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Are specific learning disorders truly specific, and are they disorders?

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ABSTRACT

Specific learning disorders, such as dyslexia and dyscalculia, are frequently studied to inform our understanding of cognitive development, genetic mechanisms and brain function. In this Opinion Paper, we discuss limitations of this research approach, including the use of arbitrary criteria to select groups of children, heterogeneity within groups and overlap between domains of learning. By drawing on evidence from cognitive science, neuroscience and genetics, we propose an alternative, dimensional framework. We argue that we need to overcome the problems associated with a categorical approach by taking into account interacting factors at multiple levels of analysis that are associated with overlapping rather than entirely distinct domains of learning. We conclude that this research strategy will allow for a richer understanding of learning and development.

1. Introduction

Specific learning disorders (SLDs) such as dyslexia and dyscalculia are studied by researchers to inform diagnosis and remediation. In addition, SLDs are investigated in efforts to advance our understanding of the mechanisms that underpin learning in the affected domains. Reading and arithmetic are considered core subjects in formal education and are predictive of educational achievement and income in later life [1]. One of the dominant frameworks used in developmental research to understand the mechanisms underlying the development of such neurocognitive abilities has been and continues to be the study of children who present with SLDs. The rationale for studying selected groups of children that present with specific deficits is that understanding what causes and correlates with their deficits can elucidate the neurocognitive mechanisms underlying the development of the affected competencies more generally.

The study of SLDs has been inspired and continues to be driven by the study of adult neuropsychological patients who present with specific deficits in one domain of cognitive processing, which are dissociated from intact functioning in another [2]. Against this background it has been hypothesized that SLDs are underpinned by core domain-specific causes (e.g., 'a defective number module' for dyscalculia [3]). The key prediction is that identifying potential core variables that explain the occurrence of SLDs will inform targeted, theory-driven interventions for children who are struggling to acquire functional levels of academic abilities. Despite criticism that has been leveled at this single-deficit view of SLDs (see e.g., [4,5]), it remains the default view and basis for research.

The quest of identifying specific neurocognitive factors that explain and predict academic skills using comparisons of groups of children with different deficits has resulted in mixed and heterogeneous evidence. In what follows, we will discuss both methodological and conceptual issues associated with investigations into SLDs that aim to inform our understanding of neurocognitive development more broadly. We close by suggesting alternative theoretical and methodological directions that, in our opinion, are necessary to further advance our understanding of the factors that are associated with individual differences in children's ability to learn across domains.

2. The trouble with classification

In both basic research and clinical practice, categorical classification schemes are applied to select groups of children for further study or clinical intervention. For example, the DSM 5 classifies SLDs as neurodevelopmental disorders characterized by difficulties acquiring and applying academic skills. It specifies that performance of at least 1.5 standard deviations below the population average or below the 7th percentile is required for greatest diagnostic certainty. Furthermore, the DSM 5 states that these difficulties must have persisted for at least six months, despite targeted interventions, and cannot be accounted for by inadequate schooling or developmental delays (e.g., intellectual disability or other neurodevelopmental disorders; [6]). Within clinical settings, the diagnosis of an SLD results in, for example, more access to remedial teaching, special programs and accommodations. At the

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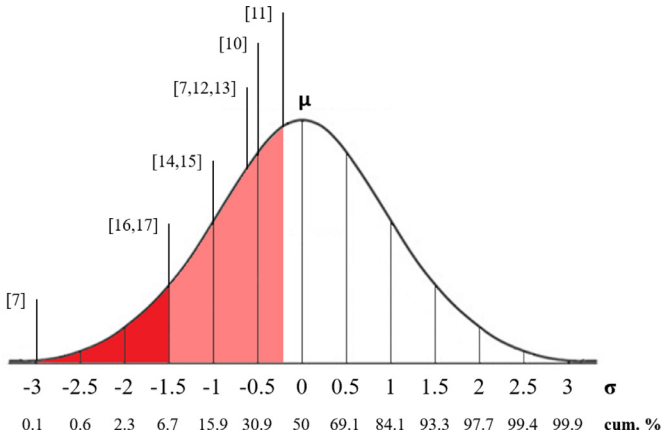

Fig. 1. Visualization of the variation in cut-off scores that have been used in a limited subset of studies of dyslexia and dyscalculia. Children scoring below 1.5 standard deviations of the population mean, and who therefore would be classified as having an SLD according to the DSM 5 criteria, fall within the dark red area of the distribution. More lenient cut-off criteria that have been used in research would result in overestimations of up to 37 percent of children being labeled as having SLDs, indicated in pink [12,14,15,17]. See [22] for a similar figure depicting the variability in selection criteria for dyscalculia.

outset, we would like to stress that the goal of this Opinion piece is not to criticize clinical practices. Instead, our aim is to discuss theoretical and methodological issues that are inherent when studying SLDs in an effort to inform and further our understanding of learning and development.

Even though the criteria for a clinical diagnosis of an SLD are clearly described, the selection criteria used to categorize participants into groups for research purposes are typically highly variable between studies (and within studies, see [7]). In view of this high variability in cut-off points, it is hardly surprising that reported prevalence estimates of SLDs have also been variable (see e.g., [8,9]). If researchers were more consistent in their classification and applied the DSM 5 criteria, the occurrence of SLDs should be, by definition, 7 percent. In fact, taking the DSM 5 criteria of adequate educational opportunities and persistency into account, 7 percent may even be an overestimation of the prevalence of SLDs. In the scientific literature, however, some of the cut-offs used have been as high as the 30th [10] or 37th percentile [11]. Such practices invariably result in a large overestimation (with reference to the diagnostic criteria) of the number of children being classified as presenting with an SLD (see Fig. 1).

Furthermore, contrary to the DSM 5 recommendations, many researchers will classify children as having an SLD based on one assessment at one time point, rather than on the presence of persistent difficulties (e.g., [13,16] but see also [18–20]). In addition, variability in the assessments that researchers use leads to variability in which children are assigned to the groups. In other words, a child could be classified as having an SLD using one measure, but as typically developing using a different, equally valid and reliable measurement tool (e.g., [21]). Critically, this heterogeneity in selection criteria implies that any attempt to uncover common patterns of performance across studies is highly unlikely to yield informative meta-analytic estimates. Indeed, if different researchers apply different selection criteria and measures for group inclusion, yet at the same time employ the same terminology to describe their groups (e.g., 'children with dyslexia' for low reading achievement), comparisons across studies as well as literature syntheses should be interpreted, at the very least, with great caution or avoided entirely.

3. Quantitative not qualitative differences

Beyond these methodological limitations in the published research literature examining the neurocognitive underpinnings of SLDs, the interpretation of such data is marred by additional problems. Assigning labels such as 'dyscalculia' or 'dyslexia' to selected groups of participants may imply that these children are qualitatively different from those that score outside of the selected cut-off. However, such reasoning is unwarranted because selection is based on an arbitrary point along the normal distribution of ability scores on a particular measure. Put differently, children who perform below any given cut-off point are not necessarily qualitatively different from those who scored above that criterion. Importantly, if groups are insufficiently different from each other, the potential absence of group differences on a particular measured variable might be interpreted as though this variable is not associated with deficits in the ability of interest. Alternatively, it might be that the between-group variability is simply too small to detect subtle effects. Relatedly, sample sizes of these ill-defined groups of children characterized as having an SLD are often very small (see e.g., [23,24]), which can lead to overestimations of effect sizes of group differences, and limits strong, replicable inferences [25,26].

Indeed, a recent analysis showed that neuroimaging studies of mental disorders (in which subjects were categorized into a diagnostic group or a control group using pattern recognition analyses), are biased by sample size: small sample sizes were found to be associated with inflated classification accuracies [27]. The important implication here is that, in neuroimaging studies of Schizophrenia, depression and ADHD with adequate statistical power there may actually not be substantial evidence in favor of specific neural differences between disorders vs. controls (see also [28] for a discussion on the lack of specific between psychiatric disorders). These finding demonstrate that the use of small sample sizes in neuroimaging research is highly problematic and obscures the heterogeneity and overlap of disorders. Such data can give the false impression that there are strong categorical boundaries between different disorders.

Because the dominant, categorical classification approach on its own does not permit inferences about qualitative differences, many researchers studying learning disorders (see e.g., [29]), and psychiatric conditions such as Autism Spectrum Disorder (ASD; see e.g., [30,31]) have instead advocated for the application of a dimensional approach as a more productive research strategy [32]. This approach is built on the premise that there is no qualitative discontinuity in the distribution from low to high performers. In this vein, such an approach embraces the individual, quantitative variability within the overall population and avoids classification based on arbitrary cut-offs. Indeed, it has been shown that studies of individual differences in typically developing children drawn from population-representative samples, can completely predict the pattern of deficits experienced by individuals with SLDs.

We can illustrate this point by considering an example from research into the cognitive underpinnings of arithmetic ability. Here, symbolic number processing (the ability to judge which of two Arabic numerals is numerically larger) has been suggested as an important scaffold for the development of arithmetic skills [33,34]. Importantly, this association between symbolic number processing and arithmetic has been investigated both in children who were selected using cut-off scores, as well as in correlational studies with unselected samples. Do their conclusions differ?

To answer this question lets consider the following: using a categorical approach, De Smedt and Gilmore [35] selected children with mathematical learning difficulties (MLD; scores below the 16th percentile on a standardized mathematics achievement test), low achieving children (LA; scores between the 16th and the 25th percentile), and typically achieving children (TA; scores above the 35th percentile). Children in all groups performed a symbolic number comparison task. A comparison of the three groups revealed that the MLD group responded more slowly than the LA and TA groups (see Fig. 2). Using a dimensional approach, Holloway and Ansari [36] investigated the correlation between arithmetic performance and symbolic number processing in an

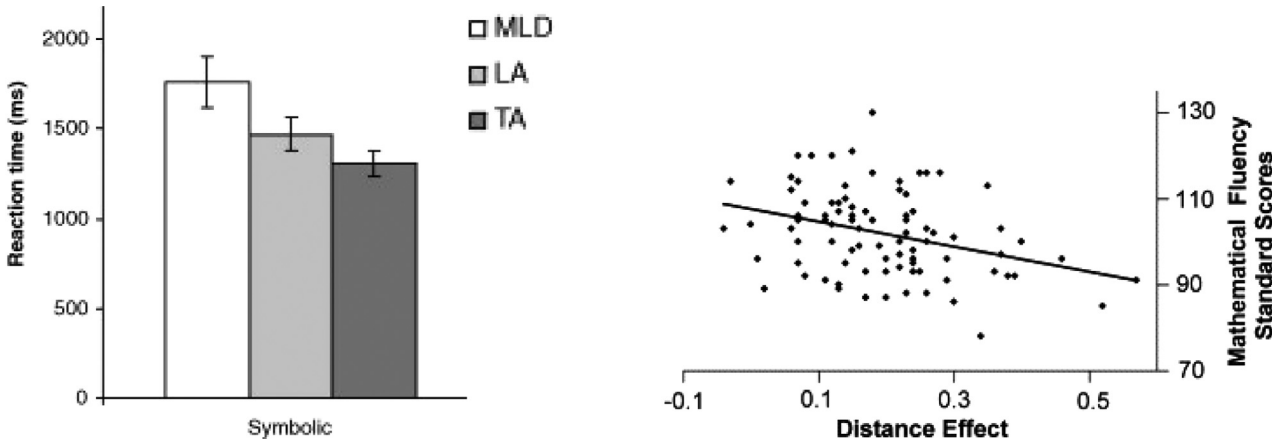

Fig. 2. The left panel depicts the results from a study using a categorical approach [35], the right panel from a study using a dimensional, individual differences approach [36].

unselected sample of children. Their findings revealed a linear association between symbolic number processing and arithmetic skills (see Fig. 2).

These two studies, using different approaches, show the same pattern of results: symbolic number processing and arithmetic ability are positively related. Hence, there is little that the categorical approach reveals about the association between symbolic number processing and arithmetic that cannot be gleaned from the continuous, dimensional approach. This is only one example of many that show that investigating individual differences can completely predict studies in which participants are categorized into groups using arbitrary cut-offs. Such examples reveal that grouping according to cut-offs does not necessarily reveal qualitatively different patterns of behavior.

4. Heterogeneity within disorders

The most prevalent view of SLDs among researchers and clinicians is that these are specific in nature. For instance, children with dyslexia display severe deficits in reading, whereas children with dyscalculia present with arithmetic deficits. Moreover, these specific behavioral profiles are thought to arise from isolated core deficits shared among children with the same label.

Studies of SLDs are guided by evidence from case studies of adult neuropsychological patients, where lesions to single brain regions have been associated with specific deficits in cognitive processing within single domains (see e.g., [37]). However, this conceptual leap from adult neuropsychological patients to children with developmental difficulties is flawed. More specifically, despite having been categorized using the same selection criteria, children belonging to the same group present with substantially variable phenotypes. For example, not all children with dyslexia show phonological deficits [38], and alternatively, other candidate correlates of dyslexia have been revealed [39–42]. Moreover, variable profiles of strengths and weaknesses across the purported core deficits have been revealed within groups of children with dyslexia [43]. Patterns of within-group phenotypic variability have also been reported in groups of children labeled as having dyscalculia. For instance, Bartelet and colleagues [44], using cluster analyses, found that the performance of children on a large battery of number processing measures resulted in seven independent clusters. Thus, even *if* one accepts the notion of SLDs, it is clear that there exists significant within-disorder heterogeneity. Therefore, no single core deficit can possibly explain all patterns of performance within groups of children with the same label (see also [45,46]). It is important to stress that this well-documented within-disorder heterogeneity is completely discounted when statistically comparing groups, since only between-group variability is investigated.

Beyond evidence from studies that have examined the cognitive correlates of SLDs, investigations of their neural correlates have further strengthened the notion that SLDs cannot be reduced to core deficits that may be reflected in isolated brain regions. For example, neuroimaging studies of children with dyslexia have uncovered aberrant brain activation in a widespread left-lateralized network, comprising inferior parietal, frontal, temporal and fusiform regions [47]. In this context, it is important to emphasize that some of these regions showing aberrant activation levels fall outside of the “neural reading network” (see e.g., [48]). This implies that there are more brain areas associated with low reading performance than would have been predicted based on a single neural deficit associated with dyslexia, in which case the neural differences would have been limited to (an isolated brain area in) the reading network. Similarly, neuroimaging studies of children with dyscalculia have predominantly focused on atypical activation in the intraparietal sulcus, given this brain region's well-documented role in numerical and mathematical processing [49]. However, the brain areas that are differentially activated in children with dyscalculia consist of a broader network, including frontal, parietal, occipitotemporal and hippocampal areas [50,51]. Such reports of aberrant neural activation in widespread brain areas, both for dyslexia and dyscalculia, imply that there is no evidence for isolated neural deficits (in terms of single brain regions) associated with either SLD.

It is important to consider that, like behavioral studies, neuroimaging studies are typically characterized by small samples of children categorized as having SLDs with variation between studies in the diagnostic criteria used to label children [52]. Therefore, it is possible that the observed heterogeneity in brain activation within and across studies is reflective not of the existence of multiple neural correlates associated with an SLD, but rather of weak and noisy estimates thereof [25,52–55]. Consequently, we would like to stress that any syntheses or meta-analytic estimates of such data should be treated with great caution and a healthy dose of skepticism.

Like cognitive and neuroimaging studies, research into the genetic basis of SLDs has also failed to reveal evidence of clear-cut, unique causal factors. In the past, researchers frequently attempted to associate single genes with behavioral outcomes [56,57]. Many of these studies have been difficult to replicate [58]. Recent research into the genetic correlates of cognitive phenotypes has demonstrated why the absence of evidence for specific candidate genes for SLDs is entirely unsurprising. Specifically, the advent of methodological and analytical tools that have made it possible to consider genetic variants across the entire genome (Genome Wide Association Studies, GWAS) and relate them to cognitive phenotypes, has consistently shown that *multiple*, not single, genetic variants correlate with cognitive phenotypes (see e.g., [59]). The repeated finding that many genetic variants are related to

specific phenotypes, with each genetic variant having a very small effect, has become known as the fourth law of behavioral genetics [60]. Such polygenic (multiple genetic variants are correlated with phenotypic variation) effects have not only been identified for intelligence [59], but also for reading ability [61], mathematical ability [62] and overall educational attainment [63]. These polygenic effects provide yet further evidence that SLDs can only be reasonably explained by adopting a framework that considers multiple causal pathways at multiple levels of analysis (see also [29]).

5. The case for overlap between disorders

Thus far, we have concentrated on the plausibility of isolated deficits. However, children with an SLD in one domain (e.g., reading), will frequently exhibit difficulties in other domains (e.g., arithmetic): this comorbidity or co-occurrence of different SLDs is remarkably high [64]. However, in the search for specific, homogeneous, and qualitatively different groups of children, researchers have not considered comorbidity sufficiently in both their diagnostic criteria and consequently in their causal models of SLDs. It is therefore not surprising that many studies have considered this comorbidity as a confound. Researchers investigating the cognitive correlates of dyslexia do not routinely include measures of arithmetic (see e.g., [65]) and, similarly, investigations focused on understanding the origin of dyscalculia rarely consider the reading skills of children (see e.g., [66]). Furthermore, when researchers do include measures of arithmetic (in the context of dyslexia) or reading (in the context of dyscalculia), these measures are often matched across groups, or included as covariates (see e.g., [67,68]).

A small body of research has investigated three accounts seeking to explain the cognitive mechanisms underlying the co-occurrence of SLDs [69]. First, the domain-specific deficit or additive account states that different SLDs arise from distinct, domain-specific correlates and that deficits in the comorbid group arise from an additive effect of the unrelated deficits of the isolated disorders [70,71]. Second, the common deficit account posits that atypical functioning in children with co-occurring difficulties arises from a single factor that explains variance in both domains of learning (e.g., phonological processing in children with math and reading problems [72]). Third, the domain-general account proposes that domain-general factors, such as working memory or attention, cause deficits across multiple domains of learning [73,74]. Unfortunately, the results from the few studies aimed at uncovering the cognitive underpinnings of co-occurring SLDs have not lead to converging evidence that unequivocally points to one of these accounts as being the most likely to explain existing data.

At the neural level, research investigating the origin of the comorbidity between SLDs is also scarce, again reflecting a bias in the literature to focus on single groups of children that present with impairments in one domain but not others. There have been some reports of neural overlap of arithmetic and reading [75–77], but those were solely based on the activation of similar regions in the context of separate tasks. Inferences from such studies are inherently limited, given that neural activation in similar regions does not imply that similar neural processes are involved (see [78]). However, recently, two neuroimaging studies have directly contrasted the neural correlates of different SLDs. Strikingly, both reported neural similarity between SLDs. Neural similarity is usually reflected in null-results (i.e., no differences between groups) and is therefore hard to interpret, since absence of evidence is not evidence of absence. However, in both studies, state-of-the-art techniques were applied to bypass the problems of drawing inferences from null-results. Specifically a DTI study in adults [79] used Bayesian statistics (which can be used to quantify evidence for the null hypothesis [80]) and reported moderate evidence in support of the null-hypothesis that there are no group differences in white matter integrity within the arcuate fasciculus and corona radiata.

In a similar vein, using multivariate subject generalization analysis, an fMRI study in school-aged children found that the neural activation patterns elicited by an arithmetic task were indistinguishable between groups of SLDs. These data provide evidence for neural similarity across SLDs and therefore speak against the notion that SLDs are associated with specific and separable neural deficits [81]. Clearly, more neuroimaging research is needed to confirm this lack of specific neural deficits for different SLDs. However, these studies suggest that specific deficits do not map one-to-one onto distinct neurocognitive processes and that the search for specific neural deficits for specific SLDs is likely to be an unwarranted and unfruitful approach. These conclusions are convergent with a recent meta-analysis that demonstrated that the neural correlates of different psychiatric disorders such as Schizophrenia, anxiety disorders and bipolar disorders may not be as specific as originally thought, and that there exists substantial overlap between disorders [77].

Evidence from genetics also shows that there is genetic overlap between different cognitive phenotypes. Findings from twin studies and GWAS have demonstrated that the same sets of genetic variants relate to different cognitive phenotypes [82,83]. Such findings led to the 'generalist genes' hypothesis [84], which posits that there is pleiotropy, or genetic correlation, between domains such as reading and arithmetic [85,86]. Specifically, research has shown that sets of genes that influence reading also play a significant role in explaining individual differences in arithmetic, and vice versa. These findings demonstrate that specific genetic influences on either reading or arithmetic are weaker than shared genetic influences [82,83]. For instance, a Genome-Wide Complex Trait Analysis study by Rimfeld and colleagues [82] revealed substantial overlap between genes involved in arithmetic and reading, even when genetic associations with intelligence were accounted for. Thus the evidence from genetics provides yet further support that different SLDs are not as separable as previously thought and, moreover, that they are characterized by a strong biological overlap. Therefore, focusing on specific, separable associations is, in our opinion, myopic (see also [87]).

When considering the evidence from genetics it is also important to return to the the distinction between qualitiative versus quantititative differences (discussed in section 3 above) between children classified as presenting with an SLD and those in the normal range of performance. Research in genetics has demonstrated that genetic variants that are associated with an SLD are also linked to individual differences in the normal range of the corresponding academic ability (i.e., reading for dyslexia and arithmetic for dyscalculia; [83,84]). Put differently, the same genes that contribute to individual differences in the lower end of the distribution, also contribute to individual differences in the upper end (see [25] for a review). Therefore, convergent with the behavioral evidence discussed above, findings from genetics do not provide evidence for qualitative differences between low and high performers. Instead, it shows that the same genes influence performance across the entire spectrum of performance. Thus genetics has revealed that similar genetic variants affect variability in seemingly different phenotypes and that the same genetic variants affect phenotypic variation across levels of ability.

In view of this, we contend that the use of the term "disorder" in SLD, which implies a qualitative difference between those diagnosed and those not diagnosed, cannot be justified. The use of the term "disorder" also assumes that the causal pathways that result in low performance could be different from the pathways to typical or high performance. In contrast, we argue, that it is more productive and reflective of the state-of-the art in behavioral sciences, neuroscience and genetics to adopt a dimensional framework. Such a framework predicts that there is no separate cause for low performance, but rather that individual differences between all learners are likely caused by the same genetic, neural, cognitive and behavioral causal factors. To gain more insight into these factors, we need to study the entire distribution. Importantly, we would like to emphasize that this should not be taken to necessarily have implications for clinical practice. Undeniably, the consequences of low performance for students' daily lives still need to

be considered and acted upon. From a scientific perspective, however, a dimensional approach will enhance our ability to better characterize causal factors for individual variability in performance (including low performance).

6. Moving beyond the study of specific learning disorders

In light of the conceptual and methodological quagmires afflicting the empirical study of SLDs, we suggest a shift away from the status quo.

Specifically, we propose that in order to make progress, researchers ought to abandon categorical, group comparison paradigms, and instead pursue a dimensional approach. As discussed throughout this paper, labeling children as having SLDs for research purposes is based on arbitrary criteria and, so far, no biological markers have been revealed to suggest that these criteria are convergent with the existence of qualitatively different groups of children. Therefore, we contend that, because those arbitrary cut-offs clearly do not result in groups that have specific and separable deficits, such an approach stifles cumulative science and curtails meaningful inferences. Adopting a dimensional framework reveals individual variability in academic abilities across the overall population and thus across the entire spectrum of performance.

Additionally, we suggest broadening this previously proposed (see e.g., [29,32]), yet rarely implemented shift towards a dimensional methodological approach. Specifically, it is clear from the above-discussed cognitive, neural and genetic research that the factors explaining performance across different domains of academic learning may not be as specific and separable as previously assumed. Therefore, we think it is necessary for researchers to carefully consider the influence of multiple, potentially shared factors on overlapping cognitive abilities, such as reading and arithmetic. We would like to emphasize that our aim is not to deny the existence of *any* specific factors. However, the evidence outlined in this Opinion article, suggests that the case for specificity may be weaker than the case for overlap. We therefore believe that the focus of research should be extended to investigating multiple, potentially overlapping factors across multiple levels of analysis, rather than solely focusing on the specificity of various domains of learning.

Against this background, we propose that more progress will be made in research on academic achievement by adopting a *dimensional* framework investigating the influence of *multiple* correlates across levels of analysis (cognitive, neural, genetic and environmental), and their interactive effects on *multiple* (not singular) phenotypes. It is important to stress that we are proposing this framework specifically for research studies that seek to uncover neurocognitive correlates of learning across domains. As emphasized above, the purpose of this Opinion paper is not to propose changes to the way that clinical diagnoses of learning disorders are made or the way in which remedial teaching or access to additional resources is organized. Although using a dimensional model in clinical settings seems appropriate in light of the research evidence put forward here, a shift away from diagnostic categories would have severe and practical implications. Specifically, for SLDs, policies regarding eligibility and cost reductions for remedial teaching would need to be revised, which would require a complete shift in thinking about SLDs among psychologists, teachers, parents, politicians, etc. It is our opinion that until researchers embrace the kind of methodology proposed here, it is unlikely that a similar shift will occur in clinical practice, policy and education.

In this revised, dimensional approach, large samples of children should be meticulously phenotyped with respect to their neurocognitive characteristics, comprising not only the typical domain-specific correlates of academic abilities identified in the past (e.g., phonological processing for reading, number processing for arithmetic), but also more domain-general correlates, such as working memory. Indeed, neurocognitive and genetic research (described above) has demonstrated that there is no evidence for single neurocognitive factors capable of explaining variation in specific academic abilities. One such domain-general factor that has begun to be investigated to explain the overlap between reading and arithmetic is procedural learning (the learning and control of skills and habits [88]). Specifically, procedural learning has been found to be associated with both reading ability [88,89] and arithmetic achievement [90]. Continuing to focus research on identifying separate explanatory factors for different abilities that are in fact highly related, would be inefficient, uninformative and limit scientific progress.

Beyond the study of individual differences, which is correlational in nature, the approach we propose can also be extended to experimental studies, such as interventions and training studies. Given the substantial case for the absence of qualitative differences, discussed above, there is no a priori reason to expect that experimental manipulations that boost performance in unselected samples would not generalize to groups of children that fall below an arbitrary cut-off. Of course, this still needs to be directly examined. If true, it would imply that evidence-informed principles of high-quality instruction can be effectively applied to high and low-performing students.

It is important to note that we have focused on research on reading and arithmetic ability, which are arguable the two domains of learning within which SLDs are most commonly studied to make inferences about general neurocognitive processing. However, we propose to extend this shift in approach beyond these two SLDs, and to include other critical cognitive processes, such as spelling, language, writing and attention. Indeed, comorbidities have also been reported between dyslexia and dyscalculia, and developmental disorders such as dysorthographia, specific language impairments, dysgraphia and ADHD (see e.g., [91–94]).

Clearly, the changes we propose represent a substantial change and rethinking for research on learning difficulties. However, similar shifts in conceptual and methodological approaches have already taken place in the study of other mental conditions (see [95]). For example, in the study of Autism Spectrum Disorder (ASD), researchers have acknowledged the fact that there is no qualitative distinction between children with and without a formal diagnosis of ASD, and that the spectrum of symptoms is continuous, albeit lower in terms of severity, in the non-diagnosed population (e.g., [31]). The approach we suggest here is conceptually similar to the National Institute of Mental Health of the United States' Research Domain Criteria (RDoC) project. The RDoC project proposes a dimensional methodological framework to study mental disorders, focused on integrating evidence at multiple levels of analysis, in order to better understand the correlates underlying individual differences in human behavior. Thus, there exists substantial precedent in other domains for what we are proposing for the study of SLDs.

While we are confident that using a dimensional approach investigating multiple correlates associated with multiple phenotypes will further our understanding of the development of cognitive abilities better than a categorical, single-deficit approach would, we do acknowledge that there are potential research questions that might be solved better using a categorical approach. For example, in the context of testing the effectiveness of intervention programs, randomized controlled trials (RCT) can be a valuable approach. RCTs require the recruitment of a sample of (low performing) children, half of whom receive an intervention program, and half of whom serve as a control group (see e.g., [96,97]). These research paradigms can be very fruitful for the development of interventions and remedial teaching programs. However, in view of the above, we suggest that it is necessary to take the inevitable heterogeneity within these samples into account by including an elaborate battery of cognitive measures. These measures could help explain why some intervention programs work better for some children than others.

Moreover, there may be instances in which groups of children are in fact qualitatively different, for example in cases of rare genetic variants (see e.g., [98]). In this case, we argue that group comparisons can in

fact be valuable research approaches. However, in the vast majority of the studies in the learning disorders literature, groups are not based on biomarkers, such as rare genetic variants, but instead are defined using arbitrary cut-offs.

Taken together, we do not wish to suggest that specific causes of learning difficulties in reading or math will never be isolated. However, for the reasons discussed above, we believe that the current research agenda is unlikely to do so, and that the insights from cognitive psychology, genetics and neuroscience suggest that such discoveries are unlikely to occur in the future.

7. Concluding remarks

The overall goal of studying children's academic abilities, such as reading and arithmetic, is to constrain evidence-informed strategies for fostering and training the neurocognitive mechanisms that underpin the development of reading and arithmetic skills. To that end, most studies have focused on deficits in children scoring at the lower end of the distribution, using group comparisons between low and high performers. In this Opinion article we have reviewed evidence that leads us to the conclusion that a multi-level, multi-factorial, dimensional framework is vastly superior compared to the status quo: a categorical approach.

Because development is not a static, predetermined process that unfolds at the same rate and via the same routes for all children, longitudinal studies that allow for the investigation of dynamic and probabilistic processes are necessary. We acknowledge that longitudinal studies are very challenging, time-consuming and expensive enterprises. However, only by investigating how interacting factors at multiple levels change longitudinally in their association with academic abilities (and therefore also low academic abilities), will we be able to gain more insight into how children acquire, use and achieve mastery in academic abilities over developmental time. This will refine our ability to determine the optimal timing and content of specific interventions for low performing children [99,100].

The change in approach we have advocated for in this Opinion piece necessitates research across multiple levels of analysis. This challenge may be addressed by consortia of multidisciplinary teams, in which expertise at various analytical levels is shared between researchers. It is clear that the type of studies that we propose are large-scale, high-powered, complex and expensive. Such studies will inevitably require researchers to share data across multiple sites, in ways that are already common in the field of genetics (e.g., COGENT Consortium, UK Biobank, GENUS).

We acknowledge that this proposed shift in methodology may be difficult to implement throughout the field immediately. Large-scale, multi-level studies are not always feasible or affordable. However, there are more fine-scale methodological adjustments that could be implemented at a smaller scale in individual labs right away. These include adopting dimensional frameworks rather than comparing groups, and stepping away from focusing on single domain-specific causal factors (e.g., only investigating the role of phonological processing in the context of reading). Additionally, there are examples of large-scale initiatives in psychology, such as the Psychological Science Accelerator Project [101] and the Many Labs Project [102], that join forces to accelerate the accumulation of generalizable evidence and to address the replication and reproducibility crises [26]. Therefore, we believe that it is possible to unite and organize multiple labs to coordinate data collection and set up studies that are more ambitious. Indeed, one of the hopes we harbor is that this Opinion paper will initiate discussions between researchers that will focus on how to make our suggestions actionable. Furthermore, we believe that adopting open science practices, such as data sharing, will play an integral role to fully implement this proposed shift in methodology. These practices not only lead to more reproducible research, but will also help build up larger databases and encourage collaboration between disciplines and research units. To this end, it is important that researchers provide elaborate descriptions of the measures they used, and that they use precise terminology when discussing their topic of interest (e.g., math and arithmetic are not the same but are often used interchangeably).

For both theoretical and practical reasons it is imperative that researchers continue to look for the causes of learning difficulties. However, to make progress in these endeavors, it is necessary to abandon outdated and questionable approaches and to embrace new methodological and conceptual directions in the quest of more informed inferences.

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Ethical statement

No empirical data were collected in the context of this Opinion Paper. We therefore have no Ethical Statement to make.

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