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Discussion forum

Don't forget about levels of explanation

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The basic point of this contribution is to emphasise that in understanding the link between neuroscience and developing educational programmes, we have to do more than pay lip-service to the fact that they operate at very distinct levels of description and abstraction. We should clearly acknowledge that there are at least three levels of relevance here – the biological, the cognitive and the behavioural. Current neuroscience straddles the biological and the cognitive and current educational practice and research straddles the behavioural and the cognitive. It is simply premature and disingenuous to claim that anything currently in the literature straddles all three levels. Or at least in such a way as to convey the impression that biological facts and theories currently have implications for educational practice that offer anything distinctive from that offered by cognitive theories. A simple example might make this point. Our concept of autism, and indeed what kinds of educational regimes might be effective in responding to it, has been profoundly influenced by developments in cognitive neuroscience in the past 20 years. If we allow that the acknowledgement of a biological basis to autism stems from work in neuroscience (interpreted broadly, and certainly including genetics) then this has been a major contribution in understanding the cause of autism.

However, we would submit that exactly what this biological basis is and indeed what specific

genes may or may not be involved, or what neurochemical pathways are compromised (or whatever), while of the greatest scientific interest, is of little or no importance to educational planning over and above the basic fact that there is a biological cause of autism. On the other hand, what has been important for possible intervention is the development of theories for understanding the cognitive basis of autism – for example that it might be caused by a deficit in “theory of mind”. This is because cognitive theories only require a translation to an adjacent level of explanation (the behavioural) before meaningful intervention principles can be developed. This level of translation is hard enough but infinitely more attainable than translations over two levels of explanation especially when there is not an intervening cognitive link (what cognitive theory of autism hangs on whether biochemical pathway X or biochemical pathway Y is the cause of autism?). The “levels” approach shows every sign of leading to successful theory development in understanding developmental disorders and linking them to their biological substrates (Morton, 2004). We believe that this is a surer footing than any available set of neuroscientific facts can provide for those interested in developing educational programmes.

None of this is to say that there can be no sound implication of neuroscientific facts and theories for educational intervention, just that this is unlikely to occur without at the very least the development of an intervening cognitive theory. We can think of no better contemporary example than the controversy surrounding DORE Achievement Centres. The DORE programme, now with more than 50 centres worldwide, advertises itself as a unique medication-free approach to help children who have been “labelled” as suffering from a developmental disorder such as dyslexia, ADHD, and dyspraxia. The essence of the programme is a series of exercises designed to stimulate the cerebellum. The rationale is based on the view that there are good neuroscientific reasons to believe that cerebellar

functioning may be implicated in many developmental disorders. Be that as it may, there is but one study (Reynolds et al., 2003) that claims the program is effective for aiding children with a developmental disorder (in this case dyslexia). However, the methodology of this study has been severely criticized in a number of ways (Snowling and Hulme, 2003, Rack et al., 2007, McArthur, 2007 and Bishop, 2007) – the principal one being that the study did not contain an appropriate control group – and led to such a controversy that when a follow-up report was published in the same journal in 2007, five of the editorial board resigned in protest. Our point is that while it may be true that cerebellar functioning is associated with a number of disorders, very much more than this has to be done to justify developing a remediation curriculum for language disorders based on physical activity. First, we would need to discover how the cerebellum is involved in language processing and in particular that part of language processing implicated in developmental disorders. Then we would need to know how physical activity could change those specific and critical features of the cerebellum and in such a way that it could restore a function that already has a developmental history. It seems that quasi-neuroscientific speculation can persuade some even in the absence of any behavioural evidence in its favour. Such is the danger of not being clear about levels of description and explanation.

So how might we do it differently? Our research project (Project KIDS) has a consideration of multiple levels of explanation at its core. Our formulation of hypotheses about the ways in which different cognitive abilities mature is driven by a cognitive model (Anderson, 1992). Over the years these hypotheses have been tested using psychometric and information processing measures targeting specific cognitive functions. Most recently we have felt confident to extend this hypothesizing to brain-based relationships using ERP recordings of children whilst they are performing key cognitive tasks.¹ We can then relate both of these

types of measures to behavioural data relating to the children's psychometric and academic performance. In this sequential and integrated way, we can consider and experimentally explore hypothesized connections between constructs at different levels of explanation rather than guessing at these connections on the basis of unrelated studies taken out of context. This work has also provided us with a control group of typically developing children against which we can compare children who have been identified, at a behavioural level, as having educational problems. One example of such a cohort is children born prematurely. We are exploring potential factors that may contribute to the behavioural sequelae of prematurity without jumping to the conclusion that any consequence must be brain-based because of the interruption to gestation. The hope is that a theory-driven approach informed by the integration of biological, cognitive and behavioural data would be more productive for the next step which is to translate the suggestions from basic research in cognitive neuroscience into appropriate interventions and evaluations of interventions. We are of the view that this integrated approach that concurrently addresses multiple levels of explanation should be encouraged as a prerequisite for the planning of educational interventions.

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