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SCIENCE AND SOCIETY

Neuroscience and education: from research to practice?

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Abstract | Cognitive neuroscience is making rapid strides in areas highly relevant to education. However, there is a gulf between current science and direct classroom applications. Most scientists would argue that filling the gulf is premature. Nevertheless, at present, teachers are at the receiving end of numerous 'brain-based learning' packages. Some of these contain alarming amounts of misinformation, yet such packages are being used in many schools. What, if anything, can neuroscientists do to help good neuroscience into education?

There is a hunger in schools for information about the brain. Teachers are keen to reap the benefits of the 'century of neuroscience' for their students. In neuroscience laboratories, considerable progress is being made in understanding the neurocognitive development underpinning essential skills taught by educators, such as numeracy and literacy. This progress is largely theoretical. The current gulf between neuroscience and education is being filled by packages and programmes claiming to be based on brain science. The speed with which such packages have gained widespread currency in schools is astonishing. This article highlights some pervasive 'neuromyths' that have taken root in education, gives a flavour of the information being presented to teachers as neuroscientific fact, and reviews recent findings in neuroscience that could be relevant to education. It also considers what, if anything, we should do now to influence the widespread misapplication of science to education.

Brain-based learning in schools

At a recent conference held to mark the launch of the Centre for Neuroscience in Education at the University of Cambridge[1], teachers reported receiving more than 70 mailshots a year encouraging them to attend courses on brain-based learning. Similar phenomena have been reported in other countries[2]. These courses suggest, for example, that children should be identified as either 'left-brained' or 'right-brained' learners, because individuals 'prefer' one type of processing[3]. Teachers are told that the left brain dominates in the processing of language, logic, mathematical formulae, number, sequence, linearity, analysis and unrelated factual information. Meanwhile, the right brain is said to dominate in the processing of forms and patterns, spatial manipulation, rhythm, images and pictures, daydreaming, and relationships in learning[3]. Teachers are advised to ensure that their classroom practice is automatically 'left- and right-brain balanced' to avoid a mismatch between learner preference and learning experience[3]. This neuromyth probably stems from an over-literal interpretation of hemispheric specialization.

Other courses for teachers advise that children's learning styles should be identified as either visual, auditory or kinaesthetic, and that children should then wear a badge labelled either V, A or K while in school, showing their learning style for the benefit of all of their teachers. Still others argue that adoption of a commercial package 'Brain Gym^{R}' ensures that 'true' education happens. Brain Gym^{R} prescribes a series of simple body movements[4] "to integrate all areas of the brain to enhance learning". Teachers are told that "in technical terms, information is received by the brainstem as an 'impress', but may be inaccessible to the front brain as an 'express'. This … locks the student into a failure syndrome. Whole-brain learning draws out the potential locked in the body and enables students to access those areas of the brain previously unavailable to them. Improvements in learning … are often immediate". It is even claimed that the child can press certain 'brain buttons' under their ribs[4] to focus the visual system for reading and writing.

Many in education accept claims such as these as established fact[5]. Scientists have already alerted society to the neuromyths that are dominant in education at present[6–8]. In addition to the left brain/right brain learning myth, neuromyths that relate to critical periods for learning and to synaptogenesis can be identified. The critical

period myth suggests that the child's brain will not work properly if it does not receive the right amount of stimulation at the right time (an insightful analysis is provided by Byrnes[9]). Direct teaching of certain skills must occur during the critical period, or the window of opportunity to educate will be missed. The synaptogenesis myth promotes the idea that more will be learned if teaching is timed with periods of synaptogenesis[7]. Educational interventions will be more effective if teachers ensure that they coincide with increases in synaptic density. Educational interventions are also sometimes suggested to be superior if they encourage 'neuroplasticity'[10], and teachers are told that neural networks can be altered by 'neuroplasticity training programmes'[10]. Teachers do not realize that, although there might be sensitive periods for some forms of learning, the effects of any type of training programme that changes behaviour will be reflected in the 'remapping' of neural networks.

Neuroscience in the classroom

These neuromyths need to be eliminated. The dominance of these myths obscures the important strides being made by cognitive neuroscience in many areas relevant to education. For example, our understanding of the neural bases of the '3 Rs' — reading, writing and arithmetic — is growing rapidly. So is our understanding of how to optimize the brain's ability to benefit from teaching. Good instructional practice can be undermined by brain-based factors such as learning anxiety, attention deficits and poor recognition of social cues. All of these factors disrupt an individual's capacity to learn, and also have an effect on other learners in the same classroom.

Reading and dyslexia. From work with adults, it is well established that a left-hemisphere network of frontal, temporoparietal and occipitotemporal regions underpins mature reading[11]. However, cross-language imaging studies show some interesting variations. These seem to depend on how the orthography (the writing system) of a language represents phonology (the sounds of the language). When learners of transparent writing systems (for example, Italian) are contrasted with learners of non-transparent (for example, English) or character-based (for example, Chinese) writing systems, highly similar brain areas are found to be active during reading[12,13]. However, mature readers of transparent orthographies show greater activity in the left planum temporale, a brain region involved in letter-sound conversion, whereas mature English readers show greater activation of an area known as the visual word form area (VWFA) in the left occipital temporal region[12]. Although originally proposed as the substrate of visual word recognition[14,15], this neural area has also been proposed to involve phonology — for example, through the computation of orthographic–phonological connections[16,17]. Its greater activation in English could reflect the several levels of spelling-sound correspondence that are important for decoding English[18] (for example, reading BOMIC by letter-sound conversion or by analogy to COMIC). Readers of Chinese show relatively more engagement of visuospatial areas, presumably for recognizing complex characters[13].

Developmentally, it is known from behavioural studies that pre-readers who can recognize phonological similarity (for example, that CAT and HAT rhyme, or that CAT and CUP share the first sound) become better readers. Imaging studies have confirmed that young readers primarily depend on the left posterior superior temporal cortex, the area identified in adult studies as the locus of phonological decoding[19] (FIG. 1). Activity in this region is also modulated by children's phonological skills. As literacy is acquired, the VWFA (described as a 'skill zone' by some developmental neuroscientists[20]) is more engaged and areas initially active in the right hemisphere are disengaged.

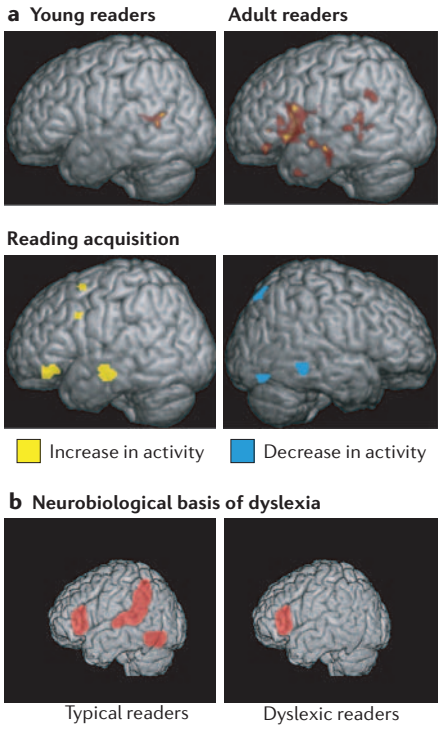

Figure 1 | **Brain areas involved in typical reading development and dyslexia measured with functional MRI. a** | Images in the top panel show the early reliance on the left posterior superior temporal cortex, which is known to be involved in phonological processing, in children learning to read, and the expansive involvement of the left parietal, temporal and frontal cortices in adult readers. Correlations between brain activity during reading and reading ability (measured on standardized tests) demonstrate increased involvement of the left temporal and frontal regions, associated with phonology and semantics, as reading develops (bottom panel). Right posterior activation declines as reading is acquired, presumably indicating reduced reliance on the systems for recognizing non-lexical forms. **b** | Summary of brain regions engaged during reading and reading-related tasks in typically developing readers (left inferior frontal gyrus, left temporoparietal cortex and left inferotemporal cortex) and readers with dyslexia (left inferior frontal gyrus only). Panel **a** reproduced, with permission, from REF. 19 © (2003) Macmillan Publishers Ltd. Panel **b** courtesy of G. Eden, Centre for the Study of Learning, Georgetown University, Washington, DC, USA.

Studies of children with developmental dyslexia (children who are failing to learn to read normally despite average intelligence and educational opportunity) show that, atypically, the right temporoparietal cortex continues to be activated during reading[21]. Children with developmental dyslexia also show significantly less activation in the usual left hemisphere sites. If targeted remediation is provided, usually through intensive tuition in phonological skills and in letter-sound conversion, activity in the left temporal and parietal areas appears to normalize[22,23]. So far, however, developmental neuroimaging studies have been short term and mostly confined to English. Theoretically motivated studies across languages are now required[24].

These developmental imaging studies show that we can begin to pin-point the neural systems responsible for the acquisition of reading skills, and that we can remediate inefficiencies in these systems. However, so far, these studies do not tell teachers 'what works' in the classroom. Most training studies have used interventions already known to be successful from educational research, and have simply documented that neural changes in the expected areas accompany behavioural changes[22,23]. So far, neuroimaging tells us little more, but, the potential is there. For example, imaging offers the possibility of identifying neural indices of a child's potential difficulties, which may be hidden from view earlier in development. We can

attempt to identify neural markers for phonological sensitivity, such as brain responses to auditory cues for rhythm[25], to identify who is at risk of later reading difficulties. Alternatively, we can seek general language markers for dyslexia[26]. In both cases, early identification of infants with poor skills would enable language interventions to prevent dyslexia long before schooling[27].

Studies could also be designed to test neural hypotheses. For example, a popular cognitive theory of developmental dyslexia proposes a cerebellar deficit[28]. A commercial exercise-based treatment programme, the DDAT (Dyslexia Dyspraxia Attention Deficit Treatment)[29], aims to remediate cerebellar difficulties. Children are encouraged to practise motor skills such as catching beanbags while standing on one leg on a cushion. This is claimed to benefit reading. Imaging studies could measure where neural changes occur in response to such remediation, to see whether permanent changes to the neural areas for reading are involved (this seems unlikely — any effects found for reading are probably short-term placebo effects).

Number and dyscalculia. Progress in understanding the underpinnings of arithmetic has been rapid since the proposal that the human brain has dedicated circuits for recognizing numerosity[30]. This 'number sense' capacity depends on parietal, prefrontal and cingulate areas, with the horizontal segment of the bilateral intraparietal sulcus (HIPS) playing a central part in the basic representation and manipulation of quantity[31]. In simple paradigms, in which participants have to decide whether, for example, 3 is larger than 5, the HIPS might be the only region specifically engaged. Activity in the HIPS is modulated by the semantic distance between numbers and by the size of numbers[32]. Other arithmetic operations are more dependent on language-based fact retrieval, such as simple multiplication, which activates the angular gyrus[33].

Some arithmetic operations depend on the mental 'number line'. This is an apparently universal mental spatial representation of number, in which smaller numbers are represented on the left side of space and larger numbers are represented on the right[34]. The interactions revealed between number and space in the parietal cortex have been particularly interesting. Manual responses to large numbers are faster when the response is on the right side of space, and vice versa for smaller numbers[35]. In line bisection tasks, in which participants have to estimate the central point of a horizontal line, midpoint estimation systematically deviates to the left if the line is made up of 2s (222222222…) and to the right if the line is made up of 9s (999999999…)[36]. The numbers automatically bias attention. Patients with visual neglect, a disorder of spatial attention following right parietal damage, systematically neglect the left side of space. These patients show a rightward bias in line bisection tasks. This rightward bias was even found for oral estimation (for example, when asked to state the numerical midpoint of 2 and 6, patients tended to give answers like 5)[37]. Therefore, numerical manipulations seem to depend crucially on intact spatial representations; indeed, blind adults who acquire numbers spatially show normal parietal distance effects[38].

So far, findings from adult neuroimaging and neuropsychological studies remain to be applied to understanding mathematical development in children. One important electroencephalogram (EEG) study showed that when 5-year-old children perform the number comparison task ("is 4 larger or smaller than 5?") they show effects at similar electrodes in the parietal cortex as adults, with similar latencies[39] (FIG. 2). However, reaction time data showed that the children were three times slower to organize the key press response. This imaging experiment raises the possibility that, neurally, young children can extract numerical information as fast as adults. The slow acquisition of calculation skills in the primary years might, therefore, reflect difficulties in understanding arithmetic notation and place value, rather than difficulties in understanding the relationship between digits and quantities. Neuroimaging studies can help us to investigate this possibility. Also of interest to teachers is the evidence for the spatial mental number line. At present, there are various models in schools for teaching children ordinal knowledge of number — that numbers come in an ordered scale of magnitude. The finding that the brain has a preferred mode of representation suggests that teachers should build on this spatial system when teaching ordinality and place value — for example, through teaching tools such as the 'empty number line'[40,41].

Developmental dyscalculia occurs when a child experiences unexpected difficulty in learning arithmetic in the absence of mental retardation despite adequate schooling and social environment[42]. One possible neural explanation is that the core quantity system in the HIPS has developed abnormally. This possibility was investigated by a functional MRI (fMRI) study of girls with Turner syndrome[43], who typically present with visuospatial and number processing deficits[44]. Sulcal morphometry using new techniques[45] revealed that the right intraparietal sulcal pattern of most patients with Turner syndrome showed aberrant branching, abnormal interruption and/or unusual orientation[43]. It was suggested that this anatomical disorganization could explain the visuospatial and arithmetic impairments found behaviourally. A study of very low birthweight children with arithmetical difficulties found reduced grey matter in the left intraparietal sulcus[46]. Control studies are now required to determine whether the parietal sulci are abnormal in other developmental syndromes that do not present with arithmetical difficulties. If parietal abnormalities characterize only children presenting with arithmetical impairments, this would imply a direct link between the brain and behaviour. Children without apparent developmental syndromes who present with unusually poor number processing in the classroom would then need to be assessed for parietal damage.

Attention, emotion and social cognition. The short attention spans of some children pose continual problems for their teachers. Children with attention deficit/hyperactivity disorder (ADHD) are particularly challenging to educate, as they are inattentive and impulsive, cruising the classroom instead of focusing on their work. Of course, all young children experience some difficulties in sustaining attention and inhibiting impulses. Perhaps attentional training might benefit all preschoolers[47], leading to educational advantages?

A recent brain imaging study claimed that 5 days of attention training significantly improved performance on tests of intelligence in 4- and 6-year-old children[48]. The children were given training exercises to improve stimulus discrimination, anticipation and conflict resolution. For example, they learned to track a cartoon cat on a computer screen by using a joystick, to anticipate the movement of a duck across a pond by moving the cat to where the duck should emerge, and to select the larger of two arrays of digits when conflict was introduced by using smaller digits to present the larger array. Attention was tested before and after the training exercises by asking children to press a computer key to indicate which direction the central fish in a row of five fish was facing. Before training, the children were also given an intelligence test, and the same test was repeated after 5 days

of training (which in itself would improve performance, due to item familiarity). Children in the control group either received the attention and intelligence tests only, or attended the laboratory for five sessions of watching popular videos. No matched computer training with animal cartoons was provided to train a control skill, such as memory. Even so, attention training did not improve performance in attention. Instead, an effect of attention training was found for one of the intelligence tests. Scores on the Matrices subtest improved by a significant 6.5 points for the trained 4-year-olds only. EEG data were then collected to determine whether neural conflict-related attentional effects familiar from adults would be found in the trained children. The effect sought was a larger frontal negativity for incongruent trials at the frontoparietal electrodes, particularly at Cz. Despite the lack of behavioural effects, an electrophysiological effect was found for the trained 6 year olds at the target electrode (Cz). For the trained 4 year olds, a 'hint of an effect' was found at a different frontal electrode (Fz). From these single electrode results, it was argued that the executive attention network can be influenced by educational interventions during development. However, as the attention intervention did not affect the children's performance in the attention tasks, further research is needed to support this conclusion. Unusually, the authors offer their training programme free through the Organization for Economic Cooperation and Development, enabling other scientists to test its effectiveness. This is to be highly commended.

The neural substrates for emotional processing are increasingly well understood. For example, the amygdala is known to be important for the interpretation of emotional and social signals, particularly from the face and eyes[49]. In adults, the degree of amygdala activation is particularly correlated with the intensity of facial expressions of fear[49]. Children, too, show amygdala activity to fearful expressions, and children with autism (who have impaired social cognition) have significantly increased amygdala volume[50]. The anatomical system involved in fear processing could be abnormal from an early age in autism, as was suggested by a recent EEG study with 3 year olds[51]. The mirror neuron system in the inferior frontal gyrus is also involved in understanding the emotional states of others[52]. The results of a recent fMRI study showed no activity in this area in children with autism when compared with typically developing children during the

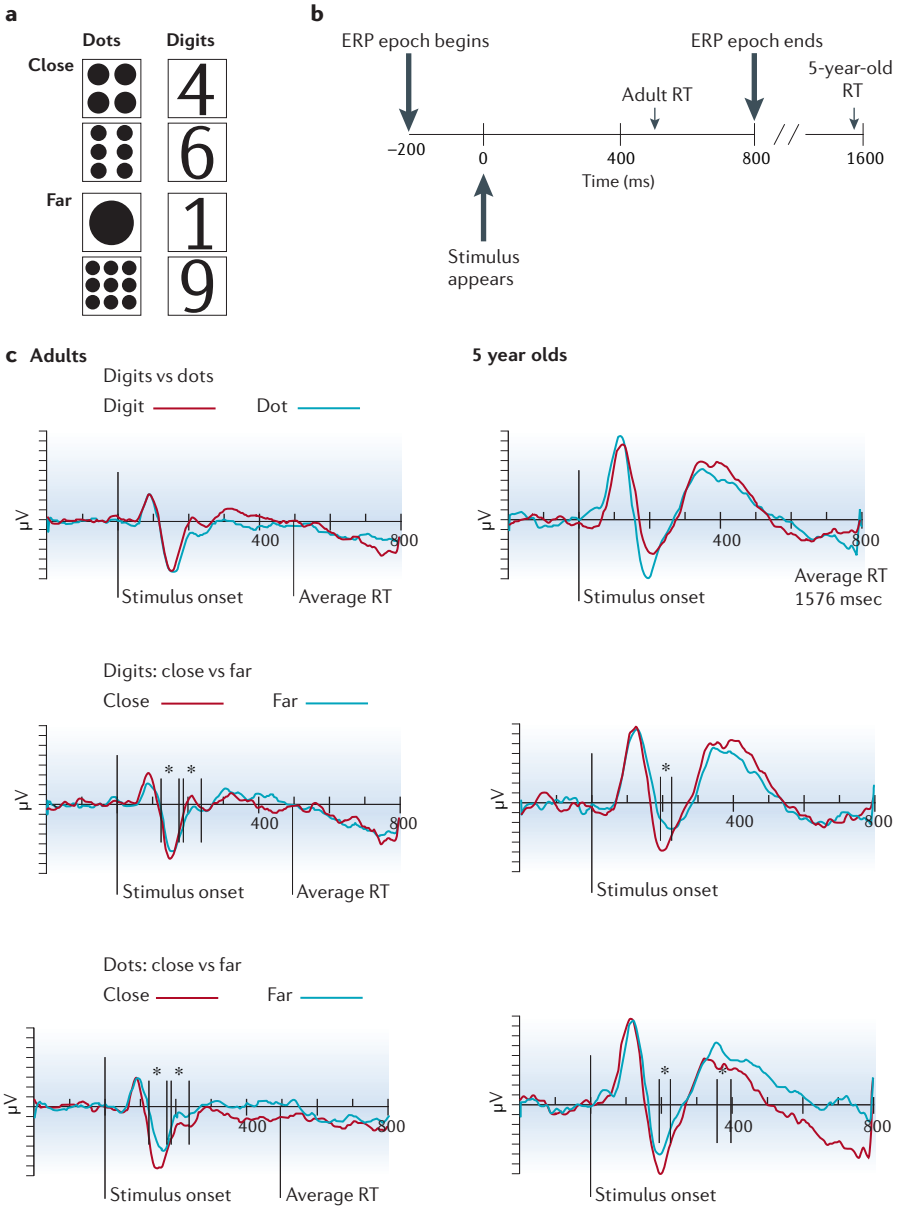

Figure 2 | **Electrophysiological recordings of activity during number processing tasks in children and adults. a** | Participants were shown numbers, represented by either dots or digits, and required to press a response key with their left hand if the numbers were smaller than 5, or with their right hand if the numbers were larger than 5. In adults, the typical finding in such tests is that responses are faster when numbers are distant (for example, 9 or 1) rather than close (6 or 4) to 5; this is called the distance effect. Behavioural data indicated distance effects for both adults and children in this task. **b** | A schematic depiction of the event-related potential (ERP) procedure. Recording of brain activity began 200 ms before and ended 800 ms after stimulus onset. Within this recording epoch, voltage changes associated with the behavioural distance effect for adults and children were found at similar parietal electrode sites. However, the schematic shows that the key press response required ~500 ms for the adults, but ~1,600 ms for the children. Whereas numbers seem to be recognized at similar latencies by children and adults, organization of the required response takes much longer for children. **c** | Representative posterior channel (91) comparing ERPs in adults and 5 year olds for the number comparison task. The *x*-axis is in milliseconds and corresponds to a 1-s epoch of recorded electroencephalogram (EEG; 200 ms baseline, 800 ms poststimulus). Top panel, notation effects (digits versus dots). The two age groups show qualitatively similar initial components (P1, N1 and P2p) with only slightly delayed peaks in the 5 year olds. Middle panel, ERP distance effect for digits in both age groups. Bottom panel, ERP distance effect for dots in both age groups. Significant differences associated with distance began in children 50 ms after adults despite children having reaction times (RTs) that were >1,000 ms longer. Asterisk denotes significant differences at $p < 0.5$. Modified, with permission, from REF. 39 © (1998) National Academy of Sciences.

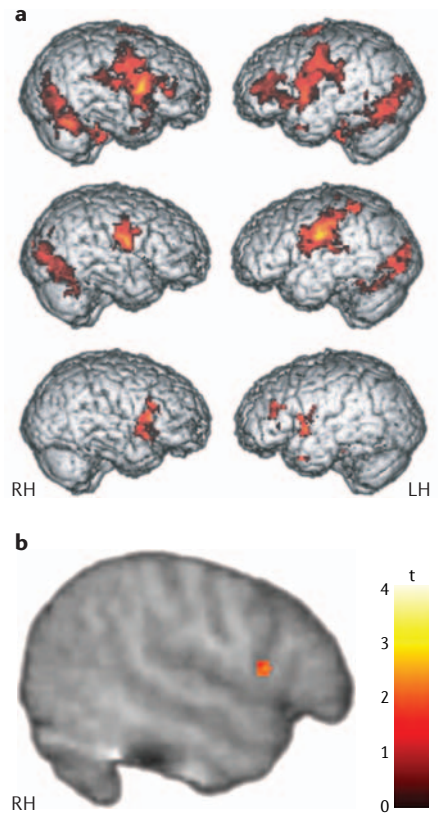

Figure 3 | **Neural activity during imitation and observation of emotional expressions for typically developing children and children with autism spectrum disorders. a** | Shows brain activation recorded during imitation of emotional expressions. Activity in the bilateral pars opercularis (stronger in the right) of the inferior frontal gyrus is seen in the typically developing group (top panel) but not in the group with autism spectrum disorders (ASD; middle panel). A between-group comparison (bottom panel) revealed that this difference is significant (t >1.83, p <0.05, corrected for multiple comparisons at the cluster level). RH, right hemisphere; LH, left hemisphere. **b** | Activity in the mirror neuron system during the observation of emotional expressions[53]. The right pars opercularis showed significantly greater activity in typically developing children than in children with ASD (t >1.83, p <0.05, small volume corrected). Reproduced, with permission, from REF. 53 © (2006) Macmillan Publishers Ltd.

imitation of emotional expressions[53] (FIG. 3). Mirror neurons appear to mediate our understanding of emotional states via imitation, allowing the translation of an observed action (such as a facial expression) into its internally felt emotional significance[52]. This translation appeared to be absent in autism.

Research such as this allows us to study the neural underpinnings of emotional processing in children in mainstream schooling. For example, children exposed to harsh discipline and physical abuse at home seem to process emotions differently from other children[54]. In later childhood they are also more likely to have conduct disorders that make them difficult to teach[55]. Such children are prone to an anger attribution bias, tending to (mis)attribute anger to the actions and statements of others[54]. So far, little neuroimaging work has been done with such children. If atypical brain development is found, and if training programmes can be devised to improve these children's reading of social signals, this would be of benefit to education. We already know that it might be possible to teach children with autism to 'read' emotions to some degree[56]. Optimal interventions for other groups of children could also be designed, with imaging data helping to pinpoint the brain networks to be targeted.

A similar logic applies to learning anxiety. Neuroimaging studies of anxiety disorders in adults focus particularly on structural and functional changes in the orbitofrontal cortex (OFC) and the temporal lobes, including the amygdala[57]. Anxiety disorders are known to increase following traumatic brain injury (TBI). A neuroimaging study of children aged 4–19 years with severe TBI showed that children with more damage to the OFC were less likely to develop anxiety disorders[58]. The authors suggested that an imbalance in the OFC–amygdala connection could influence the expression of anxiety, and pointed out that in non-human primates these connections begin to develop during gestation. Anxiety disorders can be treated, and neuroimaging in adults suggests that some beneficial treatments target the amygdala[59]. As in adults, anxiety in children appears to affect attentional systems, leading children to selectively shift attention towards threatening stimuli[60]. Again, it might be possible to devise early interventions for such children, and to use neuroimaging to identify who is most likely to benefit.

Can we bridge the gulf?

While we await such developments, can we bridge the gulf between neuroscience and education by speaking directly to teachers, and sidestepping the middlemen of the brain-based learning industry? We are trying to do this in our UK seminar series, and through the International Mind, Brain and Education Society[1,61]. For example, at the Cambridge conference, prominent neuroscientists working in areas such as literacy, numeracy, IQ, learning, social cognition and ADHD spoke directly to teachers about the scientific evidence being gathered in scientists' laboratories. The teachers were amazed by how little was known. Although there was enthusiasm for and appreciation of getting first-hand information, this was coupled with frustration at hearing that many of the brain-based programmes currently in schools had no scientific basis. The frustration arose because the neuroscientists were not telling the teachers 'what works instead'. One delegate said that the conference "Left teachers feeling [that] they had lots stripped away from them and nothing put in [its] place". Another commented that "Class teachers will take on new initiatives if they are sold on the benefits for the children. Ultimately this is where brains live!".

This last comment surely provides an insight into the success of the brain-based learning industry. Inspirational marketing ensures that teachers who attend these conferences do get 'sold' on the supposed benefits of these programmes for the children that they teach. Owing to placebo effects, these programmes may indeed bring benefits to children in the short term. However, such programmes are unlikely to yield benefits in the long term, and so many will naturally fall out of use (one teacher commented "We no longer make children wear their VAK badges"). The question for society is, should neuroscientists do anything about this misuse of science? After all, each of these programmes will have a natural life, and will then go away. Only findings for the classroom that are really based on neuroscience will endure. So should we do anything now?

At least two lessons for science and society have emerged from efforts to bring together neuroscience and education[1,62,63]. The first is the immense goodwill that teachers and educators have for neuroscience — they are very interested in neuroscience, they feel that we have the potential to make important discoveries about human learning, and they are eager to learn about these discoveries and to contribute ideas and suggestions. Many teachers have found attending these conferences an intellectually exhilarating experience. The second lesson is that neuroscientists may not be those best placed to communicate with teachers in any sustained way. The scientists are seen as too concerned to establish the rigour of their experimental manipulations, and as providing too much data. Most teachers prefer broad brush messages with a 'big picture', and being 'told what works'. Neuroscientists are not necessarily gifted at communicating with society at large, and they are appropriately cautious about saying that something 'works'.

It may be of most use to society if we as scientists foster and support a network of communicators of our research — individuals who can bridge the current gulf between neuroscience and education by providing high-quality knowledge in a digestible form. These communicators could function in a similar way to the information officers of medical charities, but, in this case, explain what neuroscience breakthroughs mean for the child in the classroom. Ideal communicators would be ex-scientists with an interest in education, perhaps attached to universities or to national education departments. They could fulfil a dual role: interpreting neuroscience from the perspective of and in the language of educators, and feeding back research questions and ideas from educators to neuroscientists. In my view, we should not remain quiet when claims that we know to be spurious are made, such as that children can organize themselves for reading and writing by pressing their 'brain buttons'. Nevertheless, it might, ultimately, be of most value to society if we empower our own middlemen, communicators who know who to consult for expert advice on the latest claims of the brain-based learning industry, and who are clearly working in the public interest and not for profit. A network of such communicators would serve us all (and our children), and would prevent society from pouring precious educational resources into scientifically spurious applications.

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1. Economic and Social Research Council Teaching and Learning Research Programme (ESRC TLRP) seminar series. *Collaborative Frameworks for Neuroscience and Education.* [online], <www.tlrp.org> Education and Brain Research: Neuroscience, Teaching and Learning conference. 25–27 July 2005, Faculty of Education, University of Cambridge, UK.
2. Stern, E. Pedagogy meets neuroscience. *Science* **310**, 745 (2005).
3. Smith, A. *Accelerated Learning in the Classroom* (Network Educational Press Ltd, Bodmin, UK, 1996).
4. Cohen, I. & Goldsmith, M. *Hands On: How to Use Brain Gym® in the Classroom* (Hands On Books, Sea Point, South Africa, 2000).
5. Hoffman, E. *Introducing Children to their Amazing Brains* (LTL Books Ltd, Middlewich, UK, 2002).
6. Organisation for Economic Co-operation and Development. *Understanding the Brain: Towards a New Learning Science* (2002).
7. Bruer, J. T. Education and the brain: a bridge too far. *Educ. Res.* **26**, 4–16 (1997).
8. Blakemore, S. J. & Frith, U. *The Learning Brain: Lessons for Education* (Blackwell, Oxford, UK, 2005).
9. Byrnes, J. P. *Minds, Brains and Learning* (Guilford Press, New York, 2001).
10. Tallal, P. Improving language and literacy is a matter of time. *Nature Rev. Neurosci.* **5**, 721–728 (2004).
11. Fiez, J. A. & Petersen, S. E. Neuroimaging studies of word reading. *Proc. Natl Acad. Sci. USA* **95**, 914–921 (1998).
12. Paulesu, E. *et al.* Dyslexia: cultural diversity and biological unity. *Science* **291**, 2165–2167 (2001).
13. Siok, W. T., Perfetti, C. A., Jin, Z. & Tan, L. H. Biological abnormality of impaired reading is constrained by culture. *Nature* **431**, 71–76 (2004).
14. Cohen, L. & Dehaene, S. Specialisation within the ventral stream: the case for the visual word form area. *Neuroimage* **22**, 466–476 (2004).
15. Dehaene, S. *et al.* The neural code for written words: a proposal. *Trends Cogn. Sci.* **9**, 335–341 (2005).
16. Price, C. J. *et al.* Cortical localisation of the visual and auditory word form areas: a reconsideration of the evidence. *Brain Lang.* **86**, 272–286 (2003).
17. Goswami, U. & Ziegler, J. C. A developmental perspective on the neural code for written words. *Trends Cogn. Sci.* (in the press).
18. Ziegler, J. & Goswami, U. Reading acquisition, developmental dyslexia, and skilled reading across languages: a psycholinguistic grain size theory. *Psychol. Bull.* **131**, 3–29 (2005).
19. Turkeltaub, P., Gareau, L., Flowers, D. L., Zeffiro, T. A. & Eden, G. F. Development of neural mechanisms for reading. *Nature Neurosci.* **6**, 767–773 (2003).
20. Pugh, K. R. *et al.* Neurobiological studies of reading and reading disability. *J. Commun. Disord.* **34**, 479–492 (2001).
21. Shaywitz, B. A. *et al.* Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biol. Psychiatry* **52**, 101–110 (2002).
22. Temple, E. *et al.* Neural deficits in children with dyslexia ameliorated by behavioural remediation: evidence from functional fMRI. *Proc. Natl Acad. Sci. USA* **100**, 2860–2865 (2003).
23. Simos, P. G. *et al.* Dyslexia-specific brain activation profile becomes normal following successful remedial training. *Neurology* **58**, 1203–1213 (2002).
24. Ziegler, J. C. & Goswami, U. Becoming literate in different languages: similar problems, different solutions. *Dev. Sci.* (in the press).
25. Goswami, U. in *Mind, Brain and Education* (eds Fischer, K. & Batro, A.) (Pontifical Academy of Sciences, Rome, in the press).
26. Molfese, D. Predicting dyslexia at 8 years of age using neonatal brain responses. *Brain Lang.* **72**, 238–245 (2000).
27. Goswami, U. Neuroscience and Education. *Brit. J. Educ. Psychol.* **74**, 1–14 (2004).
28. Nicolson, R. I. & Fawcett, A. J. Developmental dyslexia: the role of the cerebellum. *Dyslexia* **5**, 155–177 (1999).
29. Reynolds, D., Nicolson, R. I. & Hambly, H. Evaluation of an exercise-based treatment for children with reading difficulties. *Dyslexia* **9**, 48–71 (2003).
30. Dehaene, S. *The Number Sense* (Oxford Univ. Press, New York, 1997).
31. Dehaene, S., Molko, N., Cohen, L. & Wilson, A. J. Arithmetic and the brain. *Curr. Opin. Neurobiol.* **14**, 218–224 (2004).
32. Pinel, P., Dehaene, S., Riviere, D. & LeBihan, D. Modulation of parietal activation by semantic distance in a number comparison task. *Neuroimage* **14**, 1013–1026 (2001).
33. Dehaene, S., Piazza, M., Pinel, P. & Cohen, L. Three parietal circuits for number processing. *Cogn. Neuropsychol.* **20**, 487–506 (2003).
34. Dehaene, S. & Cohen, L. Towards an anatomical and functional model of number processing. *Math. Cogn.* **1**, 83–120 (1995).
35. Dehaene, S., Bossini, S. & Giraux, P. The mental representation of parity and numerical magnitude. *J. Exp. Psychol. Gen.* **122**, 371–396 (1993).
36. Hubbard, E. M., Piazza, M., Pinel, P. & Dehaene, S. Interactions between number and space in parietal cortex. *Nature Rev. Neurosci.* **6**, 435–448 (2005).
37. Zorzi, M., Priftis, K. & Umilta, C. Brain damage: neglect disrupts the mental number line. *Nature* **417**, 138–139 (2002).
38. Szucs, D. & Csépe, V. The parietal distance effect appears in both the congenitally blind and matched sighted controls in an acoustic number comparison task. *Neurosci. Lett.* **384**, 11–16 (2005).
39. Temple, E. & Posner, M. I. Brain mechanisms of quantity are similar in 5-year-old children and adults. *Proc. Natl Acad. Sci. USA* **95**, 7836–7841 (1998).
40. Bramald, R. Introducing the empty number line: the Dutch approach to teaching number skills. *Education 3–13* **28**, 5–12 (2000).
41. Griffin, S. A., Case, R. & Siegler, R. S. in *Classroom Lessons: Integrating Cognitive Theory* (ed. McGilly, K.) 25–50 (MIT Press, Cambridge, Massachusetts, 1995).
42. Kosc, L. Developmental dyscalculia. *J. Learn. Disabil.* **7**, 46–59 (1974).
43. Molko, N. *et al.* Functional and structural alterations of the intraparietal sulcus in a developmental dyscalculia of genetic origin. *Neuron* **40**, 847–858 (2003).
44. Ross, J., Zinn, A. & McCauley, E. Neurodevelopmental and psychosocial aspects of Turner Syndrome. *Ment. Retard. Dev. Disabil. Res. Rev.* **6**, 135–141 (2000).
45. Riviere, D. *et al.* Automatic recognition of cortical sulci of the human brain using a congregation of neural networks. *Med. Image Anal.* **6**, 77–92 (2002).
46. Isaacs, E. B., Edmonds, C. J., Lucas, A. & Gadian, D. G. Calculation difficulties in children of very low birthweight: a neural correlate. *Brain* **124**, 1701–1707 (2001).
47. Posner, M. I. & Rothbart, M. K. Influencing brain networks: implications for education. *Trends Cogn. Sci.* **9**, 99–103 (2005).
48. Rueda, M. R., Rothbart, M. K., McCandliss, B. D., Saccomanno, L. & Posner, M. L. Training, maturation and genetic influences on the development of executive attention. *Proc. Natl Acad. Sci. USA* **102**, 14931–14936 (2005).
49. Morris, J. S. *et al.* A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature* **383**, 812–815 (1996).
50. Schumann, C. M. *et al.* The amygdala is enlarged in children but not adolescents with autism; the hippocampus is enlarged at all ages. *J. Neurosci.* **24**, 6392–6401 (2004).
51. Dawson, G., Webb, S. J., Carver, L., Panagiotides, H. & McPartland, J. Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion. *Dev. Sci.* **7**, 340–359 (2004).
52. Carr, L. *et al.* Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. *Proc. Natl Acad. Sci. USA* **100**, 5497–5502 (2003).
53. Dapretto, M. *et al.* Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neurosci.* **9**, 28–30 (2006).
54. Schultz, D., Izard, C. E. & Bear, G. Children's emotion processing: relations to emotionality and aggression. *Dev. Psychopathol.* **16**, 371–387 (2004).
55. Scott, S., Knapp, M., Henderson, J. & Maughan, B. Financial cost of social exclusion: follow up study of antisocial children into adulthood. *Brit. Med. J.* **323**, 1–5 (2001).
56. Golan, O. & Baren-Cohen, S. Systemizing empathy: teaching adults with Asperger syndrome and high functioning autism to recognise complex emotions using interactive media. *Dev. Psychopathol.* **18**, 589–615 (2006).
57. Adolphs, R. Neural systems for recognising emotion. *Curr. Opin. Neurobiol.* **12**, 169–177 (2002).
58. Vasa, R. A. *et al.* Neuroimaging correlates of anxiety after pediatric traumatic brain injury. *Biol. Psychiatry* **55**, 208–216 (2004).
59. Rauch, S. L., Shin, L. M. & Wright, C. I. Neuroimaging studies of amygdala function in anxiety disorders. *Ann. NY Acad. Sci.* **985**, 389–410 (2003).
60. Muris, P., Merckelbach, H. & Damsma, E. Threat perception bias in nonreferred, socially anxious children. *J. Clin. Child Psychol.* **29**, 348–359 (2000).
61. International Mind, Brain and Education Society [online], <www.imbes.org>
62. *Mind, Brain and Education Useable Knowledge Conference*, 7–8 October 2004, Graduate School of Education, University of Harvard [online], <www.gse.harvard.edu/usableknowledge/mbe/index .htm>
63. *International Mind, Brain and Education Summer School*, 16–20 July 2005, Erice, Sicily.

Competing interests statement
The author declares no competing financial interests.

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