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Developmental coordination disorder: a review of research on subtypes and comorbidities

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Abstract

The interest in Developmental Coordination Disorder (DCD) has grown considerably over the last decade. Nevertheless, its etiology and prognosis are still poorly understood. The idea is growing that DCD may not be a uniform disorder. This review summarizes research on DCD, with a particular focus on subtype and comorbidity studies. The main message of the paper is that, in order to understand the etiology and prognosis of DCD, we need to have a better understanding of its nature. This requires an awareness of the existence of subtypes and comorbidities. Current theories on comorbidity phenomena are discussed in terms of their possible merit for the development of the field. Particular attention is given to the Automatization Deficit Hypothesis, a theory based on research on dyslexia.
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1. Introduction

Since Developmental Coordination Disorder (DCD) was first listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM) it has received a great deal of attention from researchers in areas such as education, psychology, kinesiology, and physical and occupational therapy. In recent years, motor journals have dedicated complete issues to the disorder (Barnett, Kooistra, & Henderson, 1998;

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Beek & van Wieringen, 2001; Henderson, 1994). But, despite this surge of interest, the etiology and prognosis of DCD are still unclear. Children diagnosed with DCD may show a variety of symptoms, and their specific needs, as well as their prognosis will differ accordingly. If we want to understand the etiology and prognosis of DCD, we need to have a better understanding of its nature. This requires an awareness of the existence of subtypes and comorbidities.

This review presents a summary of the findings on differences between children with DCD and controls. The main focus of the review, however, is on research on subtypes and comorbidities. The PsycINFO and Medline databases were searched for relevant articles, using keywords such as: Developmental Coordination Disorder, Attention Deficits, Hyperactivity, Reading Disability, Dyslexia, Specific Language Impairment, Comorbidity, and Subtypes. Publications that were referenced in these articles were added, as well as other publications known to the author.

2. What is DCD?

The fact that we still understand so little about the etiology and prognosis of DCD is partly due to inconsistencies in diagnostic criteria. These inconsistencies have led to widely diverging samples, which makes it hard to reach an overall conclusion about the findings. According to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994), Developmental Coordination Disorder is a marked impairment in the development of motor coordination, which cannot be attributed to a general medical condition or mental retardation. The diagnosis is usually based on a score on a standardized motor test, such as the Movement Assessment Battery for Children (Henderson & Sugden, 1992). To be diagnosed with DCD a child's test score should be outside the normal range. However, if the child has a known medical or neurological condition, such as cerebral palsy, the diagnosis DCD is not warranted. In case of mental retardation, the diagnosis is made only if the test score is lower than could be expected on the basis of this specific condition. An additional criterion stated in DSM IV is that the motor difficulties should have a negative impact on academic achievement or daily life activities.

Geuze, Jongmans, Schoemaker, and Smits-Engelsman (2001) conducted a review of 176 publications on DCD. They concluded that there is still little consistency in the procedures researchers use to select children for research purposes. It is unclear, for example, what constitutes the normal range of scores on a motor skills test. Different cut off scores have been used, ranging from the 5th to the 15th percentile. Furthermore, many studies do not mention how exclusion criteria were handled, while hardly any study seems to have considered the effects on academic achievement or daily life activities.

A second factor complicating our understanding of the etiology and prognosis of DCD is the ongoing debate on the nature of the disability. A review of the literature suggests that DCD is related to problems with almost any sensory or motor skill imaginable. Children with DCD have been found to show abnormalities in postural control (Wann, Mon-Williams, & Rushton, 1998; Williams & Woollacott, 1997), as

well as in fine motor skills (Smits-Engelsman, Niemeijer, & Van Galen, 2001). Further, their motor problems have been attributed to deficits in the sensory domain, the motor domain, and in sensorimotor integration.

Among the visual problems reported are inaccuracies in estimating object size (Hulme, Biggerstaff, Moran, & McKinlay, 1982; Hulme, Smart, & Moran, 1982; Hulme, Smart, Moran, & McKinlay, 1984; Lord & Hulme, 1988) and difficulties in locating an object's position in space (Schoemaker et al., 2001). Further Wilson and Maruff (1996, 1999); (Wilson, Maruff, & McKenzie, 1997) found a decreased ability to direct visual attention. Several studies have reported proprioceptive or kinaesthetic difficulties, such as a decreased kinaesthetic acuity (Coleman, Piek, & Livesey, 1997; Laszlo, Bairstow, Bartrip, & Rolfe, 1988, 1996; Piek, Pitcher, & Hay, 1999; Smyth & Mason, 1998). But the disability has also been related to a deficit in the mapping of visual and proprioceptive information (Mon-Williams, Pascal, & Wann, 1994; Mon-Williams, Wann, & Pascal, 1999; Sigmundsson, 1999; Sigmundsson, Ingvaldsen, & Whiting, 1997).

Others have found abnormalities in the execution of movements, in the absence of any perceptual component (Hoare, 1994; Raynor, 2001). And children with DCD are more variable than controls in the timing and force of muscle contractions, both in rhythmic and discrete tasks (Geuze & Kalverboer, 1987, 1994; Lundy-Ekman, Ivry, Keele, & Woollacott, 1991; Parker, Larkin, & Wade, 1997; Piek & Skinner, 1999; Volman & Geuze, 1998a, 1998b; Williams & Castro, 1997; Williams, Woollacott, & Ivry, 1992). Finally, Parush, Yochman, Cohen, and Gershon (1998) report difficulties in visuomotor integration.

These findings seem to suggest that DCD is a fairly generalized problem, affecting movement, as well as perception. But it is important to note that the findings are based on group differences, not individual data. Typically, differences between children with DCD and controls are caused by a subset of children in the DCD group. It is fairly common to find children with DCD who perform at the same level as controls on a particular measure (Wright & Sugden, 1996). Apparently, not all children diagnosed with DCD are alike. Many researchers have already suggested that there may be subtypes of DCD (e.g., Wright & Sugden, 1996).

What complicates our understanding of the etiology and prognosis of DCD even further is the comorbidity of motor difficulties and difficulties in other, non-motor areas. Many children diagnosed with DCD also show problems with attention and concentration, or specific learning disabilities, such as dyslexia and Specific Language Impairment (SLI). Although this is a widely acknowledged phenomenon, most researchers do not seem to be particularly concerned with this issue. Few attempts have been made to select 'pure' samples and, although some studies give prevalence rates of comorbidities, the possibility that the children in the sample had multiple difficulties is usually not even considered.

2.1. Subtypes, comorbidities and the etiology, nature, and prognosis of DCD

Our lack of knowledge about the prognosis of DCD is a major problem in our field. While some children seem to outgrow their motor problems, either with or

without intervention, many others continue to show poor motor skills throughout adolescence and into adulthood (Cantell, Smith, & Ahonen, 1994; Geuze & Börger, 1993; Visser, 1998; Visser, Geuze, & Kalverboer, 1998). Similar outcomes have been reported for children with learning disabilities (Sanches & Coppel, 2000) or ADHD (Rasmussen & Gillberg, 2000). If we do not understand these differences in developmental outcomes, how can we design effective intervention methods?

Rasmussen and Gillberg (2000) addressed the issue of subtypes and comorbidities in relation to developmental outcome and found some interesting results. In a longitudinal study they followed 55 children diagnosed with ADHD, either with or without comorbid symptoms of DCD. At the age of 22 the children were compared with 46 age-matched controls, without signs of ADHD or DCD. In the ADHD group as many as 58% had a poor outcome, indicated by remaining symptoms of ADHD, reading disorders, a low education level, and social problems. Furthermore, children with comorbid symptoms had the least favorable outcome.

It is quite possible that differences in developmental outcome among children with DCD also reflect the existence of subtypes. In any case, Rasmussen's findings clearly underline the importance of research on subtypes and comorbidities in order to come to a better understanding of the nature of developmental disabilities. This is a necessary step if we want to come to grips with their etiology and prognosis.

3. Research on subtypes and comorbidities

Thus far, research on subtypes and comorbidities has developed along more or less separate lines. Studies on subtypes of DCD try to find differences among the children in their performance on a series of sensorimotor measures. Typically, these studies do not look at comobidity phenomena. Comorbidity studies, on the other hand, look at relationships between DCD and other developmental disabilities, but typically do not distinguish among the children with DCD. The following sections present a review of these studies. I will argue for the importance of integrating both types of research to come to a better understanding of the nature of DCD.

3.1. Subtype studies

The earliest subtype studies focused on a descriptive analysis of the motor performance characteristics of children within the DCD population (e.g. Gubbay, 1975; Henderson & Hall, 1982). More recently, researchers have started to use cluster analysis to formalize the search for subtypes. In this approach a set of measurements is acquired and subjects are grouped together on the basis of the profiles of their scores on these measurements.

In 1994 three studies were published that used cluster analysis to detect subgroups of children with DCD (Dewey & Kaplan, 1994; Hoare, 1994; Miyahara, 1994). The subtypes reported by these studies differed, both in number and in their

characteristics. Hoare (1994) used measurements of visual perception, visuomotor integration, manual dexterity, kinesthetic acuity, balance, and running speed, and found five subtypes. Dewey and Kaplan (1994) used balance, bilateral coordination, upper limb coordination, transitive gestures, and motor sequencing and ended up with four subtypes. Miyahara, using running speed, agility, balance, strength, upper limb speed, and dexterity, also found four subtypes but these were different from the subtypes reported by Dewey and Kaplan (1994). Despite these inconsistencies there was one common result: the emergence of a subtype characterized by difficulties on all sensorimotor measures.

Wright and Sugden (1996) reported a similar finding. Using cluster analysis on a set of Movement ABC data they found a cluster with generalized problems, while other clusters were characterized by difficulties in particular areas, such as catching, fine motor skills, or balance. Overall, these findings suggest the existence of a subtype of DCD with a generalized sensorimotor deficit (or maybe, as Hoare (1994) suggests: a 'generalized perceptual dysfunction'). Such a subtype will show up regardless of the specific sensorimotor variables used in the study, whereas the presence of other subtypes depends on the inclusion and combination of particular measures.

In a review of subtype studies Macnab, Miller, and Polatajko (2001) point out that the inconsistencies in the results may have been caused by a number of factors, such as sample differences, differences in the variables used in the cluster analysis, and differences in the statistical procedures. In a replication of Hoare's study Macnab et al. (2001) found a cluster structure that was fairly similar to the one reported by Hoare (1994). This suggests that cluster analysis is a valuable technique, provided adequate information is available concerning the sample and the variables used.

But, thus far, subtype studies have contributed little to our understanding of DCD. In itself, the distinction into subtypes does not tell us much about the etiology and the prognosis of the disability. Studies are needed, therefore, to further distinguish subtypes in terms of underlying deficits, and to examine the developmental trajectories of children with different subtypes of DCD.

As Kaplan, Wilson, Dewey, and Crawford (1998) have pointed out, subtypes of DCD differ with respect to comorbidities. The prevalence of comorbidities is particularly high in children with a generalized sensorimotor deficit. This is an important finding. It indicates that the presence or absence of comorbidities has a direct relevance for the definition of subtypes of DCD. Our understanding of DCD will improve if we know why comorbidity is linked to a particular subtype.

3.2. Comorbidity studies

Many studies have examined the relation between clumsiness, attention deficits, hyperactivity, and learning disabilities. The literature is sometimes confusing, given the variety of terms used by different researchers. Reading Disability, Development Reading Disorder, and dyslexia, for example, are all used to refer to a condition in which reading ability is significantly below the norm in relation to chronological

age and overall intellectual potential. These terms should not be confused with SLI, however, which is considered to be a phonological, or phonosyntactic disorder, that specifically affects inflectional morphology and syntax (Bishop & Leonard, 2000). The literature on attention deficits and hyperactivity shows a similar maze of different terms and types of disabilities, the most common of which are Attention Deficit Disorder (ADD) and Attention Deficit-Hyperactivity Disorder (ADHD).

Research has shown that ADD, ADHD, reading disability (RD), and SLI frequently co-occur with symptoms of DCD (Dewey, Kaplan, Crawford, & Wilson, 2002; Dewey, Wilson, Crawford, & Kaplan, 2000; Gillberg, 1998; Gillberg & Kadesjö, 1998, 2000; Hill, 2001; Kadesjö & Gillberg, 1999, 2001; Kaplan, Crawford, Wilson, & Dewey, 1997; Martini, Heath, & Missiuna, 1999; Wilson & McKenzie, 1998). Hill (2001) who conducted a review of the literature on SLI, reports a substantial comorbidity between SLI and poor motor skills and suggests that SLI is, in fact, not a specific disorder of language. Similar findings have been reported in studies that looked at the relationship between DCD and ADHD. Kadesjö and Gillberg (1999) found, for example, that approximately half of the 7-year-olds they diagnosed with DCD also had moderate to severe symptoms of ADHD. In a study on children who were referred by agencies and private schools because they had learning and/or attention problems, Kaplan et al. (1998) found that comorbidity was 'the rule rather then the exception'. Among a group of 115 children only 53 were identified as 'pure cases', showing signs of DCD, RD or ADHD. Of the 62 'comorbid cases' 23 had difficulties in all areas measured.

The idea is growing, therefore, that many cases of SLI, RD, ADHD, or DCD reflect a more generalized deficit, instead of a pure language, attention, or coordination problem (e.g. Gillberg & Kadesjö, 1998). Consequently, there is an increased focus on the development of theories that identify the common cause of these symptoms (Gillberg, 1998; Gillberg & Kadesjö, 1998; Kaplan et al., 1997; Kaplan et al., 1998).

The notion of a more generalized deficit seems to breathe new life into the concept of Minimal Brain Dysfunction (see Wender, 1978, for a discussion of the concept). In its most common interpretation, popular particularly in the 1970s and 1980s, Minimal Brain Dysfunction (MBD) refers to a rather nonspecific problem in brain function, basically similar in kind to the damage connected to cerebral palsy or mental retardation (Rutter, 1984; Wender, 1978). The concept groups together a series of developmental problems that are thought to be related to brain dysfunction, such as clumsiness, attention deficits, hyperactivity, and learning disabilities. Many scientists considered MBD to be a 'waste basket hypothesis' with very little explanatory power (Kalverboer, 1978). Consequently, it was abandoned by most researchers, favoring more specific descriptions of disabilities, in the hope of finding localized brain dysfunctions.

In a sense, the renewed interest in the issue of comorbidity has led to a revaluation of the MBD concept. Most theories that guide present day research into the nature of the traditional MBD symptoms do not differ substantially from the views that dominated research in the 1980s. Research on comorbidity phenomena in relation to DCD is currently guided by two theories, labeled the 'ABD hypothesis'

and the 'DAMP hypothesis.' The following section discusses the contribution of these theories to our understanding of DCD. Further, I will suggest an alternative theory, the Automatization Deficit Hypothesis', which originates from research on dyslexia.

4. Theories on comorbidity

4.1. ABD: The atypical brain development hypothesis

According to Kaplan et al. (1998), brain imaging techniques have generally failed to support the idea that unique brain areas are associated with individual developmental disorders. Dyslexia, for example, has been linked to deficits in the left temporal-parietal area, but when other brain areas are examined, atypical brain development is visible in both hemispheres and in cortical as well as subcortical areas (Riccio & Hynd, 1996). Similarly, atypical brain development in ADHD is not limited to the prefrontal cortex and its connections, areas known to be involved in attention. Rather, it is spread throughout both hemispheres, the corpus callosum, and the striatum (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990).

Kaplan et al. (1998) conclude that the brain dysfunctions underlying these deficits are diffuse, rather than localized, and that we seem to be unable to distinguish one deficit from the other on the basis of neurological information. They suggest that there are no 'reliably identifiable, discrete developmental disorders' (Kaplan et al., 1998, p. 484). Symptoms of DCD, RD, and ADHD are considered to be a reflection of the same underlying brain deficit, labeled 'atypical brain development', or ABD (Kaplan et al., 1998). Variable expressivity, a term borrowed from genetics, is used to explain the idea that a common underlying factor, atypical brain development, can express itself in many different ways. The specific pattern of deficits is considered to depend on the extent and the location of the neurological abnormality.

The question is whether the atypical brain development hypothesis really contributes to our understanding of DCD. Linking developmental problems to a diffuse dysfunctioning of the brain is reminiscent of the MBD concept and does not seem to shed any new light on the etiology of DCD. Further, the theory has difficulty accounting for some of the 'pure cases' of disabilities. Studies on AHDH have shown, for example, that brain abnormalities in children with a pure form of ADHD are, in fact, localized, rather then diffuse. This indicates the possibility of a genetic component, specific to ADHD (Filipek et al., 1997; Semrud-Clikeman et al., 2000).

It is possible that pure cases of ADHD differ substantially from comorbid cases, not only in behavioural symptoms, but also with respect to underlying brain dysfunctions. The same may be true for pure and comorbid cases of DCD, RD, or SLI. Aram and Eisele (1994) compared the development of children with SLI and children with localized lesions in brain areas specialized in language. They noted that the symptoms in the SLI children were generally more severe and more persistent. This finding led to the conclusion that, although some children with SLI may suffer

from unilateral left hemisphere damage, many apparently have other, perhaps more extensive, neurological problems.

4.2. DAMP: The 'deficits in attention, motor control and perception' hypothesis

In 1982 and 1983 a group of Swedish researchers published the first of a series of studies on Swedish children with perceptual, motor, and attention deficits. The children, selected at the age of six and followed longitudinally, were diagnosed with MBD and showed 'neurodevelopmental deviations indicative of the clumsy child syndrome' (Gillberg, Carlstrom, Rasmussen, & Waldenstrom, 1983; Gillberg & Rasmussen, 1982; Rasmussen, Gillberg, Waldenstrom, & Svenson, 1983). In later years, the terminology used to describe these children changed from MBD into 'Deficits in Attention, Motor control and Perception' (DAMP). Over the years, the group published numerous studies demonstrating a strong relationship between attention problems, DCD, and perceptual disorders (Gillberg, 1998; Gillberg & Kadesjö, 1998). The description of DAMP does not explicitly include children with learning disabilities like dyslexia or SLI, but the symptoms are strongly related to classroom dysfunction.

Gillberg and Kadesjö (1998) argue that a generalized disorder underlies the difficulties associated with DAMP, and that we should study this conglomerate of symptoms rather than discrete disorders. It is unclear what this generalized disorder is, however. Clearly, some brain dysfunction is assumed, an assumption that is substantiated with behavioral assessments and neurological examinations (Gillberg et al., 1983). The neurological symptoms found in children with MBD or DAMP seem to suggest a dysfunctioning of certain brain areas, particularly the cerebellum and the basal ganglia (Lundy-Ekman et al., 1991). But a direct, causal link has yet to be established.

4.3. The automatization deficit hypothesis

During the last decade, research on children with dyslexia has led to the formulation of the 'Automatization Deficit Hypothesis' (Fawcett & Nicolson, 1992). This theory is relatively unknown to people working in the field of motor development, as it does not specifically address DCD. However, the Automatization Deficit Hypothesis does address motor problems in children with dyslexia. More importantly, it provides a rationale for the coexistence of a range of developmental problems, such as difficulties in articulation, reading, coordination, and attention. The theory may, therefore, prove to be of value in the search for brain dysfunctions underlying comorbid cases of DCD, ADHD, and learning disabilities.

For many years research on dyslexia was dominated by the idea that language problems are related to brain areas specialized in the processing of language, particularly the left temporal-parietal area. Nicolson and Fawcett showed, however, that motor problems and abnormalities in muscle tone are common symptoms in the majority of people with dyslexia (Fawcett & Nicolson, 1995, 1999; Nicolson & Fawcett, 1990; Nicolson, Fawcett, & Dean, 2001). The motor difficulties became

apparent during the performance of a motor task concurrent with a second, non-motor task.

According to the dual-task paradigm, such a decrease in performance as a result of a secondary task indicates a lack of automatization of the primary task. A fully automatized skill 'does not require conscious effortful monitoring and should show little or no decrement even if there are other demands upon conscious processing capacity' (Nicolson & Fawcett, 1990). Conversely, any automatization deficit will show up if conscious monitoring is made more difficult, either by stress or by some other task requiring attentional recourses.

The motor task that Fawcett and colleagues (e.g., Fawcett, Nicolson, & Dean, 1996) used in their studies was 'balancing on a beam'. They found a marked deterioration in performance in children with dyslexia when the children were asked to count backwards or respond to a tone. Controls were hardly affected by the secondary task. The findings led Fawcett et al. (1996) to conclude that dyslexia is caused by a general deficit in the ability to fully automate skills. Such a deficit is believed to lead to a lack of dexterity, or fluency, in the use of language, a problem that persists, even after an apparently successful remediation of the language disorder.

Yap and van der Leij (1994) confirmed the findings of Fawcette and colleagues regarding the breakdown of balance under the stress of a secondary task. In another replication ratings were collected on dyslexia and motor problems, but also on ADHD symptoms (Raberger & Wimmer, 1999; Wimmer, Mayringer, & Raberger, 1999). Poor dual-task balancing appeared to be limited to people with higher ADHD ratings, which was used as an argument to refute the automatization deficit hypothesis. But this finding merely indicates that the automatization deficit hypothesis does not explain the difficulties of the entire population of people with dyslexia. It may only be valid for a subgroup, showing a more generalized disorder.

One of the strengths of the Automatization Deficit Hypothesis is that the behavioral problems are linked to a clearly specified brain area, the cerebellum, which is known to play an important role in skill learning and automatization (e.g., Doyon, 1997; Kandel, Schwarz, & Jessell, 2000). Using brain imaging techniques Fawcett and Nicolson (1999) found significantly less brain activity in the right cerebellar cortex of people with dyslexia, as compared to controls, both when executing a pre-learned movement sequence and when learning a new sequence. The notion of a cerebellar deficit is in line with 'soft signs' of neurological abnormality found in children with Minimal Brain Dysfunction (Gillberg, 1985) and, more recently, children with DCD (Volman & Geuze, 1998a). These neurological symptoms include dysmetria (inaccuracy in the range and direction of movements), dysdiadochokinesia (an irregular pattern of alternating movements), mirror movements, and the choreiform twitch (a form of finger tremor), typical defects associated with cerebellar disease (Kandel et al., 2000).

In more recent research, Fawcett (2002) has further developed her causal hypothesis, linking the problems people with dyslexia face to a dysfunction of the cerebellum. She found significant problems in articulation in children with dyslexia, both in motor planning and in the speeded production of single articulatory gestures (Fawcett, 2002). She concluded that the most likely route by which cerebellar problems

lead to reading and spelling difficulties is via the role of the cerebellum in the development of articulation.

The Automatization Deficit Hypothesis may prove to be a powerful paradigm, as it is able to explain comorbid symptoms of dyslexia, ADHD, and DCD. Fluent reading and the smooth control of (particularly rapid) movements both depend heavily on learning and, eventually, automatization. A lack of automatization will cause difficulties in both areas, even if a child has normal, or above normal intelligence. Further, such a deficit will lead to a breakdown of performance in a dual task condition, or any comparable situation. In other words, children with such a deficit are easily distracted by external stimuli and may exhibit problems with attention and concentration.

Obviously, just as any 'normally developing' child has strengths and weaknesses, so will a child with an automatization deficit. Therefore, children with an automatization deficit do not have to show the same degree of difficulties in every domain. As a group, however, they can be expected to show comorbid symptoms of DCD, ADHD, and learning disabilities.

Thus far, there is no evidence that children diagnosed with DCD show signs of an automatization deficit. Very few studies have looked into these children's ability to learn a new skill, and there seem to have been no studies on automatization differences between children with DCD and controls. These studies are clearly needed to examine the possible contribution of the Automatization Deficit Hypothesis to our understanding of DCD.

5. Conclusions

There is more and more evidence indicating that DCD is not a uniform disorder. Rather, there appear to be different subtypes of disabilities, either pure or comorbid, with different causes, different treatment requirements and different outcomes. A definition of subtypes is important, as it will increase our insights into the specific problems a child experiences, and improve the diagnosis and treatment of children with these disabilities.

Although some subtype studies have been conducted, very little has been done to follow up the categorization of children in subgroups of DCD. If there are subgroups, what makes them different? How do they differ, for example, with respect to comorbidities? What are their developmental prospects, and how will they respond to different forms of treatment? A combined approach, linking the search for subtypes and comorbidities, will lead to a more thorough description of the motor and non-motor symptoms related to DCD, which may generate new ideas on its etiology and possible treatment methods.

Current research on subtypes and comorbidities indicates the existence of a subtype with a generalized sensorimotor deficit. Further, comorbidity is high among children with such a generalized deficit (Kaplan et al., 1998). Several attempts have been made to explain such comorbidity phenomena, from the MBD concept to more contemporary theories, such as the atypical brain development hypothesis and the

DAMP hypothesis. Each of these theories attributes the problems to some form of diffuse brain dysfunction. Research on people with dyslexia suggests that the dysfunction may be localized in the cerebellum and express itself in a general automatization deficit.

The notion of an automatization deficit underlying the traditional MBD phenomena seems promising. Research on the automatization of skills in subtypes of DCD may prove to be an important step in our understanding of the nature, etiology, and prognosis of this disability.

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