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Genetic and Environmental Influences on Cognition Across Development and Context

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Abstract

Genes account for between approximately 50% and 70% of the variation in cognition at the population level. However, population-level estimates of heritability potentially mask marked subgroup differences. We review the body of empirical evidence indicating that (a) genetic influences on cognition increase from infancy to adulthood, and (b) genetic influences on cognition are maximized in more advantaged socioeconomic contexts (i.e., a Gene × Socioeconomic Status interaction). We discuss potential mechanisms underlying these effects, particularly transactional models of cognitive development. Transactional models predict that people in high-opportunity contexts actively evoke and select positive learning experiences on the basis of their genetic predispositions; these learning experiences, in turn, reciprocally influence cognition. The net result of this transactional process is increasing genetic influence with increasing age and increasing environmental opportunity.

Keywords
cognitive ability, intelligence, Gene × Environment interaction, behavior genetics, cognitive development

> *Intelligence is mostly a matter of heredity, as we know from studies of identical twins reared apart. . . . Social programs that seek to raise I.Q. are bound to be futile. Cognitive inequalities, being written in the genes, are here to stay, and so are the social inequalities that arise from them. What I have just summarized, with only a hint of caricature, is the hereditarian view of intelligence.*
>
> —Jim Holt, *New York Times* Sunday Book Review, March 27, 2009

In modern industrialized populations, cognition is approximately 50% to 70% *heritable* (Bouchard & McGue, 1981). This means that genetic differences between people account for 50% to 70% of the variation in performance on tests of cognitive abilities, such as reasoning, memory, processing speed, mental rotation, and knowledge. These heritability estimates are based on studies of identical and fraternal twins raised together, identical twins separated at birth and raised apart, and adoptive families. All of these designs hinge on the question of whether more genetically related individuals (e.g., biological siblings versus adoptive siblings) are also more similar in their cognitive ability. More recently, molecular genetic studies of unrelated persons have converged on similar heritability estimates (Chabris et al., 2012; Davies et al., 2011). Despite the vociferous objections of critics of behavioral genetic research (e.g. Charney, 2012), whether genetic differences between individuals account for variation in cognition is no longer a question of serious scientific debate. As McGue (1997, p. 417) commented, "That the debate now centres on whether IQ is 50% or 70% heritable is a remarkable indication of how the nature-nurture question has shifted."

These heritability estimates have been interpreted—both by scientists and by the lay public—to mean that environmental experiences have a minimal impact on cognition. In this article, we describe an alternative interpretation of what it means for cognition to be heritable: Rather than rendering environments impotent, genetic influences on cognition are the result of accumulating environmental experiences and depend on exposure to high-quality environmental contexts over time.

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An "Educational" Example: The Heritability of Educational Attainment in the 20th Century

To illustrate how genetic influences on psychosocial outcomes can depend on the environment, we begin with an example involving generational differences in educational attainment. After World War II, there was a dramatic expansion of access to education in Norway. In 1960, the average educational attainment for Norwegian adults was 5.92 years; by 2000, it was 11.86 years (Barro & Lee, 2000). This expansion was driven by postwar increases in government-sponsored student loans and by a social climate that increasingly valued education (Kuhnle, 1986). In contrast, prewar educational opportunities in Norway were less universal, and educational attainment was much more dependent on family social class. Over this same period, the heritability of educational attainment nearly doubled, from 40% for Norwegian male twins born before 1940 to approximately 70% for those born after 1940 (Heath et al., 1985).

If it were indeed the case, as suggested by the *New York Times* quote above, that heritability imposes an upper limit on the effectiveness of social change, then why would sweeping social changes be accompanied by an increase in both the level and the heritability of educational attainment? One explanation is that, as social opportunity increases, a person's educational attainment becomes increasingly a function of his or her individual characteristics—interests, motivation, work ethic, and scholastic aptitude—rather than social position. To the extent that these individual characteristics reflect genetic differences between people, however slight, then the net result of individuals' selecting their own educational paths is greater heritability of educational attainment. This explanation implies that heritability is maximized when people are free to select their own experiences. This same process may be a key mechanism for cognitive development.

Transactional Models of Cognitive Development

Gene-environment correlation—in which environmental experiences become sorted on the basis of individuals' genetically influenced traits—is not specific to educational attainment. Rather, behavioral genetic studies have found that a broad array of presumably "environmental" experiences—such as negative life events, relationships with parents, and experiences with peers—are themselves heritable (Kendler & Baker, 2007). That is, genetically similar people (such as monozygotic twins) experience more similar environments, whereas genetically dissimilar people (such as adoptive siblings) experience less similar environments.

Transactional models posit that these gene-environment correlations are key mechanisms of cognitive development. Early genetically influenced behaviors lead a person to select (and to be selected into) particular types of environments; these environments, in turn, have causal effects on cognition and serve to reinforce the original behaviors that led to those experiences. As Dickens and Flynn (2001, p. 347) stated, "higher IQ leads one into better environments causing still higher IQ, and so on." In addition to early cognitive ability, "noncognitive" traits, such as motivation and intellectual interest, may also lead children into cognition-enhancing environments (Tucker-Drob & Harden, 2012b). For instance, higher achievement motivation may lead students to enroll in more challenging courses, spend free time engrossed in intellectually stimulating activities, and engage parents, peers, and teachers in more sophisticated discourse.

Longitudinal research has documented bidirectional associations consistent with transactional processes. For example, not only does greater parental stimulation predict children's subsequent test scores, but children's test scores also predict higher subsequent stimulation by parents (e.g., Lugo-Gil & Tamis-LeMonda, 2008; Tucker-Drob & Harden, 2012a). Moreover, children's dispositions toward engaging with stimulating learning environments predict later test scores, and children's test scores predict their later dispositions toward learning (Marsh, Trautwein, Lüdtke, Köller, & Baumert, 2005). Such positive feedback loops may yield increasing dividends. If genes influence a child's early behaviors, even small initial genetic differences can be compounded via gene-environment correlation, leading to large estimates of genetic effects. In this way, the genetic effects on individual differences in psychological development can depend on reciprocal transactions with the environment. As Scarr and McCartney (1983) explained,

> We do not think that development is precoded in the genes and merely emerges with maturation. Rather, we stress the role of the genotype in determining which environments are actually experienced and what effects they have on the developing person. (p. 425)

Transactional models propose that genetic differences between people matter for cognition because initial genetic differences lead to different environmental experiences. The "end state" of this transactional process—high levels of and high heritability of cognitive ability—is therefore expected to differ depending on the quality and availability of environmental experiences. Thus, differences in heritability between groups can provide important information about the developmental processes undergirding cognition. Contemporary research in behavioral genetics of cognition has identified two dimensions along which heritability differs: age/development and socioeconomic advantage. Below, we summarize results from these two streams of research and describe how these results can be understood within the framework of transactional models.

Developmental Changes in Heritability

Children are born with all of their genes, and they experience an ever-wider array of environmental inputs as they develop. One might therefore expect that genetic variation will account for less and less variation in psychological outcomes with age. However, in contrast to this intuitive hypothesis, genetic influences on cognition actually *increase* substantially with age. Aggregated results from 11 unique longitudinal twin and adoption studies of cognition are shown in Figure 1. In infancy, genes account for less than 25% of the variation in cognition, whereas the shared family environment accounts for approximately 60%. By adolescence, however, genes account for approximately 70% of the variation in cognition, and the shared environment accounts for virtually no variation. These age-related patterns were identified in cross-sectional analyses originally by McCartney, Harris, and Bernieri (1990) and McGue, Bouchard, Iacono, and Lykken (1993), and more recently by Haworth et al. (2009).

We can understand the developmental increase in the heritability of cognition within the transactional framework. As children select and evoke experiences in line with their genetic predispositions, and as these experiences, in turn, stimulate their cognitive development, early genetic influences on cognition will become amplified. This compounding process is expected to become accelerated as children gain increasingly more autonomy in selecting their peer groups, afterschool activities, academic courses, and other positive learning experiences.

A second explanation for the developmental increase in heritability is that "new" genes that did not previously influence cognition may become activated later in development. For example, the biological changes of puberty may trigger changes in gene expression, or genetic differences that were not previously relevant for cognition may become relevant as children's social contexts change. In fact, both "new" gene activation and gene-environment transactions may contribute to developmental increases in the heritability of cognition, and the relative importance of each process may differ across the lifespan. Longitudinal behavioral genetic studies have indicated that activation of "new" genes may be the primary mechanism underlying increasing heritability in early childhood, whereas transactional processes may be the primary mechanism underlying increasing heritability in middle childhood and adolescence (Briley & Tucker-Drob, in press).

Unfortunately, much of what is known about the behavioral genetics of cognitive development has been derived from convenience samples of twins in the United States and from representative samples of twins from less racially and socioeconomically diverse populations. Thus, the trend of increasing heritability with age may not apply as well to groups with low socioeconomic status (SES). Next we discuss emerging research on the question of whether the heritability of cognition differs as a function of SES.

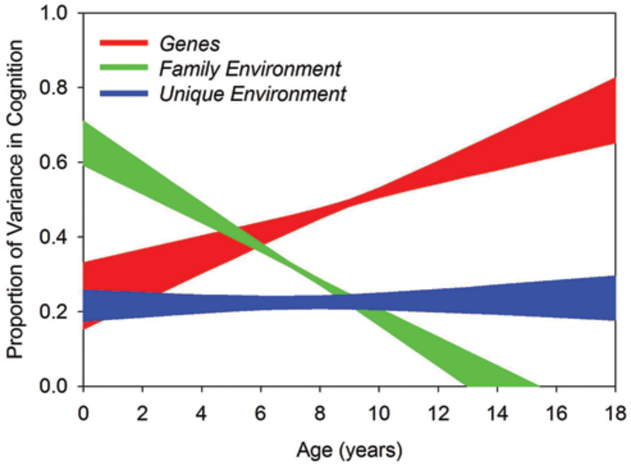

Fig. 1. Proportion of variance in cognition as a function of age. Shading around each line represents the imprecision of the estimate (± 1 *SE*). The family environment, often termed the shared environment, represents environmental influences that make siblings raised in the same family more similar to one another. The unique environment, often termed the nonshared environment, represents environmental influences that differentiate siblings raised in the same family. Data were aggregated from published reports, based on 11 unique longitudinal twin and adoption samples (weighted by the precision of the individual estimates): the Colorado Adoption Project (Petrill et al., 2004), the Early Childhood Longitudinal Study—Birth Cohort (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011), the Longitudinal Twin Study (Bishop et al., 2003), the Louisville Twin Project (McArdle, 1986), the MacArthur Longitudinal Twin Study (Cherny et al., 2001), a Moscow community sample (Malykh, Zyrianova, & Kuravsky, 2003), the Netherlands Twin Registry (Hoekstra, Bartels, & Boomsma, 2007; Polderman et al., 2006; van Soelen et al., 2011), the Twins Early Development Study (Davis, Haworth, & Plomin, 2009), and the Western Reserve Reading Project (Hart, Petrill, Deater-Deckard, & Thompson, 2007). Articles were identified by searching abstracts in PsycINFO. From the search results, we included longitudinal studies with samples of siblings with varying degrees of genetic relatedness, complete cross-time and within-time sibling correlations (or parameters from behavioral genetic models producing expectations for these correlations), measurement using an objective cognition/intelligence test, and participants under age 19 at both baseline and at least one follow-up measurement occasion.

Socioeconomic Differences in Heritability

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. SES, which is typically measured using parental income, educational attainment, occupational status, or some combination of the three, is an omnibus marker of the quality of environmental opportunity. In high-SES contexts, children have abundant opportunities to select and evoke positive learning experiences on the basis of their genetically influenced motivations and proclivities. In low-SES contexts, children are less likely to receive adequate opportunities for cognitively stimulating experiences, both at home and in school. For example, children from disadvantaged backgrounds typically have less access to enriching books and other learning materials, less rigorous academic experiences, and lower quality interactions with both peers and adults (Duncan & Murnane, 2011). Because low-SES contexts do not support transactional processes, it is predicted that genetic potentials for cognitive development are not fully realized (Bronfenbrenner & Ceci, 1994).

Indeed, research on Gene × SES interaction has indicated that genetic influences on cognition are suppressed by socioeconomic disadvantage. For children in low-SES contexts, the heritability of cognition approaches zero, whereas for children in advantaged contexts, genes account for as much as 80% of individual differences in cognition (see Fig. 2). This Gene × SES interaction has been found in young children (Scarr-Salapatek, 1971; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), adolescents (Harden, Turkheimer, & Loehlin, 2007; Rowe, Jacobson, & van den Oord, 1999), and adults (Bates, Lewis, & Weiss, in press). Moreover, although socioeconomic disparities in cognition and achievement are often interpreted as being the result of inequalities in education, Tucker-Drob, Rhemtulla, Harden, Turkheimer, and Fask (2011) found evidence for a Gene × SES interaction on infants' cognitive development between 10 months and 2 years of age, more than 3 years before the typical age of kindergarten entry. Specifically, for children in high-SES homes, genetic influences on cognition increased from approximately 0% at 10 months to 50% at 2 years, whereas for children in low-SES homes, genetic influences on infant cognition remained very close to 0% across the study period. That is, disadvantaged children did *not* show the expected developmental increase in the heritability of cognition. In follow-up work with this sample, a similar Gene × SES interaction was found on school-readiness skills (specifically mathematics) at age 4 years (Rhemtulla & Tucker-Drob, 2012). However, the interaction at 4 years was found to be entirely independent of the Gene × SES interaction earlier in development. This result suggests that Gene × SES interactions on cognition occur throughout infancy and early childhood, not because early life disadvantages have left indelible effects on cognition, but rather because low SES

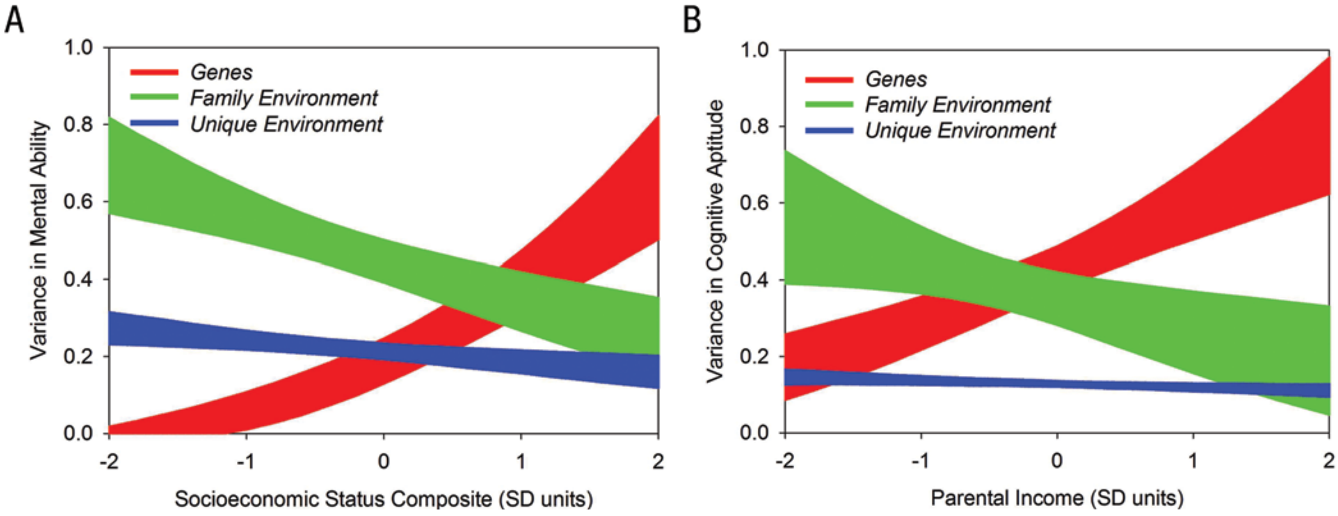

Fig. 2. Variance in mental ability as a function of SES in late infancy (age 2 years) (A). Data come from a nationally representative sample of American twins, 25% of whom lived below the poverty line (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). Variance in cognitive aptitude as a function of parental income in adolescence (age 17 years) (B). Data come from a positively selected sample of adolescent twins who sat for the National Merit Scholarship Qualifying Test (Harden, Turkheimer, & Loehlin, 2007), very few of whom were likely to be living in poverty. Because a Gene × SES interaction was detected in this more positively selected sample, Harden et al. (2007) concluded that "genotype-by-environment interactions in cognitive development are not limited to severely disadvantaged environments, as has been previously suggested." Shading around each line represents the imprecision of the estimate (± 1 *SE*). The family environment, often termed the shared environment, represents environmental influences that make siblings raised in the same family more similar to one another. The unique environment, often termed the nonshared environment, represents environmental influences that differentiate siblings raised in the same family.

children are recurrently exposed to poor environments that have novel, yet analogous, interactions with their genes at different ages.

Although a number of studies have replicated Gene × SES interactions on cognition, a handful of notable studies with sound designs have failed to replicate these effects (see Hanscombe et al., 2012 for a review). It is noteworthy that these failures to replicate have predominantly been in northern European nations, where social welfare systems are more comprehensive, whereas most of the positive results have been obtained in the United States, where social class differences in educational opportunity are vast. Socioeconomic disadvantage may not disrupt gene-environment transactions to the same extent in countries that ensure access to adequate medical care and high-quality education. Future research should identify the specific circumstances in which these Gene × SES interactions hold, by taking into account both macroenvironmental contexts (e.g., regional and national characteristics) and school- and family-level differences in economic opportunity and constraint.

Conclusions and Outlook

The results reviewed here suggest a provocative reconceptualization of the relationship between social opportunity and the magnitude of heritable variation in cognition. We began this article with a quote that illustrates the common view that heritability estimates provide an “upper bound” on the effects of social intervention—if cognition is very heritable, then the environment cannot matter as much. In fact, research on how the heritability of cognition differs across development and across context suggests that *genetic influences on cognition are maximized by environmental opportunity*. The highest heritability estimates are obtained for older children and adolescents from economically advantaged homes—that is, among children who have the autonomy to select environmental experiences consistent with their own interests and who have an array of high-quality experiences to choose from. As 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.

Distinguishing transactional processes from the “direct” influences of genes is more than a simple academic exercise. As Plomin, DeFries, and Loehlin (1977) wrote:

> Although formally it may not matter one whit in which way the effects of the genes are mediated, in practice it often matters quite a few whits, especially if one should happen to be interested in intervening in the process. (p. 321)

Indeed, child-driven transactions may be critical for intervention success. For example, Epps and Huston (2007) found that a poverty intervention changed parenting behaviors indirectly through effects on child behaviors; there was no immediate, direct effect of the intervention on parenting behaviors. In other words, the intervention was unable to directly influence parents to provide higher quality care but was able to change child behaviors to evoke more effective care from their parents. By determining the specific environmental transactions that amplify genetic influences across development and across contexts, researchers may uncover new opportunities for environmental intervention.

Recommended Reading

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Nisbett, R. E., Aronson, J., Blair, C., Dickens, W., Flynn, J., Halpern, D. F., & Turkheimer, E. (2012). Intelligence: New findings and theoretical developments. *American Psychologist*, 67, 130–159. A survey of the past 15 years of research on intelligence, including a section containing a review of empirical studies on “Social Class and Heritability of Cognitive Ability.”

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Tucker-Drob, E. M., & Harden, K. P. (2012b). See reference list. A recent study detailing a possible mechanism through which Gene × SES interactions might operate.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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