Chapter XVII

THE NEUROBIOLOGICAL BASIS OF DEVELOPMENTAL DYSLEXIA—CURRENT FINDINGS AND AREAS OF FUTURE RESEARCH

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ABSTRACT

A growing body of evidence suggests that developmental dyslexia might arise from impaired information processing, which is based on neural deficiencies. During the last 15 years, research on the neurobiological basis of dyslexia has dealt mainly with 3 different issues of interest: The first one concerns abnormal morphological findings like unusual asymmetries of the temporal plana and deviations of the corpus callosum and cerebellum. The second one concentrates on the analysis of abnormal activation patterns of the left hemispherical frontal and temporal lobe, namely, of the angular gyrus and the Broca’s area. Researchers assume that these unusual patterns are connected with phonological deficits. The third one refers to global impairments of subcortical systems that might play a major role in the development of dyslexia. An aberration of magno-cells of the geniculate nucleus is supposed to affect rapid information processing, which results in phonemic discrimination and awareness difficulties. In summary, the evidence for a relation between unusual asymmetries of the temporal plana and dyslexia seems to be very modest at best. A decision between the second and the third approach is beyond the scope of the available empirical data. It still remains unclear whether the information processing deficit is restricted to phonology only, or if there is a general deficit of rapid information processing.

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In the middle ages and far into modern times, literacy was regarded as an extraordinary ability. Nowadays, in western culture every adult spends about two hours per day reading—especially at work—and approximately 90% of all jobs require literacy (Klicpera & Gasteiger-Klicpera 1995, p. 5). At the beginning of the last century, different authors—mainly physicians—independently of each other mentioned dyslexia for the first time. For example, Hinshelwood (1900) described this phenomenon as “poor reading and spelling in the absence of any other academic problems” and termed it “word blindness”. At this time, the presumed causes were lesions in the field of the angular gyrus (Freund, 1889), birth traumata (Fisher, 1910), prenatal disease and deficient development during the embryonic stage (Hinshelwood, 1917). In particular, the role of the angular gyrus was discussed, since a lesion in this region results in alexia.

Medical literature of the beginning 20th century distinguished between a congenital and an acquired form of dyslexia (Pickle, 1998). Since then, the discrepancy between reading and spelling abilities on the one hand and intelligence on the other hand has served as a diagnostic criterion of developmental dyslexia. In recent times, this procedure has been subject to strong criticism because of the generally weak correlations between reading and spelling abilities and IQ ratios (Fletcher, Francis, Rourke, B. A. Shaywitz, & S. E. Shaywitz, 1992; Siegel, 1992; Stanovich, 1991). The discussion mainly concentrates on the question, whether diagnosis of dyslexia via IQ ratios is adequate or whether more reasonable classification systems should be applied. According to Zielinski (1998, p. 108), the insufficient definition represents an artificial psychometric construct of dubious utility. S. E. Shaywitz, Fletcher, and B. A. Shaywitz (1996), as well complain about the lack of empirical evidence:

For too many years, the road to the identification of a reading disability has been littered with artificial barriers requiring children (and adults) to meet arbitrarily imposed criteria such as those requiring an ability-achievement discrepancy. Moreover, and of most concern, we now know that these definitions more often served administrative needs than reflected valid biological underpinning. (p. 213)

So far, however, it is unclear whether developmental dyslexia should be regarded as a deficit or as a developmental lag (Francis, S. E. Shaywitz, Stuebing, B. A. Shaywitz, & Fletcher, 1996). In the latter case, developmental dyslexia just represents the lower section of the normal distribution of reading ability in the total population and, therefore, does not justify a discrepancy-based diagnosis (Francis et al., 1996).

Whether there are distinguishable subgroups of children with poor reading and spelling abilities and which diagnostic criteria should be employed in this case is not yet sufficiently clarified (Fletcher et al., 1997; Francis et al., 1996; Morris et al., 1998; S. E. Shaywitz, Fletcher, et al., 1996; Stanovich, Siegel, Gottardo, Chiappe, & Sidhu, 1997). Therefore, almost all studies draw on the discrepancy-based definition. Nevertheless, a distinction between different subgroups may be crucial for the assignment of different aetiologies underlying each subgroup. Only few research groups completely refuse the distinction between “ordinary” poor readers and diagnosed dyslexics (S. E. Shaywitz et al., 1998; Nagarajan et al., 1999). Others rely, for example, on the distinction between surface- and
phonological dyslexia as defined by the dual route theory (Castles & Coltheart, 1993; Coltheart, Curtis, Atkins, & Haller, 1993; Spinelli et al., 1997).

Current research on the neurobiological correlates of developmental dyslexia is dominated by three distinct approaches: First, following the work of Galaburda, Sherman, Rosen, Aboitiz, & Geschwind (1985), who observed unusual asymmetries of the temporal plana of dyslectic persons in post mortem studies, researchers have looked for relations between brain morphology and the occurrence of developmental dyslexia. Second, based on the findings of research involving imaging technologies, other scientists (e.g., S. E. Shaywitz, 1998) attribute dyslexia to the impairment of higher cognitive processes of speech processing. They assume that deficits are caused by a defective phonological module. The third approach asserts that dyslexia is due to deficits in lower sub-cortical systems involved in visual and auditory information processing which subsequently impair higher cognitive functions. This hypothesis is based on histological findings in the area of the lateral and medial geniculate nucleus, as well as on psycho-physiological observations.

Supporters of both the second and third approaches agree with the fact that developmental dyslexia is a consequence of phonological weakness. However, they localise the “true” cause of developmental dyslexia on different stages of information processing (Miles & Stelmack, 1994).

ANATOMICAL AND HISTOLOGICAL FINDINGS IN THE REGION OF THE PLANÀ TEMPORÁLIA, THE CORPUS CALLOSUM AND CEREBELLUM

Throughout the last century, researchers reported anatomical asymmetries of the brain, but until the 1960’s these insights were largely ignored. In 1968, Geschwind and Levitsky discovered an asymmetry of the temporal planum—a triangular region on the upper surface of the temporal lobe within the sylvian fissure—with the left temporal planum being larger than the right one. They interpreted this finding as the neurobiological substrate of the lateralisation of language in the brain. Geschwind and Levitsky found the asymmetry in about 65% of the adult population (see also Morgan & Hynd, 1998; Rumsey, Donohue, et al. 1997).

In the 1980’s, Galaburda and colleagues (Galaburda et al., 1985; Galaburda, 1989) examined the brains of seven young deceased developmental dyslexics. They found an unusual asymmetry of the temporal plana, specifically, an enlargement towards the right temporal lobe in all seven brains. Furthermore, abnormal cell groups (neuronal ectopias) were discovered in the left-hemispherical inferior frontal and superior temporal gyrus (Schultz et al., 1994). Whether the sample in Galaburda’s studies is representative of dyslexics in general remains in doubt (Beaton, 2002).

Since an asymmetry of the temporal plana can already be observed in foetal and neonatal brains (Neuhäuser, 1994; Schultz et al., 1994), these findings led to the assumption that dyslexics show abnormal brain development during the prenatal stage (Rumsey, Donohue, et al., 1997). Furthermore, Galaburda (1989) concluded that developmental dyslexia is related to unusual morphology of the temporal plana, even if the symmetry or reversed asymmetry of
the brain regions themselves is not classified as pathological. Recently, anomalies of the corpus callosum and cerebellum became a focus of research as well.

Reversed Asymmetries and Unusual Symmetries of the Planum Temporale

Recent in-vivo studies, particularly those using magnetic resonance tomography (MRI), have only partly acknowledged the findings of Galaburda (1989) or even produced contradictory results (Morgan & Hynd, 1998). Schultz et al. (1994) examined 14 dyslectic and 14 non-dyslectic, right-handed children aged between 7.5 and 9.7 years. MRI sagittal images of the temporal and parietal lobes were taken. Using the MRI data, the volumes and surface areas of various brain structures were estimated. Additionally, the researchers calculated an asymmetry index of the surfaces of the temporal plana. Initially, multiple significant differences between the control and experimental group were found, but none of the effects remained significant when the statistical analysis controlled for age. An insignificant mean difference of about three months of age was thus responsible for the ostensible relations between diagnosis (dyslexic versus control) and brain morphology.

Another MRI study was carried out by Rumsey, Donohue, et al. (1997). In the same way as Schultz et al. (1994), they determined the volumes and surfaces of the temporal and parietal plana and calculated asymmetry indices for these brain regions. They could replicate the results of Geschwind and Levitsky (1968), but did not find unusual asymmetries in the brains of dyslectic persons. Eckert et al. (2003) also failed to reproduce unusual asymmetries or size for the temporal lobes, including the temporal plana and Heschl’s gyri (primary auditory cortex). Measures of the temporal lobe did not differentiate between dyslexics and controls.

In contrast to the aforementioned studies, Dalby, Elbro, & Stødkilde-Jørgensen (1998) measured the length of the lateral fissure, the volume of the subcortical part of the temporal lobes and the volume of the entire temporal lobes of normally talented adolescents, dyslexics (discrepancy between IQ and reading performance) and adolescents with generally poor reading abilities (without IQ discrepancy). The group of dyslexics showed significantly more symmetry or a reversed asymmetry of the temporal lobes compared to both other groups.

In summary, the evidence for a relation between unusual asymmetries of the temporal plana and dyslexia seems to be very modest at best. The different studies have yielded contradictory findings, and most researchers were not able to reproduce the unusual asymmetries first reported by Galaburda and colleagues (Galaburda et al., 1985; Galaburda, 1989). The results of Schultz et al. (1994) indicate that morphological aberrations may be the consequence of other confounding variables like age or sex. Most likely, reversed asymmetries of the temporal lobes are neither the cause for developmental dyslexia, nor predictors of individual performance in reading and spelling.

The Corpus Callosum and Developmental Dyslexia

Due to the discussion about the symmetry or asymmetry of the temporal plana, the corpus callosum has become a focus of research (Rumsey, 1996). Since the right-hemispherical
temporal lobe of dyslectic persons is presumably enlarged, some researchers assumed alterations in the size of the splenium (posterior situated fifth part of the corpus callosum), because the commissural fibers of the left and right temporal lobe cross this region (Rumsey, 1996). Contrary to these presumptions, Hynd et al. (1995) discovered in their MRI studies significant differences in the size of the genu (anterior situated fifth part of the corpus callosum), but not in the size of the splenium. The cross sectional area of the genu was on average 25% smaller in the dyslexics.

Using different methods for statistical shape analysis, von Plessen et al. (2002) reported a shorter corpus callosum in dyslectic children, which was due to a shorter posterior midbody region, whereas the genu seemed to be normal. According to the authors, dyslexics presumably fail to undergo the massive myelination in this region during reading acquisition.

The Cerebellar Deficit Hypothesis

Recently, a new perspective was brought up by Nicolson, Fawcett and Dean (1995). Based on the fact that many dyslectic children exhibit problems in motor skills, automation and other tasks that include cerebellar involvement, Fawcett and Nicolson (1999) suggested that a mild cerebellar dysfunction might cause many of these problems. They have proposed a motor theory of speech perception, which views dyslexia as the result of articulatory deficits, problems in the automation of skill and knowledge and motor skill impairments (Nicolson & Fawcett, 1990; Nicolson, Fawcett, & Dean, 2001a, 2001b). According to their approach, impairments of several distinct routes accumulate to a phonological core deficit. Nicholson et al. (2001a) suggest that cerebellar deficits constitute an alternative and perhaps parallel mechanism to magnocellular abnormality, which is discussed later.

In line with this assumption, several groups of researchers observed morphological deviations within the cerebellum of dyslexics. Rae et al. (2002) reported morphometric differences in the ratios of the grey matter of the left and right cerebellar hemispheres. In contrast to the controls, who showed a larger right hemisphere, dyslexics were found to have symmetric cerebellar grey matter. Moreover, Rae et al. (1998) found significantly altered biochemical metabolite ratios within the right, but not the left, hemisphere of the cerebellum. These changes are indicative of altered patterns of cell density in the cerebellum of dyslectic individuals. Most likely this finding is due to smaller numbers of large neurons. This finding was confirmed by Eckert et al. (2003). Male dyslexics exhibited significantly smaller right cerebellar anterior lobes as well as smaller inferior temporal lobes as compared to controls. Using these distinguishing features, Eckert et al. correctly classified about 80 % of the participants.

Finch, Nicolson, and Fawcett (2002) re-examined the post-mortem tissue that was studied by Galaburda et al. (1985) years before. They investigated the morphology of the cerebellum and its connections, especially the constituents of the olivio-cerebello-dentate pathway, which they considered to be the major cerebellar-subcortical structure involved in language-related tasks. Finch et al. (2002) report fewer small cells and more large cells in the cerebellum and in the inferior olive of dyslexics compared to controls, but they did not find any significant differences in cerebellar asymmetry.
At the moment, there is a controversial debate whether a cerebellar deficit is the cause, correlate or consequence of developmental dyslexia. Moreover, it remains questionable whether this specific deficit exists at all (Beaton, 2002; Bishop, 2002). While different authors agree that many of the difficulties of developmental dyslexics are consistent with a cerebellar deficit, multiple alternative explanations stand to reason (Beaton, 2002; Bishop, 2002; Ivry & Justus, 2001; Zeffiro & Eden, 2001). Therefore, at present the cerebellar deficit hypothesis and especially the motor theory of speech perception remain speculative.

Neurophysiological and psychological findings concerning cortical processes of speech perception. In recent years, many studies have been published which used new imaging techniques, above all positron emission tomography (PET) and functional magnetic resonance tomography (fMRI). The aim of these studies was to find differences in the activation patterns of higher cortical speech processing systems in dyslectic as compared to non-dyslectic persons (e.g., Fulbright et al., 1997; Paulesu et al., 1996; Pugh et al., 1996; Pugh et al., 1997; Pugh et al., 2000; Rumsey et al., 1994; Rumsey, Nace, et al., 1997; Rumsey et al., 1999; B. A. Shaywitz et al., 1996; S. E. Shaywitz, B. A. Shaywitz, et al., 1996; S. E. Shaywitz et al., 1998; Horwitz, Rumsey, & Donohue, 1998; Ruff, Cardebat, Marie, & Démonet, 2002). The so-called subtraction paradigm was employed in all these studies. This means that activation patterns observed during a complex activity (e.g., reading) are subtracted from the activation patterns measured during a state of rest or non-specific activity, for example, during the fixation of a cross in the center of a screen. Some studies (S. E. Shaywitz et al., 1998; Pugh et al., 1996; Georgiewa et al., 1999) used more complex subtraction mechanisms, in which the activity patterns of several hierarchical tasks were subtracted from each other. By means of this procedure, researchers attempt to identify differences in the activation patterns of different cognitive subprocesses (e.g., orthographic, phonological and lexical/semantic subprocesses of reading, Pugh et al., 1996).

This procedure entails many potential difficulties. The main point of criticism is the assumption that the addition or removal of a cognitive operation from a complex task leaves the other operations unaffected. A good deal of research in cognitive neuropsychology challenges the conception of serial and unidirectional information processing in the human brain. Instead it suggests that all components of these systems are extremely interactive (Pugh et al., 1996; Rumsey, 1996). Thus, the addition or removal of a single operation can highly influence the way in which the whole neural network structure functions. By contrast, the research of activation patterns using this subtraction paradigm is based on the assumption of a strictly hierarchically organised system of information processing (see also the discussion between Poeppel 1996a, 1996b and Démonet, Fiez, Paulesu, Petersen, & Zatorre, 1996). However, despite of its methodological problems, this approach has improved our understanding of developmental dyslexia.

Phonological information processing involves a complex interaction of different brain structures (Démonet et al., 1996). PET studies found activation samples of the left hemisphere in Brodman area 21 (inferior and medial temporal gyrus), area 22/42 (Wernike’s area), area 39 (angular gyrus), area 40 (supramarginal gyrus), area 44 (inferior frontal gyrus, Broca’s area), and in further frontal and temporal-parietal areas (see Poeppel, 1996a). Although the results vary strongly in dependence of the experimental design, they are all consistent with traditional neurological models of speech processing. Roughly speaking,
Form and pattern of visual information during reading is processed within the primary and secondary visual cortex and is then forwarded to the angular gyrus. This region is regarded as the reading center within the human brain. Subsequently, the information is passed on to Wernicke’s area, the "sensory language center" and finally—via the Fasciculus arcuatus—transmitted into Broca’s area, the "motor language center". From there, depending on the task setting, the information takes its way to the appropriate brain regions (Trepel, 1999, p. 222).

**Activation Centers and Levels during Reading**

In their fMRI studies, Pugh et al. (1996, 1997), Fulbright et al. (1997), B. A. Shaywitz et al. (1996) and S. E. Shaywitz, B. A. Shaywitz, et al. (1996) observed the reading process of normal reading persons and could thus confirm numerous results of the lesion research. In order to discover neurological correlates of different components of the reading process (visual-spatial, orthographic, phonological and lexical-semantic operations), they applied a complex system of hierarchical subtraction mechanisms. For each component, they found an involvement of various networks in left-hemispherical brain regions (Fulbright et al., 1997). During orthographic processing (e.g., detection of letters), the extra striate regions (Brodmann area 18, 19) in the occipital lobe play a particularly important role (Pugh et al., 1996). Phonological recoding especially activates the inferior frontal gyrus (and to a smaller extent also the superior and medial frontal gyrus, Pugh et al., 1996). The most interesting result was observed during lexical-semantic decoding. Namely, a network of cortical structures of the temporal lobe seems to be involved in it (medial temporal gyrus, superior frontal gyrus; Pugh et al., 1996) but in addition a strong activation of the inferior frontal gyrus was also observed during this task. Seemingly, the lexical-semantic component does not function independently of the orthographic component but rather relies on it. According to Pugh et al. (1996), these insights have important implications for models of the reading process, particularly the dual route theory. There seems to be no lexical (direct) access without simultaneous activation of phonological recoding strategies (indirect route). The researchers assume that the inferior frontal gyrus operates in a multi-functional way: "[We] assume that this system is relevant to both assembling a phonological representation from print and to transferring this information into short-term store ". According to S. E. Shaywitz (1997) and Fulbright et al. (1997) these results suggest that developmental dyslexia will finally be traced to a purely phonological deficit.

In another fMRI study, S. E. Shaywitz et al. (1998) compared the activation patterns of dyslectic and non-dyslectic readers and applied the same system of subtraction mechanisms. The non-dyslectic readers showed an increase in activity with an increase in task difficulty in a large posterior area within the left hemisphere, including Wernike’s area, the angular gyrus, and the extrastriate and striate cortex. There was no comparable increase in activity observable within the dyslectic readers. S. E. Shaywitz et al. (1998) interpret these findings as the neurobiological basis of an insufficiently developed phonological consciousness and the inability to segment words into their single phonemes. Instead, dyslexics showed a systematic over-activation of frontal brain structures, in particular the inferior frontal gyrus. The researchers suggested that this over-activation could be due to greater effort during reading.
and, therefore, be an indication of compensative efforts. The finding that the dyslectic readers showed a substantially weaker and more unsystematic increase of activity in the range of the angular gyrus confirms the results of traditional lesion research, according to which a lesion in this area usually leads to an alexia (Birbaumer & Schmidt, 1996, p. 406).

The results of Rumsey, Nace, et al. (1997) and Rumsey et al. (1999) point into a similar direction: In comparison to the control group, dyslexics showed substantially more zones of activation and deactivation, whereby the activity patterns rather differed in intensity than localisation. Rumsey, Nace, et al. (1997) likewise underline the significant roles of the inferior temporal gyrus and the entire temporal lobe of the left hemisphere in processing orthographical and phonological tasks. Besides, they found different correlations between reading ability and the intensity of the activation of the left-hemispherical angular gyrus in non-dyslectic as compared to dyslectic readers. In non-dyslectic readers, the correlation was highly positive, whereas it was highly negative in dyslexics. Therefore, a high activity within the range of the angular gyrus rather seems to disturb the reading process in dyslexics (Rumsey et al., 1999). With reference to S. E. Shaywitz et al. (1998), Rumsey and colleagues (1999) interpret their results as a confirmation of the hypothesis that a disturbance within the range of the left-hemispherical angular gyrus might cause developmental dyslexia.

Georgiewa et al. (1999) used a similar design as S.E. Shaywitz et al. (1998) and found a significantly stronger activation of the Broca’s area and the left-hemispherical inferior temporal gyrus in non-dyslectic children during a phonological transformation task. However, there were no differences during reading of regular german words. Therefore, they assumed deficiencies in the processing of compound phonological units, for example, in connection with grapheme-phoneme-correspondences, but no deficits in the processing of phonologically regular words.

Developmental Dyslexia as a Phenomenon of Functional Disconnection

Some researchers view developmental dyslexia as a disconnection-syndrome and thus examined the hypothesis that, in the long run, dyslexia is due to the separation of different brain areas: Similarly to Pugh et al. (1996), Paulesu and colleagues (1996) used a system of various tasks and subtraction mechanisms in order to examine activation patterns during different phonological operations (phonological short-term memory and phonological similarity judgement) by means of PET. In contrast to normal readers, dyslexics displayed no activation of the left insula. Furthermore, the synchronization of the activated brain regions was weak as compared to the controls. On the basis of these results and other preceding studies, Paulesu et al. suggested that the inferior frontal gyrus functions as a rehearsal system whose the task is limited to the processing of individual phonemes. The supramarginal gyrus is estimated to play an important role in phonological short-term memory and sections of the superior temporal gyrus seem to process assembled phonological units on the basis of whole words. Thereby, the insula might function as a bridge between the two brain regions. According to Paulesu et al., a lack of activation in this region leads to the separation of the anterior and posterior language regions of the temporal and frontal lobes.

Horwitz et al. (1998) pursued another approach. They used PET images to correlate the intensity of activation in different left-hemispherical areas with each other. A high correlation
between the brain activity of different frontal and temporal regions and the activity of the angular gyrus was observed in normal readers. The dyslectic participants, however, showed very poor correlations. According to Horwitz and colleagues, the absence of functional connections of the angular gyrus with corresponding brain regions—in particular the inferior frontal gyrus and the superior temporal gyrus—in dyslexics is compatible with models of the reading process in acquired dyslexia.

Pugh et al. (2000) likewise analysed correlations between the activity of different brain regions and the angular gyrus. Only under conditions, which required phonological abilities, dyslexics did not show correlations between the activity of the left-hemispherical angular gyrus and other ipsilateral posterior areas. The correlation of the right-hemispherical areas did not differ from the control group. Furthermore, the authors found no differences in tasks that did not depend on phonological abilities. Pugh et al. interpreted these results as a confirmation of their hypothesis that dyslexia is the consequence of a specific phonological deficit.

To summarize, although the observations of individual groups of researchers differ in detail, the use of imaging techniques has corroborated the results of the traditional lesion research. All quoted studies report deviated activation patterns, which can be interpreted within the context of traditional lesion research. However, these results do not necessarily permit conclusions about the cause of developmental dyslexia, nor do they lead to potential approaches for therapy.

A handicap of some of the older studies was the examination of grown-up dyslexics only, because their findings could as well be attributed to compensatory strategies. However, newer studies, for example, Corina et al. (2001), Georgiewa et al. (1999, 2002) and B. A. Shaywitz et al. (2002) focused on children and teenagers and replicated the before mentioned results. Meanwhile, there is evidence, that deficiencies of the neural system of dyslexics are already present within the first grades in elementary school. According to B. A. Shaywitz et al. (2002), developmental dyslexia in children must be seen within the same framework as developmental dyslexia and acquired dyslexia in adults.

It has to be noted, though, that findings in children at the age of 8 may not easily be transferred to pre-school-age children. After all, acquisition of reading and spelling leads to functional restructuring within neural networks (Chase, 1996; Castro-Calda, Petersson, Reis, Stone-Elander, & Ingvar, 1998). Altered activation patterns may therefore reflect consequences of a failure in this restructuring process instead of being the true cause of developmental dyslexia.

Another point of criticism is the limited temporal and spatial resolution of imaging technologies (Poeppel, 1996a; Chase, 1996; Bookheimer & Dapretto, 1996).

**Dysfunctions of Basic Information Processing**

Within the first half of the 20th century, researchers favoured the hypothesis that deficits of visual information processing lead to developmental dyslexia. Orton (1925), for example, assumed that these individuals suffered from a neuro-visual deficit based on insufficient lateralisation and hemispherical dominance in the brain. This concept was displaced by models of information processing that emphasized disturbed language processing (Näslund,
In the meantime, the connection between a lack of phonological awareness and developmental dyslexia has been well established (Bradley & Bryant, 1978; Liberman, Shankweiler, Fischer, & Carter, 1974; Schneider, 1997; Zielinski, 1998; Wimmer, 1996). The hypothesis of deficient processes of visual and auditory perception nevertheless has been brought up again (Eden, VanMeter, Rumsey, & Zeffiro, 1996), because the mechanisms underlying deficits in phonological awareness remain unclear.

The Transient Visual System

Since permanent use is made of the visual perception system during reading and writing, disturbed processes of perception can have a major impact on performance. Visual confusion (e.g., letter reversal, distortion, blurring and superimposition) can complicate the reading process and the visual-linguistic integration of information (Stein & Walsh, 1997; Eden, VanMeter, Rumsey, & Zeffiro, 1996).

Initially, research in this field concentrated on the observation of temporal resolution of visual stimuli, for example, via the use of flicker fusion rates (Martin & Lovegrove, 1987). By means of the method of limits, the temporal resolution capability of the visual system can be determined as a function of the spatial resolution of the visual stimulus: The finer grained the visual stimulus, the lower the temporal resolution (Lovegrove, Heddle, & Slaghuis, 1980). For this purpose, researchers normally use gratings with sinusoidal varying luminescence.

Martin and Lovegrove (1987) observed a characteristic decrease of the flicker fusion frequency in dyslexic children. It was possible to differentiate between dyslexics and controls on the basis of this frequency. Moreover, approximately 75% of the dyslectic children showed a slower increase of the temporal resolution capability when stimuli with gradually reduced spatial resolution were presented (Lovegrove et al., 1980). These findings were repeatedly confirmed (Demb, Boynton, Best, & Heeger, 1998; Eden, Stein, H. M. Wood, & F. B. Wood, 1995; Lovegrove, 1993; Slaghuis & Ryan, 1998). However, other researchers failed to reproduce them (Barnard, Crewther, & Crewther, 1998; Gross-Glenn et al., 1995; Johannes, Kussmaul, Münte, & Mangun, 1996; Kronbichler, Hutzler, & Wimmer, 2002; Williams, Stuart, Castles, & McAnally, 2003). Bednarek and Grabowski (2002) found an oversensitivity to luminance contrast of low spatial gratings in developmental dyslexics. Farrag, Kehr, and Abel-Naser (2002) reported prolonged P100 latency of visual evoked potentials in response to stimuli of low spatial frequency but not in response to stimuli of high spatial frequency.

Interestingly, Borsting et al. (1996), Slaghuis and Ryan (1998), and Spinelli et al. (1997) only observed a reduced temporal capability in dyslectic children who had problems with segmenting and analysing words (i.e., children with a phonological deficit) but not in dyslectic children without phonological problems. These findings point to a possible relationship between insufficiencies in the temporal resolution of rapid visual stimuli and phonological deficits. In contrast to these studies, Williams et al. (2003) reported different outcomes of contrast sensitivity tasks. Neither in the dyslectic group, nor in phonological subgroups of developmental dyslexics they found general deficits in magnocellular functioning.
Lovegrove (1993) interprets the results of his studies in the context of neuropsychological models of visual perception processing and stresses the role of two different subsystems, namely, the transient and sustained system. The transient visual system is characterised by high transmission rates and reacts predominantly to movements. The sustained system on the other hand is sensitive for visual stimuli of high spatial frequency.

The neuronal equivalent for these two functional subsystems can be found in the parvocellular and the magnocellular fibres (organised in six different layers) of the lateral geniculate nucleus, a thalamic body (Birbaumer & Schmidt, 1996, p. 392). The parvocells are small cells that transmit information concerning colour, shape and detail from the central fovea to the visual cortex in the occipital lobe (Wittmann & Pöppel, 1999). The magnocells have large, fast transmitting axons that convey information about brightness contrasts from the movement fields distributed across the entire retina (Kolb & Wishaw, 1993, p. 63; Birbaumer & Schmidt, 1996, p. 391).

Electrophysiological measurements have revealed that the magnocells are characterized by substantially higher transmission rates than the parvocells. Therefore, the magnocells can react much more quickly to fast moving stimuli. Altogether, the parvocellular pathways process visual stimuli of low temporal frequency and high spatial detail, whereas the magnocellular pathways process stimuli of high temporal frequency and low spatial complexity (Wittmann & Pöppel, 1999).

In post-mortem studies, Galaburda and Livingstone (1993) found characteristic differences between non-impaired and dyslectic persons within the area of the lateral geniculate nucleus: The magnocells of the dyslectic persons were on average 27% smaller and more variable in form and size. Furthermore, Galaburda, Menard and Rosen (1994) examined the medial geniculate nucleus, the thalamic body of the auditory system, and also discovered deformations of the magnocells.

By the use of fMRI, Eden, VanMeter, Rumsey, Maisog, et al. (1996) verified these phenomena in living persons as well. They examined responses to stimuli of low contrast and high temporal frequency in the V5/mt area—a part of the magnocellular pathways and the primary visual cortex. Bilateral activity in this area was observed in the normal readers but in only one of the dyslectic participants. However, both dyslectic and normal readers showed identical activation patterns in the primary and secondary visual cortex (V1 and V2) while looking at static stimuli. This points to a normal function of the parvocellular system. Demb, Boynton, & Heeger (1999) could replicate most of these findings. They also used fMRI in order to measure the activation patterns in the entire occipital lobe. In comparison to controls, dyslexics showed reduced activity within the range of MT+ and V1 on test conditions that addressed particularly the magnocellular fibres (low spatial in connection with high temporal resolution and low brightness). Those subjects who showed intense brain activity within the range of V1 and MT+ in this condition had a superior speed of reading and better temporal resolution capability. However, on test conditions that addressed the parvocellular fibres, there were no differences. Both Eden, VanMeter, Rumsey, Maisog, et al. (1996) and Demb et al. (1999) interpret their results as confirmation of the hypothesis that a magnocellular defect leads to a disturbance of the processing of rapid stimuli which in turn causes developmental dyslexia.
Possibly, a weakness of the magnocells could negatively affect the oculomotor system. The interaction of magnocellular and parvocellular pathways is fundamental for the effectiveness of the reading process. Reading is characterized by an interrelation of fixations and saccades. During a fixation, the parvocellular system can efficiently process the form and pattern of stimuli that are projected stably on the fovea. This is ensured by their functional characteristic of high spatial resolution. By contrast, the magnocells process information about the rough position of the stimuli on the entire retina. This is of crucial importance to fixate the relevant objects by means of a saccadic eye movement. Activated by a fast modification of the image on the retina, the magnocellular pathways suppress any activity within the parvocellular layers. Otherwise, the slow electrophysiological characteristics of the parvocellular pathways would lead to the interference of two subsequently fixated visual stimuli, for example, two sections of a text. If this tuning is not successful because of weak magnocellular fibres, the images on the retina acquired during successive saccades can no longer be separated clearly (Wittmann & Pöppel, 1999). A suboptimal position of letters on the fovea due to a lack of spatial guidance can occur as well (Stein & Walsh, 1997). The resulting visual confusions can cause letter reversal, distortion, blurring and superimposition and, therefore, complicate the reading process considerably.

It has repeatedly been shown that the saccades of dyslectic children are substantially less precise and more variable in speed compared with the saccades of children of the same age (Biscaldi, Fischer, & Aiple, 1994; Biscaldi, Gezeck, & Stuhr, 1998; Fischer, Biscaldi, & Hartnegg, 1998). Other aspects of the oculomotor system of dyslectic children as, for example, vergence and fixation control, are equally impaired (Demb et al., 1998; Eden, Stein, H. M. Wood, & F. B. Wood, 1994; Eden et al., 1995). Still further investigation is needed to corroborate the idea that these findings can be explained by a magnocellular impairment. However, it is obvious that imprecise eye movements complicate the reading process (Biscaldi et al., 1994).

**Auditory Information Processing**

Galaburda et al. (1994) examined the medial geniculate nucleus of five dyslectic and seven control brains. In the dyslectic sample, the neurons on the left side were significantly smaller than those on the right, whereas the controls showed no asymmetry. Moreover, the neurons were generally smaller and their size was more variable than those of the control sample. The brains had already been studied by Galaburda and Livingstone (1993) and showed aberrations in other regions as well, for example, in the lateral geniculate nucleus. However, the small size of the sample of five dyslectic and seven control brains does not permit a generalisation of the results. On the other hand, the findings are consistent with a variety of reported behavioural deficits concerning auditory information processing.

Whereas there is substantial support for a general deficit in visual perception and control of saccadic eye movements, it remains unclear whether there is a general auditory dysfunction as well. The current discussion is dominated by two different hypotheses: The first one assumes that the phonological processing of consonants and vowels at a segmental level constitutes the core deficit in developmental dyslexia. The second hypothesis focuses on deficits of discrimination abilities of rapid acoustic stimuli or even a general dysfunction of
temporal information processing across modalities and argues that this can give rise to at least some subtypes of developmental dyslexia. In the light of this approach the well documented phonological processing deficits are just an epiphenomenon.

Tallal (1980) first introduced the concept of deficient processing of rapid acoustic stimuli to the field of specific language impairments (SLI) and dyslexia (Rosen & Manganari, 2001). Tallal and colleagues were convinced that it is necessary to start with the examination of basic auditory functions before higher order speech deficits are taken into consideration (Tallal, Miller, & Fitch, 1993). They analysed the performance of children with SLI and with dyslexia in a variety of tests. Children with SLI as well as those developmental dyslexics with a phonological deficit showed characteristic problems in the identification of rapidly presented sinusoidal tones of different frequency. If the interstimulus interval (ISI) of two tones was shorter than 350 ms, the performance of the impaired children dramatically decreased. They could no longer discriminate whether two sounds in rapid succession had different frequencies or not. These results were replicated by Reed (1989).

Nagarajan et al. (1999) examined the processing of rapid sequences of tones by means of magneto-encephalography (MEG) and found the same characteristic impairment. They assumed that this was due to an unusual strong and long lasting post stimulus inhibition in dyslexics, which resulted in drastically reduced discrimination ability if the ISI was 200 ms and less.

Schulte-Körne, Reimel, Bartling, & Remschmidt (1998) studied the mismatch negativity (MMN) in response to speech and tone stimuli. The MMN is a negative component of event related potentials (ERP), which is elicited automatically (i.e., without active participation of the listener) about 100 to 300 ms after the presentation of unexpected stimuli. Compared to the control sample, dyslectic children showed an attenuated MMN in response to speech, but not to tone stimuli. According to Schulte-Körne et al. (1998), these results disprove the hypothesis of general auditory deficits in dyslexics and instead indicate a pre-attentive speech perception dysfunction. Kujala et al. (2000) used the same paradigm in order to measure the neural activity in pre-attentive auditory stimulus discrimination. They presented pairs and sequences of tones with deviant stimuli differing from the standard-stimuli only in the length of the ISIs. In accordance with Schulte-Körne et al. (1998), there was no difference in the MMN in response to the pairs of tones between dyslectic and normal adults. The MMN in response to sequences, however, revealed a major difference: In the control sample, the deviant tone sequences elicited two consecutive MMN, while the dyslexic subjects totally lacked the first MMN. This finding again supports the hypothesis of a basic nonlinguistic auditory-information processing deficit in dyslexic individuals.

Other researchers (e.g., Mody, Studdert-Kennedy, & Brady, 1997; Rosen & Manganari, 2001; Share, Jorm, MacLean, & Matthews, 2002) found conflicting results. Their studies are consistent with a speech-specific but not with a general auditory deficit. Rosen and Manganari (2001) underline “that an auditory deficit is neither necessary nor sufficient to cause dyslexia. This, of course, does not mean, that a general auditory deficit could not be a contributing factor for dyslexia in a subset of children”. By contrast, Wolff (2002) observed inordinate difficulty of dyslexics in reproducing simple rhythms by finger tapping. They also showed difficulties in reproducing a rhythmic order of nonsense syllables—especially when they had to synchronize their performance to an external metronome. These results point to an
even more extended deficit in temporal perception processing. An alternative explanation for the heterogeneous results may be the lack of selective attention to visual and auditory stimuli (Facoetti et al., 2003; Hari & Renvall, 2001).

Interestingly, hemispherical differences in the ERPs following linguistic and non-linguistic auditory stimuli have been found for newborns with and without risks for developmental dyslexia. For example, Molefese (2000) reported differences in the automatic processing of acoustic stimuli, which were already present at birth. In a longitudinal study, he examined the ERPs of 186 newborns evoked by linguistic and non-linguistic stimuli and repeated his investigation annually. At the age of eight, 17 children turned out to be developmental dyslexics, and 7 had a general reading disability. Using six different measures of peak latency and baseline to peak amplitudes of the ERPs recorded at birth, he was able to calculate a discriminant function that correctly predicted reading and spelling problems at the age of eight. The function identified 22 of the 24 dyslectic or poor reading children, whereas 19 of 24 randomly chosen children were rejected. If this approach turns out to be reliable in prospective studies, the risk for developmental dyslexia could be diagnosed at a very early age with a better chance of effective intervention and remediation.

In line with this study, Guttorm, Leppänen, Richardson and Lyytinen (2001) reported differences between newborns with and without familial risk for dyslexia in mean event-related brain responses to various consonant-vowel-syllables. Clear hemispherical differences were seen in responses to the syllable /ga/. The high-risk group showed stronger positivation in the right hemisphere at latencies between 50 and 170 ms, which possibly reflects diminished desynchronisation of brain activity. Rippon and Brunswick (2000) as well observed a general absence of task-related desynchronisation and a stronger right-hemispherical activation that characterised the EEG of dyslectic children at the age of ten during a phonological processing task.

The effects that a magnocellular defect may exert on auditory perception processing have not yet been clarified. This is, among other reasons, due to the fact that the auditory system has received very little investigative attention in comparison to the visual system (Kolb & Wishaw, 1993, p. 66). According to Tallal and colleagues (Tallal et al., 1993; Tallal, Miller, Jenkins, & Merzenich, 1997), deficits of the magnocells, in the end, may lead to an impaired ability to segment and discriminate speech sounds. This in turn may distort the development of phonological and orthographic representations that are essential for the acquisition of reading and spelling.

CONCLUSION

The majority of the mentioned studies are consistent with the hypothesis of the occurrence of developmental dyslexia in connection with a general deficit of information processing. A conceivable explanation for conflicting results could possibly lie in the confounding of different subgroups.

In summary, the evidence for a relation between unusual asymmetries of the temporal plana and dyslexia appears to be very modest. By contrast, a growing body of evidence suggests that developmental dyslexia might arise from impaired information processing that is based on neural deficiencies. However, whether the information processing deficit is
restricted to phonology or a general deficit of rapid information processing, is yet to be resolved.

REFERENCES


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