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Discussions

The Distance Between Genotype and Human Intelligence Is Long, Indirect, and Densely Mediated by Experience

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Jensen (Jensen, 1998) stated that the "holy grail" of intelligence research was the elucidation of the biological basis for its' variations. Gargus and Haier (2025) expand on this notion, suggesting that a more complete understanding of the molecular biology of general intelligence will provide actionable plans to increase the intelligence of individuals. While the approach described by Gargus and Haier is hypothesis-driven, the distance between genotype and phenotype is long, indirect, and densely mediated by experience. Common strategies do not capture nonlinear biological interactions, feedback loops, or developmental and environmental influences, i.e., they provide no insight into how or why a phenotype emerges. The main difficulty with the molecular account of intelligence advanced by Gargus and Haier (2025) is not that the biological pathways they discuss are implausible, rather, it is that the phenotypes themselves are so complex that even well-replicated genetic associations give only limited traction on questions of causation. Based on the hidden influence of gene-environment interplay, small effect sizes of identified gene variants, the low generalizability of GWAS signals across time and place, and the fact that parental genotypes can shape offspring outcomes through environmentally-mediated pathways rather than direct biological transmission, we think the quest to find global (and timeless) genes for intelligence is not simply difficult, but may be futile from the start. A focus on the role of educational opportunity, family environment, and socio-economic circumstance might yield more immediately tangible strategies to promote human intelligence.

Body height is easily and accurately measured, and there is no dispute about the adequacy of its quantification. Its heritability is *high*, with twin and adoption studies indicating that 70-90% of the variation in height is heritable, and it is a prototypic polygenic trait with many additive genetic effects contributing to the phenotype. Thousands of DNA regions have been found to be *associated* with variations in height, but, until recently, these contributed to less than 10% of its variance. Such a surprising discrepancy exemplifies the "missing heritability" problem.

Recently, the missing heritability of height has been narrowed significantly, with large genome-wide association studies (GWASs) revealing the common alleles that additively account for most of the variation in height (Bicknell et al., 2025). Notably, this level of explanatory power required a study with 5 million participants and the inclusion of over 12,000 genetic variants. Even so, 10-20% of variation in height remains unexplained by DNA variants. The resolution of this discrepancy might require a still larger sample size in order to detect the smallest genetic influences. Alternatively, this discrepancy may arise because even a trait as "simple" as height is nevertheless subject to small levels of Gene x Environment (GxE) interactions (e.g., height can be stunted by nutritional deficits; Grasgruber et al., 2014) and GE correlations (as height predicts higher future wages [Thompson et al., 2023], and a wealthy household can provide a better diet for the children).

Unlike body height, the measurement of IQ is far from simple, and its quantification is fraught with inaccuracies and debates about its very nature. Still, IQ (or at least its common metrics) is highly heritable, with some estimates approaching that of body height. In fact, IQ is one of the most heritable of "psychological" traits, with estimates (depending on population and age of estimate) consistently ranging from 50-80%. Unlike height, the causal chain of IQ seems to be much, much more plagued by GxE interactions and GE correlations.

Gargus & Haier (2025) propose a strategy which could potentially elucidate the causal genetic mechanisms for intelligence. Rather than a scatter-shot approach inherent to many GWASs, these authors suggest that a more productive approach should be hypothesis-directed. First, they argue that any search for intelligence-associated genes should focus on Human Accelerated Regions (HARs) of the brain. HARs are evolutionary markers that have undergone significant changes after human/chimpanzee divergence, which in principle, can be specific to Human Lineage Traits (HLTs). This could be a valuable approach, if we assume that the processes that underlie general cognitive abilities are uniquely human. In itself, this premise is debatable (c.f.,

Burkart et al., 2016; Matzel & Sauce, 2017), but nevertheless, it is a starting point from which to focus the search for genetic variants that might be specific to human intelligence (a point that we will return to below). To add to this approach, Gargus & Haier suggest that rare genetic variants are well suited to account for variations in intelligence both between and within families, and that rare, protein-truncating variants in genes that are intolerant of loss are well-situated to account for developmental disorders relevant to cognition. They discuss eight such variants that could reasonably be expected to impair the expression of general intelligence, and these might well account for some instances of low intelligence. However, we are less optimistic that these variants could contribute broadly to variations within the range of *normal* intelligence (i.e., IQs > 80). It is also worth mentioning that the variants-of-interest discussed by Gargus and Haier are severely limited (by the authors' intention to generate *specific* hypotheses), and a survey of 100 researchers would be likely to yield a list of variants-of-interest that is orders of magnitude longer. Thus, variations in intelligence within the "normal" range may well require an approach that goes beyond that described by Gargus & Haier, and may not be (owing to GxE interactions and GE correlations) entirely transparent to typical GWASs.

GE correlations take several forms (passive, evocative, and active). Passive interactions reflect (for example) the observation that a child born to high IQ parents are likely to find themselves in an environment that is cognitively enriched, e.g., the presence of chessboards and books related to chess. Next, these same children may *evoke* a response from their environment, e.g., their teachers may recognize a precocious desire to play chess and might steer them toward chess club. Lastly, these individuals might *actively* seek cognitive challenges that match their cognitive talents, e.g., they may request that their parents take them to Washington Square where they can test their chess skills against similarly-minded chess aficionados. Fortunately for them, their parents (not coincidentally) may live in Manhattan, a high SES environment where chess culture can be easily accessed.

GE correlations can *strongly* influence estimates of heritability. Two quick examples can clarify the role of opportunity and resources in the establishment of metrics of heritability. First, the heritability of IQ increases with age. When IQ is first quantifiable (around age four), its heritability is relatively low (e.g., h = .2). However, by middle age, it's heritability can be quite high (h > .5). In the context of GE correlations, this is not surprising. Imagine two twins with initially similar (but not identical) IQs. Given a similar history (the presence of chessboards, evocating teachers, and their desire to play chess in Washington Square), they gravitate along similar tracks to arrive at a similar destination, becoming even *more* similar with time. Second, in low SES environments, the heritability of IQ is low relative to that observed in high SES environments. Again, this is a reflection of the role of GE correlations. In the high SES environment, the *opportunity* exists to exploit the home and educational resources, and to find like-minded chess players. Importantly, in neither of these cases do the common shared genes increase (despite increases in the estimated heritability). Instead, a smaller number of shared genes can be matched to an environmental niche where innate dispositions can be exploited.

GWAS is a technique that compares the genomes of many people to identify genetic variations (e.g., single nucleotide polymorphisms; SNPs) that are associated with a particular trait, and could in principle resolve genetic variations that are emblematic of variations in that trait. GWASs for intelligence and educational attainment find that this trait is highly polygenic, and is associated with thousands of genetic variants, each of which alone typically account for 0.01-0.02% of variance in the trait. Even with 1000s of associated variants, only a small portion (typically 20-30%) of the variation in intelligence and educational attainment can be accounted for (e.g., Chen et al., 2025; Hill et al., 2018). This missing heritability is not resolved by the computation of polygenic scores (PGS; the sum of weighted GWAS effect sizes across variants) as a PGS cannot capture nonlinear biological interactions, feedback loops, or developmental and environmental influences, i.e., they provide no insight into how or why a phenotype emerges. This is particularly problematic with regard to intelligence or educational attainment, which as described above, is the product of a complex web of GxE interactions and hidden GE correlations.

To account for missing heritability, some 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 only act additively. Because of this, and since we can presume that variants with the largest effect were the first to be recognized, the number of additive DNA variants necessary to account for IQ's heritability would be extremely high. Each genetic region from GWAS seems to explains less than 0.01% of the total variance in intelligence (Chabris et al., 2012), which suggests that a *minimum* number of independent, additive DNA variants necessary to explain IQ's heritability would be over 9000 (Sauce & Matzel, 2018), or almost half of the 20 thousand protein-coding genes in the human genome. A gene may have multiple independent SNPs associated with it, since some genes are immense and include many different regulatory regions, but still, this quick scale-check already paints an unlikely picture for additivity. Furthermore, 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 becomes effectively neutral because genetic drift then dominates the weak effects of selection, especially in small populations such as the ancestral populations of our species. Were this the case, for most of our evolutionary history the causal variants behind the variation in human intelligence would have been under neutrality, at the whim of genetic drift. We shouldn't expect all genetic effects in IQ to be pure additive effects. And as we know empirically

now, there are many non-additive genetic effects that complicate the story via GE interactions and correlations.

The main difficulty with the molecular account of intelligence advanced by Gargus and Haier (2025) is not that the biological pathways they discuss are implausible. Rather, it is that the phenotypes themselves are so complex that even well-replicated genetic associations give only limited traction on questions of causation. Unlike traits such as height (which are comparatively straightforward to measure and emerge from fewer and simpler developmental pathways), intelligence and educational attainment are the emerging product of neural, cognitive, developmental, social, and historical processes, all of which probably interact in multiple ways. The central problem is not simply that genetic effect sizes are small, but that the distance between genotype and phenotype is long, indirect, and densely mediated by experience.

Let's check first educational attainment, which plays a prominent role in the Gargus and Haier (2025) framework as both an outcome of interest and a proxy for intelligence. Years of schooling are shaped as much by institutional policies, family resources, labor-market incentives, and cultural expectations as by cognitive capacity. Two individuals with similar cognitive potential may follow radically different educational trajectories depending on *opportunity structures* that have little to do with the neurobiology of cognition. In a technique called "GWAS-by-subtraction", Demange et al. (2021) inserted the results from the GWAS for educational attainment and the GWAS for cognitive performance (Lee et al., 2018) in a genomic structural equation model to "split" the effect from genetic variants found in the GWAS for educational attainment. They found that 57% of the genetic variation in educational attainment comes from non-cognitive sources (i.e., is *not* explained by genetic effects on cognitive performance). These non-cognitive sources, some indirect evidence suggests, include personality traits, motivation, delayed gratification, social skills, work/study ethics, and even seemingly "unrelated" traits like beauty and height, presumably because the beautiful and tall are treated and seen through more educationally-conducive lenses (Bauldry et al., 2016; Demange et al., 2021). That complexity and mixture of contributions does not lessen the empirical success of educational-attainment GWAS, but it does caution against treating associated loci as direct windows into the biology of intelligence.

Intelligence and educational attainment are closely related and subject to similar influences. IQ scores are often derived from performance across diverse tasks, and the positive manifold (correlation between tasks) is hierarchically structured across multiple skills segregated into many separate domains, each with somewhat different genetic influences and different associations with brain areas and neural activity. As such, intelligence as a construct may be several conceptual steps removed from any single biological mechanism, such as proposed in models of mutualism, or in bottom-up cognitive networks, or in bioecological models of gene-environment interplay (Kovacs & Conway, 2016; Matzel et al., 2020; van der Maas et al., 2006). Genes for general intelligence need not operate on a single general process like information processing or neural efficiency, and instead, may act on a mosaic of processes of more-or-less importance (depending on context, task, and developmental stage), e.g., attention, executive control, memory, abstraction, emotional regulation, physical health, access to learning opportunities, etc.

Gene-environment interplay further erodes any potential for clean causal interpretation. Intelligence and educational attainment are among the traits most strongly shaped by gene-environment correlations, including passive, evocative, and active forms. Individuals do not typically encounter environments in an arbitrary fashion. Genetic propensities influence the environments they are born into, the responses they elicit from others, and the experiences they actively seek. Over time, these processes amplify initial differences, producing developmental trajectories in which genetic and environmental influences are inseparable in both practice and principle (Harden et al., 2007; Howe et al., 2022; Nivard et al., 2024; Sauce & Matzel, 2018; Turkheimer et al., 2003; van der Maas et al., 2006). This framework helps explain why the heritability of intelligence increases with age and why heritability estimates are higher in socioeconomically advantaged environments, even though the underlying genetic variants remain unchanged (Harden et al., 2007; Tucker-Drob et al., 2013; Turkheimer et al., 2003)

These dynamics have direct implications for how the GWAS findings emphasized by Gargus and Haier should be interpreted. In the presence of strong GE correlations, a substantial portion of GWAS signal may reflect genetic predictors of environmental exposure rather than biological mechanisms that directly influence cognition. Some associated variants may therefore be "going along for the ride," tagging social pathways through which genetic differences are expressed. This concern is magnified by the fact that most large GWAS of intelligence and educational attainment rely on relatively homogeneous samples drawn from modern, high-income societies. The resulting associations may be tightly coupled to historically specific institutions, e.g., schooling systems, labor markets, and cultural norms, rather than to general biological constraints on intelligence. We were left with the impression that Gargus and Haier (2025) might expect that the "genetic effects" they describe would be likely applicable for humans in general (across all social classes, countries, and time), as if we would one day find *the* genes for intelligence. We don't believe that outcome is likely. Even contrasting GWASs of contemporary people, GWASs (for a variety of traits) based on Europeans populations have their predictive accuracy decreased by 25-75% when applied to Americans, Asians, or Africans (Martin et al., 2019; Wang et al., 2023). Based on the low generalizability of GWASs, we think the quest to find global (and timeless) genes for intelligence is not simply difficult, but may be futile from the start.

One further complication for identifying the "causal genes" for intelligence and educational attainment lies in the growing evidence for indirect genetic effects. Parental genotypes can shape offspring outcomes through environmentally-mediated pathways rather than direct biological

transmission. Family-based and within-sibship study designs consistently show that polygenic scores for cognitive and educational traits are substantially attenuated once shared family environments are accounted for. For example, polygenic score prediction for cognitive traits (intelligence and educational achievement) were on average 60% greater between families than within families, but this was not the case for non-cognitive traits such as height and BMI. That discrepancy is largely explained by socio-economic context and passive GE correlations rather than direct genetic influence (Selzam et al., 2019). These findings imply that a substantial portion of what is often interpreted as "genetic" signal in population-based GWASs reflects environmentally-mediated parental effects—sometimes referred to as genetic nurture—rather than causal effects of inherited alleles on cognitive development. In contrast, similar attenuation is not observed for traits such as height, underscoring that indirect genetic effects are not a generic property of GWAS, but are especially pronounced for socially structured traits like education and intelligence.

It gets even more complicated. A recent study suggests that indirect genetic effects on educational attainment and related cognitive traits are not confined to the nuclear family, but are embedded in broader processes of social stratification and assortative mating that operate across generations (Nivard et al., 2024). Extended-pedigree analyses indicate that a substantial share of the genetic signal detected in GWAS of education reflects dynastic processes rather than environmentally-mediated parental nurture alone, with indirect genetic effects largely attributable to multi-generational transmission of social advantage (Nivard et al., 2024). At the same time, evidence from population-scale sibling and geographic designs shows that polygenic score associations for educational attainment are inflated by GE correlations arising from migration, neighborhood sorting, and other forms of social stratification that are imperfectly controlled by standard ancestry adjustments (Abdellaoui et al., 2022). Crucially, another recent work on indirect assortative mating further demonstrates that partner similarity in educational attainment is poorly explained by direct assortment on education itself, and instead reflects matching on latent social and familial factors that are correlated with both genotype and environment (Sunde et al., 2025). These processes induce long-range correlations between genetic and environmental influences, biasing estimates of both genetic transmission and environmental effects unless explicitly modeled. Taken together, this literature underscores a sobering conclusion: For intelligence and educational attainment, many genetic associations index pathways of social reproduction and stratification rather than biological mechanisms that can be straightforwardly interpreted as causal for individual differences in cognitive ability.

These points highlight a distinction that is sometimes blurred in discussions of cognition and evolution: The causes of variation within modern populations need not resemble the causes of differences between species or ancestral populations. Genetic variants that explain why some individuals outperform others in contemporary societies may have little to do with the evolutionary processes that produced human cognitive capacities in the first place. The analogy to height is instructive here. Much of the variation in stature observed across countries today reflects nutrition and wealth, not the selective pressures that once differentiated primates' body size (Peñuelas et al., 2017; Perkins et al., 2016). Present-day genetic associations with educational attainment and intelligence may be similarly contingent on recent social and physical environments rather than deep evolutionary causes.

This distinction bears directly on the emphasis Gargus and Haier place on Human Accelerated Regions (HARs). HARs are, by definition, genomic regions that differentiate modern humans from other primates, and their proximity to neurodevelopmental genes makes them compelling candidates for understanding the evolution of human cognition. However, strong gene-environment correlation complicates the inference that variants responsible for within-population variation in intelligence must map onto regions that drove between-species differences. The mechanisms that enabled the emergence of human cognitive capacities may differ substantially from those that generate individual differences in cognitive performance within contemporary, resource-rich societies. In this sense, HARs may be better suited to illuminating the origins of human cognition than to explaining the distribution of intelligence among modern individuals.

These considerations also temper our enthusiasm for interpreting specific genes or pathways (however biologically plausible) as definitive explanations of intelligence differences. The gene-level discussions offered by Gargus and Haier (2025) necessarily rest on correlational evidence and account for small fractions of total variance in the traits. Note that even the largest and most successful GWAS on cognition to date only explain/predict 5-15% of the individual differences in IQ scores, school grades, or level of education (Okbay et al., 2022). It is easy to overinterpret these genetic effects by constructing post hoc narratives that link molecular function to cognition. Without stronger causal leverage, these narratives are merely describing patterns rather than explaining causal processes.

A final issue should be briefly addressed, as it weighs heavily in discussions of the heritability of intelligence. The high heritability of IQ among twins raised apart is often cited as strong evidence for the primacy of genetic influences on intelligence (Wakefield, 2013). However, the *difference* between heritability estimates of IQ among twins raised together versus twins raised apart is a classic and often repeated observation in the field of behavioral genetics. While heritability estimates are high in both cases, large-scale studies and meta-analyses indicate that the heritability of IQ in MZ twins drops from ~.86 in those raised together to ~.7 in twins raised apart (Bouchard et al., 1990; Bouchard & McGue, 1981).

The drop in heritability from ~0.86 to ~0.70 is thought to reflect the influence of familial environment on the development of intelligence. These environmental effects are the product of passive GE correlations, i.e., the environment imposed by parents onto their children. However, although

separated-twin studies are intended to control for the effects of famial environment, even these passive correlations are not entirely mitigated by these designs, since adoptive parents tend to be more advantaged, stable, and financially secure relative to those parents who place their children in adoption (Cadoret, 1995), i.e., adopted children are often raised in advantaged and homogenous environments where opportunities are prolific. Unquestionably, adoption from impoverished into wealthy environments can promote dramatic increases in IQ (e.g., Duyme et al., 1999; O'Connor et al., 2000; van Ijzendoorn et al., 2005). More important to the present discussion, evocative and active GE correlations are not substantially reduced when a child moves to an adoptive home, i.e., the adopted child's genetic dispositions can *evoke* commensurate responses from the environment (e.g., teachers that recognize a proclivity for chess) and that child might *actively* seek out challenges appropriate for his/her dispositions (e.g., the child might ask for a trip to Washington Square where he/she can develop their skills). Again, since adoptive parents tend to be more wealthy and stable, adopted children are likely to find themselves in environments where small dispositions can be readily pursued and exploited. In total, twin adoption data does not negate the conclusion that environmental influences contribute heavily to estimates of the heritability of IQ (Sauce & Matzel, 2018).

Conclusions

Intelligence is a polygenic trait, and its ultimate expression arises from complex interactions and correlations between genes and environment. Large scale, high resolution GWASs have identified thousands of potential genetic contributions, and further GWASs are likely to identify DNA variants with diminishingly small effects. Despite this, only a fraction of the heritability of intelligence has been explained. And even that fraction, we know, is highly confounded by interactions and correlations with the environment.

Where, then, might progress come from? Advances are unlikely to arise from association studies alone, regardless of the sample size or resolution of small effects. Instead, genuine insight will require designs that engage causation more directly (Burt et al., 2018). Mendelian randomization offers one possible route, and although many researchers are productive in employing this approach, its assumptions are particularly fragile for traits characterized by high pleiotropy and GE correlations (Sanderson et al., 2022). In addition, the field of behavior genetics is starting to embrace study designs that tap deep into causation: natural experiments (for example, during changes of relevant policies/laws or major events like covid or famines; He et al., 2018; Zhou et al., 2025), quasi-experimental designs like regression discontinuity (for example, making use of arbitrary cut-off in school entry; Judd et al. 2022), and longitudinal studies tracking genetic effects across multiple environments.

Jensen (1998) stated that the "holy grail" of intelligence research was the elucidation of the biological basis for its' variations. Gargus and Haier expand on this notion, suggesting that a more complete understanding of the molecular biology of general intelligence will provide actionable plans to increase the intelligence of individuals. While this goal may be in part achievable (particularly in cases of intelligence below the normal range), it cannot be completely productive absent a more immediately tangible focus on the role of educational opportunity, family environment, and socio-economic dynamics that contribute to the development of intelligence.

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