



Perceptual learning as a possible new approach for remediation and prevention of developmental dyslexia



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ABSTRACT

Learning to read is extremely difficult for about 10% of children across cultures because they are affected by developmental dyslexia (DD). According to the dominant view, DD is considered an auditory-phonological processing deficit. However, accumulating evidence from developmental and clinical vision science, suggests that the basic cross-modal letter-to-speech sound integration deficit in DD might arise from a mild atypical development of the magnocellular-dorsal pathway which also contains the main fronto-parietal attentional network. Letters have to be precisely selected from irrelevant and cluttering letters by rapid orienting of visual attention before the correct letter-to-speech sound integration applies. Our aim is to review the literature supporting a possible role of perceptual learning (PL) in helping to solve the puzzle called DD. PL is defined as improvement of perceptual skills with practice. Based on the previous literature showing how PL is able to selectively change visual abilities, we here propose to use PL to improve the impaired visual functions characterizing DD and, in particular, the visual deficits that could be developmentally related to an early magnocellular-dorsal pathway and selective attention dysfunction. The crucial visual attention deficits that are causally linked to DD could be, indeed, strongly reduced by training the magnocellular-dorsal pathway with the PL, and learning to read for children with DD would not be anymore such a difficult task. This new remediation approach – not involving any phonological or orthographic training – could be also used to develop new prevention programs for pre-reading children at DD risk.

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1. Developmental dyslexia and the dominant auditory-phonological view

Individuals with developmental dyslexia (DD) have difficulties with accurate or fluent word recognition and spelling despite adequate instruction, intelligence and sensory abilities. DD is defined by difficulties with decoding, whereas by comparison comprehension is more intact (American Psychiatric Association, 1994). Many researchers use the terms DD and “reading disability” interchangeably. Research suggests that DD represents the low end of a normal distribution of word reading ability (e.g., Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992). Some works do not support the external validity of the distinction between age-referenced and IQ-referenced definitions in terms of underlying neuropsychology or appropriate treatments (e.g., Jimenez et al., 2009). Prevalence estimates depend on the definition of DD. A common definition

sets the cutoff for reading achievement 1.5 standard deviations below the mean for age, and identifies 7% of the population as affected by DD. A similar IQ–achievement discrepancy definition identifies a similar proportion. A significant male predominance exists (1.5–3:1), however, the sex difference in referred samples is higher (3–6:1), because boys with DD come to clinical attention more often than girls. DD is co-morbid with attention-deficit hyperactivity disorder, developmental dyscalculia, language impairment and speech-sound disorder (see Peterson & Pennington, 2012 for a recent review). Predisposing candidate genes have been identified (e.g., Marino et al., 2012; see Galaburda, LoTurco, Ramus, Fitch, & Rosen, 2006 for a review), and evidence shows gene by environment interaction (e.g., Mascheretti et al., 2013; Mascheretti et al., 2014; Rosenberg, Pennington, Willcutt, & Olson, 2012).

DD is often associated to an impaired phonological awareness, the auditory analysis of spoken language that relates the letter-to-speech sound integration. The phonological awareness theory remains the most compelling to date in order to explain this disorder, although the auditory word form processing deficit also interact with other cognitive risk factors (see Gabrieli, 2009 for a review). Impaired auditory and phonological processing is

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assumed to characterize the core problem in DD (e.g., [Hornickel & Kraus, 2013](#), see [Gabrieli, 2009](#); [Goswami, 2003, 2011](#) for reviews). A suggested but unproven hypothesis is that this phonological awareness deficit impairs the ability to map speech sounds onto their homologous visual letters, which in turn prevents the attainment of fluent reading levels (see [Vellutino, Fletcher, Snowling, & Scanlon, 2004](#) for a review). Evidence suggests that co-morbidity with DD is mediated by shared causative and neurocognitive risk factors (e.g., [Franceschini, Gori, Ruffino, Pedrolli, & Facoetti, 2012](#); [Franceschini et al., 2013](#)).

2. Developmental dyslexia as a letter-to-speech sound integration deficit

The hypothesis that DD arises specifically from a deficit of phonological awareness is still debated because of the circular relationship between reading ability and phonological skills acquisition. No studies has provides clear evidence that there is a causal link between phonological awareness and reading and spelling acquisition, because no studies have controlled for existing literacy skills in their participants, and the possible effect of these skills on phonological awareness tasks ([Castles & Coltheart, 2004](#)).

[Dehaene and et al. \(2010\)](#) measured brain responses to spoken and written language in adults of variable literacy (10 were illiterate, 22 became literate as adults, and 31 were literate in childhood) by using functional magnetic resonance imaging (fMRI). Literacy enhanced phonological activation to speech sound in the planum temporal and superior temporal cortex (STC). Other studies have demonstrated that learning to read in adulthood can have a significant effect on the structure of brain regions that are important for skilled reading (e.g., [Carreiras et al., 2009](#)). Several changes occurred even when literacy was acquired in adulthood ([Carreiras et al., 2009](#); [Dehaene et al., 2010](#)), emphasizing that both childhood and adulthood reading acquisition can profoundly refine the neurobiological organization of the auditory-phonological reading network (see [Blomert, 2011](#) for a recent review).

Similar functional and structural “impairments” of the phonological network involving the auditory word form area (AWFA) in individuals with DD could be simply explained by the widespread lack of reading experience that distinguishes this neurodevelopmental learning disorder. A child with DD reads in 1 year the same number of words as a good reader reads in 2 days ([Cunningham & Stanovich, 1998](#)).

[Blau et al. \(2009\)](#) used fMRI to investigate the neural processing of letters and speech sounds in unisensory (visual or auditory) and multisensory (audiovisual congruent and audiovisual incongruent) conditions in adults with DD. The data revealed that an under-activate STC for the integration of letters and speech sounds. This reduced audiovisual integration predicts performance on phonological awareness tasks. Another fMRI study by [Blau et al. \(2010\)](#) in children with DD supports the view that letter-to-speech sound integration is an emergent property of learning to read that develops weakly in individuals with DD. DD readers did not suppress STC activity to incongruent letter-speech sound pairs, indicating a less efficient discrimination of those stimuli from existing audiovisual pairs.

Thus, the typical auditory and phonological deficits in DD might be the consequence of the reading failure resulting from a deviant interactive specialization of the neural systems dedicated to the letter-to-speech sound integration (see [Karmiloff-Smith, 1998](#); [Johnson, 2011](#) for reviews).

In fact, learning to read visual words requires a novel integration of two distinct neurocognitive systems: a visual system that allows the recognition of a visual word from a crowd of letter features and a phonological language system (AWFA) that allows the recognition and the production of spoken words from a crowd of phonetic features ([Schlaggar & McCandliss, 2007](#)).

The occipito-temporal sensitivity to print emerges when children learn letter-to-speech sound integration (e.g., [Brem et al., 2010](#)). A tentative model of functional anatomical pathways involved in visual perception of words suggests that letter strings are first processed in the ventral occipital regions (V1–V4) contralateral to the stimuli, building up increasingly abstract visual representations. For stimuli in the left visual field, information is conveyed from the right to the left hemisphere through fiber tracts in the splenium of the corpus callosum ([Fabbro et al. \(2001\)](#)). This right hemisphere mediated pathway and the direct left hemisphere pathway eventually converge in a structure within the left-hemispheric fusiform gyrus (the visual word form area, VWFA), where retinotopic coding is lost (see [McCandliss, Cohen, & Dehaene, 2003](#) for a review). [Dehaene et al. \(2010\)](#) demonstrated that literacy enhanced the left fusiform activation evoked by writing, but also broadly enhanced visual responses in the occipital cortex, extending to area V1, emphasizing that reading acquisition can profoundly refine cortical organization not only of the auditory-phonological network but also of the visual-orthographic network (see [Blomert, 2011](#) for a recent review). Importantly, left and right fronto-parietal network ([Corbetta & Shulman, 2002](#); [Corbetta & Shulman, 2011](#)) strongly modulate both the visual and auditory word pathway by temporal and spatial selective attention ([McCandliss et al., 2003](#)).

Although the neurocognitive causes of DD are still hotly debated (see [Vidyasagar & Pammer, 2010](#) vs. [Goswami, 2011](#)), all researchers agree that the main challenge is the remediation, that is, how to get dyslexic children to read more accurately words in less time. The most common approach has been to devise sophisticated remediation programs that train sub-skills of reading, especially phonological skills and auditory perception. Although rather successful, the improvements in these sub-skills do not automatically transfer in better reading abilities (e.g., [Agnew, Dorn, & Eden, 2004](#); see [Strong, Torgerson, Torgerson, & Hulme, 2011](#) for a recent review). [Zorzi et al. \(2012\)](#) showed that a simple manipulation of letter spacing substantially improved text reading performance on the fly (without any training) in a large, unselected sample of Italian and French DD children. Extra-large letter spacing helps reading, because dyslexics are abnormally affected by visual crowding, a perceptual phenomenon (see [Levi, 2008](#); [Pelli & Tillman, 2008](#); [Whitney & Levi, 2011](#), for reviews) with detrimental effects on letter recognition that is modulated by the spacing between letters (see also [Perea & Gomez, 2012](#)). Spatial attention deficits in children with DD (see [Vidyasagar & Pammer, 2010](#); [Facoetti, 2012](#) for a review) might impair their ability to focus on each successive letter in a visual word while suppressing the influence of the adjacent letters in standard typeface. Because the influence of neighboring letter features is systematically related their proximity to the attended letter, increasing the spacing between letters should reduce the interfering effects of crowding, allowing these children to more readily focus spatial attention on and recognize each successive letter within a word form (e.g., [Facoetti et al., 2010a](#); [Facoetti et al., 2010b](#); [McCandliss, 2012](#)). Accordingly, in people with DD, the speed and comprehension of reading on a small handheld e-reader device – formatted to display few words per line – significantly improved, when compared with traditional presentations on paper ([Schneps et al., 2013a](#); [Schneps et al., 2013b](#)).

3. Visual attention deficits in developmental dyslexia

Visual attention deficit is now considered a cause of DD, independent from the auditory-phonological abilities ([Franceschini et al., 2012](#); [Gabrieli & Norton, 2012](#)). The visual-orthographic system receives stimulus-driven (bottom-up) as well as goal-directed (top-down) attentional influence that modulates all visual processing levels from V1 to VWFA (see [Corbetta & Shulman, 2002](#);

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