DYSLEXIA – AN OVERVIEW OF ASSESSMENT AND TREATMENT METHODS

Evelin Witruk[1] & Arndt Wilcke[2]

[1] University of Leipzig, Institute of Psychology II
[2] Fraunhofer Institute for Cell Therapy and Immunology, Leipzig
Translational Centre for Regenerative Medicine (TRM), Leipzig

Abstract

This article will give an overview of the different methods of assessment and treatment currently used in the field of dyslexia with a special focus on genetic research. Based on the modification and extension of the multilevel model of Valtin (1989, modified by Witruk, 1993b), assessment and treatment methods will be discussed due to their primary objectives. These methods will be described regarding primary causes (biological risk factors), secondary causes (partial performance deficits), primary symptoms (reading and writing problems) and secondary symptoms (emotional and behavioural disorders).

Keywords: Multilevel model of dyslexia, genetics, magnocellular deficit, partial performance deficit, working memory, complex training programme.

The history of the dyslexia research is controversial and led to many contradictory theories and results up to the present day. It is now just over 110 years since Morgan (1896) first published his famous account of Percy, a dyslexic boy of 14 years. The current state of dyslexia research can be characterised by the distinction of scientists in groups of protagonists of a visual versus a phonological/auditory deficit on the one hand and in groups of protagonists of a low, basic level versus a higher level deficit on the other hand. A lot of contradictory results and theories posed the question about specificity and homogeneity of different deficits in dyslexic individuals. The model of Habib (2000) provides an integration of perceptive and cognitive deficits on the basis of a common temporal processing deficit, which can be analysed on a low, basic level and/or on a higher complex level of performance. The individual combination of partial deficits on the low and the higher level produces the specificity of the symptoms.

Dyslexia is defined by the World Health Organisation (WHO) as a specific and significant impairment in the acquisition of reading often connected with a disorder in acquisition of writing. These disorders appear in the presence of a normal or above-average intelligence. During the last century, hundreds of scientists searched for the specific sources of these disabilities. A lot of contradictory results posed the question of about the specificity of dyslexia and specificity of deficits in its subtypes. The different prevalence rates of dyslexia in the world vary from 1% in Scandinavian countries, 2% around the region of Beijing, 3-5% in Germany, 8-10% in UK and USA. The relation of boys to girls is about 4:1.

The multilevel model of dyslexia

The multilevel model of dyslexia calls for two causal and two symptomatic levels which are superimposed in time, with one flowing from and having repercussions on the others (cf. Table 1).

On the level of primary causes, it is assumed that there exist biological risk factors interacting with environmental stressors and believed to express themselves in functional and structural neuroanatomical characteristics of dyslexic individuals. The intervention on this level can aim at the compensation or at the restoration of these biological risk factors.

On the one hand, the secondary causes that grow out of the primary ones refer to partial performance deficits in the fields of visual and auditory perception, motor patterns and long-term and working memory. Here, the treatment involves a functional training, which is highly selected in the main imported deficit function and assumes a generalisation and stabilisation of the complex action system of reading and writing. In principle, the cause levels can be identified even before such children start formal education.

On the other hand, primary symptoms can be detected only in the specificity of failures in reading and orthography, for example on the basis of error, time and eye movement analyses. In time, these latent failures and the responses from these children`s environment lead to a vicious circle of secondary symptoms made up of the four stages of anxiety, blocking, avoidance, compensation and lowering of motivation, as described by Betz and Breuninger (1982). These effects underscore the existential observational relevance of written language and the consequences of its impairments. Secondary symptoms may have repercussions on primary symptoms and on such causes as destabilisation and blocking, though there has hardly been any research on this yet.

Thus, the psychopathology of dyslexia provides some clues about possible working memory deficits and the way they must be integrated into a person`s overall pattern of disability. Let us now examine, with the help of a demand-related information processing approach, the relevancy of working memory to the regulation of our behaviour.

Assessment of dyslexia

1. Assessment methods regarding primary causes

In the case of dyslexia, the debate between nature or nurture as possible primary causes had been clarified using

Table 1

Multilevel model (developed by Valtin, 1989, modified by Witruk, 1993b).

		Assessment	Treatment
Causes	*Primary*	Biological risk factors	Compensatory training
	Secondary	Partial performance deficit	Training of basic functions
Symptoms	*Primary*	Reading and writing tests	Rehabilitative exercises of reading and writing
	Secondary	Personality questionnaire, observation	Complex training, Psychotherapy

family and twin studies. With help of these studies it was possible to identify a strong hereditary (i.e. genetic) component of this disease.

In the following section it is assumed that most of the readers of this journal are interested in genetics as the biological basis of dyslexia, but are no experts in the field of genetics. Therefore, supporting background information is added to enable a complete understanding of the results.

The following subsection represents a summary of the article of Wilcke & Boltze, 2010.

1.1 Genetics of dyslexia

1.1.1 Family studies

First of all, one has to decide if a disease is mostly caused by genes or if genetic disposition plays only a marginal role in its development. To answer this question, family studies are the method of choice.

In family studies, the proportion of diseased relatives of affected individuals (i.e. dyslexic relatives of dyslexic persons) is compared to the general prevalence in the whole population. This results in λ, a measure to estimate familial aggregation. A $\lambda>2$ indicates a strong hereditary disposition.

$$\lambda = \frac{\text{Percentage of affected relatives}}{\text{Percentage in the whole population (prevalence)}}$$

First studies showed a significant familial aggregation (Hallgren, 1950) that could be replicated in several studies (Finucci et al., 1976; Vogler et al., 1985): more than 40% of the siblings/parents of a dyslexic were dyslexics, too. Given a prevalence of 4%, this leads to a $\lambda=10$, indicating an eminent role of genes in this disease.

Since λ only gives a rough estimate of the relation between environmental influences (nurture) and hereditary disposition (nature) and is influenced by the partially shared environment of family members, in a second step more precise data have to be gained. This is done by twin studies.

1.1.2 Twin studies

Twin studies provide exact data to which extent a disease is caused by genes. These studies are based on the comparison of monozygotic (MZ) and dizygotic twins (DZ), because monozygotic twin are genetically to 100% identical while dizygotic twins share only 50% of their genes.

The genetic proportion of a disease is estimated by the heritability index h^2. It is based on the correlation of the disease in monozygotic twins, r^2_{MZ} and the correlation in dizygotic twins, r^2_{DZ}. Thereby, h^2 can range from 0 (no genetic influence) to 1 (caused completely by genes).

$$h^2 = (r_{MZ} - r_{DZ}) \times 2$$

In dyslexia, large scale twin studies were performed by Stevenson (1991) and Olson et al. (1994), resulting in an average h^2 of 0.6 across different measures of reading and writing. This indicates that 60% of variance in dyslexia is caused by genes.

Once the question if genes had a major influence on dyslexia was solved, attempts were made to identify the involved genes.

1.1.3 Linkage and association studies

Linkage studies

Linkage studies are a way to narrow the genomic region where relevant disease genes are expected. This is necessary because complete sequencing of several genomes is not yet possible for a reasonable

amount of money and was not possible at all, when first linkage analyses were done.

Linkage is the association of genes and/or markers that lie near each other on a chromosome. Linked genes and markers tend to be inherited together.

If several genetic loci are linked, they stay together during transmission to the next generation. This is used in linkage analyses. Due to the linkage of several loci it is sufficient to determine only one genetic locus (by using a specific marker) to get information about the whole region. If the studied region harbours a disease gene, it is always linked to the used marker. So it is possible to screen the whole genome for regions with a limited amount of markers. Those marker variants that appear in most affected people but infrequently in healthy controls are linked with the disease gene sought after.

In dyslexia, first linkage analyses were restricted on the study of single chromosomes. Early studies found hints on chromosomes 15 (Smith et al., 1983) and 6

(Smith, Kimberling & Pennington, 1991). Further studies, especially on chromosome 6, could narrow down the region of interest to 6p21-22, examined in several subsequent association studies (Wilcke & Boltze, 2010). Further promising regions are currently 15q15 (Morris et al., 2000), 15q21 (Nöthen et al., 1999) and 18p11 (Fisher et al., 2002).

Due to improved technical possibilities genome wide scans were made possible in the last decade (Fisher et al., 1999; Kaminen et al., 2003, Igo et al., 2006; an overview is given in Scerri & Schulte-Körne, 2010).

Using linkage analyses, until now at least nine different chromosomal regions could be identified where several disease genes are suspected. Since those regions are connected with dyslexia, they are called DYX regions.

Due to the complex nature of these analyses, linkage studies currently do not provide final results, but provide hints where more detailed association studies might identify disease genes.

Table 2
Overview of DYX regions.

Shown are DYX-regions, their localisation in the genome, possible candidate genes and the number of supporting/non supporting studies. Numbers in brackets indicate a connection found to reading relevant skills (e.g. phonological awareness), but not to reading itself. (Scerri&Schulte-Körne, 2010, mod.)

DYX-region	Chromosomal region	Number of positive studies	Number of negative studies	Candidate gene(s)
DYX1	15q21	6	10	*DYX1C1*
DYX2	6p22.3-p21.3	7	9	*DCDC2; KIAA0319*
DYX3	2p16-p15	4 (+1)	4	*MRPL19; C2ORF3*
DYX4	6q11.2-q12	1	8	-
DYX5	3p12-q13	2 (+1)	5	*ROBO1*
DYX6	18p11.2	3	7	-
DYX7	11p15.5	1 (+1)	7	-
DYX8	1p36-p34	3	9	*KIAA0319L*
DYX9	Xq27.2-q28	1 (+1)	5	-

Association studies

Association studies focus on genes previously identified in linkage studies as potential candidates. While linkage analyses derive their data from analysis of families, association studies compare different populations, i.e. affected people vs. controls. It is tested if a certain genetic variant appears more often in affected people than in healthy controls.

One of the most abundant variants of genetic variation are SNPs. A SNP (Single Nucleotide Polymorphism) means that a single base at a certain position in the genome is different in some individuals, and that these individuals comprise at least 1% of the population. A SNP can be neutral, i.e. it has no consequences, or it can lead to different gene products or to a change in gene expression. One gene often has a large number of SNPs. Theoretically it would be possible to test every SNP for association. Fortunately, and in some respects similarly to linkage analyses, correlation of SNPs can be exploited as adjacent SNPs are mostly inherited together. This phenomenon is called "linkage disequilibrium". So it is sufficient to study only some SNPs of a gene to cover its whole variance (Figure 1).
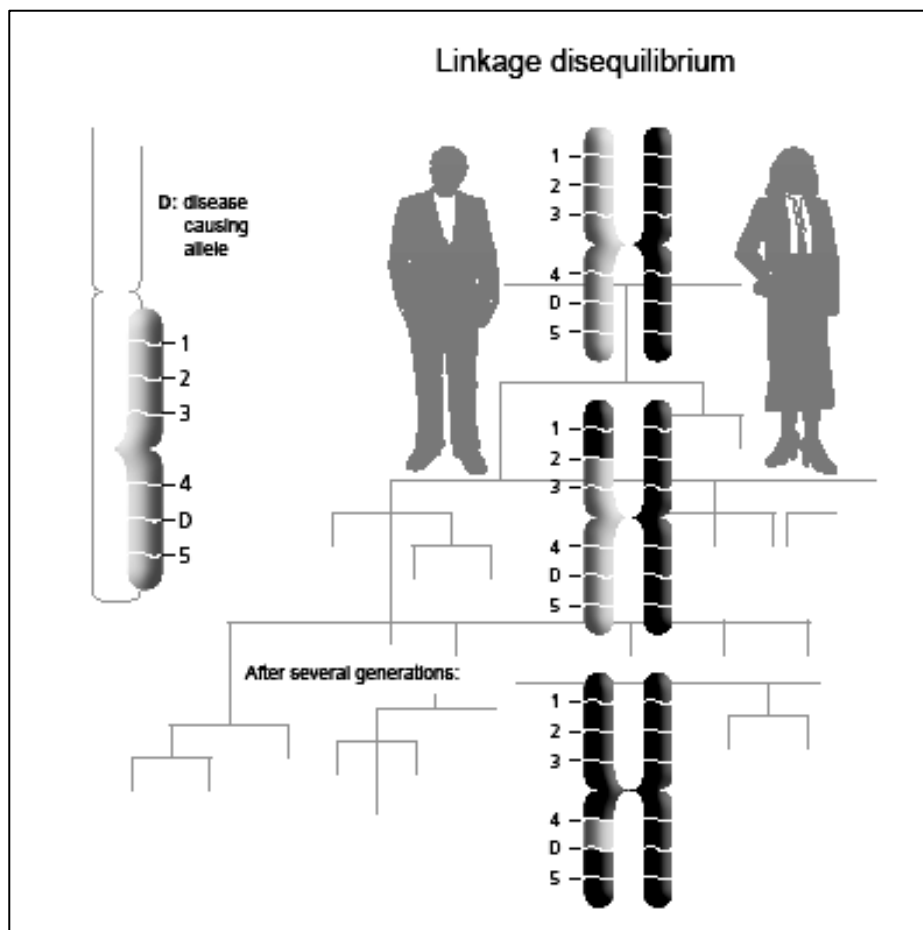
Linkage disequilibrium exists only in a narrow chromosomal region. Above: Linkage between all SNPs (1-5+D). This represents the condition in linkage analyses. Below: Only two SNPs (4 and D) are in linkage. There is a linkage disequilibrium between these two SNPs. This represents the condition in association analyses.

Figure 1. Linkage disequilibrium.

One important point in dyslexia is the complexity of this disease. Complexity exists in respect to the phenotype and the genotype, i.e. dyslexia is not caused by one or two genes with tremendous influence on disease development, but by many genes with little influence per gene. So it is possible that one person has some genetic risk variants and some protective variants that compensate each other. Depending on the number and type of genetic risk variants, a mild, moderate or severe type of dyslexia is developed (Figure 2).

In dyslexia first association studies concentrated on chromosomal region 6p22.1 (DYX2), the region best replicated in several linkage studies. Cope et al. (2005) identified a gene in this region that was found to be associated with dyslexia: KIAA0319. Shortly after, a second gene in this region was identified: DCDC2 (Meng et al., 2005; Schuhmacher et al., 2006). Whether there is an interaction between these two genes or not, is still under discussion (Harold et al., 2006). The role of these two genes seems to be moderated by language. While DCDC2 was found to be associated with dyslexia in English as well as German dyslexics (Wilcke et al., 2009), the SNPs associating in KIAA0319 could not be replicated in a German study (Wilcke, 2007).

Further genes currently in the focus of genetic dyslexia research are DYX1C1 in region DYX1 (Taipale et al., 2003), MRPL19 and C2ORF3 in region DYX3 (Anthoni et al., 2007), ROBO1 in DYX5 (Hannula-Jouppi et al., 2005) and KIAA0319L in DYX9 (Couto et al., 2008).
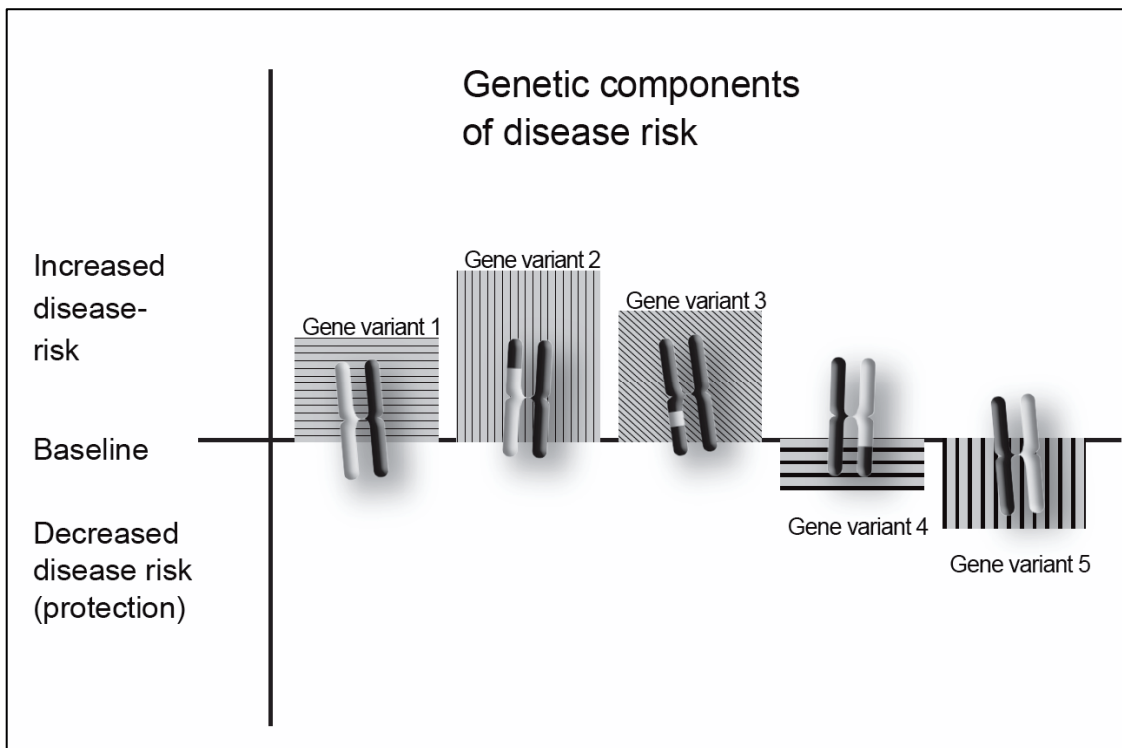
Five gene variants are shown: Three of them (1-3) increase the risk of getting a certain disease, e.g. dyslexia. Two decrease the risk. The final risk is a result of addition/subtraction of all genetic variants

Figure 2. Model for an additive genetic risk.

.

Interestingly, most of the above mentioned genes play a direct or indirect role in neuronal migration, i.e. movement of nerve cells, especially during early brain development. Since not all genes can be discussed here, DCDC2 and KIAA0319 shall serve as examples due to their location within DYX2, the most frequently replicated chromosomal region in dyslexia.

DCDC2 (*Doublecortin Domain Containing 2*) interacts with a protein called doublecortin. It is present during embryonal brain development, whereas no expressed doublecortin is found in adult neurons, illustrating its role during neurogenesis. It bundles and stabilises microtubuli (Francis et al., 1999), a part of the cytoskeleton used for migration.

KIAA0319´s role could not completely elucidated yet. But tests in mice showed a significant reduction in neuronal migration if the expression of KIAA0319 was minimised. It seems to influence neurons during the migration to their final destination along the glia fibers (Figure 3). If KIAA0319 is suppressed, neurons do not bind to the glia fibers, and no migration takes place (Paracchini et al., 2006).

So a central question in dyslexia research is, if there is a connection between genes and altered neuronal structures in dyslexics.

1.2 Neuroanatomical findings in the visual perception of dyslexics

Several studies indicated that dyslexics have a deficit in processing rapid sequences of visual stimuli as well as a low flicker-fusion frequency, i.e. the frequency when a sequence of light flashes appears as continuous light (Stanley & Hall, 1973).
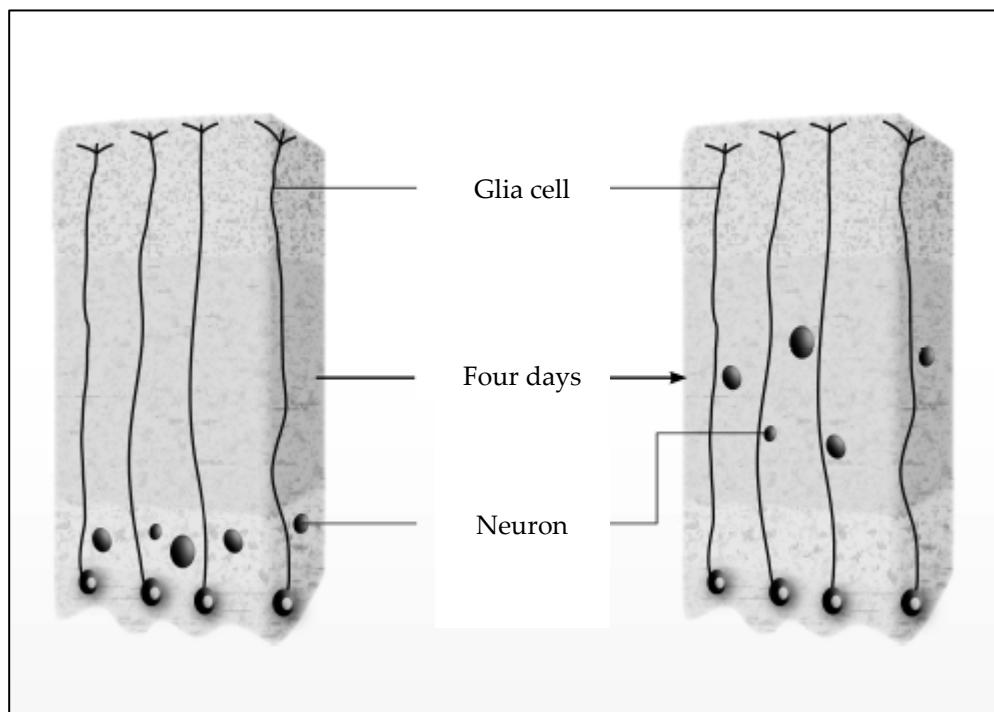
Shown is the migration of neurons along the glia cells. Migration takes place from the ventricular zone across the intermediate zone to the cortical plate along the radial glia fibers.

Figure 3. Radial neuronal migration.

Therefore, anatomical brain studies focused on detailed examination of the visual system, especially the lateral geniculate nucleus (LGN). The LGN has a central role in visual processing because almost all axons of the optical nerve are ending there. Furthermore, information from cortex and thalamus is integrated before it reaches the primary visual cortex.

There are two types of cells in the LGN: magnocells and parvocells. The magnocellular system is responsible for contrast- and movement perception while the parvocellular system is responsible for colour perception and object recognition (Livingstone & Hubel, 1988).

Since the LGN is eminent in visual perception, Livingstone et al. (1991) and Galaburda & Livingstone (1993) studied post mortem brains of dyslexics and found obvious differences in LGN´s structure as well as in the size of the magnocells (Figure 4).

Dyslexics partly showed diminished magnocells with a deviant structure, possibly indicating a disturbed neuronal migration in early development. This poses the question how diminished magnocells could lead to disturbed reading.

Reading is a combination of fixations and saccades. During fixations, the eye rests at a certain point of the text, and form and pattern of this target stimulus are projected to the fovea of the eye. During saccades, the eye moves very fast from point of the text to the next. So reading is no continuous, but a saltatory perception.

During fixations, the parvosystem is active, while during saccades the magnosystem is active. Both systems interact, leading to clearly separated perceptions (Figure 5).

Parvocells

Magnocells

Control   100μm   Dyslexic
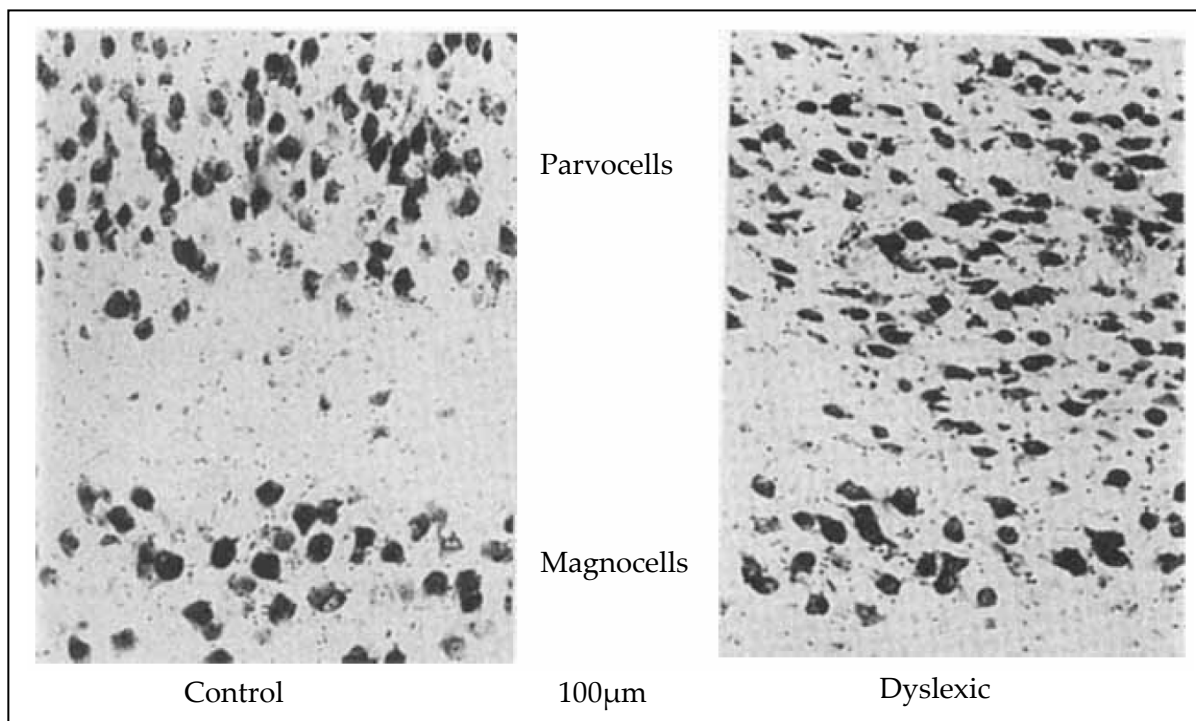
Depicted are magno- and parvocellular layers in the LGN. The dyslexic brain shows a disorganised layer structure and the magnocells are mostly smaller and more variable in size and form than in the control brain.

Figure 4. Comparision of the LGN in dyslexics and controls.
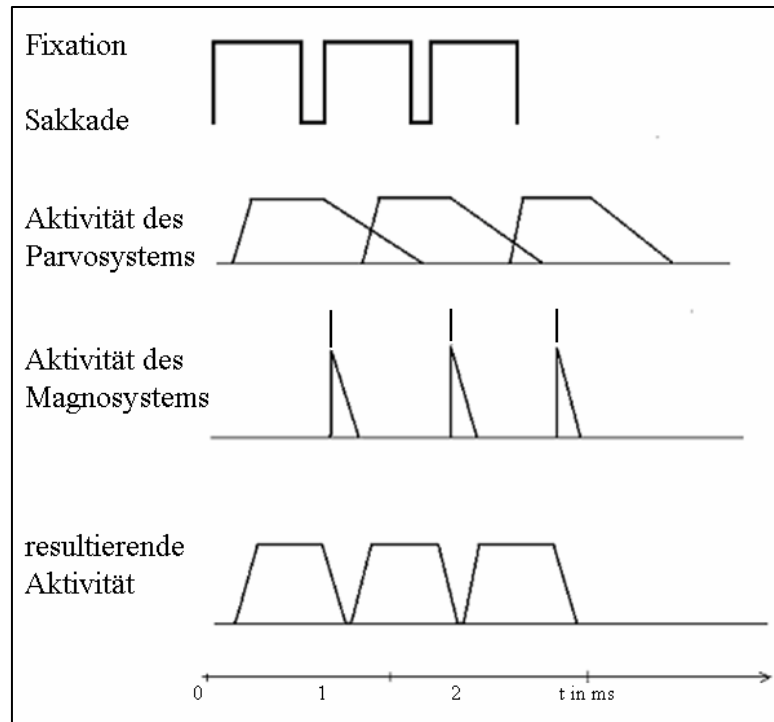
During fixations, information is processed in the parvosystem. The magnosystem processes the spatial orientiation of the actual stimulus to place it correctly on the fovea using a saccade (Lehmkuhle et al., 1993).

Figure 5. Interaction of parvo- and magnosystem.

So reading represents an interaction of mango- and parvocells. Since parvocells have a low line speed, the visual information of fixation A would still be present at the time of fixation B resulting in a blurred perception. This is prohibited by the magnosystem. It surpresses the remaining activity of the parvosystem, resulting in two clear and distinct perceptions.

This function would be affected in the case of diminished or disorganised magnocells. The "Breitmeyer effect" occurs (Breitmeyer, 1980, 1993). During reading, the Breitmeyer effect leads to the following situation: The beginning of a word (first fixation) is clearly visible, but further fixations result in blurred vision – the word is no longer readable (Figure 6). This also affects writing, because the own handwriting is perceived in the same way.
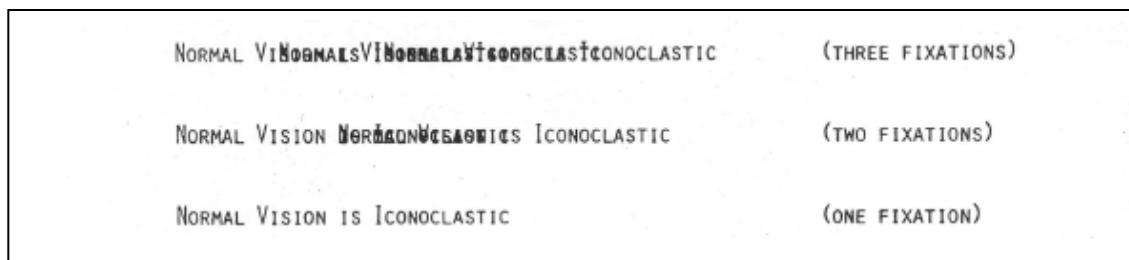
Shown is the overlay effect resulting from an insufficient magnosystem (Breitmeyer, 1980)

Figure 6. The Breitmeyer effect.

Results concerning the magnocellular deficit could be replicated in some studies (Eden et al., 1996; Sperling et al., 2003) while other doubted this effect (Vellutino et al., 2004). An overview of this discussion is given in Nandakumar & Leat (2008).

Given the existence of the Breitmeyer effect this would offer an interesting potential chain of causation from genes to reading: Most of the genes currently associating with dyslexia are involved in neuronal migration. Neuroanatomical studies of dyslexic brains have shown a deviant structure and abnormal cell sizes in an area relevant for reading. And these deviations itself could be caused by disturbed neuronal migration.

2 Assessment regarding secondary causes - Partial performance deficits

2.1 Overview

The secondary causes of dyslexia were proofed in partial deficits in basic functions and not in complex actions of reading and writing. In principle, primary and secondary causes can be identified even before such children start formal education. Intervention and prevention can therefore also start before. Partial deficits can involve auditory and/or visual perception, tactile perception, working memory, long-term memory, motor functions and integration functions (Figure 7).
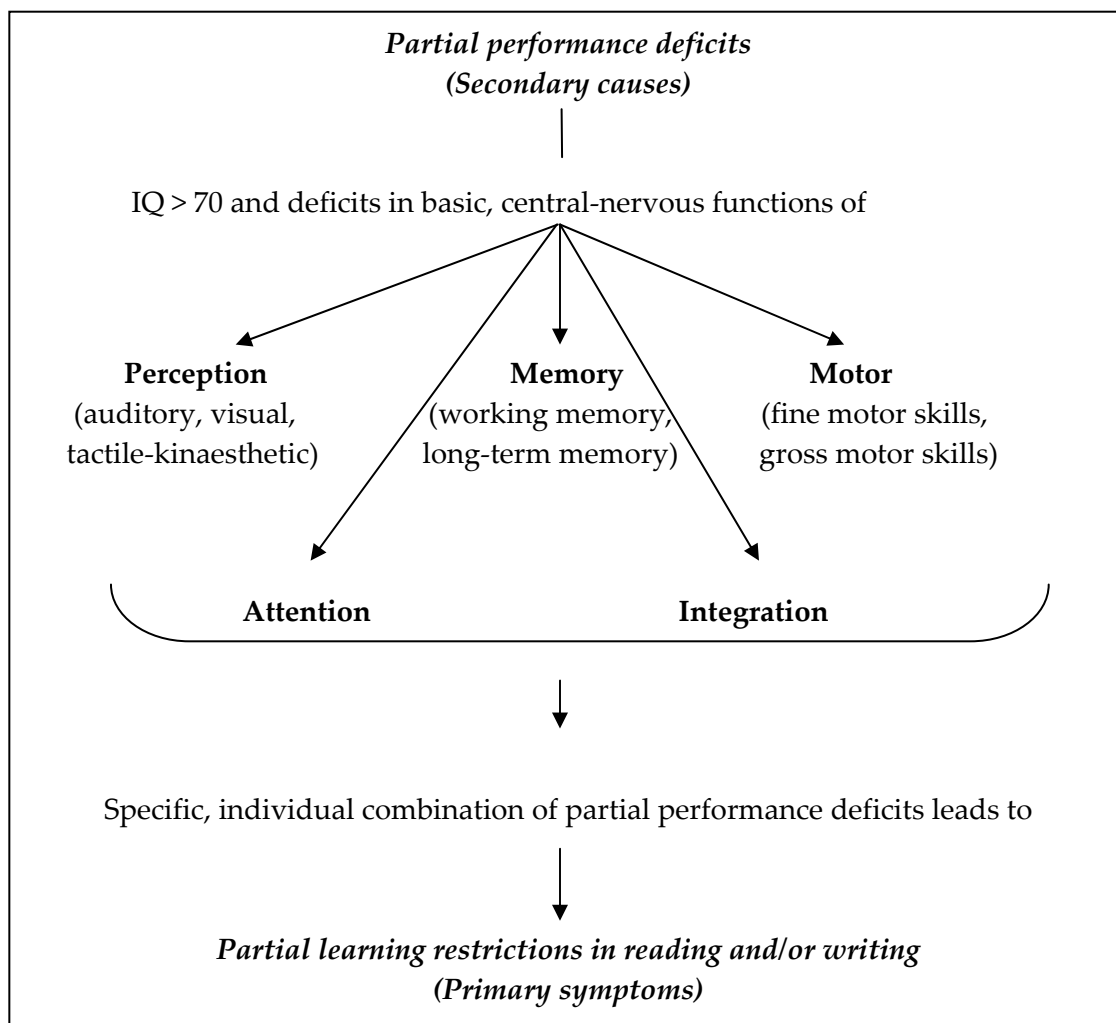

Figure 7. Partial performance deficits.

Partial deficits are related to basic functions, which are preconditions for higher complex activities like language, reading and writing (Figure 8).

The results regarding the deficits of auditory or phonological working memory seem to be present with relatively high consistency (Witruk & Ho, 2010). Deficits of visual working memory appear to depend strongly on the types of material used. Studies in which visual but nameable stimuli were used could be related to phonological decoding and to the phonological loop. The lower automation of Central Executive processes in dyslexics could be verified.

2.2 Working memory in dyslexia

Impairment of working memory performance in dyslexic children and adults has been found for visual and auditory presentation of stimuli with different paradigms and types of material.

2.2.1 Visual working memory in dyslexia

Regarding deficits in visual working memory, several results are available. So and Siegel (1997) found deficits for Chinese poor readers in visual working memory tasks (free visual reproduction of character lists with and without phonological, visual and semantic similarities). Ho and Bryant (1997) have reported that early visual memory skills are predictive of later reading performance in Chinese. Recent findings of Ho and her colleagues (2002, 2004, 2006) also suggest that the major difficulties of Chinese dyslexic children lie in visual-orthographic processing while some dyslexic children have difficulties in visual motion perception.

Ellis (1981) reported four visual matching experiments based on the Posner Paradigm with different material in which he was not able to find deficits for dyslexics if the two stimuli were not nameable. Significant deficits for dyslexics were shown if the visual stimuli were phonologically similar letters. He interpreted these results as naming deficits. Vellutino's findings (1987) also speak against a general deficit of the visual working memory. His dyslexic children were able to reproduce unknown Hebrew words and letters just as well as normal reading children. If the word list was in English, the dyslexic
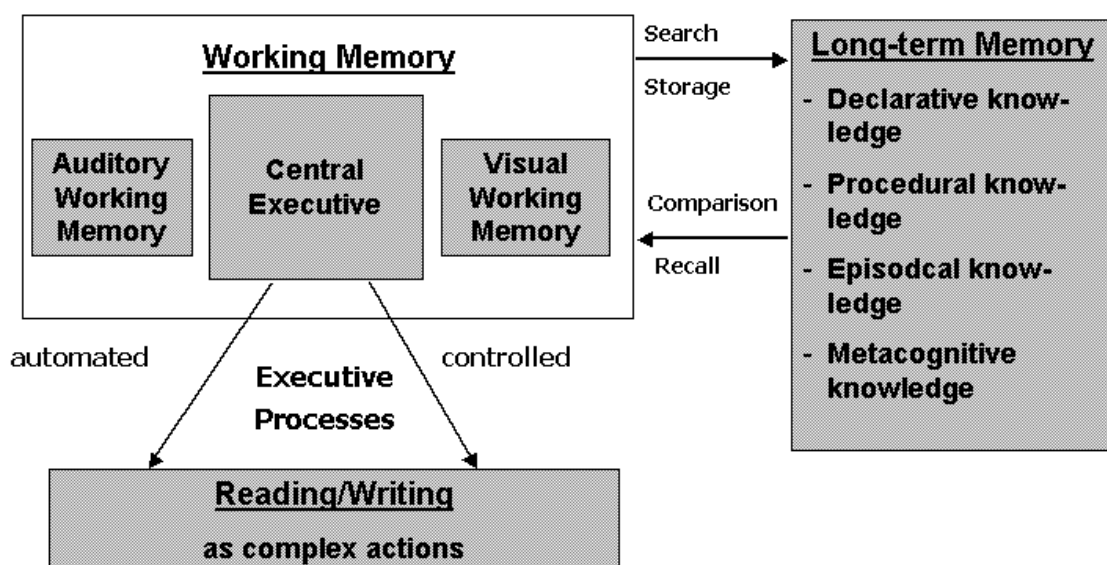

Figure 8. Working memory functions during reading and writing (Witruk, 2003).

children performed significantly poorer than the control group. Vellutino's interpretation refers to a deficit of dyslexics during storage and recall of linguistic information. Likewise Barnea, Lamm, Epstein, and Pratt (1994) mainly found deficits for Hebrew speaking dyslexic children with series of lexical and visual stimuli.

Using visual matching tasks, Willows, Kruk, and Corcos (1993) found deficits of dyslexic children with letters from the - to them unknown - Hebrew alphabet. These deficits in accuracy and speed were stronger in 6-year-old children than in 8-year-olds.

Compensation effects for deficits of visual working memory were shown in a study by Witruk and Rosendahl (1999) for visual matching tasks and visual serial recall tasks. For these visual working memory tasks they found significant adaptations towards the control group in a longitudinal and cross-sectional comparison between 7- and 9-year-old dyslexic children. For visual matching tasks Witruk (1993a, 1999) and Witruk, Ho and Schuster (2002) found a material-specific, nongeneral deficit in dyslexic children. For the accuracy parameter, significantly higher error rates were observed for dyslexic children with letters and dot patterns but not with line patterns.

2.2.2 Auditory working memory in dyslexia

The current discussion explores whether the reading and spelling difficulties of dyslexic children are based on auditory working memory deficits or on specific phonological working memory deficits with linguistic material like phonemes, syllables or words. Some studies show that the dyslexia deficit is based on the auditory field in general and also involves phonology. For example, Lachmann (2007) found

a lower Mismatch Negativity in dyslexic children in comparison to nondyslexic children for linguistic stimuli but also for tone series. Mismatch Negativity represents auditory perception, discrimination, and memory processes on different levels (prior to semantic representations). Measured as a component of an acoustical evoked potential, it represents vast pre-attentional stimulus discrimination and memory comparisons. Auditory working memory deficits for nonlinguistic material were also found by Fischer (2007) for tone pairs, by Helenius, Uutela and Hari (1999) and, Hari and Renvall (2001) for tone series. Schulte-Koerne (2001) found that a smaller value of the Mismatch Negativity occurs in German dyslexic children compared to nondyslexic children for the passive perception, discrimination and memory comparison of verbal stimuli but not for nonverbal, auditory stimuli (sine tones).

Regarding the deficits of phonological working memory, research evidence has been more convergent. The most often used paradigm is the so-called memory span for numbers, words, and pseudowords. Deficits in phonological abilities and of phonological working memory in dyslexic Canadian children are described by Siegel and Linder (1984). Ho, Law and Ng (2000) and Ho and Lai (1999) were able to validate these phonological deficits in Chinese dyslexic children. According to Everatt, Groeger, Smythe, Baalam, Richardson, and McNamara (2001), phonological working memory deficits on sequential information (such as in digit span tasks) could be the root-cause of some other deficits and are evident across child and adult populations.

Gathercole and Baddeley (1993) found delays of development regarding articulation speed, rehearsal of nonwords, and memory span for words in 8-, 11- and 15-year-old dyslexic children. Phonological

deficits which were found in 8- and 11-year-old dyslexic children were not found in 15-year-old dyslexic children. Thus, with regard to phonological working memory, one can call it a later onset in the tendency of compensation. Gathercole, Alloway, Willis and Adams (2006) found that phonological working memory skills represent an important constraint on the acquisition of skills and knowledge in reading and mathematics.

2.2.3 Central Executive functions in dyslexia

Proof of deficits in dyslexics in relation to Central Executive functions were found only in a few investigations. Schneider (2001) reported a stronger activation of the frontal lobe in dyslexic children during mental rotation and sound connecting tasks. She interpreted these results as a stronger involvement of the Central Executive in dyslexic children on the basis of an inefficient automation. The tasks used by Siegel and Ryan (1989) involved executive functions during word recognition after sentence completion and counting. They found generalized working memory deficits in dyslexic children (age 7-13), while children with arithmetic deficits had only a deficit in processing numerical information.

3 Assessment of primary symptoms - Failures in reading and writing

The diagnostic criteria in the ICD 10 are based on the analysis of failures in reading and writing in comparison to a better intelligence. This discrepancy between the IQ and reading and writing performance is the basic assumption. Sometimes this discrepancy is quantified. The typical errors in reading and writing are:
- Loss of letters, word parts, whole words,

- Reversal errors of letters or mirror errors (like "u" and "n", "b" and "d"),
- Adding of letters, word parts or whole words,
- Low reading speed,
- Low level of reading understanding.

For the assessment of the primary symptoms it is necessary:
- to measure the reading performance with a reading test (for example the Zurich Reading Test (ZRT) from Linder and Grissemann, 2000),
- to measure the writing performance with a writing test (for example the Westermann writing test (WRT 6+) from Rathenow, Vöge and Laupenmühlen, 1980),
- to use a combined reading and writing test like the Salzburg reading and writing test (SLRT) from Landerl, Wimmer, & Moser (1997) and
- for the proof of the discrepancy to use an intelligence test like the Cultural Fair Test (CFT) from Weiss (1987).

4 Assessment of secondary symptoms

Secondary symptoms can be developed in dependence on the feedback of the environment. The interactive behaviour of parents, peers and teachers with the dyslexic child has a high relevance for its self-esteem. The labilisation and the decrease of the self-esteem are the beginning of the development of emotional and behavioural disorders.

Betz and Breuninger (1982) describe four stages of the development of emotional and behavioural disorders:

1. After the first weeks in school negative attributions (of the failures) developed by the child and by the environment are starting. The first supporting activities by the parents will be experienced by the child as repression.

2. The dyslexic child tries to get success by the producing of compensating behaviour like clownery, violent behaviour or stealing presents for peers. But often the environment can not accept this behaviour and punishes it.

3. In the third stage the anxiety increases and leads to avoidance behaviour like absence from school, blocking and avoidance of reading and writing demands.

4. In the fourth stage these disordered emotions and behaviour will be stabilized by the decrease of motivation, disidentification and the misunderstanding of the environment (for example the mistrust of parents in cases of successful performance of the child).

If a dyslexic child has reached this fourth stage it is not possible to exercise reading and writing but it is necessary to reduce the disordered behaviour and the anxiety and to stabilize its self-esteem. That means a psychotherapy or a complex program including the parents are strongly recommended.

Treatment methods

1 Treatment on the level of primary causes

Examples of treatment methods regarding primary reasons are the following ones:

- Coloured glasses, transparencies and coloured paper of books should reduce the contrast and therefore the magnocellular activation. A compensation of the magnocellular deficit is expected.

- Reading windows should reduce the Breitmeyer- or "smear over effect".

- Prism glasses were developed for the stabilisation of the fixation point on the line.

- The cinesiological training like the "Brain Gym" program from Dennison and Dennison (1991) is based on the assumption of a hemispherical coordination deficit and wants to activate both hemispheres in the same time by special body exercises (e.g. symmetrical and cross-middle movements of the arms and legs).

- Dietary intervention, targeting specific biochemical anomalies were also investigated as a feasible treatment option.

2 Intervention regarding secondary causes

- Tachistoscopic visual perception training (Gutezeit, 1977) is based on a very short presentation (0.5 sec.) of single words and word groups, which can be repeated until the child or the children group can read or write them. The training can be realized by an overhead projector or by a PC with LCD projector. Gutezeit has evaluated this training for dyslexic children of the 3rd grade and found significant improvements of the reading and writing performance.

- Working memory training (Witruk, 2003)

- Visual perception training (Frostig, 1974)

- Training of auditory functions (Warnke, 1998)

- Training of integration functions (Karma, 2003)

3 Intervention regarding primary symptoms

Special rehabilitative classes (2nd and 3rd grade) offer particular didactics developed by Weigt (1994) with script oriented playing therapy, with additional supporting hand signals, graphical signs for peculiarities of orthography and with a

morphemic rule system for the better understanding of the construction of the German script. These special rehabilitative classes were evaluated by Witruk (1993b).

A very good impact on the school career and the personality development of the dyslexic children could be found.
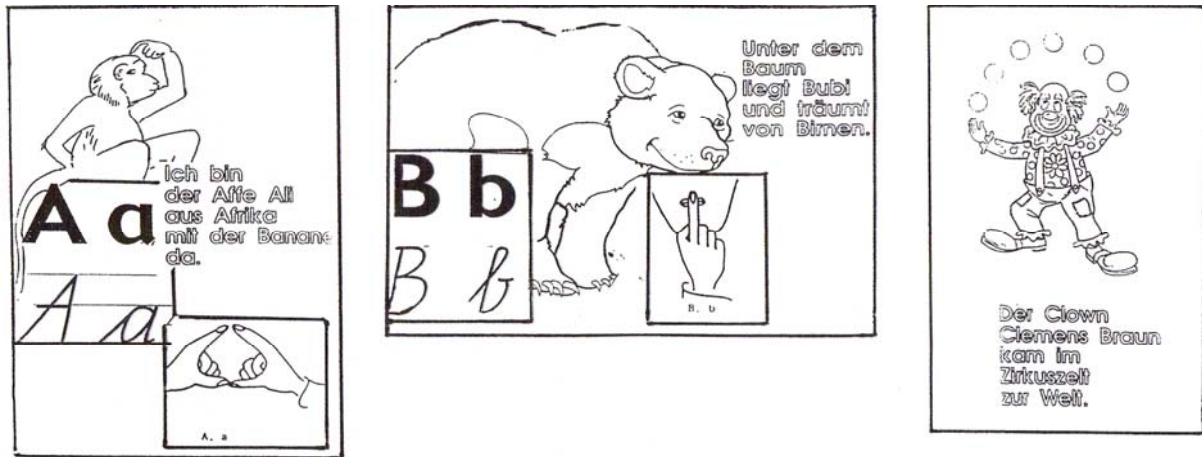

Figure 9. Special learning material in the rehabilitative classes.

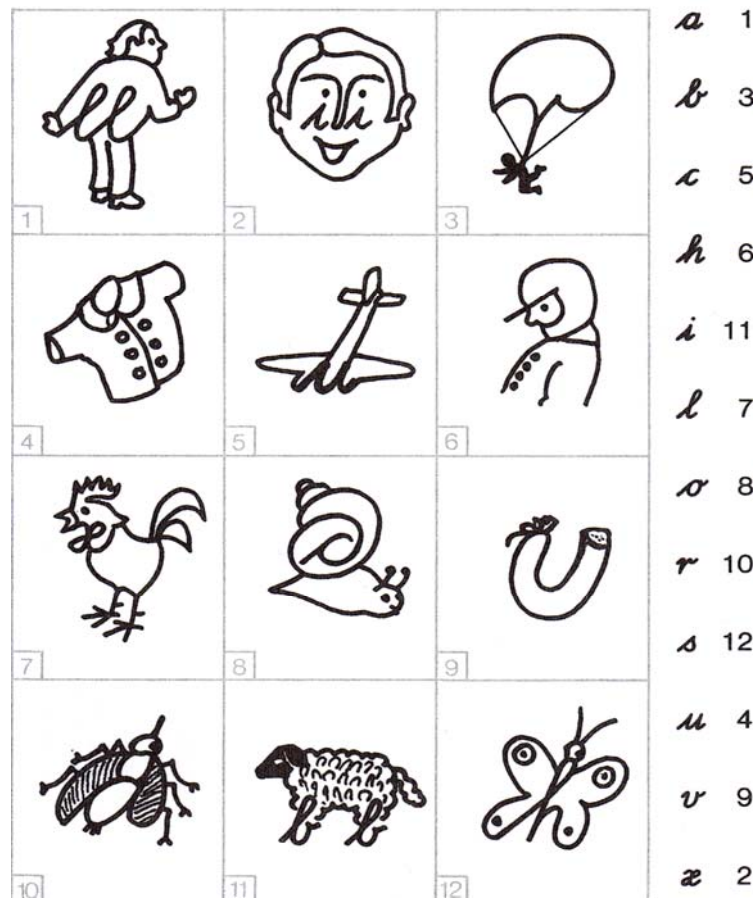Private reading and writing learning institutes offer special didactics.

Figure 10. Reading exercise for dyslexic children "Which letter is in which picture?"
(von Maydell & Vogel, 1977).

4   Intervention regarding secondary symptoms

The complex training programme developed by Betz and Breuninger (1982) is integrating three modules:

1   Group psychotherapy with children differentiated in children with high anxiety and children with violent behaviour,

2   Parent working meetings with psycho-education, exchange of experiences and information about the progress of the intervention,

3   Training of the orthography by using special didactics of success (for example exercise by self-control system, optimization of the learning organization, registration of correct responses - not of errors - in the dictate).

Single- or group psychotherapy of the dyslexic child (client-centred, non-directive psychotherapy, behaviour therapy or psychoanalyses) or systemic therapy are integrating the whole family.
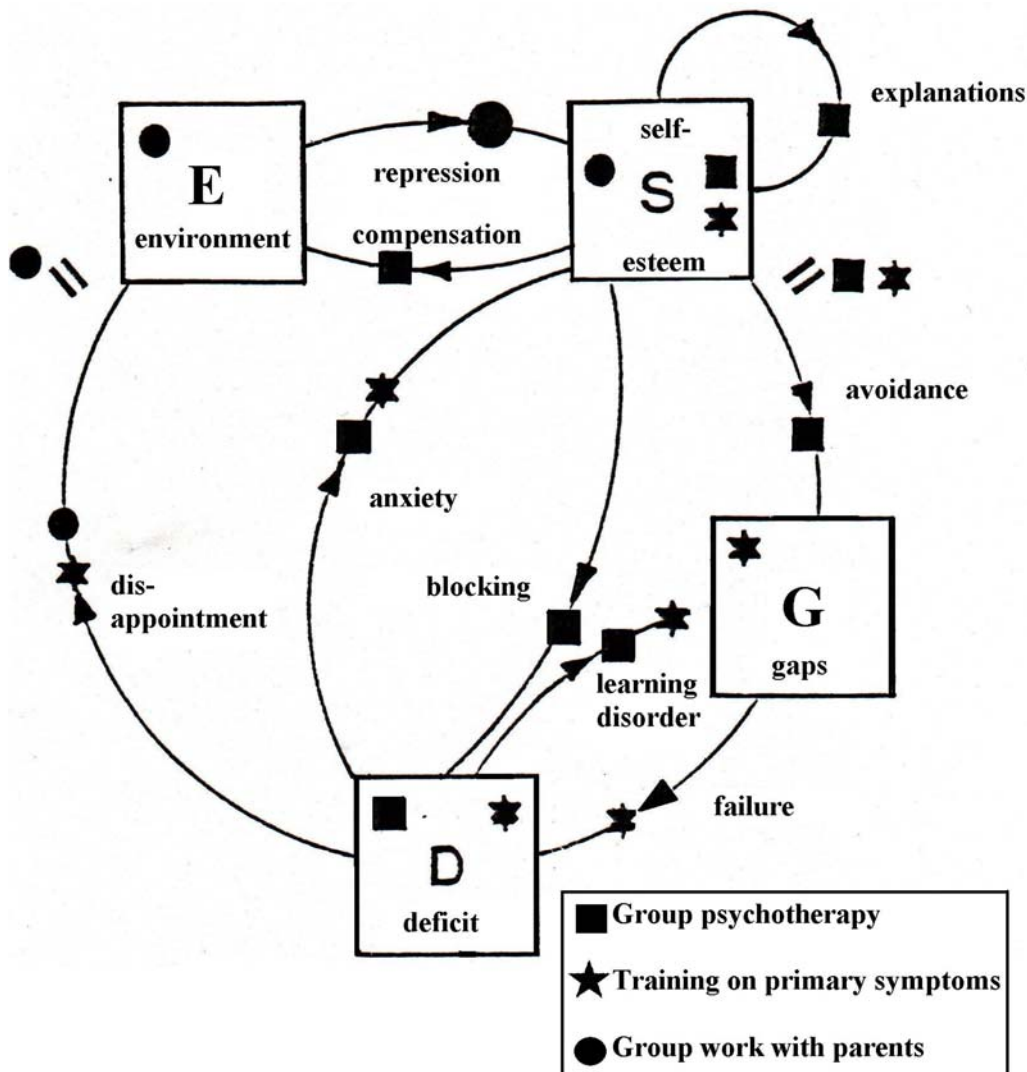

Figure 11. Complex training programme developed by Betz and Breuninger (1982).

Affiliations

Prof. Dr. Evelin Witruk
University of Leipzig
Faculty of Biosciences, Pharmacy and Psychology
Institute of Psychology II
Seeburgstr. 14 - 20
04103 Leipzig, Germany
eMail: witruk@rz.uni-leipzig.de

References

Anthoni, H., Zucchelli, M., Matsson, H., Müller-Myhsok, B., Fransson, I., Schumacher, J., Massinen, S., Onkamo, P., Warnke, A., Griesemann, H., Hoffmann, P., Nopola-Hemmi, J., Lyytinen, H., Schulte-Körne, G., Kere, J., Nöthen, M., Peyrard-Janvid, M. (2007). A locus on 2p12 containing the co-regulated MRPL19 and C2ORF3 genes is associated to dyslexia. *Human molecular genetics*, 16(6), 667-677.

Barnea, A., Lamm, O., Epstein, R., & Pratt, H. (1994). Brain potentials from dyslexic children recorded during short-term memory tasks. *International Journal of Neuroscience, 74*, 227-237.

Betz, D., & Breuninger, H. (1982). *Teufelskreis Lernstörung*. München: PVU.

Breitmeyer, B. G. (1980). Unmasking visual masking: A look at the "why" behind the veil of "how". *Psychology Review*, 380, 453-492.

Breitmeyer, B.G. (1993). The roles of sustained (P) and transient (T) channels in reading and reading disability. In: S.F. Wright & R. Groner (Hrsg.). *Facts of dyslexia and its remediation*. London/ Amsterdam/New York: Elsevier Science Publishers.

Breitmeyer, B. G., & Ritter, A. (1986). Visual persistence and the effect of eccentric viewing, element size, and frame duration on bistable stroboscopic motion percepts. *Perception & Psychophysics, 39 (4)*, 275-280.

Cope, N., Harold, D., Hill, G., Moskvina, V., Stevenson, J., Holmans, P. Owen, M. J., O'Donovan, M. C., Williams, J. (2005). Strong evidence that KIAA0319 on chromosome 6p is a susceptibility gene for developmental dyslexia. *American Journal of Human Genetics*, 76(4), 581-591.

Couto, J.M., Gomez, L., Wigg, K., Cate-Carter, T., Archibald, J., Anderson, B., Tannock, R., Kerr, E.N., Lovett, M.W., Humphries, T., Barr, C.L. (2008). The KIAA0319-like (KIAA0319L) gene on chromosome 1p34 as a candidate for reading disabilities. *Journal of neurogenetics*, 22(4), 295-313.

Dennison, P. E., & Dennison, G. (1991). *Lehrerhandbuch Brain-Gym*. Freiburg.

Eden, G.F., VanMeter, J.W., Rumsey, J. M., Zeffiro, T.A. (1996). The visual deficit theory of developmental dyslexia. *Neuroimage*, 4(3), 108-117.

Ellis, N. (1981). Visual and name coding in dyslexic children. *Psychological Research, 43*, 201-218.

Everatt, J., Groeger, J., Smythe, I., Balaam, S., Richardson, J., & McNamara, S. (2001). *Dyslexia and deficits in short-term memory: phonological versus sequential explanations*. Abstract on 5th BDA International Conference. York.

Finucci, J.M., Guthrie, J.T., Childs, A.L., Abbey, H, Childs, B. (1976). The genetics of specific reading disability. *Annual Review of Human Genetics*, 40, 1-23.

Fischer, B. (2007). Studien zur sprachfreien auditiven Differenzierung bei Legasthenie. *Forum Logopädie, 3(21)*, 30-35.

Fisher, S.E., Francks, C., Marlow, A.J., MacPhie, I.L., Newbury, D.F., Cardon, L.R., Ishikawa-Brush, Y., Richardson, A.J., Talcott, J.B., Gayan, J., Olson, R.K., Pennington, B.F., Smith, S.D., DeFries, J.C., Stein, J.F., Monaco, A.P. (2002). Independent genome.wide scans identify a chromosome 18 quantitative-trait locus influencing dyslexia. *Nature Genetics*, 30(1), 86-91.

Fisher, S.E., Stein, J.F., Monaco, A.P. (1999). A genome-wide search strategy for identifying quantitative trait loci involved in reading and spelling disability (developmental dyslexia). *European child & adolescent psychiatry*. 8, Suppl. 3, 47-51.

Francis, F., Koulakoff, A., Boucher, D., Chafey, P., Schaar, B., Vinet, M.C., Friocourt, G., McDonnell, N., Reiner, O., Kahn, A. (1999). Doublecortin is a developmentally regulated, microtubule-associated protein expressed in migrating and differentiating neurons. *Neuron*, 23, 247-256.

Frostig, M. (1974). *Visuelle Wahrnehmungsförderung. Übungs- und Beobachtungsfolge für den Elementar- und Primarbereich.* Anweisungsheft und Heft 1 - 3. (für deutsche Verhältnisse bearbeitet und herausgegeben von Anton und Erika Reinartz). Hannover: Schroedel.

Galaburda, A., Livingstone, M. (1993). Evidence for a magnocellular defect in developmental dyslexia. *Annals of the New York Academy of Sciences*, 682, 70-82.

Gathercole, S. E., Alloway, T. P., Willis, C. S., & Adams, A. M. (2006). Working memory in children with reading disabilities. *Journal of Experimental Child Psychology. 93*, 265-281.

Gathercole, S. E., & Baddeley, A. D. (1993). *Working memory and language*. East Sussex, Hove: Lawrence Erlbaum Associates Ltd.

Gutezeit, G. (1977). *Projektionstachistoskopisches Übungsprogramm für leserechtschreibschwache Schüler.* Göttingen: Hogrefe.

Habib, M. (2000). The neurological basis of developmental dyslexia: An overview and working hypothesis. *Brain, 123 (12)*, 2373-2399.

Hallgren, B. (1950). Specific dyslexia (congenital word blindness). A clinical and genetic study. *Acta Psychiatrica et Neurologica*, Suppl. 65, i-xi and 1287.

Hannula-Jouppi, K., Kaminen-Ahola, N., Taipale, M., Eklund, R., Nopola-Hemmi, J., Kääriäinen, H., Kere, J. (2005). The axon guidance receptor gene ROBO1 is a candidate gene for developmental dyslexia. *PloS Genetics*, 1(4), e50.

Hari, R., & Renvall, H. (2001). Impaired processing of rapid stimulus sequences in dyslexia. *Trends in Cognitive Science. 5*, 525–532.

Harold, D., Paracchini, S., Scerri, T., Dennis, M., Cope, N., Hill, G., Moskvina, V., Walter, J., Richardson, A.J., Owen, M.J., Stein, J.F., Green, E.D., O'Donovan, M.C., Williams, J., Monaco, A.P. (2006). Further evidence that the KIAA0319 gene confers susceptibility to developmental dyslexia. *Molecular Psychiatry*, 11(12), 1085-91.

Helenius, P., Uutela, K., & Hari, R. (1999). Auditory stream segregation in dyslexic adults. *Brain. 122*, 907-913.

Ho, C. S.-H., & Bryant, P. (1997). Learning to read Chinese beyond the logographic phase. *Reading Research Quarterly, 32*, 276-289.

Ho, C. S.-H., & Lai, D. N.-C. (1999). Naming-speed deficits and phonological memory deficits in Chinese developmental dyslexia. *Learning and Individual Differences, 11*, 173-186.

Ho, C. S.-H., Law, T. P., & Ng, P. M. (2000). The phonological deficit hypothesis in Chinese developmental dyslexia. *Reading and Writing: An Interdisciplinary Journal. 13*, 57-79.

Ho, C. S.-H., Chan, D. W.-O., Tsang, S.-M., & Lee, S.-H. (2002). The cognitive profile and multiple-deficit hypothesis in Chinese developmental dyslexia. *Developmental Psychology, 38 (4)*, 543-553.

Ho, C. S.-H., Chan, D. W.-O., Lee, S.-H., Tsang, S.-M., & Luan, V. H. (2004). Cogntive Profiling and preliminary subtyping in Chinese developmental dyslexia. *Cognition, 91*, 43-75.

Ho, C. S.-H., Chan, D. W.-O., Tsang, S.-M., Lee, S.-H., & Chung K. K. H. (2006). Word learning deficit among Chinese dyslexic children. *Journal of Child Language, 33*, 145-161.

Igo, R.P. Jr., Chapman, N.H., Berninger, V.W., Matsushita, M., Brkanac, Z., Rothstein, J.H., Holzman, T., Nielsen, K., Raskind, W.H., Wijsman, E.M. (2006). Genomewide scan for real-word reading subphenotypes of dyslexia: Novel chromosome 13 locus and genetic complexity. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)*, 141B, 15-27.

Kaminen, N., Hannula-Jouppi, K., Kestilä, M. Lahermo, P., Muller, K., Kaaranen, M., Myllyluoma, B., Voutilainen, A., Lyytinen, H., Nopola-Hemmi, J, Kere, J. (2003). A genome scan for developmental dyslexia confirms linkage to chromosome 2p11 and suggests a new locus on 7q32. *Journal of medical genetics*, 40(5), 340-345.

Karma, K. (2003). *AUDILEX 2.0 (Deutsche Bearbeitung von Bernd Richter).* Helsinki: Comp-Aid Ltd.

Lachmann, T. (2007). *Basic determinants of specific learning disabilities in reading: Experimental exploration of component functions in reading and of deficits in their coordination.* Kaiserslautern: Verlag der Universität Kaiserslautern.

Landerl, K., Wimmer, H., & Moser, E. (1997). *Salzburger Lese- und Rechtschreibtest (SLRT; 1. Auflage).* Göttingen: Hogrefe.

Lehmkuhle, S., Garzia, R.P., Turner, L., Hash, T., Baro, J.A. (1993). A defective visual pathway in children with reading disability. *The New England journal of medicine*, 328, 989–96.

Linder, M., & Grissemann, H. (2000). *Züricher Lesetest (ZLT).* (mit neuer deutscher Rechtschreibung, 6. Auflage). Göttingen: Hogrefe.

Livingstone, M.S., Rosen, G.D., Drislane, F.W., Galaburda, A.M. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 88(18), 7943-7947.

Livingstone, M.S., Hubel, D.H. (1988). Do the relative mapping densities of the magno- and parvocellular system vary with eccentricity? *The journal of neuroscience*, 8(11), 4334-4339.

Meng, H., Hager, K., Held, M., Page, G.P., Olson, R.K., Pennington, B.F., DeFries, J.C., Smith, S.D., Gruen, J.R. (2005). TDT-association analysis of EKN1 and dyslexia in a Colorado twin cohort. *Human Genetics*, 118(1), 87-90.

Morgan, W. P. (1896). A case study of congenital word blindness. *British Medical Journal, 2,* 1378.

Morris, D.W., Robinson, L., Turic, D., Duke, M., Webb, V., Milham, C., Hopkin, E., Pound, K., Fernando, S., Easton, M., Hamshere, M., Williams, N., McGuffin, P., Stevenson, J., Krawczak, M., Owen, M.J., O'Donovan, M.C., Williams, J. (2000). Family-based association mapping provides evidence for a gene for reading disability on chromosome 15q. *Human Molecular Genetics*, 9(5), 843-848.

Nandakumar, K., Leat, S.J. (2008). Dyslexia: A review of two theories. *Clinical & experimental optometry*, 91(4), 333-340.

Nöthen, M.M., Schulte-Körne, G., Grimm, T., Cichon, S., Vogt, I.R., Müller-Myhsok, B., Propping, P, Remschmidt, H. (1999). Genetic linkage analysis with dyslexia: Evidence for linkage of spelling disability to chromosome 15. *European Child & Adolescent Psychiatry*, 8 Suppl.3, 56-59.

Olson, R.K., Forsberg, H., Wise, b. (1994). Genes, environment, and development of orthographic skills. In: Berninger, V.W. (Hrsg.). *The varieties of orthographic knowledge I: theoretical and developmental issues*. 27-71.

Rathenow, P., Vöge, J., & Laupenmühlen, D. (1980). *Westermann Rechtschreibtest 6 + (WRT 6+)*, 1. Auflage. Göttingen: Hogrefe.

Scerri, T.S., Schulte-Körne, G. (2010). Genetics of developmental dyslexia. *European child & adolescent psychiatry*, 19(3), 179-197.

Schneider, A. (2001). *EEG-coherence-analysis for examining a general automatizational deficit in dyslexic children.* Proceedings of 5th BDA International Conference, York.

Schulte-Körne, G. (2001). *Lese-/Rechtschreibstörung und Sprachwahrnehmung*. München: Waxmann.

Schuhmacher, J., Anthoni, H., Dahdouh, F., König, I. R., Hillmer, A. M., Kluck, N., Manthey, M., Plume, E., Warnke, A., Remschmidt, H., Hülsmann, J. Cichon, S., Lindgren, C. M., Propping, P., Zuchelli, M., Ziegler, A., Peyrard-Janvid, M., Schulte-Körne, G., Nöthen, M. M., Kere, J. (2006). Strong genetic evidene of DCDC2 as a susceptibility gene for dyslexia, *American Journal of Human Genetics*, 78(1), 52-62.

Siegel, L. S., & Linder, B. A. (1984). Short-Term Memory Processes in Children With Reading and Arithmetic Learning Disabilities. *Developmental Psychology, 20, 2,* 200-207.

Siegel, L. S., & Ryan, E. B. (1989). The Development of Working Memory in Normally Achieving and Subtypes of Learning Disabled Children. *Child Development, 60,* 973-980.

Smith, S.D., Kimberling, W.J., Pennington, B.F., Lubs, H.A. (1983). Specific reading disability: Identification of an inherited form through linkage analysis. *Science,* 219, 1345-1347.

Smith, S.D., Kimberling, W.J., Pennington, B.F. (1991). Screening for multiple genes influencing dyslexia. *Reading and Writing. An Interdisciplinary Journal*, 3, 285-298.

So, D., & Siegel, L. S. (1997). Learning to read Chinese: Semantic, syntactic, phonological and working memory skills in normally achieving and poor Chinese readers. *Reading and Writing: An Interdisciplinary Journal, 9,* 1-21.

Sperling, A.J., Lu, Z, Manis, F.R., Seidenberg, M.S. (2003). Selective magnocellular deficits in dyslexia: A

„phantom contour" study. *Neuropsychologia*, 41(10), 1422-1429.

Stanley, G. & Hall, R. (1973). Short-term visual information processing in dyslexics. *Child development*, 44(4), 841-844.

Stevenson, J. (1991). Which aspects of processing text mediate genetic effects ? *Reading and Writing. An Interdisciplinary Journal*, 3, 249-269.

Taipale, M., Kaminen, N., Nopola-Hemmi, J., Haltia, T., Myllyluoma, B., Lyytinen, H., Muller, K., Kaaranen, M., Lindsberg, P.J., Hannula-Jouppi, K., Kere, J. (2003). A candidate gene for developmental dyslexia encodes a nuclear tetratricopeptide repeat domain protein dynamically regulated in brain. *Proceedings of the National Academy of Sciences of the United States of America*, 100(20), 11553-11558.

Valtin, R. (1989). Dyslexia in the German language. In P. G. Aaron and R. M. Joshi (Eds.), *Reading and Writing Disorders in Different Orthographic Systems*, (pp. 119-135). Dordrecht: Kluwer Academic Publishers.

Vellutino, F. R. (1987). Dyslexia. *Scientific American, 256, 3*, 1-42.

Vellutino, F.R., Fletcher, J.M., Snowling, M.J., Scanlon, D.M. (2004). Specific reading disability (dyslexia): What have we learned in the past four decades? *Journal of child psychology and psychiatry*, 45(1), 2-40.

Vogler, G.P., DeFries, J.C., Decker, S.N. (1995). Family history as an indicator of risk for reading disability. *Journal of Learning Disability*, 18, 419-421.

Von Maydell, I., & Vogel, H. (1977). *Übungen für Legastheniker*. Braunschweig: Westermann Lernspielverlag GmbH.

Warnke, F. (1998). *CD-Lateraltraining 1. und 2. MediTECH,* Wedemark.

Weigt, R. (1994). *Lesen- und Schreibenlernen kann jeder!? Methodische Hilfen bei Lese-Rechtschreib-Schwäche.* Neuwied, Kriftel, Berlin: Luchterhand.

Weiss, R. H. (1987). *Grundintelligenztest Skala 2 - CFT 20.* Göttingen: Hogrefe.

Wilcke, A. (2007). Genetische Grundlagen der Legasthenie. Diplomarbeit, Universität Leipzig.

Wilcke, A. & Boltze, J. (2010). Genetische Grundlagen der Legasthenie. In: Witruk, E., Riha, D., Teichert, A., Haase, N., Stück, M. (Eds.): *Learning, Adjustment, and Stress Disorders*. Bern: Peter Lang.

Wilcke, A. Weissfuss, J. Kirsten, H., Wolfram, G., Boltze, J., Ahnert, P. (2009). The role of gene DCDC2 in German dyslexics. *Annals of Dyslexia*, 59(1), 1-11.

Willows, D. M., Kruk, R., & Corcos, E. (Eds.) (1993). *Visual Processing in Reading and Writing Disabilities.* Hillsdale, NJ: Lawrence Erlbaum Publishers.

http://gl.yorku.ca/GlProfProfiles.nsf/PublicationsNav/828B3A398C593140852574C20065C21C?OpenDocument&ExpandView&count=1000&subnavigation=facultyWitruk, E. (1993a). Memory Deficits of Dyslexic Children. In P. Tallal, A. M. Galaburda, R. R. Llinás, & C. von Euler (Eds.), *Temporal information processing in the nervous system. Special reference to dyslexia and dysphasia.* Annals of the New York Academy of Sciences, 682, (pp. 430-435). New York: The New York Academy of Sciences.

Witruk, E. (1993b). Long-term Effects of Rehabilitative Interventions For Dyslexic Children. In P. Tallal, A. M. Galaburda, R. R. Llinás, & C. von Euler (Eds.), *Temporal information processing in the nervous system. Special reference to*

dyslexia and dysphasia. Annals of the New York Academy of Sciences, 682, (pp. 426-429). New York: The New York Academy of Sciences.

Witruk, E. (1999). Working memory performance in dyslexic children. In E. Witruk, & T. Lachmann (Eds.), *Basic Mechanisms of Language and Language Disorders.* Leipzig: Leipziger Universitätsverlag.

Witruk, E. (2003). Training of working memory performance in dyslexics. *Psychology Science, 45, I,* 94-100.

Witruk, E., & Ho, C. S.-H. (subm. 2010). Working memory in Cantonese speaking and German dyslexic children. *Australian Journal of Learning Difficulties.* Routledge.

Witruk, E., Ho, C. S.-H., & Schuster, U. (2002). Working memory in dyslexic children - How general is the deficit? In E. Witruk, A. D. Friederici , & T. Lachmann (Eds.), *Basic Functions of Language and Language Disorders,* (pp. 281-297). Boston: Kluwer Academic Publishers.

Witruk, E., & Rosendahl, W. (1999). Modalitäts-und Anforderungsspezifik von Arbeitsgedächtnisleistungen bei Legasthenikern. *In Kongreßbericht der XXIII. Arbeits- und Fortbildungstagung der Deutschen Gesellschaft für Sprachheilpädagogik e.V.,* Würzburg 1999.