What can the study of genetics offer to educators?

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Running head: Genetics and education

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Abstract

This article explores the potential contribution of modern genetic methods and findings to education. It is familiar to hear that the ‘gene’ for this or that behavior has been discovered, or that certain skills are ‘highly heritable’. Can this help educators? To explore this, we describe the methods used to relate genetic variation to individual differences in high-level behaviors such as academic skills and educational achievement. These methods include twin studies and genome-wide association studies. We address the key question of what genetic data imply about the ability of educators to optimize educational outcomes for children across the range of abilities.

Introduction

Researchers investigating the genetics of behavior face a challenge. Broadly speaking, they have discovered that many cognitive abilities, and indeed educational achievement itself, are highly heritable. That is, many of the differences between children in their cognitive skills and their educational performance appear to be of genetic origin. Yet when genetics researchers interact with educators, the key message (which we endorse here) is that the discovery of genetic effects should not be taken to imply that these outcomes – how good a child's cognitive skills will be, how well he or she will do at school – are inevitable or determined by one's genetic make-up. Genetic effects may reduce or even disappear if the environment is changed (in this case, the environment of the classroom, the broader educational system, the family, and society). So what, then, should educators take from studies that report high heritability of, say, intelligence or reading ability or self-efficacy? What does this mean for teaching practices? What does it mean for teachers' own teaching abilities – are these inherited too? Can these abilities be changed?

The field of genetics has a dual history. On the one hand, the study of heritable traits – those that can pass from parent to offspring – has informed a key component of evolutionary theory. This long-standing approach does not require knowledge of the underlying mechanism. Among other things, it has guided improvements in farming and animal husbandry through selective breeding to exaggerate desirable traits. It has also led to recognition of the ways that disabling traits can run in families, but also can occur spontaneously.

On the other hand, there is the more recent study of the biological mechanisms of inheritance, starting with the discovery of chromosomes and then the structure of DNA. As we shall see, there still remains a gap between these two traditions, so

genetics does not yet comprise a seamless, unified field; this can contribute to the challenges of ascertaining practical implications.

How can genetics research be relevant to educational practitioners or those working at the intersection of education and neuroscience? One answer is that the field uses methods to help us understand mechanisms of learning that are complementary to those used in psychology, neuroscience and education research. Genetics can contribute to the multi-disciplinary objective of investigating the causal mechanisms underpinning learning so that we understand why current educational methods work, as well as what future educational methods might also work and for whom (Mareschal, Butterworth & Tolmie, 2013; Thomas, 2013).

The application of genetics to education is exemplified by two recent studies. First, using a *behavioral genetic* approach, where similarities in behavior are compared between identical and non-identical twin pairs (in this case, around 13,000 16 year olds in the UK), Krapohl et al. (2014) demonstrated that examination performance in secondary school was highly heritable, with 62% of the variation in examination results explained by genetic similarity. Second, using a *molecular genetic* approach, Rietveld et al. (2013) correlated variation in individual letters of DNA code across the genome with educational achievement in around 125,000 individuals, looking for actual genes implicated in educational outcomes. However, not much of the variation in educational achievement was explained using this method. Below, we will consider both these studies in a wider context. In the meantime, it is worth noting the very large sample sizes necessary to carry out this kind of research and the associated challenge of moving from such studies to implications for smaller groups, or even individuals.

It is timely to consider the relationship between genetics and education, given that researchers have now begun to make recommendations for education based on genetic research. For example, in the recently published book 'G is for Genes', Asbury and Plomin (2014) made three types of proposals for educators and policymakers: (1) embrace genetic variation in abilities; (2) tailor educational curricula to allow maximization of children's different genetic potential and encourage children to play a role in this process; and (3) invest in alleviating the limiting effects of deprived backgrounds early in development.

This message seems clear. However, genetics results do not yet readily mesh with psychology and education. To illustrate, here is a paradox. Height is 80% heritable. That is, environmental variation does not predict much of the difference in height between people; mostly it is down to the height of their parents (Wood et al., 2014). However, the average height of men has gone up 11cm in 150 years (Hatton, 2013; no similar historical data were available for women). Presumably, this historical increase is due to better nutrition, healthcare, and so forth – that is, it is due to environmental factors. So does environment have a strong influence on height or not? Of course, height is not that important for education. However, the same paradox has emerged for intelligence as well, where it is known as the *Flynn effect* (e.g., Flynn, 2009). Intelligence is highly heritable yet has increased over generations, presumably due to improvements in education and/or society encouraging more practice in the sorts of abstract reasoning tasks that intelligence tests measure. So, based on these contradictory findings, does the environment have a strong influence on intelligence, or not?

Actually, these are not paradoxes, because heritability is about *individual differences* (e.g., the rank order in the class), not about *population means* (e.g., how

well the whole class is doing). If the environment changes for all of us (nutrition for height, practice on abstract reasoning for intelligence tests), we can all move up, even if the rank order among us remains the same (and is present for largely genetic reasons). Thus, results about heritability are not a genetic 'ball and chain' with respect to human potential. Huge changes could be made in the environment that would impact on the population mean performance of a skill set, but these changes might have little impact on the rank order of individual differences within the population for these skills, or indeed the causes of differences between individuals (which might, for instance, be largely genetic). To make the point more strongly, as a society, there are things that people haven't yet thought of doing that if we all did tomorrow, differences between us would be heritable.

What then does heritability imply for educators? If genetics is not deterministic, how does evidence of heritability help us? The answer is that these data are telling us that our environment, right now, for a given population of children, is allowing X% of the variation in ability or achievement between children to come from genetic sources (whatever X might be). This result might surprise us. We might want to do something to understand why the statistic arises. And, potentially, we may as a society want to change the situation. Crucially, the implication is that changing environmental factors can influence the expression or relevance of heritable traits.

However, there remain many unanswered questions. One question is whether better education will tend to increase or decrease heritability. If the educational environment is optimized, the remaining differences between us are more likely to be due to our genetic makeup. Indeed, if we follow Asbury and Plomin's advice and align environments with genetic differences to maximize potential (so that, say, kids with a talent for soccer take more soccer classes), this will exaggerate measurements

of heritability. But it doesn't have to work this way. Environments can be matched with genetic differences in such a way as to reduce the effect of those differences. For example, children with genetic make-ups that put them at risk of developing atypically can be provided with strategically designed environmental inputs to reduce their differences: this is the rationale behind intervening for children with inherited learning disabilities. Recent studies show how this work of individualizing educational techniques is beginning, for instance in tailoring interventions for conduct disorder according to different possible genetic causes (Frederickson, Jones, Warren, Deakes & Allen, 2013), and identifying which individuals will benefit most from a working memory intervention depending on their genetic make-up (Söderqvist, Matsson, Peyrard-Janvid, Kere & Klingberg, 2013).

A second question is, if we find 'genes for education', what will they look like? What will they do? As we see below, the picture emerging is that there will likely be many, many such genes. Of course, they are likely to be involved with the brain, in its construction and cognitive function, since the brain underlies cognition and learning. However, genes that are relevant to educational outcomes may also turn out to be those that influence other aspects of the individual, such as their emotions, their fitness, their response to stress, and their immune systems. Much remains to be discovered.

In the next section, we consider some of the principal methods of genetic research and findings that are relevant to education, before considering the challenge of integrating them to produce practical implications for education.

Genetic methods and recent findings relevant to education

Education is the clearest example of an environmental influence on a person's development. However, *behavioral genetic research* shows that educational environments interact with people's unique genetic profiles, leading to huge individual differences in motivation, learning, ability, and achievement (e.g., Kovas, Haworth, Dale, & Plomin, 2007). Today, quantitative genetic research involves large representative samples and utilizes the latest analytic and statistical methodology - providing deeper and deeper insights into the mechanisms underlying child development. Quantitative genetic methods include family designs, such as twin studies, adoption studies, and a recently developed 'adoption at conception design', where children are conceived through IVF technology with the possibility of donor sperm, donor eggs, and surrogacy, and therefore can be divided into several groups stratified by different degree of genetic relatedness between parents and children (Harold et al., 2012). A new addition to the quantitative genetic methodology toolbox - Genome-wide Complex Trait Analysis (GCTA) - estimates genetic influences on complex traits using genome-wide genotypes in large samples of unrelated individuals (Plomin & Deary, 2014). Comparing GCTA results to the results of family studies provides important insights into the genetic architecture of complex traits.

Many of the recent behavioral genetic findings might require shifts in our conceptualizations of the causal mechanisms underlying observed variability in educationally relevant traits. Quantitative genetic research challenges the mistaken view of genetic influences as deterministic. In reality, heritability only reflects the influence made by genetic factors in specific environments. For example, moderate to high heritability of most educationally relevant traits in the UK may reflect the uniformity of the UK Curriculum and teaching standards (Kovas et al., 2007). Indeed,

there is some evidence from international comparisons, suggesting that with a higher degree of variation in school types and quality, genes explain less variation in academic ability and achievement (e.g., Petrill, Deater-Deckard, Thompson, Schatschneider, & DeThorne, 2007). Heritability represents the proportion of variation in behavior explained by genetic factors compared to environmental factors; if the environment simply has a wider range of variation for a given population, genetic factors will necessarily explain less of the variation in behavior, and so the measured heritability will be lower.

Several recent studies suggest that genetic effects are dynamic rather than static - that the same genes may be expressed differently in different environments and at different stages of development. For example, several studies have found that genetic effects on general cognitive ability increase with age (e.g., Haworth, Kovas, Petrill, & Plomin, 2007; Kovas et al., 2007; Haworth et al., 2010). One recent study found that heritability of general intelligence was significantly lower than of literacy and numeracy achievement in the early school years, but increased by age 12 and became equal to that of literacy and numeracy (Kovas, Voronin et al., 2013).

One recent study of over 13,000 twins from 6 different populations (Kovas, Garon-Carrier et al., in review) found that motivation (enjoyment and self-perceived ability in different school subjects) is only modestly correlated even in monozygotic twins, suggesting that motivation forms largely under the influence of individual specific environmental factors. The moderate heritability of motivation, demonstrated in this study, is similar to the heritability of general cognitive ability in the early school years, suggesting that genetic effects are equally important for motivational and intellectual development. The study also showed that studying in the same classroom did not increase similarity among the twins in motivation, which is

consistent with previous findings of the absence of increased twin similarity in achievement and cognition when studying in the same class (Kovas, et al., 2007; Byrne, Coventry, Olson, Wadsworth, Samuelsson, Petrill, Willcutt & Corley, 2010).

This lack of a shared classroom environment effect may reflect the achievements of modern education: as a society, we provide all children access to quality teaching. This allows most children to reach a certain level, beyond which the differences are explained largely by genetic differences and unique experiences. In a less egalitarian society, the influence of educational shared environment on motivation and achievement would likely be much greater. A less optimistic conclusion can also be drawn: it is possible that the lack of the teacher effect reflects the absence of effective individualized educational methods. In other words, the lack of effect reflects a large diversity of pedagogical and instructional opportunities available that are not necessarily well matched to individual student needs.

The quantitative genetic methodology has also been applied to the important question of whether learning disability should be conceptualized as categorically different or etiologically linked to the normal individual variation. Research suggests that learning disabilities (for example, very low mathematical performance) lie on the same etiological continuum as ability. In other words, the *same* genetic and environmental influences are involved in placing someone at the very low end of the continuum, as are involved in placing one person just slightly below another in terms of achievement at the high end. It is the number and combination of such factors that determine each particular position (Plomin & Kovas, 2005; Plomin et al., 2009; Butterworth & Kovas, 2013). It is however possible that different factors operate at the very low or very high extremes of the variation. These possibilities will be

definitively tested in large-scale molecular genetic studies that examine individuals' DNA code (Plomin & Deary, 2014).

Multivariate genetic designs extend the principles of the twin method to address theoretically meaningful questions about the relationships between measures of educational interest. For example, if the same genes affect different traits (a biological phenomenon called *pleiotropy*), a *genetic correlation* is observed between the traits. The multivariate approach has been employed to address many fundamental questions in education, such as the relationship between reading and math abilities and disabilities (Hart, Petrill, & Kamp-Dush, 2010; Hart, Petrill, Thompson, & Plomin, 2009; Kovas et al., 2007); the association between reading, math, and attentional skills (Hart, et al., 2010); relationships between motivation and achievement over time (Luo et al., 2011); and relationship between reading and measures of the environment (Harlaar et al., 2011). Pleiotropy has been found across all of these traits, meaning that many genetic effects are general rather than specific to any one trait. For learning disabilities, substantial genetic co-morbidity has also been found, in that genetic correlations are high between reading, mathematics and language disabilities, and moderate between learning disabilities and other developmental difficulties, such as ADHD (Butterworth & Kovas, 2013; Plomin & Kovas, 2005). The same genes contribute to several disabilities.

Two recent multivariate twin studies examined the genetic architecture of academic achievement and found several important results for education. First, the heritability of achievement (measured by the state examination performance) was moderate for all school subjects, including mathematics, language, science, art and business (Rimfeld et al., in press). Second, the moderate-to-high observed correlations in performance in different academic subjects were largely explained by overlapping

genetic influences. Third, this large overlap in genetic influences on different academic subjects was not reduced (or reduced by very little) once intelligence was controlled for. In other words, to a large extent the same genes affected exam performance in different subjects (e.g., mathematics and art), even after accounting for any shared effects with intelligence. Fourth, genetic influences on achievement in core school subjects partly overlap with genetic factors affecting intelligence, as well as a whole range of other traits, including self-efficacy, personality, psychopathology, behavioral problems, health, wellbeing and even perceptions of home and school environment (Krapohl et al., 2014).

Quantitative genetic research has also provided important insights into environmental mechanisms. For example, family environment contributes very little to similarity between children in the same home*;* perceptions of environments are themselves partly heritable; and the links between educational outcomes (e.g., school achievement) and learning environments (e.g., classroom atmosphere) are partly genetic, potentially because environments are subjectively perceived (Plomin, DeFries, & Loehlin, 1977). Children evoke responses from parents and teachers in part for genetic reasons (sometimes called *evocative gene-environment correlation*); and children actively create environments that foster their genetic propensities (sometimes called *active gene-environment correlation*). Together, these examples show that a passive model in which the environment directly causes differences in children's development has to give way to an active model in which children create their own experiences, for example, by selecting and modifying their environments and by constructing perceptions of their experience and re-constructing their experiences in memory (Plomin, 1994).

Quantitative genetic designs remain valuable, providing a more refined genetic investigation of educationally relevant traits and paving the way for new molecular genetic investigations. The rapidly advancing molecular genetics aims to identify the actual genes that are involved in variation in traits. Today's behavioral geneticists have at their disposal a whole range of molecular genetic tools. These include new and continuously improving technologies (e.g., *microarrays* that allow to genotype hundreds of thousands of DNA markers simultaneously), statistical methodologies (e.g., *whole-genome sequencing* analyses), and increasing understanding of the biological processes (e.g., *epigenetic regulation* by which environments regulate genetic effects). Molecular genetics is possibly the fastest developing area in the history of human science. The area is still in its infancy, but has already provided many important insights into the origins and mechanisms of individual differences.

One method within molecular genetics that has seen particularly widespread use is Genome Wide Association Studies or GWAS. In this method, common variations in individual letters of the DNA code found within human populations can be correlated to variations in some trait or behavior (about 300 million letters of the DNA code show common variation in human populations, out of the total 3 billion, or 0.1%). Those variations showing reliable correlations may indicate that the genes within which the DNA letters reside are contributing to biological pathways that produce the observed variation in the trait. The method allows snapshots of the whole genome to identify regions that may be important for producing variation (Edwards, Beesley, French & Dunning, 2013). However, the method requires large sample sizes, because the correlations between variation in individual DNA letters and high-level traits tend to be very small. GWAS have been successful in identifying biological

pathways for disease (Hindorff et al., 2009; Visscher et al., 2012), and they have the potential to inform our understanding of biological mechanisms of learning.

GWAS studies have recently been applied to educationally relevant skills such as language, reading, and mathematical skills. For example, Meaburn et al. (2008) carried out a GWAS exploring genetic sources of variation in reading ability in over 5000 children. Notably, these authors found no statistically reliable associations between individual DNA letter variations and reading ability. The key implication is that individual genetic effects are weak (too small to be detected in this study) and that many, many genetic variations must contribute to the variation in reading itself. There have now been a further five well-powered GWA studies published since 2013 exploring reading, some in conjunction with other abilities such as language and mathematics, and one focused on disabilities, all with large samples from 2000-5000 individuals (Davis et al., 2014; Eicher et al., 2013; Gialluisi et al., 2014; Harlaar et al., 2014; Luciano et al., 2013). None of the studies identified any individual DNA sequence variants that both replicated and were also genome-wide statistically significant, the key criteria for a robust finding. Together, the studies indicate that the biggest effect sizes for associations between genes and reading and/or language ability are much smaller than researchers initially expected, implying that smallest effect sizes must be extremely small. In other words, variations in hundreds, if not thousands of sequences of DNA contribute to variation in educationally relevant skills.

Rietveld et al. (2013) recently performed a GWA study investigating variations in genetic code that correlate with educational attainment, in a very large sample of 125,559 individuals. Educational attainment was measured either as the number of years of schooling or as a binary variable of whether the individual had completed

college or not. The approach taken was to employ crude measures in order to get large samples with good statistical power to detect associations. Previous studies have suggested a heritability of around 40% for educational attainment, that is, the majority of the variation is of environmental origin but a substantial chunk is genetic. Rietveld et al. found only a small number of DNA code variations that were genome-wide statistically significant and which replicated (that is, only 3), with effect sizes of around 0.02% each (corresponding to 1 month of schooling). In total, all measured genetic variation only predicted 2% of the variation in educational attainment. Two points are worth noting. First, what mechanisms might the genetic variation be influencing? Rietveld et al. related the observed associations to pathways impacting on health, cognition, and the central nervous system, and identified one potential brain mechanism, the anterior caudate nucleus involved in controlling goal-directed action. Second, the total variation in educational attainment explained by the molecular genetics study, at 2%, falls far short of the heritability measured by behavioral genetic methods, of around 40%. This gap between molecular and behavioral approaches is a general issue, known as the problem of *missing heritability* (e.g., Manolio et al., 2009). It demonstrates that the two historical traditions in genetics, of studying heritable traits versus studying the biological mechanisms of inheritance, have yet to be unified.

For educators, there are two major problems with data like those from the Rietveld et al. study. First, the predictive power of genetic variation is small. Second, the data describe large populations, whereas educators are interested in individuals. How can we proceed? One way forward is to use what is known as a *polygenic score*. This involves aggregating the small effects of many DNA variants to create a single score for an individual, based on the particular variants the individual has and whether

these variants have positive or negative correlations with the ability in question. This score is only probabilistic: it indicates an individual's risk of a good or bad outcome. This type of evidence, however, might help inform the creation of guidelines to help make decisions for a particular child about the sorts of techniques or educational environments that may produce better outcomes.

It is clear from the range of methods and the recency of many of these findings that genetics is a fast-moving field, yet it faces many challenges. Indeed, it is possible that the information on our complete genomic profiles will be routinely available long before we can truly utilize this knowledge. The process of tracing a path from each genetic variant to behavior may take a long time – and further leveraging of this knowledge will also require understanding of the mutual impact of individuals and their environments. Research into the exact mechanisms by which each gene affects a trait is complex and involves multiple levels: from gene expression profiles, to specific protein functions, to physiology, and often to the structure and function of the brain viewed in a developmental context (Thomas, Forrester & Ronald, in press). It is currently difficult to foresee very specific applications of molecular genetic research to education, but this new area of scientific endeavor offers much promise.

Integration and translation

Despite its promise, we need to be clear about the difficulties involved in applying genetic research to education. Educational neuroscience holds that complete accounts of learning – focusing on just this aspect of education – require explicit integration of previously diverse strands of research, including that on brain function. The evidence discussed above suggests we need to include genetics in this integrative effort, but this is challenging given the order of complexity that may be involved.

The missing heritability issue illustrates why. One plausible explanation of the small effect sizes associated with specific genetic characteristics in Rietveld et al. (2013) and other GWAS studies is that simple main effects are scarce, and a large proportion of variation in individual outcomes stems from the cumulative impact of higher order interactions between genetic variants, environmental events (not just general 'factors') and neurophysiological functions. If so, the work required to track these interactions will be considerable, and both missing heritability and missing environmental influences will present challenges, since the same complex interactions obscure both.

The 'polygenic score' approach assumes a simpler model in which genetic effects are many but essentially additive. Its success in predicting individual outcomes will therefore indicate how far we actually need to explore complex gene x brain x environment interactions. Even if this approach does prove productive, however, we will still need to understand how these additive influences operate and what we might do about them – and this depends on working out *which* outcomes we are interested in, and measuring these appropriately.

A recent adoption study by Beaver et al. (2014) study highlights the dangers. The authors claimed to show an absence of environmental influence from parenting style on variation in later verbal IQ, with the implication that genetic factors were primarily responsible for IQ differences. However, their measure of IQ was simplistic (the short version of Peabody Picture Vocabulary Test), and their measure of parenting was limited to eight items predominantly focused on attachment, which has little obvious direct relevance to verbal ability. Even when variables are selected on good theoretical grounds, GWAS and polygenic score methods demand large samples. This almost inevitably means that non-genetic measures are restricted in scope,

because they are lowest common denominator solutions chosen to ensure manageable data collection. The measures of educational attainment used by Rietveld et al. (2013) are characteristically blunted: years of completed schooling, and whether or not participants had a college degree. However, impoverished measures entail limited conclusions.

Finally, if *translational* research is the objective, we need to bring our conclusions to bear on intervention at level of the individual learner. The difficulties are illustrated by research on developmental disorders with a range of genetic polymorphisms, such as Williams syndrome (WS). WS is a neurogenetic disorder caused by the deletion of a stretch of DNA from one copy of chromosome 7, containing around 28 genes. Broadbent et al. (2014) investigated two cases studies with different, smaller genetic deletions that were subsets of these 28 compared against a wider WS group. Despite identified deletions, the two cases exhibited unexpected cognitive profiles: the case with the more typical deletion pattern showed the more atypical cognitive profile compared to usual WS, with better non-verbal reasoning performance than would be expected, while the other case exhibited profound impairments although the majority of genes in the WS 'Critical Region' were intact. This constrained situation should have offered straightforward insights into the roles of individual genes, particularly given the small number of genes involved and that the case studies represented deletions of subsets of the WS Critical Region. That it did not supports the view that elucidating which specific genes play what role seems likely to be challenging - let alone then anticipating the appropriate form of intervention.

Given these complexities, we need to start by determining *what* we are trying to achieve:

- Improved outcomes for those at the lower end of the range? Pushing for good, consistent standards in educational and home environments would be the best approach here, and if *apparent* heritability increased as a result, this would buy space to understand better the mechanisms involved. We are a long way from this at present, though.
- Enhanced benefits for those at the higher end? This is implicit in Asbury and Plomin's (2013) contention that everyone's genetic endowment should be helped to flourish, but it is controversial territory given its eugenicist resonances. However, if resilience, for instance, has genetic influences, might we not reasonably want to increase the impact of these?
- Improved outcomes for those with more extreme developmental disorders with a known genetic influence? This is the most achievable goal in the short-term, but still challenging. However, the use of genetic markers for dyslexia (Schulte-Körne et al., 2007) as a basis for early intervention using established phonological training techniques illustrates the potential benefits.

We need to make reasoned choices about these possibilities in order to steer work over the next 10-20 years. To ignore the influence of genetic factors is not an option: if the objective is to build *full* models of the factors shaping learning processes as a basis for informed, evidence-based educational practice, how can we not take genetics into account?

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