Current Genetic Discoveries and Education: *Strengths, Opportunities, and Limitations*

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ABSTRACT—This article notes that many key positive developments in education originated in research on the structure and genetics of abilities, providing primary evidence for ability in disadvantaged groups and playing a critical role in demonstrating the existence of developmental learning disorders and effective interventions. It is argued that new work in genetics offers similar hope, but that widely held beliefs about genetic equipotentiality and brain plasticity act as roadblocks to research improving educational outcomes. It is suggested that rebuilding the collaborative links between educationalists and psychometric and genetic research that functioned so effectively in the early development of universal education can again underpin improved educational outcomes.

INTRODUCTION

Just as the first images of Earth taken from space proved to be perspective altering, so too images of neuronal migration and proliferation under genetic control in the fetal brain not only change our understanding of cognition (Galaburda, LoTurco, Ramus, Fitch, & Rosen, 2006) but also cast the job of education in a new perspective. This perspective offers a substantial leap in our understanding of the difficulties faced by children as they strive to absorb the content and the tools of culture. It can allow both earlier, more timely delivery of interventions and lead to new ways for educators to help children and adults with difficulties in learning. Taking advantage of these opportunities depends critically on adopting a model in which genetic information can be translated into educational outcomes. This article addresses significant roadblocks to the generation and translation of information from genetics into educational practice, touching on the validity of twin research, limits to neocortical plasticity, the magnitude of individual differences, and the implications of gene discovery for educational practice.

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EFFECTS ON EDUCATION OF PSYCHOMETRIC AND NEUROPSYCHOLOGICAL RESEARCH

Abilities research, genetics, and cognitive neuropsychology are domains of psychological science of particular relevance to the education. Significant misgivings exist about the past present relevance of work in these fields for current and future practice, especially where genetics and brain science are concerned (Hirsh-Pasek & Bruer, 2007). These perspectives have, however, placed an enormous, almost definitive stamp on education since they arose early in the past century. Indeed, they have accompanied the development of universal education itself.

Arguably, the most compelling outcome of ability research was the finding that mental abilities show a predictable increase across development, and that different abilities such as math and spatial reasoning are highly correlated, a finding replicated in measures of the full the breadth of human ability including not only analytic but also practical and creative abilities find a strong general factor of ability (Sternberg, 2006). These two findings of developmental increase and cross-sectional coherence can be seen in at the heart of the design of universal education and the design of school systems: Progressively, more difficult content is introduced over successive years, with difficulty level being held roughly equal across subjects within a given year. Many schools supplement this scheme with support for individualized teaching—acknowledging the interaction between ability and teaching such that different children suit different rates and even forms of content presentation, implemented as streaming or "setting" based on grouping children of similar ability. These two

principles map directly onto the developmental progression of ability from infancy into the late teens uncovered by early research on intelligence, as well as the large stable differences in ability between children (Binet, 1905/1916). Importantly, because Binet and Spearman argued that intelligence tests should avoid material that depended on previous experience, ability tests were able to demonstrate talent in groups previously argued to be "unsuited" to advanced education. Thus, as the system of schooling and written examinations became common in the 1860s, the primary evidence for the equal aptitude of women for education came from psychometric ability testing, which demonstrated the equal talent of women and supported opening access to higher education for women (who score at the same average level as men on ability measures; Deary, Irwing, Der, & Bates, 2007).

The neuropsychological model was more relevant to recognizing the needs of children with learning disorders, providing both the diagnostic instruments and the remediation strategies in disorders as diverse as attention deficit hyperactivity disorder (ADHD), dyslexia, autism, and specific language impairment. Prior to the interest of cognitive psychology and the advent of sophisticated neuropsychological testing, these disorders were often not recognized, misclassified as general dullness, or were simply greatly underdiagnosed. Research since these early discoveries has continued to revolutionize the ways in which we understand and approach treatment for learning disabilities—for instance, researchers in autism have recently demonstrated that autism is unlikely to be a single disease but rather appears to fractionate into at least three distinct and independent components involving affective processing, language, and flexibility of thinking: each with distinct biological bases and therapeutic needs (Ronald et al., 2006). Psychometric work on the developmental progression of abilities, and the identification of specific abilities, then, has had a critical role in shaping the creation and delivery of universal education.

BARRIERS TO INTEGRATING MIND, BRAIN, AND EDUCATION

Despite their role in differential diagnosis and early, effective therapy, there are currently very strong barriers to the translation of genetic research into education: To quote, Blake and Gardner (2007) on the introduction to Harvard of the Mind, Brain, and Education Program, a common question asked of them was "Can't you leave out the brain?"

The idea that the brain is not relevant to education highlights an "explanatory gap" where, while nearly all researchers have long since agreed that nearly all human behaviors have a substantial heritability (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), there remains a strong thread of discourse that rejects a role of genetics or even of biology in normal individual differences in cognition and learning. In this article, I use the device of referring to comments in six recent public discussions of genetics I have heard broadcast or have attended in the past year, to frame a discussion of the causes of barriers to including genetics and the brain in education experienced by Blake and Gardner (2007).

Over 2006–2007, I have heard the following comments (anonymized to avoid embarrassment):

1. "The assumptions of twins studies are invalid—we can discount their results" (professor of sociology).
2. "The heritability studied by twin researchers is, of course irrelevant, but I will talk today about interactions" (professor of population genetics).
3. "Language can't be genetic, can it? Certainly reading can't … it's learned" (two professors of linguistics).
4. "Gene × Environment interactions mean there are no main effects, no 'risk' genes" (research council executive). Attempts to apply genetics to traits relevant to education are logically impossible, as, while genes exist within individuals, cultural processes such as education "are transactions, and the two are therefore incommensurable" (professor of sociology).
5. "Genetics is just a fishing expedition … there's no theory" (professors of philosophy and sociology).
6. "Genes can't really limit development, because we now know that the brain is constantly generating new neurons in response to the environment" (professor of sociology, science journalist, two professors of education).

I argue below that each of these views is in whole or in part untrue, mislead the public about the nature of individual differences, and the reasonable outcomes that changes in education can be expected to produce, and create barriers to mutual respect and communication between psychology, genetics on the one hand, and educators on the other hand. Because sophisticated research and long-term research initiatives are required to discover and translate genetic information into opportunities for improving childhood learning, it is important that these barriers are broken down. I briefly address each of these issues in turn below.

1 and 2: Twin Studies and Heritability Research Are Invalid

The traditional twin study is one of a family of research designs using individual differences in genetic and environmental similarity to divide the curve of scores in a population into components, reflecting additive and nonadditive genetic effects, shared or within-family environment, and a residual component consisting of variance unaccounted for by these other effects.

The twin method has one very important assumption: that of equal environments for MZ and DZ twins. Observers often

believe that this is unlikely to be true for twins, given their identical appearance. Of course, this criticism embodies an implicit theory of development: It acknowledges that physical appearance is genetic (allowing MZ twins to be identified by parents) and further implies that similar treatment is triggered by sharing of traits such as height and eye color, such that parents give both members of an MZ pair exceptional reading skills, high or low IQ, or even autism. Even more paradoxically, because heritability reflects excess similarity in MZ twins compared to DZ twins, the environmental theory implies that parents drive siblings and DZ twins apart in their scores on these traits, somehow choosing one child to give neuroticism or intelligence based on a physical trait such as eye color. Quite how this might occur is not specified. However, it is not necessary to critique this counterintuitive implicit theory of development: Instead, the assumption of equal environments can, and has been, tested in two very insightful ways.

One test of the equal environment assumption is to examine what happens in cases of mistaken zygosity, when DZ twins are as identical or vice-versa. Careful work on such cases indicated that zygosity beliefs do not explain heritable similarities and may even reduce them (Kendler, Neale, Kessler, & Heath, 1994). Recently, a dramatically more powerful examination of the assumption was proposed, using molecular genetics to directly measure genetic relatedness in nontwin siblings, creating an assumption-free equivalent of a continuous dimension of "twinness" based on chance variation in gene sharing for which causes any pair of siblings to differ by a small fraction from 50% gene sharing. Despite involving no twins, and thus avoiding the possibility of special identity effects, this study showed an identical high heritability for IQ to that in a conventional MZ–DZ sample from the same population (Visscher et al., 2006).

A second criticism often raised is that as twin studies often show modest to zero effects of family environment, the models *must* be flawed. First, studies in populations with extremely low social resources do detect effects of family environment (Turkheimer et al., 2003). But the general lack of shared environment effects in many studies is not a weakness but *a success story for western education and parenting*. Low environment effects mean that the school system carefully crafted and optimized over the past century, seems able, largely but not entirely, to compensate for differences between family environments, at least in gross outcomes of IQ, grades, and so forth. Education should have a humble pride in reducing shared environment to close to zero.

What then of the related claim that heritability is irrelevant, now that we can directly study particular genes? It is the case that if we could measure all genes directly, we would not need to statistically assess heritability—it would appear as a large number of measured or "fixed" genetic effects as opposed to a latent or "random" effect to use the statistical jargon. However, we are far from knowing all the genes affecting behavior, and until then, heritability remains critical for modeling. In education, heritability remains our most powerful tool for predicting the nature of the genes that exist, of modeling the consequences of genes even before they are known, and of quantifying both genetic diversity within disorders and gene–environment interactions. A powerful example of the utility of heritability is in revealing overlaps and distinctions between disorders. For instance, research reported in this journal and elsewhere indicates that rather than reflecting environmental effects, almost all the general of *g*-factor of ability underlying is due to shared genetic influences that increase across development (Plomin, Kovas, & Haworth, 2007). By contrast, the same techniques suggest that the global trait of autism is an amalgam of at least three genetically distinct vulnerabilities (Ronald et al., 2006). Both these findings from heritability research are useful guides to assessment, remediation, and research.

3: "Language Can't Be Genetic, Can It? It's Learned"

Individuals differ significantly in their biological preparedness for reading as reflected in the high rates and high heritability of dyslexia. Nevertheless, statements such as "we know there are no genes for reading" are routinely made by educationalists and researchers alike. The statement is amenable to several interpretations. One simple interpretation is that "reading is not influenced by genes." Read in this way, the claim is false: Although home environment and effects of social and economic status on reading decrease sharply after age 5 in societies where universal education begins at that age (Byrne et al., 2005), normal variation in reading and spelling is one of the most heritable of human traits, with some elements of reading not much less heritable than height (Bates et al., 2007). A similar result holds for cognition more broadly, including general intelligence, with heritability rising from around 0.3 in childhood and increasing through adulthood up to around 0.8 in old age (Deary, Spinath, & Bates, 2006).

A second interpretation of the claim that there cannot be genes for reading is that this would be logically impossible: That the 5,000-year period during which reading has been developed by humans is too short to have allowed evolutionary changes to support reading. Recent evidence, however, demonstrates that several novel gene mutations have spread across entire groups of humans in this time (blue eyes and processing the sugars in milk are good examples) and that a number of novel alleles have spread across the globe over since the advent of civilization (Mekel-Bobrov et al., 2005).

A third interpretation of the claim that "there are no genes for reading" might intend it as claiming that, although there are genetic effects on reading, and some allele frequencies may even be changing in response to selection for reading, no genes (or brain systems) can have evolved specifically to support

reading. This is a testable hypothesis, and the answer may have important impacts on education. If no functional cognitive changes have evolved to support reading, the lexical and grammatical systems of reading may be created by "recycling" cortical regions able of whose circuits can be programmed for novel functions, at least during early development (Dehaene, 2007), possibly at the cost of decreases in the ability to represent information that would otherwise use these areas.

This recycling hypothesis predicts that reading occupies brain tissue that would otherwise be available for processing other information—perhaps regions evolved to detect and store complex visual patterns, as exemplified in face processing. One finding that mitigates against this view is that it implies that differences in reading are mediated by brain structures that evolved for generic reasoning, or for perceptual or linguistic tasks. Many claims of this nature have been made, but no tasks of reasoning, vision, or acoustic processing have been found, which remotely well predict single-word reading, and perceptual tasks uniformly show very modest relations to reading (Barry, Yasin, & Bishop, 2007). This suggests that reading may be a distinct ability of the human mind, not entirely explained away by recourse to either language evolution or object recognition, and that reading may therefore possess a specific genetic architecture and evolutionary history.

Given that reading differences are not explained by general cognitive or perceptual differences, my group is exploring the hypothesis that the 12 genes and chromosome regions so far associated with reading reflect recently evolved changes in neuronal migration patterns, allowing the use of graphical symbols to express grammar and to access semantics, based on the selective benefits of written communication. The neuronal changes, if any, made to allow a generative symbol processing system paralleling spoken language are unclear but discoverable. Several authors have suggested that the origins of language lie in systems designed for fine motoric actions or signals, and this may have ensured that modifications required to support writing and perhaps the mirror process or reading were relatively undemanding or even preexisting (Corballis, 2003). It seems likely, however, that selection for reading may have required at least some significant genetic modifications in existing systems to rewire the brain for reading, as hinted at by the functional role of the first of many genes involved in reading that appear to impact the nature of the migration diagram of the brain—for instance by the genes *ROBO1* and *DCDC2* expressed early in fetal brain development (Galaburda et al., 2006).

4: Gene × Environment Interaction: Genes, Environments, and Social Transactions

A fourth criticism of genetics suggests that applications of genetics to traits relevant to education are logically impossible. This deep skepticism of the ability of genetic research to uncover causes of traits important in sociology and education is important to address. If correct, it implies that genetics cannot be meaningfully applied, and not just to traits such as intelligence and language but also to any social trait, such as personality, altruism, sexuality, criminality. A philosophical version of this claim would suggest that social behaviors do not exist in individuals. Prima facie evidence against this view is provided in the existence of large individual differences within highly similar environments (Rowe, 2001). Far more common is the belief that it is unreasonable to speak of "risk genes," as any apparent "risk" associated with a gene in one environment will be balanced by a matching benefit or protective effect in another environment. Taken seriously, this otherwise comforting view of individuals as equal but different would suggest that there are many children for whom knowledge is decreased by exposure to school or that there are youngsters for whom exposure to standard literature programs would decrease literacy. In fact, exposure to school leads to year-on-year learning for all children, albeit at different rates. Likewise, although there is substantial genetic evidence that some children will benefit most from phonics while others would benefit from lexical development (learning whole words), there is no support for different ways of teaching reading programs that reverse the dyslexia and good-reader groups.

Empirically, rather than showing crossover effects where alleles are a risk factor in one environment and protective in other environments, genes appear to largely act as multipliers of the effects of the environment (Caspi et al., 2002). There is some evidence in psychiatric genetics for Gene × Gene interaction, where allelic effects depend on other genes: For instance, in the dopaminergic system, the effect of a gene decreasing dopamine sensitivity is contingent on whether other elements of the dopamine system are causing low, average, or high levels of dopamine (Mattay et al., 2003). This same article, however, demonstrates the main effect of overall dopamine levels. Thus, interaction at a Gene × Gene level does not imply a lack of net genetic main effects.

5: Is Genetics a Fishing Expedition? Is That a Bad Thing?

The quote expressing the idea that genetics is just a fishing expedition and that geneticists have no theories and therefore no understanding of the traits they seek to explain perhaps summarize the lack of contact between genetics and education at present (it would otherwise be hard to be as curtly dismissive of the talents of others). The broad claim that genetics is a "fishing expedition" is false. Genome-wide studies of variation do, by design, cast a wide net, but many other genetic studies are deeply guided by theory: For instance, candidate gene research, in which a process–dopamine metabolism or the effects of breast feeding—is established, a theory for its role in the disorder is developed,

one or more genes for the protein discovered, and the role of this specific candidate tested (Caspi et al., 2007; Mattay et al., 2003).

Given that we have no adequate biological models for understanding most processes in education, fishing expeditions (technically called genome-wide association studies) are necessary, wise, and effective (The Wellcome Trust Case Control Consortium, 2007). There is no alternative method for catching fish than to go fishing. In this case, the "fish" are potential cures for some of the most common and devastating disorders affecting us, and the "fishing" is bringing home catches related to normal cognitive variation (Bates et al., 2007; Butcher et al., 2004; Posthuma et al., 2005), turning rather immediately into theories of brain development, with clear implications for diagnosis, and even potential treatments (Galaburda et al., 2006).

6: How Plastic is the Brain?

A final, commonly expressed belief that acts to diminish the translation of genetic research into educational outcomes is the idea that new functional neurons can be generated in the neocortex throughout life and that this universal ability creates a pool of plasticity, allowing compensation for early difficulties. However, although there is evidence for tiny amounts of neurogenesis in the olfactory regions of the adult brain, innovative experiments have provided powerful evidence that very little, if any, neurogenesis occur in the neocortex after birth in humans (Bhardwaj et al., 2006). This study assessed the creation date of neuronal cells based on levels of radioactive isotopes emitted during the period in which aboveground nuclear testing was practiced, creating a global date signal as these isotopes were incorporated during fetal development but were absent before and immediately after the advent and banning of nuclear testing. As such, they give a high degree of certainty that neurogenesis does not occur, outside a handful of neurons destined to replace neurons exposed to the environment, particularly in the olfactory bulb (Bartlett et al., 1998).

PROSPECTS

If we take seriously the notion that genes will interact with environments, then research is genetics is by definition critical to delivering effective education predicated on the individual child's genetic makeup, and one that can be accomplished with current genetic technology.

What would reducing these barriers to communication between educators and genetics researchers achieve? The advent of genetic information about cognition has to date had very positive effects. Dyslexia, prior to the advent of genetic models in the early 1980s, was largely and variously viewed as nonexistent, a result of poor care by parents, poor teaching, or laziness on the part of the child. Compelling evidence for high heritability did not result in abandonment or ostracism of children at risk for reading disability. Instead, a period of great activity in brain sciences, behavioral therapies, and teaching methods was designed to allow all children to access written language. A similar pattern of positive change occurred in autism. Previously believed due to "refrigerator mothers," evidence for high heritability (Folstein & Rutter, 1977) helped focus work on the cognitive basis of autism (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), the fractionation of its components (Ronald et al., 2006), and therapeutic innovations, as well as a public acceptance of the disorder and a dramatic increase in therapeutic resources consequent on a spectrum diagnosis.

If genetic tests were ever to become available for disorders such as dyslexia and autism, it seems likely that their effects could be highly beneficial. Genetic tests can assess risk well before any behavioral signs, allowing focused intervention much earlier than ever before. More speculatively, understanding the molecular genetic pathways of disorders such as autism may lead to entirely novel, biologically informed therapies. This seems ambitious and remote, given our lack of neurologically understanding for complex disorders. The dramatic response of phenylketonuria (PKU) and diabetes to simple biochemical interventions, however, suggests that substantial improvements or even elimination may be possible when the triggers of gene expression in pathways to dyslexia, autism, ADHD, and even general intelligence (as in the case of PKU) are understood.

CONCLUSIONS

We are moving out of an era in which opinions about genetics of cognition were to some extent reflections of individual's attitude toward genetics, with people falling along a continuum from skeptics to enthusiasts, with most people falling in the middle, but the debate dominated from either end (Rutter, 2005). Now however, individual gene variants are being found, with observable effects on brain development and on traits such as reading and autism. Knowledge of the genetic basis of behavior affords great opportunity, not least of which is that differences in educational aptitude can be acknowledged and addressed, rather than reflexively blaming teachers for "leaving children behind"). This new knowledge seems likely to promote not only acceptance and additional resources for tailored programs (as is beginning to occur for autism and dyslexia) but also earlier and more effective intervention. Achieving this, however, requires a shared decision to encourage a partnership between genetics and education in vigorous pursuit of the genetic and environmental basis of cognition and educational outcomes.

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