Review

Developmental Dyscalculia: heterogeneity might not mean different mechanisms

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Research indicates that developmental dyscalculia (DD; a mathematical deficiency) involves a single brain area abnormality – in the intraparietal sulcus. This is surprising because, (i) the behavioural deficits are heterogeneous, (ii) multiple problems are most common in most cases (co-morbidity) and (iii) different aspects of intact number processing are represented in different brain areas. Hence, progress in the study of DD might be limited by conceptual issues. This work looks at biological and cognitive findings within DD and delineates frameworks for studying the neurocognitive basis of DD. We offer three alternative frameworks. These proposed frameworks have the potential of facilitating future discussions, work in the field and have implications for studies of similar disorders like dyslexia and attention-deficit/hyperactivity disorder.

From pure developmental dyscalculia to co-morbidity

Most diagnostic criteria use the term developmental dyscalculia (DD) to describe moderate to extreme difficulties in fluent numerical computations that cannot be attributable to sensory difficulties, low IQ or educational deprivation [1,2]. Epidemiological studies have indicated that DD is as common as reading disorders and affects 3.5%–6.5% of the school-age population [2]. Paradoxically, DD is an unexpectedly neglected area by both clinicians and researchers, despite its importance in health management [3], schooling, everyday life and employment.

Current research points to a single biological marker in DD: an intraparietal sulcus (IPS) abnormality (Figure 1) [4–6]. This is surprising because cognitive deficits seen in DD are heterogeneous [7], and functional brain imaging and brain lesion studies demonstrate that various aspects of intact number processing undoubtedly involve not only the IPS but also additional brain areas [8–10].

Recently, Wilson and Dehaene [11] wrote a review revolving around the idea of DD being because of a core numerical deficit (Boxes 1 and 2) involving a single brain area (similar to the first framework we propose later). They still suggested that other subtypes of DD could exist and would involve brain areas other than the IPS. Our departure point is heterogeneity in etiology and in the manifestation of maths difficulties. Accordingly, we critically evaluate the core problem and offer additional frameworks of thought (Figure 2) [12].

We would like to draw a distinction between DD and mathematical learning disabilities (MLD). Both are disorders in mathematics with no other non-numerical disorder. The term DD is reserved here for a deficit in core numerical abilities (e.g. difficulty in processing quantities) and a relatively specific malfunction at the behavioural level (first framework; Figure 2a). By contrast, MLD are caused by several cognitive deficits such as deficient working memory, visual-spatial processing or attention. Accordingly, DD and MLD would manifest in different behaviours at early stages of development. However, they sometimes manifest in similar behaviours later in life because of the influence of various developmental factors [13] such as schooling.

We offer three alternative frameworks for the origin of DD or MLD and their cognitive deficits. These frameworks can direct theoretical work and help reveal the causal relationship between neurocognitive mechanisms and behaviour. The first framework indicates that a single restricted biological deficit gives rise to a specific developmental disorder (Box 1). However, as is the case with many developmental disorders, multiple problems are most common and pure disorders apply to a minority of cases only. Hence, two other frameworks are suggested. The second framework indicates a variety of cognitive deficits because of a single or multiple instances of biological damage (Box 3). Each cognitive deficit produces a different mathematical deficiency and as a whole, they create the behavioural manifestations of MLD. The third framework indicates that the neurocognitive damage that causes DD could produce other behavioural disorders that are unrelated to DD, namely co-morbidity (e.g. DD + dyslexia) (Figure 2).

It should be noted that very little is known about the molecular biological origins of DD or MLD and there are very few longitudinal studies that examine developmental aspects of these disorders. We emphasize brain dysfunction as a possible origin. However, DD or MLD can involve genetic or environmental factors. Accordingly, the links between the biological, cognitive and behavioural levels are, in most cases, tentative.

In what follows we outline three frameworks, demonstrate their viability and explore important barriers to embracing the particular frameworks.

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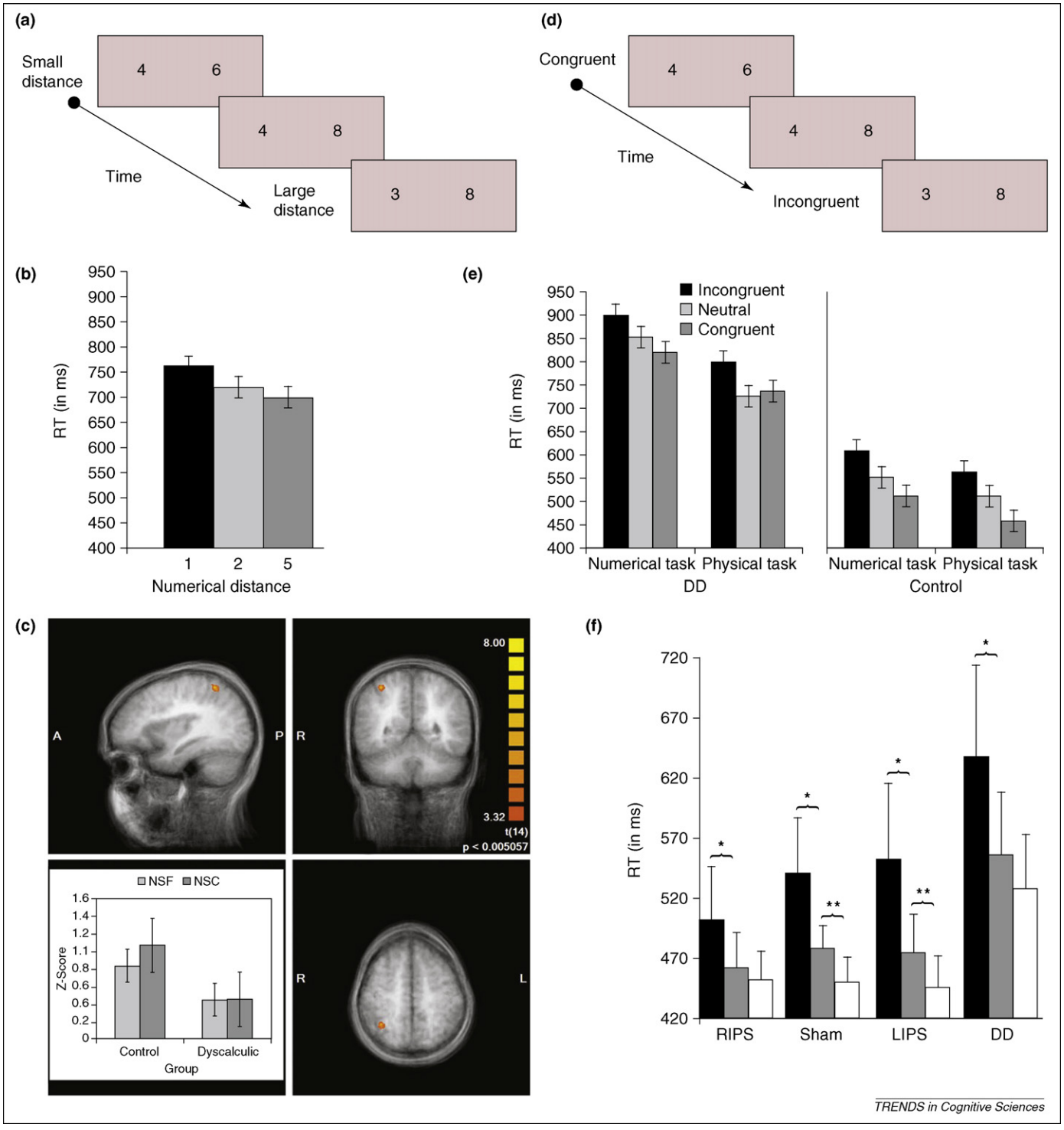

Figure 1. The distance and size congruity effects. **(a)** A typical task that produces the distance effect (DE). When participants are asked to compare two digits, they respond faster when the digits are numerically further apart from one another (e.g. 3–8) than when they are closer (e.g. 4–6). This negative correlation between reaction time and numerical distance is termed DE [56]. DE is considered to reflect access to an analogue representation of numerosity. **(b)** Typical behavioural results, which appear both in DD subjects and controls [17,18]. **(c)** DE involves IPS activation [57]. Right IPS DE for comparisons of non-symbolic stimuli (NSF, non-symbolic far distance; NSC, non-symbolic close distance) is reduced in children suffering from DD [5]. **(d)** Trials in the numerical Stroop task are characterized by independent manipulation of both numerical and physical distances. The two dimensions can be congruent (e.g. 4–6) or incongruent (e.g. 3–8) [58]. Participants process both dimensions automatically; they cannot ignore either dimension and respond faster to the congruent trials than to the incongruent trials [58–60]. **(e)** Typical and atypical behavioural results of the numerical Stroop task. Controls showed both facilitation (response to congruent trials faster than to neutral trials) and interference (response to neutral trials faster than to incongruent trials), whereas DD subjects showed a pattern similar to children at the end of first grade [61], that is, a lack of facilitation and a smaller overall effect [17]. **(f)** TMS to the right IPS (RIPS) but not to left IPS (LIPS) or other brain locations (Sham) produce a DD-like pattern of reaction time (RT). Error bars depict one standard error of the mean. * < 0.05, ** < 0.005. Part (c) reproduced, with permission from Ref. [5]. Part (f) reproduced, with permission, from Ref. [6].

Framework 1: a unique cognitive deficit because of a unique pathophysiology

It has been argued that dyscalculia is the result of specific disabilities in basic numerical processing [14,15], for example, a deficit in quantity processing [16–19] rather than in general cognitive abilities such as working memory [20] (Figure 2a and Box 1). Studies of distance, size congruity effects (Figure 1) and counting (Box 2) support the