Special issue: Editorial

From dyslexia to dyslexias, from dysgraphia to dysgraphias, from a cause to causes: A look at current research on developmental dyslexia and dysgraphia

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Interpreting the reading and spelling profiles of children with developmental dyslexia and/or dysgraphia has traditionally proven a challenging task. In particular, problems seem to arise when trying to provide a unitary account of all individuals with developmental dyslexia or of all individuals with developmental dysgraphia. These attempts to provide a single account or a single generalization to cover all aspects of dyslexia consistently result in empirical problems, inconsistent results, and failures to replicate. The articles included in this issue suggest that the key to explaining this pattern may lie in the selectivity and heterogeneity of developmental dyslexia and dysgraphia types, in the effect of modulating factors such as orthographies, and in the need to search for various (rather than unitary) causes underlying the deficit (in general, spanning auditory, phonological, visual mechanisms etc. on one hand, and orthographic-specific impairments on the other). Indeed, these various causes lead to a variety of specific developmental dyslexias and dysgraphias.

1. Types of developmental dyslexia

Many studies have reported selective developmental reading deficits and have showed distinct error types and effects on reading, depending on deficits to different components in the reading process. To date, 17 types of developmental dyslexia have been identified and reported (depending on how they are counted), which show properties very similar to the parallel acquired dyslexia, a disturbance previously examined in patients with acquired dyslexia, which has found very limited space in the developmental literature. In attentional dyslexia, letters migrate between neighboring words but are correctly identified and maintain their correct relative position within the word.

Another type of dyslexia reported in adults with acquired dyslexia, but only sparsely reported in its developmental form, is dyslexia of reading without meaning (Although it is sometimes termed “hyperlexia”, we prefer not to use this term here, because the problem in this type of dyslexia is not that they read too well, as suggested by the “hyper” in hyperlexia). The study by Castles and colleagues describes in detail two children with this type dyslexia in its developmental form. These children showed very good oral reading of words and nonwords but were impaired in their word comprehension. These results suggest that reading without meaning can also be clearly demonstrated in a developmental form and with characteristics similar to those of the parallel, acquired dyslexia. The study also provides important evidence of the existence of the direct lexical route for reading. The participants read well via the lexical route (connecting the orthographic input lexicon with the phonological output lexicon), as demonstrated by their good reading of irregular words, but were still unable to understand the words they read, suggesting that their semantics (or semantic route) is impaired.

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spelling deficits, as proposed by Castles (2006); Castles et al. (2006); Castles and Coltheart (1993); Coltheart et al. (1983); Friedmann and Lukov (2008); Friedmann and Nachman-Katz (2004); Friedmann and Rahamim (2007); Marshall (1984) and Temple (1997).

Interestingly, the typical reading tests for developmental dyslexia, which assess nonword reading, reading of regular words, words, and word and nonword reading does not suffice, and tests should be elaborated to become sensitive to all subtypes of dyslexia. Furthermore, analysis of the number of errors is not sufficient, and one should analyze and report error rates as well as error rates when describing the reading of individuals with developmental dyslexia. For instance, the same error rates in reading a word list could indicate surface dyslexia if the errors are regularizations and neglect dyslexia if the errors are letter omissions and substitutions on one side of the word. The report of effects on reading (namely, which written stimuli are more susceptible to errors) is also relevant in this respect: whereas developmental surface dyslexia is expected to be pronounced in irregular words, but not in regular words and nonwords, developmental neglect dyslexia should manifest itself in both regular and irregular words, and in both words and nonwords.

Furthermore, given the large variability and different profiles of different children with developmental dyslexia and dysgraphia, looking at the results only at the group level does not suffice; hence, studies of a group of children with developmental dyslexia or dysgraphia would benefit from a report of the performance of each of the individuals in the group.

Other types of developmental dyslexia and dysgraphia are reported in other articles in this issue: Funnell and Pitchford described two children with surface dyslexia and three children with phonological dyslexia following left hemisphere stroke in childhood (and another child with surface dyslexia following a right hemisphere stroke). McLean, Castles, Coltheart, and Stuart described three children with developmental surface dyslexia and two with dyslexia that could be interpreted on the basis of the existing data as developmental phonological dyslexia or developmental visual dyslexia. The participants in De Luca et al.’s study showed a reading pattern typical of surface dyslexia.

These reports of children with various selective types of developmental dyslexia thus join a growing body of studies providing robust evidence for the existence of subtypes of developmental dyslexia. When error patterns of developmental dyslexia and dysgraphia were carefully examined, similarities emerged with subtypes of acquired dyslexia. This has been reported for developmental surface dyslexia (Broom and Doctor, 1995a; Castles et al., 2006; Castles and Coltheart, 1993, 1996; Coltheart et al., 1983; Friedmann and Lukov, 2008; Judica et al., 2002; Masterson, 2000; Temple, 1997; Valdois et al., 2003), developmental phonological dyslexia (Broom and Doctor, 1995b; Howard and Best, 1996; Temple, 1997; Temple and Marshall, 1983; Valdois et al., 2003), developmental reading-without-meaning dyslexia (Glosser et al., 1997), developmental deep dyslexia (Siegel, 1985; Stuart and Howard, 1995; Temple, 1988, 2003), and developmental peripheral dyslexias: developmental letter position dyslexia (Friedmann and Haddad-Hanna, in press; Friedmann and Rahamim, 2007; Friedmann et al., 2010), developmental attentional dyslexia (Rayner et al., 1989), and developmental neglect dyslexia (neglexia, Friedmann and Nachman-Katz, 2004; Nachman-Katz and Friedmann, 2007, 2010). For a comprehensive survey of this literature, see Brunsdon et al. (2002), Castles and Coltheart (1993), Castles et al. (1999, 2006), and Temple (1997).

For the reader who is more acquainted with research on acquired disturbances, the existence of different types of dyslexia and dysgraphia (which differ with respect to error types and effects on reading) and the need for analysis of error types and effects may seem straightforward. However, for reasons which would require a separate analysis, a large part of the research on developmental dyslexia and dysgraphia has followed different assumptions. We suggest that, given the parallelism between types of acquired and developmental dyslexia and dysgraphia, similar rules can be applied for research in developmental disorders.

2. Factors modulating developmental dyslexia and dysgraphia: effects of language and age

Not only do the different subtypes of developmental dyslexia, derived from impairments in different reading components, create different profiles, they can also change as a function of orthography. Interpreting dyslexia in orthographically consistent languages has proven difficult in part as an effect of the Anglocentrism (Share, 2008) of most literature, i.e., the tendency to use English, an orthographically irregular language, as the yardstick for interpreting the reading and writing disorders.

Three studies of the special issue contribute to characterizing the reading deficits of readers of orthographically consistent languages. Marinus and de Jong’s contribution focuses on Dutch children with dyslexia. It is well known that children with (certain types of) dyslexia show a quite marked length effect (e.g., Spinelli et al., 2005; Ziegler et al., 2003). Marinus and de Jong examine the relative role of letter length, phoneme length, and presence of digraphs in the reading of these children. De Luca and colleagues try to disentangle the role of global components in the reading deficit of Italian readers with developmental dyslexia. Based on an fMRI paradigm, Wimmer and colleagues examine the profile of brain activation of Austrian adolescents and adults with developmental dyslexia in a phonological lexical decision task within a dual-route perspective, and report distinct patterns of activation for the dyslexia group both for processes requiring the lexical route and for processes requiring the sublexical route.

Profiles of deficit can change also as a function of age. Angelelli and colleagues show that the spelling impairment of Italian children with dysgraphia assumes different characteristics in different grades: younger (third grade) children show an undifferentiated spelling deficit involving regular words, nonwords, and words with unpredictable spelling (irregular
words). By contrast, older (fifth grade) children are prevalently impaired in writing words with unpredictable transcription. These findings indicate that the children with dysgraphia learning a shallow orthography suffered from delayed acquisition of the sub-word-level routine together with a long-lasting deficit of orthographic lexical acquisition. Due to their different developmental trajectories, the joint effect of these two disturbances yields different spelling profiles as a function of literacy expertise.

Overall, there is an explanatory risk in assuming the homogeneity of reading and spelling deficit profiles when interpreting the causal mechanisms of these disturbances or when examining the neuroimaging correlates of the disturbance. As mentioned above, this problem is accentuated by the presence of important differences between orthographies. In reviewing the evidence on cross-linguistic studies of dyslexia, Hadzibeganovic and colleagues make the important point that profiles of brain activation should not be compared across languages disregarding the variability of the types of dyslexias and dysgraphias and their variability as a function of the characteristics of the orthographies.

3. From cause to causes of developmental dyslexia and dysgraphia

One can look in a similar way at the tendency to search for a single, unitary underlying cause of developmental dyslexia. There have been countless proposals to this aim. However, failures to replicate have been the norm rather than the exception in this area of research. Below, we suggest that the reason for this is that there is no such unitary explanation, given the different profiles of developmental dyslexia and dysgraphia, and different profiles may have different underlying causes.

One such example of an explanation that was suggested as a unitary account for developmental dyslexia, and that was repeatedly found not to apply to many individuals with developmental dyslexia, is the phonological impairment hypothesis. In its various forms, this account proposes that developmental dyslexia (as a unitary phenomenon) results from a phonological deficit or from poor phonemic awareness (Frith, 1997; Goswami, 2002; Marshall et al., 2001; Snowling, 1998; Stanovich, 1988). Although this claim may be true for some children with dyslexia (specifically, phonological and deep dyslexia), it does not hold for other types of dyslexia. Research over the past 10 years has proved that children can have pronounced developmental dyslexia but still show normal phonemic awareness and normal phonological skills (Castles and Coltheart, 2004; Friedmann and Rahamim, 2007; Lukov and Friedmann, 2004; Valdois et al., 2003; see Marshall, 1998 and McCloskey and Rapp, 2000 for discussions of this point). In this issue, Funnell and Pitchford report that the three children with surface dyslexia they tested excelled on a phoneme segmentation task, showing, again, that not all children with dyslexia have impaired phonological abilities.

Several other interpretations of dyslexia have also been proposed. One well-known early example is Pavlidis’s proposal of a selective eye movement deficit in dyslexia (Pavlidis, 1981; for counter evidence, see Brown et al., 1983; Olson et al., 1983; Stanley et al., 1983). An extremely large body of literature has focused on the hypothesis that dyslexia is due to abnormalities of the magnocellular component of the visual system, which is specialized for processing fast temporal information (e.g., Stein and Walsh, 1997), a deficit that would cut across phonological, visual, or motor deficits. Accordingly, dyslexics are seen as impaired in processing incoming sensory information in any of these domains. However, evidence supporting this hypothesis is at best controversial (for a review see Skottun, 2000). Nicolson et al. (2001) proposed the intriguing idea that impairments in the reading and writing of children with dyslexia are in fact caused by disorders of cerebellar development, considered critical for development of the ability to perform skills automatically (for counter evidence, see, for example, Raberger and Wimmer, 2003). More recently, Ahissar (2007) proposed the anchoring-deficit hypothesis, according to which dyslexics fail to form a perceptual anchor in tasks that require the encoding and comparison of several stimuli, such as tones or pseudowords. Again, failures to confirm the predictions of the hypothesis (Di Filippo et al., 2008; Ziegler, 2008) have been reported.

No systematic attempt is made here to review this extremely large body of literature. However, it is hard not to be surprised by the widespread inconsistency of findings in this area of research. In the special issue, two papers critically examine causal interpretations of dyslexia. McLean and colleagues show that prolonged attentional blink (or sluggish attentional shifting) is not specifically associated with reading disturbances, as previously proposed by Hari and Renvall (2001). Namely, they show that deficits in children with dyslexia appear tied to general components of performance rather than to prolonged attentional blink. Further, their analyses indicate that this interpretation has the potential to accommodate previous findings of attentional blink deficits in children with dyslexia. In two experiments, Georgiou and colleagues examine two separate hypotheses that link deficits in auditory temporal processing to dyslexia: the P-center or beat detection hypothesis (Goswami et al., 2002) and the rapid auditory processing hypothesis (Tallal, 1980). Results are clear-cut in showing that, as a group, children with dyslexia who speak an orthographically consistent language such as Greek do not show auditory processing deficits.

In commenting upon this literature, particularly the anchoring hypothesis (Ahissar, 2007), Di Filippo et al. (2008, p. F45) propose that “rather than searching for yet another unitary explanation of dyslexia, such as a deficit in setting up a perceptual anchor, recent work suggests that much can be gained in understanding dyslexia by taking into account the heterogeneity of dyslexia both in terms of reading performance and the deficits in various reading components.” An intriguing approach in this vein is to examine the reading disturbance within the so-called co-morbidity perspective. Phenotypic analyses indicate that several developmental deficits may arise from multiple cognitive deficits rather than a single primary cognitive deficit. This approach is illustrated in the paper by Willcutt and colleagues, in which reading disturbances and attention-deficit hyperactivity disorders (ADHD) are examined in the same group of children. The two disturbances show both common and dissociated cognitive weaknesses. Phonemic awareness, verbal reasoning, and working memory are impaired in children with dyslexia, whereas inhibitory control is deficient in children with ADHD. By contrast, both groups of
children display a common deficit in processing speed, a shared weakness due to a genetic influence as assessed by twin analysis. Thus, several perceptual and cognitive deficits may interact in producing the complex reading profiles that are present in developmental dyslexia and dysgraphia.

The contribution of Leppänen and colleagues on the relationship between atypical processing of sound frequency at birth and later literacy skills can be seen in this perspective. They examine auditory brain event-related potentials for tones varying in pitch in children with and without a familial risk background of dyslexia and conclude that a proportion of dyslexic readers with familial risk background are affected by atypical auditory processing. Thus, their findings indicate that a selective early deficit in auditory processing may contribute to shaping the later development of reading. These authors stress that “atypical auditory processing is unlikely to suffice as a sole explanation for dyslexia but rather as one risk factor”. Within the co-morbidity perspective, the challenge will be to investigate which perceptual and cognitive deficits independently contribute to produce the complex profiles of reading and spelling deficits observed in developmental dyslexia and dysgraphia. Conceivably, some of the deficits that have failed to represent unitary interpretations of the reading disturbance may well be re-interpreted within this theoretical perspective.

Overall, we feel that the papers of the special issue contribute to the process of clarifying the selectivity and heterogeneity of reading and spelling profiles and offer several new insights for studying the causes of these disturbances. Given the various profiles of developmental dyslexia and dysgraphia and selective impairments of reading and spelling, a study of the underlying causes that takes into account this heterogeneity promises to bear fruit and provide more precise explanations.

REFERENCES


