

# Linking ADHD – Dyslexia and Specific Learning Difficulties

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## INTRODUCTION

The knowledge of the simultaneous co-existence of two or more conditions is very useful, both from the theoretical and from the practical point of view because when one exists, then it is likely to expect the appearance of the co-existing condition. Dyslexia and attention deficit hyperactivity disorder (ADHD) usually co-exist and are lifelong developmental neurobiological and usually hereditary conditions, with an international ratio of 4 boys to 1 girl, which confirms their biological origin (Geschwind, 1986).

However, a major biological difference distinguishes the two conditions: ADHD is a ‘God-created’ universal and fundamental brain function, which is why it has its own dedicated brain center in the prefrontal lobe; dyslexia on the other hand has no dedicated brain center because reading and writing are human-taught inventions of limited time span.

## DYSLEXIA

Dyslexia is a specific learning disability (SLD) of the written language. Its fundamental diagnostic criterion is a serious difficulty in reading and, specifically, in the very slow reading speed that dyslexics face throughout their life internationally, irrespective of language, race or culture. A fundamental prerequisite for the existence of a SLD is normal or above IQ. In contrast, general learning

difficulties result from negative intellectual, psychoeducational and environmental factors and present the regular ratio of 1 boy to 1 girl, internationally.

Dyslexia, the most common SLD, accounts for about 80 per cent of all SLD and affects between 3–12 per cent of the population (Pavlidis, 1981, 1985, 1990, 1990a; Eden & Vaidya, 2008). Dyslexia is characterized by severe difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. Secondary consequences may include problems in reading comprehension and reduced reading experience, which can impede growth of vocabulary and background knowledge.

There are many definitions of dyslexia but the most complete and clinically valid is the one by Critchley (1981), who defined dyslexia as follows:

‘Developmental dyslexia is a learning disability which initially shows itself by difficulty in learning to read, and later by erratic spelling and by lack of facility in manipulating written as opposed to spoken words. The condition is cognitive in essence, and usually genetically determined. It is not due to intellectual inadequacy or to lack of socio-cultural opportunity, or to emotional factors, or to any known structural brain-deficit. It probably represents a specific maturational defect which tends to lessen as the child grows older, and is capable of considerable improvement, especially when appropriate remedial help is afforded at the earliest opportunity.’

Although all agree that the cause of dyslexia is neurobiological and one of the most heritable (Grigorenko, 2001), the exact cause of dyslexia is a fiercely debated issue with many competing theories trying to explain its origins. The ‘phonological deficit theory’ (Vellutino, Fletcher, Snowling, & Scanlon, 2004) is the most popular but it is also a troubled theory (Ramus, Rosen, Dakin, Day, Castellote, White, & Frith, 2003; Pavlidis & Giannouli, 2003; Pavlidis, 2013). Unlike dyslexia, ADHD is characterised by a set of inappropriate behaviours, such as inattention, hyperactivity and impulsivity, the combination of which, in particular inattention, negatively affects the process of learning (Conners, 1990; Currie et al., 2008).

In order for a theory to be reliable and valid, such as the ‘Neuro-Sequential and Visuo-Linguistic Transfer Deficit Theory’ (Pavlidis, 2013; Pavlidis & Giannouli, 2003), it is mandatory to describe and, even better, to predict the reality (e.g. the symptomatology and the various research findings). The ‘Neuro-Sequential and Visuo-Linguistic Transfer Deficit Theory’ states that the main cause of dyslexia is neurological, and manifests itself in both verbal and non-verbal tasks, which call upon mechanisms that constitute important components of the reading process, such as sequencing, concentration and eye movements (Pavlidis, 1981; 2013; Ojemann & Mateer, 2005). Furthermore, it is unequivocally accepted that if the cause of a problematic condition is successfully treated, then the very same problem and its symptoms should disappear. Consequently, in nations with 100 per cent phonologically consistent and transparent languages (like Turkish and Finnish) dyslexia should not exist because the risk of the erroneous phoneme to grapheme correspondence is eliminated. This is not, however, the case. Interestingly enough, the prevalence of dyslexia in transparent languages is similar to the percentage being reported for the nations with phonologically inconsistent, opaque languages, such as English, where there are many phonological expressions of the same grapheme. Such findings not only challenge the foundations of the Phonological Deficit Theory of dyslexia (Torgesen, 2004; Vellutino et al., 2004; Ziegler

& Goswami, 2005), but also prove it unreliable. It seems that not the phonological awareness but the morpho-grammatical structure of a language is the more important determinant of the kind of spelling errors made by dyslexics (Pavlidis & Giannouli, 2003).

A theory could not be valid if it violates fundamental scientific principles – something that the phonological deficit theory does (e.g. tautology – meaning that a problem cannot be explained by the problem itself). Reading has two fundamental stages: decoding and comprehension. The first stage of reading (decoding) refers to the ability to correctly convert a grapheme (e.g. a letter) to its corresponding phoneme (sound). Phonological awareness in the written form refers exactly to the same skill, namely to the ability to correctly convert a grapheme (e.g. a letter) to its corresponding phoneme (sound). Therefore, in the written language, phonological awareness and decoding are one and the same process, hence phonological awareness cannot be the cause of the dyslexia (decoding deficit) because it contravenes the basic rule of science – tautology – as it is summarized in Table 15.1

## ADHD

ADHD is one of the most frequently diagnosed developmental behavioural disorders in school-age children and its prevalence is estimated to be 5–8 per cent of all school-age children, irrespective of socioeconomic status and ethnicity. ADHD has been shown to be associated with SLD (especially dyslexia).

ADHD in children has been one of the most widely studied, debated and treated disorders and is defined as a disorder of attention, self-regulation and cross-temporal organization of behaviour (Barkley, 1994). Functional imaging and fMRI studies uncovered abnormalities in the frontal lobes of individuals with ADHD (Rubia et al., 2007), whereas brain-imaging studies showed reversed asymmetry of hemisphere structures. Evidence from neuropsychological studies suggests that the dopaminergic system plays an important role in the pathology of ADHD and possibly accounts for the problems in executive functioning, such as problem-solving strategies, inhibitory control at a cognitive and/or motor level, working memory, self-regulation, cognitive flexibility, interference control or planning (Swanson, et al., 2001). The Diagnostic and Statistical Manual of Mental disorders (DSM-IV-R) of the American Psychiatric Association (APA, 2000) for the diagnosis of ADHD requires that the individual must have at least six out of the nine symptoms of either inattention or hyperactivity–impulsivity, or a combination of them for at least 6 months. These symptoms must be present in at least two different settings, e.g. home or school, appear before the age of 7, and cause a dysfunction in at least one setting, e.g. school, home, work. These symptoms must be inappropriate for their developmental level. People with schizophrenia, mental retardation and autism are excluded from the diagnosis. The diagnostic questionnaire is filled out either by the teacher or the parent and the answer to each question is a yes or no. Most of the questions start with the word frequently,

**Table 15.1 Why the phonological deficit theory as a cause of dyslexia is wrong**

- Phonological awareness is the same as the first stage of reading (decoding, the conversion of graphemes into phonemes). Thus, the Phonological Deficit Theory tries to explain the reading difficulties of dyslexia with the reading difficulty itself. Hence, the Phonological Theory is tautological and violates this fundamental rule of science (tautology).
- Difference between oral and written language. The same brain centres control oral and written language, hence, if it was a language problem, then dyslexics should have the same problems in written and oral language, but internationally they do well oral language and fail in the visible language (writing). Hence, dyslexia cannot be caused by a language-phonological deficit. Therefore, the Phonological Theory is incorrect.
- If a perfect phonological awareness is reached through teaching, then dyslexia should be completely cured, which is not the case. The same should have applied in languages with perfectly consistent phonology, such as Finnish, Turkish, where dyslexia should also not exist. But it does exist in similar percentages as in phonologically inconsistent languages like English because dyslexia has biological etiology, thus its universal existence is mandatory. Therefore, the Phonological Theory and the non-existence of dyslexia are mutually exclusive. Therefore, either dyslexia does not exist or the Phonological Theory is incorrect.
- The Phonological Theory predicts the same kind of spelling errors (phonological), irrespective of the phonological consistency of the language, but dyslexics make completely different spelling errors in Greek and in English (Pavlidis & Giannouli, 2003). Therefore, the Phonological Theory is incorrect.
- If the Phonological Theory was correct, then any questionnaire that does not include questions about phonology should not have a high correlation with the actual diagnosis of dyslexia. This assumption is proven to be wrong because the results of the Pavlidis Questionnaire, which does not include any questions on phonology, are in agreement in 96% of the cases with the actual diagnosis of dyslexia (Xystrou & Pavlidis, 2004).

which introduces a high level of subjectivity because it means different things to different people. That is why there is a major disagreement between diagnoses (Lahey et al., 2006; Lee et al., 2008).

According to DSM-IV, if a child fulfils the aforementioned criteria, then they can be diagnosed with one of the three types of ADHD: (1) ADHD, predominantly inattentive type, (2) ADHD, predominantly hyperactive–impulsive type, or (3) ADHD, combined type, which is the most diagnosed type in children (55 per cent) (Heward, 2009).

### **ADHD–DYSLEXIA: SIMILARITIES AND DIFFERENCES**

Dyslexia constitutes the most common SLD and one of the most frequently diagnosed developmental disorders in school-age children, along with ADHD.

Dyslexia and ADHD are characterised by different developmental deficits (see Table 15.2). Dyslexia is an SLD with difficulties manifested in reading, writing and sometimes in math, which hamper the children's educational development, thus widening the gap between the reading level achieved by the child and the expected level of the child's development according to its intellectual abilities and daily effort. On the other hand, ADHD is characterised as a behavioural and emotional disorder that is manifested in attention and concentrated difficulties, executive functions deficits (working memory, response inhibition, planning) and, in most cases, SLD are secondary to ADHD.

Furthermore, their diagnostic criteria are far from being the same. For evaluation of ADHD, the child's observation is rated not directly, but indirectly by parents and teachers. On the contrary, the evaluation of dyslexia is carried out by direct psychoeducational tests of the child's performance (Eden & Vaidya, 2008; Pavlidis, 1990a). In ADHD the completion of behavioural rating scales, aptitude testing, and physiological and neurological testing are also used.

Internationally, the diagnosis of dyslexia is performed by subjective psychoeducational tests and the earliest it can be done with some certainty-accuracy is after the middle of the 2nd grade because the pupil must be at least 1.5 years behind his class in reading. Similarly, the current diagnostic criteria, as set by DSM-IV and ICD-10, are even more unreliable, as shown by Lee et al. (2008) who found that the diagnoses of the same children based on the answers of the same 419 mothers in the two similar questionnaires (DSM-IV and ICD-10) agreed only in 11 per cent of the cases and disagreed 89 per cent. However, the objective and biological ophthalmokinesis test (known as Pavlidis Test) achieves an accurate prognosis and diagnosis of dyslexia and the diagnosis of ADHD from preschool age because it is not based on writing or reading, but on the objective-biological ophthalmokinesis (Jost, 1997; Pavlidis & Samaras, 2005; Pavlidis, 2013). The early diagnosis or the prognosis from preschool age is important because it maximizes the effectiveness of the individualised treatment, and at the same time prevents or ameliorates both the learning difficulties and the secondary psychosocial problems, i.e. low self-esteem resulting from humiliating school failure and negative criticism at home and in school.

It is widely accepted that the accurate and timely diagnosis of these two disorders leads to their effective but different treatments. The effective treatment of dyslexia or ADHD requires an individualized intervention program adjusted every time to the child's specific psychoeducational needs. Research has found that the most effective treatment of ADHD is the combination of medication and psychological support, especially via cognitive behaviour therapy (Barkley, 1996). Medication helps temporarily two-thirds of the children with ADHD to focus, reduce his/her excessive fidgeting and hyperactivity, improve the ability to selectively attend to relevant stimuli, and offers a better efficiency of specific and/or general cognitive processes, but these drugs are very potent and should be used with caution and only if all other modes of treatment have failed. Psychosocial treatments teach children social skills and psychoeducational treatments teach a number of alternative–better behavioral strategies.

Despite the aforementioned differences, and prior to the discussion of the comorbidity of ADHD and dyslexia, it should be stressed that the neurological disorders appear worldwide, and always with similar rate and characteristics. As a consequence, the indisputable neurobiological cause of ADHD and dyslexia (neurological disorders) dictates a uniform and global epidemiology, symptomatology and expression of their nature across nations, languages and cultures.

**Table 15.2 Dyslexia–ADHD: similarities and differences**

| Criteria                                      | Dyslexia | ADHD |
|---|----------|------|
| <i>Neurobiological + hereditary aetiology</i> | Yes      | Yes  |
| Dedicated brain centre(s)                     | No       | Yes  |
| Specific learning disability                  | Yes      | No   |
| <i>Behavioural disorder</i>                   | No       | Yes  |
| <i>Pharmacological treatment</i>              | No       | Yes  |
| <i>Psychoeducational treatment</i>            | Yes      | No   |

### COMORBIDITY: ADHD–DYSLEXIA AND SPECIFIC LEARNING DIFFICULTIES

The term comorbidity describes the simultaneous occurrence of two or more unrelated conditions/disorders. ADHD co-occurs (is comorbid) with other neurobiological disorders more than dyslexia. Kronenberger & Dunn (2003) reported that among behavioural disorders, ADHD is the most frequently associated with dyslexia.

Strong associations-comorbidity was reported between ADHD, dyslexia and other SLD in different samples and settings (Gooch, Snowling, & Hulme, 2011).

Researchers suggested three distinct levels of developmental comorbidity that can be related with the particular time of appearance of the comorbid disorder/s that co-exist with ADHD. According to Taurines et al. (2010), the first developmental level – the precomorbidity – covers the time period from birth to childhood when ADHD may co-exist with comorbid disorders such as autism and sleep disorders. The second developmental level – the simultaneous comorbidity – covers the time period from childhood to puberty when ADHD may co-exist with SLD (e.g. dyslexia, developmental coordination disorder (DCD), or specific difficulties in reading, spelling or mathematics). The third developmental level – post-comorbidity – covers the time period from puberty to adulthood when ADHD may co-exist with comorbid disorders such as stress, depression, or personality and behavioral disorders.

Faraone et al. (1993) suggested that ADHD and dyslexia are transmitted independently in families and that their co-occurrence may be due to non-random mating. According to this, spouses of those with ADHD had significantly higher rates of dyslexia than spouses of those without ADHD. Friedman, Chhabildaw, Budhiraja, Willcutt, and Pennington (2003) could not provide support for this finding in their study because they could not sufficiently explain the larger part of comorbid cases of ADHD + dyslexia. Pennington, Groisser, and Welsh (1993) looked at the hereditary transmission of the basic symptoms of attention deficit disorder ([ADD] hyperactivity, impulsivity) on dyslexic subjects in a study of twins. The comorbid disorders ADD + dyslexia were found to be inherited in 45 per cent of the cases, while the comorbid disorder ADHD/Impulsivity + dyslexia was inherited in 5 per cent of the cases. At the same time, researchers reported that 95 per cent of a comorbid phenotype (ADD + dyslexia) was attributed to

commonly shared genetic factors, compared to 21 per cent of comorbid phenotype (ADHD/impulsivity + dyslexia). In cross-sex comparisons, it was found that ADD co-exists with dyslexia without significant differences between boys and girls. More recently, a study of 700 twins by Greven et al. (2011) showed that the comorbid disorder ADD/Hyperactivity + dyslexia is inherited in very high percentages.

Despite the aforementioned growing evidence of heritability of the comorbid ADHD + dyslexia disorder, Willcutt et al. (2002) stated that ADHD-only subjects appeared impaired on executive functions but not on phonological tasks; dyslexia-only subjects exhibited phonological processing deficits but not executive functions deficits; and the comorbid subjects showed a sum of the previous deficits. In other words, their model predicted that a different neuropsychological deficit is the cause of each disorder: a phonological deficit relates to dyslexia and an inhibition deficit explains ADHD. Supporting evidence came from the study of Raberger & Wimmer (2003) who reported slower rapid automatized naming (RAN) in dyslexia and in comorbid phenotype, but not in ADHD-only.

Rucklidge & Tannock (2002) suggested that the neuropsychological deficits of the comorbid group are different from the simple additive combination of the deficits associated with dyslexia-only and ADHD-only groups because there is a significant interaction between dyslexia and ADHD on at least some of the neurocognitive weaknesses. So, individuals with dyslexia and ADHD generally seem to have slower naming speed, specifically concerning naming letters and digits tasks in dyslexia, and for object and colour tasks in ADHD (Ghelani, Sidhu, Jain, & Tannock 2004). Rucklidge & Tannock (2002) reported that the comorbid group exhibited more severe naming and working memory deficits (characteristic of dyslexia), as well as inhibition deficits and reaction times (characteristic of ADHD) in relation to both single disorder groups. McGee, Brodeur, Symons, Andrade, & Fahie (2004) studied time perception and auditory working memory in children with ADHD, children with dyslexia and children with both disorders. They found that children with both ADHD and dyslexia faced more difficulties in 'estimating the duration of a task' than the single disordered groups, suggesting the importance of time estimation in differentiating ADHD and dyslexia (De Jong, Oosterlaan, & Sergeant, 2006). Another important impairment described in the comorbid group is the processing speed deficit that appears to relate to both dyslexia and ADHD. It seems a shared cognitive factor that can explain the comorbidity of the two disorders (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Thus, the neuropsychological background of co-occurring dyslexia and ADHD can be better understood as the co-existence of phonological short-term memory deficits and central executive deficits sustaining an incomplete dissociation of executive and linguistic function in dyslexia and ADHD (Tiffin-Richards, Hasselhorn, Woemmer, Rothenberg, & Banaschewski, 2008).

The 'endophenotypes' are heritable quantitative traits between the genotype and the behavioural phenotype that reflect an individual's liability or risk to develop or

manifest a given disorder (Castellanos & Tannock, 2002). Taking into consideration this statement, Gooch, Snowling, and Hulme (2011) investigated the possible 'cognitive endophenotypes' of the two disorders in children with dyslexia and/or ADHD symptoms by measuring children's performances on phonological skills, executive function and time perception. The latter was tested with two tasks: time reproduction, in which deficits were related to ADHD but not dyslexia, and duration discrimination, which revealed deficits associated with both disorders. In the duration discrimination task, children with dyslexia and ADHD performed in a similar way to those with ADHD-only, but performed worse than children with dyslexia-only, indicating symptoms of inattention among the children with dyslexia, rather than literacy difficulties, which are usually specific to dyslexics. So, this particular outcome of the study didn't support what Castellanos and Tannock (2002) described. All in all, their findings were in line with the claim that ADHD and dyslexia are products of different cognitive deficits, and that they may be attributable to common genes whose influences are likely to be pleiotropic (Willcutt et al., 2002), meaning that the same influences affect more than one phenotype. Similarly, Germano, Galiano, and Curalo (2010) stated that there are such pleiotropic genes and candidate chromosome regions responsible for the comorbid disorder ADHD + dyslexia. In the first cartography of genes, which included assessment of reading ability, Loo et al. (2004) studied a sample of 233 ADHD subjects and their siblings. Their results defined/designated for the first time two candidate chromosome regions (16p, 17q) responsible for the co-existence of ADHD + dyslexia. The comorbid children ADHD + dyslexia showed a phenotype with more severe cognitive deficits and worse neuropsychological outcomes compared with children with only ADHD or dyslexia.

Bird, Gould, and Staghezza (1993) reported that over half of all ADHD children have comorbid disorders and each comorbid disorder increases the impairment of the ADHD child. He also stated that the rates of comorbid disorders can differ according to the subtype of ADHD. Although ADHD affects the ability to learn, it is not a learning disability in itself, so treating the symptoms of ADHD will not correct the learning disorder that the child might have.

Various researchers have found evidence that ADD without hyperactivity is related to LD, especially in math. Children with ADD without hyperactivity encounter difficulties in reading and math computation because their poor working memory, combined with the inattention, prevents them from learning the arbitrary symbol systems involved in reading and math. Likewise, Zentall (1990) argued that poor cognitive style (inattention, disorganisation) is associated with math computation deficits, whereas cognitive ability (IQ, memory) and reading are correlated with decreased comprehension and problem solving. These findings suggest that the cognitive deficits of students with ADD without hyperactivity underlie LD.

Pennington (2002) claimed that is common to find high rates of ADHD in LD populations. This association, however, may be an artifact of a definitional overlap. In an epidemiological sample in which ADHD, dyslexia or math disability

(dyscalculia) were defined independently, Shaywitz et al. (2002) reported that 11 per cent of the ADHD children had either dyslexia or dyscalculia and 33 per cent of the dyslexics and/or dyscalculic children had ADHD. Research has shown that 25–50 per cent of children with ADHD have SLD (Barkely, 1994) whereas Semrud-Clikeman et al. (1992) indicated a prevalence of learning difficulties in children with ADHD: 15–50 per cent for reading, 24–60 per cent for math, 24–60 per cent for spelling. Dyslexia and ADHD co-occur in 25–50 per cent of the cases (Willcutt & Pennington, 2000). Between the two disorders, there is a bidirectional relationship, children with reading, math or spelling deficits who also have ADHD have more severe learning difficulties than those with only one of the conditions.

In the Dunnedin epidemiologic sample, about 80 per cent of 11-year-old children were identified with ADHD who, as a consequence, had dyslexia or related spelling or written language problems (McGee, et al., 2004). Interestingly, a study, carried out in Sweden by Katesjo and Gillberg (2001), followed up a big population of pupils in general schools between the ages of 4 and 7. After their diagnosis, these pupils were allocated different categories, such as ADHD, borderline ADHD, without ADHD, DCD, ODD, asperger, dyslexia and moderate learning difficulties. Researchers reported that 40 per cent of the pupils had comorbid dyslexia + ADHD, 29 per cent had comorbid dyslexia + borderline ADHD, and only 7 per cent had pure dyslexia without ADHD. More recently, Pauc (2005) attempted to replicate these findings of Kadesjo and Gillberg. He tried to define the exact percentage of various cases of comorbidity in a sample of 100 children, aged 4–15 years old, who had been allocated various developmental disorders, as shown in Table 15.3.

ADHD coexists more often with other disorders (80.4 per cent of the cases) than with dyslexia (51.6 per cent of the cases). When a child is diagnosed with ADHD, they are more likely to also have dyslexia than the other way round. The attention deficit subgroup coexists more frequently with dyslexia than with the hyperactivity–impulsivity one. Even far more interesting is the fact that the former 31 per cent and 21 per cent of ADHD and dyslexia comorbidity is shaken off when the number of children with ADHD without hyperactivity is included in the analysis.

ADHD has a strong negative effect on academic performance. For example, 25–70 per cent have learning disabilities, 90 per cent have low school performance, 50–70 per cent have poor peer relationships, 32 per cent do not complete high school, 75–95 per cent do not finish university on time, 50 per cent are less likely to go into higher education and 46 per cent were expelled from school (Currie et al., 2008).

Velting and Whitehurst (1997) found that inattention–hyperactivity between ages 6 and 7 was closely related with poorer reading skills. Children with the inattentive form of ADHD are more susceptible to learning difficulties, and (Willcutt et al. 2002; Willcutt et al. 2007) put forward that common genetic influences may predispose children to both reading difficulties and elevations of

**Table 15.3 Comorbidity of ADHD with ODD, Tourette, dyslexia and DCD**

|          | Comorbidity |          |      |          |     |
|----------|-------------|----------|------|----------|-----|
|          | ODD         | Tourette | ADHD | Dyslexia | DCD |
| ODD      |             | 10%      | 25%  | 45%      | 95% |
| Tourette | 13%         |          | 38%  | 25%      | 81% |
| ADHD     | 14%         | 17%      |      | 31%      | 89% |
| Dyslexia | 17%         | 8%       | 21%  |          | 85% |
| DCD      | 21%         | 14%      | 35%  | 50%      |     |

Source: Pauc (2005).

**Table 15.4 Comorbidity of ADHD with SLD (69.8%)**

| Problem                  | Comorbidity % |
|--------------------------|---------------|
| Difficulty in reading    | 26.7%         |
| Difficulty in arithmetic | 31.4%         |
| Difficulty in spelling   | 30.2%         |
| Difficulty in writing    | 65.1%         |

Source: Mayes, Calhoun, and Crowell 2000.

inattention. Mayes, Calhoun, and Crowell (2000) reported that in their study of the 119 children aged 8–16 years old, and previously diagnosed as having SLD and/or ADHD, 61.3 per cent of the cases exhibited learning difficulties and 72.3 per cent showed ADHD. Interestingly, 69.8 per cent of the children with ADHD had shown an additional SLD (e.g. reading and/or spelling difficulty in arithmetic) (see Table 15.4).

Seidman, Biederman, Monuteaux, Coyle, and Faraone (2001) evaluated children with and without SLDs, both in reading and in arithmetic, on a broad sequence of neuropsychological tests. In their analysis, children with ADHD + RD (reading difficulties) performed significantly worse than ADHD and controls in the tests. The researchers contended that more severe neuropsychological dysfunctions were associated with comorbid LD in the ADHD group especially when arithmetic difficulty is present. Raberger and Wimmer (2003) found that early attention problems may be associated with concurrent and later reading problems, but not vice versa. Similarly, Ghelani et al. (2004,) in an attempt to evaluate reading processing, applied a sequence of tests of reading component processes to adolescents with ADHD, RD (reading difficulties), ADHD + RD (reading difficulties) and controls. Regarding the lexical access and the text reading, researchers found that both RD (reading difficulties) and RD (reading difficulties) + ADHD groups showed significant difficulties in all measures. The ADHD group showed adequate reading with subtle difficulties in text, word speed and accuracy, and slight difficulties in silent reading. The RD group obtained scores on reading comprehension comparable to both ADHD and the

**Table 15.5 Comorbidity of ADHD with dyslexia and DCD in boys and girls**

|                        | Dyslexia   | ADHD       | DCD        |
|------------------------|------------|------------|------------|
| Boys ( <i>n</i> = 136) | 90 (66.2%) | 94 (69.1%) | 19 (14.7%) |
| Girls ( <i>n</i> = 43) | 36 (83.7%) | 22 (51.2%) | 10 (24.4%) |

Source: Kaplan, Dewy, Crawford, and Wilson (2001).

control group. And, as expected, the comorbid group ADHD + RD performed similarly to the RD group on the silent reading and had better scores on the oral comprehension. The noteworthy finding is that both RD and RD + ADHD groups showed generalized impairment in naming speed for all types of stimuli.

Around 50 per cent of ADHD cases also have severe enough motor problems to be diagnosed as DCD (Pitcher, Piek, & Hay, 2003). Researchers, such as Katesjo & Gillberg (2001), reported a higher prevalence of boys compared to girls for both ADHD and DCD. Kaplan, Dewy, Crawford, and Wilson (2001) studied seven disorders: dyslexia, ADHD, DCD, ODD (oppositional defiant disorder), CD (conduct disorder), depression and stress, and gathered data from a sample of 179 children from 430 families, aged 8.2–16.9. The most striking outcome of their study was that from the 116 children diagnosed with ADHD, 63 showed a comorbid reading difficulty, 29 of them had a comorbid DCD, the 39 of them had a comorbid ODD and only 5 of them had a stress disorder. The children's percentage rates having dyslexia, ADHD and DCD can be seen in Table 15.5.

When ADHD and DCD are comorbid, the outcome tends to be more severe than when each disorder occurs alone (Pitcher et al., 2003; Visser, 2003). This implies that there may be an exclusive shared aetiology of the comorbidity that is distinct from the factors influencing either of the separate disorders. One major difficulty in exploring the comorbidity of these two disorders is the difference between the bodies of research existing for each disorder. ADHD has been extensively researched, with many genetic studies confirming the high heritability of ADHD and its subtypes (Levy & Swanson, 2001) and many well-developed measures in questionnaire or interview form and/or numerous neuropsychological measures (Taylor et al., 2004). DCD, however, is much less extensively researched and the existing literature and its aetiology is often confusing.

This confusion is often due to varying selection criteria, such as different cut-off scores being used in different studies (Piek, Pitcher, & Hay, 1999), and also due to the overlap that many symptoms of DCD have with other disorders, such as learning difficulties (Peters, Barnett, & Henderson, 2001). Although the link between ADHD and motor problems has been recognised for many years, there have been few studies that have investigated the motor problems in relation to the three distinct subtypes identified by the DSM-IV. One link that appears to have been established is that between inattentive symptomatology and poor fine motor skills (Pitcher et al., 2003). There is also evidence that suggests that gross motor deficits are more likely to occur in ADHD-combined type compared to ADHD-inattentive type (Visser, 2003). Differences in the prevalence of boys and girls for different

subtypes also suggests a greater relationship between DCD and ADHD-inattentive type, as a greater proportion of girls are found with ADHD-inattentive type compared with ADHD-combined type (Lahey et al., 2006).

## CONCLUSION

In conclusion, there is robust evidence of the clinically significant coexistence of ADHD and SLD, especially with dyslexia. However, some aspects of their association remains unclear, for instance it is unknown whether the association of SLD and ADHD is comparable across ages. The association between inattention and dyslexia–SLD is stronger than between the hyperactive–impulsive or combined type of symptoms of ADHD. On the contrary, the impulsive type is more strongly associated with behavioural problems than with academic ones. When the two conditions co-exist, their problems are more severe and treatment becomes more difficult. If someone has ADHD, then they are more likely to also have dyslexia than the other way round. Scientific literature on ADHD is mainly based on research in boys because ADHD is more frequently observed in boys than in girls (Biederman & Faraone, 2005). The challenge for future research is to develop a better understanding of the nature and interaction of the genetic and environmental influences that produce the overlapping of the cognitive and behavioural profiles associated with these conditions. Most importantly, the prognosis and diagnosis of dyslexia and ADHD has to move from the existing controversial subjectivity to the biological objectivity, which in turn will lead to earlier and more effective methods of treatment that will ameliorate not only the LD but also the consequent psychosocial problems.

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