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Thinking positively: The genetics of high intelligence

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ABSTRACT

High intelligence (general cognitive ability) is fundamental to the human capital that drives societies in the information age. Understanding the origins of this intellectual capital is important for government policy, for neuroscience, and for genetics. For genetics, a key question is whether the genetic causes of high intelligence are qualitatively or quantitatively different from the normal distribution of intelligence. We report results from a sibling and twin study of high intelligence and its links with the normal distribution. We identified 360,000 sibling pairs and 9000 twin pairs from 3 million 18-year-old males with cognitive assessments administered as part of conscription to military service in Sweden between 1968 and 2010. We found that high intelligence is familial, heritable, and caused by the same genetic and environmental factors responsible for the normal distribution of intelligence. High intelligence is a good candidate for "positive genetics" — going beyond the negative effects of DNA sequence variation on disease and disorders to consider the positive end of the distribution of genetic effects.

1. Introduction

High intelligence is precious human capital for advancing and maintaining society in the information age, as documented in studies that demonstrate that high intelligence is responsible for exceptional performance in many societally-valued outcomes (Kell, Lubinski, & Benbow, 2013; Lubinski, Benbow, Webb, & Bleske-Rechek, 2006; Rindermann & Thompson, 2011). Understanding the genetic and environmental origins of high intelligence is crucial for government policy (for example, for education in the STEM subjects of science, technology, engineering and mathematics), for neuroscience (for investigating the high-performance brain), and for genetics. A key question for genetic research is the extent to which the aetiology of high intelligence differs from the aetiology of the normal distribution of intelligence. More specifically, do the same genes affect both high intelligence and the rest of the distribution to the same extent? It cannot be assumed that the aetiology of high intelligence is the same. For example, very low intelligence (severe intellectual disability) differs aetiologically from the normal distribution, as proposed initially by Lionel Penrose (1938). In quantitative genetic studies (Nichols, 1984; Reichenberg et al., in preparation), a critical piece of evidence is that siblings of individuals with severe intellectual disability have an average IQ near 100, whereas siblings of those with mild intellectual disability have an average IQ of around 85, about one standard deviation below the population mean. In recent molecular genetic studies, rare non-inherited mutations appear to be a major source of severe intellectual disability (Ellison, Rosenfeld, & Shaffer, 2013).

One of the earliest studies in behavioural genetics was Galton's *Hereditary Genius* (1869), an analysis of family pedigrees for brains as well as beauty and brawn. Since there

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was no satisfactory way at the time to measure intelligence, Galton had to rely on reputation as an index of eminence, which he found to be highly familial. Since Spearman's (1904) seminal work on general cognitive ability (*g*) over a century ago, research has focused on intelligence as a general factor that indexes what diverse tests of cognitive abilities have in common (Jensen, 1998). Intelligence was the target of the first twin and adoption studies in the 1920s (Burks, 1928; Freeman, Holzinger, & Mitchell, 1928; Merriman, 1924; Theis, 1924), and continues to be among the most studied traits in behavioural genetics (Plomin, DeFries, Knopik, & Neiderhiser, 2013).

For these reasons, it is surprising that few behavioural genetic studies have focused on high intelligence (Plomin & Haworth, 2009). We review these studies below, but we begin with hypotheses about why genetic and environmental factors might differ for high intelligence (the Discontinuity Hypothesis), and why the results might be similar (the Continuity Hypothesis).

2. The Discontinuity Hypothesis

The Discontinuity Hypothesis posits different environmental and genetic aetiologies for high intelligence in contrast to the rest of the distribution (Petrill, Kovas, Hart, Thompson, & Plomin, 2009). Although the evidence showing substantial heritability for the normal distribution of intelligence is one of the most consistently documented findings in the behavioural sciences (Deary, Johnson, & Houlihan, 2009), researchers in the field of expert training have argued that "differences in early experiences, preferences, opportunities, habits, training, and practice are the real determinants of excellence" (Howe, Davidson, & Sloboda, 1998, p. 403). A recent special issue of the journal Intelligence examines this environmental view of the acquisition of expertise (Detterman, 2014), including its relationship to genetic research (Plomin, Shakeshaft, McMillan, & Trzaskowski, 2014). Although the critical importance of deliberate practice is most often considered in the domain of specialist skills such as games, arts and sports, intelligence is also sometimes viewed as acquired expertise rather than inherited talent (Sternberg, 1999). If one accepts the overwhelming evidence showing substantial heritability for variation in the normal range of intelligence, the expert training position would suggest a discontinuity in the sense that it assumes that excellence is primarily due to environmental factors. Quantitative genetic research such as the twin method can test this hypothesis by investigating whether environmental influence is more important for high intelligence as compared to the rest of the distribution. Another more subtle environmental source of discontinuity can also be tested: the hypothesis that "differences in early experiences" are especially important for excellence would lead to the prediction that shared environment – environmental factors that make family members similar – should be greater for high intelligence.

Genetic reasons for discontinuity are also plausible, beginning with the folk wisdom that there could be "genes for genius." The most persuasive case for genetic discontinuity for genius has been made by David Lykken (1998). He notes that a key problem of genius is "its mysterious irrepressibility and its ability to arise from the most unpromising of lineages and to flourish even in the meanest of circumstances" (p. 29). He proposed that genius emerges from unique combinations of genes; he referred to these higher-order nonadditive (epistatic) interactions as emergenic (Lykken, 1982, 2006). The emergenesis hypothesis does not necessarily predict that different genes affect high intelligence, but it does predict that genetic effects are nonadditive for high intelligence. The hallmark of an epistatic trait is one for which identical twins, who share all their genes, are more than twice as similar as fraternal twins and other first-degree relatives, who share on average 50% of their segregating genes. The twin design can test this hypothesis that nonadditive genetic effects are greater for high intelligence as well as testing the "genes for genius" hypothesis that different genes are responsible for high intelligence.

For both environmental and genetic discontinuity hypotheses, a crucial issue is the cut-off used to define high ability. If the cut-off is extremely high, scientific research gives way to case studies, as has been recently avowed by a leader in research on expert training, who advocated case studies of the "less than a handful of individuals… with the very highest levels of performance" (Ericsson, 2014). In genetics, too, there is interest in the very highest levels of performance. For example, Galton benchmarked the top 1 in a million (.0001%) as "illustrious" and the top 250 in a million (.025%) as "eminent" (Galton, 1869), and Lykken referred to "genius" although he did not suggest a specific cut-off. Such extreme cut-offs are beyond the reach of quantitative genetics research or gene-hunting research, both of which require large sample sizes. However, once genes accounting for at least a few percent of the variance at any level of performance are identified, they can be used with adequate power as a polygenic score in research on even "a handful of individuals with the very highest levels of performance" (Plomin & Deary, 2014). This is beginning to happen in the world of elite athletic performance where, contrary to the Discontinuity Hypothesis, the same genes appear to be associated additively with both ordinary and extraordinary performance (Epstein, 2013).

3. The Continuity Hypothesis

The Continuity Hypothesis posits that high performance is the quantitative extreme of the same environmental and genetic factors responsible for the rest of the normal distribution. From an environmental perspective, the prodigious practice and concentrated effort of high performers might be only quantitatively (e.g., number of hours of deliberate practice) but not qualitatively different from the factors responsible for the rest of the distribution. In terms of genetics, the Continuity Hypothesis is the foundation for quantitative genetic theory (Fisher, 1918). If multiple genes affect a trait, their joint effects are distributed as a normal bell-shaped curve, which means that the same genes affect the low and high extremes of such polygenic traits. Molecular genetic research has begun to confirm this polygenic prediction as genes are identified that contribute to the heritability of complex dimensions and disorders (Plomin, Haworth, & Davis, 2009). For example, genes identified by their association with obesity are associated with body weight throughout the distribution of weight (Speliotes et al., 2010).

4. Quantitative genetic analysis of high intelligence

When genes associated with intelligence are identified, they will provide a strong competitive test of these two hypotheses by assessing the extent to which genes