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The Practical and Principled Problems with Educational Neuroscience

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Keywords: educational neuroscience; education; instruction; neuroscience; mind, brain, and education.

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### **Abstract**

The core claim of educational neuroscience is that neuroscience can improve teaching in the classroom. Many strong claims are made about the successes and the promise of this new discipline. By contrast, I show that there are no current examples of neuroscience motivating new and effective teaching methods, and argue that neuroscience is unlikely to improve teaching in the future. The reasons are two-fold. First, in practice, it is easier to characterize the cognitive capacities of children on the basis of behavioral measures than on the basis of brain measures. As a consequence, neuroscience rarely offers insights into instruction above and beyond psychology. Second, in principle, the theoretical motivations underpinning educational neuroscience are misguided, and this makes it difficult to design or assess new teaching methods on the basis of neuroscience. Regarding the design of instruction, it is widely assumed that remedial instruction should target the underlying deficits associated with learning disorders, and neuroscience is used to characterize the deficit. However, the most effective forms of instruction may often rely on developing compensatory (non-impaired) skills. Neuroscience cannot determine whether instruction should target impaired or non-impaired skills. More importantly, regarding the assessment of instruction, the only relevant issue is whether the child learns, as reflected in behavior. Evidence that the brain changed in response to instruction is irrelevant. At the same time, an important goal for neuroscience is to characterize how the brain changes in response to learning, and this includes learning in the classroom. Neuroscientists cannot help educators, but educators can help neuroscientists.

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There is growing interest in the claim that neuroscience can improve education. New journals (*Mind, Brain, and Education; Trends in Neuroscience and Education*), research centres (e.g., *Cambridge Centre for Neuroscience in Education*), as well as research degrees and conferences have been established. A number of recent high-profile papers in *Nature, Nature Neuroscience, Nature Reviews Neuroscience, Science, and Neuron* (Butterworth, Varma, & Laurillard, 2011; Carew & Magsamen, 2010; Eden & Moats, 2002; Gabrieli, 2009; Goswami, 2006; McCandliss, 2010; Meltzoff, Kuhl, Movellan, & Sejnowski, 2009; Sigman, Peña, Goldin, & Ribeiro, 2014) all attest to the promise of this approach. Although there are a growing number of sceptics (e.g., Alferink & Farmer-Dougan, 2010; Bishop, 2013; Coltheart & McArthur, 2012; Della Sala & Anderson, 2012; Schrag, 2011a), it is widely claimed that the future is bright for forging links between neuroscience and education, and indeed, this collaborative approach is often described as a new discipline and given the name *Educational Neuroscience*.

The most fundamental claim associated with educational neuroscience is that new insights about the brain can improve classroom teaching. Neuroscientists contribute because their findings are used to develop new teaching practices. Strong claims of this sort are common. For example:

We believe that understanding the brain mechanisms that underlie learning and teaching could transform educational strategies and enable us to design educational programmes that optimize learning for people of all ages and of all needs. (Blakemore & Frith, 2005, p. 459)

Teachers should also play a central role by adopting these new teaching methods in their classrooms. Furthermore, it is claimed that teaching teachers about neuroscience will inspire better instruction (Carew & Magsamen, 2010; Coch & Ansari, 2009;

Dehaene, 2009; Dubinsky, 2010; Eden & Moats, 2002; Gabrieli, 2009; Goswami, 2006; Katzir & Pare-Blagoev, 2006; McCandliss, 2010; Meltzoff et al., 2009; Pickering & Howard-Jones, 2007; Sigman et al., 2014). As Dehaene (2011) puts it, "...educators who can visualize how the child's brain works will, spontaneously, conceive better ways of teaching" (p. 26). Recommendation 2 (of 4) from a Royal Society (2011) report on neuroscience and education is to provide teacher training in neuroscience, and an increasing number of researchers are calling for reform of teacher training to include neuroscience (e.g., Ansari, 2005; Ansari, Coch, & De Smedt, 2011; Dubinsky, Roehrig, & Varma, 2013; Sigman et al., 2014).

In addition, there are claim that neuroscience can help to diagnose learning disorders early when instruction can be most effective, and claims that neuroscience can lead to medical treatments that facilitate learning in persons with learning disorders. I will briefly consider these later hypotheses, but the focus of this article concerns the core claim of educational neuroscience; namely, that neuroscience can improve teaching in the classroom. Here I side with the sceptics, and show that educational neuroscience has not yet contributed to any new and useful teaching practices, and detail why educational neuroscience is unlikely to improve classroom instruction in the future.

The paper is organized as follows. First I review the empirical success of educational neuroscience thus far. I conclude that there are no examples of neuroscience motivating new teaching methods that are effective. Indeed, few novel forms of instruction have even been proposed. Second, I argue that the theoretical motivations underpinning educational neuroscience are misguided. The core problem is that the goal of understanding the brain is irrelevant to designing and assessing teaching strategies. This raises serious questions of whether educational neuroscience

will prove useful for classroom instruction in the future. Third, I give a case study of educational neuroscience in the domain of literacy instruction that highlights both the practical and principled problems with this approach. Fourth, I briefly consider the claims that educational neuroscience can be used for diagnosis or to improve medical treatments for learning disorders, and finally, I conclude by noting that whereas neuroscience cannot help education in the classroom, the question of how education impacts the brain is fundamental to neuroscience.

### **The Empirical Failures of Educational Neuroscience**

As a first step in evaluating the promise of educational neuroscience it is worth considering the achievements thus far. Unfortunately, a careful reading of the literature reveals that the successes are either (1) trivial, in the sense that the recommendations are self-evident, (2) misleading, in the sense that the recommendations are already well established (based on behavioral studies), or (3) unwarranted, in the sense that the recommendations are based on misrepresentations of neuroscience or the conclusions do not follow from the neuroscience.

### **Trivial Justifications for Educational Neuroscience**

When highlighting the successes and promise of educational neuroscience authors often use neuroscience to motivate claims that are obvious and long understood. Perhaps the most common example is the claim that imaging studies reveal the potential of the brain to learn beyond early childhood years. According to many advocates of educational neuroscience, it is important to pass on this insight to teachers (e.g., Blakemore & Frith, 2005; Dubinsky, 2010; Goswami, 2006, 2008; Oliver, 2011; Tallel, 2004). For example:

Until relatively recently, it was widely believed that the adult brain was incapable of change....But research is beginning to show that this view of the

brain is too pessimistic: the adult brain is flexible, it can grow new cells and make new connections, at least in some regions, such as the hippocampus.

Although laying down new information becomes less efficient with age, we believe it is important to make people aware that there is no age limit for learning. (Blakemore & Frith, 2005, p. 460)

or:

Studies of the brain also suggest that it is never too late to learn. Some neural structures are still developing in the mid-twenties (e.g. the frontal cortex), and experience-dependent plasticity means that fibre connections continue to form to represent new learning throughout adulthood... This principle of learning provides empirical support for the efficacy of lifelong access to education ('lifelong learning'). (p 394, Goswami, 2008)

Two comments are worth making. First, the claim that neuroscience has only recently demonstrated that the brain is plastic is a misreading of the literature. Studies have provided evidence that new neurons in the dentate gyrus (part of the hippocampal formation) develop in adulthood (Eriksson et al., 1998), a phenomenon known as neurogenesis. This did challenge the long-standing assumption that new neurons do not form in adult humans. But the hypothesis that neurons change their connections in adulthood has long been established. Indeed, unless one is a dualist, the brain necessarily changes whenever learning takes place. Second, and more important for present purposes, teachers already know that learning continues throughout life. There would be no point in school, universities, and adult education otherwise. It is unclear what benefit or encouragement can be taken from the fact that the brain changes in response to learning throughout life. Phrases like "brain-based" learning (e.g., Jensen, 2008), "brain-compatible" learning (e.g., Tate, 2005),

“brain-friendly” learning (Perez, 2008), “brain-targeted” learning (e.g., Hardiman, 2003) should be seen for what they are -- tautologies. When learning occurs in the classroom, then it was mediated by neural changes.

Neuroscience is also used to motivate the conclusion that emotion is relevant to learning in schools. For instance:

It is increasingly recognized that efficient learning does not take place when the learner is experiencing fear or stress... The main emotional system within the brain is the limbic system, a set of structures incorporating the amygdala and hippocampus. The ‘emotional brain’ (LeDoux, 1996) has strong connections with frontal cortex (the major site for reasoning and problem solving). When a learner is stressed or fearful, connections with frontal cortex become impaired, with a negative impact on learning. (Goswami, 2004, p. 10)

Similar points are made repeatedly (e.g., Gruart, 2014; Hardiman, 2003; Hart, 1983; Zull, 2002). But again, this conclusion is trivial: Everyone knows that stressed or fearful students make poor learners. Many other examples of educational neuroscience supporting trivial conclusions have been made, including: adequate sleep and diet is important for learning (e.g., Sigman et al., 2014); that an enriched environment and exercise is good for learning whereas neglect, abuse, and malnutrition is bad (e.g., Carew & Magsamen, 2010), that learning is a social phenomenon, and as a consequence, learning with others is often more effective than learning alone (Goswami, 2008), motivation and study in quiet environments improves learning (e.g., Knowland, & Thomas, 2014), etc. Given that we already know all this, it is hard to argue that neuroscience has influenced teaching in any of these situations.

### **Misleading Justifications for Educational Neuroscience**

When neuroscience is used to make less obvious claims regarding instruction it is almost always the case that the instruction was first motivated by behavioral data. So again, the neuroscience is not contributing anything new to education.

For example, US children are often not introduced to a second language until secondary school given the widespread assumption that English might suffer from learning a second language too early. However, careful assessment of language skills shows that early exposure to a second language does not compromise the first language, and that mastery of two languages is best when the two languages are learnt early (Petitto, Katerelos, Levy, Gauna, Tetreaut, & Ferrao, 2001). Petitto and colleagues have gone on to show in fMRI studies that the blood-oxygen-level dependent (BOLD) signal in early bilinguals while they speak in either language looks more like the BOLD signal in monolingual speakers compared to bilinguals who learned one language early and one late. Based on the brain imaging, Petitto makes strong claims about educational neuroscience:

We have dedicated ourselves both to launching the discipline as well as to promoting *educational neuroscience* [italics in original], an exciting and timely new field that provides a most relevant level of analysis for addressing today's core problems in education... Here, we will show how language research, be it in monolinguals or bilinguals, in educational neuroscience has the fullest potential to fundamentally advance contemporary educational policy and practice—and soon. (Petitto, 2009, p. 185).

The problem with this is that the neuroscience is all but irrelevant to the argument that children should be taught a second language early – the critical behavioral data

preceded the neuroscience, and whatever the fMRI results showed the conclusion would be the same.

Another finding from psychology that is often cited in support of educational neuroscience is the so-called “testing effect”. Typically teachers give tests in order to assess students’ learning, and the educational value of testing is largely ignored. However, the testing of information often improves learning more than additional study. For instance, Roediger and Kaprike (2006) asked students to read fact-filled passages about various subjects, and then students were asked to take a seven-minute test on the passage, or re-read the passage for another seven minutes. After a short retention interval students remembered most in the extra study condition, but after a two-day delay, memory was substantially better in the test condition. Indeed, Roediger et al. (2011) showed that the testing effect works in the classroom, with robust advantage in a classroom of 11-12 year olds who learned in a quiz compared to non-quiz condition. What is striking about the testing effect is that students (and teachers) do not appreciate the power of the effect. When students were asked to predict how well they will remember something following testing vs. study conditions, they did not appreciate that their memory will be better in the test condition (also see Rohrer & Pashler, 2010).

Although these findings are purely behavioral, the results are often presented in support of educational neuroscience. For example, Carew and Magsamen (2010) summarized this work in a review article published in *Neuron* entitled “Neuroscience and Education: An Ideal Partnership for Producing Evidence-Based Solutions to Guide 21<sup>st</sup> Century Learning” and write:

Moreover, since memory retrieval and the consolidation of learning are basic components of education *and* are deeply explored topics in neuroscience, what a great place to start building bridges to span the two disciplines. (p. 686-687 )

But again, neuroscience has had nothing to contribute to the findings, and Carew and Magsamen make no proposal about how neuroscience could contribute to education above and beyond what psychology has already established. Roediger, Finn, & Weinstein (2012) also nicely highlight the relation between their work and that of educational neuroscience:

Of course, neural processes underlie any cognitive processes, so in a broad (and vacuous) sense our chapter can be conceived as being about neuroscience and education. However, we certainly do not make this claim. We seek to generalize findings from behavioral studies to possible educational practice (p. 128).

Or consider suggestions regarding mathematics. A number of imaging studies suggest that regions of the parietal cortex are specialized for numerical quantity (e.g., Cantlon, Brannon, Carter, Pelphrey, 2006; Dehaene, Piazza, Pinel, & Cohen, 2003) and that these regions are functionally impaired in dyscalculic subjects (Kucian et al., 2006; Price, Holloway, Räsänen, Vesterinen, & Ansari, 2007). Although imaging results are somewhat mixed (e.g., Rotzer et al., 2008), the findings are generally taken to support the long-standing hypothesis that a “number sense” plays a foundational role in more complex mathematics (e.g., Gallistel & Gelman, 2000; Gelman & Gallistel, 1978). In addition, the neuroscience is often taken to support the claim that remediation programs in mathematics should target the number sense. For instance, Butterworth et al. (2011) argue that instruction in set enumeration (naming the number of items in a set) and comparison (comparing the number of items in different

sets to decide which set has more) should be emphasized. This is in line with previous suggestions by Gelman and Gallistel (1978) based on behavioral data, and there is no indication how the neuroscience provides any additional insight into how instruction should be designed. Similarly, Dehaene (2007) uses neuroscience to argue that a number sense can be improved in children by encouraging finger counting, using concrete analogies to make fractions clearer (e.g., considering proportions of a pie when adding the fractions  $1/2$  and  $1/3$ ), using number lines to understand the concepts of zero and negative numbers, etc. Indeed, Dehaene takes the neuroscience to support earlier educational practices that emphasized the importance of making the concept of numbers concrete, writing: “Number sense –indeed, common sense—is making a comeback” (p. 297). So once again, it seems that neuroscience is not providing any novel ideas about how to best carry out instruction (see Schrag, 2011a for similar critique of the value of educational neuroscience to math).

A somewhat different result was reached by Supekar et al. (2013) who carried out functional and structural MRI analyses on grade 3 children (aged 8-9) prior to an intensive eight-week one-on-one math tutoring programme that focused on conceptual aspects of number knowledge and speeded practice on counting strategies (conceptual and procedural skills, respectively). The critical finding was that hippocampal volume and intrinsic connectivity of the hippocampus with dorsolateral and ventrolateral prefrontal cortices and the basal ganglia predicted training gains, whereas various behavioral measures did not. That is, brain regions associated with learning and memory, rather than arithmetic processing, predicted learning outcomes best. Based on these findings the authors conclude: “Characterization of predictive biomarkers in each child may facilitate the development of targeted training and intervention programs” (p. 8234). But again this conclusion is unjustified. The

behavioral findings suggest that the intervention was successful (children improved overall), but the authors do not provide any suggestions about how the biomarkers in each child might be used. Indeed, it is not clear that studies that highlight the importance of the parietal cortex (Dehaene et al., 2003) vs. the hippocampus and related structures (Supekar et al., 2013) have different implications for education.

Finally, consider the case of literacy instruction. A great deal of research in psychology suggests that phonics instruction is better than many alternative approaches to teaching literacy (e.g., National Reading Panel, 2000). Indeed, based on behavioral research, phonics is currently legally required instruction in all UK state schools and is a standard teaching method in the US and Canada. More recently, neuroscience has been used to support phonics instruction based on the finding that dyslexic children often show abnormal brain activation or structure in regions associated with phonological processing (e.g., Heim & Grande, 2012; Meyers et al., 2015; Peterson & Pennington, 2012; Saygin et al., 2013). Below I consider in more detail whether the neuroscience licences any strong conclusions regarding phonics instruction, but for present purposes, the main point is that neuroimaging data is often used to support a form of instruction that already has widespread support based on behavioral data.

I do not think I am being unfairly selective in my review of the evidence. To illustrate the lack of any novel or useful contribution to classroom teaching it is worth considering a recent article published in *Nature Neuroscience* that summarizes the best achievements of educational neuroscience thus far (Sigman, et al., 2014). The authors organized their review in four sections. First they note that a good diet, exercise, and sleep are important for learning, and cite evidence that all three factors impact the brain. But you did not need neuroscience to tell you that these factors have

an impact on learning, and the neuroscience is irrelevant to the proposed solutions (e.g., starting school later in the day during the teenage years). Second, they note that medical interventions can facilitate language learning outside the classroom. For instance, providing hearing disabled children with cochlear implants before 3 years of age has a positive impact on language learning. This is indeed an important outcome, but is not an example of neuroscience improving teaching in the classroom. I consider medical treatments briefly below. Third, they argue that neuroscience is relevant to developing policies regarding bilingual education. But as noted above, the relevant findings were first advanced based on behavioural work. Finally, the authors highlight imaging data suggesting that dyslexia is associated with phonological impairments, and claim that EEG data that may be useful for early diagnosis of dyslexia. But again, the psychological evidence for phonological deficits in dyslexia long predated the neuroscience, and there are strong reasons to question the value of neuroscience in the early diagnosis of dyslexia (see below).

### **Unwarranted Uses of Neuroscience to Motivate Instruction**

In addition, neuroscience is often mischaracterized in order to support conclusions regarding education. For example, Tommerdahl (2010) argues that Parallel Distributed Processing (PDP) models of word naming provide one of the best examples of teaching being informed by neuroscience. Tommerdahl highlights the biological constraints on PDP models and notes that PDP models of word naming learn to map letters to sounds. She uses this to support phonics instruction.

However, PDP models of reading are very far from biologically constrained. Indeed, advocates of PDP models often distance their models from neurobiology:

...there are clearly many aspects of the standard PDP framework that do not emulate known aspects of neurophysiology: the lack of separate excitatory and

inhibitory cell populations, the purely linear integration of inputs with no consideration of dendritic geometry, the use of a real-valued symmetric activation function, no consideration of metabolic constraints, and the propagation of error signals back through forward-going connections, to mention only a few. However, as has repeatedly been emphasized, PDP models are generally not intended to emulate all aspects of the underlying neural substrate: The models are intended to abstract away from many details (Plaut & McClelland, 2010, p. 287).

Furthermore, the way PDP models of word naming learn is unrealistic in a number of ways. First, the models are presented the correct pronunciation of each word every time. This is very unlike the conditions in which children learn to read (e.g., Share, 1995). Second, the models tend to be trained in an “interleaved” manner so that they learn all words slowly in parallel. Again this is very unlike learning in children. Third, the PDP model Tommerdahl appears to be referring to in order to support phonics (Seidenberg & McClelland, 1989) only learned to translate letters to sounds for monosyllable words. Accordingly, it is unjustified to use this model as evidence for phonics as opposed to alternative approaches that emphasize the role of semantic or morphological processes in instruction. Whatever the merits of PDP models and their relevance to literacy instruction, the biological plausibility of these models should not be used to support phonics instruction.

Similarly, Fisher, Goswami, and Geake (2010) cite recent Bayesian models of neural processing as providing evidence relevant to educational neuroscience. They claim that Bayesian models learn better under noisy conditions, citing a paper by Ma, Beck, Lathan, and Pouget (2006). According to Fisher et al.: “Insights like these may be critical to advancing a new field like educational neuroscience, where educators

are continually faced with “noisy systems” (vis. Children!).” (p. 73). Apart from the fact that the authors do not specify how this insight might help, it is not true that Bayesian models perform better under noisy conditions (they deal optimally with noise, but the more noise, the worse they perform). Instead, Ma et al. showed that it is easier to implement certain Bayesian computations in neurons that have a certain form of noise (Poisson noise). The link between Bayesian models of cortical computation and education is hard to see, and indeed, there are reasons to question the claim that brains compute in a Bayesian-like way (e.g., Bowers & Davis, 2012; Jones & Love, 2011)

More often neuroscience is mischaracterized in more extreme ways to make unwarranted conclusions. For example, claims regarding left and right brain functioning, brain learning styles, that we only use 10% of our brain, etc. are used to justify a wide variety of instructional programs (typically very expensive) that have little or no empirical support. A number of authors (e.g., Goswami, 2004; Howard-Jones, 2014a) argue that educational neuroscience is useful in this context in that teacher training in neuroscience puts teachers in a stronger position to reject bogus methods justified with neuroscience. That is, educational neuroscience can be a good antidote to so-called “neuromyths”. But given the common misuse of neuroscience by advocates of educational neuroscience, this is not a promising solution. Rather than introducing neuroscience to teacher training it would be better to just avoid all forms of instruction that are motivated by neuroscience.

Even when the neuroscience is well characterized there are logical problems relating the results to recommendation for classroom instruction. So even here the conclusions regarding education are unwarranted. I consider these problems next.

### **Logical Problems with Educational Neuroscience**

When educational neuroscience is challenged, the most common criticism is that the gap between neuroscience and education is too wide to be of any use; Bruer (1997) introduced the phrase “a bridge too far”. This analogy might be taken to suggest that we just don’t know enough in order to apply neuroscience to education, and that future advances might bridge this gap (although Bruer himself raises doubts about whether neuroscience will ever be relevant to classroom teaching). I disagree that future advances will help. The fundamental problem is not with the quality of the neuroscience but in the logical flaws motivating educational neuroscience. As a consequence, neuroscience is poorly suited for designing new forms of instruction as well as assessing the efficacy of instruction, as detailed below.

### **Educational Neuroscience Provides a Misguided Approach to Designing**

#### **Instruction**

One of the primary reasons why educational neuroscience is thought to be so promising is that it can provide unique insights into the nature of learning disorders. This is considered to be critical because instruction can then target these deficits. A colourful example of this reasoning can be found in the following analogy:

My firm conviction is that every teacher should have some notion of how reading operates in the child’s brain. Those of us who have spent many hours debugging computer programs or repairing broken washing machines (as I have done) know that the main difficulty in accomplishing these tasks consists in figuring out what the machine actually does to accomplish a task. To have any hope of success, one must try to picture the state in which it is stuck, in order to understand how it interprets the incoming signals and to identify which interventions will bring it back to the desired states.

Children' brains can also be considered formidable machines whose function is to learn. Each day spent at school modifies a mind-boggling number of synapses. Neuronal preferences switch, strategies emerge, novel routines are laid down, and new networks begin to communicate with each other. If teachers, like the repairman, can gain an understanding of all these internal transformations, I am convinced that they will be better equipped to discover new and more efficient education strategies.... (Dehaene, 2009, p. 232-233)

As detailed below psychology is generally in a better position to identify and characterize the learning difficulties of a child. But for the sake of argument, consider the case in which neuroscience does identify a cognitive deficit that was missed in behavioral studies. Does this constitute an example where neuroscience is poised to improve instruction? No it does not.

The problem is that is not clear that interventions should target an underlying disorder. Instead, interventions might be best when they are designed to enhance alternative skills (compensatory approach) as opposed to ameliorating the deficits themselves (restitutive approach). Indeed, in the context of acquired brain disorders (rather than developmental disorders), there is strong evidence that a compensatory (or sometimes called "strategy training") approach is more effective than the restitutive approach (Cicerone et al., 2005). So when neuroscience identifies a novel explanation for some cognitive deficit it is not clear what one should do with this new insight. One conclusion would be to develop an intervention that targets this disorder; the other would be to focus on other skills or some combination of skills. This is not very helpful.

### **Educational Neuroscience Provides a Misguided Approach to Assessing the Efficacy of Instruction**

But the most serious difficulty is that educational neuroscientists often lose sight of the only thing that matters from the point of view of the teacher and the child, namely, whether the child learns. Many authors highlight the fact that the brain changes in response to teaching, and take this as relevant to the assessment of teaching instruction. For example, Oliver (2011) writes: “Imaging studies can provide confirmatory evidence of behavioral changes” (p 216). But this is misguided: Behavioral change cannot be confirmed (or disconfirmed) by neuroscience. Behavior changes or not, and if it does, it is a given that the brain has changed in some way. Similarly, according to a recent government report in the UK, neuroscience can be a “tool for science-based education policy, which can help assess the performance and impact of different educational approaches” (Royal Society, 2011, p.9). Again this is confused thinking. The only relevant measure of performance is behavioral, and the only way to assess change in performance is to measure behavior. Any observed brain changes may or may not have an impact on the relevant behavior.

Indeed, sometimes it is claimed that neuroscience provides a more “direct” way of measuring the impact of learning than behavior itself. For example, Goswami (2004) writes:

Although it is frequently assumed that specific experiences have an effect on children, neuroimaging offers ways of investigating this assumption directly... For example, on the basis of the cerebellar theory of dyslexia, remedial programmes are available that are designed to improve motor function. It is claimed that these programmes will also improve reading. Whether this is in fact the case can be measured directly via neuroimaging (p. 9).

But this is getting the things exactly backwards. The direct test to see whether these teaching instructions improve reading is to measure reading. All that matters is whether the child reads better. Coltheart and McArthur (2012) make the same point regarding the faulty logic of looking at brain as opposed to behavior when assessing the efficacy of an educational intervention.

A possible defence of educational neuroscience is that imaging can provide a more sensitive measure of learning, such that it can pick up changes before they are manifest in performance. If so, it might be argued that imaging results could provide some guide as to what teaching practices should be pursued further, with the assumption that more training will manifest itself in behavior as well (e.g., James, 2010). There are two problems with this. The first (practical) difficulty is that claims regarding the greater sensitivity of fMRI compared to behavioral measures may reflect false-positive results. Indeed, many recent papers are highlighting how unreliable many imaging results can be (e.g., Button et al., 2013; David et al., 2013; Vul & Kanwisher, 2010). Accordingly, reports of significant BOLD changes in the absence of behavioral change should be treated with caution. The second (in principle) difficulty is that if performance does not improve in a reasonable amount of training, the instruction does not work. Rather than using (expensive) fMRI to look for a weak training effect that may or may not manifest itself in behavior following further training, researchers should explore whether more extensive (cheaper) behavioral training does indeed result in useful changes in behavior in a practical amount of time.

Another possible defence of educational neuroscience is that it can help explain why a given behavioral intervention worked. For example, imagine that a specific method of instruction was effective in teaching children algebra, and further,

this instruction led to changes in the BOLD signal in language centres of the brain. This would provide some evidence that language was somehow involved in the improved performance (ignoring all the difficulties in linking changes in BOLD signals to the underlying skills that support performance; see below). However, the results do not provide any insight into whether it was the best form of instruction. In the current example, perhaps there is a much better way to teach algebra that would engage another (e.g., spatial) part of the brain.

In sum, neuroscience is not well suited for proposing new forms of instruction, nor for assessing the efficacy of instruction. This may help explain why there are no examples of neuroscience improving instruction in the classroom.

At the same time, it is important to emphasize that psychology does have an important role to play in improving teaching in the classroom, and indeed, as noted above, educational neuroscience often takes credit for findings reported in psychology. More generally, a wide variety of findings from psychology provide specific and well motivated suggestions about how to improve educations across a variety of domains (e.g., Dunlosky, Rawson, Marsh, Nathan, & Willingham, 2013; Roediger, 2013; Roediger & Pyc, 2012). The critical difference between psychology and neuroscience that explains this contrasting success is that psychology is concerned with behavior, and behavior is the only relevant metric when assessing the value of an instructional intervention. Even areas of neuroscience that focus on both brain and behavior, such as cognitive neuroscience, are only relevant to education to the extent that they provide new insights into behavior. Of course, theories regarding how the brain supports behavior are relevant to psychologists and neuroscientists, but for the teacher, all that matters are hypotheses regarding behavior that can be used to design better forms of instruction.

This is not to dismiss the relevance of all forms of theory, only theories that do not have implications for behavior. Indeed, theory from a variety of domains has already led to teaching innovations. For example, Gigerenzer and colleagues have developed a theory of “ecological rationality” according to which human reasoning is based on an adaptive toolbox of simple heuristics that work well in specific contexts. Based on this theory a range of heuristics have been hypothesized, and this in turn has led to proposals regarding how to improve reasoning across a range of tasks and conditions (e.g., Gigerenzer & Todd, 1999). Outside psychology altogether, linguistics has provided a detailed theory of how the English orthographic system is designed: rather than the common view that letters are primarily designed to represent sounds, English is a morphophonemic system in which spellings have evolved to represent an interrelation of morphology, etymology and phonology. As Venezky (1999, p 4) wrote:

“English orthography is not a failed phonetic transcription system, invented out of madness or perversity. Instead, it is a more complex system that preserves bits of history (i.e., etymology), facilitates understanding, and also translates into sound”.

This has obvious implications for literacy instruction given that learning and memory is better when information is encoded in a meaningful and well-organized manner (e.g., Bower, Clark, Lesgold, & Winzenz, 1969). Indeed, based on insights from linguistics and psychology, P. Bowers & Kirby (2010) have developed new methods and tools to teach literacy to young children. It is hard to see how theories of brain function could lead to these sorts of teaching innovations, and again, behavioral testing is the only method to assess the efficacy of these proposals.

### **Case study of Educational Neuroscience When Applied to Literacy Instruction**

Some of the strongest claims regarding the success and promise of educational neuroscience have been made with regards to literacy instruction (e.g., Gabrieli, 2009; Katzir & Pare-Blagov, 2006; Howard-Jones, 2014b). For this reason it is worth going into more detail regarding the practical and principled problems in relating neuroscience to education.

### **Practical Problems**

Most of neuroscience research taken to be relevant to literacy instruction was designed to characterize the deficits associated with dyslexia. The hope is that a better understanding of the deficit will determine what skills need to be targeted in remedial instruction. The practical problem with this agenda is that psychology does a better job in identifying the cognitive deficits of dyslexia. Not only is it difficult to relate brain states to cognitive disorders, the results from neuroscience are often highly mixed.

For example, many fMRI studies have reported abnormal BOLD signals in the dyslexic brain; most often the temporoparietal cortex involved in phonological processes, and the ventral occipito-temporal cortex (often referred to as the visual word form area, or VWFA) involved in representing the visual forms of words (e.g., Brunswick, McCrory, Price, Frith, & Frith, 1999; Kronbichler et al., 2006; Paulesu et al., 2001; Rumsey, Nace, & Donohue, 1997; Shaywitz et al., 2007). Abnormal activation in either of these areas has been used to support the claim that phonological deficits are responsible for the dyslexia. The most common conclusion is that instruction should target phonological processes, typically phonics (e.g., Dehaene, 2009), but also in other ways (e.g., Temple et al., 2003).

A first point to note is that inferring a phonological deficit in dyslexia on the basis of an abnormal BOLD signal in the temporoparietal cortex might seem

reasonable given its involvement in phonological processing. But why make the same conclusion on the basis of an abnormal BOLD signal in the VWFA? The standard argument is that the abnormal BOLD signal in the VWFA is secondary to the primary dysfunction of the temporoparietal cortex (McCandliss & Noble, 2003; Pugh et al., 2000). This is plausible, but it also highlights a difficulty in relating the locus of an abnormal BOLD signal to claims regarding the locus of the deficit: Just as an abnormal BOLD signal in VWFA is attributed to a core deficit outside the VWFA, an abnormal BOLD signal in the temporoparietal cortex may be attributed to a core deficit outside the temporoparietal cortex. In which case, this abnormal BOLD signal does not provide unequivocal evidence for a phonological basis of dyslexia.

Furthermore, a meta-analysis of neuroimaging studies of the dyslexic brain have found many areas of abnormal BOLD activation, including the under activation of inferior parietal, superior temporal, middle and inferior temporal, fusiform, and inferior frontal gyrus of the left hemisphere. There was also over activation in the primary motor cortex and anterior insula in the left hemisphere (Richlan, Kronbichler, & Wimmer, 2009). The wide range of abnormal BOLD signals across studies *may* all reflect a disordered phonological processing system within the temporoparietal cortex, but it is difficult to make a strong case that phonology is the root cause of dyslexia based on these findings.

Another set of findings taken to support a phonological basis of dyslexia is that the abnormal BOLD signals in phonological areas often become more normal following phonics and related phonological interventions (Barquero, Davis, & Cutting, 2014; Eden et al., 2004; Heim & Grande, 2012 for review see; Shaywitz et al., 2004; Simos et al., 2002). But again, BOLD signal changes are not restricted to reading areas. For example, Shaywitz et al., (2004) reported that a dyslexic group

who received phonics instruction showed increased BOLD activation in the VWFA compared to a dyslexic group who did not receive special instruction (a finding taken to be consistent with a phonological account of dyslexia). However, the dyslexic group who received phonics instruction also showed an increased BOLD signal in the inferior frontal gyrus and a decrease in the BOLD signal in the caudate (a brain region that is not known to be involved in reading). Furthermore, the dyslexic group who received phonics instruction only showed an increased BOLD activation in the caudate compared to a control (non dyslexic) group. If anything, the results should be taken as a puzzle for a phonological theory of dyslexia.

But even if future studies show that successful phonics instruction leads to a selective normalization in the BOLD signal in temporoparietal cortex, it is difficult to conclude that changes in temporoparietal cortex mediated the behavioral change. Consider a recent paper by Olulade, Napoliello and Eden (2013) that showed that developmental dyslexia was associated with a reduced BOLD signal in V5/MT (visual cortex) in response to a visual motion processing task. Furthermore, the authors found that a phonologically based reading intervention lead to increased activation in V5/MT in response to a visual processing task, such that brain activation in this visual region became more normal. Although this imaging result might appear to support the claim that visual deficits are associated with dyslexia, Oludade et al. argued that the return to normal in the V5/MT BOLD signal was an incidental by-product of learning elsewhere (the phonological system). This is plausible, but it again highlights the difficulty in associating BOLD signal changes with the locus of a cognitive disorder. Making causal claims regarding the locus of learning (as opposed to processing) is particularly difficult using fMRI because the BOLD signal is not linked to the biological processes that support learning (Dorjee & Bowers, 2012).

Somewhat more consistent findings have been reported with structural fMRI studies that provide evidence that various measures of reading related skills are correlated with increase white matter associated with the left arcuate fasciculus. For instance, Saygin et al. (2013) found increased white matter was associated with increased phonological awareness skills of pre-reading and early-reading kindergarten children. Similarly, Meyers et al. (2015) found evidence that increased white matter in kindergarten children predicted reading performance in Grade 3. The arcuate fasciculus is thought to link the visual word form area with inferior parietal regions associated with phonological knowledge and therefore may support grapheme-to-phoneme-conversion. Given that increased myelination provides a measure of stronger connectivity between regions, these findings suggest that skilled reading is linked with the ability to convert graphemes to phonemes. This in turn is often taken as evidence in support of phonics.

Although the structural fMRI findings may provide stronger evidence in support of the claim that phonological processing deficits are related in dyslexia it should be noted that the arcuate fasciculus also projects to the angular gyrus that may play a role in the translation of orthography to semantics (Vigneau et al., 2006). So in principle these results may also relate to the skill of linking visual word forms to meaning (which may suggest different conclusions regarding instruction). It is also worth noting that these studies were carried out on children who are very likely being taught to read with phonics. Whether the same association between reading and the arcuate fasciculus would be found following a different type of instruction that does not emphasize grapheme-phoneme correspondences to the same extent (e.g., reading instruction that also highlights the morphological structure of words) is not yet clear.

But in any case, the most important point is that we already know that phonological processes are implicated in dyslexia in alphabetic languages. Psychological tests show that most children with reading difficulties have difficulties with a range of phonological skills. For example, a recent meta-analysis of 235 behavioral studies (Melby-Lervåg et al., 2012) showed that dyslexics have the greatest difficulties in phonemic awareness in relation to typically developing children of the same age (pooled effect size estimate: -1.37) as well as deficits in rime awareness and verbal short-term memory (pooled effect size estimates: -0.93, and verbal short-term memory, -0.71). The neuroscience described above, at best, is providing confirmatory (and less direct) evidence in support of what we already know. According, statements like this are unjustified:

Whether neuroscience can be informative to educational theory and practice is not debatable - it has been. For example, behavioral data were not decisive in determining whether dyslexia was primarily a visual perceptual disorder, or whether phonology was the more fundamental problem (for a review, see (McCardle, Scarborough, & Catts, 2001). Brain imaging data (e.g., Rumsey et al., 1992) showed reduced activation in left temporo-parietal cortex, a region known from other studies to support phonology, thus strongly supporting the phonological theory. (Willingham, 2009, p. 544)

If educational neuroscience was only providing converging theoretical motivation for phonics instruction, then at least it would be supporting an instructional method that has been shown to improve reading. However, neuroscience is often used to support interventions that have little or no empirical support. For example, Bishop (2013) reviewed all studies between 2003-2011 that reported measures of brain function in children before and after an intervention for language

learning difficulties and found serious methodological problems in them all. Three of these studies assessed an intervention called Fast ForWord that is based on the hypothesis that a rapid auditory temporal processing deficit underlies dyslexia. In order to treat dyslexia, Fast ForWord uses a set of computer programmes that trains children on processing sounds in an attempt to ameliorate this deficit. Although all three imaging studies were taken to support this approach, a detailed meta-analysis of behavioral outcomes showed no benefit to this form of instruction (Strong, Torgerson, Torgerson, & Hulme, 2011). Another of the neuroimaging studies claimed that a programme called “Earobics” designed to improve reading improves the cortical representation of speech. But again, not only did Bishop find the study flawed, behavioral assessments have failed to support the conclusion that it improves literacy (Pokorni, Worthington, & Jamison, 2004). Similarly, an intervention designed to improve phonological and motor skills in children with developmental disorders of speech and language was flawed (although this was not specially about literacy). Nevertheless, the results from neuroscience are often taken as more informative than psychological studies, consistent with research that shows that people are often worse at distinguishing good from poor explanation of psychological phenomena when irrelevant neuroscience is added to the mix (Weisberg, Keil, et al., 2008).

### **Principled Problems Applying Neuroscience to Literacy Instruction**

The first principled problem with making any conclusions regarding literacy instruction on the basis of these neuroimaging results is that it is unclear whether instruction should target processes that are inferred to be impaired. Instead, as noted above, it is equally plausible that instruction should target other skills. Indeed, some of the neuroimaging results could be taken as evidence that compensatory (non-phonological) processes play a key role in improvement. For instance, Hoefft et al.

(2011) carried out a longitudinal study with children with and without dyslexia and found that greater right prefrontal BOLD activation at the beginning of the experiment predicted later reading gains in dyslexics. The authors concluded that this frontal activation may reflect compensatory processes engaged by individuals with phonological deficits (also see Shaywitz & Shaywitz, 2005).

Does this evidence for compensatory processes in dyslexia constitute a potential contribution to instruction? At best, it suggests that something above and beyond phonics is appropriate, but nothing more than that. Based on the evidence that there are abnormal activations in phonological and non-phonological regions of the brain it is hard to make any suggestions regarding how to better design literacy instruction.

The second principled problem of applying neuroscience to literacy instruction is that neuroscience is irrelevant for assessing the success of any intervention. For example, consider again the hypothesis literacy instruction should be designed to teach children the logic of their writing system, with English speakers being taught of the joint constraints of phonology, morphology, and etymology on word spellings (Bowers & Kirby, 2010). This is an alternative to phonics instruction that selectively stresses letter-sound correspondences. In any future study that compares the relative efficacy of these two approaches, it is the behavior of the children not the normalization of their brain that is relevant to consider. Furthermore, any subsequent neuroscience data that provides some insight into how a new form of instruction works is irrelevant to assessing the efficacy of the instruction. For example, if it turns out that teaching children the logic of the writing system is the more effective approach to literacy instruction, the finding that some brain area associated with morphological processing is also normalized does not constitute an example of a

success of educational neuroscience: This finding will not have motivated the instruction in the first place, nor constitute the relevant measure of the success of the method.

I have gone into great detail into the practical and principled problems of educational neuroscience in the domain of literacy instruction, but equivalent problems apply to all domains of instruction. It is hard to see how neuroscience will ever improve teaching in the classroom.

### **Other Forms of Educational Neuroscience**

#### **Diagnosis**

Another common claim is that educational neuroscience can be used as a tool to predict developmental learning disorders before they manifest in behavior. This will allow teachers to provide specialized instructions to children early when they have the greatest effect. Note, this form of educational neuroscience is not subject to the criticisms above because the design and assessment of instruction could be based entirely on behavioral evidence.

Perhaps the strongest claims regarding educational neuroscience and diagnosis has been made in the domain of reading disorders. For instance, Gabrieli (2009) writes:

Perhaps the most practical, near-term synergy between education and cognitive neuroscience arises from an integration of behavioral and brain measures in the service of predicting reading difficulty and then offering intervention to avoid reading failure

But again, there are reasons to be cautious about making any strong conclusions.

First consider an fMRI by Hoefft et al. (2007). The authors tested children between 8 and 12 years of age identified by teachers at the start of a school year as

being at risk for reading difficulty. Children received a standardized test of decoding, 12 additional behavioral measures of language and reading, and underwent brain imaging. The behavioral and brain measures taken at the beginning of the school year were then related to the children's decoding ability at the end of the same school year. The critical finding was that behavioral and imaging results accounted for 65% and 57% of the variance in end-of-year decoding performance, respectively, but the combination of behavioral and brain measures accounted for significantly more of the variance (81%). That is, the imaging added unique variance that could be used to predict performance.

Is this a success? The first point to note is that a significant increase in prediction is often of little practical utility when deciding whether a given child should be given remedial instruction (e.g., Bishop, 2000). The issue is whether the neuroscience improves the sensitivity of prediction (the proportion of children with a learning disorder that are identified as having a disorder) and the specificity of prediction (the proportion children without a learning disorder that are identified as not having a disorder) in the relevant population to such an extent as to make the test clinically useful. The authors have not demonstrated this.

Second, the unique contribution of the imaging data in the Hoeft et al. (2007) study almost certainly rests on leaving out other relevant behavioral predictors. For instance, the study did not consider low-level visual and attentional factors that also explain unique variation in reading performance after controlling for various reading related tasks (Franceschini et al., 2012). Indeed, Hoeft et al. (2007) did not include one of the strongest predictors of future dyslexia into their regression studies, namely, whether one (or both) of the parents are dyslexic. If all these factors were entered in a

regression then the unique contribution of imaging data would almost certainly be reduced.

Similar claims have been made on the basis of structural MRI scans (Hoeft et al., 2007; Meyers et al., 2015; Saygin et al., 2013) where abnormal brain structures have been reported in children prior to the diagnosis of dyslexia. But again, the value of the neuroscience as a diagnostic tool depends on whether the increase in sensitivity and specificity in the relevant population is clinically relevant. This has not been shown. And again in all cases, all the relevant behavioral measures have not been included in the regressions, so there may be simpler methods to improve prediction.

Even more dramatic claims have been made on the basis of event-related potentials (ERPs). Here a number of authors that have suggested the ERPs collected soon after birth (Guttorm, Leppänen, Hämäläinen, Eklund, & Lyytinen, 2010; Guttorm, Leppanen, Poikkeus, Eklund, Lyytinen, & Lyytinen, 2005; Molfese, 2000), or prior to reading instruction (Maurer, et al, 2009), are anomalous in children who will go on to develop dyslexia, and accordingly, it is claimed that ERPs can be used to diagnosis very early.

However, there are reasons to be cautious regarding these studies. For example, Bishop (2007) reviewed a series of ERP studies that claimed to provide evidence that poor auditory temporal processing deficits play a role in dyslexia. She noted a range of problems across the studies, including that they tended to be underpowered, and that the statistical analyses of the studies were unsystematic, with the possibility of false positives given the number of possible differences in a waveform that can be used to predict later reading performance. These problems make it difficult to make any strong conclusions in these studies despite the fact that the results were based on averaging ERPs across groups of individuals. It is much

more challenging to make predictions on the basis of ERPs at the individual level, and there is no indication that EEG studies designed to make early predictions of dyslexia are immune to these methodological criticisms (e.g., Maurer, et al., 2009, noted that the limited sample size of their study limited their ability to make some key conclusions).

The task of making diagnosis very early (predicting dyslexia soon after birth) is especially difficult given the lack of stability in language difficulties during early childhood (e.g., Nation, Plunkett, and Bishop, 2015). But even if we assume that neurobiological markers will in the future become clinically accurate for diagnosis, another question that is rarely considered is whether this can inform instruction? It is widely claimed that it is important to make diagnosis early, but at least in the case of literacy, the most effective forms of instruction require children to work with letters in order to convert them to sounds (Hatcher et al., 2006) or appreciate both the phonological and morphological structure of words (e.g., P. Bowers et al., 2010). These forms of intervention cannot be started before the child is starting to read. Furthermore, the forms of instruction that can be carried out earlier (training on sounds; e.g., Temple et al., 2003) have been found to be ineffective (Strong et al., 2011). So at least in the domain of literacy, it is unclear if there is any benefit to identifying the disorder very early.

In sum, the problems with using educational neuroscience as a diagnostic tool are more practical than principled, but it is far from clear that this is a promising approach. Indeed, if early diagnosis is going to be one of the main contributions of educational neuroscience it will be important to demonstrate that early instruction is important (or even possible) in ameliorating the learning difficulty.

### **Medical Treatments**

Briefly, advocates of educational neuroscience sometimes note how neuroscience has introduced medical breakthroughs that have educational implications. For instance, Goswami (2009) highlights that neuroscience contributed to the development of cochlear implants that are effective in improving language learning in hearing disabled children. And in contrast with her criticism of educational neuroscience more generally, Bishop (2013) notes how so called “smart drugs” may be helpful to treat attention deficit disorders and notes that in the future a range of medications may be useful for range of developmental disorders.

I agree. This is a domain in which neuroscience can improve learning, but this is quite a different version of educational neuroscience. Indeed, almost all of the work involves neuroscientists and medics. The only role for educators here is to teach these children who are now better prepared to learn. Schrag (2011b) provides an excellent summary of the promise of neuroscience to improve education through medical interventions. He calls this link between neuroscience and education an “...alternative kind of marriage, one that needn’t be consummated in school classrooms and might not involve teachers”.

### **Summary**

In sum, it is hard to see how neuroscience is relevant to teaching in the classroom. At present the strong claims regarding the successes of educational neuroscience are either (1) trivial, in the sense that the recommendations are self-evident, (2) misleading, in the sense that the recommendations are already well established (based on behavioral studies), or (3) unwarranted, in the sense that the recommendations are based on misrepresentations of neuroscience or the conclusions do not follow from neuroscience. From my reading of the literature there are no examples of novel and useful suggestions for teaching based on neuroscience thus far.

More importantly, there are principled reasons to think that educational neuroscience will not help improve teaching methods in the future. First, the common approach of using neuroscience to improve our understanding of a learning difficulty is problematic as it is not clear whether remedial instruction should target the deficits or target the preserved skills of children. The only way to find out is to carry out behavioral studies in psychology. Second, changes in brain states are irrelevant for evaluating the efficacy of an instruction. What matters is not whether the brain changes, but whether the child learns as expressed in behavior.

At the same time, it is important to emphasize that an important goal for neuroscience is to characterize how the brain changes in response to learning, and this includes learning in the classroom. So although neuroscientists cannot help teachers in the classroom, teachers can help neuroscientists by changing the brains of their students (by teaching). This suggests that critics of educational neuroscience might reconsider the analogy of a bridge too far. Perhaps a more appropriate analogy is that there is a one-way street linking education to neuroscience. Educational neuroscience is trying to travel in the wrong direction.

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