Chapter 11

Education 2.0: genetically-informed models for school and teaching

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Overview

School is intended to raise life-course outcomes for children and society. What we do in school to achieve this necessarily depends intimately on our beliefs about how children’s minds work. Key questions include the following: do children differ in their ability to learn, or are they all identical? Is the mind a blank slate or a complex set of specialized cognitive mechanisms? Do differences in learning simply reflect prior experience? Are these differences remediable through experience and neuronal plasticity? Would successful teaching minimize or increase differences in capability between children? Are differences in children largely due to gene-by-environment (G×E) interactions, such that all children can achieve equal outcomes given the correct special environments or teaching styles? This article uses behaviour and molecular genetic examples to address these questions. Of course, not all answers are clear, and many are surprisingly preliminary given the importance of education to society. It is suggested that neuroscience indicates that children’s minds are made up of multiple genetically and psychologically distinct mechanisms underlying learning; that children differ in the functioning of these systems; and that much of this difference is genetic in origin and general in nature, with limits to brain plasticity. A systems framework is presented linking these basic biological bases to the lifespan thriving which school is intended to foster. This systems model suggests that the principal outcome of neuroscience research will be to focus attention on how non-neuroscientific choices impact learning: teacher selection and training, teaching methods and curriculum. The chapter concludes by suggesting that much of education is based on different answers to the research questions posed here than those flowing from current neuroscience. If we are to maximize the capability of all children, it is suggested that it will be important to achieve consilience between what we know about the mind and what educationalists do.

11.1 Introduction

To discuss even a fraction of the information flowing in a single year from biological and genetic approaches relevant to understanding children’s learning would take an encyclopaedia. As noted in other chapters in this volume, our understanding of disorders such as dyslexia, language impairment, autism, etc. is voluminous. Rather than summarizing the information of relevance
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for teaching or curriculum regarding research on some fraction of this information, here I focus on
describing how genetic and biological research on individual differences provides frameworks that
help us understand what education can do for children, how education may work and what it is
reasonable to expect from education. The central emphasis of this chapter is on a framework linking
basic biological factors to the outcomes which education fosters, such as prosperity and independ-
ence, via an intervening layer of cognitive systems. It is these cognitive systems that interact with
education and which are the proximal causes of behaviour that education is designed to enhance.

Education appears to be important for a range of reasons, not least of which is its strong asso-
ciation with social position (Gottfredson, 2004). At an international level too, education is highly
correlated with technological prowess and with GNP (gross national product) per capita. This
seems to be due to the association of education with the skills and adaptability of employees
(Rindermann & Ceci, 2009). Finally, in societies with a broad electoral franchise, the task of
ensuring that citizens are culturally literate was deemed important enough that education is free
and compulsory until late adolescence. Doing a good job at education, then, seems critical, and
for that reason around 5–7% of the GNP of developed nations is devoted to education, provided
by most states at low or zero direct cost.

But what does education do—what happens at school, and can behaviour genetic research tell us
anything about this critical social enterprise? What would be seen as a mainstream view from the
 genetics of behaviour? Most important among the results from research, I suggest, are the follow-
ing three. First, the finding that the mind is not a holistic instrument of infinite capacity, but rather
a collection of dissociable specialized systems (see, for instance articles in Rapp, 2002), under-
pinned by distinct genetic mechanisms (e.g. Bates, Castles et al., 2007). Second is that much of this
genetic variance is shared across the distinct components of the mind, creating a general ability
factor explaining much of the differences between children (Deary, Spinath & Bates, 2006; Kovas
& Plomin, 2006). Third and arguably most important is that much of these differences between
children in mental capacity originate in genetic differences (Royal Society Working Group, 2011).

Some key ‘explanatory gaps’ remain, however, between educators’ understandings of what
children’s biology brings to the classroom compared to what neurobiological and genetic research
indicates. Recent commentaries have documented a desire to ‘leave out the brain’ in education (Blake
& Gardner, 2007). This view that brains don’t matter is in contradiction to the three claims noted
earlier, and also represents a strong thread of discourse in education rejecting a role of biology in
cognition and learning, and therefore in education (Hirsh-Pasek & Bruer, 2007). This view is similar
to that expressed humorously in Garrison Keillor’s radio broadcasts when he described the fictional
US Midwestern town of Lake Wobegon where ‘all the women are strong, all the men are good looking,
and all the children are above average’. From ‘no child left behind’ to anguished newspaper headlines
regarding differential success at entering university predicted from class background, this idea that
education should be able to achieve equal outcomes for all children irrespective of the child distracts
attention from understanding effective strategies for educating. Indeed, as argued later in this chapter,
those practices more likely to increase the capabilities of children may also magnify these differ-
ces (e.g. see Figure 11.2). Because evidence that the mind is componential, and with large genetic
individual differences in the performance on these components is basic to answering this question of
what education should be capable of, it forms the topic for this first section in the present chapter.¹

¹ Much of this argument depends on information regarding the different perspectives on learning and develop-
ome emerging from the individual differences, genetics and neuropsychology perspectives. In order to
avoid distracting from the central argument presented here, a discussion of the way in which behaviour–
genetic models of cognition can be integrated with those flowing from neuropsychology and differential
psychology is presented in appendix 1.
Differences on educationally-relevant traits have a genetic component

Nearly all researchers have long since agreed that nearly all human behaviours have a substantial heritability (Turkheimer, Haley, Waldron, D’Onofrio & Gottesman, 2003). All educationally relevant traits from reading ability (Bates, Luciano et al., 2007; Haworth et al., 2010; Olson & Byrne, 2005; Paracchini, Scerri & Monaco, 2007) to autism (Ronald et al., 2006b) to intelligence (Deary et al., 2006) are heritable. Indeed, these three are among the most heritable. The same is true for non-cognitive resources such as conscientiousness, and curiosity (Luciano, Wainwright, Wright & Martin, 2006).

Most of our information about the genetics of educationally relevant traits comes from twin studies. Perhaps the single most challenging, but important finding from twin research is that fraternal twins’ scores on ability measures and educational outcomes correlate much less than do those of identical twins, despite shared social status, neighbourhoods, parents, diet, housing and, often, schools and teachers (see Chapter 9, this volume). This sharp drop in correlation is as expected for traits that are highly heritable, and suggests that family environments have modest effects (Plomin, 1991). A caveat is that some (Turkheimer et al., 2003) but not all (Asbury, Wachs & Plomin, 2005; van der Sluis, Willemsen, de Geus, Boomsma & Posthuma, 2008) studies in populations with extremely low social position show large effects of family environment on cognition which suppress genetic variance (Tucker-Drob, Rhemtulla, Harden, Turkheimer & Fask, 2011). The general lack of shared environment effects is a success story for education (Bates, 2008a). They mean that our school systems, carefully crafted and optimized over the last century, largely but not entirely, equalize differences between family environments and the ability to profit from education they provide (Haworth, Asbury, Dale & Plomin, 2011). I think we should take pride, therefore, in having reduced shared-environment as close to zero as we have, while focusing on effective school interventions which address residual shared environmental effects in low-socioeconomic status groups. Teacher quality appears to be a critical factor in this respect (Taylor, Roehrig, Soden Hensler, Connor & Schatschneider, 2010), suggesting that more rigorous teacher selection and training may be the single most significant factor in addressing social status effects on the cognitive underpinnings of capability.

Molecular genetics: learning has mechanisms

If minds and brains are complex cognitive mechanisms as indicated by differential, genetic and neuropsychological research (see Appendix 1), then education is the process of working with these mechanisms to maximize the capability of the developing child. Molecular genetics is in its relative infancy compared to psychology, and currently few, if any, biological mechanisms are well understood, especially for complex cognitive systems such as memory and reasoning. However, the last decade has seen a dramatic increase in genetic knowledge, mostly based on increases in the power of molecular genetics, available now at relatively very low cost. Beyond the ultimate benefit of showing the mechanisms underlying learning, concrete genetic associations give a sense of reality to difficulties in learning which are often claimed to be malingering or an unwarranted call for special attention. A second finding of direct relevance to educators is that molecular research has confirmed that the same genes are responsible for the full range of normal variation, for instance, in reading ability and intelligence seen in the classroom as are involved in severe clinical reading disorder or dyslexia (Bates, Luciano et al., 2007) and learning disability (Kovas & Plomin, 2006) respectively (see Chapter 10, this volume). This tells us that biology does not apply to only special cases, but that all children face the limits and potentials of their brain...
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mechanisms for learning. Concrete genetic associations thus provide evidence for the reality of
difficulties in learning and for continuity between normal and abnormal.

Moving to candidate genes, researchers have begun to find the pathways in which these effects
operate. In the case of reading, all of the genes discovered to date appear to play a role in fetal
neuronal migration. **DYX1C1**, for instance, affects the short-range laminar migration of axons,
resulting in both inappropriately increased and foreshortened intracortical connectivity (Rosen
et al., 2007). These specific genes alter neuronal migration in ways that are believed to underlie the
cognitive difficulties encountered in dyslexia. At this gene-specific level, the same variants
involved in clinical dyslexia are reliably found in normal samples, unselected for dyslexia. Thus
**KIAA0139** (Luciano et al., 2007), **DCDC2** (Lind et al., 2009) and **DYX1C1** (Bates et al., 2010) have
all been linked to normal variation in reading ability. The study of other traits relevant for educa-
tion such as language and long-term storage and learning is in its infancy, but already gene effects
are being found which mediate differences in learning mechanisms in spoken language (Bates
et al., 2011; Hannula-Jouppi et al., 2005) and for memory or the retention and recall of informa-
tion over time (Papassotiropoulos et al., 2006). Both these traits are, of course, of direct relevance
to understanding the mechanisms of learning in the classroom.

Understanding the underlying biochemistry of these mechanisms casts a powerful light on the
origins of differences in learning in the classroom. The new genetics opens possibilities for earlier
and more targeted diagnosis (these opportunities for translational research are outlined briefly in
Appendix 2). The nature of the structural biological differences coded for by these genes may
also carry some implications for the kinds of plasticity teachers should expect in children. These
factors are discussed next.

**Plasticity and limits to plasticity in complex systems**

Breakthroughs allowing researchers to selectively knockdown genes of interest during development
(in animal models, of course) have recently allowed the functional effect of genes implicated in
dyslexia to be visualized directly. This work is ongoing, but strongly suggests that the biological
basis of dyslexia lies in failures of neuronal migration (Gabel, Gibson, Gruen & LoTurco, 2010).
These knockdown mice have psychological disorders plausibly related (although distantly, of
course) to those found in dyslexia—for instance, reduced working memory in **DYX1C1** knock-
down models (Szalkowski et al., 2011), a trait also altered by polymorphisms in this gene in
human subjects (Bates et al., 2010). For education, perhaps the most important generalizable
conclusion flowing out of this research lies in the timing and complexity of the processes involved
in brain development. This in turn affects the kind of change (plasticity) that we will observe in
education.

The brain is ‘plastic’ in the psychological sense of supporting learning, and in the biological
sense underpinning this, with connection weights and even connections being almost continu-
ously remodelled throughout the brain and underlying learning. However, the term ‘brain plastic-
ity’ is often wielded like a light-sabre in education—implying that anything is possible (Bates,
2008a). It is near certain that education cannot replace a temperature-unstable version of the
catechol-O-methyltransferase molecule with a more stable version, or a **KIBRA** allele enabling
better retention, anymore than we should expect Lamarckian processes to know to look on chro-
mosome 22 for the **COMT** gene and within this for the guanine/adenine substitution that under-
lies a change in thermo-stability of the catechol-O-methyltransferase enzyme important in the
functional availability of dopamine and other catecholamines. The effect of genetic differences
such as these on educational outcomes is a matter of empirical study. It is logically possible that
selective application of an educational remedy might obviate all effects of some genetic differences,
but this must be empirically shown. It is also plausible that differences in hippocampal function
coded by KIBRA and frontal functions affected by genes such as COMT affect the relative capability
of children in ways that education cannot remove but must simply build upon.

Genetic differences are not restricted to neurotransmitter or membrane plasticity effects, but
also underlie the differentiation and migration of neurons into the developing brain-space,
unfolding a complex, ordered, pattern of compartmentalized connectivity. As the individual is
developing their psychological characteristics, these genes along with thousands of others then
play repeated roles in learning. An example of the effects of knocking down the current best
candidate genes for dyslexia is shown in Figure 11.1. This figure shows (in the leftmost pane) a
normal cortex with its typical complex multilayered structure, laid down in utero. In each of the
panels to the right, the effect of knocking down a dyslexia-linked gene is shown. In each case,
hundreds of millions of neuronal cell bodies fail to migrate out of the ventricular zone to
construct the complex vertically organized laminar cortex (Meng et al., 2005).

This complex matrix of neuronal migration, much of it occurring during gestation, has implica-
tions for expectations about cognitive plasticity in response to interventions. Beyond the remod-
elling of connectivity among existing cells and systems, the evidence for new functional neurons
in the neocortex, as it were ‘on demand’ throughout life, is almost completely negative. Studies of
neuronal plasticity have highlighted the general lack of neuronal cell replication in the brain out-
side a single layer of a small region of the hippocampus and (possibly) the olfactory bulb where
neurons are exposed to the environment (Bartlett et al., 1998). Production of new neocortical
cells, while technically feasible, has not to my knowledge been observed in humans. Bhardwaj
et al. (2006) addressed this question in a highly innovative and insightful way, utilizing variation
in atmospheric radiation following nuclear testing to ‘carbon date’ the timing of neuron formation
based on isotope levels in cells. Postmortem studies conducted by Bhardwaj et al. indicated clearly
that all neocortical neuronal cells were tagged with the radiation level present during gestation:
compelling evidence against the routine generation of new neocortical neurons post-birth in
humans. While behaviour can change, learning is unlikely to be able to roll-back development and
re-engineer the billions of cells and their complex migrations and connections involved in neo-
cortex, compensating altered DNA code using experience. We might hope to develop the technol-
ology to grow new functional cortical neurons, and thus remediate the effects of structural alterations
by remodelling neuronal structure. This would have to activate developmental migration proc-
esses normally terminated years before training in reading begins. These reactivated processes

\[\text{Fig. 11.1} \] Normally-occurring neocortical neuronal migration (‘Control’) and the effects of RNA
interference (RNAi) gene-knockout for three candidate dyslexia genes: KIAA0319 and DCDC2 on
chromosome 6, and DYX1C1 on chromosome 15. Reproduced from Cerebral Cortex, 17, Disruption
of neuronal migration by RNAi of Dyx1c1 results in neocortical and hippocampal malformations,
Glen D. Rosen, Jilin Bai, Yu Wang, Christopher G. Fiondella, Steven W. Threlkeld, Joseph J. LoTurco,
and Albert M. Galaburda, Copyright (2007) with permission from Oxford University Press.
would then have to operate within a large already-developed brain; organizing cell differentiation, cell body, axonal and dendritic migration, in an environment quite distinct from that of the relatively ‘empty’ (albeit scaffolded) space in which normal fetal migration occurs.

**Tabula rasa: genes, modules, mechanisms and innateness**

A final education-related outcome of behaviour and molecular genetic research (along with neuropsychological patients, and normal and abnormal developmental psychology) is the light it casts on the centuries-old debate about the organization of the mind caricatured as the ‘tabula rasa’ versus ideas of innate representation associated with Locke (1690/2009) and Kant (1788/1996) respectively. Research indicates compellingly that we are not blank slates, but rather are collections of mechanisms.

The research articulated earlier on the complex mechanistic systems of gene effects unfolding in the womb and beyond, along with the observation of large genetic differences in this developmental programme and limited evidence for neuron-level neocortical plasticity after birth is of relevance for the tabula rasa view of the child’s mind. Biological research suggests that education builds on an underlying biology determining the nature and complexity of primitive concepts in the young learner’s mind. Consilience between different fields is often an indication of an important insight into nature’s organization. It is valuable then to see that this view from behaviour and molecular genetics as specifying the neuronal architecture of the brain is compatible with a burgeoning body of work in developmental psychology supporting the view of the mind as composed of a number of primitives (including numerosity) out of which more complex concepts are assembled (Bloom, 2004; Carey, 1992, 2000, 2004, 2009).

Our own work in dyslexia is framed within a modular view of mind in which distinct mechanisms not only receive different inputs, but also run different types of algorithm on those inputs (Coltheart, Rastle, Perry, Langdon & Ziegler, 2001; Pinker, 1991). The finding that different subtypes of dyslexia appear to have different genetic bases is compatible with the view that the box-and-arrow diagrams of neuropsychology (see Appendix 1) are reflected in genes programming for development (Bates, Castles et al., 2007). A range of other models, however, continue to be supported as compatible to varying degrees with adult cognitive data. Some implement just one kind of computation (e.g. a neural network) and place a deeper burden on the stimuli to which people are exposed (e.g. written language) as the basis for the emergence of differential sensitivity to different features in different brain regions (e.g. regions of cortex specialized for letter-groups, words and meanings: Harn & Seidenberg, 2001). Variations on this model suggest that the brain ends up being modular, but that this emerges from a uniform substrate with only weak constraints on innate capability present (Karmiloff-Smith, Scerif & Ansari, 2003; Scerif & Karmiloff-Smith, 2005). Thus while most mainstream experts agree that the brain ends up being specialized, there are divergent views on the role of the environment in development. This in turn raises the ‘nature nurture’ debate: a discussion that has been central to discussions of mind and education.

The preceding sections suggest that there are significant differences between children in their ability to reason and learn and that specialized skills and talents such as language, spatial skills and memory are also scripted in our genome in important ways. It may be, however, that these apparent differences do not reflect main effects of genes raising or lowering the capacity to learn or reason, but rather that they generate a set of trade-offs such that all children have similar net capacities, but which must be accessed in different ways. A similar though less well-specified position is that genes act only in interaction with the environment and that psychological development is so complex and interactive that main effects are unlikely to cause differences between children.
As Pinker (2002) has noted, this 'no-debate; it's all a transaction' position meets little resistance as it appears sophisticated and reasonable. In particular it appears to offer hope for change: perhaps learning outcomes are merely complex, and better diet, smaller classes, brain gym, or attention to learning styles could make differences in outcomes go away. Research highlighted in several chapters of the present book suggests, however, that this is not the case to more than a limited degree. Despite the apparent sophistication of the 'there is no-debate' position on nature and nurture, 'Nature and nurture won't go away' for very good reasons (Pinker, 2004). Understanding nature is critical in understanding what is common to human beings and for understanding the implications of children differing in nature as well as in nurture.

**Nature, nurture and differences between children in an effective school**

The kinds of relationship of genes to environment are diverse, and often shaped by natural selection. As might be expected for important traits, evolution and selection has often tightly controlled many aspects of development: what Waddington called highly 'canalized' traits. This term refers to traits where redundant genetics systems are designed to robustly achieve a particular phenotype in almost any obtaining environment. That is why all humans have blood that carries oxygen, two more-or-less functioning eyes, ears etc. and a very precise set of many dozens of cognitive (cortical) maps and brain sub-systems to parse these inputs in robustly human manners: working memory, phonological loops, visual motion areas, social values and even mechanisms for evaluating group membership and other core evolved needs (Tooby, Cosmides & Barrett, 2003). The innate equipment of the brain is eager for certain very particular kinds of environmental input, but individuals differ in what they cognitively can do with a given input, and also, non-cognitively, in which input they prefer (Heckman, 2007) and evoke in their environment (Haworth et al., 2011). From these effects flow the large (often very large) main effects of genes.

Genes also interact with each other, and, because of these 'G×G' interactions, effects of even small changes can be extremely wide-ranging and powerful; the gene MECP2, for example, regulates over 1000 other genes and has widespread effects on brain function, cognition and disease (Joyner et al., 2009). Indeed while the great majority of DNA is not expressed as protein, significant sections of this non-expressed DNA underlie the regulation of gene expression itself, forming a complex operating system for the body (Qureshi, Mattick & Mehler, 2010).

Finally, genes interact with the environment: G×E. There are again diverse forms of G×E effect, including covariation, evocative and mechanistic interactions (Plomin, 1994). Interactions with the environment are of particular relevance to education, as schooling involves providing environments to children without which they would not develop the capabilities they acquire in interaction with education. In this case gene alleles are usually expected to multiply the effect of environments, reflecting the observed differences in children in their rates of learning (steeper or shallower slopes). Thus alleles affecting rate of learning will shift a child with a higher learning rate even further forward relative to her peers when the learning environment is optimized. Therefore while it might be expected that improvements in education would ameliorate prior environmental insufficiencies, leading to reduced differences between children, better teaching may act to magnify differences between children.

In their paper 'The rhetoric and reality of gap closing' Ceci and Papierno (2005) point to a large body of evidence showing that 'when the “have-nots” gain the “haves” gain even more'. This situation is illustrated graphically in Figure 11.2. Strong empirical evidence for this multiplier model comes from the decades-long programme of research into the lifespan developmental of talent of Baltes group, in his 'testing-the-limits' paradigm. Baltes concluded that intensive and organized
training in memory benefited all subjects, and lead to exceptionally high levels of performance. However, after intensive training, subjects’ maximum performance asymptoted. Moreover existing differences were not washed out with practice, but rather the exact opposite result was obtained: original differences were magnified by massed practice (Baltes & Kliegl, 1992). Similar to Baltes, Detterman and Ruthsatz (1999) suggest that exceptional achievement is the product of

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**Fig. 11.2** Rhetoric and reality of gap closing: four ways in which education might impact differences between students. Note: the top-left panel depicts a normal distribution of ability prior to education beginning. The three additional plots reflect three possible effects of education on student capability. The top-right panel shows the expectation if children do not differ in their ability to gain from education: Education will reduce variance, with the initially less-capable students being remediated, falling closer to the more capable student's performance. The lower-left panel represents a simple shift, in which education preserves rank-order differences and variance, raising the average scores of all students equally. The final lower-right hand shows the empirically observed effect described by Ceci & Papierno (2005) regarding the ‘rhetoric and reality of gap closing’: all students gain capability, but the initially most capable students gain even more, increasing variance.
(at least) intelligence, domain specific talent or skill and practice. This formula has been supported in case studies of musical expertise (Ruthsatz, Detterman, Griscom & Cirullo, 2008) musical prodigy (Ruthsatz & Detterman, 2003), and even domain-specific skill in savants (Ruthsatz et al., 2008). These empirical results contrast sharply with the widely cited model of talent proposed by Howe, Davidson and Sloboda (1998). Howe et al. argued for a version of the blank slate model, denying the existence of talent as anything other than early environmental choices, exposures and opportunities. In this model all variance in talented performance is allocated to practice. Detterman, Gabriel & Ruthsatz (1998) call this claim ‘absurd environmentalism’ for ignoring any role of individual differences. The results of Detterman, and, especially compelling due to their magnitude and duration, of Baltes’s group suggest that this model is wrong and that the observation of Ceci and Papierno (2005) is the norm.

A second form of interaction that has been proposed to explain differences in children are so-called ‘cross-over’ gene–environment interactions. This kind of interaction maps neatly onto the learning-styles literature (see Chapter 13, this volume), and the related suggestion that all children could learn equally well, but that they will respond to different environments. In order to sustain the notion that all children performing equally well when given the learning environment that suits their style, these models need to propose that environments which are good for some children are bad for others (otherwise some children would do better than others across the board, reducing the model to the ‘general ability + specific talents’ compelling model (Spearman, 1927; Vernon, 1950)). While intuitively appealing, placed into a concrete context this model leads to paradoxical expectations. For instance, to take the example of reading, the cross-over G×E model would predict that on beginning school lessons based on phonics (or whole-language models), some children would learn to read, while others (with different alleles) would not only fail to learn as rapidly or even fail to learn at all, but may unlearn what reading skills they had with each exposure to the (for them) toxic school-based reading method. In practice, except in cases of disease or injury, reading improves with exposure to teaching for all children. Study of these models in psychiatry suggests that this particular form of interaction of exactly counterbalanced benefits and costs in different environments are highly unlikely to be detectable, other than as false-positives, a position supported both empirically (Munafo & Flint, 2009; Risch et al., 2009) and theoretically (Maes et al., 2006).2

Strong multiplicative G×E effects with weak (if any) cross-over interactions mean that good schools will increase rather than decrease differences between children; by building on and multiplying psychological capabilities, school will act to exaggerate even small underlying differences in biological capacity. A critical consequence of differences between children is that while we can set standards for performance, and can raise the performance of all children within practical limits, we cannot hope to meet targets defined in terms such that no child can be left behind any other. This suggests that education policy ought, therefore, to focus on maximizing individual children’s capabilities rather than reducing differences between children. Of course this is not a carte blanche for ignoring differences. One statistic, for example, which does suggest a current failing in education is the finding that children equated for intelligence quotient (IQ) at age 11 do better at age 17 according to their social class. This is prima facie evidence that schools taking able children from lower social class backgrounds are failing to provide them with the opportunities to

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2 A lack of interaction effects is also relevant for claims about selective dietary requirements and brain development also claimed to be relevant for differences in educational outcomes, e.g. the effects of breastfeeding on intelligence e (Caspi et al., 2007) where replications now have found either opposite (Steer, Davey Smith, Emmett, Hibbelsn & Golding, 2010), or null effects (Martin et al., 2010).
11.3 A three-level cognitive systems framework: educating for capability

Understanding that already in the brain, billions of genetic decisions have led to the migration and differentiation of stem cells into precisely located cell bodies with their myriad connections is a perspective-changing event. Education is not working with a blank slate, nor with a magic space of infinite capacity, but with a delicate organ comprised of numerous specialized, limited capacity mechanisms. A brain in which some skills are innate: not just perception with its dozens of cortical maps in various tonotopic, retinotopic and other representational frames, but also behaviours as diverse as number skill and social relationships (Carey, 2009). Despite these specialized mechanisms, many tasks critical for success in society are effortful, require practice, and are subject to forgetting. This highlights limitations on our cognitive skill, but also focuses our attention on the role of education in achieving effortful, difficult learning. In this section I wish to bring together earlier material into a framework capable of representing biological capacities and differences, psychological content and processes, and the desired objective biographical goals of education, such as productive employment, and better health and social relationships. The section first introduces such a model. Using this framework, the mechanism of education in driving these goals is discussed focusing on three factors: what kinds of teaching lead to effective learning, which methods can effectively deliver this teaching, and what the outcomes of effective education will be in terms of between student differences.

If models that treat biology as uniform, or subsume it in a transaction where biology places no limits on performance, are unhelpful in understanding the mind, what framework can contain biological elements (such as G-coupled proteins and axonal guidance mechanisms), psychological mechanisms (such as memory and reasoning), as well as content (such as knowledge of procedures, memory for facts, or storage of phonemes in a buffer), and also valued outcomes such as being offered a job, keeping a stable relationship, or starting a successful company which motivate our interest in education? Here I suggest a framework in which these three factors are viewed as three distinct layers in a bio-psycho-social framework. This model is presented in Figure 11.3.

This bio-psycho-social model proposes that individual differences in behaviour span three domains: (1) biological capacities—which have no psychological properties; (2) psychological adaptations—psychological constructs such as values, memory, vocabulary, reasoning skills, which are formed under the joint influences of basic tendencies and external factors such as life-events and cultural norms; and (3) objective biography—observable outcomes explained by the interplay of external stimuli (also observable) with psychological adaptations. This model specifies the proximal cause of objective biography as the psychological layer, with influences from basic tendencies being fully mediated by psychological adaptations—with no direct links to behaviour. Education is explicitly represented as an external stimulus interacting with psychological adaptations (the decision to form and fund education is, of course, part of the objective biography of citizens and politicians).

In this model, we no longer expect social environment and genes to form an inseparable or inscrutable nexus, but rather to function as distinct layers of a bio-psycho-social system, with cognition in the middle layer. Cognition is dependent on underlying biological capacities, but it is this psychological layer with which education interacts and which generates observed behaviours. In this model, teachers and schools are elements of the external environment, interacting with the current mental state to generate new mental capacities. Elements of the left hand pane are tightly linked to genetic code. Measured in this way, they will often (but not always) have close to 1.0 heritabilities—as has been observed for components of reading (Olson, Forsberg & Wise, 1994).
A key benefit of this three-layer model is the affordance of a conceptual framework for separating basic genetic and biological factors from the proximal causes of behaviour in the form of psychological content and skills. This has the potential to defuse misleading debates about ‘nature’ and ‘nurture’ which often tend to lose both in a ‘complex transaction’, suggesting that development is so exceptionally complicated and intrinsically interactive that nothing true can be said about how nature (genes and their programmes) unfolds (Pinker, 2002). It is of course important that scientists retain a respect for the complexity of their subject, moderating extreme claims so as not to suppress new approaches unnecessarily and not to simplify that which is abstract and complex. This can lead, however, to a failure to progress scientifically. There is a very good reason that biologists do not think it sophisticated to say when comparing a tall and short variant of plant differing in a gene allele that it would be necessarily simplistic to account for changed height in terms of the gene due to this invoking nature. Instead they would examine and report the mechanism by which the allele’s expression leads to growth. They would trace out the pathways, promoters and regulators of the allele searching for additional genes affecting plant height. This is, of course, exactly what occurs in human medicine to great benefit, and is occurring for the trait of height in humans (Lango Allen et al., 2010; Yang et al., 2010). If we are to understand variation in normal development, as we do in disease it is important that we avoid reflexively retreating to concepts such as pervasive interaction at the expense of concentrating on the mechanisms of nature.

This perspective suggests answers to several important questions about the role of education in helping children absorb the content and the tools of culture. To show the utility of the framework, I focus briefly on two: what teaching methods work? And what impact do differences between teachers have in education?

![Diagram of bio-psycho-social model for education](image-url)
How does school raise capabilities?

Parents, psychologists and even moral philosophers widely agree on things they would like their children and fellow citizens to have, for instance, the ability to gain and keep well-paid work, to have fulfilling social relations, to obey reasonable laws (Sen, 2009), to cope with challenges and master new skills (Ryff & Keyes, 1995), and to share a common cultural vocabulary allowing efficient communication and cooperation (Hirsch, 1999). Views on how school might achieve this vary dramatically. On the one hand school is viewed as a place where children construct their own reality with as little structure of guidance being imposed from teachers as possible. A key role for teachers in this model is simply to encourage the child whatever they are doing. Sternberg (2000) in his article ‘In search of the zipperump-a-zoo’ suggests, perhaps somewhat tongue in cheek, a model of school performance in which pupils (using himself as an example) simply produce what is expected of them based on largely capricious expectations. Rather opposed to this view, is the idea that school is a place for explicit teaching of widely agreed facts about the world, theories of why these facts are so, and skills in how to do things, from painting to playing an instrument, to writing, and designing and constructing objects. This latter approach favours explicit instruction over personal construction. Rather than ask children to construct reality, it seeks to expand their capabilities by direct instruction: ‘When you greet someone, shake their hand. Here is how: look the other person in the eye, look at their hand: grasp it firmly but not harshly, and, again, make eye contact’. Rather than presenting people and events value and date free and leaving children to extract what they can from these, events are used as examples of concepts and their development over time: ‘At Trafalgar in 1805, Britain was at war with Napoleon’s France and faced the combined fleets of France and Spain. Lord Nelson showed exemplary valour…’. It favours also using practical tests to ingrain knowledge: for instance, using an abstract concept in practical situations with repeated practice.

Somewhat aligned with this dimension running from modern structured learning to post-modern constructivism, are questions of whether school should teach knowledge, or raise basic abilities such as reasoning and memory, and in a related theme, whether school should focus on creativity rather than knowledge and skill acquisition. Experiments that can be interpreted as raising basic abilities such as intelligence are likely to be widely reported (Jaeggi, Buschkuehl, Jonides & Perrig, 2008; Rauscher, Shaw & Ky, 1993) and even sold commercially, but we are less likely to read about significant caveats (Sternberg, 2008), or critiques (Moody, 2009) and failures of replication (Chabris, 1999; Steele, Bass & Crook, 1999; Stough, Kerkin, Bates & Mangan, 1994).

The bio-psycho-social model is of value here in distinguishing between two very distinct meanings attributable to words such as ‘memory’ which have both biological and psychological interpretations. To the extent that ‘memory’ is understood as a basic biological mechanism, we cannot expect education to give a person a better memory any more than we could expect education to rewrite a person’s DNA to give the more effective version of the KIBRA gene. If ‘memory’ is, however, considered psychologically as the ability to store and recall knowledge, then memory can plainly be improved through practice and testing. The job of effective education then, becomes not equalizing children through neuronal plasticity or other mechanisms, but enhancing psychological capability by creating normative expectations and teaching situations and activities that increase desired objective biographical outcomes.

A similar approach can be taken in understanding the choice to teach creativity or understanding of existing knowledge. ‘To give a recent example, Sternberg has argued for the former, stating that we should be ‘teaching creativity not memorization’ (Sternberg, 2010). This option (if choice is required) perhaps sounds attractive: after all who would not rather have the founder of Nokia, the inventor of a high capacity battery, or the creators at Apple as fellow citizens than someone who
memorized the periodic table but did nothing new? However the statement masks several assumptions. The first is that creativity can be taught and that it is independent of intelligence and openness. Both these propositions have been powerfully critiqued (Brody, 2003a, 2003b). A second lies in the use of the term ‘memorisation’. Why not ‘learning’, or ‘understanding’? A suggestion that ‘school should teach creativity instead of understanding’ perhaps does not sound as compelling. In the bio-psycho-social model, effective education is about increasing capability by doing what works. The question then becomes empirical: what works to maximize children’s lifespan capability? This places a large focus for research and practice on understanding and implementing effective teaching that transfers to improvements in average measured outcomes. Paradoxically then, neuroscience tell us that education is about teaching.

The bio-psycho-social model suggests that a core difference between an educated and an uneducated mind consists in acquiring a large store of knowledge and skills. If this is correct, then a key question is to resolve the debate as to whether skills and knowledge require systematic practice and work in committing them to memory, or if learning should be similar to play, in that it is discovery-based and self-directed. In this latter view, rather than teachers teaching the facts that others agree on and giving practice with this knowledge, children should construct reality for themselves (Mayer, 2004). Likewise, testing is seen as having little or no role in learning, serving no positive purpose and acting to divide children, either along class lines reflecting coaching at home, or by giving damaging and self-fulfilling feedback to those unlucky enough to score badly on the test day. Any apparent learning as a result of study for tests is seen as fragile and quickly forgotten due to its rote nature.

While widely supported amongst educationists (Mayer, 2004), this view is quite opposite to traditional models of learning. To quote one of the oldest educational theorists ‘Exercise in repeatedly recalling a thing strengthens the memory’ (Aristotle, 350 BCE/2007). What do the data say (see Chapter 7, this volume)? Testing dramatically increases learning, especially over longer time periods (Roediger & Karpicke, 2006). Testing provides opportunities for retrieval, and retrieving memories appears to enhance them (Karpicke & Roediger, 2008). Speaking informally, the brain gets the message that retrieved memories are in use and valuable, and enhances their durability. We often hear, too, that feedback on poor performance must be damaging (via lowering self-esteem, it is suggested). Again, however, evidence suggests that feedback after testing further enhances learning over the testing effect (Butler, Karpicke & Roediger, 2008). Even the idea that rote learning only ‘teaches the test’ rather than building generalizable knowledge is false (McDaniel, Howard & Einstein, 2009). Why does structured learning and testing seem to work so well? Verbatim knowledge is often valuable in its own right—either in creating a social currency for compact communication (‘There is a tide in the affairs of men. . .’) or for precise recipes where quantities matter (seven eights are 56, not 57). This is true not only for learning one’s times-tables, but of all facts and axioms: these building blocks of knowledge cannot be derived, but rather form the basis of derived knowledge, and the basis for augmenting our ability to manipulate information. In the bio-psycho-social model, this augmentation takes place in the psychological layer and is what raises capability by multiplying the effectiveness of biological ability.

Rote as one end of a dimension of depth of knowledge

Rote learning where meaning was denied would be reprehensible. But learning the skills that multiply capability nearly always involves typically rote-learning and practice. Take an example from mathematics in the form of matrix algebra. Matrix algebra is ubiquitous in engineering, mathematics and statistics. Like the card game Bridge, it consists of abstract and arbitrary rules which meet a goal: in this case for making thinking about complex problems very much easier by...
abstracting away potentially enormous sets of arithmetic operations into single symbols. The pay
off is immense: \( A \cdot B^{-1} \) can express in five characters what might be several million underlying
mathematical operations on many thousands of numbers in large matrices. It is used in dozens of
fields from accounting and forecasting to statistical modelling the behaviour of complex systems
and materials in science and technology. Learning of matrix algebra involves some insight, but
developing a facility in doing matrix algebra requires dozens of hours of practice. While the goal
of matrix algebra is not arbitrary, the rules and symbolisms of matrix algebra are: indeed, as in
calculus, alternative formulations competed early on. Any person wanting to use this technology
must learn the symbolism and rules of transformation by rote so they become habitual.

The opposite of rote learning might be characterized by conceptual knowledge in which the
student can recognize a concept in various forms and media, manipulate it comfortably, and relate
it to other knowledge. We can, however, often be surprised by how shallow our own knowledge is.
We see this in children who can draw a square but not a diamond, though neither has been taught
by rote. In what sense does the child understand the concept ‘square’ when they cannot also draw
a diamond? Or the concept diamond, when they cannot draw one? As adults we may feel beyond
this stage, but it appears we are not. Imagine if you will a simple object: say a cube. Most readers
will think they have deep knowledge of a cube: You could probably define it (‘a symmetrical
three-dimensional shape, either solid or hollow, contained by six equal squares’); recognize it in a
sentence, and visually, recognize it translated in different orientations, distances or materials. You
can relate its properties in mathematical terms of angle (90°), orientation of sides (orthogonal),
equality (of edge lengths). You can also compute characteristics such as area or volume. Imagine
a cube in front of you. How many sides does it have? A moment’s inspection says six. Now, is our
knowledge of this simplest three-dimensional figure ‘rote’ or deep? Refraining from verbal or
mathematical solutions, imagine placing a finger on one corner, rocking the cube over and pick-
ing the cube up by placing a second finger on the diametrically opposite corner. Can you see how
the cube can spin on the axis you have created? Now: how many corners are there around the
middle of the cube? Few people answer this question correctly without turning to verbal knowl-
edge. Clearly a continuum of representation exists, with much of it shallower than we think, and
elaborated mostly by extensive use and testing.

Neuroscience in the classroom

In the bio-psycho-social model, school multiplies capability by providing new knowledge and
methods of manipulating this knowledge. Differences in educational technique and their effects
appear in this model as transactions between the child’s layer two, and teachers and teaching sys-
tems in layer three (see Figure 11.3). Variables in this transaction include teacher differences, class
sizes and other factors. The model focuses attention on the optimal trade-offs of factors such as
teacher quality and class size (given the costs of each). Other variables include setting of children
by ability and non-cognitive factors such as classroom discipline. Each of these factors can be
examined empirically. Here I mention briefly two recurrent issues: class size and teacher quality.

Class-size reductions have been argued to be important (Krueger, 1999) and having large effects
on learning. This is important, because in a system in which salary and pension costs dominate
expenditure, class size is the single most expensive variable to change in education. It therefore has
to have a very large effect to warrant the opportunity cost of decreasing class size. Effects of class
size, however, are typically found to be small, often approaching zero (Hanushek, 1998; Hoxby,
2000). Moreover, because reductions in class size lead to reductions in teacher pay over the
medium term, such interventions may further reduce teacher quality where funds are limited
(Jepsen & Rivkin, 2009).
This latter conclusion by Jepson and Rivkin (2009) focuses attention on the concept of teacher quality. Views on the impact of teacher ability and subject matter knowledge on educational outcomes vary. At one extreme, it is suggested to be largely irrelevant (Hanushek, 1998). Others conclude that teacher’s cognitive ability is an important factor in raising student capabilities. Some data are available. Teacher effects have been examined in large samples (Leigh, 2010). This study controlled for between-student differences, looking at the effect of teachers on the children they taught, controlling for the previous year results of those children. This was a large study with data on 10,000 teachers and over 90,000 pupils. There was evidence for a general ability to teach, with significant between-teacher effects on student literacy and on numeracy. Between-teacher differences also influence the likelihood of individual children maximizing the expression of genetic differences in ability, with good evidence for a strong specific role of teachers on effective teaching of reading (Taylor et al., 2010). Similar data suggest significant lifetime health and income benefits of teacher quality early as kindergarten (Chetty et al., 2011). The nature of teacher quality is, however, unclear at present and warrants more research.

The impact of reduced prestige and pay relative to other occupational choices in lowering the ability of entrants to teacher training is often highlighted. Objectively, teacher cognitive scores appear to have fallen. For instance, over the past decades the ratio of trainee teachers with average IQ (100) versus high ability (IQs of 130 or above) appears to have fallen from approximately equal numbers in both groups to current ratios of 4:1 (Murnane, 1992). Studies of teacher-trainees also indicate that some teachers lack a full understanding of the material that they will be required to teach as assessed by an ability to gain good passes in the tests that their pupils will sit, for instance submissions to the Donaldson report on education indicated that teachers often need multiple attempts to gain good passes in primary school mathematics tests, and say that they often learn the material they will teach one lesson ahead of the pupils. This has been attributed to a cultural norm in schools that ‘undermines the need to have a sound understanding of what is being taught’ (Donaldson, 2011). Links of teacher cognition and knowledge to student improvements in student capabilities may repay research investment.

11.4 Conclusion

Genetics research and research on the brain suggests that children bring to school very large and robust differences in their learning capacity, with large proportions of these differences reflecting heritable differences (Royal Society Working Group, 2011), but also that it is decisions about how we teach that will have the biggest impact on raising children’s capabilities. This highlights two implications of neuroscience research. The first is that because education works via biological systems that differ between children, differences between children in turn are magnified by effective education. The second is that improving education can be aided by reliable answers to practical questions about effective teaching techniques: Under what circumstances does learning best occur? How are skills like painting best taught? How is historical knowledge best taught? Do dates help or hinder children’s understanding and recall? Should classes be small? Can a mix of large instructional classes and small tutorials and practice sessions deliver better educational outcomes? Does teacher ability and knowledge matter? Should children be ‘set’ in classes with others of like-ability? Solid, well-referenced and readily accessible answers to these questions are basic to optimizing the capabilities of children, and yet surprisingly hard to find. Debates in the news are often fuelled by apparent disagreement over core facts: are exams easier today than in the past? Work by the Royal Society of Chemistry (Royal Society of Chemistry, 2008) appears to demonstrate compellingly that this is the case. But this information is often available only in a scientific report rather than in open, well-referenced, live and contestable summaries, easily accessible to journalists,
employers and parents alike. While there are numerous special committee reports and documents into education, these date rapidly and necessarily have a limited authorship. Often too, there simply do not seem to be robust reliable studies to address particular questions. It would be hoped, then, that the coming years will see a much tighter integration across academic departments of education, psychology and other disciplines basic to learning, as well as with policymakers, parents and community. My vote would be to invest modest sums in answering these questions and to educate the educational debate with facts about how education works.

11.5 Appendix 1: describing and explaining the mind

At least three branches of psychology have emerged to explain differences between individuals: neuropsychology, individual differences research and behaviour genetics. The three have distinct perspectives on the mind, but can be integrated under an umbrella of cognitive genetics, providing a framework for understanding the biological bases of learning relevant for education (Bates, 2008b). These three approaches to cognition are shown in Figure 11.4 (differential psychology model of intelligence); Figure 11.5 (neuropsychological model of specialized cognitive mechanisms and their connections); and Figure 11.6, showing a behaviour genetic model of reading and elements of language and working memory.

The three figures have quite different forms, reflecting the focus of researchers in the three domains. The results of all three of these measures are of course reconcilable, and highlight two facts of broad importance for education. The differential psychology approach is shown in Figure 11.4. This depicts the relationships amongst 13 cognitive ability measures, focusing on the covariance among these measures. Of relevance to education, this covariation approach revealed that a single general factor account for around 40–50% of the variance in any diverse and arbitrarily large battery of tests (Carroll, 1993).

Fig. 11.4 Differential psychology model of cognition: The factor structure of ability as measured in the Wechsler Adult Intelligence Scale III. General ability accounts for around half the variance in performance, with domains of ability also exhibiting some structure (for instance, verbal and perceptual factors) and substantial variance among the specific abilities. It is these specific abilities, or even more fractionated forms thereof, which appear in neuropsychological models, such as that of Figure 11.5.
This general or g factor is heritable (Deary et al., 2006) and highly stable across the lifespan (Deary, Whalley, Lemmon, Crawford & Starr, 2000). General ability measures are highly correlated with a range of life-course outcomes, and account for much of the variance in educational outcomes. For instance, in a study of some 70,000 children followed from age 11 to their GCSE exams, IQ measures at age 11 accounted for 81% of school grade variance at age 16 (Deary, Strand, Smith & Fernandes, 2007). This suggests that children differ not merely on what they know at a given moment, but on their ability to learn. Thus even lengthy exposure to education tends to be highly rank-preserving. Figure 11.4 also highlights how hierarchical modelling within individual differences captures clusters of variance beyond g. Shown here are representing visuospatial and

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**Fig. 11.5** Reading, language and working memory in cognitive neuropsychology. On the right-hand side are shown components of the dual-route cascaded system for reading (Coltheart et al., 2001). Data from patients supports dissociations between the ability to read aloud pseudo-words such as ‘SLINT’ and the ability to read-aloud irregular words such as ‘YACHT’ (Castles, Bates, Luciano, Martin & Coltheart, 2005; Castles & Holmes, 1996). In the centre we see semantics and the executive components of working memory (Baddeley, 2007). These are represented as separate modules because dissociations again suggest that there are patients with acquired brain damage who retain the ability to read but no longer access meaning from written language, and other patients who retain meaning despite losing the ability to read. Finally on the left hand side of the model are components of mind implicated in specific language disorder, specifically the systems for speech sound analysis, for brief storage of phonological strings, independent of meaning, and connections supporting rehearsal in short-term memory. Adapted from Baddeley, A. D., Working memory: Multiple models, multiple mechanisms. In H. L. Roediger, Y. Dudai & S. M. Fitzpatrick (Eds.), *Science of Memory: Concepts*, 2007, with permission from Oxford University Press.
verbal ability intermediate between the single tests themselves and g or general ability. These two facts about cognition: that it contains both a substantial general component and a substructure of specialized faculties emerged in the 1920s (Spearman, 1927) and have been widely agreed since the 1950s (Vernon, 1950; Neisser et al., 1996).

Figure 11.5 shows the second powerful organizing framework in psychology: that of cognitive neuropsychology (Rapp, 2002). By focusing on double dissociations in patients, cognitive neuropsychology has built models of cognition based on the cascaded flow of information through distinct processing systems revealed in cases of developmental and acquired cognitive deficit (Shallice, 1988). A critical piece of information from this model is the suggestion that the mind has a complex internal structure, with concepts and processes of the mind being basic building blocks of its function, rather than arbitrary arrangements imposed by external processes such as education.

The individual differences and neuropsychological models appear, on the face of it, incompatible: one contains a latent, unobserved g factor and de-emphasizes specialized mechanisms; the other contains explicit boxes and arrows and focuses almost entirely on mechanisms and information transfer, rather than correlated abilities (which are uninformative about the dissociability of components of ability Shallice, 1988). Elsewhere, I argued that this apparent discrepancy is, however, illusory: both sets of information can be represented in a common framework (Bates, 2008b). An example of such a common framework is that of behaviour genetics as described below.

The behaviour genetics model (Figure 11.6) closely maps onto the model from neuropsychology. Both models capture irreducible components of the mind. The difference is that for behaviour genetics, evidence for these modules is based on developmental genetic dissociations flowing from Mendel’s laws, rather than on acquired differences in patients. Research on the genetics of autism, for instance, recently demonstrated that autism fractionates 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., 2006a). It is interesting to note
that behaviour-genetic research (Bates, Castles et al., 2007) also replicates the dissociation of sur-
face and phonological dyslexia found in acquired dyslexia. This genetic linkage suggests that the
origin of the dissociable mechanisms found in the adult mind, and exposed by damage to the
adult brain, do not arise simply from environmental regularities promoting specialization
(Karmiloff-Smith, 1998), but that this specialization is itself programmed (Pinker, 2002).

As well as articulating the separately heritable mechanisms of the mind found in neuropsychology,
behaviour genetics also highlights an aspect of the mind that does not emerge naturally from
neuropsychological models (because of their focus on double dissociation): namely the general
and group ability components of cognition captured in individual differences research. General
ability or 'g' is not a mental mechanism per se, hence there is no 'g' box in neuropsychological
models (though g is more tightly linked to brain volumes and functional differences in parieto-
frontal and infero-temporal regions of the brain supporting attention, language, and working
memory than with other regions of the brain (Jung & Haier, 2007)). Nevertheless, g accounts for
around half the variance in cognitive ability and this is an important finding for education: it
implies that children performing poorly (or well) on one element of their schooling are likely to
be performing similarly poorly (or well) on other domains of their learning. Genetic studies
(reviewed in Deary, Spinath & Bates, 2006) indicate that not only is a large portion of stable cogni-
tive differences heritable, but that almost all of the correlations between different mental abilities
are accounted for by genetic influences on multiple cognitive traits. Our own research into the
genetics of reading and language supports this idea. The genetics of reading is mostly overlapping:
not only at the level of comprehension where general ability is predominant, but in the very spe-
cific subtasks of reading: building a sight vocabulary of whole words and building a facility for
decoding letters into sound. Genes then as often have very general effects (Kovas, Harlaar, Petrill
& Plomin, 2005) just as they also have very specific effects (Bates, Castles et al., 2007; Wilmer
et al., 2010).

11.6 Appendix 2: risks and future developments

Gene discovery creates the possibility of early diagnostic testing based on large numbers of aggre-
gated genetic risk factors. Because genes associated with reading are present at birth, genetic
knowledge allows an assessment of specific risk years before signs of reading delay could ever be
detected by conceivable behavioural testing. This affords both much more effective allocation of
this expensive resource to at-risk individuals, much earlier intervention and, possibly, interven-
tions tailored to particular risk factors. As the discovery of specific genes for cognitive develop-
ment continues, affordable effective prognostic testing is now emerging. As a result of genome-wide
association studies (GWAS) assessing around one million single nucleotide polymorphisms
(SNPs) in each individual for around £200, we can also soon expect dramatic improvements in
early (pre-symptomatic) diagnosis of risk based on prediction functions. For many complex dis-
orders, thousands of genes seem likely to each play very small, but cumulative roles. Prediction
functions aggregate thousands of tiny effects distributed across the genome and impacting
on a complex disorder like schizophrenia or autism. In the case of height, this has been effective,
suggesting that the present methods of genomic testing capture around 40% of the genetic basis of
height (Lango Allen et al., 2010; Yang et al., 2010). These levels of prediction afford great opportu-
nity. When genetic tests become available for disorders such as dyslexia and autism, it seems likely
that their effects could be beneficial. Genetic tests can assess risk well before any behavioural 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. While this seems remote given our lack of understanding of complex disorders at a neurological level, the dramatic response of apparently inevitable disorders such as phenylketonuria (PKU) and insulin-dependent diabetes to very simple biochemical interventions, suggests that substantial improvements, or even elimination may be possible when the triggers of gene expression in pathways to dyslexia, autism, attention deficit hyperactivity disorder and even general intelligence (as in the case of PKU) are understood. Secondly, and as seen in several other disorders already, understanding the genetic basis of differences in educational aptitude is likely to transform these into facts that we can acknowledge and address, rather than unexplained outcomes for which teachers are blamed as ‘leaving children behind.’ This new knowledge seems likely to promote not only acceptance and additional resources for tailored programmes (as is beginning to occur for autism and dyslexia) but also earlier and more effective intervention.

While early genetic testing offers the hope that we might put in place ameliorative cognitive interventions well before any negative social consequences of falling behind in school can ever emerge, this power, of course, enables various forms of GATTACA-like society. On the hopeful side, many important changes in education were accompanied by research results from individual differences and genetics: from socially inclusive education, where Binet and others provided evidence for ability in disadvantaged groups, to equal provision for education for both sexes, which was preceded by research from Burt and Moore (1912) who concluded in their paper in the *Journal of Experimental Pedagogy* that ‘with few exceptions innate sex differences in mental constitution are astonishingly small – far smaller than common belief and common practice would lead us to expect’ (Burt & Moore, 1912), a result supported by more sophisticated contemporary studies also showing almost equal average ability across the sexes (Deary, Irving, Der & Bates, 2007). More, not less opportunity and resources for the less able, has likewise accompanied research on the biology of differences. Thus compelling evidence for high heritability of dyslexia did not result in abandonment or ostracism of children at risk for reading disability. Instead research on behavioural therapies and teaching methods continued to occur, with the goal of enabling 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 genetic components (Ronald et al., 2006a), and therapeutic innovations, as well as a public acceptance of the disorder, and a dramatic increase in therapeutic resources consequent on a spectrum diagnosis.

References


AQ: Is the Chetty et al. 2011 paper in press or has it been published? Please supply details if so


REFERENCES


