Neuroscience, education and special education

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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 oxidise 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, Scheuerpfug, Zillesen, Fallgater & Warnke, 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 paths 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, Schmid & 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 ERPs).

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.

Figure 2: A child wearing a specially adapted headcap for measuring ERPs (event related potentials). I am grateful to Professor Mark Johnson, Director of the Cognitive and Brain Development Centre, Birkbeck College, London, for this image.

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 effects of different kinds of educational input on learning; and an increased understanding of individual differences in learning and the best ways to suit input to learner. I will now describe briefly some recent neuroscience studies in certain areas of cognitive development and try to give a flavour of how their methods could contribute to questions that are more specific to special education.

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 (Holllins, 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 single 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, Mencel, 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 temporop-occipital areas increases with reading skill (see, for example, Shaywitz, Shaywitz, Pugh, Mencel, 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 & Papanico1ou, 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 neuropsychological 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 neuropsychology 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, Berns, 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.


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