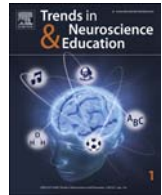


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Editorial

Developmental dyscalculia: Fresh perspectives

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ABSTRACT

This issue of Trends in Neuroscience in Education offers some fresh perspectives on developmental dyscalculia. Here we present an overview of different theoretical approaches to identifying and defining developmental dyscalculia, and a consideration of critical measurement and experimental issues. We note a series of important caveats that must be applied when interpreting the existing research base. While there is currently no generally agreed upon *functional* definition of developmental dyscalculia (DD), the papers collected here represent the wide range of educational and research issues that must be considered when applying neuroscience techniques to the study of developmental disorders of number.

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1. Introduction

Mathematical skills are increasingly important if individuals are to thrive in today's technologically-oriented society. However, evidence suggests that many adults in developed societies possess quite immature mathematical abilities. A 2011 Department for Business, Innovation and Skills survey in the United Kingdom found that 49% of the adult population could only attain standards comparable to 11 year-old children in mathematics (whereas 14.9% achieved such standards in literacy). Furthermore, 23.7% of adults reached only the standards typical for 9 year-old children (compared to 7.1% for literacy). As may be expected from these figures, research on mathematical learning problems lags well behind research on literacy problems, and takes longer to affect educational instruction. For example, during the period of 1985–2006 nearly 5 times as many research papers were published on 'dyslexia' compared to 'dyscalculia' [51]. Hence, it is not surprising that there is *no* generally agreed upon *functional* definition of developmental dyscalculia (DD). In fact, conditions which may or may not be equivalent to DD are labelled by many different names (Box 1). Here we provisionally define DD at the widest possible *phenomenological* level. We define it as persistently weak mathematical performance of developmental origin, related to the weakness of some kind(s) of cognitive function (s) and/or representation(s); appearing when *concurrent* motivation to study mathematics and access to appropriate mathematics education is normal. Research suggests that most individuals who are weak in mathematics do *not* have DD. Here, we will consider DD at the levels of behavioural phenomena, cognitive functions and neural underpinnings, pointing to important controversies in research.

1.1. Behavioural phenomena (operational definition)

At the level of *behavioural phenomena* DD is usually defined operationally as a condition where mathematical achievement is (much) lower than average. Criterion validity is typically provided by

standardized mathematical tests. However, mathematics is a collection of various competences, and is not a well-defined skill as in the case of reading. Consequently, the content of different standardized tests of mathematics can differ markedly. For example, some tests may rely on the interpretation of verbal problems, while others rely on calculations with Arabic digits. Test content *always* differs when tests are aimed at different age groups. Thus, different standardized tests do not necessarily measure the same 'kind' of mathematics and/or the same kinds of skills supporting mathematics. This variability affects diagnosis. Further, there is no agreement on the particular threshold or 'cut-off' test score under which a child should be defined as having DD.

If DD is a specific weakness of mathematics, then false positive diagnoses can only be avoided by testing whether other functions are indeed preserved [48]. However, there is no agreement on the kind of non-mathematical control variables which should be selected (e.g. intelligence and/or reading), nor on whether discrepancy between intelligence scores and mathematical test outcomes should be considered [51]. Indeed, while mathematical problems often appear on their own [61,69], they are also frequently co-morbid with other learning problems [43], especially with reading and spelling problems [46,59]. These discrepancies raise important questions concerning whether co-morbid states of DD are as typical (or even more typical) forms of the disorder than 'pure' DD [44]. It is also important to establish whether co-morbid states represent profiles with qualitatively different mathematical impairments relative to pure states; whether they rely on the co-occurrence of independent cognitive impairments; and whether they rely on causal relations between impairments. The theoretical issue of whether co-morbidity is seen as core to the disorder or it is allowed in the definition at all obviously has strong implications for defining DD.

1.2. Prevalence

These uncertainties concerning which behavioural phenomena should be considered central to DD naturally make it difficult to establish prevalence rates for DD. Prevalence studies have used highly

Box 1—Terms used to describe conditions which may or may not be equivalent with DD.

Dyscalculia
Developmental Dyscalculia (DD)
Arithmetic-related learning disabilities (AD)
Arithmetical disability (ARITHD)
Arithmetic Learning Disability (ALD)
Mathematical Disability (MD)
Mathematics Learning Disability (MLD)
Mathematical Learning Difficulty (MLD)

variable cutoff criteria, ranging from the 3rd to the 25th percentile, and studies differ in whether they have relied on control variables, in which control variables were selected, or whether control variables were considered at all. When control variables are thought to be important for the definition of DD, then prevalence estimates are affected by the intercorrelation of criterion and control variables. Prevalence estimates from 17 studies range between 1.3% and 10.3% (-2 SD to -0.68 SD below the mean in terms of a normal distribution). The mean of these estimates is about 5–6%, and there seem to be no consistent gender differences in DD (see review in [17]; especially Table 1 and Fig. 1). Experimental studies examining the functional basis of DD often ignore prevalence estimates and use very liberal cutoff scores, sometimes selecting children below the 35th (-0.38 SD) and 45th (-0.12 SD) percentiles as representative of low-achievement mathematics groups (see Table 1 in [51]). Such cut-offs include children within the normal range of performance. The extreme variability of children included in different samples means that it is difficult to compare experimental results across DD studies. Hence, in order to be able to differentiate between qualitatively different cognitive profiles, some researchers have classified children fitting various levels of cutoff criteria into different groups (e.g. [25,51]).

1.3. Cognitive functions

This variability with regard to criterion validity (testing instrument, cut-off score and control variables) contributes to the uncertainty about which cognitive function(s) and/or which mental representation(s) is/are affected in DD. One debate concerns whether there are qualitative differences in the cognitive profiles of children with DD [68]. DD may originate from the impairment or weakness of a *single* cognitive representation or function [64]; it may result from weakness in a constellation of *multiple* representations/functions, or indeed, it may be an *umbrella term*, denoting mathematical weakness of unrelated and/or variable functional origins [36]. Theories in adult cognitive psychology and cognitive neuroscience typically follow a modular view, preferring to identify a single function underlying a condition like DD. On the other hand, developmental researchers have shown that mathematical weakness appears in many forms. Hence, search for a single underlying cause of DD may not be an optimal strategy. As long as the underlying factors behind the various kinds of mathematical weaknesses are not understood, it is simply not possible to decide whether various weaknesses stem from the same underlying condition.

The literature offers a wide range of cognitive functions which may be impaired in DD. A popular view is that DD is the consequence of the deficit of a core amodal [49] magnitude representation often called the ‘number sense’ [15]. There are various versions of this ‘core deficit’ hypothesis [7,55]. Other researchers relate DD to impaired links between the magnitude representation and number symbols [14,62]; or to suboptimal automatic activation of the magnitude representation [63]. Yet others link DD to impairment in verbal and visual working

memory [6,23,24,31,53,54,77], impairment in spatial processing [60,61], impairment in attentional function [3,30,77], impairment in inhibitory function [5,6,20,77] and impairment in phonological ability [78]. All these different cognitive functions seem to play important roles in mathematics, and hence can be plausibly related to DD.

It has also been proposed that the field should distinguish between *subtypes* of DD depending on children’s different mathematical profiles, which may be related to different cognitive impairments. For example, some children show weakness in mathematical fact retrieval (which provides shortcuts in both simple and complex arithmetic; [69]), while others show immature procedural/strategy choices, and others appear to have inefficient visuo-spatial manipulations [1,22,79,80]. Rubinsten and Henik [64] suggested that the term ‘Mathematical Disabilities’ should be used as an umbrella term, while the term ‘DD’ should be reserved for core deficits of the number sense. This suggestion raises the question of whether DD should be conceptualised as representing a quantitative extreme of the cognitive skills associated with mathematical achievement (the tail of the normal distribution), or whether it represents a discontinuous *qualitative* difference between DD and typically developing children.

1.4. Heritability

DD is of *developmental* origin, that is, it is not acquired through mental or physical events experienced by an individual who had age-appropriate mathematical skill during an earlier period of life. Rather, DD seems a deficiency of cognitive development that is inherent to an individual. One suggestion is that such an inherent deficiency has a genetic basis [37,72]. However, even the best-built genetic system needs crucial environmental input to achieve its potential. Hence, it is difficult to exclude the possibility that DD is the result of environmental factors which were not forthcoming at the appropriate time earlier in developmental history. Such factors, for example motivational and emotional factors and/or inadequate teaching, may be absent at the time of diagnosis, but may have contributed to DD *in the past*. That is, inherent developmental problems do not necessarily require genetic explanations but may result from (unknown) suboptimal past environmental inputs.

On the other hand, DD shows familial aggregation, which may be attributed to genetic factors [72]. However, besides genetic factors familial cultural and parental attitudes towards mathematics can probably also explain some familial aggregation effects [76]. In fact, a study of twins found that 49% of monozygotic twins (who share all of their genes) and 32% of dizygotic twins had DD, which points to moderate genetic influence [44]. Similarly, a large-scale twin study concluded that mathematical achievement is influenced moderately by *both* genetic and environmental factors, and that mathematical weakness is the quantitative extreme of the distribution of these factors rather than some kind of discontinuous qualitative difference [38]. Importantly, genetic explanations do not provide evidence for the heritability of an isolated number-specific factor. Various basic cognitive abilities, like overall memory capacity or speed of processing, may be under genetic influence, and all these factors can in turn influence mathematical development. Therefore, in principle, heterogeneous genetic influences on more than one cognitive factor may affect mathematical performance.

1.5. A developmental perspective

A critical issue in developmental disorders is how to define ‘age-appropriate’ mathematical skill (see e.g. [18,28]). Is a diagnosis of DD meaningful if a child has age-appropriate mathematical skills until age 7, but later shows signs of learning disability, even though there is no indication of mental/physical trauma [47]

Such questions emerge because we do not yet understand the complexities of *developmental change*. Yet developmental change is a central issue for mathematics, where skill learning requires the incremental acquisition of several layers of information, which must be built on top of each other during primary and secondary schooling. Children continuously learn new information about mathematics that radically change their understanding, change their solution strategies and change the representations that are mobilised [71,73,83]. Hence, while various individual developmental trajectories are possible [79], at least some aspects of mathematical development require a strict succession of learning stages. Suboptimal learning pathways can likely trigger a ‘cascade of mathematics failure’ especially in low socio-economic status children which makes early interventions especially important ([34]; p66; [29]).

Further, a certain cognitive capacity (for example, good verbal and spatial memory) may not be relevant for mathematics learning until (say) age 7, but subsequently may become absolutely indispensable. In such cases, DD may appear after such a cognitive mechanism becomes of central importance for mathematics performance. Temporal variability in development also leads to instability or uncertainty in DD diagnoses. For example, a child who fits DD criteria in one year may show better performance the following year, taking them above the threshold for diagnosis. In such cases poor performance may be attributed to transient factors such as motivational factors, or to *temporarily delayed development*, rather than to a central and persisting cognitive impairment. Studies suggest that about 50–60% of children with DD have a persistent condition [47,70,74]. Around 95% of children with DD show long-term weak mathematical performance [70].

Importantly, even the subtype of DD can show temporal variability within individuals [74]. Hence, it is critical to conduct longitudinal studies, replicating test results at different time points, to demonstrate the persistent nature of DD and the stability of the different proposed subtypes of DD. Longitudinal assessments (which are very expensive) are also important because the analysis of growth curves enables the determination of whether DD is best understood as a persistent *developmental delay*, or is better captured by theoretical models suggesting qualitatively different development [47]. Overall, a truly developmental perspective of developmental disorders requires a full description of *typical and atypical developmental trajectories* [35]. Models based on adult data are at best insufficient with respect to explaining the emergence of DD and the cognitive complexities of mathematical learning.

1.6. Neuroimaging in DD

To date, there are surprisingly few studies examining the brain correlates of DD. A popular theory, originating from neuroscience studies, is that DD is related to impairment of the core magnitude representation. This theory is often called the deficient number module deficit theory [7,55]. This core number module or ‘number sense’ is thought to reside in the bilateral intraparietal sulci of the brain (IPS). Some notes of caution are appropriate, however. While children with DD do indeed seem to show *structural* abnormalities in the parietal cortex [33,58,66], the functional significance of these findings is not yet clear. Firstly, the tests and tasks used in some behavioural and neuroimaging studies lack clarity with respect to the functions that they measure, and their criterion validity is unclear. Various papers may label functionally dissimilar measures as ‘number sense’ measures (for example, speeded dot pattern comparison, symbolic number comparison, counting dots, positioning numbers on a number line). Hence, the relationship between theoretical labelling and the actual measures used can be questioned in some cases. Secondly, some tests thought to

measure core capacity, such as non-symbolic dot-comparison tasks, can be strongly affected by visual stimulus properties which confound a clear interpretation of performance [26,27,86]. Thirdly, in studies where non-numerical control conditions are lacking, there is a need for caution when drawing number-specific conclusions on the basis of visually-loaded tests. Fourthly, with regard to brain substrates, 4 out of 6 *functional* MRI studies investigating the deficient number module/core IPS deficit hypothesis did not provide supporting neuro-imaging data [12,39,41,42,87]. Further, 5 out of 6 of these studies did not provide supporting behavioural data (*ibid* and [52]), indeed, only 1 out of the 6 studies provided both supporting behavioural and imaging data [57]. Electroencephalographic (EEG) investigation of DD could also not find evidence for a deficient core magnitude module [75,88]. A further study demonstrated altered IPS function in children with DD relative to controls in a *working memory* task [58]. As structural and functional brain differences between DD and control children appear in various brain regions besides the IPS [12,39,42,52,65,66], the absence of sufficient studies make it difficult to properly evaluate the deficient number module/core IPS deficit theory. Indeed, a whole *network of brain* regions seems to be affected in DD, rather than a single area [21]. This is in fact more consistent with the variability of cognitive impairments observed in DD.

With regard to evaluating the deficient number module/core IPS deficit hypothesis, it is also important to point out that the IPS is involved in many different cognitive functions in addition to the proposed number sense. The IPS seems to be involved in most cognitive functions that are important for mathematics, like working memory [10,11,19,45,81], attention [10,13,67,82], inhibitory function [8,50] and spatial processing [85]. Therefore, impairment of any of these important functions could plausibly explain IPS abnormalities in DD [87]. Consequently, when IPS effects are demonstrated with no supporting behavioural data, it cannot be concluded that these demonstrations support the deficient number module IPS deficit theory. Such conclusions are in fact invalid ‘reverse inferences’, because the inference that the imaging findings relate to the cognitive functions that are *assumed* to be tested may not be justified [56].

This inferential point also highlights a frequent *implicit* assumption in neuroimaging studies, which is that brain data are stronger than behavioural data (see [84]). Yet brain data are typically not interpretable if behavioural data are missing. Finally, the frequent *causal interpretation* of brain data should be noted. Brain and behaviour are interdependent. Whatever we do behaviourally can be traced back to brain function, therefore finding a brain difference between two groups of participants with different behavioural characteristics (e.g. different maths scores) does not imply that the neural difference is causal in the behavioural difference. It may well be that a third factor (e.g. reduced maths tuition in the past in the weaker group) caused both the brain and the behavioural differences. In this example, too little tuition may inhibit the optimal development of certain parts of the brain *and* may lead to worse performance. That is, the brain difference itself may be the consequence of differences in tuition and not an independent (primary) cause of the differences in mathematical behaviour. Hence, merely detecting a brain difference is not a valid basis for concluding that a certain brain area is damaged or dysfunctional for biological reasons, and that this is the cause of a behavioural difference.

1.7. Intervention

Persistent mathematical weakness is often labelled ‘Mathematical Disability’. The 2010 Equality Act of the United Kingdom defines disability as ‘a physical or mental impairment that has

a substantial and long-term negative effect on your ability to do normal daily activities'. If DD is an example of such a serious inherent handicap, then the most important question for educational neuroscience is whether this handicap can be remediated by appropriate interventions. In fact, theories proposing the lack of a core module may suggest to educators an irreversible condition. At the moment there are very few intervention studies with mixed results on the impact of training magnitude comparison (see [9]). Other studies investigated training effects in response to working memory tasks [32,40]. The study of *individual differences* is particularly important for optimising interventions, and indeed a focus on individual differences also offers a *strong test of theories*. For example, a theory may look convincing in group studies, but may prove inefficient at the individual intervention level. Finally, it is important to note that intervention efforts may be hampered by participants' attitudes towards maths, for example mathematics anxiety, which results in participants avoiding mathematics-related situations [2,18]. Attitudes of pupils and teachers may be particularly important when it comes to explaining gender differences in mathematics [4,16].

2. Conclusion

This issue of Trends in Neuroscience and Education presents several alternative theoretical approaches to DD. We hope that these fresh perspectives will bring the field closer to an accepted functional definition of DD. Meanwhile, it is clear that operational diagnoses should rely on the criterion validity provided by standardized tests. Indeed, it is advantageous to use more than one test, aiming at different content and to record performance in a range of control variables as well, so that generalisability across samples can be assessed. Conclusions are stronger if diagnoses are confirmed by longitudinal replication. A particularly important goal for the field of DD is to describe individual differences across typical and atypical developmental pathways using longitudinal data and the wide range of psychological constructs implicated in DD. Distinctions are required between (biological) markers/correlates/consequences of DD and (biological) causes of DD. Behavioural and imaging studies should test theories against each other rather than focus on a single theory, and emotional, motivational and anxiety-related aspects of mathematics also need to be considered.

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