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**Motivational Factors as Mechanisms of Gene-Environment Transactions in
Cognitive Development and Academic Achievement**

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Genetic differences between people are statistically associated with differences in their cognitive development and academic achievement (Plomin & Deary, 2014; Rietveld, 2013; Shakeshaft et al., 2013). Differences in the types and quality of environments experienced are also associated with differences in cognitive development and academic achievement (Duncan & Murnane, 2011; Huston & Bentley, 2010). While these simple observations have historically been viewed as incompatible with one another, the contemporary scientist and indeed even the educated layperson, will be quick to point out the fallacy in this apparent paradox: Rather than competing with one another, genetic and environmental influences act synergistically to affect human development. The recent mainstream acceptance of *interactionism* (Tabery 2014), however, still leaves open many scientific questions regarding mechanism. Most generally, what are the specific biological, social, and developmental processes through which genetic and environmental factors work together to influence human development?

In this chapter I describe a set of theoretical models that posit dynamic developmental mechanisms through which genetic and environmental factors *transact*, leading children to become nonrandomly matched to educationally-relevant environmental experiences that foster academic achievement. I pay particular attention the role of motivational factors as driving forces in these dynamic transactions, and I describe how these processes may give rise to gene-by-environment interactions. First, I begin with an overview of how the basic behavioral genetic paradigm is used to estimate the statistical contributions of genetic and environmental factors to

individual differences in psychological outcomes such as achievement test scores, GPA, and achievement motivation.

A Short Primer on Behavioral Genetic Methodology

Classical behavioral genetic methodology capitalizes on data from samples of sets of individuals who vary across sets in their degree of genetic relatedness (e.g. identical vs. fraternal twins, close-in-age biological siblings vs. close-in-age adoptive siblings) and/or shared rearing environment (e.g. siblings raised together vs. siblings raised apart) to build statistical models that estimate genetic and environmental contributions to variation in one or more outcomes of interest (e.g. motivational factors, personality traits, achievement test scores or GPA). Typically, total variation in an outcome is decomposed into three components: a genetic component, a shared environmental component, and a nonshared environmental component. The magnitude of variance in an outcome attributable to the genetic component is inferred from the extent to which, holding the amount of objectively shared rearing environment constant, more genetically similar individuals (e.g. identical twins raised together) resemble one another on that outcome more than do less genetically similar individuals (e.g. fraternal twins raised together). The magnitude of variance in an outcome attributable to the shared environmental component is inferred from the extent to which, holding genetic relatedness constant, individuals reared together (e.g. genetically unrelated adoptive siblings) resemble one another on that outcome more than do individuals reared apart (e.g. random pairs of individuals). It can also be inferred from the extent to which genetically related individuals reared together (e.g. identical twins reared together and fraternal twins reared together) resemble one another on the outcome to a greater extent than can be attributed to genetic relatedness alone. Finally, the magnitude of variance in an outcome attributable to the non-shared environmental component is inferred from

the extent to which individuals are even more dissimilar on an outcome than would be expected from differences in their rearing environment and genetic makeup. For instance, the extent to which identical twins raised together (who have nearly identical genetic makeup and are raised in the same homes by the same parents and often attend the same school) differ on an outcome (to a greater extent than would be expected on the basis of measurement error alone) is attributable to the nonshared environment.

It is important to keep in mind that behavioral genetic methods are only useful for studying variation that exists in the population sampled. Behavioral genetic methods are able to provide insight into the extent to which differences in genetic sequence between people are statistically associated with individual differences in their outcomes, but they are unable to provide direct insight into the extent to which portions of genetic sequence that are invariant across individuals give rise to universals shared by all humans. For instance, behavioral genetic methods cannot be used to determine the role of genetics in the fact that (nearly) all humans have ten fingers and ten toes, or in the fact that (nearly) all human adults are capable of producing and understanding complex language. Similarly, behavioral genetic methods are able to provide insight into the extent to which variation in environmental experiences that naturally exists in the population sampled is statistically related to individual differences in the outcomes under investigation, but they are (like all observational methods in the social sciences) not able to provide direct insight into the extent to which environments not experienced by participants in the sample (including interventions or policies that have yet to be implemented), or environments that are universally experienced by all participants in the sample (e.g. going to school) are related to the outcomes under investigation. This is an important and oftentimes underappreciated point: High estimates of heritability on an outcome **do not** place constraints on whether a new

intervention or policy can be effective in influencing that outcome. Behavioral genetic methods can of course be informative about the effects of existing interventions or policies that vary (either naturally, or as a result of experimental control) in the population sampled. Indeed, as I discuss in the final section of this chapter, the application of behavioral genetic designs to randomized experiments is a potentially fruitful avenue for understanding how individuals might differentially respond to interventions (Tucker-Drob, 2011; Plomin & Haworth, 2010) and how interventions might change not just mean levels of an outcome (e.g. academic achievement) but also the distribution of levels of that outcome across individuals and families.

The merits, assumptions and limitations of various behavioral genetic approaches have been discussed at length elsewhere (Turkheimer, 2015; McGue et al., 2005) and I will not repeat them here. However, the reader should be aware that evidence for genetic influences on cognitive and educational outcomes does not derive from one particular paradigm, but rather from an assortment of different types of studies including twin, extended family, adoption, and most recently molecular-genetic. Because each method relies on somewhat different assumptions, violations of which have different implications for model estimates, and because the general pattern of results regarding genetic influences on cognitive and educational outcomes has been robust to the particular method employed, the general body of behavioral genetic work rests of very solid ground (for an accessible overview, see Munafò, 2016). Arguments about whether there are statistical associations between genotype and cognitive and educational outcomes are outdated. The associations exist and, on average, they are moderate in magnitude. An important question that remains is what the specific mechanisms are that give rise to these associations. Thus, the remainder of this chapter focuses on a class of theoretical models that

proposal a dynamic developmental mechanism through which genetic influences on cognitive and educational outcomes come to be realized.

Transactional Models of Cognitive Development and Academic Achievement

According to transactional models of cognitive development and academic achievement, individuals differ in the experiences that they select, evoke, and attend to, on the basis of their genetically influenced interests, goals, aptitudes, and motivations. These environments, in turn, have causal effects on their cognitive development and academic achievement. Because environmental experiences are nonrandomly experienced on the basis of genetically-influenced psychological and behavioral tendencies, the causal effects of environmental experience on learning results in the statistical differentiation of individuals' educational outcomes by genotype. Thus, in contrast to the lay view that genetic influences compete with experiential influence, transactional models hold that genetic influences on cognition and achievement occur- at least in part- by way of environmental experience.

One of the first explicit proposals of the transactional hypothesis was by Hayes (1962), who made the following four-point argument:

“(a) Differences in motivation may be genetically determined. (b) These motivational differences, along with differences in environment, cause differences in experience. (c) Differences in experience lead to differences in ability. (d) The differences commonly referred to as intellectual are nothing more than differences in acquired abilities” (p. 303).

In other words, according to Hayes, genetically-influenced motivational factors, what he referred to as “experience producing drives,” play instrumental roles in what environments are experienced by individuals and variation in experience leads to variation in intellectual

development, such that genetic influences in motivational factors give rise to individual differences intellectual development.

Transactional models also build on Scarr and McCartney's (1983) developmental theory of genotype-environment correlation, which itself builds on the work of Plomin, DeFries, & Loehlin (1977). Genotype-environment correlation (rGE) refers to the correlations that arise between genetic differences between people and differences in the environments that they experience. Plomin et al. (1977) developed a tripartite taxonomy of rGE. Passive rGE arise when children who are reared by their biological parents inherit genes from the same individuals who provide them with their rearing environment. For example, children raised by more educationally motivated parents not only inherit a disposition toward educational motivation, but are also raised in a family environment in which high academic achievement is valued and promoted. Active rGE occurs when children actively choose experiences from their environment on the basis of their genetically influenced traits. For example, children who are disposed toward high academic motivation may enroll in more rigorous coursework and seek out extracurricular activities that promote positive academic skills. Evocative rGE (originally termed reactive rGE) arise when children evoke different experiences from individuals and institutions within their broader environmental contexts on the basis of their genetically influenced traits. For example, children disposed toward high motivation may be more likely to respond positively to attention from teachers, thus positively reinforcing teachers' tendency to provide them with further time and attention. Both active and evocative forms of rGE are hypothesized to have central roles in transactional processes between children and their environments. As proposed by Scarr & McCartney (1983,), "the degree to which experience is influenced by individual genotypes increases with development and with the shift from passive to active genotype → environment

effects, as individuals select their own experiences” (p. 427)... “and build niches that are correlated with their talents, interests, and personality characteristics (p. 433)” with age.

Other notable contributions to the development of the transactional perspective come from the work of Sameroff. Sameroff (1975) wrote that “the constants in development are not some set of traits but rather the processes by which these traits are maintained in the transactions between organism and environment.” More recently Sameroff & McKenzie (2003) wrote that

“the development of the child is a product of the continuous dynamic interactions of the child and the experience provided by his or her family and social context. What is central to the transactional model is the equal emphasis placed on the bidirectional effects of the child and of the environment. Experiences provided by the environment are not viewed as independent of the child.”

Like the theory of Scarr and McCartney (1983), Sameroff’s transactional perspective is a more general framework of psychological development that was not specifically developed with cognition or academic achievement in mind. Unlike that of Scarr and McCartney (1983), however, Sameroff’s perspective does not directly address the role of genotype in the transactional process. It does, however, consider “constitution” (Figure 1).

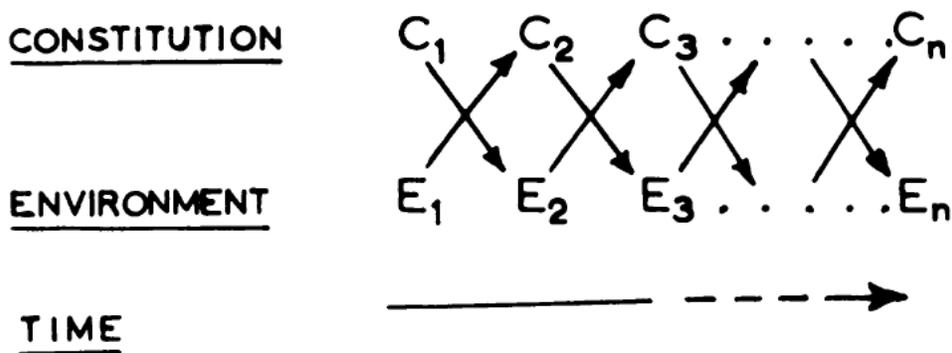


Figure 1. A representation of Sameroff’s Transactional perspective. From Sameroff (1975).

In their bioecological model, Bronfenbrenner and Ceci (1994) further expanded upon the concept of reciprocal causation between the child and his or her immediate environment, explicitly hypothesizing that such transactions are a primary basis for genetic effects on adaptive psychological outcomes, including intelligence.

“Human development takes place through processes of progressively more complex reciprocal interaction between an active, evolving biopsychological human organism and the persons, objects, and symbols in its immediate environment. To be effective, the interaction must occur on a fairly regular basis over extended periods of time. Such enduring forms of interaction in the immediate environment are referred to henceforth as *proximal processes*... Proximal processes serve as a mechanism for actualizing genetic potential for effective psychological development” (p. 572)

Importantly, as indicated by the quote above, Bronfenbrenner and Ceci (1994) hypothesized that proximal processes must occur recurrently over prolonged periods of time, and that their effects on psychological development accumulate progressively over time.

Recently, transactional models have been mathematically formalized. Dickens and Flynn (2001), for instance developed a simulation model of “strong reciprocal causation between phenotypic IQ and environment” (p.345) in which initial genetically influenced individual differences in cognitive ability lead to more cognitively stimulating environments, which in turn lead to higher cognitive ability, leading to “a positive correlation between environment and genotype that masks the potency of environment” (p. 345). Beam, Turkheimer, Dickens, and Davis (2015) adapted the Dickens and Flynn (2001) model as a structural equation model, which they fit to longitudinal IQ data from the Louisville Twin Study. They concluded that the

transactional model (which allows latent genetic factors to predict subsequent latent environmental factors) provided a better fit to the data than a conventional autoregressive simplex model (which models time-point-to-time-point stability of IQ as the simple result of time-point-to-time-point stability of genetic and environmental factors, but does not allow associations between genetic and later environmental factors).

Fundamental to the Dickens and Flynn (2001) model is the postulation (also found in Bronfenbrenner and Ceci's 1994 bioecological model) that, in order for environmental experiences to have meaningful effects on cognitive development, they must systematically recur over extended periods of time. Experiences that are systematic and recurring, Dickens and Flynn (2001) have argued, stem from socially entrenched and institutionalized processes (e.g. social class, race, historical period, and culture) and from gene-environment transactions. On this latter point, Dickens and Flynn (2001) have reasoned that experiences selected on the basis of relatively stable and enduring genetically-influenced tendencies will tend to recur systematically over time. Apart from those that result of macro-societal forces in which individuals are deeply embedded, experiences that result from nongenetic factors, Dickens and Flynn (2001) argued, have a stronger tendency to be arbitrary, tend not to recur, and therefore tend to have unappreciable and ephemeral effects. This postulation is crucial to the prediction that transactional processes lead to the differentiation of individuals by genotype, rather than simply by initial states, over time (Tucker-Drob & Harden, 2012a).

Motivational Factors as Propulsive Forces in Academically-Relevant Gene-Environment Transactions

What are the specific genetically-influenced factors that lead individuals to differentially select evoke achievement-relevant environments? Some authors (e.g. Dickens & Flynn, 2001;

Beam et al., 2015) have suggested that early genetically-influenced individual differences in cognitive ability lead to differentiation of environmental experience, which in turn further differentiates individuals by cognitive ability. Others, including the early work of Hayes (1962) and the influential work of Scarr and McCartney (1983) have placed strong emphases on genetically influenced variation in motivations, interests, and personality as propelling individuals to differentially select and evoke environmental niches. High levels of motivational factors such as intellectual interest and achievement motivation may lead children to actively choose more intellectually stimulating peer groups, coursework, and extracurricular experiences from the ecologies in which they are embedded. At the same time, behaviors stemming from such motivational factors may, when observed by others, may evoke from them more stimulating interactions, attract more achievement-oriented friendship networks, and lead teachers and parents to provide individuals with greater and/or higher quality experiences. Motivational factors are also likely to be related to the extent to which different children attend to, deeply process, and expend effort even in the same educational setting. On the whole, differences in the amount and quality of environments experienced and differences in the extent to which these environments are attended to, lead both to differences in the cognitive development and academic achievement and to differences in the motivational traits that lead to the different experiences in the first place (Tucker-Drob and Harden, 2012b; Figure 2),.

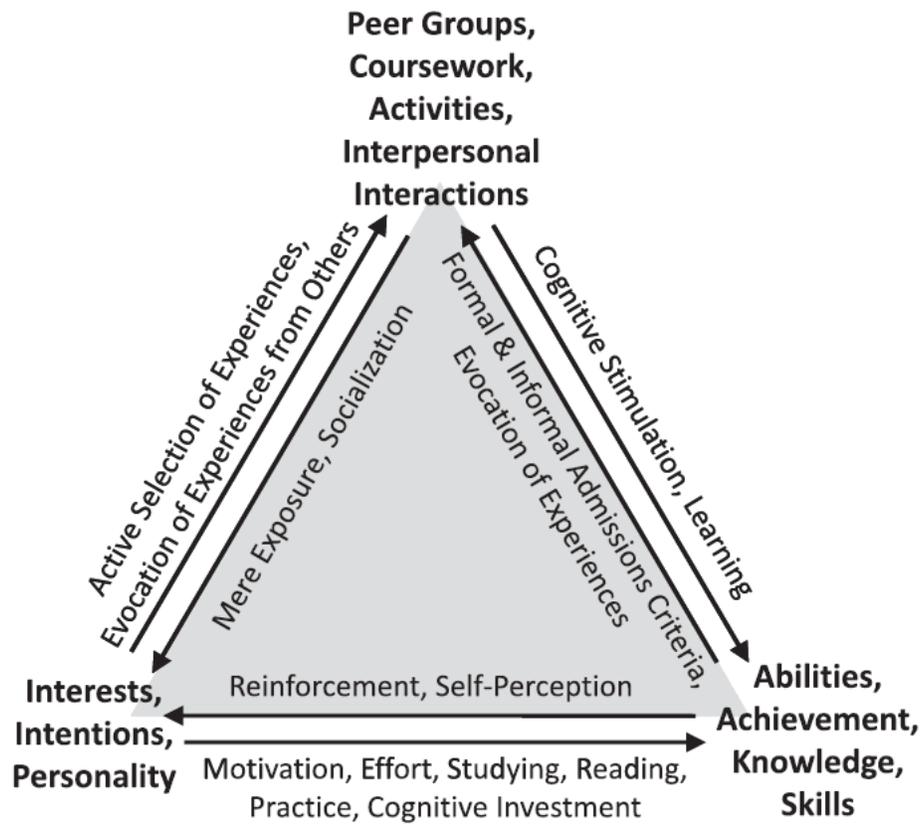


Figure 2. A conceptual model for the mutual relations between motivational factors, proximal environments, and achievement. From Tucker-Drob & Harden (2012b).

Tucker-Drob & Harden (in press) recently reviewed the evidence relevant to the roles of a broad constellation of motivational factors in the processes by which individuals come to nonrandomly experience different academically-relevant environments as functions of their genotypes. These included Openness, Conscientiousness, Intellectual Interest, Academic Interest, Self-Perceived Ability, Grit, Self-Control, Achievement Goal Orientations, Intelligence Mindsets, Expectancies, and Values. We suggested six general criteria that should be fulfilled in order for a motivational factor to be implicated in academically-relevant gene-environment transactions. **(1)** The motivational factor should be correlated with academic achievement in observational data, as a correlation is typically a necessary, though not sufficient, condition of

causality within a naturally occurring system. **(2)** The motivational factor should statistically predict achievement above and beyond both cognitive ability (2a) and the Big Five personality factors (2b), as incremental prediction is necessary to rule out simple third variable (and “jangle”) confounds attributable to overlap with the most well-studied psychological dimensions of individual differences. **(3)** In order to serve as a mechanism by which genotypes become matched to experiences, the motivational factor must be heritable. **(4)** In order for the motivational factor to mediate genetic effects on achievement, achievement must be influenced by some of the same genes that influence the motivational factor; i.e. there should be a nonzero genetic correlation between the motivational factor and achievement. **(5)** The direction of causation within the naturally occurring system, as tested using longitudinal methods such as cross-lagged panel analysis, should at least partially be from the motivational factor to achievement. **(6)** As a direct test of the role of gene-environment correlation in the motivation-achievement association, measured academically-relevant environments should at least partially mediate genetic links between the motivational factor and achievement.

Based on our literature review (Tucker-Drob & Harden, in press), we were able to verify that nearly all of the motivational factors considered are correlated with academic achievement at nontrivial levels (Criterion 1), and in many cases such associations were robust to controls for intelligence (Criterion 2a). We found that many motivational factors, however, were not well-studied using behavioral genetic methods. Notable exceptions include Openness and Conscientiousness, which as major dimensions of personality have been highly studied in genetically-informed samples, as well as – but to a much lesser extent- intellectual and academic interest, self-perceived ability, and self-control, all of which have been found to be moderately heritable (Criterion 3). We found that there had been very little, if any, behavioral genetic work

on Grit, Achievement Goal Orientations, Mindsets, or Expectancies and Values. In the cases of Conscientiousness, Openness, Intellectual Interest, and Self Perceived ability, there was also evidence that genetic factors at least partially mediate associations with academic achievement (Criterion 4). There was emerging evidence that many of the motivational factors longitudinal predict achievement, even when controlling for past achievement, indicating that the direction of causation may at least partially originate from the motivational factors (Criterion 5). Finally, with the exception of Expectancies and Values, we were unable to identify strong longitudinal research testing for mediation of the motivational factor-achievement association by measured environments. Nor did we find any work that tested such mediation using genetically-informed methods (Criterion 6). We suggested that measuring environmental factors that children are able to dynamically select and evoke and that are relevant to achievement may indeed be one of the biggest ongoing challenges in empirical tests of transactional models. Finally, we found that the extents to which motivational factors relate to one another and to the Big Five personality traits were not well studied, and it was therefore unclear whether many of the commonly studied represent the same, independent, or partially overlapping dimensions of individual differences (Criterion 2b).

Recently, my colleagues and I published an article reporting results of a project that has attempted to fill many of the above-identified gaps in the literature (Tucker-Drob, Briley, Engelhardt, Mann, & Harden, in press). Using data that we collected from a racially, ethnically, and socioeconomically diverse population-based sample of 811 third through eighth grade twins and triplets from the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013), we examined how seven popular character traits (*grit*, *intellectual curiosity*, *intellectual self-concept*, *mastery orientation*, *educational value*, *intelligence mindset*, and *test motivation*) relate to measures of

the Big Five personality traits, (b) relate to one another, (c) are associated with genetic and environmental variance components, and whether such effects operate through common dimensions of individual differences, and (d) are related to verbal knowledge and academic achievement above through genetic and environmental pathways both before and after controlling for fluid intelligence. We found that the character measures correlated moderately with one another and with measures of Openness and Conscientiousness from the Big Five Inventory (BFI; John, Naumann, & Soto, 2008). When these measures were included in a factor analyses, two latent factors emerged: (1) a latent factor that we named Openness, upon which Intellectual Self-Concept, Intellectual Curiosity (Need for Cognition), and BFI Openness loaded appreciably, and (2) a latent factor that we named Conscientiousness, upon which grit, Intellectual Curiosity, Mastery Orientation, Educational Value, Intelligence Mindset, and BFI Conscientiousness loaded appreciably. Both latent factors (which were correlated at $r = .44$) were influenced approximately 50% by genetic factors and 50% by nonshared environmental factors. For nearly all of the individual measures, there were also residual genetic and nonshared environmental influences that were not accounted for by the latent Openness and Conscientiousness factors. There was no indication of shared environmental influence at either the factor or the measure-specific levels. Both when character was examined at the level of the Openness and Conscientiousness factors and when it was examined at the level of the individual measures (Figure 3), relations with verbal knowledge and academic achievement were positive, and persisted after controlling for fluid intelligence. Consistent with the predictions of transactional models, genetic factors primarily mediated these associations. Nonshared environmental mediation was generally trivial and inconsistent across tasks.

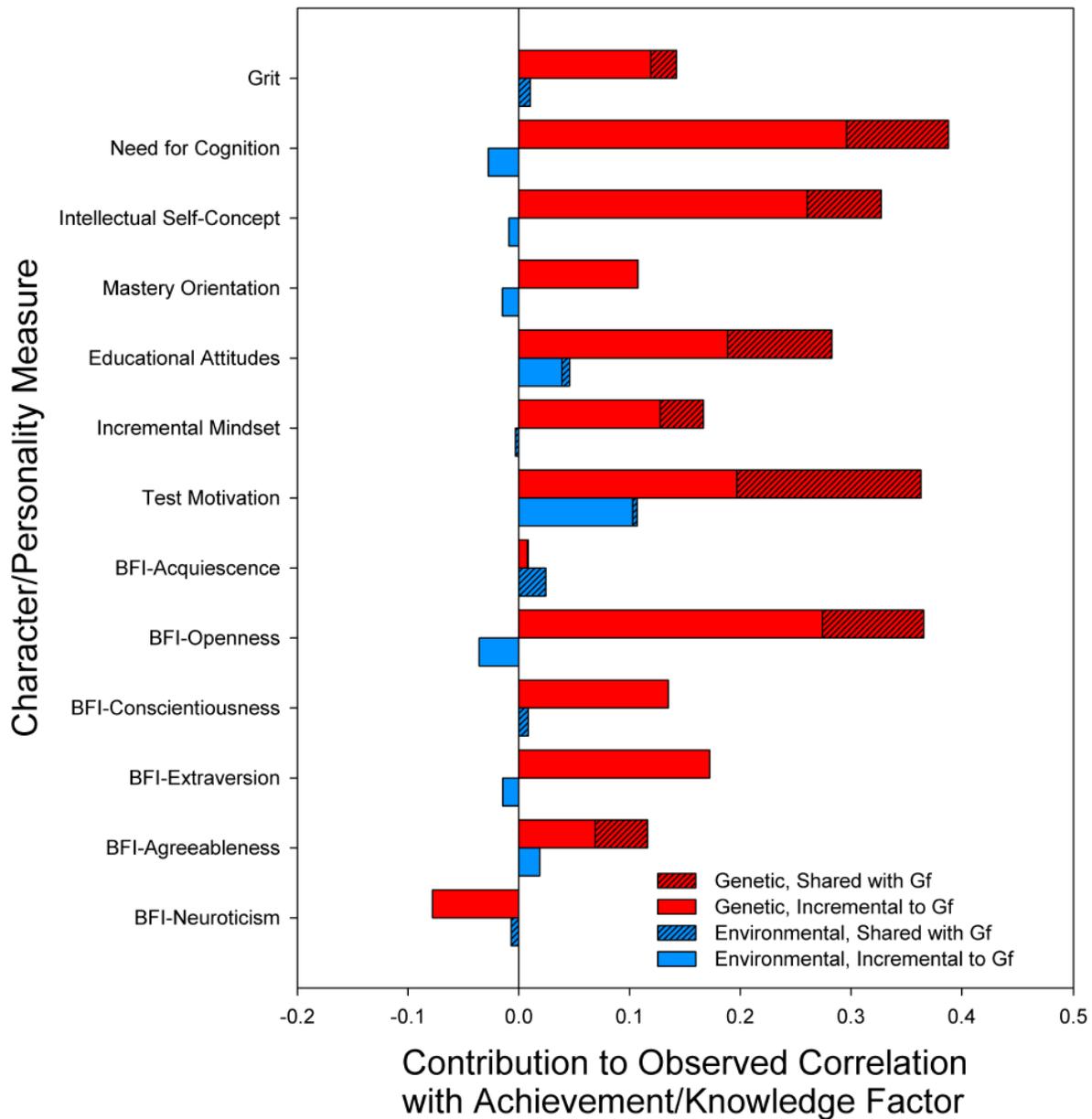


Figure 3. Barplot representing correlations between the character/Big Five Inventory scores and a latent achievement/ knowledge factor. The sum of the paired red (i.e., genetically mediated contribution) and blue (i.e., environmentally mediated contribution) bars represents the net model-implied correlation. The cross-hatched portion of the red and blue bars represents genetic and environmental contributions to associations between character and achievement

shared with fluid intelligence. The solid portion of the red and blue bars represents genetic and environmental contributions to associations between the character and achievement incremental to fluid intelligence. Shared and incremental effects sum to the total genetic and environmental effects. For instances in which the shared and incremental effects were in opposite directions, the aggregated effect is displayed. From Tucker-Drob et al. (in press).

Transactional Processes as Mechanisms of Developmental Increases in Heritability

A highly robust and perhaps equally counterintuitive finding from the past quarter century of behavioral genetic research is that of developmental changes in the heritability of cognitive abilities. Some researchers (e.g. Fryer & Levitt; Spelke, 2005) have speculate that genetic influences on psychological outcomes should be strongest in early life and decrease with age, as the effects of environmental influences accumulate and account for a larger and larger share of the individual differences pie. However, the empirical pattern of developmental changes in in the heritability of cognitive abilities is exactly the reverse. Genetic influences on cognitive abilities account for very small proportions of variance during infancy, with proportions increasing continuously over the course of child development, such that by late adolescence, genetic influences on cognitive abilities is between approximately 60% and 70% (Haworth et al., 2010; McCartney, Harris, and Bernieri (1990; Tucker-Drob Briley, & Harden, 2013; Briley & Tucker-Drob, in press).

Two general classes of mechanisms have the potential to account for this pattern (Briley & Tucker-Drob, 2013; Plomin, 1986). *Innovation* refers to a circumstance in which novel genetic factors, not previously relevant for cognitive abilities in early development, become relevant for cognitive abilities at later ages. This can occur because portions of genetic code are not yet transcribed until later in development, at which point they become epigenetically activated (Reik

et al., 2007; Bocklandt et al., 2011; Hannum et al., 2013; Horvath, 2013). Innovation can also occur when genetic factors that are expressed early in life influence non-cognitive but not cognitive abilities, and become increasingly relevant for cognitive abilities over the course of development. *Amplification* refers to a circumstance in which the same genetic factors relevant for cognitive abilities in early life have increasingly large effects on those abilities with age, such that their effects are amplified over development.

In a series of papers, Daniel Briley and I (Briley & Tucker-Drob, 2013; Briley & Tucker-Drob, in press; Tucker-Drob & Briley, 2014) have meta-analyzed longitudinal behavioral genetic studies of cognitive ability to examine the extent to which genetic influences on cognitive abilities persist forward and the extent to which novel genetic influences arise over time. We find that over the first decade of life, increasing heritability is driven by innovation processes, in which genetic factors not previously relevant for cognitive abilities become relevant at later ages. In the second decade of life, amplification process become the predominant drivers of increasing heritability: Heritability of cognitive abilities continues increasing during middle childhood and adolescence by way of amplifying the effects of genetic factors relevant for cognitive ability since approximately age 10 years.

Transactional models provide a plausible explanation for both the innovation pattern observed in infancy and early childhood and the amplification pattern observed in middle childhood and adolescence. Under transactional models, genetically influenced motivational factors are initially irrelevant for cognitive development. As time passes, and as children have increasing autonomy to select their experiences, they differentially accrue different environment experiences as a function of their genetically influenced motivational factors. Genetic influences on motivational factors that were originally irrelevant for cognitive abilities are expected to

become relevant for cognitive abilities over time- i.e. innovation. Once genetic influences on motivational factors become coupled to cognitive abilities, transactional processes are expected to continue, further differentiating children's experiences, and hence their cognitive abilities, by genotype – i.e. amplification. Consistent with more conventional wisdom, such a transactional perspective postulates that the effects of environmental experience on cognitive abilities accrue over time. However, because environments are nonrandomly experienced on the basis of genetically-influenced factors, the result is increasing heritability of cognitive abilities over development.

Transactional Processes as Mechanisms of Gene-by-Environment Interactions

Thus far, this paper has discussed *gene-environment transactions*, which are dynamic processes in which individuals come to be differentially exposed to environmental experiences on the basis of genetically-influenced dispositional factors, *and* these environments in turn affect their cognitive development and academic achievement. *Gene-by-environment interactions* are conceptually and mathematically distinct phenomena whereby genetic differences between people are associated with differences in effects of an environmental input on their psychological development *and* the magnitude of genetic effect on an outcome is stronger in some environmental contexts than in others (Plomin, DeFries, & Loehlin, 1977). Interestingly, macro-environmental contexts may modulate the magnitude of heritability by way of constraining or facilitating transactional processes. In other words, *gene-environment transactions* may serve as a basis for *gene-by-environment interactions*. This hypothesis has been stated by a number of separate authors over the past two decades, as exemplified by the following quotes:

“The entire theory [of gene-environment correlation] depends on people having a varied environment from which to choose and construct experiences. The theory does not apply,

therefore, to people with few choices or few opportunities for experiences that match their genotypes” (Scarr, 1992, p. 9).

“Heritability (assessed by h^2) varies markedly and systematically as a function of levels of proximal process” (Bronfenbrenner & Ceci, 1994, p. 570).

“Under a transactional model of cognitive development, children are expected to select and evoke their environmental experiences on the basis of genetically influenced dispositions, but this process depends on the existence of adequate opportunities for such experiences” (Tucker-Drob, Briley, & Harden, 2013).

“Genes without sufficient match to suitable environments lose influence on development” (Beam et al., in press).

One of the most commonly mentioned macro-environmental dimension hypothesized to be associated with differences in the efficiency of academically- and intellectually-relevant transactional processes is childhood socioeconomic status. Children living in lower socioeconomic status settings are provided with fewer opportunities to seek out high quality educational experiences, and live under conditions of hardship that may limit the ability of those around them to be attentive to and supportive of their interests, talents, and goals. Consistent with this hypothesis, a number of studies (Scarr-Salapatek, 1971; Rowe, Jacobson, & Van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003; Harden, Turkheimer, & Loehlin, 2007; Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011; Bates, Lewis, &

Weiss, 2013) have reported that genetic influences on cognitive ability and academic achievement are suppressed under conditions of socioeconomic privation. A recent meta-analysis (Tucker-Drob & Bates, 2016) confirms this Gene-by-Childhood Socioeconomic Status interaction in the United States (Figure 4): at 2 standard deviations below the mean SES, model-implied heritability of cognitive ability and academic achievement is approximately 24%, with progressive increases in heritability throughout the range of SES, such that at 2 standard deviations above the mean SES, model-implied heritability is approximately 61%. Interestingly, our meta-analysis indicated that such an interaction is not apparent in samples from Western Europe and Australia, with the difference between US and Western European/Australian interaction effects sizes itself being statistically significant. Sensitivity analyses indicated robustness of this cross-national pattern to the choices of samples and measures included, and there was no significant evidence of p-hacking or publication bias that could have biased or distorted estimates (Simonsohn, Nelson, & Simmons, 2014). One provocative interpretation, then, of these cross-national differences is that opportunities for cognitively- and academically-relevant gene-environment transaction are far less stratified by socioeconomic status in Western Europe and Australia than they are in the United States.

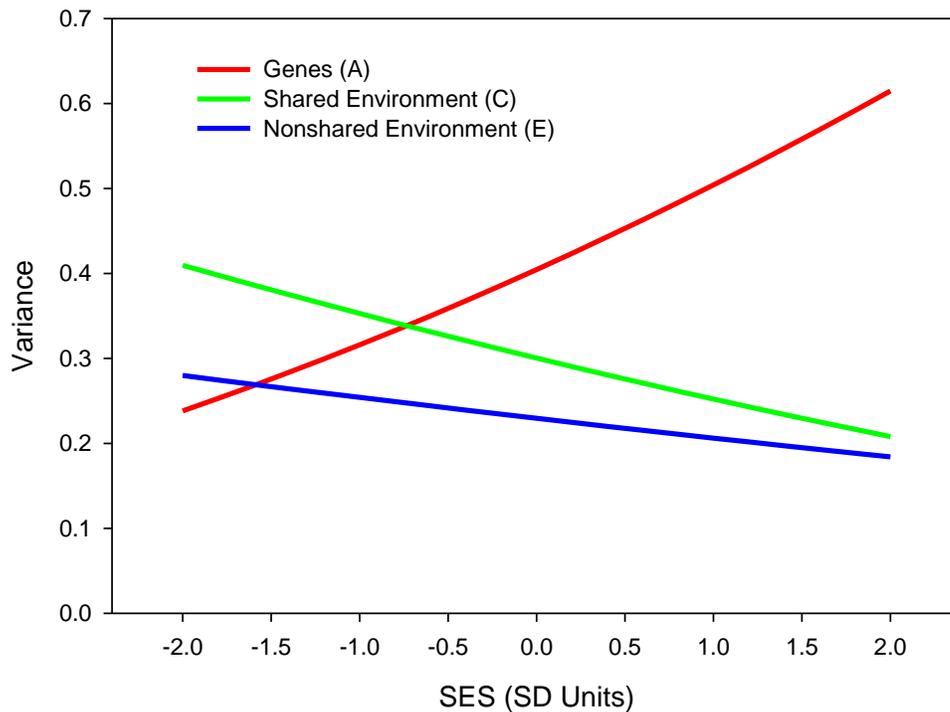


Figure 4. Meta-analytic results for Gene-by-Socioeconomic Status interaction on intelligence and achievement in the United States. From Tucker-Drob & Bates (2016). The *x*-axis represents family socioeconomic status (SES) and the *y*-axis represents variance in intelligence and achievement that is explained by genetic, shared environmental, and nonshared environmental factors. For further explanation of the meaning of genetic, shared environmental, and nonshared environmental variance components, see section titled “*A Short Primer on Behavioral Genetic Methodology.*”

My colleagues and I have conducted series of studies probing whether socioeconomic status moderates the role of motivational factors on academic achievement. In one study (Tucker-Drob & Briley, 2012) of N=375,000 US high school students, we investigated whether family SES moderated the relation between domain-specific interests and domain-specific knowledge in eleven academic, vocational/professional, and recreational domains, including art, literature, music, biological sciences, physical sciences, and sports. Consistent with our hypothesis that higher SES contexts afford children greater opportunities to pursue learning

experiences on the basis of their interests, we found that interest was appreciably more related to knowledge at higher levels of SES for all domains except for farming. In another study (Tucker-Drob, Cheung, & Briley, 2014) of approximately 400,000 high school students from 57 countries, we investigated moderation of science interest-science achievement associations by family SES, school SES, and National Gross Domestic Product. Again, consistent with the hypothesis that higher SES contexts allow children to select and evoke learning opportunities on the basis of their interests, we found that family SES positively moderated interest-achievement associations, such that science interest was a stronger predictor of science achievement test scores at higher levels of family and school SES. Importantly, however, the magnitude of moderation varied by country, with one of the largest interaction estimates obtained in the US subsample. We also found strong moderation of the within-country science interest-achievement association by National GDP: In the richest countries, the standardized association between interest in achievement was over .30, but in the poorest countries, the association was essentially 0. The correlation between log transformed National GDP and the country-specific effect-size representing the science interest-science achievement association was .753, 95% CI = [.639, .867]).

In two separate behavioral genetic studies of US children, we have found that this SES-by-Interest interaction mediates the Gene-by-Childhood SES interaction on achievement discussed earlier. Tucker-Drob & Harden (2012b) used a sample of 777 pairs of American high school twins (i.e., N = 1,554 individuals) we fit bivariate gene-by-environment interaction models to examine the association between intellectual interest and an academic achievement composite measure comprised of English Usage, Mathematics Usage, Social Science Reading, Natural Science Reading, and Word Usage. Results indicated that for low SES students, genetic

variance in intellectual interest was unrelated to academic achievement, but that for high SES students, genetic variance in intellectual interest accounted for approximately 30% of the variance in academic achievement. This interaction with genes for intellectual interest accounted for the previously identified gene-by-childhood SES interaction on achievement. In a separate sample of 650 pairs of preschool-age twins, we (Tucker-Drob & Harden, 2012c) similarly found that genetic influences on learning motivation were unrelated to early mathematics skills in low SES children, but accounted for approximately 30% of the variance in early mathematics skills in higher SES children. This interaction with genes for motivation accounted for the previously identified gene-by-childhood SES interaction on mathematics skills. Together these results are consistent with the hypothesis that, in the United States, higher SES affords greater opportunities for children to engage in transactional processes in which they select and evoke learning experiences on the basis of their genetically-influenced interests and motivations to learn.

Some comments are warranted. First, it is important to note that not all motivational factors may interact with SES in the same way. Our previous research has largely focused on interest, but other motivational factors, such as self-concept or self-control may interact with SES in different ways. For instance, it is possible that in high SES environments, where external support systems help children to structure their time and follow-through on their goals, individual differences in self-regulatory factors may be less important for achievement.

Second, while this section has focused on interactions involving SES, there are many other environmental factors that may interact with motivational factors to influence achievement. For instance, specific aspects of the school environment, such as teacher quality, have been found to interact with genetic influences on achievement (Taylor et al., 2010), and it is possible that motivational factors may play a role in this interaction.

Third, motivational factors may interact with one another in the prediction of student achievement. A recent series of studies has provided evidence for expectancy-by-value interactions in both engagement in educational activities (Nagengast et al., 2011) and academic achievement (Trautwein et al, 2012; Tucker-Drob et al., 2014). Although I am aware of no genetically-informed work on this topic, an exciting area for future research may be to examine whether genetic and/or environmental components of expectancies and values serve as the basis of these interaction. Expectancy-value interactions may constitute gene-by-environment interactions, environment-by-environment interactions, gene-by-gene interactions or some combination of the aforementioned.

Considering Interventions

What are the implications of transactional models, and of behavioral genetic research on motivation and achievement more generally, for policy and intervention? It is important to make clear that current knowledge regarding the developmental-genetic mechanisms of motivation and achievement is based almost exclusively on observational research and that it would therefore be inappropriate to rely on such research to make recommendations for the enactment of specific policies or interventions within society. Rather, this research is at a point in which it can be used to generate hypotheses about how new interventions or policies might be designed, and to make probabilistic statements about what sorts of policies or interventions might be more or less likely to be effective in the context of a carefully-designed program evaluation. Rigorous approaches to treatment and policy evaluation, ideally approaches that rely on randomized controlled designs, would be necessary before recommendations could be made regarding implementation outside of a research context.

It is also useful to make explicit what insights from behavioral genetics do not mean in terms of implications for intervention and policy research. Simply because an outcome is genetically-influenced does not mean that the environment does not matter. Genetic influences on outcomes rarely, if ever, account for all of the variation in psychological outcomes. Thus, even acceptance of the fallacious view that the genetic portion of the variance pie reflects an immutable component, still leaves plenty of room for a plastic component of the pie.

Moreover, genetically influenced variation in psychological outcomes are likely to often occur via environmental mechanisms. For instance, genetic influences on musical expertise occur, in part, by way of genetically-influenced variation in the propensity to practice a musical instrument, and it is the environmental experience of consistently playing the musical instrument that results in the development of musical expertise (Hambrick & Tucker-Drob, 2015). Similarly, transactional models predict that genetically-influenced variation in cognitive ability and academic achievement occur, in part, by way of variation in time, effort, and attention dedicated to learning experiences on the basis of genetically influenced motivational traits. Thus, one potentially fruitful avenue for policy and intervention research would be to first empirically trace the specific learning-relevant behaviors and experiences that motivated children engage in, and then develop programs and curricula that foster these behaviors, either through changing the motivational factors themselves or via changing the behaviors that are downstream from the motivational factors. Another potentially fruitful avenue for such research would be to examine how modulating the contextual supports for person-driven selection and evocation of learning experiences might shift both overall average levels of achievement and heterogeneity in achievement outcomes. It may also be advantageous to develop and test interventions that increase opportunities for highly motivated children to select and evoke environments while at

the same time restrict opportunities for children who are low in motivation to select suboptimal learning experiences. Indeed, work described earlier on gene-by-environment interaction highlights the potential utility of a dual emphasis on both personal and contextual factors in the development of policy and intervention hypotheses.

The finding that shared environmental influences on motivational factors are low, if not entirely absent, does call into question the common wisdom that the socializing effects of between-family variation in environmental experiences are a primary mechanism of naturally existing variation in these factors. Trivial estimates of shared environmental influence on measures of motivational outcomes imply that either 1) family environments have differential effects on the motivational outcomes of individuals within the same family and/or 2) environmental experiences that naturally vary (at nontrivial prevalence rates) in the general population aren't very potent for motivational outcomes. The implication of trivial shared environmental influences on motivational factors for intervention research is that, in order to be successful in producing a nontrivial average causal effect on motivational factors, an intervention would likely need to implement a treatment that isn't already varying at the family-level within the general population. As my colleagues and I (Tucker-Drob et al., in press) have previously stated, the lack of evidence for shared environmental influence on the character measures examined does "not inform the question of whether interventions or policies that have yet to be implemented, did not naturally occur for children in the current sample, or were universally experienced by all children in the sample could potentially make children raised together more similar in their character."

Finally, it is important to keep in mind that treatment effects, as they are typically estimated in the context of a randomized controlled experiment, are estimates of average causal

effects of the treatment across individuals. Treatment effects, however, may not be the same for all individuals within the population sampled. Methods for the estimating variability in, and correlates of, individual causal effects exist (Tucker-Drob, 2011), but such approaches are rarely (or inappropriately) implemented. Studying heterogeneity in treatment effects, however, can be tremendously valuable. Such knowledge could be used to 1) choose the most appropriate intervention for an individual student or subpopulation of students, 2) produce the best informed a priori estimate of how much of an effect to expect for a particular student or subpopulation of students, and of the potential range of magnitude of effect to be expected, 3) identify subpopulations of students that are most likely to benefit from a policy or intervention, and those that are likely to not benefit or to even react adversely. In fact, the incorporation of randomized experimental approaches and behavioral genetic approaches can be used to test whether the treatment under study magnifies and/or constricts genetic and environmental influences on the outcomes of interest. One potential goal of an intervention might be to both increase overall average levels of achievement and reduce between-family (shared environmental) variation in achievement (i.e. reduce achievement gaps). One underappreciated consequence of such a result is that, all else being equal, a greater proportion of remaining variation in achievement will be associated with genetic factors. As my colleagues and I (Tucker-Drob, Briley, & Harden, 2013) have previously hypothesized, “a social, educational, and economic opportunities increase in a society, genetic differences will account for increasing variation in cognition—and perhaps ultimately in educational and economic attainment.” Effective interventions that boost overall achievement and narrow socioeconomic inequalities in achievement outcomes may have similar effects, i.e. to increase the relative salience of genetic influences on those outcomes. This, however, is not a necessary outcome of all interventions that boost mean achievement; some

interventions may boost achievement while increasing between-family disparities. Ceci and Papierno (2005) have described such a situation as being one in which “the ‘have-nots’ gain but the ‘haves’ gain even more.” Whether such an outcome is, on balance, desirable from a policy or social justice perspective is a matter of values. Regardless, the question of heterogeneity in treatment effect is an important scientific question that can be used to inform policy decisions.

Conclusions

In this chapter, I have described a set of theoretical models that posit dynamic developmental mechanisms through which individuals become nonrandomly matched to environmental experiences on the basis of their genetically-influenced traits, and these experiences, in turn have causal effects on their cognitive development and academic achievement. While early genetically-influenced levels of cognitive ability and scholastic aptitude may themselves be propulsive factors in such transactional processes, there is both theoretical and a growing body of empirical evidence implicating genetically-influenced motivational factors as themselves propulsive. In other words, genetically influenced individual differences in personality, interests, goals, and other motivational factors lead to differences in the types and qualities of academically-relevant environments that children select, evoke, and attend to, leading to the differentiation of individual’s cognitive and educational outcomes by genotype over time, such that statistical associations between genetic factors and cognition and achievement increase over infant, child, and adolescent development. If macro-environmental factors, such as socioeconomic status, are related to the efficiencies of such dynamic processes, genetic influences on cognitive and educational outcomes will differ as systematic functions of macroenvironmental measures, i.e. a gene-by-environment interaction. Future work will be necessary to identify and test the specific motivational factors responsible for gene-environment

transactions, to identify the specific educationally-relevant environments that come to be correlated with genotypes over time, and to further delineate the macro-environmental conditions under which transactional processes are modulated. Such work may ultimately help to inform the design of policies and interventions that would then need to be evaluated using rigorous randomized controlled methods before being implemented in society at large.

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