

# The Paradox of Intelligence: Heritability *and* Malleability Coexist in Hidden Gene-Environment Interplay

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Intelligence can have an extremely high heritability, but also be malleable; a paradox that has been the source of continuous controversy. Here we attempt to clarify the issue, and advance a frequently overlooked solution to the paradox: Intelligence is a trait with unusual properties that create a large reservoir of hidden gene–environment (GE) networks, allowing for the contribution of high genetic *and* environmental influences on individual differences in IQ. GE interplay is difficult to specify with current methods, and is underestimated in standard metrics of heritability (thus inflating estimates of “genetic” effects). We describe empirical evidence for GE interplay in intelligence, with malleability existing on top of heritability. The evidence covers cognitive gains consequent to adoption/immigration, changes in IQ’s heritability across life span and socioeconomic status, gains in IQ over time consequent to societal development (the Flynn effect), the slowdown of age-related cognitive decline, and the gains in intelligence from early education. The GE solution has novel implications for enduring problems, including our inability to identify intelligence-related genes (also known as IQ’s “missing heritability”), and the loss of initial benefits from early intervention programs (such as “Head Start”). The GE solution can be a powerful guide to future research, and may also aid policies to overcome barriers to the development of intelligence, particularly in impoverished and underprivileged populations.

## **Public Significance Statement**

This integrative review advances the idea that gene–environment interplay underlies differences in human intelligence, and that the high heritability of IQ is not merely a product of genetic variation. Gene–environment correlations and interactions are obscured by typical research approaches, but their contribution to the development of intelligence explains a paradoxical body of evidence from recent decades. Our proposed new approach has important implications for strategies to understand and promote intelligence.

*Keywords:* intelligence, heritability, gene–environment interplay, adoption, early intervention

When you have eliminated the impossible, whatever remains, however improbable, must be the truth.

—Sherlock Holmes in *The Sign of the Four* by Arthur Conan Doyle

## **The Debate Over the Heritability of Intelligence**

Some of us have blue eyes, while others have green or brown eyes. Some are tall and others are short. Some people are outgoing while others are shy. Individual variations are far-reaching and can be found in both physical and psychological traits. “Heritability” is a statistic that, as commonly interpreted, captures how much of the variation on a trait is due to genetic differences. Heritability can be estimated for any trait, and it ranges from 0.0 (meaning that the trait has no genetic component) to 1.0 (meaning that the trait is

completely heritable). For example, the heritability of breast cancer is 0.27; the heritability of body mass index is 0.59; and the heritability of Type 1 diabetes is 0.88 (Hyttinen, Kaprio, Kinnunen, Koskenvuo, & Tuomilehto, 2003; Lichtenstein et al., 2000; Silventoinen, Magnusson, Tynelius, Kaprio, & Rasmussen, 2008). Using similar methods, the heritability of general intelligence is estimated to be as high as 0.8 (although, as we discuss below, this value will vary depending on where and when it is estimated). To put that in perspective, the heritability of other “highly heritable” psychological traits rarely approach the level of IQ (see Bouchard, 2004, for extensive examples). The most comparable is schizophrenia (heritability of 0.64; Lichtenstein et al., 2009), while alcoholism (0.50), neuroticism (0.48; Riemann et al., 1997), and major depression (0.40; Sullivan et al., 2000) are markedly lower.

The high heritability of intelligence has captured the attention of many researchers across diverse disciplines, and has spurred a century-long debate which still endures (e.g., Gottfredson, 1997; Jensen, 1969; Lewontin, 1970; Tabery, 2014). In retrospect, that controversy generated more heat than light, and confusion is still widespread. Even within the field of psychology, many appear unclear about the implications of the heritability of IQ, and are unaware of the impact of gene–environment interplay on estimates

This article was published Online First October 30, 2017.

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of heritability. This confusion has profound functional implications. Not only is IQ a recognizably consistent measure, it also independently predicts real outcomes such as academic grades, income, social mobility, happiness, marital stability and satisfaction, general health, longevity, reduced risk of accidents, reduced risk of drug addiction, and reduced likelihood of committing violence and crimes (Gottfredson, 2001; Mackintosh, 2011). A clear understanding of the causes of variation in intelligence is critical for future research, and its potential applications to society are self-evident.

Recently, Robert Plomin, a prominent behavioral geneticist who has performed seminal studies on the heritability of intelligence, said in an interview with the magazine *The Spectator*:

At one time, people thought family members were similar because of the environment, but it turns out that the answer—in psychopathology or personality, and in cognition postadolescence—the answer is that it's all genetic! What runs in families is genetic! (Wakefield, 2013)

When the interviewer interjected that the environment *must* have an effect on what adults becomes, Plomin replied

I did an adoption study on weight, IQ and cognitive abilities, and parents who don't see their children after the first few hours of life are just as similar in terms of both weight and IQ to them after adolescence as are parents who reared their own kids. And adopted parents are zero similar! (Wakefield, 2013)

Plomin certainly did not mean “similar” in the highly deterministic way that the general public would have understood it. And, as we argue below, even what Plomin did mean by “similar” (as high independent genetic variance) is likely to be incomplete.

The influence of genes on IQ, are not as powerful or constrictive as might be assumed. As we describe here, intelligence seems to be quite malleable, and changes in the environment can, by *interacting* with genes, explain a great deal of differences in IQ across families, life span, socioeconomic status, and generations.

Here we will provide evidence and rationale for the conclusion that environmental interactions and correlations with genes (rather than genes alone) are key determinants of an individual's IQ. Although the role of gene–environment interplay (interaction and correlation) is not a new idea, even within the field of behavior genetics the implications of these effects for variations in traits is just beginning to be appreciated. As we will discuss, the gene–environment interplay in intelligence can be especially elusive, and is often disregarded or ignored, even by many in the immediate field (Finkel & Pedersen, 2016).

## An Improbable Solution Emerges: Theoretical Reasons for GE Interplay

### What Heritability is and What it is not

Genetic variation is a major determinant of most traits, and this maxim has been described as the “first law of behavior genetics” (Turkheimer, 2000). Ronald Fisher, one of the fathers of both population genetics and modern statistics, invented in the 1920s the now ubiquitous analysis of variance (ANOVA) as a way to break down the total variation of any trait into genetic and nongenetic components. Using this or other related statistical

methods, we can describe the simple split of variance in the following way:

$$VP = Vg + Ve$$

VP is the total variance (the squared deviation from the mean value) of the phenotype (or trait) in the population. Note that VP here is split into genetic factors and nongenetic factors:  $Vg$  is the variance associated with genetic differences in the population, and  $Ve$  is the variance of everything else, including shared environmental effects, unique environmental effects, epigenetic effects, and errors. Unfortunately, Ronald Fisher misleadingly described  $Ve$  as the “environmental variance,” while it is really the total variance left over after explaining a portion of the phenotypic variance with the genetic model (Templeton, 2006). Later, we discuss the importance of this seemingly subtle conceptual difference. From this, we can then represent heritability mathematically as  $Vg/VP$ . Or in plain English, heritability is the proportion of genetic variation in a trait in relation to the total variation. The more variation  $Vg$  can “capture” during the split, the higher the estimate of heritability.

While seemingly straightforward, in the natural environment there are many complications associated with the calculation of heritability. Often, we do not *know* which genes to focus on, and it is expensive (and interpretatively complex) to scan for the whole genome. In addition, we do not know the ways in which genes work together to produce variations in a trait. Due to these complexities, both human and nonhuman animal studies have historically used an indirect approach to estimate heritability: genetic *relatedness* as a proxy for genes per se. (Genetic relatedness is easier to measure because while we cannot specify the genes behind a trait, we typically *can* identify daughters, uncles, siblings, or other relatives.) The problem with this trick is that individuals who are related also tend to share environments. Do cousins have the same temperament because they are genetically similar or because they grew up with similar family traditions? For nonhuman animals raised under controlled conditions, researchers can split “genetic relatedness” from “shared environment” by distributing family members with known genetic variations into distinct environments (or the same environment for all different families, which accomplishes the same goal). For humans, we look for cases where these splits happened naturally, such as cases of adoption, siblings raised apart, and families with twins.

For the present purposes, we can limit our discussion now to the “twin method,” a strategy that is frequently used to estimate heritability in humans. The strategy exploited by twin studies is to compare monozygotic, “identical” twins (henceforth called “MZ twins”) to dizygotic, “fraternal” twins (henceforth called “DZ twins”). MZ twins share approximately 100% of genetic material, while DZ twins share on average 50%, just like any other siblings. The assumption of twin studies is that the shared family environment experienced by MZ and DZ twins will be relatively similar, while only the shared genetic material will differ. Therefore, the discrepancy between how “similar” MZ and DZ twins are can be used to estimate how heritable a trait is. For IQ scores, if the values among MZ twins correlate more than the values among DZ twins, this discrepancy in correlation is inferred to be the consequence of genetic influences.

Here is a simplified formula to calculate heritability based on differences between MZ and DZ twins:

$$\text{Heritability} = 2(r_{MZ} - r_{DZ})$$

$r_{MZ}$  = the average correlation of a measured trait among MZ twins  
 $r_{DZ}$  = the average correlation of a measured trait among DZ twins

Because the difference in the resemblance of MZ and DZ twins is due to the difference in sharing either 100% or 50% of genes, the difference between  $r_{MZ}$  and  $r_{DZ}$  is multiplied by 2, yielding an index of the proportion of the trait that is heritable. (For more on the twin method and other models for estimating heritability in humans, see [Tenesa & Haley, 2013](#)).

There are a number of concerns one can raise about the estimates of heritability obtained from twin studies. MZ twins usually share a placenta, while DZ twins never do, and this may cause different patterns of gene expression ([Gordon et al., 2012](#)). And of course MZ twins may be treated differently than DZ twins, or might be encouraged to make more similar decisions in life ([Kendler et al., 1993](#); [Richardson & Norgate, 2005](#)). But these often-discussed caveats (e.g., [Eysenck, 1979](#)) do not form the crux of our concerns here. The problems we have in mind are more substantive in nature, and they hinder people's appreciation of the malleability of intelligence as well as its gene–environment interplay. There is first a problem of interpretation: Heritability is *not* the equivalent of genetic determination. Heritability is an estimate of the causes of differences in a trait among people (a population statistic), whereas genetic determination is a matter of what causes a trait to be expressed in an individual. *Heritability is a measure of what is associated with variation in the trait (its causes of variation), and not a measure of what causes the trait.* Second, there is a problem sometimes overlooked even by experts: Estimates of heritability (owing to biases in the underlying calculations) grossly underestimate gene–environment interplay.

In some instances, genetically *determined* traits are associated with very low estimates of heritability. For example, the number of fingers on a human hand is genetically determined: The genes related to this trait code for instructions that lead to five fingers in almost everyone in any normal environment. However, the heritability of number of fingers in humans is very low. That is because the vast majority of variation in finger number is purely environmental, with traumatic amputations and prenatal complications being the leading causes (while genetic coding for other than five fingers is rare in humans). In contrast, traits that are not genetically determined can sometimes be highly heritable. For example, the tendency to vote in an election has a heritability of 0.53 ([Fowler et al., 2008](#)), and the affiliation to Democrat (liberal) versus Republican (conservative) parties in the U.S. has a heritability of 0.46 ([Settle et al., 2009](#)). If this strikes the reader as odd, remember: A heritable *characteristic* is *not* one that is passed down in the genes, and neither is political affiliation, at least not in any sense where this term has explanatory power. In fact, there might be dozens of correlations and interactions between genes and environmental factors such as wealth, education, and choices in life that contribute to one's political affiliation. Unfortunately, typical measures of heritability grossly underestimate GE interplay, as we explore next.

### First Dibs to Genetic Effects: Why Heritability can Underestimate GE Interplay

The construct of “general intelligence” (or the *g* factor) captures the remarkable phenomenon that performance across

diverse cognitive tasks are positively correlated. In other words, people who do well in one task tend to do well in many other tasks. This principle is well-supported by decades of evidence, and is a central feature of many (if not the majority of) modern theories of intelligence ([Mackintosh, 2011](#)). At first glance, the existence of general intelligence might suggest that one “thing” explains much of the variability in intelligence. Such a casual assumption would severely limit our appreciation of the basis for intelligence. It could bias us into a straightforward genetic view where there is no space (nor need) for a multitude of correlations and interactions with the environment. Although general intelligence is a single, abstract construct, it needn't be a single, concrete property of the brain. Instead, the higher-order construct of *g* may emerge from the interplay between a multitude of systems, including genetic *networks*, influences from psychological traits like working memory capacity, attentional capacity, motivation and personality, and reciprocal relations between cognitive and environmental processes (as held, e.g., by on the “mutualism” model of intelligence originally proposed in [van der Maas et al., 2006](#)). Intelligence is a hugely complex trait, and we should expect a high number of moving parts behind differences across individuals.

At this point, it is important to define “GE interplay.” GE interplay denotes both correlations as well as interactions between genes and environment. Gene–environment *correlation* means that individuals with particular genotypes for a trait are more likely to experience particular relevant environments. Meanwhile, a gene–environment *interaction* means that there is a difference in the *effect* of a given relevant environment on individuals depending on their genotypes for that trait. These effects will be explored in more detail below.

While historically ignored (or at least minimized), the study of GE interplay as a cause of variation in a trait is starting to gain momentum in our current scientific age of big data, complex networks, and systems biology ([Chandler, Chari, & Dworkin, 2013](#); [Geschwind & Konopka, 2009](#); [Lazer et al., 2009](#); [Philip Chen & Zhang, 2014](#); [Rockman, 2008](#)). Given their importance, we should recognize where GE interplays are captured in the estimation of heritability. The following formula is representative of a relatively complete model of heritability (and one that is used extensively in contemporary analyses):

$$VP = \underbrace{Ga + G \times G}_{\text{Genetic Variance}} + Ec + \underbrace{rGE + G \times E}_{\substack{\text{GE Interplay} \\ \text{Malleability}}} + Ee$$

As in the simpler formula described earlier, VP is again the total variance of the phenotype (trait) in the population. Genetic variance (formerly  $V_g$ ) is generally broken down into two parts: the variance associated with additive genetic variation ( $G_a$ ), and the variance associated with gene–gene interactions ( $G \times G$ ). Additive genetic variation is also known as “independent” genetic variation because it is the portion transmissible from a single parent to the offspring, no matter what the other parent contributes.  $E_c$  is the variance associated with shared environments in the population. (In human studies, it is the environment shared by siblings in the same family.) Making up what we call “GE interplay,”  $rGE$  is the variance associated with gene–environment correlations, while the  $G \times E$  is the variance associated with



gene–environment interactions.<sup>1</sup> Finally,  $E_e$  is everything else (now a smaller “everything else” compared with the earlier equation), which includes the variance associated with “unique” environments (in human studies, these are aspects of the environment that differ among siblings, such as being a firstborn vs. a later-born child), personal decisions (like when one sibling takes the initiative and “decides” to follow a cognitively challenging line of study), epigenetic effects (the regulation of DNA transcription without alteration of the original sequence, through mechanisms like DNA methylation, histone modifications and noncoding RNAs), and error.

Independent environmental effects ( $E_c$  and  $E_e$ ) as well as GE interplay (rGE and  $G \times E$ ) are the determinants of a trait’s “malleability.” The more the environment causes (directly or indirectly) the variation in a trait, the higher the trait’s malleability. Keep in mind, however, that even if a trait is not particularly malleable, interventions are still possible, albeit much harder to detect and/or implement. If there is no typical environment (within the normally encountered range of environments) that affects the trait, there may still be *special* environments (e.g., one created in response to our knowledge of the underlying biology) which can affect it. For example, the highly heritable intellectual disability that arises from phenylketonuria can be avoided if the carrier maintains a special diet that would not typically be encountered (Hanley, Clarke, & Schoonheydt, 1987).

As we discussed above, in the simple equation  $VP = V_g + V_e$ ,  $V_e$  is defined as the left-over variance (residual variance) that is not explained by  $V_g$ . In the complex equation, something similar happens: the indirect effects of genetic interactions, gene–environment correlations, and gene–environment interactions are relegated to the residual genetic variance left over that was not explained by additive genetic variance (Templeton, 2006). Because additive genetic effects ( $G_a$ ) are accounted for before rGE and  $G \times E$ , any contribution from genes to the variation in a trait will be overestimated as the direct effect from genes (Templeton, 2006). And, of course, any contribution from the environment to the variation in a trait will be underestimated because its fair share of the variance was already appropriated by  $G_a$ . In other words, Fisher designed the quantitative genetic parameters in his model in such a way that (as we like to say) *first dibs went to genetic effects*. This is a problem intrinsic to partitioning variance under any classic statistical tool (such as ANOVA or simple regression): Variance (outcomes) with multiple correlated sources (predictors) can be misrepresented as belonging to the source first/better specified in the analysis. In the case of intelligence, while researchers can precisely specify genetic relatedness, they do not understand, posit, and/or search for all of the rGE and  $G \times E$  sources, and so default to the assumption that the variance comes from  $G$ . As pointed by Tenesa and Haley (2013), lack of modeling frequently leads to an overestimation of genetic effects. And this problem is not limited to twin studies. Any model (e.g., the ACED model for the estimation of heritability) where GE interplay is not specified, there is a high risk of GE becoming incorporated into additive genetic effects, which of course bias the estimates of heritability (Tenesa & Haley, 2013).

What exactly are additive, independent genetic effects ( $G_a$ )? Does it imply that a single isolated gene produces the trait? In any literal sense, this cannot be true. If you put a strand of DNA double helix in a tube and wait, no distinguishable trait will emerge from it. And what about the GE interplay of gene–environment correlations (rGE) and gene–environment interactions ( $G \times E$ )? Do

these parameters merely refer to the evident truth that an egg needs both genes and environment to create an adult chicken? The answer is “no.” As we discussed earlier, these parameters are related to *causes of variation*, not deterministic causation. Additivity or independence is conceptually analogous to what a statistician would describe as a “main effect:” it is how much a predictor/input variable (a gene) relates to a predicted/output variable (a trait) when averaging across the other predictor variables (environment and other genes). An rGE effect is a statistical correlation between two predictor variables. In turn, a  $G \times E$  effect is conceptually close to a statistical interaction: it is when the effect of a variable (gene) with another variable (an environmental factor) has multiplicative consequences, with the result (variation in a trait) being more than the sum of its parts.<sup>2</sup>

Now that we understand what additive effects and GE interplay mean conceptually, it is useful to explore rGE and  $G \times E$  more concretely.

**Gene–environment correlation (rGE).** Behavior geneticists commonly classify the gene–environment correlation into three different types: passive, reactive (or evocative), and active (Dick, 2011; Plomin et al., 1977). The philosopher Ned Block (Block, 1995) gives an interesting example to illustrate these types. Suppose there are genes that predispose humans to musical abilities. Now suppose that children with those genes tend to have parents who provide them with an environment conducive to developing those abilities, including music lessons, concerts, access to an extensive music collection, musical discussion over dinner, and so forth. Assume also that the children who have a genetic disadvantage also have an environment that hampers their musical abilities (as could reasonably be expected, because their parents were likely to be musically disadvantaged). In this scenario, there will be a correlation between genes and environment that will move children toward both extremes of the distribution. These types of gene–environment correlations are called “passive covariance” because they do not depend on what the child does. Parents create a home environment that is influenced by their own heritable characteristics, which correlates with the genetic material they pass to their biological children. Passive covariance can be controlled in heritability estimates by using, for example, cases of adoption, because adopted children with musical ability genes will not be more likely to be raised by music aficionado parents. In contrast, “reactive covariance” is a matter of the environment *reacting* to

<sup>1</sup> Here we are treating all types of rGE and  $G \times E$  under the umbrella term of GE interplay. As noted in (Rutter & Silberg, 2002), these two forms of GE are commonly intertwined in real life, and are difficult to assess separately (though some modern statistical methods can help). In addition, it is likely that from an evolutionary perspective, both rGE and  $G \times E$  reflect adaptive effects of  $G$  on  $E$  as well as of  $E$  on  $G$ , so we should indeed expect both effects to contribute to individual differences (Rutter & Silberg, 2002). And more to the main argument developed here, rGEs and  $G \times E$ s are *together* a solution to explain why intelligence can possibly have high heritability as well as high malleability.

<sup>2</sup> Technically, the interaction in  $G \times E$  is similar, but not the same as a statistical interaction. A statistical interaction will only occur when there is variation in both  $G$  and  $E$  (Rutter & Silberg, 2002). Cases of potentially high  $G \times E$  where an important environmental factor is the same to most people (e.g., exposure to pollen when studying the trait of hay fever) will result in no statistical interaction (Rutter & Silberg, 2002). In addition, as we already noted, the interactions of  $G \times E$  can be masked by main, independent effects.

the person's traits, as when a school gives advanced tutoring classes to children who exhibit musical talent, or when friends devote a lot of time to help practicing for a performance. (In a real example, Tucker-Drob and Harden (2012) found a case of reactive covariance in children using a twin design. In it, genetic predispositions to higher cognitive abilities in 2-year-old children lead to more and higher quality cognitive stimulation by parents in the form of a dyadic task. This stimulation, then, led to children to a better reading ability at 4 years of age.) Finally, "active covariance" occurs when a person *creates* the correlation between genes and environment by self-selecting his or her experiences, as when a musically able child practices musical themes in her imagination or pays attention to the songs created by other musicians.

As noted by Block (1995), reactive as well as active covariance cannot be estimated without specific hypotheses about how the environment affects a trait. Due to our vast ignorance regarding the development and expression of human intelligence, a significant portion of rGE is mostly beyond the reach of current (and ethically permissible) methods in genetics. As we mentioned earlier, *when estimating the heritability of IQ, those gene-environment correlations that we do not recognize or do not know how to measure will be attributed to the genetic component.* (For a more sophisticated treatment on the genetics and mathematics of this issue regarding hypothetical traits, see Templeton, 2006.)

**Gene-environment interactions ( $G \times E$ ).** Distinct from cases of gene-environment correlations, gene-environment interactions mean that genotypes vary in their environmental *sensitivity*. Continuing the example on musical abilities, a musically talented child who frequently listens to a rap song might have her musical ability sharpened if she attends to the song's complexity and the depth of the lyrics. In contrast, a child who is musically impaired might exhibit no sharpening of musical ability when listening to the exact same song because she ignores all cognitively and artistically engaging aspects of the song, focusing instead only on the profanities. This is because during  $G \times E$  interactions, genetically different individuals have different experiences (i.e., pay attention to, absorb, or respond differently) to the same environmental stimulation.

Gene-environment interactions are hard to measure in studies of human traits because we do not know much about the genotypes and environmental states that are relevant to a given trait, much less how they react together (Bailey, 1997). And, as we have seen for the case of gene-environment correlations, cases of unspecified gene-environment interactions can also lead to overestimation of genetic effects (Tenesa & Haley, 2013).

As we discussed above, the method of estimating heritability adopts a convention that genes dominate environment; that which is not understood is (by mathematical default) attributed to genes! The genetic component gets first dibs on the causes of variation of a trait. In the earlier example on musical abilities, the estimate of heritability is likely to be quite high, although that estimate reflects a large contribution of hidden rGE and  $G \times E$ . In this case, the estimate of heritability is misleading, because what is doing much of the actual work in creating differences in musical ability is advanced tutoring, devoted friends, heavy practice, and inspiration from our culture. Heritability estimates will credit genes with creating the differences in musical abilities, but those differences would not exist if genes and environment were uncorrelated.

In fact, gene and environmental effects are quite hard to separate. Urie Bronfenbrenner and Stephen Ceci proposed a bioecological model where reciprocal causation between G and E during development (via processes that are repetitive, increasingly complex, and spread out over time, such as the ones on musical abilities presented earlier) create tightly knit synergistic effects (Bronfenbrenner & Ceci, 1994). They argued that heritability is frequently environmentally loaded, and that a heritability of .80 for IQ does not mean that there will only be 20% left for environmental influence. As Bronfenbrenner and Ceci (1994) write:

Actualized genetic potential involves substantial environmental components. Hence  $h^2$  cannot be interpreted as an estimate of the proportion of variance in a given developmental outcome that is completely free of environmental influence. On the contrary, environmental factors are seen as playing a major role in determining which individual capacities are realized and to what extent. (p. 583)

The convention of first dibs to genetics, however, is regarded by many behavior geneticists as a perfectly sensible strategy. An often-referenced passage in Roberts (1967) states that:

The genotype may influence the phenotype either by means of biochemical or other processes, labeled for convenience as "development," or by means of influencing the animal's choice of environment. But this second pathway, just as much as the first, is a genetic one; formally it matters not one whit whether the effects of the genes are mediated through the external environment or directly through, say, the ribosomes.

This assertion can be misleading and misrepresents the independent influence of the environment. If we define genetic effects this way, every single phenomenon on Earth that has been touched by a living organism could be said to be genetic! And this, to our dismay, is not far from what researchers with the gene-centric view of nature actually claim, such as the ethologist Richard Dawkins in his book *The Extended Phenotype* (see Jablonka, 2004 for a critical analysis of the book). At a nominal level, the assertion may be "true," but in such a trivial and vague way that it would have no explanatory value. The range of opportunities and stimuli that individuals might encounter are not merely static features of the environment always available to those genetically disposed to exploit them. As noted by the neurogeneticist Michael Meaney, heritability would be better described as representing the exclusively genetic influence on the variation of a trait if, and only if, there is zero Gene  $\times$  Environment contribution (Meaney, 2010).

### The Iceberg of Hidden Interactions on Evolutionarily Critical Traits

As we have seen, the hidden nature of rGE in the variation of intelligence comes from our inability to specify factors that contribute to active and reactive correlations. The hidden nature of  $G \times E$  reflects, like for rGE, a similar lack of knowledge on the modulation of intelligence. But in addition to that, as we see now, the search for  $G \times E$  is made even harder due to a probable "hidden iceberg" in the past and current evolution of intelligence. Evidence from evolutionary genetics strongly suggests that traits related to survival and reproduction (like intelligence) have a large reservoir of hidden variance in the form of interactions (Merilä & Sheldon, 1999). This type of diversity comes from both gene-gene

and gene–environment interactions, and is generally referred to as “hidden” variation because it has the potential to affect a trait, but is not expressed under typical/current conditions (Le Rouzic & Carlborg, 2008). These interactions capture genetic variation and accumulate mutations that stay latent for long periods of time, because natural selection is “blind” to anything other than additive, independent effects (Hermisson & Wagner, 2004). And because additive genetic variation is what fuels evolution by natural selection, important traits like intelligence might only continue to evolve at a fast pace because of constant new  $G \times G$  and  $G \times E$  hidden effects. A useful analogy to represent this is the iceberg. The tip of the iceberg (the independent genetic variance) is the only type of variance that can be seen by natural selection. And it is also the only effect that is not “residual” in most methods in quantitative genetics, like the ones seen for heritability here. Just as we see only the tip of the iceberg, the effects of interactions cannot be seen by natural selection and are much harder to detect during empirical measurements.

A recent meta-analysis on interventions to increase intelligence in humans (Protzko, 2015) suggested that “under increased demands from the environment (due to an intervention) we can raise IQ. Once those demands are removed, the system adapts to the new, reduced demands. Protzko’s conclusions suggest that  $G \times E$  interactions influence IQ in a way similar to muscular mass, where some people are more genetically prone to build muscle, but that tendency also continuously interacts with circumstances: if you play in a professional league, your environment will buff you further (until you get fired or retire), and if are an astronaut in the International Space Station, your environment will lead to muscular atrophy (until you return to Earth). So, one could ask: What are the advantages of being weak? And why aren’t all humans “blessed” with hypertrophied muscles cells like gorillas? No one knows for sure, but it is not hard to imagine how our ecology and history might have sometimes benefited weak humans. Or, better, humans that could match their strength according to their needs in a period of a few months (thus, conserving resources). Similarly, one could ask: What are the advantages of having a low IQ? There might be many. And maybe intelligence’s plasticity is useful to match its environment as to save precious resources from our energy-hungry brain.<sup>3</sup>

In fact, evolution is not only a part of our argument here on the hidden iceberg. The present article is also inspired by recent changes in the field of evolutionary biology. Some psychologists, and more so behavior geneticists, claim that there is nothing new about the recent evidence on GE interplay as it relates to the interpretation of what heritability means and to the interpretation of the broader questions regarding the mixing of nature and nurture in the development of complex traits. We disagree, and are immediately reminded of an analogy in the field of evolutionary biology. Studies of  $G \times G$  interactions were only recently possible, as were the assessment of epigenetic effects, niche construction, and interacting phenotypes (such as in the case for social evolution, seen in Moore, Brodie, & Wolf, 1997). These findings and ideas make up today what modern researchers call The Extended Evolutionary Synthesis, which provides a new framework to think about and understand evolutionary phenomena that differs from the gene-centric conception that dominated evolutionary thinking for almost a century (Pigliucci, 2007). The extended evolutionary synthesis revisits some neglected and/or unknown factors at play in evolu-

tion, including developmental processes, the role of epigenetic inheritance and plasticity, as well as networks of interactions between genes and environment (Danchin et al., 2011; Laland et al., 2015). In sum, the goal of the new synthesis is, in Danchin et al. (2011) words: “to go beyond DNA in order to build a broader conception of evolution.”

Something analogous, we imagine, could be in the making for the fields studying the genetics of psychological (and other complex) traits, and GE interplay might be critical to it. Intelligence is a prime candidate, since this trait presents a unique case of established moderate-to-high heritability while at the same time exhibiting remarkable malleability. Intelligence has potential for gigantic GE interplay, although the way this interplay manifests itself in the variation in IQ has contributed to its earlier dismissal (as we discuss in the next section).

We can conclude so far that there is not only a common problem with the interpretation of heritability’s *meaning* (genetic determination vs. genetic cause of variation), but heritability estimates themselves overlook indirect environmental/genetic effects. Furthermore, intelligence is a trait with a huge potential for gene–environment correlations and interaction given its vast psychological and biological complexity. But how do we *know* that the environment or GE interplay exerts a central influence on the variation of intelligence? In fact, a multitude of empirical evidence indicates that intelligence has a substantial malleability coexisting with its high heritability.

### Empirical Evidence for IQ’s Malleability on Top of its Heritability

Many studies provide strong empirical evidence for the malleability of IQ existing on top of its heritability. Tables 1, 2, and 3 provide a summary of some of the relevant data, and many of the articles included there are described in more detail below. The tables are not an exhaustive review of the relevant literature, which is quite extensive. A search of the National Library of Medicine for the terms “IQ” [or] “intelligence” [and] “genes” [and] “environment” returns 173 references, many of which are unrelated to the present topic (e.g., inbreeding or testosterone levels). Conversely, many relevant topics (such as early intervention effects on academic performance) are not returned in such a search. This literature is not only difficult to capture in a formal search, it is impractical to systematically summarize owing to the vastly different (and not quantitatively comparable) dependent measures relevant to this topic (e.g., “academic success,” “executive control,” “IQ”). Consequently, these tables are only intended to sum-

<sup>3</sup> The reader might assume that there is convincing evidence that intelligence is highly stable (i.e., not plastic) across the life span (Deary, 2014). And indeed, that is the case. However, that stability does not mean that actual cognitive capacity does not change (an adult is clearly superior to a child). Rather, it means that an individual’s IQ relative to his or her age cohorts tend not to change with age. While this is an interesting fact, it says little about the malleability of intelligence or the presence of GE interplay. The relative stability of IQ allows for changes in environmental factors and of GE interplay in the same way that it needs to allow for changes in genetic factors (given that humans are not born already expressing all their genes). As we describe below, massive environmental manipulations can underlie similarly large changes in the IQ of some individuals, despite the fact the environments and IQs are relatively stable for *most* individuals.



Table 1  
*Evidence for IQ Gains From Adoption and Immigration*

Authors	Sample/ <i>n</i>	Summary	Comments	See page
Schiff et al., 1982	32 males and females born to unskilled workers and abandoned at birth.	Low SES 4-month-olds adopted into high SES families (top 13 %) exhibit a 14-point increase in IQ in 5 years.	Confined to France. Dramatic increase in IQ (relative to match controls) instantiated by a simple shift in economic environment.	n/a
Duyme et al., 1999	65 4- to 6-year-old male and female adoptees.	Impoverished 4- to 6-year-olds were adopted into a range of SES families. In adolescence, adoption was associated with 7.7 (low SES) to 19.5 (high SES) point increases in IQ.	Confined to France. Degree of increase in IQ following adoption is dependent of degree of increase in SES status.	9
Kendler et al., 2015	436 full male siblings, separated at birth, tested at 18–20 years.	Pairs of separated siblings (one raised in biological family, one in adopted family) were compared. In adoptive families with $\geq 2.5$ steps higher education status than biological parents (i.e., the difference between no high school and some postsecondary education), the adopted-away siblings tested 7.6 IQ points higher.	Confined to Sweden, with <i>relatively</i> low income inequality.	9
van Ijzendoorn et al., 2008	Meta-analysis of 75 studies of 3,800 children from 19 countries.	Compared the intellectual development of children living in orphanages with that of children living with their adoptive families. On average, children growing up in orphanages had an IQ that was 16.5 points lower than their peers who were adopted.	Orphanages in countries with a higher Human Development showed a smaller detrimental effect in children's intelligence (reduction of 11.9 IQ points) than orphanages in countries with a lower Index (reduction of 21 IQ points).	10
Winick et al., 1975	205 female Korean orphans adopted in infancy.	Korean orphans (an underdeveloped country at the time) were adopted during early life by U.S. parents. After 6+ years, the mean IQ of the children who were malnourished prior to adoption was 10 to 40 points higher than the IQ of malnourished children living with their biological families in Korea.	Smaller increases in IQ were observed for adopted orphans who were initially moderately- or well-nourished.	10
Rutter, 1998	111 male and female adoptees (adopted prior to 24 months of age).	Children who had been adopted into families in the United Kingdom following severe privation (in Romania) for up to 24 months of life exhibited cognitive scores within the normal range at age 4 years (2 years post UK adoption). Children whose experiences of deprivation were limited to the first 6 months of life had cognitive scores that were lower than, but not statistically different from, early-placed, nondeprived adoptees.	The strongest predictor of level of cognitive functioning at 4 years was the children's age at entry to the United Kingdom.	n/a
O'Connor et al., 2000	111 male and female adoptees (adopted prior to 24 months of age).	Follow-up of sample from Rutter et al., 1998. Longitudinal data (4 and 6 years postadoption) were available on 111 Romanian adoptees placed into United Kingdom homes before 24 months of age. There was partial catch-up among late-placed Romanian children, but children adopted at an earlier age exhibited greater gains.	Improvements 4 years were maintained longitudinally, but there was no further evidence of recovery, suggestive of limits on the role of environment in the gene-environment interaction.	10
van Ijzendoorn et al., 2005	Meta-analysis of 18 studies; 17,767 multinational children.	Adopted children scored higher on IQ tests (+17.6 points) and academic performance than their nonadopted siblings or peers who stayed behind.	Although improved, school performance and language abilities lagged behind, and more adopted children developed learning difficulties.	10
Rindermann and Thompson, 2016	Comprehensive public data available from 92 nations.	Adult immigration (which typically represents migration from lower to higher SES environment) was associated with a 1–4 point increase in IQ over a 10-year period, with greater gains associated with immigration to countries with higher educational quality.	Small gains relative to infant/child adoption data, possibly due to the relatively smaller SES gains achieved by adult immigrants.	10

*Note.* Principal evidence for the malleability of intelligence by gene-environment interplay (i.e., observations that cannot be accommodated by an “all gene” or “all environment” interpretation). Unless noted, IQ values represent means. “See pages” denotes page numbers in the present article, and “n/a” indicates that the results were not discussed within this article.

Table 2  
*Evidence for Changes in Estimates of Heritability Across the Lifespan and Socioeconomic Status*

Authors	Sample/n	Summary	Comments	See page
Lyons et al., 2009	1,237 twins, followed from age 20–55.	Genes accounted for most of the stability in IQ from age 20 to age 55 (71.3% of the longitudinal correlation was genetic). However, a change in IQ of 10 points was observed in half the individuals, and a change of at least 20 points in one fifth of the individuals. These increases in IQ could be accounted for (83.1% of the longitudinal correlation) by aspects of the environment not shared by twins.	These results indicate that correlations (e.g., in the IQ of parents and children) can exist independently of changes in the mean values.	11
Turkheimer et al., 2003	320 pairs of 7-year-old twins.	Twins in low and high SES homes were assessed. The proportions of IQ variance attributable to genes and environment vary nonlinearly with SES. The authors' model suggests that in impoverished families, 60% of the variance in IQ is accounted for by the shared environment, and the contribution of genes is close to zero; in affluent families, the result is almost exactly the reverse.	The implication of this data is that correlational evidence of the heritability of IQ obtained from studies of adoptive families (which tend to be more homogeneous and affluent) is likely to underestimate the impact of family environment on a child's IQ.	12
Tucker-Drob et al., 2011	750 pairs of 10-month-old twins followed for 14 months.	Cognitive ability was measured at age 10 months and again at age 2 years. At 10 months, genes accounted for negligible variation in mental ability across all levels of SES. However, at 2 years, genes accounted for nearly 50% of the variation in mental ability of children in high-SES homes, but only negligible variation in mental ability of children raised in low-SES homes.	Estimates of heritability of intelligence increase with age, but do so primarily in environments that are more conducive to cognitive development.	12
Harden et al., 2007	839 pairs of adolescent twins	Shared environmental influences were stronger for adolescents from poorer homes, while genetic influences were stronger for adolescents from more affluent homes. Results suggest that environmental differences between middle- to upper-class families influence the expression of genetic potential for intelligence.	Similar to results reported by Turkheimer et al. (2003), with an older population of twins.	12
Tucker-Drob et al., 2013	Nonsystematic meta-analysis and modeling of data from multiple prior studies.	Genetic influences on cognition increase from infancy to adulthood, and these influences are maximized in more advantaged socioeconomic contexts (i.e., a Gene $\times$ Socioeconomic Status interaction). The authors argue 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.	Indicates an increasing genetic influence with increasing age and increasing environmental opportunity. Support the role of the gene-environment correlation.	13
Tucker-Drob and Bates, 2015	A meta-analysis of 14 studies comprising 24,926 twin pairs in the U.S., Western Europe, and Australia.	In developed countries, changes in heritability might depend on the environment that the poorest SES segments experience. In this article, the authors report that in the U.S. data, clear support exists for moderately sized gene $\times$ SES effects, with heritability increasing with SES. In Western Europe and Australia, gene $\times$ SES effects were close to zero.	The authors suggest that the different estimates of heritability in the U.S., Western Europe, and Australia might be due to the practice in the later countries of providing universal access to high quality education and health care.	n/a

*Note.* Principal evidence for the malleability of intelligence by gene-environment interplay (i.e., observations that cannot be accommodated by an "all gene" or "all environment" interpretation). Unless noted, IQ values represent means. "See pages" denotes page numbers in the present article, and "n/a" indicates that the results were not discussed within this article.



Table 3  
*The Flynn Effect Reflecting the Outcome of Gene–Environment Interactions and Correlations*

Authors	Sample/ <i>n</i>	Summary	Comments	See page
Flynn, 1984	Standardization samples of U.S. participants for intelligence scales from 1932 to 1978.	Found that every Stanford-Binet Scale, WISC, WAIS, WISC-R, WAIS-R, and WPPSI standardization sample from 1932 to 1978 established norms of a higher standard than its predecessor (i.e., tests increased in difficulty to maintain an average score of 100). This pattern indicates that Americans did progressively better on IQ tests over a 46-year period (gaining the equivalent of 13.8 IQ points).		13
Lynn and Hampson, 1986	~270,000 British citizens (across 10 studies) who took IQ tests multiple times across years.	Determined that national mean intelligence had been rising in Britain by 1.71 IQ points per decade between 1932 and 1982 (increases of 7.70 IQ points per decade between 1950 and 1980 were found in a separate analysis of Japanese samples).	In Britain IQ gains were greatest at the lower end of the intelligence distribution, indicating a contraction in the range of intelligence among the contemporary population.	13
Trahan et al., 2014	Meta-analysis (285 studies; <i>n</i> = 14,031) of the “Flynn effect” since 1951.	Across <i>industrialized</i> societies worldwide, a meta-analysis an average increase of 17.6 IQ points occurred between 1951–2011, translating to an average increase of approximately 2.9 IQ points per decade.	While the “Flynn effect” is a worldwide phenomenon, it is observed primarily in societies undergoing industrialization and modernization.	13
Dutton et al., 2017	Samples of 8- to 15-year-old Kuwaitis in 2006 ( <i>n</i> = 6,529) and 2015 ( <i>n</i> = 6,431) in 2015.	The Flynn Effect can reverse course during repeals of modern educational practices or deindustrialization. Here, the authors observed a negative Flynn Effect amounting to a loss of 6.2 points in a decade.	Based on consideration of several factors, the authors conclude that a shift to nonsecular education resulted in a general decline in interest in education and a less analytically-focused educational curriculum.	n/a

*Note.* Principal evidence for the malleability of intelligence by gene-environment interplay (i.e., observations that cannot be accommodated by an “all gene” or “all environment” interpretation). Unless noted, IQ values represent means. “See pages” denotes page numbers in the present article, and “n/a” indicates that the results were not discussed in this article.

marize some key results that cannot be accommodated by either the “all genes” or “all environments” approach to our understanding of intelligence.

### **IQ Gains From Adoption and Immigration**

Adoption studies allow researchers to separate the effects of the environment and genes by comparing an adopted child with his or her biological siblings who were not adopted, or with other peers who were left behind. Being raised by different parents changes not only a child’s family environment, but also all factors contributed by the neighborhood, peer, and school environments. These often-drastic environmental changes make adoption studies a particularly powerful method to assess the malleability of intelligence. It should be reiterated that if the change in environment is small, evidence can be found to support the all-gene view of IQ. However, by their design, these studies cannot capture the potential for IQ’s malleability (though they are certainly important to show its limits and granularity). Although we will discuss such studies where appropriate, our emphasis here is on studies that illustrate the potential for gene–environment interplay.

In a French adoption study, Duyme et al. (1999) examined a group of 65 impoverished children adopted relatively late in life (between 4 and 6 years of age) who had an average IQ of 77 before adoption. When measured during adolescence, these adopted children showed significant gains in IQ, and the new values were

significantly correlated with preadoption IQ (indicating that the measurements were reliable). The size of the gain was dependent on the socioeconomic status (SES) of the adoptive families: an average gain of 7.7 IQ points in low SES adoptive families and 19.5 IQ points in high SES adoptive families. These IQ gains are far from trivial. To put them in perspective, a successful college graduate is, on average, 15 IQ points above the average of 100, and a child is considered “gifted” in the U.S. if he or she is 30 IQ points above average.

Even in countries with very low inequality and relatively homogeneous sociocultural environment, changing from a relatively poor and uneducated family to a wealthier and educated family can substantially increase IQ. In a recent adoption study done in Sweden, Kendler et al. (2015) assessed the IQs of 436 pairs of separated siblings where at least one member was reared by biological parents and the other by adoptive parents. Adoption by parents with higher level of education was associated with a significant increase of 4.4 points in the child’s IQ in adulthood. Interestingly, the authors also found that in families with at least 2.5 steps higher education status than biological parents (which is the difference between no high school and some postsecondary education), the adopted-away siblings had 7.6 IQ points higher on average than their home-reared adopted siblings. On the other extreme, sibling sets in which the biological parental educational status was at least two steps higher than that of the adoptive

parents, the adopted-away siblings had an IQ on average 3.8 points lower than their home-reared siblings (Kendler et al., 2015). This result suggests some role of GE interplay, as it seems that higher biological parental education (which is itself likely to correlate strongly with the biological parents' IQ) was worse than higher adoptive parental education for stimulating intellectual development.

The studies above can be contrasted with a small study ( $n = 38$ ) involving a full cross-fostering design by Capron and Duyme (1989). Like in the above, the authors here found that the mean IQ of children reared in upper SES homes was significantly superior to those reared in low SES homes. The IQ gains from high SES environments was 12 points; smaller than the 19.5 points from Duyme et al. (1999), but still remarkably high! However, children born to upper SES families but adopted early into lower SES families had mean IQ of 107.5 whereas the mean IQ of children born into low SES families but adopted early into high SES families had a mean IQ of only 103.6 (Capron & Duyme, 1989). One would expect the means to travel extensively in the opposite direction if the environment was as potent as it appears in aggregated data because these contrasting environments (top vs. bottom 13% of French society) are several standard deviations apart. So, while these data are compatible with GE interplay, it suggests that additive genetic effects might sometimes be strong enough to counteract improvement in SES.

In a meta-analysis, van Ijzendoorn et al. (2008) considered 75 studies (totaling more than 3,800 children in 19 different countries) to compare the intellectual development of children living in orphanages with that of children living with adoptive families. On average, children growing up in orphanages had an IQ that was 16.5 points lower than their peers who were adopted. Not surprisingly, orphanages in countries with a higher Human Development Index (a combined measure of life expectancy, literacy, education, standards of living, and quality of life) had smaller detrimental effects on children's intelligence (reduction of 11.9 IQ points) than countries with a lower index (reduction of 21 IQ points). Also, children in orphanages with the most favorable caregiver-child ratio (maximally three children per caregiver) did not significantly differ from their adopted peers. These observations suggest that the typical orphanage has environmental conditions that are detrimental to the development of intelligence. In other words, environmental conditions related to orphanages are causes of variation in IQ.

Environmental effects on intelligence from international adoption are potentially much more powerful than those observed within a country or region. Economically prosperous countries can have up to nine times the GDP per capita of economically undeveloped nations. (By comparison, adoption within the U.S. from a low SES family to a higher SES family is commonly associated with a two to four times increase in family income.) The poorest 5% of the U.S. population, for example, are richer than 60% of the world (Milanovic, 2013). In addition, there are many other environmental factors relevant to IQ that differ drastically across nations, such as educational opportunities, parental expectations and pressure, motivation, a culture of intellectualism versus anti-intellectualism, the availability of cognitively demanding jobs, and so forth.

Winick et al. (1975) examined 205 Korean orphans (all of the viable cases from a single adoption service from 1959–1967) who were adopted during early life by U.S. parents, and divided cases

into three categories (according to the conditions of the children before adoption): malnourished ( $n = 59$ ), moderately nourished ( $n = 76$ ), and a control of well-nourished children ( $n = 70$ ). (Keep in mind that Korea as a whole was a poor and underdeveloped country at the time of the adoptions during the 1960s.) After at least 6 years with their American parents, the children were assessed for IQ. The mean IQ of the previously malnourished group was 102; the moderately nourished group, 106; and the well-nourished group, 112. Strikingly, the mean IQ of the children from the previously malnourished category was 10 to 40 points higher than the IQ of malnourished children living with their biological families in Korea or other poor populations (Galler et al., 1983; Hertzog et al., 1972; Liu et al., 2003; S. A. Richardson, 1976). In a similar analysis, O'Connor et al. (2000) examined 111 children from Romania who were adopted (after the collapse of the Soviet Union) by families in the United Kingdom at the age of 4. The authors found a considerable catch-up in children's cognitive abilities from the time at the adoption to just 2 years later, at age 6 (although these adopted Romanian children were still slightly below the average IQ of adopted United Kingdom children.)

A meta-analysis of 62 studies from a multitude of countries (totaling 18,000 adopted children) found an average increase in IQ of 17.6 points within several years of adoption (van Ijzendoorn et al., 2005)—a remarkable cognitive gain over their biological, nonadopted siblings and their peers who stayed behind. The size of the IQ gains described in this meta-analysis is higher than typical for studies of adoption. That, we believe, is because the meta-analysis considered studies where children came from extremely low SES and were adopted by families in a developed country (so, as pointed out above, they experienced a more dramatic environmental change than would be typical for within-nation adoptions). Furthermore, no significant differences were observed between the ultimate IQs of the adopted children and their environmental siblings/peers. In other words, the new environment made the initially "dull" children climb up the IQ ladder as high as the typical child in their new environment.

While early adoption associated with immigration can have a dramatic positive impact on IQ, the effects of adult immigration (absent adoption) are less encouraging. Rindermann and Thompson (2016) compared immigrants with natives worldwide across a 10- to 15-year period. Rindermann and Thompson reported that generally, immigration was associated with a small drift in IQ toward that of the native inhabitants. Depending on the quality of the host country's educational system and economic resources, increases in IQ of one to four points were typical; a level much lower than observed in typical adoption studies (Rindermann & Thompson, 2016). However, because most immigrants are not adopted (so would not obtain the benefits of adoption into higher SES families) and many are adults at the time of immigration, we might anticipate that the effects of immigration would be smaller than would accompany the subset of cases of immigration represented by adoption. Thus, Rindermann and Thompson's analysis suggests that while immigration can itself impact IQ, age at the time of immigration and access to the resources of the host country are likely (as described above) to influence the degree of change in IQ.

Now we are left with a thorny question: How exactly can environmental factors explain the huge IQ gains associated with adoption? It is well established that the effect of shared environment component,  $E_c$ , on IQ is usually low. In an elegant study by

Charles Murray, for example, he compared thousands of siblings who have grown up in the same home, with the same parents, but who have different IQs, and found little relevance of shared environment (Murray, 1998). More recent studies also estimate  $E_c$  to be low, fluctuating around 10%–30%, (e.g., Johnson et al., 2010; Lyons et al., 2009). And therein lies the problem: In the current models of heritability, environmental factors would need a colossal change in order to create such massive variations in IQ. The gains seen in adoption studies tend to be around 15 IQ points, or one standard deviation. Therefore, we would need an environmental improvement of five standard deviations to support a corresponding increase of 15 IQ points. And if we take into account that some environmental factors do not change much across SES and countries, the required improvement in relevant environments might be over six standard deviations. That is the equivalent of a boy from the slums of the Bronx in New York City being adopted by the CEO of a Fortune 500 company (a five standard deviation change in U.S. income distribution), or a girl from a lower-class family in Kenya immigrating to Norway (a six standard deviation change in global income distribution). Both examples are clearly too extreme to accommodate the typical case of adoption and immigration. To make matters worse, in most adoption studies that have measured it, shared environmental effects on IQ tend to fade by adulthood. Does that mean, then, that the environment has little room to be a relevant cause of variation in IQ? Yes, but only if we limit the consideration of environmental impact to  $E_c$ , or, in other words, only if we consider the independent effects of the (shared) environment. As we have seen, although it is recognized that gene and environment can be correlated, the assumption has been that it did not matter whether genes expressed themselves purely through biological mechanisms or through environmentally mediated paths. Genetics always got first dibs. However, as proposed by Dickens and Flynn (2001),  $G \times E$  correlations “and the mechanism we believe causes it radically alter the implications of heritability estimates for the potential effects of environment on IQ.” To put it another way, the synergistic (whole-more-than-the-sum) effects of  $G \times E$  interactions could easily explain the malleability of intelligence seen in adoption and immigration.

The evidence on intelligence’s malleability from adoption studies may appear to be in conflict with the common assertion that intelligence has highly heritability. As we saw, Plomin stated that

parents who don’t see their children after the first few hours of life are just as similar in terms of both weight and IQ to them after adolescence as are parents who reared their own kids. And adopted parents are zero similar! (Wakefield, 2013)

Similarly, psychologist Charles Locurto argued that standard adoption studies provide little evidence for the malleability of IQ (Locurto, 1990). He based his conclusion on the fact that the IQ of adopted individuals, even many years after the adoption, is still highly correlated with the biological parents (and, as we pointed earlier, the environmental influence of the adoptive family is drastically reduced). How can these patterns be reconciled with the massive IQ gains following adoption?

Indeed, substantial evidence shows that the correlations of adopted children with their biological parents are high, and the correlations with their adoptive family members are near zero by late adolescence

(examples in Horn, 1983; Plomin et al., 1997). However, this pattern of correlations does not imply that the impact from the environment must be small. In fact, massive IQ changes consequent to adoption are entirely compatible with high IQ correlations with biological parents. Put more generally, the magnitude of a correlation is independent of changes in means. (While this is a mathematical truth, its implications can be easily overlooked when interpreting correlations.) A recent study explored this issue empirically by performing a longitudinal assessment of intelligence in thousands of twins, first at age 20, and later, at age 55 (Lyons et al., 2009). Using standard methods for separating the causes of variation between genetic components, shared environment, and unique environment, the authors were able to infer that the genetic component was responsible for most of the observed stability in IQ from age 20 to age 55 (71.3% of the longitudinal correlation was genetic). There was, however, a change of 10 IQ points in half the individuals, and a change of at least 20 points in one fifth of the individuals. The authors concluded that these massive changes were overwhelmingly (83.1% of the longitudinal correlation) due to aspects of the environment not shared by twins. Therefore, genetic factors were primarily responsible for stability, and environmental factors were primarily responsible for changes in the actual value of IQ. Does that mean, then, that genes are at least the main force for the stability of IQ? As discussed next, the answer is “it depends.” The very correlation between genetic effects and IQ (or, in other words, the heritability of IQ) also varies across life span and SES.

### Heritability Changes Across Life Span and Socioeconomic Status

The heritability of body weight is quite high at 5 years of age (heritability = 0.95), but it typically *decreases* across the life span, plateauing at approximately 0.60 by 50 years of age. This decline in heritability reflects the fact that while genes set some initial parameters for body weight, lifestyle choices have more dominant later influence. While genetics can determine the physiological predisposition for body fat, such influence is less pronounced as lifestyle choices accumulate with age. The changing heritability of body weight illustrates that this trait is not genetically *determined*, but rather, is quite malleable despite the initially strong genetic influence.

Like body weight, intelligence is also subject to a change in its heritability across the life span. However, the pattern of change for intelligence is quite different than that observed for body weight, and many other traits. Intelligence can be reliably quantified beginning at about age 4–5. In populations of this age, the heritability of IQ is estimated at approximately 0.22. By 16 years of age, the heritability of IQ is estimated to be 0.62. Even more striking, by age 50 (at which time the heritability of body weight has declined precipitously), the heritability of IQ is commonly estimated to be 0.80 (with estimates ranging as high as 0.90; Bouchard, 1997; Haworth et al., 2009). This increase in heritability is not simply an artifact of changes in our ability to measure IQ. While IQ can be difficult to measure accurately at age 4–5, its measurement at age 16 is as reliable as it is later in life. Instead, the increase in IQ’s heritability with age probably reflects an underlying role of GE interplay in creating IQ differences between individuals.

Gene–environment interplay may underlie the increase in IQ’s heritability in a way that may not be immediately intuitive. Importantly, the genome is largely established at birth, so a population is not



gaining much genetic variation as it ages (except for age-dependent genes). This suggests that the increase in heritability across life span cannot be explained purely by changes in independent genetic effects. Alternatively, it is reasonable to expect that an individual's intelligence influences that individual's attraction toward a particular cognitive environment. Individuals with disparate intelligence are likely to find themselves pursuing very different cognitive challenges, while those with similar cognitive abilities are likely to gravitate to more similar cognitive challenges. Thus, those with similar IQs become *more* similar, while those with disparate IQs become less similar, with the net effect being an increase in the estimate of heritability. As this runaway process occurs (via the passive, active, and reactive rGE described above), genetic differences that underlie early differences in IQ can be amplified by the accumulation of cognitive challenges offered by different environments (Lykken, Bouchard, McGue, & Tellegen, 1993; McGue, Bacon, & Lykken, 1993). Children with slightly higher IQs end up mated with the environment (and choose an environment) that is appropriate for their cognitive abilities, which can in turn promote further gains in intelligence. Conversely, children with slightly lower IQs may gravitate toward less challenging environments, and may come to have relatively lower IQs as adults.

Part of the increase in heritability across life span could also be due to  $Ga \times Ec$  interactions as well, and not only correlations. Interactions with the shared/familial environment are known to inflate estimates of  $Ga$  for a trait (Lathrope, Lalouel, & Jacquard, 1984). The  $Ec$  behind IQ variation is quite high during infancy (around 0.55) and early adolescence (around 0.30), but falls to single digits by ages around 17 to 23 (Flynn, 2016). So, during late teenage years and early adulthood,  $Ec$  could be channeled toward heritability by interacting with differences in genes to explain the sudden and quick raise in IQ's heritability. And this process could be cumulative, as intelligence is expected to have a high number of  $G \times E$  interactions later in life (as feedback loops and networks accumulate), and have fewer of them early in life (which as we have described is the opposite for what is expected for heritability in most traits). This theme is also discussed by Tucker-Drob and Briley (2014), and may have repercussions for the way we understand the genetic stability of intelligence. As  $Ga \times Ec$  compound with age, the effects of shared environments become ever more tied to genotypic differences. The authors suggest that the accumulation of  $Ga \times Ec$

may also help to explain why the stability of the shared environment increases to such a high level. As recurrent objectively shared experiences increasingly differentiate individuals on the basis of their genes, it is possible that the only remaining shared environmental main effects are those that have resulted from particularly severe and lasting early environmental experiences that all humans respond similarly to (Tucker-Drob & Briley, 2014)

It is important to note that  $Ga \times Ec$  does not need to be completely cumulative. The reduction in  $Ec$  and the increase in heritability across age could, in part, be because as people age, both the environments as well as the genetic effects relevant to IQ get more diversified (due, respectively, to cognitively complexity of adult life, and to genes with late onset). That, in turn, can lead to more opportunities for  $Ga \times Ec$  to happen. (Remember that  $Ec$  is not only about the factors related to the household, but also about factors related to school quality, friendship circles, socioeconomic status, etc.). Hence, if for a 6-year-old many of the  $Ec$  and  $Ga$  do not interact, by age 18 the number and complexity of  $Ec$

and  $Ga$  is such that there are many more opportunities for "collisions" in  $Ga \times Ec$ . This  $Ga \times Ec$  would be interpreted as  $Ga$  and would inflate estimates of heritability in adulthood. Caution is required here, though. Heritability reaches its peak much later in life, whereas  $Ec$  has mostly faded by the early 20s. Hence, this noncumulative  $Ga \times Ec$  cannot in isolation explain all of IQ's heritability increase across life span.

To summarize, because methods in quantitative genetics usually give first dibs to genetics, the rGE and  $G \times E$  effects creating the IQ gap is counted as an independent genetic effect. Thus, the fact that IQ's heritability increases with age is, counterintuitively, evidence of GE interplay, a process described by others to reflect the "transactional model" of heritability (Tucker-Drob et al., 2013).

Interestingly, the same pattern of changes in heritability that occurs across life span happens across socioeconomic status. In a seminal study, Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman (2003) estimated genetic and environmental effects on IQ in 7-year-old twins in high- and low-SES families. About 25% percent of the families sampled had incomes below the U.S. poverty line (which comprises about 15% of the U.S. population), and the median annual income of the sample was equivalent today to \$37,000, which is moderately lower than the median income of the U.S. population as whole (\$52,000). Results from that study showed that among affluent families, most of IQ's variation was associated with genetic variation, and almost none was associated with shared familial environment (heritability of 0.72, with the rest associated with unique environments). However, among the poorest families, the reverse was true: most of variation in IQ was associated with the shared familial environment, and little with genetic variation (heritability of 0.10). These results suggest that differences in family background matter more when that background is relatively impoverished. Subsequent twin studies have found a similar pattern. Tucker-Drob, Rhemtulla, Harden, Turkheimer, and Fask (2011) measured cognitive abilities in infant twins, 25% of whom lived below the U.S. poverty line. At age 10 months, genes had almost zero effect on the variation in mental ability across all levels of SES, which is expected given what we have seen about changes in heritability across life span. At 2 years of age, children raised in high-SES homes (two standard deviations above the sample's mean SES) had genes accounting for nearly 50% of the variation in mental ability (with 35% of shared environment). On the other hand, 2-year-olds raised in low-SES homes (two standard deviations below the mean SES) had genes accounting for less than 5% of the variation in mental ability (with 70% of shared environment). In a third study, Harden, Turkheimer, and Loehlin (2007) assessed a sample of adolescent twins.<sup>4</sup> Among those from the lowest SES families (the lower 40th percentile of U.S. family income, equivalent to \$40,000 today), genetic influences accounted for 39% of the variance in cognitive abilities (with 45% attributed to shared environment). Meanwhile, among the twins from the wealthiest families (94th percentile of U.S. income, equivalent to \$200,000 today), genetic effects accounted for 55% of the variance in cognitive abilities, and 35% was attributable to shared environmental influences.

<sup>4</sup> Note that quantitative conclusions based on the studies of Turkheimer et al. (2003) and Tucker-Drob et al. (2011) are somewhat complicated by a lack of detail regarding the range of incomes sampled, or the average incomes of the upper SES brackets. However, the reported results are qualitatively consistent with those reported by Harden et al. (2007), and this latter study does not suffer from the same ambiguities.



The adolescents sampled were all takers of the National Merit Scholarship Qualifying Test, and so we expect very few to be living in extreme poverty. Because of that, [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.”

The results above suggest that environmental differences between low-, middle-, and high-SES families influence the expression of genetic potential for intelligence. Differences in genes are more accentuated in favorable environments, while on the other extreme, differences in familial environment are strongest for IQ’s variation among poor families. In fact, [Bronfenbrenner and Ceci \(1994\)](#) predicted these results and the importance of GE interplay in their bioecological model a decade before any empirical support (see discussion in section First Dibs to Genetic Effects). This pattern was confirmed in a more recent meta-analysis ([Tucker-Drob, Briley, & Harden, 2013](#)). Based on aggregated data from 11 studies that followed twin and adopted samples from birth to 18 years of age, [Tucker-Drob et al. \(2013\)](#) reported that in infancy, genes accounted for less than 25% of the variability in IQ, whereas the shared family environment accounted for approximately 60%. By late adolescence, this pattern had reversed, with genes accounting for 70% of the variability in IQ and the shared family environment accounting for near 0%. In the same article, [Tucker-Drob et al. \(2013\)](#) traced a beautiful parallel between the meta-analysis results for heritability changes across life span with previous results (already detailed here) of heritability changes across socioeconomic status by [Harden et al., 2007](#) and [Tucker-Drob et al., 2011](#). In the parallel made by [Tucker-Drob et al. \(2013\)](#), the graph for heritability by life span and the graph for heritability by SES are remarkably similar! In both cases, the heritability of IQ is low precisely when GE is expected to be low (in young age due fewer relevant experiences, and in poor populations due to lack of opportunities), while heritability of IQ is high when GE is expected to be high (in old age due to accumulated experience, and in wealthy populations due to more opportunities). It’s hard to imagine how Ga alone could have affected the continuum of SES and life span in a similar manner to explain the same pattern of heritability changes. Independent genetic effects cannot be the only important way in which genes affect IQ’s heritability— $G \times E$  and/or rGE effects matter.

Common descriptions give the misimpression that IQ’s heritability is always high, and that the shared environment plays only a trivial role.<sup>5</sup> Given the data on IQ and SES discussed above, the opposite is more likely true. In a global scale, 80% of humans are below the U.S.’s and Europe’s poverty lines (for an in-depth and insightful analysis on global inequality, see [Milanovic, 2013](#)), and so the shared environment is likely to be an extremely powerful cause of variation in intelligence in most populations. Moreover, it is plausible that these high values of the shared environment component reflect (the same way high values of the genetic component probably do) a high  $G \times E$  (and/or rGE) contribution to the estimate of heritability.

## The Flynn Effect

IQ scores are standardized, and so the average score of a population is necessarily 100. As tests are revised, the test maker designs each revision to maintain that average (based initially on large preliminary test samples). Consequently, if the population

were to become “smarter,” the test maker would need to increase the test’s difficulty in order to maintain the average score of 100. This is exactly what occurred throughout the 20th century, where in France for instance (where IQ scores were obtained for the entire population of 18-year-old males), IQ increased at least 20 points just between 1950 and 1980. Thus, although the average IQ score remained constant, the actual intelligence of test takers had increased. This phenomenon, referred to as the “Flynn Effect,” was first formally described in detail in 1984 by James Flynn ([Flynn, 1984](#)).

Although the Flynn effect is a worldwide phenomenon, it occurs predominantly in countries transitioning into what we consider today to be a “developed” society, both in social aspects like education and health access, as well as in economic aspects like per capita GDP and industrialization. In the United States, IQ increased by approximately 14 points between the years 1932 and 1978 ([Flynn, 1984](#)), and similar gains of three IQ points per decade were observed during the last century in France, Great Britain, the Netherlands, Australia, Canada, Germany, and Japan ([Lynn & Hampson, 1986](#)). In recent decades, gains in IQ also began to emerge in developing countries such as Turkey, Sudan, and Dominica ([Khaleefa et al., 2008](#); [Meisenberg et al., 2005](#); [Rindermann et al., 2013](#)). Worldwide, a meta-analysis of 53 studies conducted in industrialized societies showed an increase of 17.6 IQ points occurred between 1951 and 2011, translating to an average increase of approximately 2.9 IQ points per decade ([Trahan et al., 2014](#)). These increases are far from trivial. According to the Wechsler IQ classification scheme, this increase translates to an equivalent shift (when comparing across the 20th century) from “average” to “superior” intelligence.

Many explanations for the Flynn Effect have been considered, such as genetic heterosis (the “hybrid vigor” effect from miscegenation; [Mingroni, 2007](#)), improvements in nutrition/health ([Lynn, 2009](#)), reduced pathogen stress ([Eppig, Fincher, & Thornhill, 2010](#)), reduced family size ([Sundet, Borren, & Tambs, 2008](#)), and test-takers’ familiarity with formal testing methods ([Tuddenham,](#)

<sup>5</sup> Some have argued that the relationship between environmental and phenotypical variance has been tested for, and those tests generally show little or no reason to suspect a  $G \times E$  interaction. Were we to accept those earlier results at face value, they would be in clear conflict with the data reviewed above (as well as our broader conclusions). These arguments against  $G \times E$  influences are based on what has been described as studies of “environmentality” (e.g., [Plomin & DeFries, 1979](#); [Thompson, Determan, & Plomin, 1993](#)). To precisely perform such analyses, it would be necessary to quantify environmental variance. The difficulty arises in that while genetic variance (or its proxy, familial resemblance) can be precisely specified, estimates of environmental variance are at best qualitative. Environmental history is vague or unknown, it changes with time, the critical components of the environment are a matter of speculation, and any measurement of environmental variance would be “nominal” at best. Ronald Fisher was aware of this problem, and his revolutionary quantitative techniques were only able to split a trait’s variation by giving precedence to what could be known: the genetic effects (see section *First dibs to genetic effects*). In this same tradition, rather than directly measure environmental variance, studies of environmentality compute the ratio of genetic and environmental covariance to the phenotypic correlation, yielding estimates of bivariate heritability and environmentality. In other words, this strategy assumes that the “left over” environmental variance is the inverse side of heritability. Thus, it is unavoidable that environmental effects will necessarily be low; *all of the  $G \times E$  interaction (which was not empirically determined) was already assigned to  $Vg$ !*

1948). Although each of those factors may have some small explanatory value, they explain (both alone and in combination) only a small portion of the increase in intelligence throughout this era (Lynn, 2009; Woodley, 2011).

In contrast to the earlier attempts to explain this phenomenon, Dickens and Flynn (2001) described a model with high explanatory value in which people's IQs were shaped by both environment and genes, but in which environments were *matched* to individuals' IQs. Dickens and Flynn argued that, as we have seen here, "heritability" includes both a direct effect of the genotype on IQ and also indirect effects such that the genotype interacts with and shapes the environment, and that these *combined* influences determine an individual's ultimate cognitive capacity. That is, those with a greater IQ tend to seek stimulating environments that further increase IQ, and importantly, that more stimulating environments are available to those that reside in developed societies. These reciprocal effects result in GE interplay, or what Dickens and Flynn (2001) call "multiplier effects." There are many changes that come with a country's development that contribute to the Flynn Effect. James Flynn currently proposes that developed countries have more complex jobs, and have adopted a widespread, formal education focused in abstract and scientific thinking. To quote Flynn (Flynn, 2012):

Our ancestors lived in a world that was concrete and utilitarian. In 1900, schoolchildren were asked, "What are the capitals of the 46 states?" Today they are asked, "If rural representatives dominated a state legislature, where would they put the capital?" (The answer is that, because they hate big cities, they would put the state capital [of New York] in Albany rather than New York City.) *In other words, we take applying logic to hypothetical situations seriously.*

In a recent meta-analysis on the Flynn Effect, Pietschnig and Voracek (2015) examined 271 independent samples spanning one century and four million participants from 31 countries. The authors used multiple metaregressions on annual IQ test score changes, and included a wide range of predictor variables, including genetic miscegenation, blood lead levels, nutrition, pathogen stress, family size, test taking behavior, technology, education, and GE multipliers effects. After analyzing this extensive data set, the authors determined that IQ gains were most heavily associated with what they describe as hybrid factors (interacting biological and environmental factors), the most relevant of which were improvements of education, improvements in nutrition, and the GE multipliers proposed by Flynn (and discussed above). These results reinforce the conclusion that GE interplay is critical to the increases in intelligence that emerged across the 20th Century.

### Cognitive Aging

In normative terms, IQ tends to be stable across the life span, for example, an individual of average IQ at age 16 is likely to exhibit average IQ at age 90 (Deary, 2014). However, *absolute* performance on the IQ test declines precipitously as we age, such that by age 90, an individual will obtain a *raw score* (one that has not been age-normalized) that is comparable to a typical 8-year-old. This later effect (the impact of which can be observed across a multitude of cognitive tasks) is widely recognized and described as "cognitive aging" (Gerstorf et al., 2008; Salthouse & Ferrer-Caja, 2003). Numerous factors contribute to cognitive aging, some of

which are related to the organic brain impairments that accumulate with age (Fischer et al., 2010; Kramer et al., 2004; Salthouse & Ferrer-Caja, 2003).

Though the general decline in cognitive capacity from aging seems to be ultimately unavoidable, it may be possible to slow the rate of decline. For example, it seems that improvements in health care and general fitness, as well as cognitive stimulation, play a greater role in cognitive function among the elderly than it does for younger individuals. In a rather unique study, Staff et al. (2014) compared two groups from Scotland (the Aberdeen, 1921 and 1936 Birth Cohort) who were born in different decades. When contrasting both groups at age 77, the later, more "modern" group outscored the earlier group by 16.5 IQ points, which was a significant increase relative to their IQ gap recorded in their teens. What might account for this dramatic difference between the two generations? It is not hard to imagine that the elderly in the more modern group had access to more modern medicine, had better dietary habits, and tended to exercise more often. Another plausible environmental cause of variation in the IQ of older people is, of course, improved technology and greater access to it.

In a recent study, Bordone et al. (2015) contrasted cognitive tests taken by Europeans at around 60 years of age during multiple time points between 2002 and 2012. They found that cognitive test scores of individuals in later years were higher compared with those from earlier years, a result that is predicted by the Flynn Effect. What is remarkable here was that the study also quantified the individual's use of modern technology. The time frame that they considered (2002–2012) was one in which intense technological progress became widely available to the general population (note that the first "smart" phone was released in 2007). Indeed, test participants near the end of this time frame were significantly more likely to report use of modern technology such as PCs, the Internet, and mobile phones. Even after controlling for sex, age, and education, the authors found that the adoption of modern technology explained as much as 54% of the observed increase in IQ among the more modern sample. The authors concluded that "Part of the Flynn effect observed for the *older population* in Europe is caused by an increasing level of cognitive stimulation brought forth by the permeation of everyday lives with cognitive challenges related to technological innovation and interactive media" (Bordone et al., 2015). In accordance with these results, a qualitative review suggested that computerized cognitive training programs (classic cognitive training tasks as well as neuropsychological software and video games) can delay cognitive decline of older adults (Kueider, Parisi, Gross, & Rebok, 2012). Thus, although it is indisputable that aging is associated with a decline in general cognitive capacity, it is interesting to speculate that some of the age-related decline in cognitive capacity can be overcome by "cognitive training" and other environmental manipulations. While direct evidence with humans is still incomplete (Kramer et al., 2004), supporting experimental evidence has been obtained with laboratory animals (Markowska & Savonenko, 2002a, 2002b; Matzel et al., 2011; Matzel, Wass, Kolata, Light, & Colas, 2009; Tranter & Koutstaal, 2008).

### Early Education and Cognitive Engagement

Early education and programs focused on cognitive stimulation in children can have dramatic effects on later academic success.

Montessori preschools, for example, have a curriculum strongly based on developing executive functions, such as reducing disorder, impulsivity, and inattention, and promoting self-discipline, independent thinking, and orderliness. “Executive functions” (involving attention, impulse control, allocation of working memory, and orchestration of thought and action) are widely asserted to be core features of intelligence (Miller & Cohen, 2001). In one particular study, preschool age children were randomly picked from a lottery to enter a Montessori public school and were compared over time with individuals who were also in the lottery but were not picked (Lillard & Else-Quest, 2006). Upon completion of kindergarten (age 5) and sixth grade (age 12) Montessori students performed better on specific tests of executive functions than their peers who attended other schools during preschool. Furthermore, they received better grades in reading and math.

In a recent review, Diamond and Lee (2011) looked at studies of children 4- to 12-years-old who underwent intervention programs aimed at promoting executive functions, including progressively more challenging preschool school curricula, computer and non-computer games, and mindfulness training. The authors concluded that such training was overall associated with improved academic performance. Interestingly, participants of lower SES typically exhibited initially poorer executive functions, and these individuals benefited more from early intervention than those of initially higher SES or executive skills. This pattern is consistent with the idea we discussed before that the impact of  $G \times E$  and  $rGE$  on intelligence depends on a cognitively challenging environment.

While the studies on early education have not explicitly assessed the impact of early cognitive engagement on later IQ, we have already seen here that IQ is highly predictive of functional cognitive-dependent outcomes such as years of education, academic grades, income, and career prestige (Gottfredson, 2001; Mackintosh, 2011). Thus, it is easy to imagine that one of the benefits of these early intervention programs is at least in part a reflection of their effects on IQ. As support for such a possibility, it is established that IQ is strongly related to measures of cognitive control, such as selective attention and the implementation of working memory (Cowan, Fristoe, Elliott, Brunner, & Saults, 2006; Shipstead & Engle, 2013).

Based both on independent analyses and systematic reviews of available literature, the Nobel laureate in economics James Heckman has written extensively on the cognitive benefits of early education. In a summary of his analyses (<https://heckmanequation.org/resource/invest-in-earlychildhood-development-reduce-deficits-strengthen-the-economy/>), Heckman concludes that:

the highest rate of return in childhood development comes from investing [in education] as early as possible, from birth to age five in disadvantaged families. . . . Skills beget skills in a complementary and dynamic way. Those seeking to reduce deficits and strengthen the economy should make significant investments in early childhood education.

Heckman’s conclusions, even though based as much on speculation as actual data, are consistent with what Flynn described earlier as “multiplier effects,” that is, those with an initially greater IQ tend to seek stimulating environments that further increase IQ. The direct effect of the gene–environment interplay might initially be quite small, but the cycle of feedback could (over the course of

a lifetime) ultimately lead to a large increase in the IQ of the population.

Government-sponsored early intervention programs such as Head Start have been the subject of ongoing criticisms, echoed in the popular media, that their benefits appear to dissipate within a year or two of the program’s termination (DeParle, 1993). In a recent meta-analysis of the results from 39 independent studies, Protzko (2015) concluded that while early (and relatively brief) environmental interventions (such as Head Start) do raise the IQ of otherwise impoverished individuals, these beneficial effects are not sustained after the intervention ends. While these results may not be immediately encouraging, keep in mind that after an intervention, the participant is likely to return to the cognitively impoverished environment from which he or she was selected. But, as Protzko (2015) writes “The interventions show a strong effect on IQ that does not immediately snap back but instead gradually fades over years.” Absent the opportunity to assimilate into an environment that is matched to their new cognitive capacity (a forced loss in gene–environment correlation), it would be difficult to maintain or amplify the initial benefits afforded by the early intervention. Thus, much like the intergenerational Flynn effect, increases in IQ might be amplified, or at least sustained, by greater access to opportunities that often are inequitably distributed. In simpler terms, the analysis of Protzko (2015) should not lead us to conclude that early intervention programs such as Head Start can have no long-term benefits. Rather, these results highlight the need to provide participants with continuing opportunities that would allow them to capitalize on what might otherwise be transient gains in cognitive abilities.

### Implications of the Hidden GE Solution to the Missing Heritability of IQ

In pursuit of the actual genes that underlie causes of variation in complex traits, modern methods like genome-wide association studies (or GWAS) have been used to examine hundreds of thousands of DNA variants in thousands of individuals. Among complex behavioral traits and disorders, DNA variants identified so far usually explain, combined, less than 5% of individual differences (Manolio et al., 2009). In contrast, family studies, like the twin studies discussed above, typically report “genetic” influences accounting for 30% to 50% of individual differences in many behavioral traits (Turkheimer, 2000). The Big Five personality traits have heritabilities ranging from 0.40 to 0.60 (Bouchard, 2004), while autism spectrum disorder is currently estimated at 0.38 (Hallmayer et al., 2011), and schizophrenia at 0.64 (Lichtenstein et al., 2009). How is it that these metrics of heritability can be so high and yet the sum of DNA variants detected in GWA studies explain only a fraction of differences among individuals? Where is the remaining genetic variation? This question is widely known as the problem of the “missing heritability.”

As we have seen, heritability estimates of IQ can reach as high as 80% of the variance explained by “genetic” effects. However, few DNA regions from GWAS have emerged for intelligence, and all variants combined usually explain less than 8% of the total variance (Butcher et al., 2008; Loo et al., 2012; Rietveld et al., 2013). Disconcertingly, these DNA regions rarely replicate in independent samples, tend to have much lower effect sizes than reported in their original studies, and never account in isolation for



more than 0.2% of variance in IQ (Chabris et al., 2012; Manuck & McCaffery, 2014). In other words, despite intense efforts, genome-wide association studies of intelligence have failed to find the genes that account for the “genetic” variance predicted by twin studies.

In their efforts to resolve the “missing heritability” of IQ, many theoreticians have proposed that the problem resides in the lack of power to detect variants with very small effects (see DeYoung & Clark, 2012, for an extensive review). This explanation, however, assumes that causal variants act additively (again, meaning that the variation effect of each gene is independent from the others). Because of this, and because we can presume that variants with the largest effect were the first, or easiest, to be identified, the number of causal variants necessary to account for IQ’s heritability would be extremely high. Each genetic region from GWAS seems to explain, on average, less than 0.01% of the total variance in intelligence (Chabris et al., 2012). Given that estimate, the minimum number of causal variants necessary to explain IQ’s heritability would be over nine thousand! Regardless as to how many of these variants were to be actual protein-coding genes, this would still be a doubtfully high number of functional units in an evolutionary sense. Intelligence is a trait closely related to evolutionary fitness, and so each additive causal variant identified in the DNA is “visible” to natural selection. If a mutation leads to +0.5 IQ points independently, that mutation would have been strongly favored. However, if the effect of each additive variant is too small (e.g., a mutation leading to +0.001 IQ points), each mutation become effectively neutral because genetic drift then dominates the weak effects of selection, especially in small populations such as the ancestral populations of our species (for more on this phenomenon in evolutionary genetics and its implication to genetic additivity in general, see Goodnight, 1988; Ohta, 1973). Were this the case, for most of our evolutionary history (and perhaps even today) the causal variants behind the variation in human intelligence would have been under neutrality, at the whim of genetic drift. We think that even the most arduous proponents of “small, additive variants” as an explanation to IQ’s missing heritability would find it difficult to bite that particular bullet.

Instead of searching for ever smaller, additive DNA variants, another potential solution to the missing heritability problem involves the recognition of  $G \times E$  interactions.  $G \times E$  interactions could be diluting the real additive, independent genetic effects because exposures to a predisposing environment are limited in each study population and because the genetic variation examined has little effect outside that environment (Manuck & McCaffery, 2014).

As we have discussed, even though the concepts of biological networks with  $G \times G$ , rGE, and  $G \times E$  effects are not new, they are widely underappreciated. Even massive genome-wide association studies often evaluate only additive, independent genetic effects and completely ignore interactions.  $G \times E$  effects are only tested (and usually in follow-up and less visible studies) for those variables that had a statistically significant independent main effect. For example, Harlaar et al. (2005) used a large sample of 7-year-old twins to assess GE interplay in five DNA markers related to intelligence over a couple of simple ratings of family/household environments. First, the authors found GE correlations with their measures of the “proximate” family environment (disorganization and discipline in the household) rather than distal

measures (maternal education and father’s occupational class), which suggests reactive rather than passive rGE. More importantly, the authors also found significant GE interactions between the DNA markers and discipline, education and occupation (Harlaar et al., 2005). Although positive, these results had small effect sizes, and the DNA markers used were obtained by standard GWAS techniques that search for independent genetic effects. Maybe these additive markers might not represent the IQ genes with the largest interaction with the environments studied. Given the complexity of individual differences in intelligence, it is plausible that many DNA markers with  $G \times E$  effects might be epistatic markers or epigenetic markers. Unfortunately, genetic variations that have an interaction effects but no detectable main effects are almost universally missed in typical analyses. These nonadditive markers require complex study designs, and with such designs, some markers have been found in plants and animals for various traits (see examples in Goes, Sauce, & Peripato, 2012; Kooke et al., 2015; Sauce, de Brito, & Peripato, 2012; Zhao et al., 2017).

In a different study, Docherty, Kovas, and Plomin (2011) used a large sample of 10-year-old children to test the effect of 10 DNA markers for mathematical ability across multiple environments. There were 10 environmental measures related to the children’s homes and schools, such as levels of household disorganization, parental discipline, teacher’s quality, peer attitudes, and SES. (Those are relatively extensive measures in the current literature, though, of course, there is still many more slices of relevant environments that could potentially contribute.) The authors found significant  $G \times E$  effects in two of the DNA markers. Of course, this study suffers from a caveat described above, that is, the DNA markers studied are additive, not interaction markers.

Similar to what we have suggested here, Chaste and Leboyer (2012) have argued that a reason for the difficulty in detecting genes associated with autism (and the common inability to replicate those genes that *have* been identified) might happen due to an insensitivity to  $G \times E$  interactions. If the effect of a gene is observed in a sample of subjects frequently exposed to a particular environmental risk but not in those infrequently exposed, the source of nonreplication will remain a “known unknown.” But our capacity to detect genes behind a trait (and to make a priori predictions) could be increased if in designing studies, we attended more to the role of environment, a strategy that has begun to yield success in the study of plant phenotypes (Crossa, 2012; El-Soda, Malosetti, Zwaan, Koornneef, & Aarts, 2014).

In addition to incorporating specific models of the environment, the detection of  $G \times E$  would benefit from additional power (Dick et al., 2015; Manuck & McCaffery, 2014). However, as discussed above, the literature on evolutionary genetics indicates that traits critical to fitness (such as intelligence) will have relatively small additive genetic effects, but much bigger  $G \times G$  and  $G \times E$  effects. So, detecting  $G \times E$  may require still larger sample sizes, though,  $G \times E$  should not suffer from the problem of paradoxically small effects that are typically observed for  $G_a$ . Surprisingly, the  $G \times E$  solution is mentioned only briefly, when at all, in commentaries on missing heritability.

The solution of hidden GE interplay to the paradox of high heritability and high malleability of intelligence has, then, two interesting implications to IQ’s missing heritability. The first, named phantom heritability, implies that the true heritability of IQ



(the heritability estimated from only independent genetic effects, not interactions with the environment) is much lower than we think. In a computational model created by Bailey (1997), when gene–environment covariance was added to a model of causes of variation of intelligence, and heritability was estimated by the usual method, the accepted values of heritability were found to be inflated. In other words, the first dibs to genetic components overestimates the heritability of IQ that is attributable purely to genetics. In the context of genome-wide association studies, this means that the additive, independent genes that *have* been identified might explain a much greater percentage of a trait’s heritability than the reported values suggest (for more on phantom heritability, see Zuk et al., 2012). A second, related implication is that genome-wide association studies cannot find genes that contribute to substantial differences in IQ because they are primarily sensitive to additive, independent effects. As a complex trait that was critical to survival and reproduction, intelligence probably emerges from many hard to detect interactions that are hidden under the surface of the iceberg. Therefore, we are likely to identify many more genes of interest if we are sensitive to  $G \times G$ ,  $rGE$ , and  $G \times E$  interactions. Both of the above implications are interdependent. However, we believe that Implication 2 would lead to a more productive research path to understand individual differences in intelligence.

Searching for  $G \times G$ ,  $rGE$ , and  $G \times E$  effects in human intelligence will require a theory-driven approach with a substantial knowledge of the development and neuroscience of IQ. It will necessarily be resource-intensive, requiring time as well as behavioral, statistical, molecular, and computational techniques. Detecting interactions among genes and among genes and environment is an increasingly recognized problem in genetics (Manuck & McCaffery, 2014; Tyler et al., 2009). As pointed out by Eichler et al. (2010), solving the problem of missing heritability of complex traits will need methods that detect interactions implemented in a systems biology framework. The high-throughput technology of current genome-wide association studies alone will not solve this problem. The search for candidate  $G \times E$ , for example, could benefit greatly from a more thoughtful selection of genetic and environment variants, attention to scaling of the data (which can artificially deplete interaction effects), focused study designs, and so forth (for more details on these and other recommendations, see Dick et al., 2015). Fortunately, the first pieces for a new methodology are already on the horizon (Darabos et al., 2014; Fan et al., 2011; Hahn et al., 2003), with some promising results on the search for genes related to traits like pancreatic cancer (Duell et al., 2008) and cardiovascular disease (Bentley et al., 2010).

## Conclusions

Even though the heritability of intelligence is high (at least in some populations), evidence from multiple lines of research suggests that variation in intelligence is greatly affected by normal environmental variation. In other words, one can say that IQ has a high heritability *and* a high malleability.

The high heritability of intelligence could have emerged from independent genetic effects, while its high malleability could have arisen from independent environmental effects. However, in isolation, these possibilities have little explanatory value. Accordingly, because intelligence is demonstrably malleable, independent

genetic effects cannot possibly run the show. Likewise, because intelligence is demonstrably heritable, independent environmental effects cannot possibly run the show. This leads us to the conclusion that gene–environment interplay is the ring master. While seemingly straightforward, this conclusion has been sublimated by methodological/conceptual biases (the first dibs to genetics) and its elusive nature (the hidden iceberg of interactions). Here, we have presented evidence that the GE solution is theoretically and empirically sound, even though at first glance it seems improbable. Paraphrasing Sherlock Holmes’ maxim: Because we have eliminated the now implausible options, whatever remains, however well hidden, must be the truth (or at least a closer approximation).

However, we are reminded of a view that has prevailed for many decades. As noted above, many have argued (e.g., Roberts, 1967) that the “real” cause of the observed variations in intelligence has nothing to do with interactions. By this argument, nongenetic “causes” are not causes at all because they act on genetic differences. Genetics could be influenced or amplified by the environment, but the underlying *cause* would still be genetic. We disagree with this premise. Absent the availability of a rich, diverse environment to choose from, the amplification process could not be engaged. In this regard, the environment matters a great deal. In an analogous case, one could say that all prejudice, opportunity, favoritism, economic circumstance, services, education, and so forth are not causes of observed variations in wealth among adults, which instead, is solely the product of the inheritance of wealth. While inheritance may be principal contributor to wealth, of course people sometimes succeed from the bottom up, while others can fail from the top down. Wealth inequality can be substantially affected in a country that has more opportunities for those who inherited less wealth, as it can be affected by personal decisions and of course, luck. (In genetic studies on IQ, this is considered part of the  $Ee$  effect. For a detailed, and novel exploration of the role of personal decision on changes in IQ, see Flynn, 2016).

Differences in the environment, we believe, are also causes of variations in intelligence. The environment that humans created has as much potential information and sources of causation as the DNA sequences that were created by evolution. The “potentiality” in the genes of 100,000 BCE individuals of our species (if they were transported in a time machine to modern age) is the same type of potentiality in the environment of modern civilization (if a human ancestor of a different species, such as an Australopithecus, was raised here and then returned to his peers). Or as elegantly pointed out by Purcell (2002)

$G \times E$  is often conceptualized as genetic control of *sensitivity* to different environments. A related phenomenon, G-E correlation ( $rGE$ ) represents genetic *control* of exposure to different environments. Equivalently, of course,  $G \times E$  is the environmental control of differential gene effects, whereas  $rGE$  is the environmental control of gene frequency.

Unfortunately, it is sometimes easy to miss this equivalency about GE interplay.

There is no absolute answer to the question “What *is* the heritability of IQ?” The answer to this question is “it depends.” It depends on not only the genetic contribution to IQ, but also on the degree and type of variability (or lack of variability) in the environment (see Sternberg, 1997, for practical implications). Much of the confusion and trepidation that is associated with the topic of

intelligence in general, and intelligence research in particular, could be averted if we distanced ourselves from a reductionist view of biology/psychology (for a recent discussion on these trepidations, see Hayden, 2013). Given the present state of evidence, we must reconsider the prevailing view on the heritability of intelligence, replacing it with one that acknowledges its malleability together with its heritability, as well as the principal role played by GE interplay. Such a view is critical to future research efforts and may guide the field into new, exciting paths. Understanding how these “extra-genetic” influences interact with our genes will not only contribute to the elucidation of intelligence, but as importantly, may provide critical insights into policies to overcome barriers to the development of intelligence, particularly in impoverished, underprivileged, and otherwise neglected populations.

## References

- Bailey, R. C. (1997). Hereditarian scientific fallacies. *Genetica*, *99*, 125–133. <http://dx.doi.org/10.1007/BF02259516>
- Bentley, J. P., Asselbergs, F. W., Coffey, C. S., Hebert, P. R., Moore, J. H., Hillege, H. L., & van Gilst, W. H. (2010). Cardiovascular risk associated with interactions among polymorphisms in genes from the renin-angiotensin, bradykinin, and fibrinolytic systems. *PLoS ONE*, *5*, e12757. <http://dx.doi.org/10.1371/journal.pone.0012757>
- Block, N. (1995). How heritability misleads about race. *Cognition*, *56*, 99–128. [http://dx.doi.org/10.1016/0010-0277\(95\)00678-R](http://dx.doi.org/10.1016/0010-0277(95)00678-R)
- Bordone, V., Scherbov, S., & Steiber, N. (2015). Smarter every day: The deceleration of population ageing in terms of cognition. *Intelligence*, *52*, 90–96. <http://dx.doi.org/10.1016/j.intell.2015.07.005>
- Bouchard, T. J. (1997). IQ similarity in twins reared apart: Findings and responses to critics. In R. J. Sternberg & E. L. Grigorenko (Eds.), *Intelligence: Heredity and Environment* (pp. 126–160). New York, NY: Cambridge University Press.
- Bouchard, T. J., Jr. (2004). Genetic influence on human psychological traits. A Survey. *Current Directions in Psychological Science*, *13*, 148–151. <http://dx.doi.org/10.1111/j.0963-7214.2004.00295.x>
- Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, *101*, 568–586. <http://dx.doi.org/10.1037/0033-295X.101.4.568>
- Butcher, L. M., Davis, O. S. P., Craig, I. W., & Plomin, R. (2008). Genome-wide quantitative trait locus association scan of general cognitive ability using pooled DNA and 500K single nucleotide polymorphism microarrays. *Genes Brain & Behavior*, *7*, 435–446. <http://dx.doi.org/10.1111/j.1601-183X.2007.00368.x>
- Capron, C., & Duyme, M. (1989). Assessment of effects of socio-economic status on IQ in a full cross-fostering study. *Nature*, *340*, 552–554. <http://dx.doi.org/10.1038/340552a0>
- Chabris, C. F., Hebert, B. M., Benjamin, D. J., Beauchamp, J., Cesarini, D., van der Loos, M., . . . Laibson, D. (2012). Most reported genetic associations with general intelligence are probably false positives. *Psychological Science*, *23*, 1314–1323. <http://dx.doi.org/10.1177/0956797611435528>
- Chandler, C. H., Chari, S., & Dworkin, I. (2013). Does your gene need a background check? How genetic background impacts the analysis of mutations, genes, and evolution. *Trends in Genetics*, *29*, 358–366. <http://dx.doi.org/10.1016/j.tig.2013.01.009>
- Chaste, P., & Leboyer, M. (2012). Autism risk factors: Genes, environment, and gene-environment interactions. *Dialogues in Clinical Neuroscience*, *14*, 281–292.
- Cowan, N., Fristoe, N. M., Elliott, E. M., Brunner, R. P., & Saults, J. S. (2006). Scope of attention, control of attention, and intelligence in children and adults. *Memory & Cognition*, *34*, 1754–1768. <http://dx.doi.org/10.3758/BF03195936>
- Crossa, J. (2012). From genotype × environment interaction to gene × environment interaction. *Current Genomics*, *13*, 225–244. <http://dx.doi.org/10.2174/138920212800543066>
- Danchin, É., Charmantier, A., Champagne, F. A., Mesoudi, A., Pujol, B., & Blanchet, S. (2011). Beyond DNA: Integrating inclusive inheritance into an extended theory of evolution. *Nature Reviews Genetics*, *12*, 475–486. <http://dx.doi.org/10.1038/nrg3028>
- Darabos, C., White, M. J., Graham, B. E., Leung, D. N., Williams, S. M., & Moore, J. H. (2014). The multiscale backbone of the human phenotype network based on biological pathways. *BioData Mining*, *7*, 1. <http://dx.doi.org/10.1186/1756-0381-7-1>
- Deary, I. J. (2014). The stability of intelligence from childhood to old age. *Current Directions in Psychological Science*, *23*, 239–245. <http://dx.doi.org/10.1177/0963721414536905>
- DeParle, J. (1993, March). Sharp criticism for head start, even by friends. *The New York Times*. Retrieved from <http://www.nytimes.com/1993/03/19/us/sharp-criticism-for-head-start-even-by-friends.html>
- DeYoung, C. G., & Clark, R. (2012). The gene in its natural habitat: The importance of gene-trait interactions. *Development and Psychopathology*, *24*, 1307–1318. <http://dx.doi.org/10.1017/S0954579412000727>
- Diamond, A., & Lee, K. (2011). Interventions shown to aid executive function development in children 4 to 12 years old. *Science*, *333*, 959–964. <http://dx.doi.org/10.1126/science.1204529>
- Dick, D. M. (2011). Gene-environment interaction in psychological traits and disorders. *Annual Review of Clinical Psychology*, *7*, 383–409. <http://dx.doi.org/10.1146/annurev-clinpsy-032210-104518>
- Dick, D. M., Agrawal, A., Keller, M. C., Adkins, A., Aliev, F., Monroe, S., . . . Sher, K. J. (2015). Candidate gene-environment interaction research: Reflections and recommendations. *Perspectives on Psychological Science*, *10*, 37–59. <http://dx.doi.org/10.1177/1745691614556682>
- Dickens, W. T., & Flynn, J. R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review*, *108*, 346–369. <http://dx.doi.org/10.1037/0033-295X.108.2.346>
- Docherty, S. J., Kovas, Y., & Plomin, R. (2011). Gene-environment interaction in the etiology of mathematical ability using SNP sets. *Behavior Genetics*, *41*, 141–154. <http://dx.doi.org/10.1007/s10519-010-9405-6>
- Doyle, A. C. (1890). *The Sign of the Four*. Philadelphia, PA: Lippincott's Monthly Magazine.
- Duell, E. J., Bracci, P. M., Moore, J. H., Burk, R. D., Kelsey, K. T., & Holly, E. A. (2008). Detecting pathway-based gene-gene and gene-environment interactions in pancreatic cancer. *Cancer Epidemiology, Biomarkers & Prevention*, *17*, 1470–1479. <http://dx.doi.org/10.1158/1055-9965.EPI-07-2797>
- Dutton, E., Bakhiet, S. F., Essa, Y. A. S., Blahmar, T. A., & Hakami, S. M. A. (2017). A negative Flynn effect in Kuwait: The same effect as in Europe but with seemingly different causes. *Personality and Individual Differences*, *114*, 69–72. <http://dx.doi.org/10.1016/j.paid.2017.03.060>
- Duyme, M., Dumaret, A. C., & Tomkiewicz, S. (1999). How can we boost IQs of “dull children?” A late adoption study. *Proceedings of the National Academy of Sciences of the United States of America*, *96*, 8790–8794. <http://doi.org/10.1073/pnas.96.15.8790>
- Eichler, E. E., Flint, J., Gibson, G., Kong, A., Leal, S. M., Moore, J. H., & Nadeau, J. H. (2010). Missing heritability and strategies for finding the underlying causes of complex disease. *Nature Reviews Genetics*, *11*, 446–450. <http://dx.doi.org/10.1038/nrg2809>
- El-Soda, M., Malosetti, M., Zwaan, B. J., Koornneef, M., & Aarts, M. G. M. (2014). Genotype×environment interaction QTL mapping in plants: Lessons from Arabidopsis. *Trends in Plant Science*, *19*, 390–398. <http://dx.doi.org/10.1016/j.tplants.2014.01.001>

- Eppig, C., Fincher, C. L., & Thornhill, R. (2010). Parasite prevalence and the worldwide distribution of cognitive ability. *Proceedings Biological Sciences/The Royal Society*, 277, 3801–3808. <http://dx.doi.org/10.1098/rspb.2010.0973>
- Eysenck, H. J. (1979). *The Structure and Measurement of Intelligence*. Berlin, Heidelberg: Springer Berlin Heidelberg. <http://dx.doi.org/10.1007/978-3-642-67075-6>
- Fan, R., Zhong, M., Wang, S., Zhang, Y., Andrew, A., Karagas, M., . . . Moore, J. H. (2011). Entropy-based information gain approaches to detect and to characterize gene-gene and gene-environment interactions/correlations of complex diseases. *Genetic Epidemiology*, 35, 706–721. <http://dx.doi.org/10.1002/gepi.20621>
- Finkel, D., & Pedersen, N. L. (2016). Gene-Environment Interplay in Adulthood. *Behavior Genetics*, 46, 1–3. <http://dx.doi.org/10.1007/s10519-015-9775-x>
- Fischer, H., Nyberg, L., Karlsson, S., Karlsson, P., Brehmer, Y., Rieckmann, A., . . . Bäckman, L. (2010). Simulating neurocognitive aging: Effects of a dopaminergic antagonist on brain activity during working memory. *Biological Psychiatry*, 67, 575–580. <http://dx.doi.org/10.1016/j.biopsych.2009.12.013>
- Flynn, J. R. (1984). The mean IQ of Americans: Massive gains 1932 to 1978. *Psychological Bulletin*, 95, 29–51. <http://dx.doi.org/10.1037/0033-2909.95.1.29>
- Flynn, J. R. (2012, February). Thinking in more sophisticated ways. *The New York Times*. Retrieved from <http://www.nytimes.com/roomfordebate/2012/02/26/are-people-getting-dumber/thinking-in-more-sophisticated-ways>
- Flynn, J. R. (2016). *Does your family make you smarter?: Nature, nurture, and human autonomy*. New York, NY: Cambridge University Press. <http://dx.doi.org/10.1017/CBO97811316576694>
- Fowler, J. H., Baker, L., & Dawes, C. T. (2008). Genetic variation in political participation. *The American Political Science Review*, 102, 233–248. <http://dx.doi.org/10.1017/S0003055408080209>
- Galler, J. R., Ramsey, F., Solimano, G., Lowell, W. E., & Mason, E. (1983). The influence of early malnutrition on subsequent behavioral development. I. Degree of impairment in intellectual performance. *Journal of the American Academy of Child Psychiatry*, 22, 8–15. <http://dx.doi.org/10.1097/00004583-198301000-00002>
- Gerstorff, D., Siedlecki, K. L., Tucker-Drob, E. M., & Salthouse, T. A. (2008). Executive dysfunctions across adulthood: Measurement properties and correlates of the DEX self-report questionnaire. *Neuropsychology, Development, and Cognition Section B, Aging, Neuropsychology and Cognition*, 15, 424–445. <http://dx.doi.org/10.1080/13825580701640374>
- Geschwind, D. H., & Konopka, G. (2009). Neuroscience in the era of functional genomics and systems biology. *Nature*, 461, 908–915. <http://dx.doi.org/10.1038/nature08537>
- Góes, C. P., Sauce, B., & Peripato, A. C. (2012). Milk ejection in mice LG/J x SM/J. *Mammalian Genome*, 23, 770–779. <http://dx.doi.org/10.1007/s00335-012-9431-1>
- Goodnight, C. J. (1988). Epistasis and the effect of founder events on the additive genetic variance. *Evolution: International Journal of Organic Evolution*, 42, 441–454. <http://dx.doi.org/10.1111/j.1558-5646.1988.tb04151.x>
- Gordon, L., Joo, J. E., Powell, J. E., Ollikainen, M., Novakovic, B., Li, X., . . . Saffery, R. (2012). Neonatal DNA methylation profile in human twins is specified by a complex interplay between intrauterine environmental and genetic factors, subject to tissue-specific influence. *Genome Research*, 22, 1395–1406. <http://dx.doi.org/10.1101/gr.136598.111>
- Gottfredson, L. S. (1997). Mainstream science on intelligence: An editorial with 52 signatories, history, and bibliography. *Intelligence*, 24, 13–23. [http://dx.doi.org/10.1016/S0160-2896\(97\)90011-8](http://dx.doi.org/10.1016/S0160-2896(97)90011-8)
- Gottfredson, L. S. (2001). Intelligence and the American ambivalence toward talent. In N. Colangelo & S. G. Assouline (Eds.). *Talent development IV: Proceedings from the 1998 Henry B. and Jocelyn Wallace National Research Symposium on Talent Development* (pp. 41–58). Scottsdale, AZ: Great Potential Press.
- Hahn, L. W., Ritchie, M. D., & Moore, J. H. (2003). Multifactor dimensionality reduction software for detecting gene-gene and gene-environment interactions. *Bioinformatics*, 19, 376–382. <http://dx.doi.org/10.1093/bioinformatics/btf869>
- Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., . . . Risch, N. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry*, 68, 1095–1102. <http://dx.doi.org/10.1001/archgenpsychiatry.2011.76>
- Hanley, W. B., Clarke, J. T., & Schoonheydt, W. (1987). Maternal phenylketonuria (PKU)—A review. *Clinical Biochemistry*, 20, 149–156. [http://dx.doi.org/10.1016/S0009-9120\(87\)80112-1](http://dx.doi.org/10.1016/S0009-9120(87)80112-1)
- Harden, K. P., Turkheimer, E., & Loehlin, J. C. (2007). Genotype by environment interaction in adolescents' cognitive aptitude. *Behavior Genetics*, 37, 273–283. <http://dx.doi.org/10.1007/s10519-006-9113-4>
- Harlaar, N., Butcher, L. M., Meaburn, E., Sham, P., Craig, I. W., & Plomin, R. (2005). A behavioural genomic analysis of DNA markers associated with general cognitive ability in 7-year-olds. *Journal of Child Psychology and Psychiatry*, 46, 1097–1107. <http://dx.doi.org/10.1111/j.1469-7610.2005.01515.x>
- Haworth, C. M., Wright, M. J., Luciano, M., Martin, N. G., de Geus, E. J. C., van Beijsterveldt, C. E. M., . . . Plomin, R. (2009). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, 15, 1112–1120.
- Hayden, E. C. (2013). Ethics: Taboo genetics. *Nature*, 502, 26–28. <http://dx.doi.org/10.1038/502026a>
- Hermisson, J., & Wagner, G. P. (2004). The population genetic theory of hidden variation and genetic robustness. *Genetics*, 168, 2271–2284. <http://dx.doi.org/10.1534/genetics.104.029173>
- Hertzog, M. E., Birch, H. G., Richardson, S. A., & Tizard, J. (1972). Intellectual levels of school children severely malnourished during the first two years of life. *Pediatrics*, 49, 814–824.
- Horn, J. M. (1983). The Texas adoption project: Adopted children and their intellectual resemblance to biological and adoptive parents. *Child Development*, 54, 268–275. <http://dx.doi.org/10.2307/1129690>
- Hyttinen, V., Kaprio, J., Kinnunen, L., Koskenvuo, M., & Tuomilehto, J. (2003). Genetic liability of type 1 diabetes and the onset age among 22,650 young Finnish twin pairs: A nationwide follow-up study. *Diabetes*, 52, 1052–1055. <http://dx.doi.org/10.2337/diabetes.52.4.1052>
- Jablonska, E. (2004). From replicators to heritably varying phenotypic traits: The extended phenotype revisited. *Biology & Philosophy*, 19, 353–375. <http://dx.doi.org/10.1023/B:BIPH.0000036112.02199.7b>
- Jensen, A. R. (1969). How much can we boost IQ and scholastic achievement? *Harvard Educational Review*, 39, 1–123. <http://dx.doi.org/10.17763/haer.39.1.13u15956627424k7>
- Johnson, W., Deary, I. J., Silventoinen, K., Tynelius, P., & Rasmussen, F. (2010). Family background buys an education in Minnesota but not in Sweden. *Psychological Science*, 21, 1266–1273. <http://doi.org/10.1177/0956797610379233>
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics*, 23, 21–27. <http://dx.doi.org/10.1007/BF01067551>
- Kendler, K. S., Turkheimer, E., Ohlsson, H., Sundquist, J., & Sundquist, K. (2015). Family environment and the malleability of cognitive ability: A Swedish national home-reared and adopted-away cosibling control study. *Proceedings of the National Academy of Sciences of the United States of America*, 112, 4612–4617. <http://dx.doi.org/10.1073/pnas.1417106112>
- Khaleefa, O., Abdelwahid, S. B., Abdulradi, F., & Lynn, R. (2008). The increase of intelligence in Sudan 1964–2006. *Personality and Individual Differences*, 45, 412–413. <http://dx.doi.org/10.1016/j.paid.2008.05.016>



- Kooke, R., Johannes, F., Wardenaar, R., Becker, F., Etcheverry, M., Colot, V., . . . Keurentjes, J. J. B. (2015). Epigenetic basis of morphological variation and phenotypic plasticity in *Arabidopsis thaliana*. *The Plant Cell Online*, 27, 337–348. <http://dx.doi.org/10.1105/tpc.114.133025>
- Kramer, A. F., Bherer, L., Colcombe, S. J., Dong, W., & Greenough, W. T. (2004). Environmental influences on cognitive and brain plasticity during aging. *The Journals of Gerontology, Series A: Biological Sciences and Medical Sciences*, 59, M940–M957. <http://dx.doi.org/10.1093/gerona/59.9.M940>
- Kueider, A. M., Parisi, J. M., Gross, A. L., & Rebok, G. W. (2012). Computerized cognitive training with older adults: A systematic review. *PLoS ONE*, 7, e40588. <http://dx.doi.org/10.1371/journal.pone.0040588>
- 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. *Biological Sciences*, 282, 20151019. <http://dx.doi.org/10.1098/rspb.2015.1019>
- Lathrope, G. M., Lalouel, J. M., & Jacquard, A. (1984). Path analysis of family resemblance and gene-environment interaction. *Biometrics*, 40, 611. <http://dx.doi.org/10.2307/2530905>
- Lazer, D., Pentland, A., Adamic, L., Aral, S., Barabasi, A.-L., Brewer, D., . . . Van Alstyne, M. (2009). Social science. Computational social science. *Science*, 323, 721–723. <http://dx.doi.org/10.1126/science.1167742>
- Le Rouzic, A., & Carlborg, O. (2008). Evolutionary potential of hidden genetic variation. *Trends in Ecology & Evolution*, 23, 33–37. <http://dx.doi.org/10.1016/j.tree.2007.09.014>
- Lewontin, R. C. (1970). Further remarks on race and the genetics of intelligence. *Bulletin of the Atomic Scientists*, 26, 23–25. <http://dx.doi.org/10.1080/00963402.1970.11457808>
- Lichtenstein, P., Holm, N. V., Verkasalo, P. K., Iliadou, A., Kaprio, J., Koskenvuo, M., . . . Hemminki, K. (2000). Environmental and heritable factors in the causation of cancer—Analyses of cohorts of twins from Sweden, Denmark, and Finland. *The New England Journal of Medicine*, 343, 78–85. <http://dx.doi.org/10.1056/NEJM200007133430201>
- Lichtenstein, P., Yip, B. H., Björk, C., Pawitan, Y., Cannon, T. D., Sullivan, P. F., & Hultman, C. M. (2009). Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: A population-based study. *Lancet*, 373, 234–239. [http://dx.doi.org/10.1016/S0140-6736\(09\)60072-6](http://dx.doi.org/10.1016/S0140-6736(09)60072-6)
- Lillard, A., & Else-Quest, N. (2006). The early years. Evaluating Montessori education. *Science*, 313, 1893–1894. <http://dx.doi.org/10.1126/science.1132362>
- Liu, J., Raine, A., Venables, P. H., Dalais, C., & Mednick, S. A. (2003). Malnutrition at age 3 years and lower cognitive ability at age 11 years: Independence from psychosocial adversity. *Archives of Pediatrics & Adolescent Medicine*, 157, 593–600. <http://dx.doi.org/10.1001/archpedi.157.6.593>
- Locurto, C. (1990). The malleability of IQ as judged from adoption studies. *Intelligence*, 14, 275–292. [http://dx.doi.org/10.1016/S0160-2896\(10\)80001-7](http://dx.doi.org/10.1016/S0160-2896(10)80001-7)
- Loo, S. K., Shtir, C., Doyle, A. E., Mick, E., McGough, J. J., McCracken, J., . . . Nelson, S. F. (2012). Genome-wide association study of intelligence: Additive effects of novel brain expressed genes. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51, 432–440.e2. <http://dx.doi.org/10.1016/j.jaac.2012.01.006>
- Lykken, D. T., Bouchard, T. J., Jr., McGue, M., & Tellegen, A. (1993). Heritability of interests: A twin study. *Journal of Applied Psychology*, 78, 649–661. <http://dx.doi.org/10.1037/0021-9010.78.4.649>
- Lynn, R. (2009). What has caused the Flynn effect? Secular increases in the Development Quotients of infants. *Intelligence*, 37, 16–24. <http://dx.doi.org/10.1016/j.intell.2008.07.008>
- Lynn, R., & Hampson, S. (1986). The rise of national intelligence: Evidence from Britain, Japan and the U.S.A. *Personality and Individual Differences*, 7, 23–32. [http://dx.doi.org/10.1016/0191-8869\(86\)90104-2](http://dx.doi.org/10.1016/0191-8869(86)90104-2)
- Lyons, M. J., York, T. P., Franz, C. E., Grant, M. D., Eaves, L. J., Jacobson, K. C., . . . Kremen, W. S. (2009). Genes determine stability and the environment determines change in cognitive ability during 35 years of adulthood. *Psychological Science*, 20, 1146–1152. <http://dx.doi.org/10.1111/j.1467-9280.2009.02425.x>
- Mackintosh, N. (2011). *IQ and Human Intelligence*. Oxford, UK: Oxford University Press.
- Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorf, L. A., Hunter, D. J., . . . Visscher, P. M. (2009). Finding the missing heritability of complex diseases. *Nature*, 461, 747–753. <http://dx.doi.org/10.1038/nature08494>
- Manuck, S. B., & McCaffery, J. M. (2014). Gene-environment interaction. *Annual Review of Psychology*, 65, 41–70. <http://dx.doi.org/10.1146/annurev-psych-010213-115100>
- Markowska, A. L., & Savonenko, A. (2002a). Protective effect of practice on cognition during aging: Implications for predictive characteristics of performance and efficacy of practice. *Neurobiology of Learning and Memory*, 78, 294–320. <http://dx.doi.org/10.1006/nlme.2002.4064>
- Markowska, A. L., & Savonenko, A. (2002b). Retardation of cognitive aging by life-long diet restriction: Implications for genetic variance. *Neurobiology of Aging*, 23, 75–86. [http://dx.doi.org/10.1016/S0197-4580\(01\)00249-4](http://dx.doi.org/10.1016/S0197-4580(01)00249-4)
- Matzel, L. D., Light, K. R., Wass, C., Colas-Zelin, D., Denman-Brice, A., Waddell, A. C., & Kolata, S. (2011). Longitudinal attentional engagement rescues mice from age-related cognitive declines and cognitive inflexibility. *Learning & Memory*, 18, 345–356. <http://dx.doi.org/10.1101/lm.2034711>
- Matzel, L. D., Wass, C., Kolata, S., Light, K., & Colas, D. C. (2009). Age-related impairments of new memories reflect failures of learning, not retention. *Learning & Memory*, 16, 590–594. <http://dx.doi.org/10.1101/lm.1503209>
- McGue, M., Bacon, S., & Lykken, D. T. (1993). Personality stability and change in early adulthood: A behavioral genetic analysis. *Developmental Psychology*, 29, 96–109. <http://dx.doi.org/10.1037/0012-1649.29.1.96>
- Meaney, M. J. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, 81, 41–79. <http://dx.doi.org/10.1111/j.1467-8624.2009.01381.x>
- Meisenberg, G., Lawless, E., Lambert, E., & Newton, A. (2005). The Flynn effect in the Caribbean: Generational change of cognitive test performance in Dominica. *The Mankind Quarterly*, XLVI, 29–69.
- Merilä, J., & Sheldon, B. C. (1999, August). Genetic architecture of fitness and nonfitness traits: Empirical patterns and development of ideas. *Heredity*, 83, 103–109. <http://dx.doi.org/10.1046/j.1365-2540.1999.00585.x>
- Milanovic, B. (2013). Global income inequality in numbers: In history and now. *Global Policy*, 4, 198–208. <http://dx.doi.org/10.1111/1758-5899.12032>
- Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24, 167–202. <http://dx.doi.org/10.1146/annurev.neuro.24.1.167>
- Mingroni, M. A. (2007). Resolving the IQ paradox: Heterosis as a cause of the Flynn effect and other trends. *Psychological Review*, 114, 806–829. <http://dx.doi.org/10.1037/0033-295X.114.3.806>
- Moore, A. J., Brodie, E. D., III, & Wolf, J. B. (1997). Interacting phenotypes and the evolutionary process: I. Direct and indirect genetic effects of social interactions. *Evolution: International Journal of Organic Evolution*, 51, 1352–1362. <http://dx.doi.org/10.1111/j.1558-5646.1997.tb01458.x>
- Murray, C. (1998). *Income inequality and IQ*. New York, NY: AEI Press.
- O'Connor, T. G., Rutter, M., Beckett, C., Keaveney, L., & Kreppner, J. M. (2000). The effects of global severe privation on cognitive competence: Extension and longitudinal follow-up. *Child Development*, 71, 376–390. <http://dx.doi.org/10.1111/1467-8624.00151>
- Ohta, T. (1973). Slightly deleterious mutant substitutions in evolution. *Nature*, 246, 96–98. <http://dx.doi.org/10.1038/246096a0>



- Philip Chen, C. L., & Zhang, C. Y. (2014). Data-intensive applications, challenges, techniques and technologies: A survey on Big Data. *Information Sciences*, 275, 314–347. <http://dx.doi.org/10.1016/j.ins.2014.01.015>
- Pietschnig, J., & Voracek, M. (2015). One century of global IQ gains: A formal meta-analysis of the Flynn Effect (1909–2013). *Perspectives on Psychological Science*, 10, 282–306. <http://dx.doi.org/10.1177/1745691615577701>
- Pigliucci, M. (2007). Do we need an extended evolutionary synthesis? *Evolution; International Journal of Organic Evolution*, 61, 2743–2749. <http://dx.doi.org/10.1111/j.1558-5646.2007.00246.x>
- Plomin, R., & DeFries, J. C. (1979). Multivariate behavioral genetic analysis of twin data on scholastic abilities. *Behavior Genetics*, 9, 505–517. <http://dx.doi.org/10.1007/BF01067347>
- Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, 84, 309–322. <http://dx.doi.org/10.1037/0033-2909.84.2.309>
- Plomin, R., Fulker, D. W., Corley, R., & DeFries, J. C. (1997). Nature, nurture, and cognitive development from 1 to 16 years: A parent-offspring adoption study. *Psychological Science*, 8, 442–447. <http://dx.doi.org/10.1111/j.1467-9280.1997.tb00458.x>
- Protzko, J. (2015). The environment in raising early intelligence: A meta-analysis of the fadeout effect. *Intelligence*, 53, 202–210. <http://dx.doi.org/10.1016/j.intell.2015.10.006>
- Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research*, 5, 554–571. <http://dx.doi.org/10.1375/136905202762342026>
- Richardson, K., & Norgate, S. (2005). The equal environments assumption of classical twin studies may not hold. *The British Journal of Educational Psychology*, 75, 339–350. <http://dx.doi.org/10.1348/000709904X24690>
- Richardson, S. A. (1976). The relation of severe malnutrition in infancy to the intelligence of school children with differing life histories. *Pediatric Research*, 10, 57–61. <http://dx.doi.org/10.1203/00006450-197601000-00011>
- Riemann, R., Angleitner, A., & Strelau, J. (1997). Genetic and environmental influences on personality: A study of twins reared together using the self- and peer report. *Journal of Personality*, 65, 449–475. <http://dx.doi.org/10.1111/j.1467-6494.1997.tb00324.x>
- Rietveld, C. A., Medland, S. E., Derringer, J., Yang, J., Esko, T., Martin, N. W., . . . Koellinger, P. D. (2013). GWAS of 126,559 individuals identifies genetic variants associated with educational attainment. *Science*, 340, 1467–1471. <http://dx.doi.org/10.1126/science.1235488>
- Rindermann, H., & Thompson, J. (2016). The cognitive competences of immigrant and native students across the world: An analysis of gaps, possible causes and impact. *Journal of Biosocial Science*, 48, 66–93. <http://dx.doi.org/10.1017/S0021932014000480>
- Rindermann, H., Schott, T., & Baumeister, A. E. E. (2013). Flynn effect in Turkey: A comment on Kagitcibasi and Biricik (2011). *Intelligence*, 41, 178–180. <http://dx.doi.org/10.1016/j.intell.2013.02.003>
- Roberts, R. C. (1967). Some concepts and methods in quantitative genetics. In J. Hirsch (Ed.), *Behavior-genetic analysis* (pp. 214–257). New York, NY: McGraw-Hill.
- Rockman, M. V. (2008). Reverse engineering the genotype-phenotype map with natural genetic variation. *Nature*, 456, 738–744. <http://dx.doi.org/10.1038/nature07633>
- Rutter, M., & Silberg, J. (2002). Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology*, 53, 463–490. <http://dx.doi.org/10.1146/annurev.psych.53.100901.135223>
- Rutter, M. (1998). Developmental catch-up, and deficit, following adoption after severe global early privation. *Journal of Child Psychology and Psychiatry*, 39, 465–476. <http://dx.doi.org/10.1017/S002196309802236>
- Salthouse, T. A., & Ferrer-Caja, E. (2003). What needs to be explained to account for age-related effects on multiple cognitive variables? *Psychology and Aging*, 18, 91–110. <http://dx.doi.org/10.1037/0882-7974.18.1.91>
- Sauce, B., de Brito, R. A., & Peripato, A. C. (2012). Genetic architecture of nest building in mice LG/J × SM/J. *Frontiers in Genetics*, 3, 90.
- Schiff, M., Duyme, M., Dumaret, A., & Tomkiewicz, S. (1982). How much could we boost scholastic achievement and IQ scores? A direct answer from a French adoption study. *Cognition*, 12, 165–196. [http://dx.doi.org/10.1016/0010-0277\(82\)90011-7](http://dx.doi.org/10.1016/0010-0277(82)90011-7)
- Settle, J. E., Dawes, C. T., & Fowler, J. H. (2009). The Heritability of Partisan Attachment. *Political Research Quarterly*, 62, 601–613. <http://dx.doi.org/10.1177/1065912908327607>
- Shipstead, Z., & Engle, R. W. (2013). Interference within the focus of attention: Working memory tasks reflect more than temporary maintenance. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 39, 277–289. <http://dx.doi.org/10.1037/a0028467>
- Silventoinen, K., Magnusson, P. K. E., Tynelius, P., Kaprio, J., & Rasmussen, F. (2008). Heritability of body size and muscle strength in young adulthood: A study of one million Swedish men. *Genetic Epidemiology*, 32, 341–349. <http://dx.doi.org/10.1002/gepi.20308>
- Staff, R. T., Hogan, M. J., & Whalley, L. J. (2014). Aging trajectories of fluid intelligence in late life: The influence of age, practice and childhood IQ on Raven's Progressive Matrices. *Intelligence*, 47, 194–201. <http://dx.doi.org/10.1016/j.intell.2014.09.013>
- Sternberg, R. J. (1997). Educating intelligence: Infusing the triarchic theory into school instruction. In R. J. Sternberg & E. Grigorenko (Eds.), *Intelligence, heredity, and environment* (pp. 343–362). New York, NY: Cambridge University Press.
- Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and meta-analysis. *The American Journal of Psychiatry*, 157, 1552–1562. <http://dx.doi.org/10.1176/appi.ajp.157.10.1552>
- Sundet, J. M., Borren, I., & Tambs, K. (2008). The Flynn effect is partly caused by changing fertility patterns. *Intelligence*, 36, 183–191. <http://dx.doi.org/10.1016/j.intell.2007.04.002>
- Tabery, J. (2014). *Beyond versus: The struggle to understand the interaction of nature and nurture*. Cambridge, MA: MIT Press. <http://dx.doi.org/10.7551/mitpress/9780262027373.001.0001>
- Templeton, A. R. (2006). *Population genetics and microevolutionary theory*. Hoboken, NJ: Wiley-Liss. <http://dx.doi.org/10.1002/0470047356>
- Tenesa, A., & Haley, C. S. (2013). The heritability of human disease: Estimation, uses and abuses. *Nature Reviews Genetics*, 14, 139–149. <http://dx.doi.org/10.1038/nrg3377>
- Thompson, L. A., Dettmerman, D. K., & Plomin, R. (1993). Differences in heritability across groups differing in ability, revisited. *Behavior Genetics*, 23, 331–336. <http://dx.doi.org/10.1007/BF01067433>
- Trahan, L. H., Stuebing, K. K., Fletcher, J. M., & Hiscock, M. (2014). The Flynn effect: A meta-analysis. *Psychological Bulletin*, 140, 1332–1360. <http://dx.doi.org/10.1037/a0037173>
- Tranter, L. J., & Koutstaal, W. (2008). Age and flexible thinking: An experimental demonstration of the beneficial effects of increased cognitively stimulating activity on fluid intelligence in healthy older adults. *Aging, Neuropsychology and Cognition*, 15, 184–207. <http://dx.doi.org/10.1080/13825580701322163>
- Tucker-Drob, E. M., & Bates, T. C. (2015). Large Cross-National Differences in Gene x Socioeconomic Status Interaction on Intelligence. *Psychological Science*, 27, 138–149. <http://dx.doi.org/10.1177/0956797615612727>
- Tucker-Drob, E. M., & Briley, D. A. (2014). Continuity of genetic and environmental influences on cognition across the life span: A meta-analysis of longitudinal twin and adoption studies. *Psychological Bulletin*, 140, 949–979. <http://dx.doi.org/10.1037/a0035893>

- 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, 349–355. <http://dx.doi.org/10.1177/0963721413485087>
- Tucker-Drob, E. M., & Harden, K. P. (2012). Early childhood cognitive development and parental cognitive stimulation: Evidence for reciprocal gene-environment transactions. *Developmental Science*, 15, 250–259. <http://dx.doi.org/10.1111/j.1467-7687.2011.01121.x>
- Tucker-Drob, E. M., Rhemtulla, M., Harden, K. P., Turkheimer, E., & Fask, D. (2011). Emergence of a Gene x socioeconomic status interaction on infant mental ability between 10 months and 2 years. *Psychological Science*, 22, 125–133. <http://dx.doi.org/10.1177/0956797610392926>
- Tuddenham, R. D. (1948). Soldier intelligence in World Wars I and II. *American Psychologist*, 3, 54–56. <http://dx.doi.org/10.1037/h0054962>
- Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, 9, 160–164. <http://dx.doi.org/10.1111/1467-8721.00084>
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, 14, 623–628. [http://dx.doi.org/10.1046/j.0956-7976.2003.psci\\_1475.x](http://dx.doi.org/10.1046/j.0956-7976.2003.psci_1475.x)
- Tyler, A. L., Asselbergs, F. W., Williams, S. M., & Moore, J. H. (2009). Shadows of complexity: What biological networks reveal about epistasis and pleiotropy. *BioEssays*, 31, 220–227. <http://dx.doi.org/10.1002/bies.200800022>
- van der Maas, H. L. J., Dolan, C. V., Grasman, R. P. P. P., Wicherts, J. M., Huizenga, H. M., & Raijmakers, M. E. J. (2006). A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychological Review*, 113, 842–861. <http://dx.doi.org/10.1037/0033-295X.113.4.842>
- van Ijzendoorn, M. H., Juffer, F., & Poelhuis, C. W. K. (2005). Adoption and cognitive development: A meta-analytic comparison of adopted and nonadopted children's IQ and school performance. *Psychological Bulletin*, 131, 301–316. <http://dx.doi.org/10.1037/0033-2909.131.2.301>
- van Ijzendoorn, M. H., Luijk, M. P. C. M., & Juffer, F. (2008). IQ of children growing up in children's homes: A meta-analysis on IQ delays in orphanages. *Merrill-Palmer Quarterly*, 54, 341–366. <http://dx.doi.org/10.1353/mpq.0.0002>
- Wakefield, M. (2013, July). Revealed: How exam results owe more to genes than teaching. *The Spectator*. Retrieved from <http://www.spectator.co.uk/features/8970941/sorry-but-intelligence-really-is-in-the-genes/>
- Winick, M., Meyer, K. K., & Harris, R. C. (1975). Malnutrition and environmental enrichment by early adoption. *Science*, 190, 1173–1175. <http://dx.doi.org/10.1126/science.1198103>
- Woodley, M. A. (2011). Heterosis doesn't cause the Flynn effect: A critical examination of Mingroni (2007). *Psychological Review*, 118, 689–693. <http://dx.doi.org/10.1037/a0024759>
- Zhao, J., Li, S., Wang, L., Jiang, L., Yang, R., & Cui, Y. (2017). Genome-wide random regression analysis for parent-of-origin effects of body composition allometries in mouse. *Scientific Reports*, 7, 45191. <http://dx.doi.org/10.1038/srep45191>
- Zuk, O., Hechter, E., Sunyaev, S. R., & Lander, E. S. (2012). The mystery of missing heritability: Genetic interactions create phantom heritability. *Proceedings of the National Academy of Sciences of the United States of America*, 109, 1193–1198. <http://dx.doi.org/10.1073/pnas.1119675109>

Received December 7, 2016

Revision received September 21, 2017

Accepted September 25, 2017 ■



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