Neuroscience, education and special education

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The discipline of neuroscience draws from the fields of neurology, psychology, physiology and biology, but is best understood in the wider world as ‘brain science’. Of particular interest for education is the development of techniques for ‘imaging’ the brain as it performs different cognitive functions. Cognitive neuroimaging has already led to advances in understanding some of the basic functions involved in learning and raised implications for education and special education in particular. For example, neuroimaging has enabled scientists to study the very complex processes underpinning speech and language, thinking and reasoning, reading and mathematics. In this article, Professor Usha Goswami of the University of Cambridge Faculty of Education first reviews basic information on brain development. She provides a brief introduction to the tools used in neuroimaging then considers recent findings from neuroscience that seem relevant to educational questions. Professor Goswami uses this review to suggest particular ways in which neuroscience research could inform special education. In its closing sections, this article provides authoritative perspectives on some of the ‘neuromyths’ that seem to have taken root in the popular imagination and argues for increased dialogue, in the future, between the disciplines of neuroscience and education.

Key words: neuroscience, brain, dyslexia, dyscalculia, learning disabilities.

Introduction

Both educationists and neuroscientists are interested in learning and how to optimise learning. Neuroscientists investigate the processes by which the brain learns and remembers, from the molecular and cellular levels right through to brain systems (for example, the system of neural areas and pathways underpinning our ability to estimate numerosities, calculate exact quantities and solve (or not) differential equations). Neuroscientists study learning at a variety of levels. Understanding cell signalling and synaptic mechanisms (one brain cell connects to another via a synapse) is important for understanding learning, but so is examination of the functions of specific brain structures such as the cerebellum. Brain cells (or neurons) transmit information via electrical signals, which pass from cell to cell via the synapses, triggering the release of neurotransmitters (chemical messengers). There are around 100 billion neurons in the brain, each with massive connections to other neurons. Patterns of neural activity are thought to correspond to particular mental states or mental representations. Learning essentially comprises changes in connectivity: the release of neurotransmitters at the synapse can be altered, or connections between neurons can be strengthened or pruned. Successful teaching directly affects brain function by changing connectivity.

Clearly, successful learning is also dependent on the curriculum and the teacher; the context provided by the classroom and the family; and the context of the school and the wider community. All of these factors also interact with the characteristics of individual brains. For example, children with high levels of the MAOA gene (monoamine oxidase A) who experience maltreatment and adverse family environments seem to be protected from developing antisocial behaviours (Caspi, McClay, Moffitt, Mill, Martin, Craig, Taylor & Poulton, 2002). This protection may occur via moderation of their neural response to stress. It is also possible to study the effects of various medications on cognitive function. Methylphenidate (Ritalin), a medication frequently prescribed for children with ADHD (Attention Deficit Hyperactivity Disorder), has been shown to improve stimulus recognition in medicated children in terms of improving their attention to auditory and visual stimuli (as revealed by neuroimaging, see Seifert, Scheuerpflug, Zillessen, Fallgater & Wänke, 2003). Neuroimaging techniques thus offer the potential to study the effects of different medications, food additives and potential toxins on educational performance.

Cognitive neuroscience also offers techniques for studying the effect of teaching on the brain. There are already studies exploring the effects of different instructional programmes in literacy on brain function and eventually neuroscience may be able to offer methods for the early identification of special educational needs. Neuroscience is already able to assess the delivery of education for special needs in certain specialised areas such as dyslexia. At the same time, however, it is worth noting that ‘neuromyths’ abound. Some popular beliefs about what brain science can actually deliver to education are quite unrealistic. Although current brain science technologies offer exciting opportunities to educationists, they complement rather than replace traditional methods of educational enquiry.

A short introduction to brain development

Many critical aspects of brain development are complete prior to birth (see Johnson, 1997, for an overview). The
development of the brain begins during the first weeks of gestation, with the birth of the cells that compose the brain. These cells migrate to the different regions in the foetal brain, the regions where they will be employed in the mature brain, prior to birth. By seven months gestation, almost all of the neurons that will comprise the mature brain have been formed. We already know that there are very specific effects of maternal drug addiction on brain development. For example, babies with foetal alcohol syndrome are born with underdeveloped parietal lobes. As the parietal lobe is critical for numeracy, these babies develop into children with specific problems in number processing and mathematical cognition (Kopera-Frye, Dehaene & Streissguth, 1996). Despite the plasticity of the developing brain, some aspects of brain development are already complete in the womb and therefore less amenable to the effects of later environment.

Brain development following birth consists almost exclusively of the growth of fibre connections and synapses between neurons: this process is called ‘synaptogenesis’. For vision and hearing (visual and auditory cortex), there is extensive early synaptogenesis. The density of connections peaks at around 150% of adult levels between four and 12 months, and the connections are then extensively pruned. Synaptic density returns to adult levels between two and four years in the visual cortex. For other areas such as prefrontal cortex (thought to underpin planning and reasoning), density increases more slowly and peaks after the first year. Reduction to adult levels of density takes at least another 10–20 years, hence there is significant brain development in the frontal areas even in adolescence. Brain metabolism (glucose uptake, which is an approximate index of synaptic functioning) is also above adult levels in the early years. Glucose uptake peaks at about 150% of adult levels somewhere around four to five years. By the age of around ten years, brain metabolism has reduced to adult levels for most cortical regions. The general pattern is clear. Brain development consists of bursts of synaptogenesis, peaks of density, and then synapse rearrangement and stabilisation. This occurs at different times and different rates for different brain regions. This means that there are different sensitive periods for the development of different types of knowledge.

In fact, brain volume quadruples between birth and adulthood. This is because of the proliferation of connections. As the brain is highly plastic, significant new connections form all the time, even in adulthood, in response to new learning or to environmental events (such as a stroke or motorbike crash). Similarly, sensitive periods are not all-or-none. Windows of enhanced sensitivity do not ‘close’; rather the ability of the brain to benefit from specialised input changes. For example, if early visual input is lacking, the critical period for setting up a visual system is not ‘missed’ (Fagiolini & Hensch, 2000). However, the effects of early deprivation will vary depending on visual function. Functions that develop late (for example, depth perception) suffer more from early deprivation than functions that are relatively mature at birth (such as colour perception, Maurer, Lewis & Brent, 1989). This means that some abilities have a lower likelihood of achieving full potential than others when the sensitive period is missed. However, particularly for more cognitive abilities, I would argue that focused intervention always has an effect on development.

Figure 1: The major subdivision of the cerebral cortex. The different lobes are specialised for different tasks. The frontal lobe is used for planning and reasoning and controls our ability to use speech and how we react to situations emotionally. The temporal lobe is mainly concerned with memory, audition, language and object recognition. The parietal lobe controls our sense of touch and is used for spatial processing and perception. The occipital lobe is specialised for vision. Structures such as the hippocampus and the amygdala are internal to the brain, situated beneath the cerebral cortex in the midbrain.

It is also important to realise that there are large individual differences between brains. There is striking variation in the size of different brain structures and in the number of neurons that different brains use to carry out identical functions, even between genetically identical twins. Nevertheless, there is significant localisation of function across brains. A basic map of major brain subdivisions is shown in Figure 1. All adult brains show this basic structure. However, it is thought that, early in development, a number of possible developmental pathways and end states are possible. The fact that development converges on the same basic brain structure across cultures and gene pools is probably to do with the constraints on development present in the environment. Most children are exposed to very similar environments despite some cultural differences in rearing practices. Large differences in environment, such as being reared in darkness or without contact with other humans, are thankfully absent or rare. When large environmental differences exist, they have notable effects on cognitive function, some of which are beneficial. For example, neuroimaging studies show that blind adults are faster at processing auditory information than sighted adults and that congenitally deaf adults are faster at processing visual information in the peripheral field than hearing adults (see, for example, Neville, Schmidt & Kutas, 1983; Röder, Rösler & Neville, 1999; Neville & Bavelier, 2000).

Even so, neurons themselves are interchangeable in the immature system and so dramatic differences in rearing
environment can lead to different developmental outcomes. For example, deaf people’s brains use the area underpinning spoken language in hearing people to represent sign language (Neville, Bavelier, Corina, Rauschecker, Karmi, Lalwani, Braun, Clark, Jezzard & Turner, 1998). This is remarkable, as spoken language depends on auditory analysis whereas sign language depends on visual/spatial analysis. Hence the same neurons have the potential to process either auditory or visual/spatial information. Visual brain areas are recruited for braille reading (which requires tactile rather than visual analysis) in blind people (see Röder & Neville, 2003). It has even been reported that a blind adult who suffered a stroke specific to the visual areas of her brain consequently lost her proficient braille reading ability, despite the fact that her somatosensory perception abilities were unaffected (Jackson, 2000).

Another interesting perceptual possibility is that all modalities (vision, audition, touch) are initially mutually linked. Potential evidence for early multi-modal perception is that auditory stimulation during early infancy also evokes large responses in visual areas of the brain, while somatosensory responses are enhanced by white noise (Neville, 1995). If early mutual linkage of the senses is the norm, a kind of simultaneous perception or ‘synaesthesia’ could enable infants to extract schemas that are independent of particular modalities, schemas such as number, intensity and time (see Röder & Neville, 2003). For some, it may be that early synaesthesia is never lost. Around one in 2,000 adults is thought to be synaesthetic, for example experiencing distinct colours (‘photisms’) for numbers and letters. These colours are always consistent and are evoked simply by thinking about the number or letter: they are an intimate part of that person’s concept of numbers and letters. Synaesthesia seems to have beneficial effects on memory (Smilek, Dixon, Cudahy & Merikle 2002). If this mutual linkage of usually distinct sensory systems is also present in early childhood, it may explain why younger children respond so well to teaching via multi-sensory methods. Although there is as yet no research with synaesthetic children, synaesthesia is an area of growing interest for cognitive neuroscientists.

A short primer on neuroimaging tools

A quick primer on methodology is worth introducing here, as current neuroimaging techniques have important limitations. Neuroimaging is based on the assumption that any cognitive task makes specific demands on the brain. These demands are met by changes in neural activity. The brain pumps more blood to meet demand. Cognitive neuroimaging methods either measure local changes in blood flow directly (PET) or indirectly (fMRI) or measure the extremely low-voltage electrical impulses associated with brain activity (EEG and ERP).

PET (positron emission tomography) tracks blood flow via radioactive tracers. Brain areas with higher levels of blood flow have larger amounts of the tracer. Due to the tracers, PET is unsuited to children. When blood flow to particular brain areas increases, the distribution of water in the brain tissue also changes. fMRI (functional magnetic resonance imaging) depends on this property. It works by measuring the magnetic resonance signal generated by the protons of water molecules in neural cells, generating a BOLD (blood oxygenation level dependent) response. This technique depends on inserting participants into a large cylindrical magnet. It is very noisy inside the magnet and participants are given headphones to shield their ears and a panic button (the magnet is claustrophobic). Because of these factors, it has been challenging to adapt fMRI for use with children (who also move a lot, impeding scanning accuracy). However, with the advent of specially adapted coils and less claustrophobic head scanners, fMRI studies of children are growing in number. Both PET and fMRI show where brain activity is occurring (localisation of function). As images are often acquired over a few seconds (minimum possible resolution 0.5 seconds), PET and fMRI cannot tell us about the exact timing of mental events.

A different neuroimaging technique that is highly sensitive to timing is the event related potential (ERP). Sensitive electrodes are placed on the skin of the scalp in order to record brain activity. When the spontaneous natural rhythms of the brain are recorded this is called EEG (electroencephalography). When particular events are designed by the experimenter to affect spontaneous rhythms, systematic deflections in electrical activity are evoked (hence the event related potential). ERP rhythms are time-locked to specific events designed to study cognitive function and are widely measured in children. The usual technique is for the child to watch a video while wearing a headcap (like a swimming cap) that holds the electrodes. An illustration is provided in Figure 2. Another advantage of this method is that the child does not have to attend to the specific events in order for the brain to register them. For linguistic stimuli, the events can form a background noise while the child sits engrossed in a silent cartoon: the brain will respond to the auditory events in the same way. For ERP studies, neuroscientists measure (1) the latency of the potentials; (2) the amplitude (magnitude) and direction (positive
or negative) of the responses; and (3) the distribution of the activity. Exact localisation (where the responses originate) is not yet possible with ERP, but millisecond differences in timing are measurable and reasonably consistent.

Different electrical potentials (characterised in countless ERP studies) are clearly visible when the potentials are plotted against time, and are called N100, P200, N400 and so on. These labels denote negative peak at 100 ms, positive peak at 200 ms and so on. The amplitude and duration of single ERP components such as the P200 increase until age three to four years and then decrease until puberty. Decreasing amplitude usually indexes development in the school years. ERP latencies decrease within the first years of life and reach adult levels in late childhood. Therefore, decreasing latency usually indexes development in the school years. If the brain can do something 50 milliseconds faster in a ten-year-old than in a seven-year-old, this has a big effect on cognitive performance. In fact, it has been suggested that ‘g’, the general factor proposed by Spearman (1927) to underlie all individual differences in cognitive abilities, is actually speed of neural processing (Anderson, 2001).

**Selected empirical studies**

The tools of cognitive neuroscience have the potential to offer various exciting possibilities to education. For special education, these include the early diagnosis of special educational needs; the monitoring and comparison of the development of children with very poor intellectual ability seem to acquire relatively faster in a ten-year-old than in a seven-year-old, this has a big effect on cognitive performance. In fact, it has been suggested that ‘g’, the general factor proposed by Spearman (1927) to underlie all individual differences in cognitive abilities, is actually speed of neural processing (Anderson, 2001).

**Language**

One striking aspect of a number of developmental disorders is the relative sparing of the language faculty. Even children with very poor intellectual ability seem to acquire relatively normal language, particularly in terms of vocabulary. For reasons that are not yet well-understood, the brain systems important for syntactic and grammatical processing are more vulnerable than the brain systems responsible for semantic and lexical functions. These two aspects of language are also represented in different regions of the brain. Studies of non-disabled adults show that grammatical processing relies more on frontal regions of the left hemisphere, whereas semantic processing and vocabulary learning activate posterior lateral regions of both hemispheres.

ERP studies show that when English is acquired late due to auditory deprivation or late immigration to an English-speaking country, syntactic abilities do not develop at the same rate or to the same extent (Neville, Coffey, Lawson, Fischer, Emmorey & Bellugi, 1997). Children acquiring English as a second language may thus need extra support with grammatical aspects of their second language compared to support for basic vocabulary learning. Interestingly, brain imaging studies suggest that late learners do not rely on left hemisphere systems for grammatical processing, but use both hemispheres (Weber-Fox & Neville, 1996). ERP studies also show that congenitally blind people show bilateral representation of language functions (Röder et al., 2000). Blind people also process speech more efficiently (Holkins, 1989); for example, they speed up cassette tapes of normal speech, finding them too slow. Again, this shows that the development of certain sensory systems can be enhanced when other systems are impaired or absent. The implications of this for learning disability have not yet been studied.

**Reading**

In the field of specific reading disability or dyslexia, neuroimaging studies of both children and adults are widespread. These studies suggest that, at least for alphabetic scripts, the major systems for reading are lateralised to the left hemisphere. Such studies typically measure brain responses to simple word reading, using fMRI or ERPs. Reviews of such studies conclude that many neural regions are necessary for reading, as alphabetic/orthographic processing is associated with occipital, temporal and parietal areas (see, for example, Pugh, Mencl, Jenner, Katz, Frost, Lee Shaywitz & Shaywitz, 2001). The occipital-temporal areas are most active when processing visual features, letter shapes and orthography. The inferior occipital-temporal area shows electrophysiological dissociations between words (like ‘cat’) and non-words (like ‘cet’) at around 180 ms, suggesting that these representations are not purely visual but are linguistically structured. Brain activation in temporoparietal areas increases with reading skill (see, for example, Shaywitz, Shaywitz, Pugh, Mencl, Fulbright, Skudlarski, Constable, Marchione, Fletcher, Lyon & Gore, 2002) and activation is decreased in children with developmental dyslexia. There is also hyper-activation of this area in hyperlexic children (Turkeltaub, Flowers, Verbalis, Miranda, Gareau & Eden, 2004).

Studies of reading acquisition have emphasised the importance of phonological awareness (the ability to recognise and manipulate component sounds in words) across languages. Brain imaging shows that phonological processing appears to be focused on the temporoparietal junction. This appears to be the main site supporting letter-to-sound recoding and is also implicated in spelling disorders. Children with dyslexia, who typically have phonological deficits, show reduced activation in the temporoparietal junction during tasks such as deciding whether different letters rhyme (for example, P, T = yes, P, K = no). Targeted reading remediation increases activation in this area (see, for example, Simos, Fletcher, Bergman, Breier, Foorman, Castillo, Davis, Fitzgerald & Papanicolaou, 2002). This shows that special education programmes can affect very specific areas of the brain. Finally, recordings of event-related magnetic fields (MEG recordings) in children with dyslexia suggest that there is atypical organisation of the right hemisphere (Heim, Eulitz & Elbert, 2003). This is consistent with suggestions that compensation strategies adopted by the dyslexic brain require greater right hemisphere involvement in reading.
To date, it is probably fair to say that neuroimaging studies have largely confirmed what was already known about reading and its development from behavioural studies. Imaging studies have essentially confirmed the central importance of the language system for reading and writing. Brain activation is reduced in phonological areas of the language system in dyslexia and it is these areas that increase activation when targeted phonologically based reading remediation packages are administered. However, neuroscience techniques also offer a way of distinguishing between different cognitive theories. This work has not yet been done. For example, it is still argued that dyslexia may have a visual basis (see, for example, Stein & Walsh, 1997) or may be due to a deficit in the cerebellum (see, for example, Nicolson & Fawcett, 1999). Neuroimaging techniques could be used to measure activation in all three of these brain systems in the same children. Neuroimaging could also be used to measure the impact of training programmes devised in response to particular theories of dyslexia (see, for example, the DDAT, an exercise-based treatment deriving from the cerebellar hypothesis, which is based on motor exercises such as practice in catching beanbags while standing on a cushion on one leg, Reynolds, Nicolson & Hambly, 2003). If an exercise-based package actually improves reading in children with dyslexia, there should be measurable effects in the neural systems for reading.

Neuroimaging techniques also offer a potential means for distinguishing between deviance and delay when studying developmental disorders. Does the brain behave totally differently in these disorders or are affected children developing along the same trajectory as unaffected children but more slowly? In studies of linguistic disorders such as specific language impairment and dyslexia, ERP studies suggest that the language system of the child is immature rather than deviant (McArthur & Bishop, 2004; Thomson, Baldeweg & Goswami, 2004). In disorders such as autism, the brain may actually be different, lacking a ‘theory of mind’ module (Frith & Happé, 1998), or it may be that social cognitive abilities are following the usual developmental trajectory but are reliant on such impoverished input that this trajectory never looks normal (Baron-Cohen, 1997). The promise of neuroimaging is that we may actually be able to find out which of these alternative possibilities is correct. This would have enormous implications for intervention and remediation.

**Mathematics**

For mathematics, cognitive neuroscience has already gone beyond existing behavioural-cognitive models. It has been shown that there is more than one neural system for the representation of number. There is a phylogenetically old ‘number sense’ system, found in animals and infants as well as older humans, which seems to underpin knowledge about numbers and their relations (Dehaene, Dehaene-Lambertz & Cohen, 1998). This system, located bilaterally in the intraparietal areas, is activated for example when comparing two numbers (‘is three larger or smaller than five?’). Mode of presentation does not affect the location of these parietal ERP components, as responses are the same whether the comparisons involve Arabic numerals, sets of dots or number words. Developmental ERP studies have shown that young children activate exactly the same parietal areas to perform number comparison tasks (Temple & Posner, 1998).

A different type of numerical knowledge is thought to be stored verbally, in the language system (Dehaene, Spelke, Pinel, Stanescu & Tsirkin, 1999). This system also stores knowledge about poetry and overlearned verbal sequences (such as the months of the year), and underpins counting and rote-acquired knowledge such as the multiplication tables. The system seems to store ‘number facts’ rather than to compute calculations. Many simple arithmetical problems (for example, 3 + 4 or 3 x 4) are so overlearned, at least by adulthood, that they may be stored as linguistic knowledge. More complex calculation seems to involve visuospatial regions (Zago, Pesenti, Mellet, Crivello, Mazoyer & Tzourio-Mazoyer, 2001). It is thought that this may indicate the importance of visual mental imagery in multi-digit operations (that is, a sophisticated form of a number line, see Pesenti, Thioux, Seron & De Volder, 2000). Finally, a distinct parietal-premotor area is activated during finger counting and also calculation. This last observation may suggest that the neural areas activated during finger-counting (a developmental strategy for the acquisition of calculation skills) eventually come to partially underpin numerical manipulation skills in adults.

Neuroimaging studies of young children doing mathematics are still rare. One current growth area is dyscalculia. Dyscalculia is a specific difficulty in learning mathematics, despite good IQ and good performance in other curriculum areas. As there are distinct neural systems that contribute to mathematical cognition, not all children with dyscalculia may be the same in neural terms. For example, some children with dyscalculia are also dyslexic (Landerl, Bevan & Butterworth, in press). If dyslexia has a phonological basis, then it seems likely that the mathematical system affected in these children with dyscalculia will be the verbal system underpinning counting and calculation. Children with dyslexia and mathematical difficulties may not show comparable neural anomalies in the activation of the parietal and premotor number systems. Children with dyscalculia who do not have reading difficulties may show patterns of impairment in these other neural systems for number. We do not know, but neuroimaging offers a way of finding out. Knowledge of the neural basis of the difficulties experienced by different children with dyscalculia could then inform individual remedial curricula.

**General learning disability**

General learning disability, as opposed to specific learning difficulties like dyslexia and dyscalculia, encompasses many different conditions. General learning disability is usually defined on the basis of low IQ (scores below 70). About 40% of individuals with IQ below 70 have a medical background condition and this rises to 80% for individuals with IQ below 50 (Gillberg & Soderstrom, 2003). The most
common cause of learning difficulty is Down’s syndrome, which typically occurs because there is a third chromosome 21 in some cells (‘trisomy’). Many children with Down’s syndrome go on to develop the clinical features of Alzheimer’s disease as they get older. Other disorders linked to chromosomal abnormality include Fragile X syndrome, Angelman syndrome and Prader-Willi syndrome. Chromosomal abnormalities account for about 50% of cases of severe learning difficulties (Rittey, 2003). Other prenatal causes include intrauterine infection (for example, rubella) and intrauterine toxins (for example, alcohol), which can have devastating effects on brain formation. Alcohol in particular seems to show a dose-response relationship (that is, the more alcohol, the more damage is caused; Autti-Rämo, 2000). Birth asphyxia (lack of oxygen) and prematurity are other important causes.

Although brain imaging studies are rare within the area of general learning disabilities, there are some interesting neurophysiological hypotheses. For reasons that are not well understood, epilepsy is much more common in people with learning disabilities and affected individuals may suffer continual clusters of small brain seizures on a regular basis. This clearly impedes brain function. Other theories are linked to the disproportionate number of males who present with different medical conditions. For example, it has been suggested that dysregulation of the normal developmental trajectory of myelination may play a role in some disorders (Bartzokis, 2004). Myelin sheaths form around the axons (nerve fibres) of brain cells and increase neural transmission speed. Myelin also enables widely distributed neural networks to fire at the same time, which is necessary for higher-level skills like reasoning and memory. There is extensive myelination through middle age (late fifties). When myelination is dysregulated, there is abnormal development of white matter and this has been proposed to be characteristic of disorders such as Asperger syndrome and non-verbal learning disorder (Ellis & Gunter, 1999). Female hormones promote myelination, therefore acting as a protective factor.

Developmental abnormalities in the amount/thickness of myelin would be expected to particularly affect late-developing brain structures such as the frontal, temporal and parietal lobes. Interestingly, it has been proposed from brain imaging work that ‘g’, the general intelligence factor, may be specific to areas of the frontal cortex (Duncan, Seitz, Kolodny, Bor, Herzog, Ahmed, Newell & Emslie, 2000). If ‘developmental g’ is underpinned by frontal functions (see Anderson, 2001), and abnormalities in myelination particularly affect frontal regions, then there is a theoretical connection between myelination and the development of ‘g’. This is at present speculative. Nevertheless, if particular aspects of brain function can be related to general learning disability, this would inform pharmacological intervention. Meanwhile, even if they do not raise IQ, educational interventions always improve the quality of life of individuals with learning disability. This is because of their documented effects on behaviour and overall adjustment (see Gillberg & Söderstrom, 2003).

Neuromyths
An OECD report on understanding the brain coined the engaging term ‘neuromyths’ (OECD, 2002) to demonstrate the ease and rapidity with which scientific findings have also translated into misinformation regarding education. There are three myths given special attention in the OECD report, namely (1) the lay belief in hemispheric differences (‘left brain’ versus ‘right brain’ learning etc.); (2) the notion that the brain is only plastic for certain kinds of information during certain ‘critical periods’ and that therefore education in these areas must occur during the critical periods; and (3) the idea that the most effective educational interventions need to be timed with periods of synaptogenesis.

The idea of ‘left brain’ versus ‘right brain’ learning has virtually no credence in neuroscience. The idea appears to stem from the fact that there is some hemispheric specialisation in terms of the localisation of different skills. For example, many aspects of language processing are left-lateralised (although not, as we have seen, in blind people or in those who emigrate in later childhood to a new linguistic community). Some aspects of face recognition, in contrast, are right-lateralised. However, it is also a fact that there are massive cross-hemisphere connections in the normal brain. Both hemispheres work together in every cognitive task so far explored with neuroimaging, including language and face recognition tasks. So far, neuroimaging data demonstrate that both ‘left brain’ and ‘right brain’ are involved in all cognitive tasks.

Similarly, the conceptual notion of critical periods for learning has been overextended from the actual neuroscience findings. In fact, this concept stems largely from the neuroscience of the visual system rather than the neuroscience of cognition and learning. Although optimal periods for certain types of learning clearly exist in visual development, they are sensitive periods rather than critical ones. The term ‘critical period’ implies that the opportunity to learn is lost forever if the biological window is missed. In fact, there seem to be almost no cognitive capacities that can be ‘lost’ at an early age. As discussed earlier, some aspects of complex processing suffer more than others from deprivation of early environmental input (for example, depth perception in vision or grammar learning in language), but nevertheless learning is still possible. It may be better for the final performance levels achieved to educate children in, for example, other languages during the sensitive period for language acquisition. Nevertheless, the existence of a sensitive period does not mean that adults are unable to acquire competent foreign language skills later in life.

Finally, the idea that the effectiveness of educational interventions depends on whether they coincide with periods of synaptogenesis appears to be a misinterpretation of experimental work on learning in rats. This research showed that rodent brains form more connections if the young are reared in enriched and stimulating environments
(for example, Greenough, Black & Wallace, 1987; note however that these ‘enriched’ environments were within laboratory cages and did not come close to mimicking the intensely stimulating normative environment of the wild rat). Further, more connections form in response to particular environments throughout life. fMRI studies have shown that skilled pianists (adults) have enlarged cortical representations in auditory cortex, specific to piano tones (Pantev, Oostenveld, Engelien, Ross, Roberts & Hike, 1998). MEG studies show that skilled violinists have enlarged neural representations for their left fingers, those most important for playing the violin (Elbert, Pantev, Wienbruch, Rockstroh & Taub, 1996). London taxi drivers who possess ‘The Knowledge’ (detailed knowledge of the street map of London) show enlarged hippocampus formations compared to adults who do not drive taxis (Maguire, Gadian, Johnsrude, Good, Ashburner, Frackowiak & Frith, 2000; the hippocampus is a small brain area thought to be involved in spatial representation and navigation). Hippocampal volume was found to be correlated with the amount of time spent as a taxi driver, just as the cortical representation of piano tones was found to be correlated with amount of time spent in piano practice. These demonstrations do not mean that greater synaptic density predicts a greater capacity to learn. Rather, they demonstrate that the brain can always benefit from targeted inputs, even when these inputs are received exclusively during adulthood (as for taxi drivers learning ‘The Knowledge’).

Other neuromyths can also be identified. One is the idea that a person can either have a ‘male brain’ or a ‘female brain’. The terms ‘male brain’ and ‘female brain’ were coined to refer to differences in cognitive style rather than biological differences (Baron-Cohen, 2003) and applied to autism and autistic spectrum disorders. Baron-Cohen argued that men were better ‘systemisers’ (good at understanding mechanical systems) and women were better ‘empathisers’ (good at communication and understanding others). He therefore suggested that autism could be conceptualised as an ‘extreme’ form of the male brain. He did not argue that male and female brains were radically different, and that females with autism had male brains. Rather, he was using the terms ‘male’ and ‘female’ brain as a psychological shorthand for (overlapping) cognitive profiles.

A final neuromyth is the idea that ‘implicit’ learning has the potential to open new avenues educationally. Much human learning is ‘implicit’, in the sense that learning takes place in the brain despite lack of attention to and conscious awareness of what is being learnt (for example, Berens, Cohen & Mintun, 1997, but see Johnstone & Shanks, 2001). Almost all studies of implicit learning use perceptual tasks as their behavioural measures (that is, the participant gets better at responding appropriately to ‘random’ letter strings in a computer task when the ‘random’ strings are actually generated according to an underlying ‘grammar’ or rule system which can be learnt). There are no studies showing implicit learning of the cognitive skills underpinning educational achievement. These skills most likely require effortful learning and direct teaching.

Conclusions

Clearly, the potential is there for neuroscience to make exciting contributions to educational research in general and to special education in particular. Nevertheless, more bridges need to be built between basic neuroscience and research in education, and neuromyths need to be weeded out. Bruer (1997) first made the point about building bridges between the disciplines (in an article provocatively subtitled ‘a bridge too far?’) and suggested that cognitive psychologists are admirably placed to construct such bridges. He also cautioned that, while neuroscience has learnt a lot about neurons and synapses, it has not learnt nearly enough to guide educational practice in any meaningful way. In my view, this is too pessimistic. Cognitive developmental neuroscience has established a number of findings relevant to education, as discussed above, and has also enabled the discovery of neural ‘markers’ that can be used to assess development. These markers may prove very useful for investigating educational questions.

For example, consider the different ERP signatures of language processing that have emerged over the last 20 years of research (Brown & Hagoort, 1999). Different ERP parameters are robustly associated with semantic processing (for example, N400), phonetic processing (for example, mismatch negativity or MMN) and syntactic processing (for example, P600). The development of these parameters can now be investigated longitudinally in children. Certain patterns of development may turn out to be indicative of certain developmental disorders. For example, children at risk for dyslexia may show immature or atypical MMNs to phonetic distinctions, such as /b/ versus /d/ (Csepe, 2003, for Hungarian). Children with specific language impairment may have generally immature auditory systems – systems resembling those of children three to four years younger than them in terms of processing basic aspects of sound such as frequency (see McArthur & Bishop, 2004). The different ERP signatures may also change in response to targeted educational programmes. For example, the MMN to phonetic distinctions may become sharper (as indexed by faster latencies) in response to literacy tuition in phonics (see Csepe, 2003). If such findings were to be established across languages, education would have a neural tool for comparing the efficiency of different packages for remediating dyslexia. For example, one could measure whether the MMN to phonetic distinctions sharpened in response to different commercially marketed training regimes for dyslexia. This is only one example of the potential for the creative application of neuroscience techniques to important issues in special education. Bearing in mind the limitations of current technologies, it is time to think ‘outside the box’ about how available neuroscience techniques can help to answer important educational questions.
References


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Manuscript submitted: May 2004
Accepted for publication: July 2004

© NASEN 2004 British Journal of Special Education • Volume 31 • Number 4 • 2004
SYMPOSIUM NO. 14:
RESPONSE

CAN NEUROPSYCHOLOGY REALLY CHANGE
THE FACE OF SPECIAL EDUCATION?

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Journal of Learning Disabilities

Reed’s organizing thesis — “physicians cannot fully understand handicapping conditions nor can educators fully understand educational outcomes unless the physicians and educators understand the relationships between biological handicaps and educational outcome” — is certainly reasonable. Implicit in this thesis is the concept that one must use both biological and educational data bases for the optimal treatment of the child. While his reasons are unclear, he suggests that “to increase the educational sophistication of physicians would be worthy, but inappropriate for this forum. Happily, it is totally appropriate to exhort educators to become better versed in the biological nature of the handicapping conditions that identify many of the children for whom they are responsible.” While it is unclear why the onus should be on educators to expand their knowledge into biological circles and not the reverse, certain practical problems involved in this expansion might well have been considered in the paper. I shall return to these difficulties.

Reed’s laudable desire to have educators know more about potential biological bases for learning irregularities does not appear to be adequately supported in his paper. He recognizes that “the relevance of biological insults and disease for special education must be demonstrated rather than merely claimed.” While it would be difficult to question the necessity for evidence for such an assertion, Reed imputes general attitudes to both physicians and educators that surely cannot be valid. Specifically, he notes that “ignorance of the relationships between biological handicaps and learning ability has frequently led both physicians and educators to assume that such relationships do not exist.” Concerning the attitudes of physicians, he claims that they “fail to see diminished learning as an integral and permanent component of these conditions.” Regarding educators, he claims that the “possibility of a biologically based learning disorder is rarely if ever considered.” Instead, “the educator assumes that the child’s failure to learn at what is judged to be an appropriate rate must necessarily be the consequence of inappropriate teaching methods and materials or, worse, be due to interfering emotional problems or deliberate stubbornness on the part of the child.” I am sympathetic with Reed’s desire to see the rapidly expanding and learned area of neuropsychology become a force in special education. However, it is false to claim that mutual ignorance on the part of physicians and educators, specifically biological biases of the former and biological igno-
rance of the latter, is largely instrumental in the present schism between them. Such a claim ignores both the social history behind the present situation as well as the sociocultural conditions which support the present state of affairs and which will resist the integration of knowledge for which Reed argues.

**Issue of relevance of biological factors**

It is important to convince educators of the *relevance* of biology in the handicapping conditions with which they work. I think Reed misreads the educator’s attitude in this respect. I believe that educators do not necessarily deny the biological bases of many of the handicapping conditions; rather, they see them as irrelevant to the intervention/treatment process. As Reed is well aware, neuropsychology and those areas of special education concerned with the retarded and the learning disabled (many of whom were previously labeled “minimally brain damaged” and related terms) share some common ancestry. The biologically oriented thinking of Orton, which continues to stimulate research in the area of dyslexia, as well as that of Strauss and his collaborators, are two striking cases in point. In the latter case, a significant portion of the handicapping condition known today as learning disabilities has direct roots with “minimal brain dysfunction,” “minimal brain damage,” and related terms (cf., Clements, 1966). It is not, then, simply that educators have remained ignorant of the biological bases of learning. Historically, those concerned with the unique educational problems presented by these children actively pursued biological hypotheses. This history is reflected today in many laboratories, in a large-scale program by the Orton Society seeking neuroanatomical and biochemical concomitants of dyslexia, in a large electrophysiological study of learning-disabled children sponsored by the Bureau of Education for the Handicapped (E. Roy John, principal investigator), as well as in many smaller projects in laboratories throughout the country.

The relevance of this historical perspective is that for some reason etiological and, in particular, biological causes for disability have increasingly become of less concern to the practicing special educator. The attitude I frequently hear is not that biological bases do not exist but that they are simply irrelevant to the educational problems facing practitioners and their clients. One still must teach the child, whatever the cause of the handicapping condition. If I am correct that it is the alleged *irrelevance* of the biological bases, as opposed to the denial of their existence, that causes the special educator’s disinterest in this area, more convincing evidence, and evidence of a different sort than Reed presents, would be necessary to capture the special educator’s attention.

Reed presents two lines of evidence to persuade the special educator of the relevance of biological factors in educational problems. The first includes a review of such factors as neural tube defects, malnutrition and its related impacts, and low birth weight. While the distinction is sometimes made between conditions which produce undifferentiated lowering of intelligence and those which produce more selective impairment, the evidence is clear that the integrity of the neurological system is related to the system’s capability to perform intellectual-academic problems. Such a conclusion is hardly startling, it is simply
incontestable. And paradoxically, as Reed himself states, this point is somewhat irrelevant to the central thesis of why special educators appear disinterested in the biological underpinnings of behavior. He notes that the research studies reviewed concerning severe biological disorders (a) “represent a low incidence condition,” (b) “have only limited applicability to children in the United States,” and (c) have the “limitations of knowledge expressed in measures reflecting group tendencies.” There is no convincing reason here why the special educator should adopt a new-found interest in biological bases for educational problems.

Neuropsychological knowledge about adults and children

In bridging to his second line of evidence as to why special educators should be interested in biological factors — the neuropsychological analysis of three clinical cases — Reed presents an admittedly skeletal review of adult neuropsychology. Three points are particularly noteworthy. First, the years of research and clinical work noted provide an enormous knowledge base for understanding the problems of organically impaired children. Thus, that work represents probably the best justification for using a neuropsychological model within special education. In Reed’s brief history, names known to most special educators are prominent — Birch, Benton, Kephart, Reitan, Strauss — supporting my earlier point that the special educator’s disinterest in biological concomitants is not simply a matter of “ignorance.”

Second, Reed notes that “it is very useful, however, to know that adult brain-injured patients differ from matched controls on measure of abilities even when difference in IQ scores are controlled. The principal point to be made here is that batteries of tests provide genuinely useful information that cannot easily be provided in any other way.” Here is an area where special education and neuropsychology share a common belief: that the IQ, though useful in some contexts and for some functions, needs to be supplemented with information about more specific skills. Both neuropsychology and special education are heavily involved in clinical practice. Hence, they must both deal with the intraindividual characteristics of clients — those skills they perform well and those in which they are deficient. While the attendant theoretical models of the two fields may be different, their need to understand the individual case provides them with a similar framework conducive to interdisciplinary collaboration.

A third point that Reed graciously acknowledges is the more tentative status of neuropsychological knowledge with respect to children. While he notes that the “litany of barriers to adequate neuropsychological research programs with children has not stymied progress altogether,” the reader cannot likely be inspired with a high level of confidence in the child neuropsychologists’ craft. The subsequent discussion of discrepant results found in studies of children versus adults erodes one’s confidence. I here refer to the admittedly “limited value” of the research strategy utilized in which “nearly all of the pediatric neuropsychological research cited above compares groups of normal children with groups of brain-injured children.” I mention this point because my expe-
rience tells me that the neuropsychological test batteries, such as the one Reed subsequently describes, rest on no firmer foundations than those used in special education. Reed is certainly to be commended for his openness — if not for his salesmanship.

The second line of evidence Reed presents regarding the importance of biological factors is a series of three case histories. It is difficult to accept the assertion that the case studies demonstrate biological causation in view of the many caveats which precede the studies. Certainly, it is disquieting to find Reed himself stating, "It is becoming quite clear that brain lesions result in recognizable distortions in developmental patterns that have profound educational significance," followed immediately by the sentence, "it is just now becoming possible to formulate the kind of controlled studies necessary to test this hypothesis" (italics added). Reed seems to oscillate between the position that biological bases for educational disorders of a more subtle sort have been demonstrated and the position that this proposition is still formally a hypothesis.

**Issues of test validity**

Whether or not the case histories convince the reader of the biological bases for some learning problems, one needs to examine logically how such evidence could be convincing. The issue here is not whether the diagnostic information will have remedial value. (That question certainly can be raised, but it is tangential in that other case histories could readily have been selected to enhance the belief that neuropsychology has implications for educational treatment.) Rather, the issue concerns whether the tests administered relate to brain function in the way that Reed asserts. This linkage is essentially an issue of demonstrating construct validity. Reed is to be excused for not having taken on this task, for it would have involved an extensive review of clinical and experimental studies for each of the procedures in the lengthy battery of tests he describes. Without such a demonstration of the brain involvement associated with signs provided by each of the tests discussed, the reader must accept on faith the assertion that disordered brain activity is both indicated by the test scores and related to educational outcomes — the very assertion that Reed wishes to prove and the one which he acknowledges "must be demonstrated rather than merely claimed."

One is left to conclude that biological factors are relevant to a number of severe neuropsychological disorders which are of low incidence; the factors are otherwise of admittedly little relevance to most special educators. The second line of evidence for brain involvement in educational problems, the case histories, fails to prove biological causation and probably is unconvincing to the reader unfamiliar with neuropsychological tests and the attendant literature. I am sympathetic with Reed's position that some educational problems will be found to be heavily influenced by biological factors; I also am sympathetic with his stance that neurological status can profoundly affect optimal educational treatment. A recent study by Adams (1978) adequately demonstrates that the children's cerebral status — damaged or not — was significantly related to the success of different intervention strategies. The critical point here is that the
educator's belief that brain status is irrelevant may eventually be proven false: Brain status may well become relevant information in treatment planning. This assertion will, of course, have to be proven in sufficient detail so that special educators will want to look to neuropsychological data for intervention ideas.

Limitations on a special education partnership with neuropsychology

While the acceptance of neuropsychology by special educators would logically depend on its utility, other factors prohibiting its acceptance loom large. Territoriality is a fact of life in most areas of human endeavor, including the provision of human services. I think it is fair to say that the educational community has been successful in wresting much of special education's enterprise from related professions, especially those medically-related. For example, the minimal-brain-dysfunction task force document (Clements, 1966), intended to be seminal by its primarily medical authors, has laid dormant the last decade despite the intense interest aroused in the definition of the condition. Is it reasonable to expect easy entrance into special education of a psychological discipline which is related to medical practice and biology and which bases its thinking in brain–behavior relationships?

A second practical matter needs to be considered: How can the already full curricula of the special educator accommodate the demands of the neuropsychologist? Neuropsychological training is one of the most demanding of the psychological specialties; it involves a knowledge of fields additional to general psychology. In fact, neuropsychology has already begun to spawn its own sub-specialties (such as "child neuropsychology" with a heavy emphasis on development), further emphasizing the educator's insurmountable problem in trying to master this related field of knowledge.

Whether neuropsychology proves itself worthwhile to the special educator, whether neuropsychology represents a sociologically acceptable partner with education (with all the sharing of resources and authority that such acceptance represents), whether the special educator can learn enough about neuropsychology to feel comfortable with it as a working partner, and so on — these "whether's" may speak more directly to the special educators' "ignorance of the relationships between biological handicaps and learning ability."

Uncertain future

There can be little doubt that special education could use new conceptualizations of the dysfunctions its practitioners face every day. Many of the prominent conceptualizations seem either to have lost their status as "normative definitions" (Kuhn, 1962) or to have become intellectual straightjackets, both to those who practice and those who conduct research. Neuropsychology certainly presents an alternative model and a set of procedures with which to understand many of the dysfunctional behaviors special educators see in their practice. Whether special educators will come to see neuropsychologists as useful colleagues and their information as of value to educational practice is the critical question.
References


Of the recent advances in education-related research in Down syndrome, the characterization of the Down syndrome behavioral phenotype has become a potentially critical tool for shaping education and intervention in this population. This article briefly reviews the literature on brain–behavior connections in Down syndrome and identifies aspects of the Down syndrome behavioral phenotype that are potentially relevant to educators. Potential challenges to etiologically informed educational planning are discussed.

Key Words: Down syndrome; behavioral phenotypes; education; intervention

As the most common chromosomal abnormality associated with intellectual disability, Down syndrome has been the focus of more behavioral and educational research than most other genetic disorders. These efforts have led to notable milestones in the advancement of education in Down syndrome, including cases in the literature of young adults with Down syndrome attending university courses [Hamill, 2003; Casale-Giannola and Wilson Kamens, 2006] and achievements in the area of educating children and adolescents in inclusive environments [Buckley et al., 2006]. The past five years alone have brought innovations that include teacher training interventions to shape attitudes toward inclusion in Down syndrome [Campbell et al., 2003], a refined understanding of effective inclusive practice in Down syndrome [Wolpert, 2001], and new instructional approaches involving computer technology [Lloyd et al., 2006; Ortega-Tudela and Gomez-Ariza, 2006].

However, the innovation that has the potential to have the greatest impact on educational practice in Down syndrome is the characterization of the Down syndrome "behavioral phenotype," or the pattern of behavioral outcomes associated with this disorder throughout development. Research into the phenotypic outcomes associated with Down syndrome has led to a better understanding of the learning profile associated with this disorder, and has offered new information regarding the possible brain–behavior pathways leading to these outcomes. Over the past few decades, researchers have uncovered characteristic patterns of functioning in the areas of cognition, language development, social–emotional functioning, and personality-motivation [see Dykens et al., 2000; Rondal and Buckley, 2001; Fidler, 2005]. Though many questions regarding development in this population remain unanswered, researchers have gained a clearer understanding of the developmental trajectory associated with Down syndrome, and how this chromosomal abnormality impacts development in a dynamic and multisystemic way.

Amidst these advances in delineating the Down syndrome behavioral phenotype, there remains a wide gap between these research findings and the development of innovative practice [Hodapp and Fidler, 1999; Fidler et al., 2007]. While it has been argued that etiology-based information could be of importance for education in Down syndrome [Hodapp and Fidler, 1999; Freeman and Hodapp, 2000; Fidler, 2005], the use of these connections has not nearly met its full potential. It is true that relative to other disorders, greater research emphasis has been placed on identifying educational strategies that might improve outcomes in Down syndrome. Some of these recent approaches are informed by and specifically target aspects of the Down syndrome behavioral phenotype [Laws et al., 1996; Kennedy and Flynn, 2003; Iarocci et al., 2006; van Bysterveldt et al., 2006], and others do not [Garcia and Conte, 2004; Park et al., 2005; Trent et al., 2005]. Those existing syndrome-based recommendations may be potentially quite useful, but lack empirical validation [Alton, 1998; Fidler, 2005].

Though the gap between research findings and targeted practice in Down syndrome remains wide, there is evidence that educators and practitioners themselves recognize the importance of scientific progress in this area. Wolpert [2001] asked educators of children with Down syndrome in inclusive settings to identify factors that might improve the outcomes in the classroom. Among their answers was "... more informa-
tion on learning characteristics of children with Down syndrome” (p. 33). Similarly, parents seem to endorse etiology-specific modifications to their child’s educational planning [Fidler et al., 2003]. As part of an effort to address the gap between research and practice in this area, this article will first review the current literature on the pathway from brain to behavior in Down syndrome. We then identify examples of phenotypic outcomes that may have educational relevance, with particular attention to the development of cognition and information processing in Down syndrome. Additional areas that may have educational relevance, including language development, social-emotional development, and personality-motivation, are briefly summarized. We conclude with a brief discussion of the potential challenges involved in shaping educational instruction in etiologically relevant ways. Although we start our review with discussion of neurobiology, we believe these kinds of data will be most relevant to the development of animal models, which then can be used to validate biological approaches to mitigation of the syndrome’s impact on cognition function. More important for educational practice will be an understanding of the phenotypic result of these neurological sequelae of trisomy 21, and we will accordingly focus on such behavioral approaches below.

THE NEUROBIOLOGY OF DOWN SYNDROME

The brain of an individual with Down syndrome at or shortly before birth is in many respects indistinguishable from the brain of a normal individual [Brookbanks et al., 1989; Wisniewski and Schmidt-Sidor 1989; Flórez et al., 1990; Schmidt-Sidor et al., 1990; Bar-Peled et al., 1991; Pazos et al., 1994]. Normal values have been reported for brain and skull shape, brain weight, proportion of specific cerebral lobs, size of cerebellum and brain stem, and the emergence of major neurotransmitter systems. There is evidence, however, that some changes begin to emerge as early as 22 weeks gestational age [e.g., Schmidt-Sidor et al., 1990; Golden and Hyman, 1994; Wisniewski and Kida, 1994; Engidawork and Lubec, 2003] and it is clear that by the age of 6 months a number of important differences are already obvious. Some of these differences are expressed in terms of the proportion of individuals with Down syndrome who show abnormal values, rather than in terms of a uniform abnormality in all instances. This is important as it highlights the variability in this population sharing the genotypic feature of trisomy 21.

While there is a postnatal delay in myelination [Wisniewski, 1990], it is worth noting that in all cases myelination is within normal range at birth, while in 75% of the cases it is within normal range throughout early development. Neuropathological differences after 3–5 months of age include a shortening of the fronto-occipital length of the brain that appears to result from a reduction in growth of the frontal lobes, a narrowing of the superior temporal gyrus (observed in about 35% of cases), a diminished size of the brain stem and cerebellum (observed in most cases), and a 20–50% reduction in the number of cortical granular neurons [see Crome et al., 1966; Benda, 1971; Blackwood and Corellis, 1970]. In sum, the overall picture at birth or shortly thereafter shows that individuals with Down syndrome tend to fall towards the bottom of the normal range (or outside it) on most measures.

Investigations of neural function, as opposed to structure, in early infancy suggest some abnormalities: there is evidence of either delayed or aberrant auditory system development [Jiang et al., 1990], which might contribute to the widespread hearing disorders observed in Down syndrome. Obviously, such a disorder, if organic, could be related to subsequent difficulties seen in the learning of language. Hill Karrer et al. [1998] have reported delayed development of cerebral inhibition using visual event-related potentials (ERP) in a visual recognition memory paradigm.

This is only one of many studies of brain function in adolescents and young adults with Down syndrome and the existing data are somewhat equivocal. Pinter et al. [2001] used high-resolution MRI methods to analyze brain structure in 16 children (mean age 11.3 years) with Down syndrome. After correcting for overall brain volume, hippocampal, but not amygdala, volume reductions were seen in this group. This result confirms some earlier work using lower resolution MRI methods [Jernigan et al., 1993]. Kates et al. [2002] looked at a group of 12 children with Down syndrome (all males: mean age 5.94 years) and compared them with children with fragile-X, developmental language delay, or typical development. The children with Down syndrome had smaller brain volumes than any of the others, with previously unreported reductions in parietal cortex as well as the oft-reported reductions in the temporal lobe. Pinter et al. [2001], on the other hand, note the relative preservation of parietal cortex.

Overall, study of neuropathology points to problems in certain regions of the cortex, including most prominently the temporal lobe and the hippocampal formation [Wisniewski et al., 1986], the prefrontal cortex, and the cerebellum. This conclusion meshes well with what has been learned from the development of mouse models of the syndrome [e.g., Kleschevnikov et al., 2004]. These models have generally demonstrated selective impairments in the anatomy, physiology, pharmacology, and behavior, which are associated with the hippocampal formation, the prefrontal cortex, and the cerebellum.

These neurobiological findings in Down syndrome can inform education in several ways. First, as only modest abnormalities are detectable at birth, the role of development is critical in building into the pronounced profile of strengths and weaknesses observed in this population. This offers educators an important window of opportunity for intervention in the first few years of life, a point that will be revisited later. Second, the atypical development of specific brain structures suggests that some areas of functioning may become more impaired than others throughout development. In addition, early in development, many of the differences observed are expressed in terms of the proportion of individuals with Down syndrome who show abnormal values, rather than in terms of a uniform abnormality in all instances. This is in line with the probabilistic approach to understanding outcomes in individuals with genetic disorders [Dyckens, 1995], where children may be at risk for certain neurobiological features, but not all children will show those specific abnormalities. These three themes are educationally relevant and will be explored in more depth in the following sections. Additional links between brain and behavior will be noted throughout the next section where relevant findings have been reported.

REVIEW OF EDUCATIONALLY-RELEVANT LITERATURE ON DEVELOPMENT IN DOWN SYNDROME

In this section, we explore the current literature on development in Down syndrome and highlight some of
COGNITION IN DOWN SYNDROME

Early Cognition

In line with the neurobiological findings described earlier, infants with Down syndrome show relatively normal abilities in learning and memory. This does not mean that either they, or indeed normally developing infants, have the full adult range of learning and memory abilities at birth. In fact this is not the case because some parts of the brain mature postnatally and the forms of learning and memory dependent on them are not available until some time after birth. The medial temporal lobe, and particularly, the hippocampus, as well as parts of the prefrontal cortex and cerebellum, are included in this category. The fact that these late-developing structures are apparently particularly at risk in Down syndrome is probably of considerable importance [see Nadel, 1986].

In an early series of studies, Ohr and Fagen [1991, 1993] reported that 3-month-old infants with Down syndrome were entirely normal in learning about the contingencies between their own movements (leg kicking) and reinforcement, including initial learning, acquisition speed, and retention. In a later report, Ohr and Fagen [1994] showed that 9-month-old infants with Down syndrome were impaired, as a group, in learning about the contingency between arm movements and reinforcement. However, they noted that some infants with Down syndrome were able to learn. They concluded that there is a relative decline in conditionability in infants with Down syndrome compared to normally developing infants after 6 months. Mangan [1992] tested control infants and infants with Down syndrome on a variety of spatial tasks, one of which, a place-learning task, was designed especially to assess the state of function of the hippocampal system. The pattern of results was consistent with diffuse, but mild, neuropathology combined with much more extensive pathology localized to the hippocampus.

Children with Down syndrome have typically been shown to acquire basic object concept more slowly than normally developing children (e.g., Ray and Meltzoff, 1995) but with intensive training they can acquire it at more-or-less the same time as normally developing infants [Wishart, 1993]. However, a different kind of problem emerges in this task situation: instability of acquisition. Although the typical subject with Down syndrome solved various levels of the task used to assess the object concept at ages not very far from the norm, performance after acquisition could be highly variable and apparently beset by motivational difficulties. These problems, if representative of the learning style of children with Down syndrome, are extremely important in thinking about effective intervention. The results of Wishart’s studies using standard intelligence test batteries suggest that they are indeed representative. Test–retest reliability was very low because successes gained in one test might not appear upon retest, as soon as 2 weeks later. New skills show up, only to disappear shortly thereafter. One could speculate that evidence of such “rapid forgetting” is consistent with damage in the hippocampal formation but considerably more data are required before this conclusion can be accepted. These developmental differences may have important implications for educators. For example, if the learning process involves more observable regressions for children with Down syndrome than for other children, it might be important for teachers and interventionists to account for these differences with more frequent reviews of materials, and it may be important to monitor the stability with which a child with Down syndrome has acquired a skill.

Information Processing

The learning and memory problems that begin to emerge in late infancy become considerably more noticeable as the infant grows to childhood and adolescence. These effects vary as a function of the type of memory being assessed. Explicit memory involves things like facts that subjects consciously recollect, whereas implicit memory can be demonstrated indirectly, without conscious recollection. This distinction has been shown to be important in understanding organic amnesia, since most amnesics are profoundly impaired on explicit memory tasks but can be relatively normal on implicit tasks. One common kind of implicit memory test looks at skills or procedures, such as mirror-tracing; another common implicit memory test involves “priming,” where prior exposure to a word or picture can influence subsequent performance on word-stem or partial-picture completion tasks even though the subjects might not recall having seen the relevant items before.

Carlesimo et al. [1997] examined the performance of children with Down syndrome in the area of “implicit” (procedural) and “explicit” (episodic) memory paradigms, including word-stem completion, list learning, and prose recall. Robust priming effects were seen in the Down syndrome group, comparable to those observed in controls, indicating that implicit memory was intact. However, deficits were observed in both explicit memory tasks. Performance on these kinds of explicit memory paradigms has been linked to functions...
of the hippocampal system; hence, the defects suggest differential impairment in hippocampal function and thereby converge with the data from study of spatial cognition. This selective impairment of explicit, but not implicit, memory was also reported in Vicari [2001].

A great deal of research attention has been focused on deficits in the processing of verbal information in Down syndrome, and how these deficits contribute to poor language and learning outcomes [Byrne et al., 1995; Hesketh and Chapman, 1998; Laws, 1998]. Most commonly, the verbal working memory deficit in Down syndrome is measured by performance on an auditory digit span task. Poor auditory digit span performance findings in Down syndrome were first reported several decades ago [Bilvosky and Share, 1965; Rempel, 1974].

More recently, a series of studies by Hulme and Mackenzie [1992] described the development of poor verbal working memory in children with Down syndrome in great detail. They found that children in the Down syndrome group scored lower than the typical MA-matched controls on auditory digit span and mental arithmetic tasks that require verbal working memory tasks were more correlated in the typically developing children than in the Down syndrome group. Similar deficits in verbal working memory are also observable when letters are substituted for numbers in the paradigm [Varnhagen et al., 1987]. This deficit is also not subject to changes when experimental design is manipulated in order to reduce unrelated demands on individuals with Down syndrome [Marcell and Weeks, 1988; Marcell et al., 1990; Laws, 1999].

Deficits in verbal working memory may relate to neuroanatomical characteristics associated with Down syndrome, including a proportionately smaller planum temporale, which is referred to as the auditory association cortex [Frangou et al., 1997]. It is important to note, however, that the relationship between the planum temporale volume deficit and cognitive-linguistic functioning in Down syndrome remains unclear [Frangou et al., 1997].

Though there is a great deal of evidence of verbal working memory deficits in Down syndrome, it is important to note that the main evidence for this dysfunction comes from studies that measure the processing of acoustically-presented information only. Auditory digit span, auditory sentence recall, and other commonly used techniques auditory nonword repetition all involve the processing of acoustic, rather than speech-based visually presented information. Thus, one might argue that the verbal working memory difficulty in Down syndrome has only been demonstrated in the maintenance of auditorially presented information, rather than the processing of all types of verbal information. The processing of speech-based visual information in Down syndrome has not yet been shown to be equally impaired, and importantly, may not be.

In contrast with their performance on verbal processing tasks, individuals with Down syndrome show relative strengths in visuospatial processing, and show a profile of higher visuospatial processing than verbal processing [Silverstein et al., 1982; Thase et al., 1984; Pueschel et al., 1987; Wang and Bellugi, 1994; Jarrold et al., 1999; Klein and Mervis, 1999]. The Corsi span, a block tapping memory task, is most often used as the test of visuospatial processing. In terms of raw scores, most individuals without Down syndrome (both with developmental delay and typically developing) tend to show higher raw auditory digit span recall scores than Corsi span recall scores. However, individuals with Down syndrome seem to perform equally well on these tests, or even show an advantage for Corsi span recall [Hazby, 1989; Azari et al., 1994; Wang and Bellugi, 1994; Vicari et al., 1995; Jarrold and Baddeley, 1997], especially when participants were not required to include order in their responses. These results have also been demonstrated with tasks other than the Corsi and auditory digit spans [Frangou et al., 1997; Hodapp et al., 1992; Bower and Hayes, 1994; Klein and Mervis, 1999]. The visuospatial working memory advantage is also demonstrated when identical stimulus information is simply presented either visually or auditorily [Varnhagen et al., 1987].

Neuroanatomical correlates for this relative strength in Down syndrome visuospatial processing have been proposed. Pinter et al. [2001] reported what they call “striking preservation” of parietal and occipital cortical gray matter in an MRI study of 5–23-year-olds with Down syndrome. Studies have shown the importance of both parietal and occipital lobe functioning for some aspects of visuospatial processing [Black and Bernard, 1984; Jonides et al., 1993].

An information processing profile that includes strengths in visual processing and implicit memory, and deficits in verbal processing and explicit memory could potentially inform educational approaches to working with children with Down syndrome. Instruction that is verbally based—and especially auditorily mediated—might pose a greater challenge to children with Down syndrome than instruction that is presented with visual supports. Minor and subtle teaching modifications can be made to address this issue, without any noticeable disruption to a larger classroom environment. In addition, instruction that involves explicit memory, such as logic problems, may be presented with greater sensitivity given that this is an area of challenge as well. Awareness of this area of deficit might allow an educator to make informed decisions regarding prompts, wait time, and supports for a child who may have particular difficulty with this type of information processing.

Reading

The cognitive underpinnings of reading are of particular relevance for education in Down syndrome. Despite the impairments observed in other areas of cognition, many individuals with Down syndrome are able to show competence in some aspects of reading development. In particular, word identification appears to be an area of relative strength within reading skills in this population, while word attack and reading comprehension appear to be more impaired [Byrne et al., 1995; Cupples and Iacono, 2000; Kay-Raining Bird et al., 2000]. Strengths in word identification have been linked to relative strengths in visual processing [Fidler et al., 2005b], while deficits in word attack skills might be linked to difficulties with verbal processing skills in Down syndrome [Boudreau, 2002].

There is some controversy regarding the implications of this profile for reading instruction in Down syndrome [see Hodapp and Freeman, 2003 for a summary], with some arguing that visually-based approaches might be warranted [Buckley and Bird, 2002], and others emphasizing the role of phonological processing in reading in this population, despite impairments [Cupples and Iacono, 2002]. Regardless of this debate, there is evidence that children with Down syndrome recruit both their visual and verbal processing skills when reading, particularly when identifying words [Cupples and Iacono, 2000; Fidler et al., 2005b].
BRAIN–BEHAVIOR CONNECTIONS

In a series of recent studies, Pennington, Nadel and colleagues have tested several different groups of individuals with Down syndrome on a range of tasks designed to directly assess the function of specific brain systems. This “cognitive neuropsychological” approach often uses tasks first developed in animal models, where the critical underlying brain circuits can be identified and carefully studied in invasive experiments. The team started with a focus on three brain systems identified by the neuropathological data, much of which was discussed above: the hippocampal system, the prefrontal cortex, and the cerebellum. They developed a set of tasks that could, collectively, tell us something about how these brain systems are faring. In the first set of studies, Pennington et al. [2003] found evidence of specific hippocampal dysfunction in our sample of 28 adolescents, using mental age matched controls.

Little evidence of prefrontal dysfunction was observed in a battery of nonverbal tasks. Subsequent pilot work, however, suggested that verbal tasks might yield a different result, and indeed that is what is being observed (Moon et al., unpublished data). Using verbal tasks to explore the prefrontal cortex, Moon et al. found in the young (aged 5–11) and old (aged 30–41) groups strong signs of dysfunction in both the hippocampal and prefrontal systems. Deficits were observed in a range of tasks although verbal mediation was necessary to bring out the prefrontal effect. Taken as a whole, these studies show that particular problems emerge in the memory domains served by the hippocampal system and the prefrontal system. The latter impairment appears to be linked to the use of verbal test materials. The impairment in hippocampal function could in principle reflect problems in any of the structures of the hippocampal region; a recent study of two neuropsychological paradigms dependent on parahippocampal and perirhinal regions (delayed nonmatching to sample and visual paired comparison), however, suggests that these areas are functioning appropriately, and that the impairment is more likely to reflect improper development of the hippocampus itself [Dawson et al., 2001].

The prefrontal cortex, as noted already, plays an important role in a wide range of functions, including episodic/explicit memory and working memory. As noted earlier, explicit memory is impaired in individuals with Down syndrome. There has been extensive research on working memory in this population, and clear deficits have been observed in a number of studies [Varnhagen et al., 1987; Marcell and Weeks, 1988; Laws, 1998; Jarrold et al., 1999]. However, this impairment seems to be limited to verbal information; as impairments are minimal in visuospatial domains. The deficit appears to be neither a motor nor articulatory problem [Kanno and Ikeda, 2002] and may relate to the so-called phonological loop [Jarrold and Baddeley, 2001; Laws, 2002].

OTHER AREAS OF EDUCATIONAL RELEVANCE

In addition to cognitive development and information processing, there are other areas of development in Down syndrome that may be relevant to decisions made by educators. Brief descriptions of findings in the area of language development, social–emotional functioning, and personality–motivation are presented in this section.

Language Development

Individuals with Down syndrome generally show a profile of stronger receptive language skills and weaker expressive language skills. This profile seems to emerge in early childhood and become more pronounced as children progress into middle childhood and beyond [Miller, 1999]. Expressive language deficits are often manifested in terms of morphosyntactic delays [Chapman et al., 1998; Eadie et al., 2002] and difficulties with speech intelligibility [Naiga et al., 1999; Stoolman, 2003; Kumin, 2006]. And while receptive language is stronger than expressive language in most individuals with Down syndrome, some areas such as receptive syntax, are more compromised than others [Abbeduto et al., 2003]. In contrast, some areas of pragmatic functioning seem to be an area of relative strength in individuals with Down syndrome [Johnston and Stansfield, 1997; Laws and Bishop, 2004].

This language profile is potentially relevant for educational planning in several ways. First, because many children with Down syndrome have stronger receptive than expressive language skills, they often understand much more language than they can produce. To an uninformed educator who is not aware of the discrepancy between expressive and receptive language, it might be natural to address the child with input language and instruction at the level of their expressive language. But such an approach would likely involve a marked underestimation of the child’s academic and receptive abilities. When considering how best to present class material, it may be important for educators to identify and target the receptive language level of a child with Down syndrome so as to appropriately challenge them and engage them at their true level of understanding [Roberts et al., 2007].

In addition, it may be useful for educators to be aware that difficulties with expressive language—and speech intelligibility in particular—may be frustrating for children with Down syndrome in classroom contexts. Morphosyntactic difficulties demand extra motivation from children with Down syndrome to produce lengthy utterances [Miller and Leddy, 1999], and intelligibility problems may lead to situations where a child must repeat herself in order to be understood. Thus, educators may want to consider both the social and motivational consequences of expressive language difficulties. It may be beneficial to identify ways to minimize the potential for negative experiences, while allowing the child with Down syndrome to benefit from the opportunity to build their speech, language, and communication skills.

Social–Emotional Functioning

The majority of individuals with Down syndrome show strengths in various aspects of social–emotional functioning, exhibiting behaviors that suggest evidence of intact social relatedness and some measure of social competence in early childhood [Fidler et al., 2006]. In the first few years of life, markers of primary intersubjectivity, the earliest forms of dyadic social relating based on emotional displays and reciprocal signaling, are identified as emerging in a delayed, but comparable manner for this population [see Fidler, 2006 for a review]. In particular, young children with Down syndrome show evidence of primary intersubjective development in the form of increased eye gaze and vocalizations directed to other people [Gunn et al., 1982; Crown et al., 1992; Legerstee et al., 1992; Kasari et al., 1995], increased direction of positive facial displays in the form of smiles [Kasari and Freeman, 1990; Fidler et al., 2005a].
In addition, there is also support for the notion that many aspects of secondary intersubjectivity, the ability to engage with a social partner in a triadic fashion, also emerge with competence, albeit in a delayed fashion. Several studies suggest that children with Down syndrome show either MA-appropriate or even increased levels of joint attention relative to other children without Down syndrome [Mundy et al., 1988; Kasari et al., 1995; Fidler et al., 2005c]. Toddlers and preschoolers with Down syndrome also show competent triadic relating in the forms of play acts, turn taking, invitations, and object shows [Mundy et al., 1988; Sigman and Ruskin, 1999].

In the context of these strengths in the development of social relatedness and other social–emotional skills, there is evidence that children with Down syndrome may show difficulties when cognitive demands in social decision making increase. While socialization skills remain a relative strength in middle childhood [Dykens et al., 1994], and many children with Down syndrome appear to be able to form reciprocal friendships with peers [Freeman and Kasari, 2002], there is mixed evidence regarding whether individuals with Down syndrome show impairments in the ability to perform more complex social cognition tasks [Baron-Cohen et al., 1985; Baron-Cohen, 1989; Yirmiya et al., 1996; Zelazo et al., 1996; Abbeduto et al., 2001]. Williams and Wishart [in press] identify other factors that may contribute to the difficulty that individuals with Down syndrome have on social–cognitive tasks, including executive function or memory difficulties. Nevertheless, it may be that despite competencies in the area of social relatedness, as the demands and complexities of social situations increase in middle childhood and beyond, individuals with Down syndrome may show difficulties with social adaptation and selecting appropriate social strategies, especially as they enter adolescence and face changes in emotional functioning and mood [Dykens et al., 2002; Fidler et al., 2005a].

This social profile is relevant for educational planning in several ways. First, these strengths may make it possible for children with Down syndrome to learn through social techniques such as modeling, peer collaboration, social groups [Loveras and Fornells, 1998; Rogoff, 2003]. Though there is a surprising lack of empirical exploration of the efficacy of these techniques in children with Down syndrome, it could be hypothesized that such techniques would capitalize on social motivation may be a useful reinforcer for children with Down syndrome. However, it is important to consider that the strong social motivation observed in this population may serve as a distractor as well [Wishart, 1996; Fidler, 2006; see discussion below]. There is also some evidence that, because of strengths in early social relatedness in this population, affective cues put forth by a teacher or interventionist can impact learning and motivation in a particularly pronounced way [Park et al., 2005]. It may also be important for educators to be mindful of potential changes in mood and social engagement as children with Down syndrome transition into adolescence, and perhaps adopt modified strategies as these behavioral changes become evident [Dykens et al., 2002].

**Personality–Motivation**

Another aspect of the behavioral profile in Down syndrome that may be educationally relevant relates to motivational orientation and task persistence [Gunn and Cuskelly, 1991]. In laboratory settings, when presented with tasks such as puzzles and other nonsocial/nonverbal tasks, children with Down syndrome have been shown to abandon the task sooner than other children at similar developmental levels, and to adopt strategies that divert attention away from the task [Landry and Chapieski, 1990; Pitcairn and Wishart, 1994; Ruskin et al., 1994; Vlachou and Farrell, 2000; Kasari and Freeman, 2001]. This, coupled with the strengths in aspects of social initiation described in the previous section, can lead to a style that involves an over-reliance on social strategies, especially in contexts that require instrumental thinking [Kasari and Freeman, 2001]. It may be that the interaction between emerging difficulties with instrumental thinking and strengths in social relatedness lead to a personality–motivation orientation that ultimately impacts ability of a child with Down syndrome to learn effectively [Wishart, 1996; Fidler, 2006].

A style that leads children to remove themselves from challenging situations in favor of social interaction may deprive children with Down syndrome of important opportunities to challenge themselves and gain new skills through active engagement with the environment that surrounds them. Awareness of this profile may be important for educators when selecting techniques for involving individuals with Down syndrome in classroom settings. It may be important for educators to identify situations when the child with Down syndrome may be recruiting social strategies when engaging with the task at hand is more appropriate [Fidler, 2006]. Educators might also manage behavior using social consequences as reinforcement, not as a distractor during tasks that might pose a cognitive challenge [Fidler, 2006].

**CHALLENGES IN LINKING RESEARCH TO PRACTICE**

**Are Behavioral Phenotypes Modifiable?**

Though many aspects of the behavioral phenotype in Down syndrome are potentially relevant for educators, there are several challenges that must be addressed as researchers aim to translate research findings into educational practice. The first challenge relates to the notion of genes and modifiability. There is a danger in discussing the notion of behavioral outcomes associated with genetic disorders in that genetic effects can cohere fixed, nonmodifiable pathways. What we now know about the mechanisms by which genes give rise to phenotypes, particularly behavioral phenotypes, indicates that we need not worry about this danger. First, from a neurodevelopmental perspective, at every step of the way, opportunities exist to modulate the translational process. In addition, all learning and education is rooted in the notion that neurophysiological changes can be observed in response to environmental input, leading the brain to undergo various types of reorganization [Nelson, 2000]. Nelson notes that it is commonly understood that "...the success of early childhood intervention strategies rests to a great degree on the relative plasticity of the human brain (p. 222)," and this applies to children with and without genetic disorders alike.

In addition, potential evidence of the modifiability of the Down syndrome phenotypic profile has been reported in a long-term study of British inclusion in this population. Buckley et al. [2006] report that the practice of including children with Down syndrome in mainstream classrooms in England has had an impact on the phenotypic profile in older children and adolescents with Down syndrome. They note that previous studies showed that children with Down syndrome who
attended school in special education settings demonstrated a profile of strengths in socialization and daily living skills, but deficits in adaptive communication abilities [Dykens et al., 1994; Fidler et al., 2006]. However, in their sample of children with Down syndrome who underwent schooling in inclusive settings, they found that the marked deficits in adaptive communication were not observable [Buckley et al., 2006]. They argue that the social challenges associated with being educated in an inclusive setting modified the phenotypic profile and narrowed the gap between areas of strength and challenge. The authors of this study note that this warrants replication, but if supported, there would be a critical means of modifying the profile of strengths and weaknesses associated with Down syndrome.

When considering whether educational approaches can modify phenotypic profiles, it is also important to note that the pattern of strengths and weaknesses associated with genetic disorders does not simply appear in a pronounced fashion in middle childhood. There is a developmental process that leads to the more pronounced end states of relative strengths and weaknesses in any genetic disorder. This is important to recognize because there may be opportunities to target early emerging phenotypic characteristics in very young children, before dissociations in profile become pronounced [see Karnoff-Smith, 1997, 1998; Fidler, 2005, 2006]. Fidler [2005] argued that for some aspects of the Down syndrome behavioral phenotype, it may be possible to identify early developmental precursors to later more pronounced outcomes. If these early developmental precursors can be identified and targeted with empirically validated intervention techniques, this too may be another means for altering the developmental pathway and the phenotypic profile associated with Down syndrome.

**What Constitutes Empirical Support for Etiology-Specific Education/Intervention?**

Another challenge in the attempt to bridge research and practice in this area relates to the empirical validation of techniques aimed at addressing phenotype-specific dimensions. There is a small, but growing literature that describes the efficacy of educational techniques such as computer-based learning [Lloyd et al., 2006; Ortega-Tudela and Gomez-Ariza, 2006], instructional approaches to reading and its component skills [Laws et al., 1996; Moni and Jobling, 2001; Cupples and Iacono, 2002; Kennedy and Flynn, 2003; van Bysterveldt et al., 2006], and math skills [Irwin, 1991; Nye and Buckley, 2006; Ortega-Tudela and Gomez-Ariza, 2006] for children with Down syndrome. While more studies of this kind are warranted, only a few of these studies show educational benefits when using one specific technique over another Cupples and Iacono, 2002).

The question remains whether this type of empirical validation is sufficient to warrant a syndrome-specific approach to educational planning. Some might argue that the efficacy of these techniques have little to do with the phenotypic profile associated with Down syndrome—rather they simply show that one technique is superior to another regardless of the population to which it is applied. By extension, it could be argued that in order to justify a syndrome-specific set of recommendations for educational practice, there must be a set of techniques that work differentially across populations. That is, there must be techniques identified that are effective for children with Down syndrome, but not effective for children who do not have Down syndrome or the developmental profile associated with Down syndrome [see Fidler et al., 2007 for a discussion]. At present, there are relatively few examples in the literature that demonstrate such differential effects [Fey et al., 2006; Yoder and Warren, 2002]. Those that do exist suggest that the personality-motivational orientation associated with Down syndrome may be particularly important to consider when selecting educational and intervention techniques [Yoder and Warren, 2002; see Fidler et al., 2007 for a discussion of this issue].

**Is Syndrome-Specific Education Feasible?**

A third challenge to the idea of linking phenotype research into practice relates to issues of classroom management and the training of teachers. While future research may show the benefits of etiology-specific instructional approaches, it could be argued that specific techniques for different children in the classroom would be too unwieldy and would require too great of a personnel demand. It could also be argued that the training of teachers in etiology-specific instructional approaches would make teacher education programs too lengthy of a process, requiring a mastery of approaches that target any number of the many syndromes and behavioral disorders present in the student population. While adopting an etiology-specific approach in the classroom will undoubtedly place additional demands on educators, some potential approaches involve less of a diversion of resources from the larger classroom culture than others. Techniques to be developed in the future can be imbedded in naturalistic ways, and might only involve subtle adjustments in teacher decision making and presentation of material. For example, supplementing instruction with supports that rely on a favored information processing modality might not be detectable to the larger classroom, and in some instances could potentially enhance instruction for children in the classroom without disabilities. In addition, while some additional training might be involved for teachers, these can come in the form of continuing educational trainings, or useful informational materials (websites, booklets) that need not burden a teacher in training. While the details of implementation would need to be addressed in a real-world process, it is likely that the implementation of some syndrome-specific instructional approaches, if they receive empirical validation, might not necessarily pose a prohibitively large challenge to educators.

**Future Directions**

Despite the many advances that have been made in the study of brain and behavioral development in Down syndrome, there is still a great deal of progress to be made both in the basic study of development in Down syndrome and in the application of these findings to practice. In terms of the potential contributions of the neurobiological approach, future work uncovering the neurobiological causes of the cognitive, language, and behavioral impairments associated with Down syndrome will ultimately lead to creation of an ever-more precise animal model of Down syndrome. A more precise animal model of Down syndrome could make it possible to develop biological interventions that might ultimately impact development in this population. Thus, advances in this area will rely on the close collaboration of behavioral scientists who are carefully delineating the nature of the Down syndrome behavioral phenotype, neurobiologists who are able to map these phenotypic outcomes onto brain anatomy and brain...
physiology in this population, and animal model researchers who can use this information to develop an ever more precise model of the disorder. This process will be aided by the neuropsychological approach, which offers the promise of identifying exactly those areas of disproportionate cognitive impairment that might guide the mapping from behavior to brain functioning.

Given the probabilistic nature of phenotypic outcomes in genetic disorders, these approaches may also make it possible to more deeply understand the nature of within syndrome variability in the population of individuals with Down syndrome. As researchers collaborate to uncover the pathway from gene to brain to behavior, it may be possible to identify with greater precision the sources of within-syndrome variability in outcomes of interest, and it may be possible to address the needs of children who show variations around the phenotypic profile that is associated with the larger group of individuals with Down syndrome. These advances offer the hope of even more targeted educational planning and the possibility of addressing the variability in outcomes that is classically associated with this population.

Another important future direction for research in Down syndrome relates to the importance of detecting emerging phenotypes in early childhood [Fidler, 2005]. This type of research transforms the view of outcomes in Down syndrome from a static, cross-sectional approach into a dynamic, longitudinal approach to studying this population. In taking this more dynamic view, it may be possible to identify the more subtle developmental precursors to more pronounced outcomes in later childhood and adolescence. These early precursors may serve as potentially useful targets for early intervention in this population. Rather than waiting to intervene once a split profile of strengths and weaknesses has become pronounced, educators and interventionists may be able to target those early precursors in their more subtle forms, which may set development on a more optimal pathway.

Finally, as the research community sorts through the various controversies related to syndrome-specific educational approaches, continued advances have still been made in the education of individuals with Down syndrome. These efforts continue to challenge the outmoded notions that children with genetic disorders such as Down syndrome have limited educational poten-

tial. Though the goal of bridging research and practice in the study of development in Down syndrome faces challenges—especially the difficulty of collaboration among scientists across neighboring fields—it is a goal that promises greater returns than simply educating children with Down syndrome according to their severity of impairment (mild, moderate, severe, profound intellectual disability), and ignoring the complex profile associated with the disorder. It is likely that our best hope for improving outcomes in genetic disorders such as Down syndrome lies in our ability to use all of the scientific information that is available, with developmentalists, education scientists, and brain experts collaborating to generate the most effective and innovative practice approaches possible.

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OF THE RECENT ADVANCES IN EDUCATION-RELATED RESEARCH IN DOWN SYNDROME, THE CHARACTERIZATION OF THE DOWN SYNDROME BEHAVIORAL PHENOTYPE HAS BECOME A POTENTIALLY CRITICAL TOOL FOR SHAPING EDUCATION AND INTERVENTION IN THIS POPULATION. THIS ARTICLE BRIEFLY REVIEWS THE LITERATURE ON BRAIN–BEHAVIOR CONNECTIONS IN DOWN SYNDROME AND IDENTIFIES ASPECTS OF THE DOWN SYNDROME BEHAVIORAL PHENOTYPE THAT ARE POTENTIALLY RELEVANT TO EDUCATORS. POTENTIAL CHALLENGES TO ETIOLOGICALLY INFORMED EDUCATIONAL PLANNING ARE DISCUSSED.

**Key Words:** Down syndrome; behavioral phenotypes; education; intervention

**As the most common chromosomal abnormality associated with intellectual disability, Down syndrome has been the focus of more behavioral and educational research than most other genetic disorders. These efforts have led to notable milestones in the advancement of education in Down syndrome, including cases in the literature of young adults with Down syndrome attending university courses [Hamill, 2003; Casale-Giannola and Wilson Kamens, 2006] and achievements in the area of educating children and adolescents in inclusive environments [Buckley et al., 2006]. The past five years alone have brought innovations that include teacher training interventions to shape attitudes toward inclusion in Down syndrome [Campbell et al., 2003], a refined understanding of effective inclusive practice in Down syndrome [Wolpert, 2001], and new instructional approaches involving computer technology [Lloyd et al., 2006; Ortega-Tudela and Gomez-Ariza, 2006].

However, the innovation that has the potential to have the greatest impact on educational practice in Down syndrome is the characterization of the Down syndrome “behavioral phenotype,” or the pattern of behavioral outcomes associated with this disorder throughout development. Research into the phenotypic outcomes associated with Down syndrome has led to a better understanding of the learning profile associated with this disorder, and has offered new information regarding the possible brain–behavior pathways leading to these outcomes. Over the past few decades, researchers have uncovered characteristic patterns of functioning in the areas of cognition, language development, social–emotional functioning, and personality–motivation [see Dykens et al., 2000; Rondal and Buckley, 2001; Fidler, 2005]. Though many questions regarding development in this population remain unanswered, researchers have gained a clearer understanding of the developmental trajectory associated with Down syndrome, and how this chromosomal abnormality impacts development in a dynamic and multisystemic way.

Amidst these advances in delineating the Down syndrome behavioral phenotype, there remains a wide gap between these research findings and the development of innovative practice [Hodapp and Fidler, 1999; Fidler et al., 2007]. While it has been argued that etiology-based information could be of importance for education in Down syndrome [Hodapp and Fidler, 1999; Freeman and Hodapp, 2000; Fidler, 2005], the use of these connections has not nearly met its full potential. It is true that relative to other disorders, greater research emphasis has been placed on identifying educational strategies that might improve outcomes in Down syndrome. Some of these recent approaches are informed by and specifically target aspects of the Down syndrome behavioral phenotype [Laws et al., 1996; Kennedy and Flynn, 2003; Iarocci et al., 2006; van Bysterveldt et al., 2006], and others do not [Garcia and Conte, 2004; Park et al., 2005; Trent et al., 2005]. Those existing syndrome-based recommendations may be potentially quite useful, but lack empirical validation [Alton, 1998; Fidler, 2005].

Though the gap between research findings and targeted practice in Down syndrome remains wide, there is evidence that educators and practitioners themselves recognize the importance of scientific progress in this area. Wolpert [2001] asked educators of children with Down syndrome in inclusive settings to identify factors that might improve the outcomes in the classroom. Among their answers was “... more informa-
tion on learning characteristics of children with Down syndrome” (p. 33). Similarly, parents seem to endorse etiology-specific modifications to their child's educational planning [Fidler et al., 2003]. As part of an effort to address the gap between research and practice in this area, this article will first review the current literature on the pathway from brain to behavior in Down syndrome. We then identify examples of phenotypic outcomes that may have educational relevance, with particular attention to the development of cognition and information processing in Down syndrome. Additional areas that may have educational relevance, including language development, social–emotional development, and personality–motivation, are briefly summarized. We conclude with a brief discussion of the potential challenges involved in shaping educational instruction in etiologically relevant ways. Although we start our review with discussion of neurobiology, we believe these kinds of data will be most relevant to the development of animal models, which then can be used to validate biological approaches to mitigation of the syndrome's impact on cognitive function. More important for educational practice will be an understanding of the phenotypic result of these neurobiological sequelae of trisomy 21, and we will accordingly focus on such behavioral approaches below.

THE NEUROBIOLOGY OF DOWN SYNDROME

The brain of an individual with Down syndrome at or shortly before birth is in many respects indistinguishable from the brain of a normal individual [Brooksbank et al., 1989; Wisniewski and Schmidt-Sidor 1989; Flórez et al., 1990; Schmidt-Sidor et al., 1990; Bar-Peled et al., 1991; Pazos et al., 1994]. Normal values have been reported for brain and skull shape, brain weight, proportion of specific cerebral lobes, size of cerebellum and brain stem, and the emergence of major neurotransmitter systems. There is evidence, however, that some changes begin to emerge as early as 22 weeks gestational age [e.g., Schmidt-Sidor et al., 1990; Golden and Hyman, 1994; Wisniewski and Kida, 1994; Engidawork and Lubec, 2003] and it is clear that by the age of 6 months a number of important differences are already obvious. Some of these differences are expressed in terms of the proportion of individuals with Down syndrome who show abnormal values, rather than in terms of a uniform abnormality in all instances. This is important as it highlights the variability in this population sharing the genotypic feature of trisomy 21.

While there is a postnatal delay in myelination [Wisniewski, 1990], it is worth noting that in all cases myelination is within normal range at birth, while in 75% of the cases it is within normal range throughout early development. Neuropathological differences after 3–5 months of age include a shortening of the fronto-occipital length of the brain that appears to result from a reduction in growth of the frontal lobes, a narrowing of the superior temporal gyrus (observed in about 35% of cases), a diminished size of the brain stem and cerebellum (observed in most cases), and a 20–50% reduction in the number of cortical granular neurons [see Crome et al., 1986; Bendix, 1971; Blackwood and Corsellis, 1970]. In sum, the overall picture at birth or shortly thereafter shows that individuals with Down syndrome tend to fall towards the bottom of the normal range (or outside it) on most measures.

Investigations of neural function, as opposed to structure, in early infancy suggested some abnormalities: there is evidence of either delayed or aberrant auditory system development [Jiang et al., 1990], which might contribute to the widespread hearing disorders observed in Down syndrome. Obviously, such a disorder, if organic, could be related to subsequent difficulties seen in the learning of language. Hill Karrer et al. [1998] have reported delayed development of cerebral inhibition using visual event-related potentials (ERP) in a visual recognition memory paradigm. Interestingly, the few studies of brain function in adolescents and young adults with Down syndrome and the existing data are somewhat equivocal. Pinter et al. [2001] used high-resolution MRI methods to analyze brain structure in 16 children (mean age 11.3 years) with Down syndrome. After correcting for overall brain volume, hippocampal, but not amygdala, volume reductions were seen in this group. This result confirms some earlier work using lower resolution MRI methods [Jernigan et al., 1993]. Kates et al. [2002] looked at a group of 12 children with Down syndrome (all males, mean age 5.94 years) and compared them with children with fragile-X, developmental language delay, or typical development. The children with Down syndrome had smaller brain volumes than any of the others, with previously unreported reductions in parietal cortex as well as the oft-reported reductions in the temporal lobe. Pinter et al. [2001], on the other hand, note the relative preservation of parietal cortex.

Overall, study of neuropathology points to problems in certain regions of the cortex, including most prominently the temporal lobe and the hippocampal formation [Wisniewski et al., 1986], the prefrontal cortex, and the cerebellum. This conclusion meshes well with what has been learned from the development of mouse models of the syndrome [e.g., Kleschevnikov et al., 2004]. These models have generally demonstrated selective impairments in the anatomy, physiology, pharmacology, and behavior, which are associated with the hippocampal formation, the prefrontal cortex, and the cerebellum.

These neurobiological findings in Down syndrome can inform education in several ways. First, as only modest abnormalities are detectable at birth, the role of development is critical in building into the pronounced profile of strengths and weaknesses observed in this population. This offers educators an important window of opportunity for intervention in the first few years of life, a point that will be revisited later. Second, the atypical development of specific brain structures suggests that some areas of functioning may become more impaired than others throughout development. In addition, early in development, many of the differences observed are expressed in terms of the proportion of individuals with Down syndrome who show abnormal values, rather than in terms of a uniform abnormality in all instances. This is in line with the probabilistic approach to understanding outcomes in individuals with genetic disorders [Dykens, 1995], where children may be at risk for certain neurobiological features, but not all children will show those specific abnormalities. These three themes are educationally relevant and will be explored in the following sections. Additional links between brain and behavior will be noted throughout the next section where relevant findings have been reported.

REVIEW OF EDUCATIONALLY-RELEVANT LITERATURE ON DEVELOPMENT IN DOWN SYNDROME

In this section, we explore the current literature on development in Down syndrome and highlight some of
the main findings that are potentially relevant to educators and interventionists. Before we examine the phenotypic profile associated with Down syndrome, there are two important issues that should be noted. First, these findings are part of a probabilistic approach to understanding the link between syndrome and outcome in behavioral phenotype research [Dykens, 1995]. Within this approach, it is understood that there is a higher probability that children with Down syndrome will show any of these specific outcomes relative to other children who do not have Down syndrome; however, not every child with Down syndrome will exhibit each of these phenotypic features [Dykens, 1995]. Thus, educators might use this information to adopt a “ready stance” regarding areas of potential strength and weakness in children with Down syndrome, but might expect that children will likely vary in the degree to which they exhibit any one of these predisposed outcomes.

Second, researchers are currently exploring the question of whether there is a subgroup of individuals with Down syndrome who meet criteria for autism spectrum disorder. Some studies suggest that a small percentage of individuals with Down syndrome have behavioral profiles consistent with autism [Collacott et al., 1992; Ghaziuddin et al., 1992; Kent et al., 1999; Paly and Hurley, 2002; Hepburn et al., in press]. These findings are not based on studies that have employed epidemiological methods for estimating the prevalence of comorbidity in this population, so they should be taken with a note of caution. However, it is possible that Down syndrome, while a diagnosis that may show a different behavioral profile than their counterparts with Down syndrome who do not have autism, and may require a different educational approach [Hepburn et al., in press].

COGNITION IN DOWN SYNDROME

Early Cognition

In line with the neurobiological findings described earlier, infants with Down syndrome show relatively normal abilities in learning and memory. This does not mean that either they, or indeed normally developing infants, have the full adult range of learning and memory abilities at birth. In fact this is not the case because some parts of the brain mature postnatally and the forms of learning and memory dependent on them are not available until some time after birth. The medial temporal lobe, and particularly, the hippocampus, as well as parts of the prefrontal cortex and cerebellum, are included in this category. The fact that these late-developing structures are apparently particularly at risk in Down syndrome is probably of considerable importance [see Nadel, 1986].

In an early series of studies, Ohr and Fagen [1991, 1993] reported that 3-month-old infants with Down syndrome were entirely normal in learning about the contingencies between their own movements (leg kicking) and reinforcement, including initial learning, acquisition speed, and retention. In a later report, Ohr and Fagen [1994] showed that 9-month-old infants with Down syndrome were impaired, as a group, in learning about the contingency between arm movements and reinforcement. However, they noted that some infants with Down syndrome were able to learn. They concluded that there is a relative decline in conditionability in infants with Down syndrome compared to normally developing infants after 6 months. Manganello et al. [1992] reported control infants and infants with Down syndrome on a variety of spatial tasks, one of which, a place-learning task, was designed especially to assess the state of function of the hippocampal system. The pattern of results was consistent with diffuse, but mild, neuropathology combined with much more extensive pathology localized to the hippocampus.

Children with Down syndrome have typically been shown to acquire basic object concepts more slowly than normally developing infants (e.g., Rinaldi and Meltzoff, 1995) but with extensive training they can acquire it at more-or-less the same time as normally developing infants [Wishart, 1993]. However, a different kind of problem emerges in this task situation: instability of acquisition. Although the typical subject with Down syndrome solved various levels of the tasks used to assess the object concept at ages not very far from the norm, performance after acquisition could be highly variable and apparently bested by motivational difficulties. These problems, if representative of the learning style of children with Down syndrome, are extremely important in thinking about effective intervention. The results of Wishart’s studies using standard intelligence test batteries suggest that they are indeed representative. Test–retest reliability was very low because successes gained in one test might not appear upon retest, as soon as 2 weeks later. New skills show up, only to disappear shortly thereafter. One could speculate that evidence of such “rapid forgetting” is consistent with damage in the hippocampal formation but considerably more data are required before this conclusion can be accepted. These developmental differences may have important implications for educators. For example, if the learning process involves more observable regressions for children with Down syndrome than for other children, it might be important for teachers and interventionists to account for these differences with more frequent reviews of materials, and it may be important to monitor the stability with which a child with Down syndrome has acquired a skill.

Information Processing

The learning and memory problems that begin to emerge in late infancy become considerably more noticeable as the infant grows to childhood and adolescence. These effects vary as a function of the type of memory being assessed. Explicit memory involves things like facts that subjects consciously recollect, whereas implicit memory can be demonstrated indirectly, without conscious recollection. This distinction has been shown to be important in understanding organic amnesia, since most amnesics are profoundly impaired on explicit memory tasks but can be relatively normal on implicit tasks. One common kind of implicit memory test looks at skills or procedures, such as mirror-tracing; another common implicit memory test involves “priming,” where prior exposure to a word or picture can influence subsequent performance on word-stem or partial–picture completion tasks even though the subjects might not recall having seen the relevant items before.

Carlesimo et al. [1997] examined the performance of children with Down syndrome in the area of “implicit” (procedural) and “explicit” (episodic) memory paradigms, including word-stem completion, list learning, and prose recall. Robust priming effects were seen in the Down syndrome group, comparable to those observed in controls, indicating that implicit memory was intact. However, deficits were observed in both explicit memory tasks. Performance on these kinds of explicit memory paradigms has been linked to functions
of the hippocampal system; hence, the defects suggest differential impairment in hippocampal function and thereby converge with the data from study of spatial cognition. This selective impairment of explicit, but not implicit, memory was also reported in Vicari [2001].

A great deal of research attention has been focused on deficits in the processing of verbal information in Down syndrome, and how these deficits contribute to poor language and learning outcomes [Byrne et al., 1995; Hesketh and Chapman, 1998; Laws, 1998]. Most commonly, the verbal working memory deficit in Down syndrome is measured by performance on an auditory digit span task. Poor auditory digit span performance findings in Down syndrome were first reported several decades ago [Bilvosky and Share, 1965; Rempel, 1974].

More recently, a series of studies by Hulme and Mackenzie [1992] described the development of poor verbal working memory in children with Down syndrome in great detail. They found that children in the Down syndrome group scored lower than the typical MA-matched controls on auditory digit span and mental arithmetic verbal working memory tasks were more correlated in the typically developing children than in the Down syndrome group. Similar deficits in verbal working memory are also observable when letters are substituted for numbers in the paradigm [Varnhagen et al., 1987]. This deficit is also not subject to changes when experimental design is manipulated in order to reduce unrelated demands on individuals with Down syndrome [Marcell and Weeks, 1988; Marcell et al., 1989; Laws, 1999].

Deficits in verbal working memory may relate to neuroanatomical characteristics associated with Down syndrome, including a proportionately smaller planum temporale, which is referred to as the auditory association cortex [Frangou et al., 1997]. It is important to note, however, that the relationship between the planum temporale volume deficit and cognitive-linguistic functioning in Down syndrome remains unclear [Frangou et al., 1997].

Though there is a great deal of evidence of verbal working memory deficits in Down syndrome, it is important to note that the main evidence for this dysfunction comes from studies that measure the processing of acoustically-presented information only. Auditory digit span, auditory sentence recall, and other commonly used techniques auditory nonword repetition all involve the processing of acoustic, rather than speech-based visually presented information. Thus, one might argue that the verbal working memory difficulty in Down syndrome has only been demonstrated in the maintenance of auditorially presented information, rather than the processing of all types of verbal information. The processing of speech-based visual information in Down syndrome has not yet been shown to be equally impaired, and importantly, may not be.

In contrast with their performance on verbal processing tasks, individuals with Down syndrome show relative strengths in visuospatial processing, and show a profile of higher visuospatial processing than verbal processing [Silverstein et al., 1982; Thase et al., 1984; Pueschel et al., 1987; Wang and Bellugi, 1994; Jarrold et al., 1999; Klein and Mervis, 1999]. The Corsi span, a block tapping memory task, is most often used as the test of visuospatial processing. In terms of raw scores, most individuals without Down syndrome (both with developmental delay and typically developing) tend to show higher raw auditory digit span recall scores than Corsi span recall scores. However, individuals with Down syndrome seem to perform equally well on these tests, or even show an advantage for Corsi span recall [Haxby, 1989; Azari et al., 1994; Wang and Bellugi, 1994; Vicari et al., 1995; Jarrold and Baddeley, 1997], especially when participants were not required to include order in their responses. These results have also been demonstrated with tasks other than the Corsi and auditory digit span [Pueschel et al., 1987; Hodapp et al., 1992; Bower and Hayes, 1994; Klein and Mervis, 1999]. The visuospatial working memory advantage is also demonstrated when identical stimulus information is simply presented either visually or auditorily [Varnhagen et al., 1987].

Neuroanatomical correlates for this relative strength in Down syndrome visuospatial processing have been posited. Pinter et al. [2001] reported what they call “striking preservation” of parietal and occipital cortical gray matter in an MRI study of 5–23-year-olds with Down syndrome. Studies have shown the importance of both parietal and occipital lobe functioning for some aspects of visuospatial processing [Black and Bernard, 1984; Jonides et al., 1993].

An information processing profile that includes strengths in visual processing and implicit memory, and deficits in verbal processing and explicit memory could potentially inform educational approaches to working with children with Down syndrome. Instruction that is verbally based—and especially auditorily mediated—might pose a greater challenge to children with Down syndrome than instruction that is presented with visual supports. Minor and subtle teaching modifications can be made to address this issue, without any noticeable disruption to a larger classroom environment. In addition, instruction that involves explicit memory, such as logic problems, may be presented with greater sensitivity given that this is an area of challenge as well. Awareness of this area of deficit might allow an educator to make informed decisions regarding prompts, wait time, and supports for a child who may have particular difficulty with this type of information processing.

**Reading**
The cognitive underpinnings of reading are of particular relevance for education in Down syndrome. Despite the impairments observed in other areas of cognition, many individuals with Down syndrome are able to show competence in some aspects of reading development. In particular, word identification appears to be an area of relative strength within reading skills in this population, while word attack and reading comprehension appear to be more impaired [Byrne et al., 1995; Cupples and Iacono, 2000; Kay-Raining Bird et al., 2000]. Strengths in word identification have been linked to relative strengths in visual processing [Fidler et al., 2005b], while deficits in word attack skills have been linked to difficulties with verbal processing skills in Down syndrome [Boudreau, 2002].

There is some controversy regarding the implications of this profile for reading instruction in Down syndrome [see Hodapp and Freeman, 2003 for a summary], with some arguing that visually-based approaches might be warranted [Buckley and Bird, 2002], and others emphasizing the role of phonological processing in reading in this population, despite impairments [Cupples and Iacono, 2002]. Regardless of this debate, there is evidence that children with Down syndrome recruit both their visual and verbal processing skills when reading, particularly when identifying words [Cupples and Iacono, 2000; Fidler et al., 2005b].
BRAIN–BEHAVIOR CONNECTIONS

In a series of recent studies, Pennington, Nadel and colleagues have tested several different groups of individuals with Down syndrome on a range of tasks designed to directly assess the function of specific brain systems. This “cognitive neuropsychological” approach often uses tasks first developed in animal models, where the critical underlying brain circuits can be identified and carefully studied in invasive experiments. The team started with a focus on three brain systems identified by the neuropathological data, much of which was discussed above: the hippocampal system, the prefrontal cortex, and the cerebellum. They developed a set of tasks that could, collectively, tell us something about how these brain systems are faring. In the first set of studies, Pennington et al. [2003] found evidence of specific hippocampal dysfunction in our sample of 28 adolescents, using mental age matched controls.

Little evidence of prefrontal dysfunction was observed in a battery of nonverbal tasks. Subsequent pilot work, however, suggested that verbal tasks might yield a different result, and indeed that is what is being observed (Moon et al., unpublished data). Using verbal tasks to explore the prefrontal cortex, Moon et al. found in the young (aged 5–11) and old (aged 30–41) groups strong signs of dysfunction in both the hippocampal and prefrontal systems. Deficits were observed in a range of tasks although verbal mediation was necessary to bring out the prefrontal effect. Taken as a whole, these studies show that particular problems emerge in the memory domains served by the hippocampal system and the prefrontal system. The latter impairment appears to be linked to the use of verbal test materials. The impairment in hippocampal function could in principle reflect problems in any of the structures of the hippocampal region; a recent study of two neuropsychological paradigms dependent on parahippocampal and perirhinal regions (delayed non-matching to sample and visual paired comparison), however, suggests that these areas are functioning appropriately, and that the impairment is more likely to reflect improper development of the hippocampus itself [Dawson et al., 2001].

The prefrontal cortex, as noted already, plays an important role in a wide range of functions, including episodic/explicit memory and working memory. As noted earlier, explicit memory is impaired in individuals with Down syndrome. There has been extensive research on working memory in this population, and clear deficits have been observed in a number of studies [Varnhagen et al., 1987; Marcell and Weeks, 1988; Laws, 1998; Jarrold et al., 1999]. However, this impairment seems to be limited to verbal information, as impairments are minimal in visuospatial domains. The deficit appears to be neither a motor nor articulatory problem [Kanno and Ikeda, 2002] and may relate to the so-called phonological loop [Jarrold and Baddeley, 2001; Laws, 2002].

OTHER AREAS OF EDUCATIONAL RELEVANCE

In addition to cognitive development and information processing, there are other areas of development in Down syndrome that may be relevant to decisions made by educators. Brief descriptions of findings in the area of language development, social–emotional functioning, and personality–motivation are presented in this section.

Language Development

Individuals with Down syndrome generally show a profile of stronger receptive language skills and weaker expressive language skills. This profile seems to emerge in early childhood and become more pronounced as children progress into middle childhood and beyond [Miller, 1999]. Expressive language deficits are often manifested in terms of morphosyntactic delays [Chapman et al., 1998; Edie et al., 2002] and difficulties with speech intelligibility [Nicholls et al., 1999; Stoeck-Gammon, 2003; Kumin, 2006]. And while receptive language is stronger than expressive language in most individuals with Down syndrome, some areas such as receptive syntax, are more compromised than others [Abbeduto et al., 2003]. In contrast, some areas of pragmatic functioning seem to be an area of relative strength in individuals with Down syndrome [Johnston and Stansfield, 1997; Laws and Bishop, 2004].

This language profile is potentially relevant for educational planning in several ways. First, because many children with Down syndrome have stronger receptive than expressive language skills, they often understand much more language than they can produce. To an uninformed educator who is not aware of the discrepancy between expressive and receptive language, it might be natural to address the child with input language and instruction at the level of their expressive language. But such an approach would likely involve a marked underestimation of the child’s academic and receptive abilities. When considering how best to present class material, it may be important for educators to identify and target the receptive language level of a child with Down syndrome so as to appropriately challenge them and engage them at their true level of understanding [Roberts et al., 2007].

In addition, it may be useful for educators to be aware that difficulties with expressive language— and speech intelligibility in particular—may be frustrating for children with Down syndrome in classroom contexts. Morphosyntactic difficulties demand extra motivation from children with Down syndrome to produce lengthier utterances [Miller and Leddy, 1999], and intelligibility problems may lead to situations where a child must repeat herself in order to be understood. Thus, educators may want to consider both the social and motivational consequences of expressive language difficulties. It may be beneficial to identify ways to minimize the potential for negative experiences, while allowing the child with Down syndrome to benefit from the opportunity to build their speech, language, and communication skills.

Social–Emotional Functioning

The majority of individuals with Down syndrome show strengths in various aspects of social–emotional functioning, exhibiting behaviors that suggest evidence of intact social relatedness and some measure of social competence in early childhood [Fidler et al., 2006]. In the first few years of life, markers of primary intersubjectivity, the earliest forms of dyadic social relating based on emotional displays and reciprocal signaling, are identified as emerging in a delayed, but complete manner in this population [see Fidler, 2006 for a review]. In particular, young children with Down syndrome show evidence of primary intersubjective development in the form of increased eye gaze and vocalizations directed to other people [Gunn et al., 1982; Crown et al., 1992; Legerstee et al., 1992; Kasari et al., 1995], increased direction of positive facial displays in the form of smiles [Kasari and Freeman, 1990; Fidler et al., 2005a].

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In addition, there is also support for the notion that many aspects of secondary intersubjectivity, the ability to engage with a social partner in a triadic fashion, also emerge with competence, albeit in a delayed fashion. Several studies suggest that children with Down syndrome show either MA-appropriate or even increased levels of joint attention relative to other children without Down syndrome [Mundy et al., 1988; Kasari et al., 1995; Fidler et al., 2005]. Toddlers and preschoolers with Down syndrome also show competent triadic relating in the forms of play acts, turn taking, invitations, and object shows [Mundy et al., 1988; Sigman and Rusklin, 1999].

In the context of these strengths in the development of social relatedness and other social–emotional skills, there is evidence that children with Down syndrome may show difficulties when cognitive demands in social decision making increase. While socialization skills remain a relative strength in middle childhood [Dykens et al., 1994], and many children with Down syndrome appear to be able to form reciprocal friendships with peers [Freeman and Kasari, 2002], there is mixed evidence regarding whether individuals with Down syndrome show impairments in the ability perform more complex social cognition tasks [Baron-Cohen et al., 1985; Baron-Cohen, 1989; Yirmiya et al., 1996; Zelazo et al., 1996; Abbeduto et al., 2001]. Williams and Wishart [in press] identify other factors that may contribute to the difficulty that individuals with Down syndrome have on social–cognitive tasks, including executive function or memory difficulties. Nevertheless, it may be that despite competence in social relatedness, as the demands and complexities of social situations increase in middle childhood and beyond, individuals with Down syndrome may show difficulties with social adaptation and selecting appropriate social strategies, especially as they enter adolescence and face changes in emotional functioning and mood [Dykens et al., 2002; Fidler et al., 2005a].

This social profile is relevant for educational planning in several ways. First, these strengths may make it possible for children with Down syndrome to learn through social techniques such as modeling, peer collaboration, and social groups [Lloveras and Fornells, 1998; Rogoff, 2003]. Though there is a surprising lack of empirical exploration of the efficacy of these techniques in children with Down syndrome, it could be hypothesized that such techniques would capitalize on social motivation may be a useful reinforcer for children with Down syndrome. However, it is important to consider that the strong social motivation observed in this population may serve as a distractor as well [Wishart, 1996; Fidler, 2006; see discussion below]. There is also some evidence that, because of strengths in early social relatedness in this population, affective cues put forth by a teacher or interventionist can impact learning and motivation in a particularly pronounced way [Park et al., 2005]. It may also be important for educators to be mindful of potential changes in mood and social engagement as children with Down syndrome transition into adolescence, and perhaps adopt modified strategies as these behavioral changes become evident [Dykens et al., 2002].

**Personality-Motivation**

Another aspect of the behavioral profile in Down syndrome that may be educationally relevant relates to motivational orientation and task persistence [Gunn and Cuskelly, 1991]. In laboratory settings, when presented with tasks such as puzzles and other nonsocial/nonverbal tasks, children with Down syndrome have been shown to abandon the task sooner than other children at similar developmental levels, and to adopt strategies that divert attention away from the task [Landry and Chapleski, 1990; Pitcairn and Wishart, 1994; Ruskin et al., 1994; Vlachou and Farrell, 2000; Kasari and Freeman, 2001]. This, coupled with the strengths in aspects of social initiation described in the previous section, can lead to a style that involves an over-reliance on social strategies, especially in contexts that require instrumental thinking [Kasari and Freeman, 2001]. It may be that the interaction between emerging difficulties with instrumental thinking and strengths in social relatedness lead to a personality-motivation orientation that negatively impacts the ability of a child with Down syndrome to learn effectively [Wishart, 1996; Fidler, 2006].

A style that leads children to remove themselves from challenging situations in favor of social interaction may deprive children with Down syndrome of important opportunities to challenge themselves and gain new skills through active engagement with the environment that surrounds them. Awareness of this profile may be important for educators when selecting techniques for involving individuals with Down syndrome in classroom settings. It may be important for educators to identify situations when the child with Down syndrome may be recruiting social strategies when engaging with the task at hand is more appropriate [Fidler, 2006]. Educators might also manage behavior using social consequences as reinforcement, not as a distractor during tasks that might pose a cognitive challenge [Fidler, 2006].

**CHALLENGES IN LINKING RESEARCH TO PRACTICE**

**Are Behavioral Phenotypes Modifiable?**

Though many aspects of the behavioral phenotype in Down syndrome are potentially relevant for educators, there are several challenges that must be addressed as researchers aim to translate research findings into educational practice. The first challenge relates to the notion of genes and modifiability. There is a danger in discussing the notion of behavioral outcomes associated with genetic disorders in that genetic effects can confound fixed, nonmodifiable pathways. What we now know about the mechanisms by which genes give rise to phenotypes, particularly behavioral phenotypes, indicates that we need not worry about this danger. First, from a neurodevelopmental perspective, at every step of the way, opportunities exist to modulate the translational process. In addition, all learning and education is rooted in the notion that neurophysiological changes can be observed in response to environmental input, leading the brain to undergo various types of reorganization [Nelson, 2000]. Nelson notes that it is commonly understood that "... the success of early childhood intervention strategies rests to a great degree on the relative plasticity of the human brain (p. 222)," and this applies to children with and without genetic disorders alike.

In addition, potential evidence of the modifiability of the Down syndrome phenotypic profile has been reported in a long-term study of British inclusion in this population. Buckley et al. [2006] report that the practice of including children with Down syndrome in mainstream classrooms in England has had an impact on the phenotypic profile in older children and adolescents with Down syndrome. They note that previous studies showed that children with Down syndrome who
attended school in special education settings demonstrated a profile of strengths in socialization and daily living skills, but deficits in adaptive communication abilities [Dykens et al., 1994; Fidler et al., 2006]. However, in their sample of children with Down syndrome who underwent schooling in inclusive settings, they found that the marked deficits in adaptive communication were not observable [Buckley et al., 2006]. They argue that the social challenges associated with being educated in an inclusive setting modified the phenotypic profile and narrowed the gap between areas of strength and challenge. The authors of this study note that this warrants replication, but if supported, there would be a critical means of modifying the profile of strengths and weaknesses associated with Down syndrome.

When considering whether educational approaches can modify phenotypic profiles, it is also important to note that the pattern of strengths and weaknesses associated with genetic disorders does not simply appear in a pronounced fashion in middle childhood. There is a developmental process that leads to the more pronounced end states of relative strengths and weaknesses in any genetic disorder. This is important to recognize because there may be opportunities to target early emerging phenotypic characteristics in very young children, before dissociations in profile become pronounced [see Karnoff-Smith, 1997, 1998; Fidler, 2005, 2006]. Fidler [2005] argued that for some aspects of the Down syndrome behavioral phenotype, it may be possible to identify early developmental precursors to later more pronounced outcomes. If these early developmental precursors cannot be identified and targeted with empirically validated intervention techniques, this too may be another means for altering the developmental pathway and the phenotypic profile associated with Down syndrome.

**What Constitutes Empirical Support for Etiology-Specific Education/Intervention?**

Another challenge in the attempt to bridge research and practice in this area relates to the empirical validation of techniques aimed at addressing phenotype-specific dimensions. There is a small but growing literature that describes the efficacy of educational techniques such as computer-based learning (Lloyd et al., 2006; Ortega-Tudela and Gomez-Ariza, 2006), instructional approaches to reading and its component skills [Laws et al., 1996; Moni and Jobling, 2001; Cupples and Iacono, 2002; Kennedy and Flynn, 2003; van Bysterveldt et al., 2006], and math skills [Irwin, 1991; Nye and Buckley, 2006; Ortega-Tudela and Gomez-Ariza, 2006] for children with Down syndrome. While more studies of this kind are warranted, only a few of these studies show educational benefits when using one specific technique over another Cupples and Iacono, 2002].

The question remains whether this type of empirical validation is sufficient to warrant a syndrome-specific approach to educational planning. Some might argue that the efficacy of these techniques have little to do with the phenotypic profile associated with Down syndrome—rather they simply show that one technique is superior to another regardless of the population to which it is applied. By extension, it could be argued that in order to justify a syndrome-specific set of recommendations for educational practice, there must be a set of techniques that work differentially across populations. That is, there must be techniques identified that are effective for children with Down syndrome, but not effective for children who do not have Down syndrome or the developmental profile associated with Down syndrome [see Fidler et al., 2007 for a discussion]. At present, there are relatively few examples in the literature that demonstrate such differential effects [Fey et al., 2006; Yoder and Warren, 2002]. Those that do exist suggest that the personality-motivational orientation associated with Down syndrome may be particularly important to consider when selecting educational and intervention techniques [Yoder and Warren, 2002; see Fidler et al., 2007 for a discussion of this issue].

**Is Syndrome-Specific Education Feasible?**

A third challenge to the idea of linking phenotype research into practice relates to issues of classroom management and the training of teachers. While future research may show the benefits of etiology-specific instructional approaches, it could be argued that specific techniques for different children in the classroom would be too unwieldy and would require too great of a personnel demand. It could also be argued that the training of teachers in etiology-specific instructional approaches would make teacher education programs too lengthy of a process, requiring a mastery of approaches that target any number of the many syndromes and behavioral disorders present in the student population. While adopting an etiology-specific approach in the classroom will undoubtedly place additional demands on educators, some potential approaches involve less of a diversion of resources from the larger classroom culture than others. Techniques to be developed in the future can be imbedded in naturalistic ways, and might only involve subtle adjustments in teacher decision making and presentation of material. For example, supplementing instruction with supports that rely on a favored information processing modality might not be detectable to the larger classroom, and in some instances could potentially enhance instruction for children in the classroom without disabilities. In addition, while some additional training might be involved for teachers, these can come in the form of continuing educational trainings, or useful informational materials (websites, booklets) that need not burden a teacher in training. While the details of implementation would need to be addressed in a real-world process, it is likely that the implementation of some syndrome-specific instructional approaches, if they receive empirical validation, might not necessarily pose a prohibitively large challenge to educators.

**Future Directions**

Despite the many advances that have been made in the study of brain and behavioral development in Down syndrome, there is still a great deal of progress to be made both in the basic study of development in Down syndrome and in the application of these findings to practice. In terms of the potential contributions of the neurobiological approach, future work uncovering the neurobiological causes of the cognitive, language, and behavioral impairments associated with Down syndrome will ultimately lead to creation of a-ever-more precise animal model of Down syndrome. A more precise animal model of Down syndrome could make it possible to develop biological interventions that might ultimately impact development in this population. Thus, advances in this area will rely on the close collaboration of behavioral scientists who are carefully delineating the nature of the Down syndrome behavioral phenotype, neurobiologists who are able to map these phenotypic outcomes onto brain anatomy and brain...
physiology in this population, and animal model researchers who can use this information to develop an even more precise model of the disorder. This process will be aided by the neuropsychological approach, which offers the promise of identifying exactly those areas of disproportionate cognitive impairment that might guide the mapping from behavior to brain functioning.

Given the probabilistic nature of phenotypic outcomes in genetic disorders, these approaches may also make it possible to more deeply understand the nature of within syndrome variability in the population of individuals with Down syndrome. As researchers collaborate to uncover the pathway from gene to brain to behavior, it may be possible to identify with greater precision the sources of within-syndrome variability in outcomes of interest, and it may be possible to address the needs of children who show variations around the phenotypic profile that is associated with the larger group of individuals with Down syndrome. These advances offer the hope of even more targeted educational planning and the possibility of addressing the variability in outcomes that is classically associated with this population.

Another important future direction for research in Down syndrome relates to the importance of detecting emerging phenotypes in early childhood [Fidler, 2005]. This type of research transforms the view of outcomes in Down syndrome from a static, cross-sectional approach into a dynamic, longitudinal approach to studying this population. By taking this more dynamic view, it may be possible to identify the more subtle developmental precursors to more pronounced outcomes in later childhood and adolescence. These early precursors may serve as potentially useful targets for early intervention in this population. Rather than waiting to intervene once a split profile of strengths and weaknesses has become pronounced, educators and interventionists may be able to target these early precursors in their more subtle forms, which may set development on a more optimal pathway.

Finally, as the research community sorts through the various controversies related to syndrome-specific educational approaches, continued advances have still been made in the education of individuals with Down syndrome. These efforts continue to challenge the outmoded notions that children with genetic disorders such as Down syndrome have limited educational poten-

tial. Though the goal of bridging research and practice in the study of development in Down syndrome faces challenges—especially the difficulty of collaboration among scientists across neighboring fields—it is a goal that promises greater returns than simply educating children with Down syndrome according to their severity of impairment (mild, moderate, severe, profound intellectual disability) and ignoring the complex profile associated with the disorder. It is likely that our best hope for improving outcomes in genetic disorders such as Down syndrome lies in our ability to use all of the scientific information that is available, with developmentalists, education scientists, and brain experts collaborating to generate the most effective and innovative practice approaches possible.

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Reading, dyslexia and the brain

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(Received 17 August 2007; final version received 8 November 2007)

Background: Neuroimaging offers unique opportunities for understanding the acquisition of reading by children and for unravelling the mystery of developmental dyslexia. Here, I provide a selective overview of recent neuroimaging studies, drawing out implications for education and the teaching of reading.

Purpose: The different neuroimaging technologies available offer complementary techniques for revealing the biological basis of reading and dyslexia. Functional magnetic resonance imaging (fMRI) is most suited to localisation of function, and hence to investigating the neural networks that underpin efficient (or inefficient) reading. Electroencephalography (EEG) is sensitive to millisecond differences in timing, hence it is suited to studying the time course of processing; for example, it can reveal when networks relevant to phonology versus semantics are activated. Magnetic source imaging (MSI) gives information about both location in the brain and the time course of activation. I illustrate how each technology is most suited to answering particular questions about the core neural systems for reading, and how these systems interact, and what might go wrong in the dyslexic brain.

Design and methods: Following a brief overview of behavioural studies of reading acquisition in different languages, selected neuroimaging studies of typical development are discussed and analysed. Those studies including the widest age ranges of children have been selected. Neuroimaging studies of developmental dyslexia are then reviewed, focusing on (a) the neural networks recruited for reading, (b) the time course of neural activation and (c) the neural effects of remediation. Representative studies using the different methodologies are selected. It is shown that studies converge in showing that the dyslexic brain is characterised by under-activation of the key neural networks for reading.

Conclusions: Different neuroimaging methods can contribute different kinds of data relevant to key questions in education. The most informative studies with respect to causation will be longitudinal prospective studies, which are currently rare.

Keywords: reading; phonology; dyslexia; brain imaging

How universal are the neural demands made by learning to read in different languages? What are the core neural systems involved, and what goes wrong in the dyslexic brain? Current neuroimaging technologies are able to throw light on research questions such as these, as will be illustrated below. In some instances, neuroimaging technologies can contribute unique information that behavioural methodologies are simply unable to provide. This includes information about the time processes in reading, and information about the parts of the brain that are affected by remedial packages for developmental

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ISSN 0013-1881 print ISSN 1469-5847 online
© 2008 NFER
DOI: 10.1080/00131880802082625
http://www.informaworld.com
dyslexia. Some neuroimaging methodologies can gather data without requiring overt attention on the part of the child. These methodologies are particularly powerful for contributing to our understanding of the biological basis of developmental dyslexia.

The history of research on developmental dyslexia has been dominated by visual theories of the disorder, ever since Hinshelwood (1896) described it as ‘congenital word blindness’. Historically, theories of reading development also assumed that visual processing was core to individual differences in the acquisition of reading. In the 1970s, for example, there was much discussion of ‘Phonecian’ versus ‘Chinese’ reading acquisition strategies. It was assumed that children who were learning to read character-based orthographies like Chinese required excellent visual memory skills in order to distinguish between the visually complex characters that represented spoken words. Hence visual memory or ‘logographic’ strategies were assumed core to reading acquisition of languages like Chinese and Japanese. Children who were learning to read languages like Greek or Italian, which were alphabetic and transparent (each letter corresponding to one, and only one, sound) appeared to require code-breaking skills. It was assumed that once the brain had learned the symbol–sound code, reading should be largely a process of phonological assembly. Many experiments were conducted with children learning to read in English, to compare the contribution of ‘Chinese’ versus ‘Phonecian’ acquisition strategies (e.g., Baron 1979). Dual-route models of reading, originally developed using data from adults, were applied to children who were learning to read (Stuart and Coltheart 1988). It was assumed that, developmentally, children could choose to learn to read by either Chinese or Phonecian strategies.

These ideas about individual differences have not gone away (e.g., Stein and Walsh 1997; Stuart 2006), but they are looking increasingly dated with the advent of brain imaging. Neuroimaging has also shed light on the processes underpinning the development of reading in deaf children, whom it was once assumed had no choice but to rely on visual memorisation strategies (e.g., Conrad 1979). Essentially, neuroscience is showing that despite the apparently different demands on the brain made by learning to read English, Greek or Chinese, and the apparently different processing strategies used by children who are deaf or who are dyslexic, reading across orthographies depends on the adequate functioning of the phonological system. Even for languages like Chinese, which would appear reliant on visual processing, it is oral language skills that underpin the acquisition of reading.

As I show in this review, brain imaging studies demonstrate that reading begins primarily as a phonological process. In the early phases of learning to read, it is the neural structures for spoken language that are particularly active. As reading expertise develops, an area in the visual cortex originally named the ‘visual word form area’ (VWFA) becomes increasingly active (Cohen and Dehaene 2004). This area is not a logographic system, even though it is very close to the visual areas that are active during picture naming. The VWFA is also active during nonsense word reading, and as nonsense words do not have word forms in the mental lexicon, the VWFA is thought to store orthography–phonology connections at different grain sizes (Goswami and Ziegler 2006a). Deaf readers rely on the same phonological system for reading as everyone else (MacSweeney et al. 2005). Children with developmental dyslexia show selective under-activation of key phonological areas of the brain, but targeted phonology-based interventions improve levels of activation in these areas, ‘normalising’ neural activity (Simos et al. 2002).
Learning to read: behavioural data

Many behavioural studies in developmental psychology show the critical role of ‘phonological awareness’ in learning to read (for a recent review see Ziegler and Goswami 2005). Phonological awareness is thought to develop via language acquisition. Between the ages of 1 and 6 years, children acquire words at an exponential rate. For example, the average 1-year-old might have a productive vocabulary of around 50–100 words, but by the age of 6 the average child’s receptive vocabulary will contain around 14,000 words (Dollaghan 1994). In order for the brain to represent each word as a distinct and unique sequence of sounds, each entry in the ‘mental lexicon’ must incorporate phonological information along with information about meaning. For example, there must be implicit knowledge of the sounds that comprise a particular word, and the order in which they occur. Phonological awareness is essentially the child’s ability to reflect on this implicit knowledge, and to make judgements based on it. Hence phonological awareness is typically measured by a child’s ability to detect and manipulate component sounds in words, for example, by deciding whether words rhyme, or by removing the initial sound from a spoken word.

The syllable is the primary processing unit across the world’s languages (Port 2006). In fact, there is an apparently language-universal sequence in the development of phonological awareness, from syllable awareness, through ‘onset-rime’ awareness to ‘phoneme’ awareness. Syllables (‘university’ has five syllables, ‘coffee’ has two syllables) can be segmented into sub-parts called onsets and rimes. The onset is the sound or sounds before the vowel, such as the ‘spr’ sound in ‘spring’ and the ‘st’ sound in ‘sting’. The rime is the vowel and any subsequent sounds in the syllable, such as the ‘ing’ sound in ‘spring’ and ‘string’. The phoneme is the smallest unit of sound that changes meaning. ‘Spring’ and ‘string’ differ in meaning because the second sound is different in each word (‘p’ versus ‘t’ respectively). In many of the world’s languages, onsets and rimes are the same as phonemes. This is because the dominant syllable structure across the world’s languages is consonant–vowel (CV). Relatively few words in English are CV syllables (5% of English monosyllables follow a CV structure: see De Cara and Goswami 2002). Examples of English words comprised of CV syllables are ‘go’, ‘do’ and ‘yoyo’.

Behavioural studies across languages have shown that phonological sensitivity at all three linguistic levels (syllable, onset-rime, phoneme) predicts the acquisition of reading (for a review see Ziegler and Goswami 2005). Furthermore, it has been demonstrated that training phonological awareness has positive effects on reading acquisition across languages, particularly when it is combined with training about how letters or letter sequences correspond to sounds in that language (e.g., Bradley and Bryant 1983; Schneider, Roth, and Ennemoser 2000). Children with developmental dyslexia across languages appear to have specific problems in detecting and manipulating component sounds in words (called a ‘phonological deficit’: see, e.g., Snowling 2000). For example, they find it difficult to count the number of syllables in different words, to recognise rhymes, to distinguish shared phonemes and to delete phonemes or substitute one phoneme for another (Korean: Kim and Davis 2004; German: Wimmer 1996; Greek: Porpodas 1999; Hebrew: Share and Levin 1999; for a comprehensive review see Ziegler and Goswami 2005). Dyslexic children are developing some awareness of phonology, but this is a slow and effortful process. Deaf children also develop phonological codes, for example, via lip reading (‘speech reading’) and vibrational cues. This is the case even if signing is their native language. Phonology is essentially the smallest contrastive
units of a language that create new meanings. In signed languages, phonology depends on visual/manual elements, with handshapes, movements and locations combined to form signs (Sandler and Lillo-Martin 2006). For deaf children too, individual differences in phonological awareness are related to reading acquisition (e.g., Harris and Beech 1998).

Reading and learning to read: neuroimaging data

To date, most neuroimaging studies of reading have been conducted with adults (see Price and McCrory 2005 for a recent synthesis). This was partly due to the methodologies available. The most popular methods for studying the brain during the act of reading depended on imaging techniques like functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). The fMRI technique measures changes in blood flow in the brain, which take approximately 6–8 seconds to reach a maximum value (i.e., maximum activity will be measurable 6–8 seconds after reading a particular word). fMRI works by measuring the magnetic resonance signal generated by the protons of water molecules in brain cells, generating a BOLD (blood oxygenation level dependent) response. The fMRI method is excellent for the localisation of function, but because changes in brain activity are summated over time, it cannot provide information about the sequence in which different neural networks become engaged during the act of reading. In PET, radioactive tracers are injected into the bloodstream and provide an index of brain metabolism. Because of the use of radioactive tracers, PET is not suitable for studying children.

More recently, the value of the electroencephalogram (EEG) methodology for studying reading is being recognised. Neurons communicate on a millisecond scale, with the earliest stages of cognitive information processing beginning between 100 ms and 200 ms after stimulus presentation. EEG methods can measure the low-voltage changes caused by the electro-chemical activity of brain cells, thereby reflecting the direct electrical activity of neurons at the time of stimulation (e.g., at the time of seeing a word). Initially, EEG methods were less widely used in the neuroscience of reading, because it is difficult to localise function using EEG. However, developmentally, information about the time course of processing is very important. Data from EEG studies suggest that the brain has decided whether it is reading a real word or a nonsense word within 160–180 ms of presentation, for children and adults across languages (Csepe and Szucs 2003; Suaseng, Bergmann, and Wimmer 2004).

Adult studies of reading based on PET and fMRI have focused on a relatively small range of reading and reading-related tasks, and studies of children using fMRI have followed suit. Typical tasks include asking participants to read single words and then comparing brain activation to a resting condition with the eyes closed; asking participants to pick out target visual features while reading print or ‘false font’ (false font is made up of meaningless symbols matched to letters for visual features like the ‘ascenders’ in the letters b, d, k); making phonological judgements while reading words or nonsense words (e.g., ‘do these items rhyme?’: leat, jete) and making lexical decisions (e.g., pressing a button when a word is presented, and a different button when a nonsense word is presented). Adult experiments show a very consistent picture concerning the neural networks that underpin skilled reading (e.g., Price et al. 2003; Rumsey et al. 1997; see for comments on divergence Price and McCrory 2005). Word recognition in skilled readers appears to depend on a left-lateralised network of frontal, temporoparietal and occipitotemporal regions, whatever language they are reading (see Figure 1). However,
there is some additional recruitment of visuo-spatial areas for languages with non-alphabetic orthographies (e.g., left middle frontal gyrus for Chinese: see meta-analysis by Tan et al. 2005). The frontal, temporoparietal and occipitotemporal regions essentially comprise the language, auditory, cross-modal and visual areas of the brain. At a very simple level, semantic and memory processing is thought to occur in temporal and frontal areas, auditory and phonological processing in temporal areas, articulation in frontal areas, visual processing in occipital areas and cross-modal processing in parietal areas.

Although there are still relatively few neuroimaging studies of children reading, the studies that have been done show a high degree of consistency in the neural networks recruited by novice and expert readers. For example, work by Turkeltaub and colleagues has used fMRI and the false font task to compare neural activation in English-speaking participants aged from 7 to 22 years (Turkeltaub et al. 2003). Importantly, 7-year-olds can perform the ‘false font’ task as well as adults, hence changes in reading-related neural activity are likely to reflect developmental differences rather than differences in reading expertise. Turkeltaub et al. (2003) reported that adults performing their task activated the usual left hemisphere sites, including left

AG = Angular Gyrus
OT = Occipito-Temporal
pTP = posterior Temporo-Parietal
pST = posterior Superior Temporal
pIT = posterior Inferior Temporal
pMT = posterior Middle Temporal
aTP = anterior Temporo-Parietal
aT = anterior Temporal
cST = central Superior Temporal
IF = Inferior Frontal
Pc = Precentral gyrus
SM = Sensori-motor cortex

Figure 1. A schematic depiction of some of the neural areas involved in reading (left hemisphere depiction) (from Price and McCrory 2005).
posterior temporal and left inferior frontal cortex. They then restricted the analyses to children below 9 years of age. Now the main area engaged was left posterior superior temporal cortex. This region is traditionally considered the focus of phonological activity, and is thus thought to be active during grapheme–phoneme translation. As reading developed, activity in left temporal and frontal areas increased, while activity previously observed in right posterior areas declined. This pattern was interpreted as showing that reading-related activity in the brain becomes more left-lateralised with development.

In further analyses focusing just on the younger children, the researchers investigated the relationships between three core phonological skills and word processing. The three core phonological skills are usually taken to be phonological awareness, phonological memory and rapid automated naming (RAN). I will focus on the phonological awareness findings here. Turkeltaub et al. (2003) calculated partial correlations between activated brain regions and each of these three measures while controlling for the effects of the other two measures. They reported that the three different measures correlated with three distinct patterns of brain activity. Brain activity during phonological awareness tasks appeared to depend on a network of areas in left posterior superior temporal cortex (phonology and grapheme–phoneme translation) and inferior frontal gyrus (articulation). The level of the children’s phonological skills modulated the amount of activity in this network. As noted earlier, the left posterior temporal sulcus was the primary area recruited by young children at the beginning of reading development. Therefore, neuroimaging data suggest that phonological recoding to sound rather than logographic recognition is the key early reading strategy. Activity in the inferior frontal gyrus increased with reading ability. This area is also a key phonological area (Broca’s area), important for the motor production of speech. Left inferior frontal gyrus is also activated when deaf children perform phonological awareness tasks silently in fMRI studies (MacSweeney et al. 2005).

An fMRI study of 119 typically developing readers aged from 7 years to 17 years by Shaywitz and colleagues found a similar developmental pattern (Shaywitz et al. 2007). Instead of the false font task, this study used a rhyme decision task (e.g., ‘do these items rhyme?’: leat, kete), and a visual line orientation task (e.g., ‘Do [\V] and [\V] match?’). Shaywitz and his colleagues reported that networks in both left and right superior and middle frontal regions were more active in younger readers, with activity declining as reading developed. In contrast, activity in the left anterior lateral occipitotemporal region increased. This region includes the putative visual word form area (VWFA). Hence both Turkeltaub et al. (2003) and Shaywitz et al. (2007) found decreased right hemisphere involvement as reading developed, but found this for somewhat different neural networks. The difference in the behavioural tasks used (e.g., false font versus rhyme judgement) may explain some of these differences.

Overall, therefore, current neuroimaging data support a ‘single route’ model of reading development, based on a process of developing orthographic–phonological connections at different grain sizes (Ziegler and Goswami 2006). Reading is founded in phonology from the beginning (Goswami and Ziegler 2006b). The VWFA becomes more active as reading develops, reflecting the development of an orthographic lexicon containing both whole words and fragments of familiar words such as orthographic rimes (Pugh 2006). The VWFA is not a logographic or visual lexicon, able to support ‘Chinese’ processing or the ‘direct route’ from printed word to meaning postulated by ‘dual-route’ theory. Neuroimaging studies of typically developing readers show that the
neural networks for spoken language play an important developmental role in reading from the outset.

Neuroimaging studies of dyslexia

The networks recruited for reading

Neuroimaging studies of adult readers with developmental dyslexia suggest that there is atypical activation in the three important neural sites for reading, namely the left posterior temporal regions, the left inferior frontal regions and the left occipitotemporal regions (such as the VWFA). These data suggest both problems with the phonological aspects of reading and with the efficient development of an orthographic lexicon (e.g., Brunswick et al. 1999). These fMRI and PET studies typically rely on tasks such as word and nonsense word reading (e.g., ‘valley’, ‘carrot’, ‘vassey’, ‘cassot’), and the ‘false font’ task. Again, the experimental picture is largely one of convergence across orthographies. For example, adult dyslexics in Italian, French and English all showed activation of a left-lateralised neural network based around posterior inferior temporal areas and middle occipital gyrus (Paulesu et al. 2001). This was a cross-language comparison within one study. However, issues of experimental design become critical when comparing individual imaging studies across languages. When studying any kind of disability, it is crucial to equate participant groups for their overall ability in the actual tasks being used to acquire the neuroimaging data. For example, it is impossible to interpret group differences in brain activity if the dyslexics are worse at reading the nonsense words being used than the control adults. In this case, differences in neural activation could simply reflect different skill levels (i.e., behavioural differences in reading performance). Similarly, it is critical to use the same criteria for acquiring images of the brain in different studies if interpretations about cross-language differences are being drawn (e.g., Ziegler 2005). Otherwise, apparent language-based differences could simply reflect differences in the significance thresholds or other experimental criteria used to acquire the images by different research groups.

Neuroimaging studies of children with developmental dyslexia report a very similar pattern to adult data (e.g., Shaywitz et al. 2002, 2007; Simos et al. 2000). For example, Shaywitz et al. (2002) studied 70 children with dyslexia aged on average 13 years, and compared them to 74 11-year-old typically developing controls (although the controls were not matched for reading level). Using fMRI, the children were scanned while performing a variety of reading-related tasks. These were letter identification (e.g., are t and V the same letter?); single letter rhyme (e.g., do V and C rhyme?); non-word rhyming (e.g., do leat and jete rhyme?); and reading for meaning (e.g., are corn and rice in the same semantic category?). Brain activity in each condition was contrasted with activity in a baseline condition, the line orientation task (e.g., do \[\text{V}\] and \[\text{V}\] match?). Shaywitz et al. (2002) reported that the children with developmental dyslexia showed under-activation in the core left temporoparietal networks, with older dyslexics showing over-activation in right inferior frontal gyrus. The children with developmental dyslexia also showed increased activation in right temporoparietal networks. One drawback of the study, however, was that there were group differences in behavioural performance in some of the component tasks. In the non-word rhyming measure, for example, the controls [79%] were significantly better than the children with dyslexia [59%]). This means that some of the differences found in brain activation could reflect differing levels of expertise rather than differences core to having developmental dyslexia. In a subsequent study of an expanded sample, Shaywitz et al. (2007) used in-magnet non-word reading ability as a covariate to control for this problem. Shaywitz et al. compared 113 dyslexic children aged 7–18 years to
the 119 typically developing readers discussed above in the non-word rhyme and visual line orientation tasks. Compared to the typically developing children, the dyslexic children showed no age-related increase in the activity of the VWFA. Instead, activity in the left inferior frontal gyrus (speech articulation) and the left posterior medial occipitotemporal system both increased, and reading did not become left-lateralized, with continued right hemisphere involvement.

There are also a few studies in the literature exploring the neural networks recruited for reading by dyslexic children in other languages. A study of 13 German dyslexic children aged 14–16 years was reported by Kronbichler et al. (2006). They used a sentence verification task (e.g., ‘A flower needs water’ – TRUE), in an fMRI design, to try and replicate natural reading. A false font task provided the control task. Consistent with studies of English dyslexics, they found reduced activation of left occipitotemporal networks and increased activation of left inferior frontal areas. A study of eight Chinese children with developmental dyslexia reported by Siok et al. (2004) claimed biological disunity, however. Their fMRI study used a homophone judgement task, in which the children had to decide whether two different Chinese characters made the same sound (an English homophone is week – weak), and a character decision task, in which the children saw one Chinese character and had to decide whether it was a real word or not. The first task was intended to measure orthography–phonology connections, and the second orthography–semantic relations. Siok et al. (2004) reported that the Chinese dyslexics did not demonstrate the reduced activation in left temporoparietal regions that would typically be found in developmental dyslexia in English during the homophone judgement task. Instead, an area involved in visuo-spatial analysis showed reduced activity, the left middle frontal gyrus. Siok et al. (2004) used this latter finding to argue that the biological marker for developmental dyslexia in Chinese was reduced activation of left middle frontal gyrus. However, the design of this study does not yet permit this conclusion. A control group matched for reading level is also required. Reduced activation in left middle frontal gyrus when making homophone judgements in Chinese might be expected for the level of reading achieved by the children with dyslexia. If this were to be the case, then increased involvement of networks for visuo-spatial analysis as reading develops would be part of typical reading development in Chinese, rather than a unique biological marker for developmental dyslexia.

**Developmental differences in the time course of neural activation**

While fMRI studies can provide important information about the neural networks supporting reading in typically developing versus dyslexic readers, they do not provide information about the time course of neural processing. This is important, as in typically developing readers words are distinguished from non-words within around 180 ms, suggesting early contact with the VWFA and semantic sites. It seems likely that this process would be delayed in developmental dyslexia. Similarly, it seems possible that cognitive processes such as grapheme–phoneme conversion might take longer in developmental dyslexia.

A longitudinal study of 33 English-speaking children using magnetic source imaging (MSI) compared brain activation in a letter–sound task (the child sees a letter and has to provide its sound) and a simple non-word reading task (e.g., ‘lan’) at the end of kindergarten and again at the end of grade 1 (Simos et al. 2005). Magnetic source imaging depends on a combination of magneto-encephalography (MEG) and MRI. The MEG measures the magnetic fields generated by the electrical activity in the brain rather than the
electrical activity itself (the latter is measured by EEG). These magnetic fields are tiny, they are one billion times smaller than the magnetic field generated by the electricity in a lightbulb. By combining this information with MRI scans, both the time course and spatial localisation of brain activity is possible. Of the 33 children studied, 16 were thought to be at high risk of developing dyslexia.

Simos et al. (2005) reported that the high-risk group were significantly slower to show neural activity in response to both letters and non-words in kindergarten in the occipitotemporal region (320 ms compared to 210 ms for those not at risk). The high-risk group also showed atypical activation in the left inferior frontal gyrus when performing the letter–sound task, with the onset of activity increasing from 603 ms in kindergarten to 786 ms in grade 1. The typically developing readers did not show this processing time increase. Comparing the onset of activity of the three core neural networks for reading, Simos et al. (2005) reported that low-risk children showed early activity in the left occipitotemporal regions, followed by activity in temporoparietal regions, predominantly in the left hemisphere, and then bilateral activity in inferior frontal regions. In contrast, high-risk children showed little differentiation in terms of the time course of activation between the occipitotemporal and temporoparietal regions. High-risk children who were non-responsive to a phonological remediation package also being administered \( (n = 3) \) were distinct in showing earlier onset of activity in inferior frontal gyrus compared to the temporoparietal regions. Given the current dearth of time-course studies by other research groups in either English or in other languages, it is difficult to interpret these differences in terms of the cognitive components of reading. Nevertheless, Simos et al. (2005) comment that the increased inferior frontal activation probably reflects the role of compensatory articulatory processes. As noted earlier, deaf children also show increased inferior frontal activation during phonological processing tasks. This may indicate that children with phonological difficulties rely more heavily on networks for articulation when phonological processing is required.

The neural effects of remediation

Although there are a variety of remediation packages for dyslexic children based on different theories of developmental dyslexia, the most effective packages across languages appear to be those offering intensive phonological intervention (e.g., Bradley and Bryant 1983; Schneider, Roth, and Ennemoser 2000). Simos and his research group (2002) used magnetic source imaging to explore neural activation in eight children with developmental dyslexia who had received 80 hours of intensive training with such a package and who had shown significant benefits from the remediation (Simos et al. 2002). MSI scans were taken during a non-word rhyme matching task (e.g., ‘yoat’, ‘wote’) both before the intervention and following remediation. Simos et al. (2002) reported that prior to the intervention, the dyslexic children showed the expected hypoactivation of left temporoparietal regions. Following the intervention, all eight children showed a dramatic increase in the activation of left temporoparietal regions, predominantly in the left posterior superior temporal gyrus (the networks supporting grapheme–phoneme recoding in typically developing readers: see Turkeltaub et al. 2003). These activation profiles were very similar to those of eight controls who also participated in the MSI study, but who did not require remediation. Nevertheless, even after remediation neural activity was delayed in the children with dyslexia relative to the controls. The peak in left superior temporal gyrus activity occurred at 837 ms on average for the dyslexic children, and at 600 ms for the controls. The data were taken to show a normalisation
of brain function with remediation. Nevertheless, Simos et al. (2002) commented that even with intensive remediation, children with dyslexia are slow to achieve the reading fluency shown by non-dyslexic children.

Shaywitz and Shaywitz (2005) used retrospective examination of the large sample of children with developmental dyslexia reported in Shaywitz et al. (2002) to compare the different developmental trajectories for children at risk for reading difficulties. Shaywitz and Shaywitz (2005) distinguished three groups within this sample when they were young adults. The first was a group of persistently poor readers (PPR), who had met criteria for poor reading in both the 2nd/3rd and the 9th/10th grades. The second was a group of accuracy-improved poor readers (AIR), who had met criteria for poor reading in the 2nd/3rd grades but who did not meet criteria in the 9th/10th grades. The third was a control group of non-impaired readers (C), who had never met criteria for poor reading (the participants had been studied since the age of 5 years). Shaywitz and Shaywitz (2005) reported that both the PPR and the AIR groups showed hypoactivation of the core left hemisphere sites when required to manipulate phonology. For example, in a nonsense word rhyming task, both groups of young adults still showed relative hypoactivity in neural networks in left superior temporal and occipitotemporal regions. However, the groups were distinguished by their neural activity when reading real words. The AIR group still demonstrated under-activation in the usual left posterior areas for real word reading, whereas the PPR group activated the left posterior regions to the same extent as controls (this was an unexpected finding).

Shaywitz and Shaywitz (2005) then carried out further analyses based on connectivity. Connectivity analyses examine the neural areas that are functionally connected to each other during reading. The connectivity analyses suggested that reading achievement depended on memory for the PPR group, and not on the normalised functioning of the left posterior regions. The unimpaired controls demonstrated functional connectivity between left hemisphere posterior and anterior reading systems, but the PPR group demonstrated functional connectivity between left hemisphere posterior regions and right prefrontal areas associated with working memory and memory retrieval. Shaywitz and Shaywitz (2005) speculated that the PPR group were reading primarily by memory. As the words used in the scanner were high-frequency, simple words, this is quite possible. However, this design choice complicates the interpretation of the neural differences found, as the PPR group may not be able to use memory strategies to read less frequent or less simple words. For such stimuli, the PPR and AIR groups may show similar neural profiles. It may also be important that the PPR group had, in general, lower IQ scores than the AIR group. Prospective longitudinal studies comparing patterns of neural activation and connectivity in dyslexic children as high-frequency words become over-learned would clearly be very valuable.

Different technologies, different research questions: the promise of brain imaging for understanding reading and developmental dyslexia

As will be clear from the foregoing review, most studies of reading development and of developmental dyslexia have relied on fMRI. These studies have provided excellent data regarding the neural networks underpinning reading in typically developing and dyslexic readers. They have also shown that the functional organisation of the networks for reading is similar in typical development and in dyslexia. Children with developmental dyslexia do not recruit radically different neural networks when they are reading. Rather, they show hypoactivation of crucial parts of the network of areas involved in word
recognition, and an atypical pattern of continuing right hemisphere involvement. Although highly informative, these studies are essentially correlational studies. They can answer research questions about the neural demands made by learning to read in different languages, and they can answer research questions about the core neural systems involved for dyslexic and typically developing readers. They can also answer research questions about the patterns of connectivity between different neural networks. However, they cannot answer research questions about what ‘goes wrong’ in the dyslexic brain, although they can help to rule out hypotheses (e.g., about the visual basis of developmental dyslexia; see Eden and Zeffiro 1998).

Neuroimaging methods that provide data on the time course of neural processing, such as MEG (MSI) and EEG, can begin to answer causal questions. As might be expected, it has been shown using MSI that neural activation is delayed in core components of the network of areas recruited for reading by children at risk for dyslexia. However, behavioural studies showing that children with developmental dyslexia are slower to read words aloud make the same point. When EEG or MSI techniques show that core components of the reading network are activated in a different order in dyslexia compared to typical reading, this is more informative with respect to causality. For example, Simos and his colleagues have shown atypically earlier onset of activity in inferior frontal gyrus (articulation) compared to the temporoparietal regions in three children at high risk for dyslexia who appear to be non-respondent to a phonological remediation package. If robust with larger samples and diagnosed dyslexics, such findings could suggest that there are different neuro-developmental routes to word recognition for dyslexic children compared to controls. Nevertheless, these different neuro-developmental routes are not the cause of dyslexia. Rather, they illustrate the response of a dyslexic brain to being trained to learn to read.

In my view, the most informative studies with respect to causation in developmental dyslexia are longitudinal prospective studies that use brain imaging to study basic sensory processing in at-risk children, with a view to understanding the causes of the phonological deficit. Here, the most promising studies to date are those investigating basic auditory processing using methodologies sensitive to the time course of auditory processing at the millisecond level. For example, a large-scale Finnish study (the Jyväskylä Longitudinal Study of Dyslexia (JLD); see Lyytinen et al. 2004a) has followed babies at familial risk for dyslexia since birth. A large variety of behavioural and EEG measures has been taken as the children have developed. EEG measures of auditory sensory processing (evoked response potentials to speech and non-speech cues) have been found to distinguish the at-risk babies from controls even during infancy (e.g., Lyytinen et al. 2005). For example, infants at risk for developmental dyslexia were less sensitive to the auditory cue of duration at six months of age (Richardson et al. 2003). The infant participants had to discriminate between two bisyllabic speech-like stimuli with a varying silent interval (e.g. ‘ata’ versus ‘atta’). Duration discrimination was still impaired when the same children were 6.5 years of age (Lyytinen et al. 2004b).

English children with developmental dyslexia are also impaired in this duration discrimination task (Richardson et al. 2004). In addition, English children are impaired in discriminating the rise time of amplitude envelopes at onset, which is an important auditory cue to the onset of syllables in the speech stream (Goswami et al. 2002; Richardson et al. 2004). Finnish adults with developmental dyslexia also show rise time processing impairments, and individual differences in rise time sensitivity predicted up to 35% of unique variance in phonological tasks like rhyme recognition (Hämäläinen et al. 2005). In the English studies, individual differences in rise time sensitivity predict unique
variance in both phonological awareness measures (around 20%: Richardson et al. 2004) and in reading and spelling measures (around 25%: Goswami et al. 2002). We are currently collecting EEG data comparing rise time discrimination in English children with and without dyslexia. Data so far suggest that children with developmental dyslexia indeed show atypical auditory processing of rise time stimuli, with N1 amplitude (an EEG measure of sound registration) failing to reduce as amplitude envelope rise times become extended (Thomson, Baldeweg, and Goswami 2005). This suggests that neural responses in the dyslexic brain do not distinguish between different rise times, at least for the auditory processing comparisons used in our study (15 ms versus 90 ms rise times).

Conclusion

Different neuroimaging methodologies contribute complementary data regarding the neural networks underpinning reading acquisition and developmental dyslexia. While fMRI studies can identify the core neural systems involved in reading, EEG and MEG methodologies are required to investigate the time course of activation of the different networks that contribute to word recognition, and to investigate potential sensory precursors of the phonological deficit. With respect to key questions in education, each neuroimaging method can contribute different kinds of data. For example, when evaluating the claims made for different kinds of remediation package for developmental dyslexia, fMRI will be useful in assessing whether interventions affect the core neural networks for reading, or affect a different kind of network (e.g., motivational systems). When evaluating claims that the core cognitive difficulty in developmental dyslexia lies in forming a high-quality phonological representation, methodologies that can explore the time course of sensory processing such as EEG will be most useful. Neuroimaging methods are of optimal use when they can provide experimental data that is not available from behavioural investigations. For example, it is possible in principle to identify neural markers of risk for developmental dyslexia that can be measured in pre-verbal infants and in older children without requiring their explicit attention (Szűcs and Goswami 2007). It is these areas of neuroscience that are likely to be of most potential benefit to educators.

References


Dyscalculia: neuroscience and education

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(Received 31 July 2007; final version received 8 November 2007)

Background: Developmental dyscalculia is a heterogeneous disorder with largely dissociable performance profiles. Though our current understanding of the neurofunctional foundations of (adult) numerical cognition has increased considerably during the past two decades, there are still many unanswered questions regarding the developmental pathways of numerical cognition. Most studies on developmental dyscalculia are based upon adult calculation models which may not provide an adequate theoretical framework for understanding and investigating developing calculation systems. Furthermore, the applicability of neuroscience research to pedagogy has, so far, been limited.

Purpose: After providing an overview of current conceptualisations of numerical cognition and developmental dyscalculia, the present paper (1) reviews recent research findings that are suggestive of a neurofunctional link between fingers (finger gnosis, finger-based counting and calculation) and number processing, and (2) takes the latter findings as an example to discuss how neuroscience findings may impact on educational understanding and classroom interventions.

Sources of evidence: Finger-based number representations and finger-based calculation have deep roots in human ontology and phylogeny. Recently, accumulating empirical evidence supporting the hypothesis of a neurofunctional link between fingers and numbers has emerged from both behavioural and brain imaging studies.

Main argument: Preliminary but converging research supports the notion that finger gnosis and finger use seem to be related to calculation proficiency in elementary school children. Finger-based counting and calculation may facilitate the establishment of mental number representations (possibly by fostering the mapping from concrete non-symbolic to abstract symbolic number magnitudes), which in turn seem to be the foundations for successful arithmetic achievement.

Conclusions: Based on the findings illustrated here, it is plausible to assume that finger use might be an important and complementary aid (to more traditional pedagogical methods) to establish mental number representations and/or to facilitate learning to count and calculate. Clearly, future prospective studies are needed to investigate whether the explicit use of fingers in early mathematics teaching might prove to be beneficial for typically developing children and/or might support the mapping from concrete to abstract number representations in children with and without developmental dyscalculia.

Keywords: dyscalculia; functional brain imaging; neuroscience; finger-based calculation; mental number representations

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Introduction

Arithmetic learning disorders (developmental dyscalculia) denote circumscribed and outstanding difficulties in the acquaintance of arithmetic skills. Importantly, dyscalculia is not a unitary concept and the associated cognitive profiles might vary widely between and within individuals (for overviews see Kaufmann and Nuerk 2005; Wilson and Dehaene 2007). With an estimated prevalence of 3% to 7%, developmental dyscalculia is about as frequent as developmental dyslexia (APA 1994) and, similar to dyslexia, persists into adulthood if untreated (Shalev and Gross-Tsur 2001). It is widely agreed that dyscalculia is a highly familial disorder (the risk for siblings of children suffering from dyscalculia is five to ten times higher than in the general population: Shalev et al. 2001). Though developmental dyscalculia may present as a single-deficit disorder (e.g., core deficit of ‘number sense’: Landerl, Bevan, and Butterworth 2004; for a review see Wilson and Dehaene 2007), many affected children exhibit associated cognitive problems, both within and outside the numerical domain (for respective overviews see Kaufmann and Nuerk 2005; Shalev and Gross-Tsur 2001). The frequent occurrence of comorbidities coincides with recent findings reporting a substantial genetic overlap between various developmental learning disorders such as dyscalculia, dyslexia and attention-deficit hyperactivity disorder (Plomin, Kovas, and Haworth 2007). Nevertheless, dyscalculia research is much younger than dyslexia research and most of what is known is derived from adult studies involving mature brain systems. Hence, our current understanding of the behavioural manifestations and (neuro)cognitive foundations of developmental dyscalculia remains incomplete.

The main aim of this paper is to demonstrate the need to go beyond adult calculation models when attempting to account for the peculiarities of developing brain systems and developmental disorders such as developmental dyscalculia. Moreover, the paper aims to illustrate how findings from brain imaging studies may inform educational understanding and even classroom instructions/interventions. The example discussed here concerns the association between fingers and numbers which has not been considered explicitly in adult calculation models, but which nevertheless – as we shall see – plays a fundamental role in learning to count and calculate. Butterworth (1999) merits reward for initiating the greatly renewed neuroscientific interest in the potential importance of finger use for the acquaintance of numerical skills. According to Butterworth (1999), fingers are convenient and natural tokens to represent number magnitudes which are intuitively used by young children when learning the verbal counting sequence and/or when executing first simple additions and subtractions (see also Butterworth 2005). Moreover, even adults may use finger-based back-up strategies. Thus, it may be argued that finger-based number representations and finger-based calculation are deeply rooted in human ontology and phylogeny. Indeed, converging evidence from brain imaging findings corroborate the latter notion of a neurofunctional link between fingers and numbers (Andres, Seron, and Olivier 2007; Kaufmann et al. 2008; Sato et al. 2007; Thompson et al. 2004). Before presenting the respective findings and their potential educational implications in more detail, we present a brief overview of current conceptualisations of typical and atypical developmental pathways of numerical cognition (as derived from neuropsychology and neuroscience).

Current conceptualisations of numerical cognition and developmental dyscalculia

Infants as young as 4–6 months are able to make number-based discriminations and even exhibit additive expectation behaviour (up to set sizes of three objects: Wynn 1992, 1995).
Larger set size discriminations may be mastered, too, provided the ratio of the to-be-compared object sets is large enough (e.g., babies may discriminate 8 from 16, but not 8 from 12 objects: Xu and Spelke 2000; and even 16 from 32 objects, but not 16 from 24: Xu, Spelke, and Goddard 2005). Interestingly, when continuous stimulus characteristics such as contour length and total filled area are controlled, 6-month-old babies can successfully discriminate large, but not small, numerosities (Xu, Spelke, and Goddard 2005; see also Xu 2003). Consequently, Xu (2003) and Xu, Spelke, and Goddard (2005) propose the existence of two core systems for number magnitude representation in infants: one mediating small (exact) and the other supporting large (approximate) numerical magnitudes (see also Feigenson, Dehaene, and Spelke 2004; for a similar distinction in mature brain systems see Dehaene and Cohen 1997).

In verbal individuals (i.e., children who have begun to master language) numerical concepts emerge as soon as children use counting to refer to objects. Upon entering formal education, most typically developing children demonstrate a rudimentary understanding of number relations, are able to count up to 20 and may even master simple additions and subtractions verbally when allowed to use their fingers or other reference objects. Thus, upon starting school (at age 6 in most European countries), most children already have acquired some verbal counting and calculation skills, which are considered to be the foundations for establishing more advanced school mathematics.

But what do we know about the development of these number processing and calculation skills during infancy and preschool years? How do children develop quantity knowledge and number representations? Which neurocognitive processes and mechanisms come into play when children gradually acquire abstract (symbolic) number representations during formal schooling? And which difficulties – within and outside the numerical domain – accompany developmental dyscalculia? These latter questions are at the core of current research attempts aiming to delineate the developmental pathways of numerical cognition. However, dyscalculia research is relatively young and, in the absence of an empirically validated developmental calculation model, many neuropsychological studies targeted at gaining a better understanding of the developmental pathways of typical and/or atypical numerical cognition rest on adult calculation models (e.g., Dehaene and Cohen 1995; Dehaene et al. 2003; McCloskey, Caramazza, and Basili 1985).

According to popular adult calculation models, number processing and calculation is multi-componential and the components constituting the calculation system are thought to be modularly organised (Dehaene and Cohen 1995; McCloskey, Caramazza, and Basili 1985). The modularity assumption derives from adult cognitive neuropsychology which considers double dissociations as evidence for a modular architecture of (neuro)cognitive systems and/or mental representations (Shallice 1988). For example, a double dissociation is present if cognitive ability A is preserved while ability B is deficient in one individual, and in another individual the opposite pattern emerges (i.e., preserved ability B and deficient ability A). Probably the most popular adult calculation model is the so-called ‘triple code model’ of numerical cognition (Dehaene and Cohen 1995) postulating three modularly organised but interrelated calculation components (i.e., analogue magnitude representation, auditory verbal word frame, visual Arabic number form), each of which is thought to be supported by distinct brain regions. A consistent finding in the adult literature concerns the key role of the intraparietal sulcus (IPS) for number magnitude processing (for an overview see Dehaene et al. 2003; Hubbard et al. 2005).

Up to now, numerous (mostly adult) studies have been published taking the Dehaene calculation model (Dehaene and Cohen 1995, 1997) as a starting-point upon which to test their hypotheses (for respective reviews see Dehaene et al. 2003; Hubbard et al. 2005; see
also for developmental issues Kaufmann and Nuerk 2005; Wilson and Dehaene 2007). However, because of crucial differences between developing and mature brain systems, adult models may not provide adequate theoretical frameworks for investigating developmental disorders (see Bishop 1997; Karmiloff-Smith 1992). For example, double dissociations in developmental disorders need not necessarily reflect the presence of modularly organised neurofunctional networks (Karmiloff-Smith 1997; Pennington 2006). Indeed, double dissociations have been observed in non-modular cognitive architectures as well (i.e., in the case of dyslexia the double dissociation between phonological and surface dyslexia; Harm and Seidenberg 1999; Plaut 1995).

Behavioural studies suggest that number magnitude discrimination abilities may be an innate capacity inherent to infants (Wynn 1992, 1995; Xu and Spelke 2000; Xu 2003) and even non-human species (Brannon and Roitman 2003), yet the question arises whether number magnitude processing is supported by identical brain regions in infants and adults. Although present developmental findings are consistent with a neurofunctional link between intraparietal regions and number magnitude processing, there is some controversy regarding the age-dependency of intraparietal (IPS) involvement in the formation of arithmetical skills. While some findings reveal similar activations in intraparietal regions extending across different ages during number magnitude processing (symbolic number processing: Temple and Posner 1998; non-symbolic number processing: Cantlon et al. 2006; Temple and Posner 1998), other studies suggest that the functional specialisation of the IPS for number magnitude processing increases with age (non-symbolic number processing: Ansari and Dhital 2006; symbolic number processing: Ansari et al. 2005; Kaufmann et al. 2005, 2006; Rivera et al. 2005). These conflicting results may partly be explained by methodological differences between studies, making a direct comparison across studies (and paradigms) difficult. Alternatively, and as mentioned already above, one may claim that adult models (resting on modularity assumptions) are not apt to account for the complexity of developing brain systems.

Developmental dyscalculia: single- or multiple-deficit views?
Upon adopting Dehaene’s modularly organised adult calculation model (Dehaene and Cohen 1995; Dehaene et al. 2003), some researchers propose that the neurocognitive underpinnings of developmental dyscalculia are best conceptualised as a (single) core deficit of ‘number sense’ (e.g., Butterworth 2005; Landerl, Bevan, and Butterworth 2004). The core deficit hypothesis implies that children diagnosed with developmental dyscalculia display specific difficulties to mentally represent and manipulate (non-symbolic) number magnitudes. Consequently, the core deficit of ‘number sense’ is thought to be related to a malfunctioning of intraparietal brain regions (i.e., the horizontal segment of the intraparietal sulcus (HIPS), which is supposed to mediate number magnitude processing according to Dehaene et al. 2003). In an excellent review, Wilson and Dehaene (2007) revisit this strong single-deficit view of developmental dyscalculia by arguing that the core deficit of ‘number sense’ may be only one of several possibly underlying deficits. According to Wilson and Dehaene (2007), other potential subtypes of dyscalculia – each of which being supported by distinct brain regions – may rest on (1) deficient verbal symbolic representations (manifesting themselves as arithmetic fact retrieval difficulties); (2) deficient executive functions (hampering fact retrieval as well as complex calculation); or (3) deficient spatial attention (leading to impaired quick recognition of small numerosities and possibly negatively affecting non-symbolic and symbolic number manipulations). Thus, Wilson and Dehaene (2007) propose that the behavioural
characteristics of developmental dyscalculia – and their neurocognitive underpinnings – might vary substantially between individuals, thus seriously questioning a strong single-deficit view.

Another argument challenging the single-deficit view of developmental dyscalculia is the observation that many children exhibiting problems in learning to count and calculate also have difficulties in other cognitive domains. Even within the arithmetical domain, children display quite distinguishable performance profiles (both at an intraindividual and interindividual level of analysis: Dowker 2005). Consequently, various attempts to classify developmental dyscalculia at a behavioural level have been undertaken (e.g., Geary 2000; Temple 1989, 1991; Von Aster 2000). As early as 1991, Temple reported a double dissociation between arithmetic fact retrieval (e.g., number fact knowledge such as $3 \times 5$) and procedural knowledge (‘knowing how’ to solve a complex arithmetic problem) in developmental dyscalculia (however, see for a critical discussion of double dissociations in developmental disorders Pennington 2006). The distinction between arithmetic fact and procedural knowledge was first acknowledged in the adult calculation model proposed by McCloskey and colleagues (1985). Likewise, the theoretical foundations for Geary’s (2000) and Von Aster’s (2000) efforts to classify developmental dyscalculia were grossly based on the Dehaene calculation model (Dehaene and Cohen 1995). A commonality of the latter classification attempts is their effort to further differentiate developmental dyscalculia according to specific performance profiles, which in turn imply the existence of distinct single cognitive deficits. And yet, according to Pennington (2006), any attempt to link these deficits to one – and only one – underlying neuroanatomical (and/or genetic) underpinning is likely to fail. Rather, developmental dyscalculia should be regarded as a complex and dynamic developmental disorder (for similar views of dyslexia and attention-deficit hyperactivity disorder see Bishop 1997; Pennington 2006). Interestingly, and consistent with the latter view, recent findings of quantitative genetic research report a substantial genetic overlap between quite diverse cognitive (dis)abilities such as reading, language and arithmetic (Plomin and Kovas 2005; for a review see also Plomin et al. 2007). This genetic overlap may partly explain the repeatedly reported high incidence of comorbidity of developmental disorders such as dyslexia, dyscalculia and attentional disorders (for a review see Kaufmann and Nuerk 2005), and furthermore, may also partly account for the considerable diversity of neurocognitive performance within one developmental disorder (in our case, dyscalculia).

To summarise, although single-deficit models (e.g., core deficit of ‘number sense’: Butterworth 2005; Landerl, Bevan, and Butterworth et al. 2004; ‘number fact dyscalculia’: Temple 1991) are presently predominant in the neuroscientific literature – probably because they are simpler and hence more testable – multiple-deficit models of developmental dyscalculia seem to better fit our current understanding of the complex nature of developmental disorders.¹ Thus, a change of paradigms from a modular and single-deficit view towards a dynamic, process-oriented and multiple-deficit view seems to be essential for the development of empirically validated developmental calculation models, as well as for the production of mathematics curricula meeting children’s neurocognitive development (i.e., maturation-dependent readiness to grasp number-based concepts and skills) and the generation of tailored dyscalculia intervention programmes.

Neuroscience and education: the case of developmental dyscalculia

A frequent criticism of brain imaging studies involving learning is their restricted applicability to education and classroom interventions. Indeed, the great majority of
neuroscientific research – including the realm of numerical cognition and/or developmental dyscalculia – is targeted at basic research. As neuroscience is a rather young discipline, which is further tightly connected to recent technological advances (and which underwent and still continues to undergo dramatic changes within very short periods), early respective studies are hardly comparable to more recent ones (regarding both methodological and practical issues). Further, it is important to acknowledge that significant activations reported in imaging studies reveal brain regions modulating a specific task which is not equivalent to regions being necessary to process the task at hand. In addition, and partly because of methodological and technical constraints, experimental paradigms generally focus on islets of skills rather than learning processes and mechanisms, the latter being a greater focus of interest for educational researchers and classroom teachers. In general, imaging studies are only as good as the behavioural paradigms they are implementing. Hence, the development of adequate behavioural paradigms should be based on a sophisticated understanding of the interplay between neurocognitive (including genetic) and pedagogical factors determining typical and atypical trajectories within particular cognitive domains (i.e., in our case, numerical cognition).

Recently, researchers of both disciplines (i.e., neuroscience and education) are slowly becoming aware of the urgent need to ameliorate communication and to foster common research efforts. The latter focus is reflected in continuously appearing scientific articles devoted to the topic of ‘neuroscience and education’ (Ansari and Coch 2006; Fawcett and Nicolson 2007; Goswami 2004; Szucs 2005), as well as in the newly founded scientific journal Mind, Brain and Education, whose first issue was compiled in 2007. Furthermore, Fawcett and Nicolson (2007) request the establishment of a new discipline of ‘pedagogical neuroscience’. These authors emphasise that diagnostic efforts based on behavioural and/or cognitive symptoms are not sufficient to contribute to our understanding of complex developmental disorders. Taking developmental dyslexia as an example, Fawcett and Nicolson (2007) stress the need to develop brain-based theories (by employing genetic and brain-based diagnostic methods), which eventually may advance not only our understanding of developmental disorders, but also lead to tailored interventions. Hence, there is a clear need for research designs being specifically targeted at the educational implications of neuroscience research. In order to accomplish the latter goal, educational experts must share their expertise in pedagogy, and neuroscience researchers must develop ecological paradigms that are capable of investigating cognitive processes and learning mechanisms instead of circumscribed skills.

Below, I present a brain imaging study conducted at Innsbruck Medical University which was aimed at making a first step towards bridging the gap between neuroscience and education (Kaufmann et al. 2008). Although the main aim of our study was to elucidate the link between non-symbolic numerical and spatial processing, here I will focus on the numerical task only and the potential implications for educational sciences that arose from studying it. The numerical task required participants to make simple number comparisons. Stimuli were pictures of two hands, each hand showing a different finger pattern (e.g., the right hand raising three fingers, the left one two fingers). Thus, our experimental paradigm provoked finger-based counting/number discriminations and these helped us address some questions regarding the association between fingers and numbers. Before discussing the results, I briefly present the relevant literature that led us to formulate our working hypotheses.
Fingers and numbers

Empirical evidence for a link between fingers and numbers is derived from developmental behavioural studies (Fayol, Barrouillet, and Marinthe 1998; Gracia-Baffaluy and Noel 2008; Landerl, Bevan, and Butterworth 2004; Noel 2005; Sato and Lalain 2008), patient studies (Gerstmann syndrome: Gerstmann 1940; developmental Gerstmann syndrome: Benson and Geschwind 1970; Suresh and Sebastian 2000) and brain imaging studies (fMRI: Simon et al. 2002; Thompson et al. 2004; TMS: Andres, Seron, and Olivier 2007; Roux et al. 2003; Rusconi, Walsh, and Butterworth 2005; Sato et al. 2007).

Probably the earliest report of a neurofunctional association between fingers (i.e., finger discrimination) and number processing was provided in 1940 by Gerstmann, who described a patient with a right posterior parietal lesion accompanied by symptoms combining finger agnosia (difficulties to recognise and discriminate fingers), acalculia (outstanding calculation problems), right–left disorientation and agraphia (impaired writing: see Benton 1997; for descriptions of developmental Gerstmann syndromes see, e.g., Benson and Geschwind 1970; Suresh and Sebastian 2000).2

Associations between fingers (finger gnosis) and calculation skills have also been reported in developmental behavioural studies. For instance, in typically developing preschool children, neuropsychological test scores (including finger recognition and finger discrimination) were found to be a good predictor of calculation skills one year later (Fayol, Barrouillet, and Marinthe 1998). Furthermore, the findings of Noel (2005) revealed that finger gnosis seems to be a specific predictor for numerical abilities and further suggest that the link between finger gnosis and arithmetic is not restricted to tasks relying on finger-based magnitude representations (but rather encompasses a wide range of number processing tasks). Noel (2005) argues that the latter findings are best explained by the anatomical vicinity of brain regions supporting finger gnosis and those mediating number magnitude processing and calculation.

Consistent with the latter suggestion, results of a functional magnetic resonance imaging (fMRI) study (Simon et al. 2002) revealed neighbouring and partly overlapping activations in posterior parietal brain regions for quite diverse abilities such as arithmetic and goal-directed hand movement (grasping/pointing), among others. In particular, brain regions supporting grasping (postcentral gyrus and anterior IPS) were found to border those mediating calculation (in and around the (H)IPS: see Simon et al. 2002, figure 2; for a review, see also Hubbard et al. 2005).

Further corroborating the notion of a neurofunctional link between finger use and number processing are the results of a repetitive transcranial magnetic resonance (rTMS) study revealing that both finger movements and number magnitude judgements (Arabic digits) are disrupted by left parietal stimulation in adults (i.e., angular gyrus: Rusconi, Walsh, and Butterworth 2005; see also Roux et al. 2003). Finally, two recent TMS studies assessing corticospinal excitability in hand muscles are suggestive of (1) a special role of right-hand muscles (left hemisphere) for small numerals (1–4, which were interpreted as reflecting culturally acquainted embodied finger counting strategies: Sato et al. 2007; for consistent behavioural findings see Sato and Lalain 2008); and (2) of a link between hands (but not arms and/or legs) and enumeration (numbers and letters: Andres, Seron, and Olivier 2007). Interestingly, upon investigating 16- and 17-year-old adolescents with and without a diagnosis of developmental dyscalculia (DD), Soltész and collaborators (2007) report that electrophysiological responses upon performing a simple, one-digit number comparison task were not comparable between the two groups (though both groups displayed comparable behavioural performance on this task). In particular,
and most interestingly, relative to their non-DD peers, individuals with DD displayed very specific neuropsychological performance profiles being characterised by preserved mental rotation and body part knowledge (among others) but deficient performance on mental finger rotation and finger knowledge. Thus, the latter results provide the first evidence that, in DD (or some groups of DD), deficient finger knowledge may be associated with atypical brain mechanisms for performing a basic numerical task.

The latter results are clearly exciting, but it has to be noted that all respective brain imaging studies were performed on adults and hence provide information about mature brain systems only. Indeed, despite converging behavioural evidence for an association between finger gnosis and numerical skills in children (e.g., Fayol, Barrouillet, and Marinthe 1998; Noel 2005; Sato et al. 2007), respective developmental brain imaging studies are so far lacking. This gap in the research provided our motivation for conducting a developmental fMRI study that required 8-year-old children (and young adults) to make number magnitude judgements. More specifically, we presented stimuli that consisted of pictures of two hands representing different numerosities (i.e., finger patterns). Participants were asked to indicate, by pressing a button, which hand displayed more fingers. Thus, by provoking finger-based comparison strategies, the experimental paradigm required participants to make (non-symbolic) numerical classifications.

Besides number discriminations, participants were asked to make spatial and colour discriminations, too. As a thorough discussion of this research clearly goes beyond the scope of the present paper, results and educational implications presented here will focus on the following questions: (1) do elementary school children and adults recruit identical brain regions upon solving a simple number comparison task (provoking finger-based number representations)?; and (2) is there a neurofunctional link between finger-based number representations and counting/number comparison, and if so, is there an age-related change in cerebral activation patterns related to finger-based magnitude extraction?

Results revealed highly interesting findings. Behaviourally, children and adults performed at ceiling upon making number classifications (99.3% correct). However, compared with adults, children were significantly slower (746 ms and 1017 ms respectively), although response latency patterns for different types of pairs were again comparable between age groups. In particular, both age groups were significantly quicker to classify distant relative to adjacent number pairs (i.e., displaying shorter response latencies upon comparing 1 versus 5 relative to 1 versus 2: children $p < 0.05$; adults $p < 0.001$). The latter reaction time phenomenon has been coined the ‘distance effect’ and is thought to reflect the integrity of the mental number line (Dehaene 1991). Thus, the behavioural data suggest that the task was performed flawlessly by both groups (as reflected by very high accuracy rates) and, moreover, children and adults alike processed number magnitude all the way down to the semantic numerical level (as reflected by the presence of the distance effect). However, a different picture emerged regarding brain activation patterns. In particular, activation patterns in response to non-symbolic number processing were clearly distinguishable between children and adults. Relative to adults, children recruited additional brain areas in lateral portions of anterior IPS, as well as in adjacent regions of the right post- and precentral gyrus upon making finger-based number magnitude classifications. Most interestingly, the latter regions were found to be deactivated in adults. We interpret our findings as being suggestive of an age-dependent neurofunctional link between areas supporting finger use and non-symbolic number processing (Kaufmann et al. 2008). Importantly, the latter findings imply that even in the case of comparable behavioural performance between children and adults, brain activation patterns need not be identical across age.
Potential educational implications of our findings

Our findings provide evidence for an age-dependent link between finger-use and number processing that is not only interesting for neuroscience and numerical cognition research, but may also have significant implications for educational research and even classroom teaching. The demonstration of age-dependent activation differences when solving a simple number comparison task (which was performed at ceiling by both children and adults) emphasises the importance of going beyond behavioural (performance) issues. Our findings highlight the potential benefit of incorporating our steadily increasing understanding of developing neurofunctional systems into efforts to design adequate and timely pedagogical curricula. In other words, although children may succeed in solving a particular (numerical) task as well as adults in terms of performance, children may need to put more effort than adults into orchestrating the brain regions associated most closely with the task. For instance, as illustrated above, a simple number comparison task requires 8-year-old children – but less so adults – to recruit brain areas supporting finger use, thus revealing age-dependent processing mechanisms at a neural level.

With respect to classroom teaching, the implications of the latter findings are straightforward. As brain areas mediating finger use might be co-activated whenever children need to access mental number representations, it does not seem advisable to forbid children using their fingers upon performing arithmetic problems. Rather, educators and teachers could take advantage of the fact that fingers may serve as concrete embodied tokens to represent number magnitude. Moreover, fingers mirror the base-10 number system, and moreover, are readily available to be used as back-up strategies. Thus, it is plausible to expect that the consistent use of fingers could positively affect the formation of mental number representations (by facilitating the mapping from concrete non-symbolic quantity knowledge to abstract symbolic number processing) and thus also the acquisition of calculation skills. Indeed, preliminary evidence supporting the latter claim comes from a recent intervention study demonstrating that training finger gnosis significantly improves arithmetic performance in 1st- graders (Gracia-Baffaluy and Noel 2008). Moreover, a prospective study of elementary school children revealed a predominance of split-five calculation errors (i.e., solutions deviating +5 from the correct result: Domahs, Krinzinger, and Willmes 2008). The latter authors interpreted their findings as reflecting ‘failure to keep track of “full hands” in counting or calculation’ (abstract: Domahs, Krinzinger, and Willmes 2008). Interestingly, with increasing age/schooling (i.e., grades 1 to 3) split-five errors decreased, thus suggesting that children’s reliance on mental finger patterns (whole hand/five fingers) decreases with increasing schooling/calculation proficiency.

Finally, it is not far-fetched to argue that the explicit incorporation of finger-use in numeracy intervention programs could be beneficial for the establishment of mental number representations in children suffering from developmental dyscalculia. However, in the absence of respective empirical studies, the latter claim thus far remains speculative.

Last but not least, it is important to stress that finger knowledge is not the whole story to becoming good at maths. Rather, several other skills like abstract thinking (e.g., facilitating the mapping process between concrete and symbolic arithmetic), spatial skills (enabling the formation of a spatially oriented mental number line and, moreover, our understanding of the base-10 system of the Arabic number system), working memory (enabling us to manipulate quantities when solving arithmetic tasks, to monitor multi-step procedures, etc.), language proficiency (underlying counting routines and arithmetic fact retrieval, among others) are also considered to be important for becoming a proficient calculator. Each of the latter domains is likely to mediate the acquisition and/or application of arithmetic skills and future – preferably longitudinal – research endeavours...
are needed to elucidate their impact on the development of numerical cognition (for a review see Kaufmann and Nuerk 2005; Wilson and Dehaene 2007).

To summarise, although the study described here nicely demonstrates the usefulness of tailoring research questions to pedagogical demands with respect to a core numerical skill, there is a clear need for future (prospective) research designs targeted at more complex learning processes and mechanisms within the realm of numerical cognition. The most sensible way to approach – and possibly achieve – the latter goal is to intensively foster scientific communication and expertise between neuroscience and education.

Acknowledgement

The author was supported by the Austrian Science Foundation (grant number T286-B05).

Notes

1. A major disadvantage of ‘complex models’ is that they may render the verification and/or falsification of working hypotheses difficult (because they typically encompass many dependent and/or unknown variables). Hence, researchers aiming to assess complex developmental disorders are especially required to (1) formulate very clear-cut hypotheses from the outset; (2) carefully define selection criteria for their study populations; and (3) employ paradigms that have been found previously to be adequate (and testable) for the research questions of interest. Reasons for advocating ‘complex models’ are at least twofold: first, complex models readily acknowledge modulating cognitive abilities (within and outside the numerical domain: Kaufmann and Nuerk 2005; Wilson and Dehaene 2007); and second, complex models may lead to a better understanding of the link between mind (cognitive), brain (neurofunctional) and pedagogy (behavioural and educational factors) mediating the acquaintance of number processing and calculation skills.

2. Though Benton (1997) seriously questioned the entity of the syndrome by stressing that a substantial proportion of patients exhibit some, but not all four symptoms constituting the full Gerstmann syndrome, the Gerstmann syndrome has received increased interest recently.

3. Stimuli across the three tasks were identical (only instructions varying), thus enabling us to control for domain-general perceptual and response-bound processing mechanisms. The strict control of domain-general processing mechanisms is crucial in brain imaging research as the to-be-interpreted activation patterns should be attributable to task-relevant processing solely (or as far as possible). The latter endeavour is achieved by a subtraction method whereby the cerebral activation patterns obtained in response to a control task (which is preferably identical to the experimental task in all but the variable of interest, in our case, number processing) are subtracted from the activations obtained in response to the experimental task. According to the subtraction logic, only the task-relevant – hence domain-specific – activations should remain. In order to achieve the best possible match between experimental and control tasks, we created stimuli that could be used across all three task conditions. In particular, stimuli consisted of two simultaneously displayed children’s hands with coloured thumbs. In half of the trials the palms of the two hands showed in the same direction, while in the other half they did not. The spatial task required participants to judge whether the palms of the two hands were showing in the same direction or not. Likewise, in the colour condition, individuals were asked to state whether the colours of the two thumbs were identical or not. The colour task served as a true control task. The spatial task was incorporated in the study because our main aim was to disentangle spatial and non-symbolic numerical processing (Walsh 2003; for a comprehensive review on the neurofunctional overlap between spatial and numerical processing see Hubbard et al. 2005).

References


Relevance of Neuroscience to Effective Education for Students With Reading and Other Learning Disabilities

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ABSTRACT

New directions in educational assessment and instruction are supported by recent advances in the neurosciences. Among these are early identification of potential learning problems through brief, efficient assessments of specific language skills that predict later reading outcomes; early intervention that systematically targets critical linguistic processing skills; and the necessity of stimulating all functions of a reading, writing, or computing brain. (J Child Neurol 2004;19:840-845).

Our understanding of language-based reading difficulties and their remediation has grown considerably in the past three decades, although conceptual and practical challenges in the identification, classification, and treatment of such children have been numerous. Progress in understanding reading disabilities has resulted from a comprehensive, Federally-funded research program in basic and applied sciences, including developmental psychology, cognitive psychology, cognitive neuropsychology and functional neuroimaging, pediatric neurology, genetics, and educational psychology. In pursuit of therapeutic models for reading disabilities, researchers have explored the mechanisms and processes of normal reading development, discovering that the role of visual and tactile-kinesthetic (manual) pathways is secondary to the roles of linguistic awareness and language proficiency. Even so, the functional and anatomic brain differences underlying reading disabilities are complex and involve multiple sensorimotor and cognitive anomalies. To teach reading, then, is to teach language processing by ear, by voice, by eye, and by hand.

What we have learned about reading disabilities and effective reading instruction is relevant to understanding a very large group of children. About 4 in 5 of the 6% nationally who are classified as learning disabled experience primary difficulty learning to read, write, and spell and can be diagnosed with language-related learning disabilities. However, about 17% of all students exhibit the phonologic processing problems that put them at risk of reading failure, and in high-poverty environments, many more are at risk. The National Assessment of Educational Progress reports that 38% of all students nationally experience significant difficulty learning to read by fourth grade and are "below basic"; they do not read well enough to participate in grade-level classroom instruction.

The vast majority of poor readers in our schools do not exhibit IQ achievement discrepancies on psychoeducational testing and thus would not meet the diagnostic criteria for learning disabilities and special education eligibility that have been in favor for the past 30 years. Many of these children attend schools in which reading failure is the norm. Others do not demonstrate the comorbid behavioral problems that lead to special education referrals. Moreover, those who are classified do not have unique language or cognitive characteristics when they are compared with unclassified poor readers, and children's response to instruction is predicted and accounted for by factors other than IQ, such as phonologic processing and speed of letter naming. Special education, as it is typically managed and delivered today, leaves classified students without significant gains or even specialized instruction and usually does little for the larger group of unclassified students who also need research-based treatments.

Conceptions of reading disability, reading development, and reading instruction that are informed by the neurosciences could move us toward more effective treatment modalities. Such models provide a rationale for early identification of all children at risk of reading failure, for the use of assessments designed to predict long-term reading outcomes, and for instructional approaches and methods that educate all relevant brain systems. Models consistent with the findings of neuroscience can help educators reach almost all children who struggle to acquire basic academic skills. Research is far more extensive in early reading acquisition than in other areas.
of educational performance, but other types of learning disabili-
ties and academic problems can also be addressed by research-
based interventions, including instruction in language
comprehension, written expression, mathematics reasoning and
computation, and adaptive behavior. This article offers a concep-
tualization of informed treatments of the future, consistent with
brain science, that are possible with better teacher preparation,
stronger administrative leadership, revised policies regarding stan-
dards and the allocation of resources, and wiser use of technology.

TEACHING THE BRAIN TO READ

Preparing the Brain to Read
The neural circuitry recruited for reading develops in interaction
with environmental stimulation. Studies of genetic influences on
reading, for example, indicate that about half of the variance in
achievement is attributable to biologic “wiring” or aptitudes and
half is attributable to life experience.12 The roots of literacy begin
long before entry into formal schooling with preschool develop-
ment of language. Infants until the age of 6 to 8 months are capa-
ble of processing all possible phonemes in all human languages,
but before 10 months of age, they have become attuned to the
phonologic systems and grammatic systems of their caregivers’ lan-
guage. Differentiation of phonemes in all language systems (eg, Chi-
inese phonemes in English-exposed babies who have no experience
with Chinese-speaking caregivers), which is possible for a 6-month-
old child, is no longer observed in infants over age 10 months.10 Cir-
cuity for language is generated and fine-tuned, probably from
birth, to enable what should be the period of most rapid growth
in vocabulary and syntax.

Normally developing children know the meanings of at least
5000 base words before entering first grade.14 Children who reside
in low-verbal, less educated families, however, receive about one
third the hours of verbal stimulation, and the verbal interactions
to which they are exposed are more punitive, directive, and con-
tent deprived than are those of more educated, middle class, or pro-
fessional families.15 Children who are at risk of reading failure
often demonstrate striking delays in vocabulary acquisition, in
addition to limited book exposure and other specific indicators of
risk, such as a lack of familiarity with alphabet letters or the speech
sounds they represent. To prepare preschool children for literacy,
then, community-based early childhood programs could begin at
about 18 months when dendrites are proliferating in the left hemi-
sphere and expressive language is emerging. The encouragement
of parents and caregivers to use nurturing tones and articulated
phrases with toddlers while talking about shared experiences,
even before the babies can articulate a response, is of critical
importance because posterior cortical systems for language com-
prehension are developing faster than anterior systems for verbal
production.16

Teachers and caretakers of toddlers can be taught conversa-
tional skills and awareness of their use of language during meal
times, play times, activities, and book reading.17 Teaching parents
and child care workers how to foster children’s language devel-
OPMENT accelerates children’s vocabulary acquisition and expres-
sive language and increases their chances of reading success in first
grade. Strategies such as the use of structured dialogue during

Assessment to Locate Children at Risk
Assessment procedures used by teachers from preschool onward
can measure critical predictors of later reading achievement.18 Chi-
ldren at risk can be located before they have experienced emo-
tionally damaging struggles with basic reading skills. Among the
critical indicators of later passage comprehension and reading flu-
ency that can be measured before children actually learn to read
are knowledge of letter names, the ability to identify the phonemes
or speech sounds in one-syllable words (chuse = /ch/ or /s/), the pro-
duction of the speech sounds that graphemes (letters and letter com-
binations that correspond to phonemes) represent, the ability to
sound out simple nonsense syllables using phonic correspondences
(fem, zis), and vocabulary knowledge. Moreover, such indicators
are far more reliable and predictive when the measures are timed
and the student’s speed of response or fluency is evaluated.18

As reading is learned, the normally developing brain increas-
ingly activates posterior areas involved in instant word recognition.19
Fluency, or speed of association of print with speech, is not only
a characteristic of proficient reading, but a lack of fluency in com-
ponent reading skills is one of the most enduring characteristics
of poor readers.20 In effect, the aim of early assessment is to deter-
mine whether students are on course to fluent reading as a result
of typical classroom instruction or whether they need more inten-
sive instruction that will normalize the brain’s activation profile.21
Observations of the learning brain have verified the importance of
automatizing the building blocks of reading, including speech
sound identification and recall, letter recognition, alphabet recall,
sound-symbol association, and instant word recognition.

In classrooms organized to prevent reading failure, children
are screened three times per year from kindergarten onward and
interventions are begun immediately for those who fail to reach a
predictive benchmark score on critical indicators of later reading
success. If children are “at risk,” instruction can be designed to build
both accuracy and fluency in the weak skills, and the response to
instruction can be monitored with brief, focused assessments.
Crucial to the efficacy of this endeavor, however, is the realization
that reading is a multicomponent process subsumed by several func-
tional brain networks, each recruited for a specific purpose: phono-
logic processing, orthographic processing, morphologic and
semantic processing, and syntax and discourse processing. As the
brain learns to read, the component processors must be educated
to perform specific functions well so that smooth, automatic
functioning of the reading brain is possible. Well-designed lessons will include a number of components: explicit teaching about letters, speech sounds, phonics and spelling, vocabulary, and comprehension, integrated into a coherent, systematic progression.22

Educating the Phonologic Processor
Most poor readers are slow and inaccurate at printed word recognition and are relatively weak on phonologic tasks, such as saying the individual phonemes in words, manipulating a phoneme sequence by deleting and recomposing sounds, or repeating a novel sequence of syllables.23 Children will vary, however, in the extent to which they need remedial instruction in speech sound awareness. Although the majority of poor readers demonstrate a core deficit in phonologic processing, some children are fast but inaccurate (phonologically impaired but not rate impaired), some are accurate but slow (rate impaired), and some are inaccurate and slow (with a “double deficit”).24 All core classroom programs should include systematic and explicit instruction in speech sound awareness,25 but those children who do not respond well and who require remediation can vary in their need for explicit teaching of components such as speech sound awareness, fast word identification, vocabulary, or other reading comprehension skills.26 Phonologic skill is necessary, but not sufficient, for proficient reading, and the program in use must allow the teaching emphasis to vary according to student need.

Phonologic awareness instruction should follow a developmental sequence of skill acquisition.27 Methods that draw the child’s attention to the oral-motor formation of speech sounds are particularly powerful. As the teacher models and instructs about the mouth movements that characterize each speech sound, children with a “tin ear for language” can learn to distinguish similar speech sounds such as /k/ and /g/ and can then segment and blend phonemes into whole words (/hl + /ou/ + /s/). The teacher provides something that technology cannot: modeling of the mouth forms and corrective feedback for the student who must focus on the features of the sounds. Once children learn to notice the internal details of the spoken word, they are more likely to be successful at mapping print to speech.

To become fluent, the child must be helped to unitize percepts of words and recognize them instantly, and this can be accomplished even with intermediate and older poor readers given instruction that is sufficiently intense and skillful.28 Intervention studies verify that students with reading disabilities require about 30 minutes daily in first grade and up to 2 hours daily in third grade and beyond to normalize their functional brain systems for reading and that conceptualization of the phoneme is the reference point for learning an alphabetic orthography. Lessons begin with sounds, link sounds to symbols, and link words to meaning.

Neuroimaging studies show not only that children and adults with reading disabilities are distinguished by underactivation of the temporoparietal areas of the left hemisphere but also that younger and older poor readers can activate right hemisphere networks that are atypical of good readers.29,30 Thus, remediation for biologically dyslexic students is more likely to result in compensatory adjustments in functional brain systems than to eradicate neurobiologic differences. In a well-designed treatment program, planning for students is long term. It includes direct remediation of phonologic skills while simultaneously providing compensatory tools and strategies, such as proofreading assistance for poor spelling, time extensions for slow reading, books on tape, and academic study strategies.

Educating the Orthographic Processor
Informed instruction will enable students to recognize printed words with fluency and accuracy, activating posterior cortical regions where orthographic processing takes place and where “sight word” images are stored. In the beginning stages of reading, children might know a good deal about letter sequences and print conventions through incidental exposure to them. Some children discover sound-letter correspondence from just a few encounters; many others, however, are dependent on explicit instruction that calls their attention to letter forms and letter sequences. The goal of phonics and spelling instruction is to develop the child’s explicit awareness phoneme-grapheme correspondence, which, in turn, supports recognition and recall of whole words.31

Each individual lesson should juxtapose exercises aimed at stimulating the essential components of a unitized, functional brain system to support word recognition. Fast communication among multiple processing networks is more likely if each is addressed separately and together. Partial approaches, for example, phonics instruction without direct and immediate application to reading and writing, have little justification within a neuroscientific approach. Rather, a variety of skills are practiced briefly and successively; children who are just learning to match sounds with symbols might search a printed page for target words in a text; practice naming upper and lower case letters with fluency; learn a grapheme for a speech sound and review known sound-symbol matches; review previously learned sight words; practice blending new printed words using phonic correspondences; and read text with learned words and patterns. Lesson design combines targeted work on component skills with the transfer and application of those skills in purposeful reading tasks.

Sound-symbol linkages, the meat of phonics instruction, are not sufficient to educate a well-functioning orthographic processor. Proficient recognition of larger units is necessary for fluent decoding of longer words that are often found in literary and informational text. Lesson design at this level incorporates words from the Latin and Greek layers of English but focuses on syllabication and morphology because memory for these larger units facilitates pronunciation of novel words encountered in text. Morphemes are the smallest meaningful parts of words and include inflectional endings (-s, -ed, -ing, -er, -est) and parts of compounds; Latin prefixes, suffixes, and roots; and Greek combining forms common in mathematical and scientific vocabulary. Morphemes are often spelled consistently; thus, a direct link can be made between visual print patterns and units of meaning (trans + port + ation; uni + cyc + le). Reading lessons should aim to include comprehensive, sustained instruction about word structure, including phonologic, orthographic, and morphologic correspondence units, throughout reading, writing, and vocabulary study from first grade to high school.

In summary, informed instruction about print will direct children’s attention to the anatomy of words, both spoken and written. Mental connections between subword units (sounds, syllables, and morphemes), whole words and their semantic networks,
syntax, and the structure of the text itself are established by combining skill practice with reading worthwhile material for well-defined purposes. The principles of redundancy within and across lessons and intensive practice to achieve automaticity reflect the brain's need for repeated stimulation to establish communicative pathways. Because reading fluency enhances reading comprehension and comprehension enhances reading fluency, lessons are most effective when all components are addressed in ways that promote connections among multiple brain systems. Meaning-making, a complex enterprise, can then be facilitated by a purposeful teacher with the support of technology.

Path to Comprehension

Effective teaching of beginning reading engages attention, supports memory, and encourages self-regulation and self-correction. It is not based on rote drill but rather encourages transfer of learning to reading for meaning through teacher guidance. The teacher leads the children into the insights necessary to comprehend; she does not assume comprehension simply because the words are being read. Reading together, reading alternately, and reading silently for specific purposes are the vehicles for active questioning and discussion. The teacher's role is to preview the text with the children, provide background knowledge, engage children in connecting the topic to their own experience, and promote the active use of reading comprehension strategies. Those strategies include constructing mental images, predicting, summarizing, questioning, and clarifying and are taught by the teacher's modeling or "thinking aloud" before, during, and after reading and by queries during shared reading. Technology can enhance this process if text is presented electronically, and the reader can ask for clarification and background information when words, phrases, or passages are not understood.

A continual demand of comprehension is inference-making and the mental construction of idea networks or mental models of the information presented. To this end, graphic organizers—visual maps of conceptual relationships—can help children understand relationships among ideas. The task of writing a summary or retelling a narrative requires children to form mental models of overall text structure, but most children with language learning disabilities need considerable scaffolding or teacher support to approximate this challenging task, along with specific steps to follow. Skill is developed over time with extensive practice in recognizing and formulating main ideas versus supporting details, ordering ideas in hierarchical networks, using graphic organizers and outlines, and recognizing various types of paragraphs. Through coaching and demonstration, teachers can learn to question at various levels, probing the meanings of words, phrases, sentences, and discourse that language-impaired students might misunderstand and modeling proven comprehension strategies as needed.

Technologic supports can assist teachers in matching children with text at the right difficulty level on topics of interest to the student. Computer programs now exist that parse any text into phrase-size units, so that the reader avoids word-by-word reading and can pick up the phrase contours in sentence structure. Other instructional packages provide videotaped background information about informational text in science or history to familiarize students with key concepts and terms before they tackle challenging passages.

TEACHING THE BRAIN TO WRITE

Language and writing difficulties can variously originate in graphomotor, visual-spatial, attention, memory, and language formulation processes. Beginning writers must acquire automatic transcription skills to produce well-formulated compositions. Instruction that applies what we know about the brain will incorporate early training in letter formation accuracy, phoneme-grapheme correspondences, spelling of high-frequency words, punctuation, and handwriting fluency so that attention and memory can be deployed in the service of language formulation, audience awareness, and discourse organization. Direct teaching of self-regulatory strategies enhances writing length and quality in learning-disabled students in the intermediate and middle grades.

Parallel emphasis on direct teaching of component skills within a lesson that includes guided composition differs considerably from the currently dominant "writers' workshop" approach in regular classrooms, in which cumulative skill instruction is deemphasized in favor of naturalistic, holistic, process-oriented writing activities. It is true that writing instruction for all children should be organized around real communicative goals established in social contexts so that skills are applied immediately in the service of "real writing," but skills are a necessary foundation for success. The skills part of a lesson, in which letter formation, letter naming, alphabet production, handwriting fluency, and spelling are emphasized, is brief (5 to 15 minutes, depending on students' ages). Frequent changes of activity and immediate, corrective feedback are necessary to engage short attention spans because the brain responds to novelty and to success. After working on component skills in the first part of a lesson, young children can be engaged in shared writing of a structured composition, in which teachers model and discuss the processes of planning, translation of words into print, and review of what has been written. Awareness of audience is fostered throughout writing instruction through peer and teacher conferences and publication of articles and books.

One reason for emphasizing component skills and habit formation in the beginning of the writing lesson is that practice is more likely to generalize to composition that follows immediately. It is not productive, especially for children with learning disabilities, to give negative feedback on multiple errors of spelling, grammar, and sentence structure after a composition is completed. Rather, good preparation for writing, supportive feedback during writing, the use of reference materials such as high-frequency word lists, and grading that focuses on the content and ideas are the best ways to avoid the negative emotions and aversive reactions that many students develop when writing becomes a punishing experience.

Other adaptations of instruction that help children with writing disabilities include early instruction in keyboarding and word processing, limiting handwriting instruction to either manuscript or cursive, using voice-activated text generation devices on the computer, and planning with the help of interactive software that prompts the writer to formulate key parts of the chosen genre. At the very least, a technology-based secretary can provide relief from the stress of transcription and rewriting and thus enhance motivation and enjoyment.
Learning disabilities in mathematics are more difficult to categorize and identify than disabilities in language, reading, and writing. Less research exists about what to teach, to whom, at what point in development, and with what methods. The territory of mathematical reasoning differs from that of reading; it is more conceptual than symbolic, although each academic domain encompasses conceptual and symbolic skills. To reason mathematically, the learner develops both quantitative and logical mental models that are distributed broadly in interconnected neural networks.

Many general principles important for addressing the attention, memory, and linguistic processing problems of students with reading and writing difficulties apply to math instruction as well. Automaticity in lower-level skills frees up the mental “desktop” for higher-level reasoning and problem solving. Thus, children need to learn their math facts, graphomotor notation of numbers, and execution of basic number operations through brief, frequent drills. Both mental arithmetic and paper-and-pencil production should be practiced. The teacher must explain the concepts and procedures using manipulative aids and concrete representations when necessary and then must orchestrate the transfer and application of skills to problem solving. Classroom discussion and collaboration during problem solving is as important to mathematical learning as it is to reading and writing.

NEUROSCIENCES AND EDUCATION: WHAT WOULD BRAIN-BASED EDUCATION MEAN FOR STUDENTS WITH LEARNING DIFFERENCES?

If schools, classrooms, instructional programs, and teacher education were to be organized according to principles supported by neuroscience, assessments of global maturity, intelligence, and readiness would be replaced by assessments of proficiency in specific neurodevelopmental domains: gross motor; fine motor; reasoning and problem solving; visual-spatial and nonverbal cognition; language processing at the levels of phonology, orthography, syntax, semantics, discourse, and pragmatics; attention; executive functions; and social-emotional development. Variations of development would not be used to delay entry into school or to retain children who are developmentally or academically delayed. Rather, children at risk of academic failure would be identified early—at least as early as the beginning of kindergarten, if not sooner—and given the benefit of focused intervention in small groups with similar instructional needs. Educators would not wait to see what “more time” would bring because organized instruction benefits children with developmental delays more than maturation alone. Grade retention would go the way of other antiquated, unsupportable practices. Retention would be replaced by special instruction initiated for all children who scored below critical predictive benchmarks at the beginning, middle, or end of an academic year.

Children in flexible, homogeneous intervention groups of three to six children would receive focused instruction either within the classroom or outside it. Focused intervention targets the specific component skills of reading, writing, language, or math in which a student is delayed; uses validated instructional approaches; and gives ample practice so that students become accurate and fluent in the application of component skills to academic endeavors. Additional practice occurs with the assistance of supportive technology, parent and paraprofessional tutoring, or peer-assisted learning strategies. With progress monitoring assessments given on a frequent basis, teacher teams can decide who might need even more intensive, individual work. Interventions would be delivered before expensive diagnostic testing and the development of an individual educational plan to maintain focus on the curriculum and the child’s response to instruction. When problems persist or the response to instruction continues to be poor, children would be referred for a multidisciplinary evaluation to determine the nature and extent of a handicapping condition.

The context for instruction would be schools organized to invest resources preventively. The school schedule would permit teachers to work with whole groups, small groups, and individuals for sufficient amounts of time to realize meaningful gains. Teachers would be expected by administrators to use validated instructional tools for specific purposes rather than to reinvent good practices on their own. Skillful implementation of proven practices would be the first expectation for teacher evaluation. Student growth would be the primary consideration for all instructional decision making rather than performance on or preparation for arbitrary accountability measures that might have little relevance to students’ instructional needs. Teachers would work with teams who shared common understandings about neurodevelopmental variations and the acquisition of reading, writing, language, and mathematical competencies. Schools would be adequately staffed to permit reasonable class size, small-group and individual instruction, and ancillary services as needed.

As this article has described, teaching is an enormously complex endeavor if it is done well. A skilled teacher who implements brain-compatible practices does many things, often simultaneously and with groups of diverse individuals. The teacher seeks actively to develop all of the functional components of complex behavioral and cognitive systems, separately and in combination. She or he plans instruction to include brief and varied routines to maintain attention and motivation. The teacher plans for transfer of skills to holistic application and ensures that sufficient practice occurs for long-term retention. The teacher often stands in for immature executive functions in learners, providing organization, scaffolding, and feedback as needed. If we could substitute technological solutions for the human interactions required in education, society might be less concerned with the compelling issue of teacher quality and teacher preparation.

Professional organizations such as the International Dyslexia Association, the American Federation of Teachers, and the American Speech-Language-Hearing Association have called for changes in the way teachers are taught to teach reading and the way speech-language therapists function to support literacy development in schools. Teachers are not born with disciplinary knowledge any more than physicians are born with knowledge of anatomy, yet programs of teacher preparation do not normally require teachers to learn the psychology of reading and language, the structure of language, the basics of brain-behavior relationships, or the implementation of research-based practices. When and if they do, children will be much more likely to realize the benefits of neuroscientific research. To that
end, our most urgent challenge is how to attract capable individuals into the profession of teaching, educate them for a demanding job, and support them while they are teaching. Perhaps neuroscientists can continue to inform the public not only about our marvelous insights into the brain at work but also about the sustained human effort required to realize the benefits of those discoveries.

References