

# Cognitive subtypes of dyslexia

Stefan Heim<sup>1,2\*</sup>, Julia Tschierse<sup>3</sup>, Katrin Amunts<sup>1,2,4</sup>, Marcus Wilms<sup>1</sup>, Simone Vossel<sup>1,2</sup>, Klaus Willmes<sup>5</sup>, Anna Grabowska<sup>6</sup>, and Walter Huber<sup>3</sup>

<sup>1</sup>Research Centre Jülich, Institute of Neurosciences and Biophysics, Jülich, Germany; <sup>2</sup>Brain Imaging Center West (BICW), Jülich, Germany, \*Email: s.heim@fz-juelich.de; <sup>3</sup>Department of Neurolinguistics, RWTH Aachen, Germany; <sup>4</sup>Department of Psychiatry and Psychotherapy, RWTH Aachen, Germany; <sup>5</sup>Department of Neuropsychology, RWTH Aachen, Germany; <sup>6</sup>Nencki Institute of Experimental Biology, Warsaw, Poland

Different theories conceptualise dyslexia as either a phonological, attentional, auditory, magnocellular, or automatisation deficit. Such heterogeneity suggests the existence of yet unrecognised subtypes of dyslexics suffering from distinguishable deficits. The purpose of the study was to identify cognitive subtypes of dyslexia. Out of 642 children screened for reading ability 49 dyslexics and 48 controls were tested for phonological awareness, auditory discrimination, motion detection, visual attention, and rhythm imitation. A combined cluster and discriminant analysis approach revealed three clusters of dyslexics with different cognitive deficits. Compared to reading-unimpaired children cluster no. 1 had worse phonological awareness; cluster no. 2 had higher attentional costs; cluster no. 3 performed worse in the phonological, auditory, and magnocellular tasks. These results indicate that dyslexia may result from distinct cognitive impairments. As a consequence, prevention and remediation programmes should be specifically targeted for the individual child's deficit pattern.

Key words: cluster, reading, attention, phonology, automatization, magnocellular, auditory, diagnostics

#### **INTRODUCTION**

Developmental dyslexia is the disability to learn and perform reading sufficiently in spite of average or above-average intelligence and adequate education. Up to 17.5% of all children are affected (Shaywitz 1998). The cognitive mechanisms underlying dyslexia are still a matter of debate. Numerous theoretical approaches have identified different potential causes of dyslexia.

The phonological theory (e.g. Liberman 1973, Snowling 2000) which is the most influential account for reading problems relates dyslexia to a deficit in phonological awareness, i.e. the ability to segregate and manipulate the speech sounds that form a word (e.g., deleting the first sound from "pearl" gives "earl").

In contrast, the auditory processing deficit theory

Correspondence should be addressed to S. Heim, Email: s.heim@fz-juelich.de

Received 12 November 2007, accepted 28 November 2007

(e.g. Tallal 1980) assumes that dyslexics have a deficit in (rapid) auditory processing. It is argued that due to this more basic deficit no adequate phonological representations can be built, resulting in additional phonological impairments. Thus, according to this theory, phonological problems are only secondary to the auditory deficits.

Yet other researchers conceptualise dyslexia as a visual processing deficit arising from the impairment of the visual magnocellular system in the brain (e.g. Stein and Walsh 1997). This system supports the processing of rapidly moving visual stimuli and is thus important for vision during saccadic eye movements. Dysfunction of the magnocellular system is supposed to result in blurred visual representations of e.g., letters which, as a consequence, are more difficult to distinguish.

The role of attentional deficits for the development of dyslexia is also discussed (e.g. Facoetti et al. 2001, 2003, Hari and Renvall 2001). Attentional deficits are thought to interfere with the encoding of a sequence of letters, resulting in the confusion of letters and visual

word forms. Interestingly, attentional deficits can be dissociated from phonological deficits, and both types of deficits are valid predictors for reading (dis)ability (Valdois et al. 2003).

Finally, the cerebellar theory (e.g. Nicolson et al. 1999, 2001, Nicolson and Fawcett 2005) argues that reading disabilities are a consequence of the impaired ability to automatise processes. It is assumed that the cerebellum supports the automatisation of basic articulatory and auditory abilities which are relevant for the grapheme-phoneme correspondence. Moreover, the control of eye-movements during reading is controlled (among others) by the cerebellum.

The latter four theories (auditory, visual-magnocellular, attentional, and cerebellar/automatisation) have been subsumed in the general magnocellular theory of dyslexia (Stein 2001), discussing impairment of the magnocellular system as the common cause for isolated or combined cognitive deficits resulting in reading disability. The general magnocellular theory regards phonological impairments as secondary to the other deficits. In contrast, the neurobiological model by Ramus (2004) assumes microlesions to the perisylvian cortex (mainly affecting phonological processing) as the primary cause of dyslexia. These lesions may but need not extend further into sub-cortical structures, additionally impairing other (e.g., magnocellular) cognitive functions.

All of these theories have seen supporting empirical evidence (for reviews see e.g. Ramus 2003, Démonet et al. 2004). Interestingly, however, not all dyslexics suffer from deficits in all cognitive domains or profit equally from all remediation techniques (Ramus 2003). Thus, it is possible that distinguishable phenotypes of dyslexia exist on the cognitive level (cf. Morris et al. 1998, Démonet et al. 2004, Ho et al. 2004, 2007, Ramus 2004, Lachmann et al. 2005) for which universal or distinct genetic (Olson 2002, Schulte-Körne et al. 2007) and neurobiological (Paulesu et al. 2001, Ramus 2004) causes are controversially discussed. Unravelling different subtypes of dyslexia would be an essential prerequisite for developing or applying specifically targeted and thus more efficient remediation strategies (Rüsseler 2006). These might even be administered to pre-school children (Démonet et al. 2004) before reading instruction begins.

The present study aimed at investigating subtypes of dyslexics that have specific and distinguishable deficits in one or more of the five cognitive domains, i.e., phonological awareness, auditory processing, visual-magnocellular processing, attention, and automatisation. Moreover, the relationship of phonological processing and the other cognitive abilities subsumed in the general magnocellular theory were investigated in order to obtain evidence for the evaluation of current neuro-cognitive models of dyslexia (e.g., Stein 2001, Ramus 2004).

#### **METHODS**

### **Participants**

In order to acquire volunteers for the study we sent a short project description to the headmasters and headmistresses of 40 primary schools in Aachen, Germany. Out of these 21 agreed to cooperate. In a next step detailed project descriptions containing information about the procedures of the study and an informed consent sheet created according to the Declaration of Helsinki (World Medical Association, 2000) were sent to the schools and forwarded by the teachers to the parents of their 3<sup>rd</sup> grade pupils. Six hundreds and forty-two parents agreed to let their children participate. One hundred and four of these children were later selected for further examination according to criteria listed below, from which 97 complete data sets were obtained and analysed.

#### **Procedure**

Testing was performed in two successive phases. First, the reading abilities were assessed in the schools as group tests. Second, children with normal and with deficient reading scores were further tested individually during or after school in a classroom or at home for their non-verbal intelligence, phonological awareness, auditory sound discrimination, automatisation, magnocellular functions, and visual attention. The non-verbal intelligence test was always administered first since the value was used as an independent variable for the inclusion or exclusion of the subject into the group of dyslexics or controls. The other tests providing the dependent variables of the study were administered in a pseudo-randomised order. The order of the tests was balanced over subjects to exclude systematic influences of sequence. All tests were administered by one co-author (JT) in the last third of the term. The tests

and paradigms employed for the assessment of the cognitive functions were standardised psychometric tests for which norms were available, or computerised tests involving well-established and previously published paradigms that validly tap the processes under investigation.

#### Psychometric tests and questionnaires

Reading ability

Reading ability was assessed with the Würzburger Silent Reading Test (Würzburger Leise Leseprobe, WLLP) (Küspert and Schneider 1998), a standard German test for reading speed. Participants read words at the beginning of a row and mark with a pencil the one out of four pictures displayed in the same row that is denoted by the word. The names of the three distractor pictures may be semantically related, phonologically related, or semantically and phonologically related to the target word (e.g., "Blatt" [leaf]: BLATT [target], BETT [bed – phonologically related], AST [branch – semantically related], BAUM [tree – semantically and phonologically related]). Norms (of 1997) for boys and girls in the first, second, third, and fourth grade are available in the test manual (Kuspert and Schneider 1998). According to the research criteria of the International Classification of Diseases (World Health Organisation 2006) children performing at, or below the 10th percentile were considered as dyslexic if the subsequent non-verbal intelligence test yielded an average or above-average IQ. Children performing above the 25th percentile and with comparable IQ were included into the control group.

# Non-verbal intelligence

The non-verbal IQ was assessed with the German version of the Cattell Culture Fair Test 20 (CFT 20) (Weiß 1998). In this test participants are shown series of pictures generated according to a particular logic. In subtest 1 "Series" subjects have to complete a series of pictures (e.g., white squares containing increasingly long black bars) by marking the one out of five alternatives that is the correct continuation. Subtest 2 "Classifications" requires the participants to indicate which out of five pictures had not been generated after the same principle as the others (e.g., a white square with a vertical black bar among four white squares

with horizontal black bars). In subtest 3 "Matrices" participants have to identify which out of five alternative pictures completes a set of three pictures (e.g., find a white square with two horizontally oriented black circles). Subtest 4 "Topologies" is supposed to assess logical reasoning. Subjects have to identify the one out of four alternatives that was created according to the same principle as a sample stimulus (e.g., where a dot can be inserted in a circle without placing it in a square). The performance in each subtest was assessed as the number of items that were correctly marked in a pre-defined time period (4 minutes for subtests 1 and 2, and 3 minutes for subtests 3 and 4). The CFT-20 was administered individually in its short form (Part 1) for reasons of time economy. Norms for the short form are available for different age-groups (8;7–70 years), grades (5th–10th), or school types (primary school grade 3 and 4; vocational school years 1 and 2) as IQ scores, T values, and percentiles. In the present study age-related IQ scores were used. Children were only included in the study if they had at least average non-verbal intelligence (IQ  $\geq$  85) in order to ensure that no children with learning disabilities participated.

# Phonological awerness

Phonological awareness, i.e. the ability to segregate and manipulate phonemes from given words, was tested with the German "Basic Competences for Reading and Writing" ("Basiskompetenzen für Lese-Rechtschreibleistungen" (BAKO 1-4) (Stock et al. 2003). From the seven subtests included in the BAKO, one productive (Test 4: Phoneme Exchange) and one receptive test (Test 6: Vowel Length Discrimination) were selected, for which norms (of 2002; T values and percentiles) for grades 1-4 are available in the test manual (Stock et al. 2003). In the productive test subjects heard spoken stimulus words played from a CD and had to utter the pseudo-word that results from exchanging the first two phonemes (e.g., Masse → "amsse"). In the receptive test they had to identify the one out of four auditorily presented pseudo-words in which the vowel was of a different length (e.g., maar – raas - DACK - laat). Since the scores of both tests were highly correlated (r=0.41; P<0.001), the average T value was calculated for each child, which entered the further analyses as the measure for phonological awareness.

#### Auditory sound discrimination

Auditory sound discrimination was assessed with subtest 1 of the Heidelberg Sound Discrimination Test (*Heidelberger Lautdifferenzierungstest*, H-LAD) (Brunner et al. 1998).The children indicated if minimal sound pairs (e.g., /ba/ – /pa/) were identical or different. Stimulus materials included both non-lexicalised syllables (Test 1B) and words (Tests 1A and 1C). The stimuli were played from a CD at 60 dB. The H-LAD provides norms (*T* values and percentiles) for the 2<sup>nd</sup> and 4<sup>th</sup> grade. Since the test was administered to 3<sup>rd</sup>-graders in the last term of the school year, the *T* values for the 4<sup>th</sup> grade were calculated.

#### Questionnaire "FBB-HKS"

The "Assessment Scale for Hyperkinetic Disorders" ("Fremdbeurteilungsbogen für hyperkinetische Störungen", FBB-HKS) is a questionnaire for the assessment of attentional deficit and hyperactivity disorder. It is part of the "Diagnostic Inventory for Psychological Disorders in Children and Adolescents according to ICD-10 and ("Diagnostik-System für Psychische Störungen im Kindes- und Jugendalter nach ICD-10 und DSM-IV", DSYPS-KJ) (Döpfner and Lehmkuhl 2000). Separate norms for boys and girls are available (Brühl et al. 2000) for the three symptom categories "attentional deficit", "hyperactivity", and "impulsiveness". In the present study only the ratings for the attentional deficit syndrome (ADS) were considered because these might be related to reading performance. Children classified as having comorbid ADS symptoms were not excluded from the sample. Instead, the number of children with additional ADS was included into the analysis in order to test if ADS symptoms had a substantial influence on the performance.

#### **Computerised tests**

The presentation of the stimuli during the computerised tests and the registration of the button presses were performed with PresentationTM software (Version 0.70; Neurobehavioral Systems, Albany, CA, USA).

#### Automatisation

For the assessment of the automatisation abilities a rhythm imitation paradigm was chosen (Tiffin-Richards

et al. 2001). In this paradigm the children had to click a button synchronously to an auditorily presented rhythm consisting of 5-6 beats with stimulus-onset asynchronies of 300 or 600 ms. For the present study we chose the complex rhythms no. 1, no. 4, and no. 5 from Tiffin-Richards and others (2001) which had best differentiated between dyslexic and control children in that study. Each rhythm was first presented once in order to familiarise the child with it. Then, each rhythm was repeated five times while the subjects had to imitate it by synchronously clicking the left mouse button with the right index finger. The total number of correct rhythms was used as the indicator for automatisation ability. Only those rhythms were judged as correct for which (1) the number of mouse clicks equalled the number of beats and (2) a required click was made before the next beat was played.

# Magnocellular functions

Wilms and coauthors (2005) recently presented a visual paradigm that activated area V5/MT+ as part of the magnocellular system in a functional magnetic resonance imaging study. In this paradigm the participants are presented with a radially expanding, static, or contracting random dot pattern which is well controlled for visual properties (for details see Wilms et al. 2005). It has been demonstrated that such moving stimuli are processed differently by dyslexics and by normal controls: Dyslexics have attenuated motion-onset related visual evoked potentials (Schulte-Körne et al. 2004). Therefore, in the present study the paradigm of Wilms and colleagues (2005) was adopted. The random dot pattern changed its type of motion (e.g., expand  $\rightarrow$  static, expand  $\rightarrow$  contract, static → expand, etc.) after a variable time interval of 1 s, 1.5 s, 2 s, 2.5 s, or 3 s. The time intervals and motion direction changes were pseudo-randomised. The transition probabilities were equal for all types of motion changes. The participants had to indicate the changes in motion by clicking the left mouse button as quickly as possible. The average reaction times for correct responses were taken as measures for magnocellular functioning.

#### Visual attention

In the Posner paradigm (Posner 1980) subjects had to indicate by clicking the left or right mouse button as quickly as possible at which of two positions in the left and right periphery of the computer screen a target stimulus occurred. A target stimulus could be preceded by

a cue which was either informative ("valid"), neutral, or misleading ("invalid"). In the case of a valid cue, the participant could correctly prepare the reaction. In the neutral condition, he or she was alerted that a stimulus would be presented soon but could not prepare a left or right button click. In the invalid condition attention to the shadowed position had to be redirected to the correct position of the target in order to perform the required reaction. The "cue validity effect", which is the reaction time difference between invalid and valid trials, is taken to reflect how quickly attention can be shifted to a new location. Smaller effects indicate better performance.

The Posner paradigm has been successfully applied for the identification of attentional deficits in dyslexic children (Heiervang and Hughdahl 2003). In the present study we used a version of the Posner paradigm that was previously applied by Vossel and coworkers (2006) with central cues and a ratio of 80:20 for valid vs. invalid cues. Only correct trials were analysed. The average cue validity effect was calculated for each participant and entered in the subsequent analyses.

#### Data analysis

In all analyses only subjects that had complete data sets, i.e. values for all five tested cognitive functions, were considered (n=97). All analyses were conducted with SPSS 12.0.1 for Windows (SPSS Inc., Chicago, Illinois, USA).

#### Multiple regression for reading

In order to understand the overall impact of the tested cognitive functions on the reading ability, a multiple regression analysis was performed for the entire sample.

#### Two-step cluster analysis

The existence of sub-types within the dyslexic sample was tested with a two-step cluster analysis which provides the optimum number of clusters in a given data set. The analysis was run allowing for a maximum of 15 clusters, log-likelihood distance estimation, Akaike's information criterion as clustering criterion, no noisehandling for outlier treatment, initial distance change threshold of 0, a maximum of eight branches per leaf node, and a maximum of three tree depth levels. All variables were standardised during the clustering procedure. A Bonferroni-correction was applied.

#### Discriminant analyses

First, a discriminant analysis was conducted in order to assess in respect to which of the five cognitive variables the dyslexics as one homogeneous group differed from the controls. The variables were entered step-wise with an inclusion criterion of  $P \le 0.05$  and an exclusion criterion of  $P \ge 0.10$ . All priors were set equal. Wilks' lambda was calculated for each step. For the analysis of the correct assignment of participants to a particular group the more conservative cross-validated statistics were reported. For the subsequent pair-wise discriminant analyses conducted in order to compare the clusters among themselves and against the control group, the same settings were selected.

#### **RESULTS**

# Description of the sample

Data from 97 children entered the analysis. From these, four children were excluded because no age norms for the H-LAD were available. Among the remaining 93 children (48 girls, 45 boys) there were 45 dyslexics (24 girls, 21 boys) and 48 controls (24 girls, 24 boys). The mean age was 9.3 and 9.2 years for dyslexics and controls, respectively. The average non-verbal IQ in the dyslexic group was 108.4, that in the controls 113.9. The reading score in the dyslexics was below the fifth percentile, whereas that of the controls was below the 63<sup>th</sup> percentile.

#### Reading score and cognitive functions

The multiple regression for the entire sample revealed that phonological awareness and attention were the two significant predictors for the reading score ( $r^2=0.36$ ; phonological awareness:  $\beta$ =0.42; P<0.001; attention:  $\beta = -039$ ; P < 0.001).

#### **Dyslexics vs. controls**

The two groups differed in phonological awareness (Wilks'  $\lambda$ =0.713;  $F_{1.91}$ =36.59; P<0.001) and attention (Wilks'  $\lambda$ =0.578;  $F_{2.90}$ =32.81; P<0.001). 83.7% of the dyslexics (41/49) and 79.2% of the controls (38/48) were correctly assigned to their groups by their scores on these two functions.

#### Clusters of dyslexics

The two-step cluster analysis of the data of the dyslexic sample for all five cognitive functions revealed three distinguishable clusters (Fig. 1). Four subjects for which no age-related T values for auditory sound discrimination were available were excluded. The subsequent discriminant analysis over the identified clusters and the controls supported the clustering by the correct assignment of 15/16 (93.8%), 11/15 (73.3%), and 13/14 (92.9%) dyslexi-

cs to clusters no. 1–3, respectively, yielding an overall correct assignment of 86.7%. The three subgroups of dyslexics and the control group differed significantly (all P<0.001) from one another with respect to sound discrimination (Wilks'  $\lambda$ =0.600;  $F_{3;89}$ =19.77), attention (Wilks'  $\lambda$ =0.373;  $F_{6,176}$ =18.70), phonological awareness (Wilks'  $\lambda$ =0.270;  $F_{9,211.886}$ =16.80), and magnocellular functions (Wilks'  $\lambda$ =0.232;  $F_{12,227.826}$ =14.01). Figure 1 displays the average scores for reading ability and the five cognitive functions separately for each cluster.

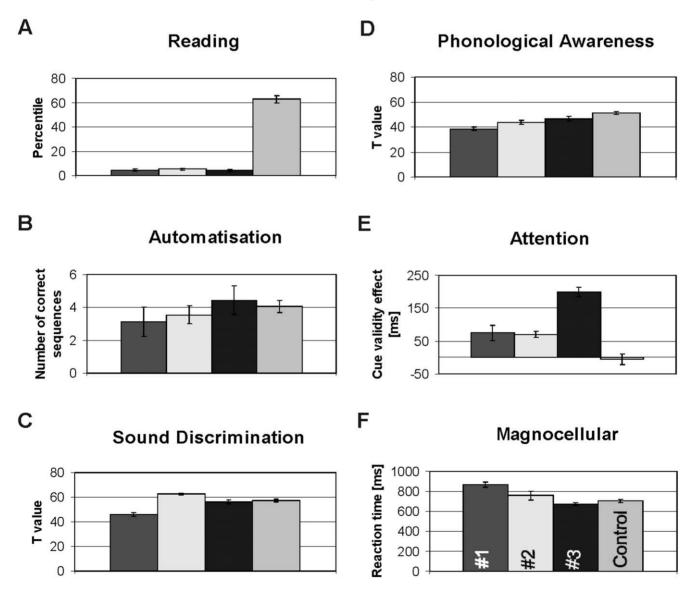


Fig. 1. Description of the dyslexia clusters (columns 1–3) and the controls (column 4). (A) Average percentile ( $\pm$  SEM) of the reading score; (B) total number of correctly imitated rhythms ( $\pm$  SEM); (C) average *T*-value ( $\pm$  SEM) for sound discrimination in the H-LAD; (D) average *T*-value ( $\pm$  SEM) for phonological awareness in the BAKO; (E) average size of the cue validity effect ( $\pm$  SEM) in the Posner paradigm; (F) average reaction time ( $\pm$  SEM) for the detection of motion changes indicative of magnocellular functions.

#### Characterisation of the dyslexia clusters

The absolute cognitive profiles characterising each cluster were identified in pair-wise discriminant analyses of each cluster versus the control group (all P<0.001). Cluster no. 1 performed worse than the control group in the tasks tapping phonological awareness (Wilks'  $\lambda$ =0.551;  $F_{1.62}$ =50.58), sound discrimination (Wilks'  $\lambda$ =0.459;  $F_{2.61}$ =35.97), and magnocellular functions (Wilks'  $\lambda$ =0.423;  $F_{3.60}$ =27.23). Children in cluster no. 2 had lower scores than the controls only in phonological awareness (Wilks'  $\lambda$ =0.791;  $F_{1.61}$ =16.16), whereas sound discrimination was even better than in the controls (Wilks'  $\lambda$ =0.679;  $F_{2.60}$ =14.19). Cluster no. 3 was characterised by a high cue validity effect indicating slow attentional reorienting (Wilks'  $\lambda = 0.587$ ;  $F_{160} = 42.25$ ).

The relative differences between the dyslexics in one cluster compared to each other cluster were also assessed with pair-wise discriminant analyses (all P<0.001). Cluster no. 1 performed worse in sound discrimination and magnocellular functions than both other clusters (cluster no. 2: sound discrimination: Wilks'  $\lambda$ =0.231;  $F_{1.29}$ =96.41; magnocellular: Wilks'  $\lambda$ =0.197;  $F_{2.28}$ =57.02; cluster no. 3: sound discrimination: Wilks'  $\lambda$ =0.190;  $F_{2.27}$ =57.57; magnocellular: Wilks'  $\lambda$ =0.428;  $F_{1,28}$ =37.48). Participants in cluster no. 2 had lower phonological awareness than cluster no. 3 (Wilks'  $\lambda$ =0.206;  $F_{3.25}$ =32.14) but performed better than cluster no. 3 in the attention and sound discrimination tasks (attention: Wilks'  $\lambda$ =0.325;  $F_{1,27}$ =56.20; sound discrimination: Wilks'  $\lambda = 0.264$ ;  $F_{2.26} = 36.28$ ).

#### Analysis of other factors

In addition to the previous analyses, potential gender differences between the dyslexic clusters were analysed. A one-factorial ANOVA yielded no effect of gender ( $F_{3.89} < 1$ ).

Finally, the numbers of children with potential comorbid ADS (according to the FBB-HKS) were identified for each subgroup of children. There were two children in the control group, one child in cluster no. 1, three children in cluster no. 2, and two children in cluster no. 3 who were classified as suffering from ADS. Over the entire sample, the FBB-HKS classification was not significantly correlated with the size of the cue-validity effect (r=-0.178, P=0.120).

#### **DISCUSSION**

We identified three sub-groups of dyslexics with distinct cognitive patterns. Cluster no. 1 performed worse than the controls in the phonological, auditory, and magnocellular tasks. In contrast, cluster no. 2 only had impaired phonological awareness, whereas cluster no. 3 had increased attentional shift costs relative to the controls.

## Distinct cognitive causes for dyslexia

The results reveal that differentiating among dyslexia subtypes with specific impairments allows a more finegrained understanding of the disorder than simply comparing "dyslexics" against controls. This is evident from the fact that the three dyslexic clusters in total differ from the controls in more cognitive functions than the dyslexic group as a whole. Moreover, the correct assignment of the dyslexics improved by differentiating between clusters of dyslexics with distinct cognitive function patterns.

These findings may explain the heterogeneity of results in previous studies (for a review see Ramus 2003) which reported deficits in some but not all of the cognitive functions for some but not all dyslexics. The data from the present cluster analysis approximately reflect the proportions of dyslexics with corresponding deficits reported in the literature (calculated by Ramus 2003, see also Ramus et al. 2003). A magnocellular decrease was observed in 35.6% of the dyslexics (Ramus 2003: 29%). 35.6% of the clustered cases had an auditory deficit that was accompanied by a phonological deficit (Ramus 2003: 39%). In the present study, 33.3% of the dyslexics had an isolated phonological effect (31.3% in Ramus et al. 2003).

# The relationship of phonological awareness and magnocellular functions

These considerations may be important for theoretical accounts for dyslexia. Whereas the neurobiological model by Ramus (2004) considers phonological deficits as primary and magnocellular impairments as secondary causes for dyslexia, the reversed order is assumed in the general magnocellular theory (Stein 2001). In our study bad phonological performance occurred in combination with decreased magnocellular and auditory performance (cluster no. 1), which is in line with both theories. However, cluster no. 2 only showed a phonological deficit, whereas the auditory abilities were even better than in the normal readers. Moreover, no cluster had isolated magnocellular deficits without phonological impairment. This pattern of results would be predicted by the Ramus (2004) model but is not compatible with the general magnocellular account. The data also go beyond the findings by Morris and colleagues (1998) who observed a phonological core deficit for all dyslexic children they examined.

#### No automatisation deficit?

With respect to the automatisation abilities the present data are not unequivocal. The dyslexics in cluster no. 1 showed a trend towards an automatisation deficit, but this result was not significant. No other dyslexia cluster revealed an automatisation deficit, either. This is in contrast to some earlier studies (Fawcett et al. 1996, Tiffin-Richards et al. 2001) that did find impaired automatisation abilities in dyslexics. In particular, we did not replicate the results of Tiffin-Richards and others (2001) from whose study the rhythms were adapted.

However, the findings are in line with the results of Ramus and colleagues (2003) who found no difference between dyslexics and controls in five different cerebellar/automatisation tasks (balance, bimanual finger tapping, repetitive finger tapping, bead threading, finger-to-thumb movement) in a sample equally large to ours. Clearly, this discrepancy between studies that do or do not obtain automatisation deficits in dyslexics requires further investigation.

With respect to the relationship of automatisation and other cognitive causes of dyslexia, it is important to note that the dyslexics in cluster no. 1, who had the worst automatisation scores, were also impaired in three other cognitive functions. This finding might suggest that the automatisation deficit is associated with a larger, multiple cognitive deficit as proposed by the general magnocellular theory (Stein 2001).

#### Attention and reading

In the present study children with a positive ADS classification were not excluded. This comorbidity may present a potential confound in the data. However, the respective analyses carried out in the present study do not support this view. First, the number of children with a positive ADS score was comparable over all clusters. Even more importantly, there were only two ADS children in cluster no. 3 which was characterised by the increased attentional shift costs. This result was further corroborated by the correlation analysis that revealed no significant relationship

between the ADS classification and the performance in the Posner task. Thus, in the present study we found no evidence that the data were significantly influenced by a comorbid ADS. Nonetheless it is possible and potentially interesting to systematically investigate the relationship of dyslexia and ADS in future studies that may also include other tasks tapping different aspects of attention, e.g. vigilance or divided attention.

# Cognitive vs. language-related impairments in dyslexia

The present study was not the first to observe that dyslexics may be classified into subgroups. Earlier work demonstrated that dyslexics differ with respect to their performance in language-related tasks. Morris and coauthors (1998) identified seven clusters of dyslexics that were either globally deficient, impaired in phonological awareness in combination with rapid naming, or showed a deficit in processing rate. Similar results for Chinese were reported by Ho and colleagues (2004) whose subjects were deficient either globally, in an orthographic task, in phonological memory, or rapid naming. King and co-workers (2007) investigated the relationship of phonological awareness and rapid naming in dyslexia. The authors found four groups that had deficits in either phonological awareness or rapid naming, both tasks, or none. Lachmann and others (2005) combined behavioral measures of word and non-word reading with a mismatch negativity (MMN) paradigm. They observed two groups of dyslexics, one impaired in word-reading and the other in non-word reading, with a reduced MMN amplitude in the first relative to the second group.

Taken together, these studies provide good insight into the language-related functions that may be affected jointly or distinctly in dyslexia. In contrast to these findings which only focus on aspects of language processing, a study by Valdois and coauthors (2003) investigated the relationship of attention and phonological awareness during reading, thus considering not only linguistic but also other cognitive influences (i.e., attention). The present data support this finding since in the multiple regression both phonological awareness and attention significantly predict reading performance. Moreover, it was in these two functions that the dyslexic group as a whole differed from the controls. However, the present study goes beyond the approach by Valdois and coleagues (2003) by including a number of cognitive functions such as attention, automatisation, and magnocellular functioning which are all discussed as being

substantial for reading. The data show the merit of this multivariate approach since many of the investigated cognitive functions prove differentially relevant for reading difficulties in dyslexia. The choice of cognitive functions included in the present study was guided by the leading theories of dyslexia but is certainly not exhaustive. Future research may include further aspects such as working memory or rapid naming in order to describe the cognitive basis of dyslexia even more fine-grained.

#### **CONCLUSIONS**

The present study demonstrates that dyslexic children can be classified into different groups with distinct cognitive patterns. There were three clusters that performed worse than the controls either only in the phonological or only in the attention tasks, or differed from the controls in phonological, auditory, and magnocellular scores. As demonstrated above, these findings may account for the heterogeneity of results in the literature concerning cognitive deficits in dyslexics.

More generally, the data reveal that a cluster-analytic approach to multiple cognitive deficits in dyslexia is advantageous. The cognitive functions tested in the present study were selected in accordance with recent cognitive theories of dyslexia and may only be a fraction of all relevant functions. Future studies may use the cluster-analytic approach to include additional cognitive functions such as working memory or lexical retrieval during picture naming. Finally, the results may inspire the identification of dyslexia subtypes on the neurobiological and genetic level.

As a consequence for diagnostics and intervention, a refined view on specific deficits of dyslexic children may motivate the more targeted use of already existing tests and remediation strategies. In particular, pre-school diagnostics before reading instruction may include cognitive functions like the ones investigated in the present study, since none of these functions require the processing of writing. The combination of early diagnostics and deficit-specific intervention may present a significant advance for ameliorating reading problems in children.

#### **ACKNOWLEDGEMENTS**

We wish to thank John Stein, Angela Fawcett, Maria Luisa Lorusso, Andrea Facoetti, Richard Olson, Margaret Snowling, Michel Habib, Piotr Jaskowski, Cecilia Marino, Marco Battaglia, Aryan Van der Leij, Peter de Jong, Heikki Lyytinen, Michel Maziade, and Marcin

Szumowski for discussing some of the basic ideas of this research with us. Moreover, we appreciate the valuable cooperation with Marion Grande, Elisabeth Bay, and Helen Schreiber.

This research was supported by the German Federal Ministry of Education and Research (BMBF 01GJ0613 to SH), the National Institute of Biomedical Imaging and Bioengineering, the National Institute of Neurological Disorders and Stroke, and the National Institute of Mental Health (KA). Further support by Helmholtz-Gemeinschaft and the Brain Imaging Center West (BMBF 01GO0204) is gratefully acknowledged. We thank all primary schools and in particular all parents and children who participated in this study.

#### REFERENCES

Brunner M, Seibert A, Dierks A, Körkel B (1998) Heidelberg Sound Discrimination Test (In German: Heidelberger Lautdifferenzierungstest, H-LAD). Göttingen: Hogrefe.

Brühl B, Döpfner M, Lehmkuhl G (2000) Assessment scale for hyperkinetic disorders (FBB-HKS) - Prevalence of hyperkinetic disorders in parents' ratings and psychometric criteria (In German: Der Fremdbeurteilungsbogen für hyperkinetische Störungen (FBB-HKS) - Prävalenz hyperkinetischer Störungen im Elternurteil und psychometrische Kriterien). Kindheit und Entwicklung 9: 115-125.

Démonet JF, Taylor MJ, Chaix Y (2004) Developmental dyslexia. Lancet 363: 1451-1460.

Döpfner M, Lehmkuhl G (2000) Diagnostic Inventory for Psychological Disorders in Children and Adolescents according to ICD-10 and DSM-IV (In German: Das Diagnostik-System zur Erfassung psychischer Störungen bei Kindern und Jugendlichen, DISYPS-KJ). Huber, Bern.

Facoetti A, Turatto M, Lorusso ML, Mascetti GG (2001) Orienting of visual attention in dyslexia: Evidence for asymmetric hemispheric control of attention. Exp Brain Res 138: 46-53.

Facoetti A, Lorusso ML, Paganoni P, Cattaneo C, Galli R, Umilta C, Mascetti GG (2003) Orienting of visual attention in dyslexia: evidence for asymmetric hemispheric control of attention. Brain Res Cogn Brain Res 16: 185-191.

Fawcett AJ, Nicolson RI, Dean P (1996) Impaired performance in children with dyslexia on a range of cerebellar tasks. Ann Dyslex 46: 259-283.

Hari R, Renvall H (2001) Impaired processing of rapid stimulus sequences in dyslexia. Trends Cogn Sci 5: 525-532.

Heiervang E, Hughdahl K (2003) Impaired visual attention in children with dyslexia. J Learn Disab 36: 68-73.

- Ho CS, Chan DW, Lee SH, Tsang SM, Luan VH (2004) Cognitive profiling and preliminary subtyping in Chinese developmental dyslexia. Cognition 91: 43–75.
- Ho CS, Chan DW, Chung KK, Lee SH, Tsang SM (2007) In search of subtypes of Chinese developmental dyslexia. J Exp Child Psychol 97: 61–83.
- King WM, Giess SA, Lombardina LJ (2007) Subtyping of children with developmental dyslexia via bootstrap aggregated clustering and the gap statistic: comparison with the double-deficit hypothesis. Int J Lang Commun Disord 42: 77–95.
- Küspert P, Schneider W (1998) Würzburger Silent Reading Test (In German: Würzburger Leise Leseprobe). Hogrefe, Göttingen.
- Lachmann T, Berti S, Kujala T, Schröger E (2005) Diagnostic subgroups of developmental dyslexia have different deficits in neural processing of tones and phonemes. IntJ Psychophysiol 56: 105–120.
- Liberman IY (1973) Segmentation of the spoken word. Bull Orton Soc 23: 65–77.
- Lovegrove WJ, Bowling A, Badcock B, Blackwood M (1980) Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. Science 280: 439–440.
- Morris RD, Stuebing KK, Fletcher JM, Shaywitz SE, Lyon GR, Shankweiler DP, Katz L, Francis DJ, Shaywitz BA (1998) Subtypes of reading disability: Variability around a phonological core. J Educ Psychol 90: 347–373.
- Nicolson RI, Fawcett AJ, Berry EL, Jenkins IH, Dean P, Brooks DJ (1999) Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. Lancet 353: 1662–1667.
- Nicolson IR, Fawcett AJ, Dean P (2001) Dyslexia, development and the cerebellum. Trends Neurosci 24: 515.
- Nicolson RI, Fawcett AJ (2005) Developmental dyslexia, learning and the cerebellum. J Neural Transm Suppl 69: 19–36.
- Olson RK (2002) Dyslexia: nature and nurture. Dyslexia 8: 143–159.
- Paulesu E, Démonet JF, Fazio F, McCrory E, Chanoine V, Brunswick N, Cappa SF, Cossu G, Habib M, Frith CF, Frith U (2001) Dyslexia: Cultural diversity and biological unity. Science 291: 2165–2167.
- Posner MI (1980) Orienting of attention. Quart J Exp Psychol 32: 3–25.
- Ramus F (2003) Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction? Curr Op Neurobiol 13: 212–218.
- Ramus F (2004) Neurobiology of dyslexia: a reinterpretation of the data. Trends Neurosci 27: 720–726.
- Ramus F, Rosen S, Dakin SC, Day BL, Castellote JM, White S, Frith U (2003) Theories of developmental dyslexia: insights

- from a multiple case study of dyslexic adults. Brain 126: 841–865.
- Rüsseler J (2006) The neurobiological basis of dyslexia (Neurobiologische Grundlagen der Lese-Rechtschreib-Schwäche). Z Neuropsychol 17: 101–111.
- Schulte-Körne G, Bartling J, Deimel W, Remschmidt H (2004) Motion-onset VEPs in dyslexia. Evidence for visual perceptual deficit. Neuroreport 15: 1075–1078.
- Schulte-Körne G, Ziegler A, Deimel W, Schumacher J, Plume E, Bachmann C, Kleensang A, Propping P, Nöthen MM, Warnke A, Remschmidt H, König IR (2007) Interrelationship and familiality of dyslexia related quantitative measures. Ann Hum Genetics 71: 160–175.
- Shaywitz SE (1998) Dyslexia. New Engl J Med 338: 307–312. Snowling MJ (2000) Dyslexia (2nd ed.). Blackwell, Oxford.
- Stein J, Walsh V (1997) To see but not to read: the magnocellular theory of dyslexia. Trends Neurosci 20: 147–152.
- Stein J (2001) The magnocellular theory of developmental dyslexia. Dyslexia 7: 12–36.
- Stock C, Marx P, Schneider W (2003) Basic Competences for Reading and Writing (In German: BAKO 1-4. Basiskompetenzen für Lese-Rechtschreibleistungen). Beltz, Göttingen.
- Tallal P (1980) Auditory temporal perception, phonics, and reading disability in children. Brain Lang 9: 182–198.
- Tiffin-Richards MC, Hasselhorn M, Richards ML, Banaschweski T, Rothenberger A (2001) Time reproduction in finger tapping tasks by children with attention-deficit hyperactivity disorder and/or dyslexia. Dyslexia 10: 299–315.
- Valdois S, Bosse ML, Ans B, Carbonnel S, Zorman M, David D, Pellat J (2003) Phonological and visual processing deficits can dissociate in developmental dyslexia: Evidence from two case studies. Read Writing 16: 541–572.
- Vossel S, Thiel CM, Fink GR (2006) Cue validity modulates the neural correlates of covert endogenous orienting of attention in parietal and frontal cortex. Neuroimage 32: 1257–1264.
- Weiß RH (1998) Test for Basic Intelligence, Scale 2 (In German: Grundintelligenz Skala 2. CFT 20). Hogrefe, Göttingen.
- Wilms M, Eickhoff SB, Specht K, Amunts K, Shah NJ, Malikovic A, Fink GR (2005) Human V5/MT+: comparison of functional and cytoarchitectonic data. Anat Embryol 210: 485–495.
- World Health Organization (2006) International Classification of Diseases ICD-10 (10th revision) URL: http://www.who.int/classifications/apps/icd/icd10online/.
- World Medical Association (2000) Declaration of Helsinki. Ethical Principles for Medical Research Involving Human Subjects. Edinburgh.