How the visual aspects can be crucial in reading acquisition: The intriguing case of crowding and developmental dyslexia

Simone Gori

Developmental and Cognitive Neuroscience Lab, Department of General Psychology, University of Padua, Padua, Italy
Child Psychopathology Unit, Scientific Institute, IRCCS Eugenio Medea, Lecco, Italy

Andrea Facoetti

Developmental and Cognitive Neuroscience Lab, Department of General Psychology, University of Padua, Padua, Italy
Child Psychopathology Unit, Scientific Institute, IRCCS Eugenio Medea, Lecco, Italy

Developmental dyslexia (DD) is the most common neurodevelopmental disorder (about 10% of children across cultures) characterized by severe difficulties in learning to read. According to the dominant view, DD is considered a phonological processing impairment that might be linked to a cross-modal, letter-to–speech sound integration deficit. However, new theories—supported by consistent data—suggest that mild deficits in low-level visual and auditory processing can lead to DD. This evidence supports the probabilistic and multifactorial approach for DD. Among others, an interesting visual deficit that is often associated with DD is excessive visual crowding. Crowding is defined as difficulty in the ability to recognize objects when surrounded by similar items. Crowding, typically observed in peripheral vision, could be modulated by attentional processes. The direct consequence of stronger crowding on reading is the inability to recognize letters when they are surrounded by other letters. This problem directly translates to reading at a slower speed and being more prone to making errors while reading. Our aim is to review the literature supporting the important role of crowding in DD. Moreover, we are interested in proposing new possible studies in order to clarify whether the observed excessive crowding could be a cause rather than an effect of DD. Finally, we also suggest possible remediation and even prevention programs that could be based on reducing the crowding in children with or at risk for DD without involving any phonological or orthographic training.

Developmental dyslexia: The same old dominant view

Developmental dyslexia

Individuals with developmental dyslexia (DD) present difficulties with accurate or fluent word recognition and spelling despite adequate instruction, intelligence, and sensory abilities. DD is characterized by difficulties with decoding while comprehension is more intact (American Psychiatric Association, 1994). DD represents the tail of a normal distribution of word reading ability (e.g., Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992). Prevalence estimates depend on the definition of DD; however, it seems that around 10% of the population can be classified as an individual with DD. A significant male predominance is consistently found with a ratio that ranges between 1.5 to three males per one female. DD presents some important comorbidity with attention deficit hyperactivity disorder, developmental dyscalculia, specific language impairment (SLI), and speech–sound disorder (see Peterson & Pennington, 2012, for a recent review).

Following earlier descriptions of high familial aggregation of the disorder (Hallgren, 1950), substantial heritability typical of a complex neurodevelopmental trait has been reported (Fisher & De Fries, 2002; Plomin & Kovas, 2005). Since the early 1980s, at least nine DD risk loci have been mapped to

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chromosomes 1, 2, 3, 6, 15, 18, and X, and candidate DD genes have been consistently reported (for reviews, see Carrion-Castillo, Franke, & Fisher, 2013; Paracchini, Scerri, & Monaco, 2007; Scerri & Schulte-Körne, 2010). Moreover, recent studies provide evidence that gene-by-environment (e.g., Mascheretti et al., 2013) and gene-by-gene (Harold et al., 2006; Ludwig et al., 2008; Powers et al., 2013) analysis can be exploited for the study of the DD etiology and assist in defining a neurodevelopmental and theoretical molecular-signaling network contributing to DD etiology (Poelmans, Buitelaar, Pauls, & Franke, 2011).

The phonological awareness theory

DD is often correlated with an impaired phonological awareness, which refers to the ability to perceive and manipulate the sounds of spoken words (Goswami & Bryant, 1990; Mattingly, 1972) and involves not only discriminating speech sounds, but also explicitly acting upon them (Castles & Coltheart, 2004). The phonological awareness theory is the most traditional approach adopted to explain DD, and it is still the dominant view. Impaired phonological processing is largely assumed to be the core deficit in DD (e.g., Hornickel & Kraus, 2013; see Gabrieli, 2009; Goswami, 2003, for reviews). A suggested hypothesis is that a phonological awareness deficit impairs the ability to map speech sounds onto homologous visual letters, preventing the attainment of fluent reading (see Vellutino, Fletcher, Snowling, & Scanlon, 2004, for a review). Recent studies suggest that comorbidity with DD is mediated by shared causative and neurocognitive risk factors (e.g., Franceschini, Gori, Ruffino, Pedrolli, & Facoetti, 2012; Franceschini et al., 2013).

The main issue related to this hypothesis is that no published study has been able to clearly call for a causality effect between phonological awareness and a reading disorder. In DD research, the commonly accepted metrics to prove a causal relationship between a neurocognitive function and DD are longitudinal and remediation studies.

Studies have reported that the phonological deficit in children with DD is still present when compared to reading level (RL) controls: younger, typical readers matched to the dyslexics on reading level (e.g., Bruck & Treiman, 1990; Stanovich & Siegel, 1994; Swan & Goswami, 1997). These results suggest a causal link between phonology and DD. However, the use of the RL control can only be a first step in research aimed at delineating the causal factors in reading difficulties. Subsequent longitudinal and remediation studies are necessary to determine for a causal link (Goswami & Bryant, 1989). To date, there are no longitudinal and remediation studies that investigate the phonological skills in DD that have controlled for existing literacy skills and grapheme-to-phoneme mapping in their participants and for the effect of these skills on phonological awareness tasks (Castles & Coltheart, 2004). Moreover, specific phonological awareness training does not automatically transfer to better reading abilities (e.g., Agnew, Dorn, & Eden, 2004; Galuschka, Ise, Krick, & Schulte-Körne, 2014; Strong, Torgerson, Torgerson, & Hulme, 2011), which, therefore, does not suggest a direct causal link between phonological awareness and reading abilities. In other words, the hypothesis that DD arises specifically from a deficit of phonological awareness is controversial because of the circular relationship between reading ability and phonological skills acquisition (Vidyasagar & Pammer, 2010).

Interestingly, Dehaene et al. (2010) measured brain responses to spoken and written language in adults of variable literacy by using 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 significantly affect the structure of the same brain areas that are important for typical readers (e.g., Carreiras et al., 2009). The brain also changes when literacy is acquired in adulthood (Carreiras et al., 2009; Dehaene et al., 2010). These results demonstrated that reading acquisition in both childhood and adulthood can profoundly refine the neurobiological organization of the auditory–phonological reading network (see Blomert, 2011, for a review). Based on the aforementioned studies, an interesting question is "Could the functional and structural impairments characterizing the phonological network in individuals with DD be a consequence of the widespread lack of reading experience that is commonly observed in individuals with DD?" It is known that a child with DD reads in 1 year the same number of words read by a typical reader in 2 days (Cunningham & Stanovich, 1998). Thus, findings of relatively less gray matter volume (GMV) in DD may represent the consequence of a limited reading experience. Consistent with previous reports, Krafnick, Flowers, Luetje, Napoliello, and Eden (2014) reported that individuals with DD showed less GMV in multiple left and right hemisphere regions, including the left superior temporal sulcus, when compared with age-matched controls. However, not all of these differences emerged when dyslexics were compared with controls matched on reading abilities with only right precentral gyrus GMV remaining significant in the second anaysis (Krafnick et al., 2014). These results indicate that the GMV differences in DD reported before are in large part the outcome of experience (e.g., disordered reading experience) compared with controls with only a fraction of the differences being driven by DD per se. Consistently,

Clark et al. (2014) found in their longitudinal study, based on structural MRI, that abnormalities in the reading network are the consequence of having different reading experiences whereas the neuroanatomical precursors of DD are predominantly in primary sensory cortices.

Interestingly, it could be that the phonological awareness deficit is a cause of SLI, which presents high comorbidity with DD (Brizzolara et al., 2006; Brizzolara et al., 2011; Chilosi et al., 2011). However, the phonological awareness deficit may not be the cause of DD itself. Considering that most of the study of DD did not exclude children with a history of SLI, the high comorbidity with DD could potentially conciliate the presence of supporting results about the causal role of phonological awareness in DD (e.g., RL design) and the absence of well-controlled studies employing powerful causal methods (longitudinal and remediation design). However, a direct consequence of controlling for history of SLI would be excluding a large number of children with DD, raising the inevitable question: Does this procedure tell us something reliable about the causes of DD? It seems pretty clear from the literature that DD is, indeed, a complex disorder characterized by a large number of deficits that combine so that the final outcome passes the threshold of diagnosis (e.g., Menghini et al., 2010). Consequently, the research of the "pure DD deficit" seems to be unsuccessful in explaining this complex disorder. Moreover, it was demonstrated that children with SLI also reported visuo-temporal attentional deficits (e.g., Dispaldro et al., 2013) showing that SLI is not exclusively a language disorder.

Traditional remediation approach for DD

Until now, the most common approach in DD remediation has been to devise sophisticated programs that train subskills of reading, especially phonological awareness. The typical tasks in phonological awareness training are phoneme deletion, phoneme counting, phoneme blending, phoneme reversal, syllable segmentation, rhyme oddity, and rhyme judgment (Castles & Coltheart, 2004). Results showed that the improvements in phonological awareness unfortunately do not automatically transfer to better reading abilities (e.g., Agnew et al., 2004; Galuschka et al., 2014; Strong et al., 2011).

Blau, van Atteveldt, Ekkebus, Goebel, and Blomert (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 the STC was underactivated for the integration of letters and speech sounds. This reduced audiovisual integration was able to predict the phonological awareness task performance. Another fMRI study by Blau et al. (2010) showed that letter-to-speech sound integration is an emergent property of learning to read that does not properly develop in children with DD. Thus, the phonological deficits in DD might be a consequence of the reading failure, resulting from a deviant interactive specialization of the neural systems dedicated to the letter-to-speech sound integration (see Johnson, 2011; Karmiloff-Smith, 1998, for reviews). Learning to read visual words requires, indeed, a novel integration of two neurocognitive systems: a visual system that allows the recognition of a visual word in a clutter of letter features and a phonological language system that is able to recognize the spoken words from a crowd of phonetic features (Schlaggar & McCandliss, 2007). Dehaene et al. (2010) showed that literacy enhanced the left fusiform activation together with enhancing the visual responses in the occipital cortex, including V1. These results demonstrated that reading acquisition can profoundly refine cortical organization in both the auditory–phonological and the visual–orthographic network (see Blomert, 2011, for a review). The remediation approach based on explicit, systematic instruction on letter-to-speech integration, also called "phonics training," appears to be the most efficient treatment in English-speaking individuals with DD (McArthur et al., 2012). By comparing the efficiency of different types of training for DD remediation, a recent meta-analysis revealed that phonics instruction is not only the most frequently investigated treatment approach, but also the only approach whose efficacy on reading and spelling performance in children and adolescents with DD is statistically confirmed (Galuschka et al., 2014).

In sum, the same old dominant view that attempted to explain DD with a single cause represented by the phonological awareness deficit remains controversial no matter how same, old, and dominant it appears at first glance; on the other hand, moving the focus more onto the letter-to-speech sound integration deficit seems to be revitalizing this traditional approach to DD.

DD: Some new fresh air

In parallel with studies supporting the phonological hypothesis in DD, new perspectives, not necessarily opposite to the dominant view, introduced fresh air into the constant fight against DD and its consequent costs. The general idea is that a lower-level deficit can be linked to DD together with the deficit in phonoloy (Goswami, Power, Lallier, & Facoetti, 2014).

Rapid auditory processing theory

One mild deficit that is often associated with DD seems to be at the level of auditory processing. More specifically, it seems that rapid auditory processing is defective in individuals with DD (Tallal, 1980, 2004). The inability to correctly process two sounds in a fast sequence can directly translate into future reading problems (e.g., Benasich & Tallal, 2002; Benasich, Thomas, Choudhury, & Leppanen, 2002). To some extent, these findings of temporal processing difficulties in the auditory system could be considered a possible neuronal basis for the phonological theory (e.g., Choudhury, Lappenen, Leevers, & Benasich, 2007; Benasich, Choudhury, Reale-Bonilla, & Roesler, 2014). Some pretty popular auditory perception trainings were developed in order to try to rehabilitate the reading difficulties in DD. These auditory perception trainings are language-based programs containing speech that is acoustically modified, similar to those used by speech and language therapists, in order to "cross-train" many different skills at the same time (Tallal, 2000). Although rather successful, the improvements in auditory perception were similar to what was found with phonological awareness training and do not automatically transfer into better reading abilities (e.g., Agnew et al., 2004; Galuschka et al., 2014; Strong et al., 2011).

Temporal sampling framework

More recently, results showing a rapid auditory processing deficit were integrated with findings on neural oscillatory mechanisms related to the temporal sampling of speech in an innovative approach to DD termed the "temporal sampling framework" (TSF) by Goswami (2011). In sum, Goswami (2011) suggests that deficits in syllabic perception at relatively low frequencies in the range of delta/theta (4–10 Hz) is the critical basis for the reading disability in DD. This hypothesis is supported by findings that show the possible role of neuronal oscillations in speech perception (Luo & Poeppel, 2007; Poeppel, Idsardi, & Van Wassenhove, 2008). Even if this approach was presented as a possible neurophysiological substrate of the phonological deficit of DD, the TSF can also be applied to the various stages of processing within the visual system, way before the phonological processing stage as suggested by Vidyasagar (2013) and successfully tested by Gori, Cecchini, Bigoni, Molteni, and Facoetti (2014b). This leads to consideration of TSF with an even more broad approach that can also integrate several low-level deficits known in DD (Gori et al., 2014b; Pammer, 2014; Vidyasagar, 2013).

The magnocellular–dorsal theory

Another dominant, albeit controversial (e.g., Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Olulade, Napoliello, & Eden, 2013; Sperling, Lu, Manis, & Seidenberg, 2005) theory is known as the magnocellular–dorsal (M–D) theory of DD (Livingstone, Rosen, Drislane, & Galaburda, 1991; Stein & Walsh, 1997), which stems from the observation that a high percentage of reading disabled children are impaired in the specific visual M–D pathway (see Boden & Giaschi, 2007; Facoetti, 2012; Gori & Facoetti, 2014; Stein & Walsh, 1997; Vidyasagar & Pammer, 2010, for reviews). The M–D pathway originates in the ganglion cells of the retina, passes through the M-layer of the lateral geniculate nucleus (LGN), and finally reaches the occipital and parietal cortices (Maunsell & Newsome, 1987). The M–D stream is considered blind to colors and responds optimally to contrast differences, low spatial frequencies, high temporal frequencies, and both real and illusory motion (e.g., Gori, Giora, & Stubbs, 2010; Gori, Giora, Yazdanbakhsh, & Mingolla, 2011; Gori, Hamburger, & Spillmann, 2006; Gori & Yazdanbakhsh, 2008; Livingstone & Hubel, 1987; Morrone et al., 2000; Ruzzoli et al., 2011; Yazdanbakhsh & Gori, 2011), which is also, surprisingly, perceived by animals without a cortex, such as fish (Gori, Agrillo, Dadda, & Bisazza, 2014a). Individuals with DD are less sensitive than typically reading controls to luminance patterns and motion displays with high temporal and low spatial frequencies (e.g., Eden et al., 1996), visual features that are known to be associated with the M–D pathway. However, they perform similarly to the controls on tasks preferentially associated with the parvocellular–ventral pathway (Gori et al., 2014b), such as those involving color and form (Merigan & Maunsell, 1993). The M–D theory can also be embedded in its multisensory (i.e., visual and auditory) version, called the temporal processing hypothesis, which suggests that children with DD have specific deficits in processing rapidly presented sensory stimuli in either the visual or auditory modalities (see Farmer & Klein, 1995; Hari & Renvall, 2001, for reviews). Importantly, the M–D temporal hypothesis explicitly claims that phonological decoding deficits in individuals with DD could arise from impairments in dynamic sensory processing of visual and auditory stimuli (e.g., Facoetti et al., 2010b; Gori et al., 2014b; Ruffino et al., 2010, 2014). It has been reported that up to 75% of dyslexic individuals show visual temporal processing deficits (Lovegrove, Martin, & Slaghuis, 1986). Moreover, a postmortem study showed that in the brain of individuals with dyslexia the M neurons of the LGN were significantly smaller than those found in normal readers' brains, and the P neurons did not differ between the two groups (Livingstone et al., 1991). This

study recently received strong support from the first in vivo study (Giraldo-Chica, Hegarty, & Schneider, in press) showing smaller LGN volume in a larger sample of individuals with DD compared to controls. Recently, Gori et al. (2014b) and Gori et al. (in press) demonstrated, for the first time, that children with DD showed a lower performance in both a task that taps the M (i.e., spatial frequency doubling illusion; Kelly, 1966) and one that taps the D (i.e., rotating tilted lines illusion, Gori & Hamburger, 2006; Gori & Yazdanbakhsh, 2008; Yazdanbakhsh & Gori, 2008, and the accordion grating, Gori et al., 2011; Gori, Giora, Yazdanbakhsh, & Mingolla, 2013; Yazdanbakhsh & Gori, 2011) portion of the M-D pathway, not only in comparison with an age-matched control group, but also with a RL control group. Some longitudinal studies provided strong evidence in the direction of a causal link between a prereading M–D deficit and future reading acquisition (e.g., Boets, Vandermosten, Cornelissen, Wouters, & Ghesquière, 2011; Boets, Wouters, van Wieringen, De Smedt, & Ghesquière, 2008; Kevan & Pammer, 2008; 2009). These studies supported the hypothesis that the M–D deficit is not caused by lack of reading abilities (effect of DD) but should be considered a core deficit of DD. Gori et al. (in press) also showed the first reported association between a genetic variance (the DCDC2-Intron deletion) and an M–D deficit in both individuals with DD and typical readers. The DCDC2-Intron deletion is a proved DD genetic risk factor (e.g., Marino et al., 2011; Marino et al., 2012; Marino et al., 2014; Mascheretti et al., 2013; Mascheretti et al., in press; Meng et al., 2005; Riva, Marino, Giorda, Molteni, & Nobile, in press). According to recent studies, the M–D pathway also seems to be specifically involved in audiovisual detection enhancements (e.g., Harrar et al., 2014; Pérez-Bellido, Soto-Faraco, & Lopez-Moliner, 2013), suggesting an additional causal link between the M–D deficit and the basic cross-modal integration dysfunction in individuals with DD. Interestingly, the M–D deficit in individuals with DD was found also in logographic languages, such as Chinese (e.g., Zhao, Qian, Bi, & Coltheart, 2014). Gori and Facoetti (2014) recently stressed the importance of showing the positive effects of a rehabilitation approach based on an M–D stream deficit. If an M–D stream deficit is really a cause of DD, it is expected that specific M–D stream training would be able to improve not only M–D functioning, but also reading abilities in individuals with DD.

In summary, some studies failed to confirm differences in high temporal, low spatial frequency stimuli perception between individuals with DD and controls (e.g., Johannes, Kussmaul, Münte, & Mangun, 1996; Schulte-Körne & Bruder, 2010; Victor, Conte, Burton, & Nass, 1993; Williams, Stuart, Castles, & McAnally, 2003, for a review). Nevertheless, sometimes questionable choices in the stimulus parameters (e.g., relative low temporal frequencies) were adopted (Stein, 2012). More importantly, around 90% of studies that specifically looked for subcortical visual M-cell deficits in individuals with DD confirmed mild M impairments in tests employing low contrast, high temporal, and low spatial frequency as recently reported by Stein (2012) in his very comprehensive literature review.

The attentional deficit theory

Interestingly, although Wright, Conlon, and Dyck (2012) suggested that magnocellular sensitivity and visual spatial attention deficits might be independent of one another, deficits in the M-pathway could influence higher visual processing stages by the D-stream. Therefore, reading difficulties could come out due to an impaired attentional orienting system (Boden & Giaschi, 2007; Hari & Renvall, 2001; Vidyasagar & Pammer, 2010), which is anatomically contained in the D-stream. Accordingly, neuroimaging studies of both typical and atypical reading development have consistently implicated regions that are known to subserve the visual attention orienting system (see Corbetta & Shulman, 2002, 2011, for reviews). Based on that, Vidyasagar (1999), probably for the first time, proposed that an attentional deficit could be the basis of DD.

Several studies employing phonological decoding tasks have shown deficient task-related activation in areas surrounding the bilateral frontoparietal attentional system in dyslexics (see Eden & Zeffiro, 1998, for a review). Although the left frontoparietal system has been linked to auditory word form processing (Pugh et al., 2000), the right frontoparietal system is a crucial component of the network subserving automatic attentional shifting (Corbetta & Shulman, 2002, 2011). Thus, developmental changes in activation of the right frontoparietal system have been linked to reading acquisition in typically developing children (Turkeltaub, Gareau, Flowers, Zeffiro, & Eden, 2003), and some studies have observed a right frontoparietal system dysfunction in dyslexics (e.g., Hoeft et al., 2006). A recent study using all-brain and data-driven analysis has shown divergent connectivity within the visual pathway and between visual association areas and prefrontal attention areas in adults and children with DD (Finn et al., 2014). Moreover, adults with DD have shown that high-frequency, repetitive transcranial magnetic stimulation improved nonword reading accuracy over the left and right inferior parietal lobules (Costanzo, Menghini, Caltagirone, Oliveri, & Vicari, 2013). Interestingly, children with autism spectrum disorders (e.g., Ronconi, Basso, Gori, & Facoetti, 2014; Ronconi et al., 2013a; Ronconi et al., 2012; Ronconi,

Gori, Ruffino, Molteni, & Facoetti, 2013b) and with SLI (Dispaldro et al., 2013) also presented attentional focusing disorders showing how attentional dysfunction can be at the basis of different developmental outcomes.

Some aforementioned data leads us to propose the M–D stream deficit as a possible neurobiological substrate of the spatial and temporal attentional deficit in DD, which is one of the current leading theories in explaining DD. 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 bottom-up as well as goal-top-down attentional influence that modulates all visual processing levels from V1 to the visual word form area (see Corbetta & Shulman, 2002; 2011; Facoetti, 2012; Laycock & Crewther, 2008; McCandliss, Cohen, & Dehaene, 2003; Vidyasagar & Pammer, 2010, for reviews). Attentional shifting improves perception in several visual tasks, such as contrast sensitivity, texture segmentation, and visual search, by intensifying the signal and enhancing spatial resolution as well as reducing the noise effect outside the focus of attention (e.g., Boyer & Ro, 2007; Carrasco, Williams, & Yeshurun, 2002; Dosher & Lu, 2000; Facoetti, 2001; Facoetti & Molteni, 2000; Montani, Facoetti, & Zorzi, 2014; Yeshurun & Rashal, 2010; see Bellocchi, Muneaux, Bastien-Toniazzo, & Ducrot, 2013; Reynolds & Chelazzi, 2004; Reynolds & Heeger, 2009, for reviews). Attentional shifting can be considered the result of the engagement mechanism onto the relevant object (e.g., the letter or grapheme that has to be mapped to its corresponding speech sound) and the subsequent disengagement mechanism from the previous object to the next one. Visual attentional shifting deficit has been systematically found in DD (see Facoetti, 2004, 2012; Hari & Renvall, 2001; Valdois, Bosse, & Tainturier, 2004; Vidyasagar & Pammer, 2010, for reviews) and more specifically in dyslexics with poor phonological decoding skills (e.g., Buchholz & McKone, 2004; Cestnick & Coltheart, 1999; Facoetti et al., 2010b; Facoetti et al., 2006; Jones, Branigan, & Kelly, 2008; Kinsey, Rose, Hansen, Richardson, & Stein, 2004; Roach & Hogben, 2007; Ruffino, Gori, Boccardi, Molteni, & Facoetti, 2014; Ruffino et al., 2010). Moreover, some evidence points toward a difficulty in excluding distracting stimuli. Sperling et al. (2005, 2006) found that the performance of adults in a visual motion detection task only correlated with reading ability in conditions with low signal-to-noise ratios. Using a visual search paradigm, Roach and Hogben (2004, 2007) measured psychophysical thresholds of individuals with DD and controls to detect a tilted target stimulus among vertical distractors showing an ineffective noise exclusion. Consistent with the multisensory "sluggish attentional shifting" (SAS) hypothesis (Hari & Renvall, 2001) and the "perceptual noise exclusion deficit" (Sperling et al., 2005), children and adults with DD are specifically impaired from rapidly engaging their attention, showing abnormal temporal masking (e.g., Di Lollo, Hanson, & McIntyre, 1983; Montgomery, Morris, Sevcik, & Clarkson, 2005; Ruffino et al., 2014; Ruffino et al., 2010). Evidence of SAS in the visual modality for children and adults with DD is provided by attentional blink results (e.g., Buchholz & Aimola-Davies, 2007; Facoetti, Ruffino, Peru, Paganoni, & Chelazzi, 2008; Hari, Valta, & Uutela, 1999; Lallier, Donnadieu, & Valdois, 2010; Visser, Boden, & Giaschi, 2004), temporal order judgment (Jaśkowski & Rusiak, 2008; Liddle, Jackson, Rorden, & Jackson, 2009), rapid multielement presentation (Bosse, Tainturier, & Valdois, 2007; Hawelka, Huber, & Wimmer, 2005), and spatial cueing tasks (Brannan & Williams, 1987; Facoetti, Lorusso, Cattaneo, Galli, & Molteni, 2005; Facoetti et al., 2010b; Facoetti et al., 2006; Roach & Hogben, 2007; Ruffino et al., 2014) that involve efficient spatial and temporal attentional shifting to rapidly displayed stimuli. Moreover, contrarily to what was recently affirmed by Goswami (2015), longitudinal studies and studies with pre-reading children at risk for DD have shown that visual attention shifting is one of the most important predictors of early reading abilities (e.g., Facoetti, Corradi, Ruffino, Gori, & Zorzi, 2010a; Ferretti, Mazzotti, & Brizzolara, 2008; Franceschini et al., 2012; Plaza & Cohen, 2007). In addition, the relationship between attentional skills in preschooler children and their future reading abilities resulted fully independent from phonological processing (Franceschini et al., 2012). These results clearly rule out the possible explanation suggested by Goswami (2015) about a supposed major role of the reading experience in explaining the attentional deficit found in children with DD.

It is proposed that the core neural deficit underlying DD is the fundamental multimodal attentional mechanism (which affects both visual and auditory perception) that mediates efficient orthographic–phonological binding (Gori & Facoetti, 2014; Hari & Renvall, 2001). Some intervention studies have clearly shown that both auditory and visual shifting of attention can be improved by training in children with both DD and/or SLI (e.g., Facoetti, Lorusso, Paganoni, Umiltà, & Mascetti, 2003; Geiger, Lettvin, & Fahle, 1994; Stevens, Fanning, Coch, Sanders, & Neville, 2008). In particular, these studies consistently demonstrated that the inhibitory aspects of attention—that are crucial for perceptual noise exclusion—can be remediated with appropriate rehabilitation programs (Facoetti et al., 2003; Geiger et al., 1994). In fact, even the so-called phonologically based treatment programs that are

typically used to rehabilitate DD (e.g., Olulade et al., 2013) have to make use of fundamental auditory attentional mechanisms. Recently, Franceschini et al. (2013) showed that playing action video games (AVG) for only 12 hr improved children's reading abilities, more so than 1 year of spontaneous reading development and more than or equal to highly demanding traditional reading treatments. These results were the outcome of an attentional training based on the AVGs that transferred directly to better reading abilities. After the AVG training, attentional and reading improvements were highly correlated even after controlling for phonological training–induced changes, showing how unfounded the phonological interpretation of these results recently suggested by Goswami (2015) was. Consequently, attentional training was found to be a crucial method to remediate DD independently from auditory–phonological approaches.

Finally, before beginning the next chapter of this review, it is important to remind the reader that there are several other visual aspects that were found to be relevant in DD that are out of the scope of this review but that are well summarized in the recent book edited by Stein and Kapoula (2012).

In summary, it seems clear that DD is a very complex disorder that is well described by a multifactorial and probabilistic model (Menghini et al., 2010). Inside this model, the visual aspects play a crucial role, and based on the scientific evidence, it is now time to seriously evaluate them even before reading acquisition. This approach could allow early identification and even prevention of DD based on prereading trainings. Among the visual aspects that are often associated with DD, one of the most prominent has not yet been mentioned: the crowding effect, which will be discussed in the following section.

DD: The intriguing case of crowding

Several studies have suggested that individuals with DD suffer from crowding more than similarly aged control readers (e.g., Bouma & Legein, 1977; Callens, Whitney, Tops, & Brysbaert, 2013; Geiger & Lettvin, 1987; Martelli, Di Filippo, Spinelli, & Zoccolotti, 2009; Moll & Jones, 2013; Montani, Facoetti, & Zorzi, in press; Moores, Cassim, & Talcott, 2011; Perea et al., 2012; Pernet, Valdois, Celsis, & Démonet, 2006; Spinelli, De Luca, Judica, & Zoccolotti, 2002; Zorzi et al., 2012). However, some outstanding questions about the link between crowding and DD remain unanswered:

- Is the observed excessive crowding in individuals with DD a cause or a simple effect of DD?
- Can training that aims to ameliorate the crowding resistance directly lead to better reading abilities in individuals with DD?
- Can training that aims to ameliorate the crowding resistance during the prereading stage reduce future DD incidence?

Visual crowding

Visual crowding occurs when an object becomes more difficult to identify when it is surrounded by other objects than when it is presented in isolation (see Pelli, 2008; Pelli & Tillman, 2008; Whitney & Levi, 2011, for reviews). Crowding is a universal perceptual phenomenon, not restricted to vision or reading. It can occur with simple objects, such as orientation gratings, and also with complex objects, such as letters and faces (Pelli & Tillman, 2008; Whitney & Levi, 2011). Recognition is impaired when objects are closer than a critical spacing (e.g., Yu, Cheung, Legge, & Chung, 2007), which is the distance between objects at which target recognition is restored (Martelli et al., 2009). Critical spacing is proportional to eccentricity. Thus, object identification is increasingly limited as objects are displayed at larger eccentricities (Bouma, 1970). However, crowding is independent of print size (Pelli et al., 2007). Crowding is also a different phenomenon from ordinary masking, with which the target disappears (Pelli, Palomares, & Majaj, 2004). On the contrary, the target remains visible in the typical crowding display, but it is ambiguous, mushed with the flankers. Moreover, the crowding effect extends over a larger region in comparison to what is observed in an ordinary masking display (Pelli et al., 2004). Finally, crowding is also independent from the surrounding suppression, in which a mask has the orientation preferred by the neuron but appears outside its receptive field as suggested by Levi, Hariharan, and Klein (2002) and demonstrated by Petrov, Popple, and McKee (2007) although both phenomena share several common properties (Petrov, Carandini, & McKee, 2005; Petrov & McKee; 2006; Petrov et al., 2007).

Possible basis of visual crowding

After many years of scientific investigation, the neural mechanisms of crowding remain debated, and several theories have been proposed to explain this phenomenon. Some theories stressed the role of the early visual cortical interaction in accounting crowding. Based on these theories, crowding occurs when the target and flanker overlap within the same neural unit or they are represented by different populations of neurons with long-range horizontal connections (Flom,

Heath, & Takahashi, 1963; Levi, 2008; Levi, Klein, & Aitsebaomo, 1985; Pelli, 2008). On the other hand, other theories argue that crowding could be the result of a limit in the resolution of spatial attention (He, Cavanagh, & Intriligator, 1996; Intriligator & Cavanagh, 2001; Strasburger, 2005; Yeshurun & Rashal, 2010). Some studies showed no or small effects of attentional cueing on crowding (Nazir, 1992; Scolari, Kohnen, Barton, & Awh, 2007; Wilkinson, Wilson, & Ellemberg, 1997); however, these studies did not control for the interactions between crowding and masking (see Whitney & Levi, 2011, for a review). After controlling that the cue did not mask the target, the attentional modulation on crowding seemed to be present (Yeshurun & Rashal, 2010). Moreover, although crowding is usually thought of as a spatial phenomenon, it also occurs in the time domain (see Whitney & Levi, 2011, for a review). It remains unclear if there is an independent mechanism specifically devoted to processing temporal crowding, but the effects of spatial crowding are correlated with those of temporal crowding (Bonneh, Sagi, & Polat, 2007), supporting the involvement of spatiotemporal and attentional mechanisms in crowding (e.g., Chakravarthi & Cavanagh, 2007; Yeshurun & Rashal, 2010). However, Freeman and Pelli (2007) proposed a bottom-up interpretation that could also fit, for example, the results by Intriligator and Cavanagh (2001) in a parsimonious model involving only low-level mechanisms. Dakin, Bex, Cass, & Watt (2009) argued that crowding does not specifically reflect an attention phenomena. On the other hand, Petrov and Meleshkevich (2011a, 2011b), based on their study on anisotropies and asymmetries in crowding, suggest that spatial attention is intimately involved in the mechanism of crowding.

Although several psychophysical studies were conducted, only a few neurophysiological studies have attempted to investigate the neural mechanisms of crowding (e.g., Anderson, Dakin, Schwarzkopf, Rees, & Greenwood, 2012; Bi, Cai, Zhou, & Fang, 2009; Chen et al., 2014; Fang & He, 2008; Freeman, Donner, & Heeger, 2011; Millin, Arman, Chung, & Tjan, 2014). Some fMRI studies (Anderson et al., 2012; Freeman et al., 2011; Kwon, Bao, Millin, & Tjan, 2014; Millin et al., 2014) showed that crowding attenuated the activation in the early visual cortex (e.g., V1). However, it is unclear whether the attenuation originates in V1 or it is a result of top-down feedback from higher cortical areas due to the low temporal resolution of fMRI. Recently, Chen et al. (2014) performed event-related potential and fMRI experiments in order to measure the cortical interaction between the target and flankers in humans. Their results showed that the crowding magnitude was strongly associated with an early suppressive cortical interaction originating in V1. As reported by these authors, spatial attention plays a critical role in the manifestation of this suppression showing that attention-dependent V1 suppression contributes to crowding at a very early stage of visual processing. Another recent study (Chicherov, Plomp, & Herzog, 2014) investigated the neural substrate of crowding using high-density EEG. These authors showed that crowding might reflect processes in high-level visual areas, such as the lateral occipital cortex. Their results suggest that crowding occurs when elements are grouped into wholes (e.g., Gori & Spillmann, 2010) and cannot be fully attributed to lower cortical areas such as V1.

Thus, the contribution of attention and of more general top-down feedback on the crowding effect remains, to date, debated. The dorsal stream role in modulating the ventral stream activation related to crowding is yet to be proved, and future studies need to be done in order to shed light on this topic.

Visual crowding and reading

Specifically, when objects are letters, which is the main focus of this review, the situation did not seem to change at all. The distance between letters (measured center-to-center) is the critical spacing (Martelli et al., 2009), which scales with eccentricity (Bouma, 1970). In the periphery of the visual field, more letters within words printed at fixed spacing will be unrecognizable because of crowding (Bouma, 1973). Consequently, the longer a word, the stronger the effect of crowding (Martelli et al., 2009). Crowding mostly affects peripheral vision in normal adult readers (Pelli et al., 2007), but it also affects central vision in school-aged children (Jeon, Hamid, Maurer, & Lewis, 2010). It is well known that letter identification is a fundamental step in visual word recognition and reading aloud (e.g., McClelland & Rumelhart, 1981; Pelli, Farell, & Moore, 2003; Perry, Ziegler, & Zorzi, 2007). Parsing of a letter string into its constituent graphemes is a key component of phonological decoding (Perry et al., 2007), which, in turn, is fundamental for reading acquisition (Goswami, 2003; Ziegler & Goswami, 2005).

There is growing evidence that children with DD are more influenced by crowding than age-matched controls even under optimal viewing conditions. In the pioneering study by Bouma and Legein (1977), children with DD and typical readers were investigated. Recognition scores of isolated or embedded letters were compared in both foveal and parafoveal vision. No difference was found between the two groups in isolated letters whereas the children with DD were impaired in the embedded letters condition. Interestingly, individual scores of embedded letters were correlated with reading skills. The so-called Bouma's

law of crowding predicts an uncrowded central window through which we can read and a crowded periphery through which we cannot (Bouma, 1970). Crowding and eccentricity determine reading rate. Typical readers are limited by letter spacing (crowding) and not font size (acuity) during ordinary text reading under adequate illumination (Pelli et al., 2007).

Geiger and Lettvin (1987) compared individuals with DD and typical readers in briefly presented letters and letter string identification across a large portion of the visual field. Although the individuals with DD showed a markedly wider area of correct identification in the peripheral field, they had a reduced accuracy for letter identification in and near the foveal field in comparison with typical readers. These results were interpreted as abnormal lateral masking in the near foveal field for individuals with DD. According to these authors, letters are self-masking: The different distinct parts of a letter mask each other. These findings suggest that individuals with DD present a peculiar spatial distribution of lateral masking across central and peripheral vision (see also Goolkasian & King, 1990; Lorusso et al., 2004; but Klein, Berry, Briand, D'Entremont, & Farmer, 1990). However, other studies that specifically investigated the crowding effect across eccentricity in individuals with DD and typical readers (e.g., the aforementioned Bouma & Legein, 1977, and the described below Martelli et al., 2009, studies) found that the disadvantage in letter identification for individuals with DD is present also in the periphery.

Spinelli et al. (2002) studied the effect of crowding on word identification in typical readers and in individuals with DD. These authors presented words either alone or embedded inside other words. Vocal reaction times of individuals with DD were slower and more sensitive to the presence of the surrounding stimuli than controls. Similar results were obtained by using the same task for isolated versus crowded strings of symbols. Interestingly, a moderate increase in interletter spacing produced faster vocal reaction times in individuals with DD whereas no effect was present in the controls. More recently, Martelli et al. (2009) tested the hypothesis that crowding effects could be responsible for the slow reading rate characterizing DD. They measured contrast thresholds for identifying letters and words as a function of stimulus duration. Thresholds were higher in individuals with DD in comparison with controls for words at a limited time exposure, but not for letters, confirming the original study by Bouma and Legein (1977). It is important to note that, with long exposure time, the thresholds were similar in the two groups, suggesting possible temporal effects of spatial attention (Facoetti et al., 2010b). Pernet et al. (2006) investigated the influence of feature analysis, memory access, and stimulus type (Latin letters, Korean letters, and geometrical figures) on crowding in typical and dyslexic readers. Participants with DD showed poorer performance than controls in memory access and a reduced identification with the crowding. Poorer performance in readers with DD may reflect impaired parafoveal/low-level processing during feature integration that may have worsened in the condition with flankers due to spatial attentional disorder.

Martelli et al. (2009) measured the spacing between a target letter and two flankers at a fixed level of performance as a function of eccentricity and size. With eccentricity, the critical spacing was significantly larger in the DD group in comparison with controls. Critical spacing was independent of stimulus size in both groups. The authors concluded that word analysis in individuals with DD is slowed because of greater crowding effects, which limit letter identification in multiletter arrays across the visual field. Crowding accounts for a large variance of children with DD slow reading speed. However, after controlling for crowding, the reading rate of children with DD remains slower than what was observed in typical readers. The persistent slow reading rate observed in DD can be simply explained in terms of a reduced reading experience as a consequence to DD itself. Crowding might not only slow down reading speed (Martelli et al., 2009; Pelli et al., 2007; Yu et al., 2007), but also might induce reading errors because crowding is accompanied by a percept that is thought to reflect pooling of features from the target and the flankers (Whitney & Levi, 2011).

In sum, several behavioral and psychophysical studies showed that individuals with DD are abnormally affected by crowding and that crowding is modulated by the spacing between objects. It could also be argued that the different spatial distribution in crowding observed in individuals with DD can be explained by their well-documented attentional deficit that could modulate the crowding effect (e.g., Petrov & Meleshkevich, 2011a, 2011b). However, the effect of attentional cuing on crowding in the absence of position uncertainty has not been shown yet. Consequently, it is unclear if the excessive crowding found in DD can be fully attributed to the attentional deficit in DD. Further studies should be conducted in order to isolate the crowding effect in DD by controlling for attention.

All these findings lead to the prediction that extra-large interletter spacing in words should reduce crowding and immediately ameliorate reading performance in individuals with DD. However, the previous studies did not control with RL participants. As mentioned above, the comparison with RL controls is the first step to call for a causal link between a neurocognitive aspect and DD. To our knowledge, the only published study including RL controls is the one by Zorzi et al. (2012). These authors 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 children with DD. In contrast, the RL controls did not show any improvement with the extra-large spacing. This result is congruent with the previous study by Spinelli et al. (2002) in which a moderate increase of the spacing between letters improved reading only in individuals with DD. Perea et al. (2012) also demonstrated that slight increases in interletter spacing improved the readability of texts aimed at children, especially those with DD. These results seem very relevant because extra-large letter spacing might help to break the vicious circle by making the reading material more easily accessible for children with DD. Recently, Schneps, Thomson, Chen, Sonnert, & Pomplun (2013a) and Schneps et al. (2013b) showed that reducing crowding by presenting fewer words in a line on a small screen improved reading abilities. Some authors interpreted this reading improvement as a consequence of the reduced amount of attention necessary to perform the task (Schneps et al., 2013a; Schneps et al., 2013b; Zorzi et al., 2012). However, this interpretation is challenged by a study on typical readers (Lee, Kwon, Legge, & Gefroh, 2010) in which the improvement in reading abilities in the periphery found by Chung (2004) after a training was correlated to reduced crowding but not to improvement in spatial attention in peripheral vision (Lee et al., 2010).

Nevertheless, all the previous reported studies investigating crowding in DD have mainly used letters or letter-like stimuli, yet it is already known that individuals with DD could have difficulties in processing such linguistic stimuli. Moores and colleagues (2011; see also Cassim, Talcott, & Moores, 2014, for evidence in a nonsearch task) measured the accuracy of the target orientation in an array of different numbers of—and differently spaced—vertically oriented distractors in adults with DD and controls. Results showed that adults with DD presented larger effects of crowding and a stronger impact of the increased numbers of distractors. These perceptual–attentional variables correlated significantly with reading and spelling. These findings extended the previous results of crowding in DD from letters to nonlinguistic and noncomplex stimuli. Although the crowding in DD is almost exclusively studied in the visual modality, there are some works that showed crowding is also different in individuals with DD in other modalities. Geiger et al. (2008) examined the performance of children with and without DD in two analogous recognition tasks: one visual and the other auditory. Individuals with DD showed more crowding near the center in comparison with typical readers. Both groups performed comparably in recognizing centrally spoken stimuli presented without peripheral interference, but in the presence of a surrounding speech mask, individuals with DD recognized the central stimuli significantly less well than typical readers. The authors suggest that these data showed how peculiar crowding is in DD in both visual and auditory modalities. Moreover, Grant et al. (2000) showed a deficit in tactile perception in individuals with DD that can be considered a homologue of the excessive crowding observed in the visual modality.

Future goals for visual crowding and reading

As reported above, three important questions remain open regarding visual crowding and reading; here we would like to suggest future research projects that may answer them in quite a conclusive fashion.

The first question that urgently needs an answer is "Is the peculiar crowding often associated with DD a cause of the reading disabilities or a simple effect of DD?" An answer to this question is crucial because, although the effects of DD on the brain and on consequent behavior can be interesting, the main aim of DD research is to find all the possible causes of DD in order to train them to actively reduce DD incidence. Our proposal is to implement a longitudinal study in which the crowding will be measured at the prereading stage, reaching even the infant stage (Farzin, Rivera, & Whitney, 2010), and the same children will be followed the next years until the diagnosis of DD can be done (which varies depending on language transparency). If the amount of crowding measured at the prereading level is a predictor of future reading abilities, a causal link between crowding and reading will be demonstrated as previously shown for the attentional deficit in DD (e.g., Franceschini et al., 2012).

Assuming that this crucial question will have a positive answer with results showing that crowding is causally linked to DD, the next question that immediately comes to mind is "Can a training able to improve crowding resistance directly lead to better reading abilities?"

Being able to answer this question will have two positive effects: To strengthen the causal link between crowding and DD and to provide a possible remediation program for DD based on crowding resistance training that can be integrated with the preexisting treatments in order to reduce the symptoms of DD. Interestingly, Geiger et al. (1994) tested a new method for DD remediation based on the learning of a "visual strategy" by a specific attentional focusing training. The experimental group improved reading skills significantly more than the control group. The ratio between central and peripheral crowding also changed after the attentional training. Recently, Franceschini et al. (2013) found that the reading abilities in children with DD improves after playing AVG. AVG are

known to reduce the crowding effect in typical readers (Green & Bavelier, 2007); consequently, it could be interesting to test the crowding before and after the AVG training and to correlate the reading and the crowding resistance improvements to see whether less crowding will result in better reading. Another possibility could be to reduce crowding with a perceptual learning approach and test if it will lead to better reading abilities. Gori and Facoetti (2014) already proposed employing perceptual learning to improve the M–D stream functioning in individuals with DD. It is known, indeed, that perceptual learning can also reduce crowding (Chung, 2004; Chung, Levi, & Tjan, 2005; Chung, Li, & Levi, 2012; Y. He, Legge, & Yu, 2013; Hussain, Webb, Astle, & McGraw, 2012; Lee et al., 2010), being, at least on paper, a perfect candidate to be employed as a training procedure for the aforementioned aim. Moreover, it would be interesting to test, after the training, if the reduced crowding will be correlated to a better performance in both attentional and M–D tasks, which provides important information about the relationship of those deficits in causing DD.

Assuming the previous questions will be answered positively and results will show that training procedures to reduce crowding produce a direct improvement in reading abilities, the remaining question will be "Could a training at the prereading stage based on increasing crowding resistance in children at risk for DD reduce the incidence of future DD?" Future studies are needed to answer this exciting question. On paper, this answer has less of a chance to be a positive one. It is, indeed, complicated by the fact that it requires a combination of a longitudinal study with a training study. On the other hand, it could also be the most important answer because the ultimate common goal in DD research is reducing the incidence of DD before the manifestation of its symptoms.

In summary, this review article aimed to connect experts of vision sciences and reading in order to better understand the role of crowding in reading disability, and pave the way for studies to be able to (a) demonstrate a causal link between crowding and DD, (b) identify a risk of DD early, (c) produce new remediation trainings, and (d) project ambitious prevention programs that potentially could stem from new insights in the topic covered by this review.

Keywords: crowding, perceptual learning, noise exclusion, reading disorder, selective attention, action video games

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Corresponding author: Simone Gori.
Email: simone.gori@unipd.it.
Address: Developmental and Cognitive Neuroscience Lab, Department of General Psychology, University of Padua, Padua, Italy.

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