutionary transition. *Proceedings of the Royal Society B: Biological Sciences*, *288*(1952), 20210538. https://doi.org/10.1098/rspb.2021.0538.

Wild, C. P. (2012). The exposome: From concept to utility. *International Journal of Epidemiology*, *41*(1), 24–32. https://doi.org/10.1093/ije/dyr236.

Yi, X., Liang, Y., Huerta-Sanchez, E., Jin, X., Cuo, Z. X. P., Pool, J. E., … Wang, J. (2010). Sequencing of 50 human exomes reveals adaptation to high altitude. *Science (New York, N.Y.)*, *329*(5987), 75–78. https://doi.org/10.1126/science.1190371.

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