The co-occurrence of neurodevelopmental problems in dyslexia

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The primary aim of this study was to explore the overlaps between dyslexia and a range of neurodevelopmental disorders and problems (NDPs), specifically symptoms of attention-deficit/hyperactivity disorder, autism spectrum disorder, atypical sensory perception and developmental coordination disorder. Capitalizing on a population-based sample of twins, secondary aims included estimating the heritability of dyslexia and reporting on the measurement characteristics of the scale used to assess dyslexia. A telephone interview regarding symptoms of dyslexia and other NDPs was conducted with parents of 1,688 nine-year-old twins. The prevalence and the heritability of dyslexia were estimated at 8 and 52%, respectively. The boy: girl ratio was 1.5:1. Results revealed that there was more than an eight-fold increase in (diagnostic proxy) NDPs prevalence in the dyslexia group as compared to typical readers. Quantitatively measured symptoms of inattention, oral language problems and atypical sensory perception significantly predicted dyslexia status in a multivariate analysis. By contrast, ASD-related inflexibility was inversely associated with dyslexia in the multivariate model. In sum, dyslexia often overlaps with other NDPs. The current study provides new knowledge supporting the position to move beyond isolated diagnostic categories into behavioural profiles of co-
occurring problems when trying to understand the pattern of strengths and needs in individuals with dyslexia.

**KEYWORDS**
ADHD, autism, comorbidity, dyslexia, perception, reading

# INTRODUCTION

Hanna is a ten-year-old girl who struggles with reading. Although her teachers have been very supportive, she constantly feels odd and unsuccessful. She reads inaccurately and very slowly compared to most ten-year-old readers. The teachers have tried many techniques to improve her reading, but the effects are not very satisfactory. Apart from reading difficulties, Hanna has problems with sports and games at school. She has poor coordination and tires easily when performing physical activities. She also gets bored with school tasks after only a few minutes, even when only listening, and often feels restless and appears ‘dreamy,’ which also makes compensating for her reading problems challenging.

Hanna’s behavioural profile is an example of neurodevelopmental ‘comorbidity.’ She has been diagnosed with dyslexia, attention-deficit/hyperactivity disorder (ADHD) and developmental coordination disorder (DCD) (American Psychiatric Association, 2013). In the present study, we approach children’s development from the perspective of dyslexia (meaning word level reading and spelling difficulties), and its overlap with other co-occurring neurodevelopmental problems (NDPs). A question then arises: Is Hanna’s reading difficulties an unusually complex case of dyslexia or is the co-occurrence with other NDPs the rule?

Dyslexia is a behaviourally defined NDP characterized by severe and persistent difficulties in acquiring fluent and accurate word reading and spelling skills, which cannot be better explained by inadequate instruction, intellectual disability, low chronological age or impairments in hearing or vision (Peterson & Pennington, 2015; World Health Organization, 1992). Apart from weaknesses in word reading, spelling accuracy and fluency, dyslexia is sometimes accompanied or preceded by oral language problems (Snowling, Hulme, & Nation, 2020). According to the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM 5), dyslexia appears under the umbrella of ‘specific learning disorders,’ and there is no longer a requirement for a discrepancy between IQ and reading achievement, for a dyslexia diagnosis to be made (American Psychiatric Association, 2013). Instead, dyslexia can be diagnosed across the IQ spectrum, even though children with higher IQ and strong executive function skills are likely to manage reading comprehension better (Snowling, 2013).

Most research has suggested that dyslexia can be said to affect 3–10% of the population, depending on the exclusionary criteria and the specific cut-offs that are used for its diagnosis (Peterson & Pennington, 2015). Many studies have found that dyslexia is under substantial genetic influence, with heritability estimates typically varying between 50 and 60% (Grigorenko, 2004; Olson & Byrne, 2005; Pennington & Olson, 2005).

For many years, dyslexia has mainly been explained within the context of the phonological theory, which postulates that dyslexic readers have difficulties with (a) processing and manipulating the sound structure of words, (b) grapheme-phoneme (i.e., letter-sound) correspondences (Bradley & Bryant, 1983; Lundberg & Høien, 1989; Snowling, 1995), and (c) rapid automatized naming (RAN) (Wolf & Bowers, 2000). However, recent studies have suggested that dyslexia typically occurs as a result of multiple deficits rather than a single phonological deficit (Carroll, Solity, & Shapiro, 2016; Fletcher & Grigorenko, 2017; Pennington, 2006) and sometimes no phonological problems at all can be observed in diagnosed cases (Snowling et al., 2020; White et al., 2006). According to Bishop (2006), single deficits can often be compensated for, but when two or three impairments co-occur, the negative effect is, at least, additive, making the disorder more profound.
In the case of dyslexia, this idea stems mostly from studies where deficits have been observed in auditory processing, visual attention and motor/balance skills. For instance, researchers have shown in experiments that some attention-related impairments in dyslexia can be linked to deficits in the auditory domain (shifts of attention) and in visual pathways (Jednoróg, Gawron, Marchewka, Heim, & Grabowska, 2014; Zoubrinetzky, Bielle, & Valdois, 2014). Poor readers show a difference in sensory processing, as their sensory input adaptation mechanisms may be impaired for a variety of stimuli: spoken and written language as well as their visual objects (Perrachione et al., 2016). Apart from atypical sensory adaptation, the perception of motion may also be affected in dyslexic readers (Gori, Seitz, Ronconi, Franceschini, & Facoetti, 2016). Some researchers have pointed to the fact that children with dyslexic difficulties often display a high rate of motor problems (Dewey, Wilson, Crawford, & Kaplan, 2000). The cerebellar theory (Nicolson, Fawcett, & Dean, 2001) states, with mixed empirical support, that poor automaticity and motor deficits in individuals with dyslexia may be associated with a dysfunction of the cerebellum (Feng et al., 2017; Vias & Dick, 2017).

Still, many of the above mentioned hypotheses of non-phonological proximal causes of dyslexia are disputable and recent research results have been mixed (Goswami, 2015). In particular, it is not well known if motor and sensory symptoms in children with dyslexia are uniquely linked to dyslexia itself or to other NDPs commonly associated with dyslexia (Ramus, 2003). Accordingly, a better understanding of the contributing factors and correlates of dyslexia is needed both theoretically and to provide adequate support to students. The current study, uniquely, aims at providing new knowledge regarding the overlap between dyslexia and several other neurodevelopmental problems, both symptoms and disorders.

1.1 Co-occurrence of neurodevelopmental problems (symptoms and disorders) in dyslexia

Accumulating evidence suggests that many children with dyslexia meet diagnostic criteria for at least one additional behaviourally defined neurodevelopmental disorder, although specific numbers on these overlaps are scarcely reported. Reading disorders seem to co-exist most clearly with ADHD and motor deficits/DCD (Fawcett & Nicolson, 1995; Gillberg, 2010; Kaplan et al., 2001; Peterson & Pennington, 2015). According to many studies, between 25 and 40% of individuals with ADHD have dyslexic problems and vice versa (August & Garfinkel, 1990; Willcutt & Pennington, 2000). Studies also support the notion that reading problems and dyslexia are more clearly associated with the inattention than the hyperactivity/impulsivity dimension of ADHD and that this association is genetically mediated (Germanò, Gagliano, & Curatolo, 2010; Mascheretti et al., 2017). A study of 179 children receiving special education at their schools reported that of the children who met criteria for a dyslexia diagnosis circa 50% met criteria for another diagnosis, including ADHD and DCD (Kaplan et al., 2001).

The association between dyslexia and autism spectrum disorder (ASD) seems to be even more complex. When it comes to word reading, the cases of children with autism who are extremely skilled in reading are particularly salient (Ostrolenk, d’Arc, Jelenic, Samson, & Mottron, 2017). For instance, Atkin and Lorch (2006) presented a case study of a nonverbal four-year-old boy with autism who had a precocious ability in word reading/decoding, a profile sometimes termed ‘hyperlexia’ (Ostrolenk et al., 2017). Though most of the children with ASD are not hyperlexic, a series of studies has pointed out that word reading/decoding skills in individuals with ASD are occasionally well-developed (Davidson & Weismer, 2014). Two previous studies have shown that among individuals with NDPs an increase in autistic symptoms is in fact linked with better word reading outcomes (Åsberg Johnels, Gillberg, & Kopp, 2019; Clair, Durkin, Conti-Ramsden, & Pickles, 2010). However, it is important to note that reading skills in ASD are highly variable at a group level, with dyslexia-like difficulties also appearing to be more common in children with autism when compared to the general population (Åsberg & Dahlgren Sandberg, 2012; White et al., 2006). Likewise, some children with dyslexia struggle when it comes to psychosocial functioning. In particular, it has been found that dyslexic children may have less pro-social behaviour and less well-developed relations with peers (Parhiala et al., 2015; Russell,
Ryder, Norwich, & Ford, 2015). Summing up, dyslexia is often not a stand-alone ‘specific learning disorder,’ but appears to co-exist with other NDPs even though many questions remain regarding the nature of these overlaps (American Psychiatric Association, 2013) and to what extent they manifest in the general population.

1.2 | General models of overlap between neurodevelopmental problems

A relatively novel concept concerning neurodevelopmental overlap is Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations (ESSENCE) (Gillberg, 2010). ESSENCE covers different kinds of neurodevelopmental difficulties and disorders in order to draw attention to the early onset of symptoms and the universal co-existence among them. According to a range of studies, the co-occurrence of diverse neurodevelopmental disorders is rather the rule than the exception in the clinical reality (Gillberg, 2010; Gillberg & Billstedt, 2000; Kaplan et al., 2001; Lundström et al., 2015). Receiving one neurodevelopmental diagnosis predicts receiving another one concurrently or later on in life, given that a comprehensive assessment of any co-occurring difficulties is provided (Miniscalco, Nygren, Hagberg, Kadesjö, & Gillberg, 2006). Symptoms and behavioural traits can also fluctuate as a function of maturation or changing environmental demands, thus potential needs for diagnoses can be added or changed over time. It should be noted that the model of neurodevelopmental co-existence does not exclude cases of single or ‘pure’ diagnoses, yet compartmentalization into distinct categories should not be a premise (Bishop & Rutter, 2008).

Gilger and Kaplan (2001) have attempted to explain neurodevelopmental co-existence by proposing another concept: atypical brain development. The atypical brain development refers to a general underlying impairment or susceptibility (Oliver, Johnson, Karmiloff-Smith, & Pennington, 2000; Powell & Bishop, 1992), where the neural development in one area contributes to the developing representations in other areas of the brain, thus serving as an integrative concept by addressing the full range of neurodevelopmental symptom overlap (Kaplan et al., 2001). Likewise, recent research examining polygenetic risk has revealed several significant associations between dyslexia risk and genetic liability to several neuropsychiatric and neurodevelopmental disorders and problems (Gialluisi et al., 2020). From this perspective it is very unlikely that a person will show major learning difficulties in only one domain of learning and development, for example, reading, without presenting any other difficulties. Other sources also support this idea by theorizing that multiple skill deficits in various syndromes can be potentially triggered by an interaction between genetic and environmental factors (Geier, 2007; Gilger & Kaplan, 2001). Both Gillberg (2010) and Kaplan et al. (2001) have argued that the term ‘comorbidity’ is inappropriate, taking into consideration the uncertainty as to whether symptoms should be interpreted as separate disorders or various manifestations of the same underlying impairment. Pauc (2005) has furthered this idea by theorizing that the frequent pattern of neurodevelopmental co-existence could be put forward as an argument for lifting the focus from disorders or diagnostic categories per se to the profile of behavioural symptoms in order to best serve the needs of the children in question.

1.3 | The current study

While it is known that reading abilities/disabilities overlap with ADHD and inattentive traits (Mascheretti et al., 2017; Willcutt et al., 2010), present knowledge is limited when it comes to the overlap with other neurodevelopmental disorders and problems. The current work aims to complement previous studies by exploring the phenotypical overlap between dyslexia and a range of other categorically and dimensionally measured NDPs, including ADHD, ASD, DCD and ASP. Identifying problems and disorders co-existing with dyslexia—and their relative prevalence rates—might facilitate our understanding of neurodevelopmental co-existence and help when designing effective support at schools and beyond the school environment. Dyslexic difficulties and NDPs are studied here in the context of a large-scale population-based sample of nine-year-old twins. Since the instruments used to assess
dyslexia—the Short Dyslexia Scale (SDS)—is new as a quite dyslexia screening tool, we also describe measurement characteristics of the SDS, such as the distribution of scores and internal consistency. The twin study design has also allowed us to estimate heritability for suspected dyslexia.

2  |  METHOD

2.1  |  Participants

This study makes use of results obtained in a cohort of twins from the ongoing longitudinal Child and Adolescents Twin Study in Sweden (CATSS), described in detail by Anckarsäter et al. (2011). The aim of the CATSS has been to include all twins born in Sweden since 1992. Starting in 2004, parents of all twins turning 9 years have been invited to participate in a telephone interview. Zygosity is concluded using a panel of 48 single nucleotide polymorphisms (SNPs). In cases where DNA samples were not available, an algorithm of five questions regarding the similarity of twins is used (Anckarsäter et al., 2011). Additionally, the CATSS is linked to the Swedish National Patient Register (NPR), which contains data on all inpatient diagnoses from 1987 through 31st of December 2016, and also includes diagnoses assigned at some outpatient clinics from 2001 and onwards.

The present study includes data from 1,688 twins (49% male) born between January 2008 and December 2009 and gathered through parental telephone interviews. The original sample included 1,880 individuals. In line with previous research on dyslexia we excluded individuals with ICD-10 diagnoses of intellectual developmental disability/mental retardation (F70-F79, n = 9), blindness and no vision (H54, n = 9), and hearing loss (H90-H91, n = 14) (World Health Organization, 1992). Information from the NPR was used to identify children with these diagnoses. Data on dyslexia-related problems were not reported by the parents of 46 individuals. Nine children had one or two missing values on the Short Dyslexia Scale (SDS) and were therefore excluded. Cases with unknown zygosity were also excluded (n = 75). Thirty more individuals were removed because there were no data from their co-twin reported. Hence, the total sample consisted of 1,688 twins (844 twin pairs, 51% females, 34% monozygotic (MZ), 66% dizygotic (DZ)). The CATSS was approved by the Regional Ethical Review Board in Stockholm (DNR 2010/597–31/1; DNR 2016/2135–31).

2.2  |  Measures

2.2.1  |  Dyslexic problems

We used a novel Swedish parent-report dyslexia questionnaire, the Short Dyslexia Scale (SDS), to assess dyslexic difficulties. The SDS is based on items from two existing instruments—the Colorado Learning Difficulties Questionnaire (CLDQ) (Willcutt et al., 2011) and the Five to Fifteen (Kadesjö et al., 2004), and was compiled by Åsberg Johnels & Lundström (unpubl). Parents report if they agree with seven statements regarding their children’s reading and writing skills with a focus on capturing dyslexic difficulties. All statements [(a) He/she has difficulties with spelling; [b] He/she has difficulties with learning to read compared to learning in other areas; [c] He/she has difficulties in understanding what he/she is reading; [d] He/she guesses a lot when he/she reads, [e] He/she does not t like to read (e.g., avoids reading books); [f] He/she has had difficulties to learn letter names, [g] He/she requires extra help with reading] are scored on a 3-point Likert scale (0 for ‘I do not agree,’ 1 for ‘I agree to some extent’ and 2 for ‘I agree’). The total scale score has a minimum of 0 and a maximum of 14. The sum score of the SDS (0–14) is used for the continuous analyses in this study. The SDS has been validated in a community sample of 137 nine-to-ten-years-olds (Parra & Wass, 2016). In that validation study, a high internal consistency of the scale (Cronbach’s $\alpha = .87$) was obtained. Moderate correlations were observed in relation to established tests of reading and spelling, namely the
phonological choice task ($r = -.37$, $p < .001$) and the orthographic choice task ($r = -.47$, $p < .001$) from the Duvan Dyslexia screening test (Wolff & Lundberg, 2003) and a spelling dictation test (Diagnostiska Läs- och skrivprov) ($r = -.41$, $p < .001$) (Järpsten & Taube, 2013). Importantly, an Area Under the Curve (AUC) of 0.80 ($p < .001$) revealed that the SDS had a high diagnostic accuracy, that means, it adequately discriminated between cases with and without suspected dyslexia as defined by test scores on the spelling test.² Parra and Wass (2016) proposed two cut-off values on the SDS: one with high sensitivity (cut-off ≥2; sensitivity = 0.92, specificity = 0.56) for screening purposes and the other one with high specificity (cut-off ≥8; sensitivity = 0.31, specificity = 0.94) as a diagnostic proxy for identifying children with clear problems. For identifying cases with suspected dyslexia in this study, we used the cut-off ≥8 to minimize false positive results.³

2.2.2  |  Neurodevelopmental problems

Neurodevelopmental problems were assessed with the Autism-Tics, ADHD and other Comorbidities inventory (A-TAC) (Hansson et al., 2005). The A-TAC is an easy-to-administer parent interview that is designed to be carried out over the telephone by laymen (Larson et al., 2010). It is designed to screen for virtually all common child and adolescent psychiatric conditions. All questions within the A-TAC were formulated by an expert group at the Gillberg Neuropsychiatry Center/Department of Child and Adolescence Psychiatry at the University of Gothenburg. Parents report if their children are engaged in certain behaviours and/or experience difficulties in different areas. The items are scored on a 3-point Likert scale (0 for ‘no,’ 0.5 for “yes to some extent” and 1 for yes). The A-TAC has been validated cross-sectionally and longitudinally, and is considered to be one of a few screening instruments that take into consideration various aspects of multiple co-existing conditions (Larson et al., 2010; Larson et al., 2013; Mårland et al., 2017). The following subscales are used in the present study (Cronbach’s $\alpha$ from Anckarsäter et al., 2011): ASD (17 items, $\alpha = .86$), ADHD (19 items, $\alpha = .92$), atypical sensory perception (ASP; termed ‘perception’ in A-TAC; items, $\alpha = .62$), coordination problems (an indicator for developmental coordination disorder [DCD], termed ‘motor control’ in A-TAC) (1 item). ASD was measured by three subscales tapping flexibility (5 items, $\alpha = .70$), language (6 items, $\alpha = .66$) and social interaction (6 items, $\alpha = .77$). ADHD was measured by two subscales: impulsiveness and activity (10 items, $\alpha = .87$) and concentration and attention (9 items, $\alpha = .90$).

Screening cut-offs with high sensitivity and diagnostic cut-offs with high specificity have been identified for the NDPs assessed with A-TAC (Larson et al., 2010; Mårland et al., 2017). In the present study, the diagnostic cut-offs were chosen in order to identify children with high levels of symptoms. Such a conservative approach in defining NDPs corresponds well with clinicians’ diagnosis of the named disorders. ASP is the only category that has not been validated in relation to any corresponding current diagnostic group; instead the perception scale has been validated against the diagnostic concept of deficits in attention, motor control and perception (DAMP) and recently found to relate phenotypically and genetically with autistic traits (Taylor et al., 2018). In the following, sensitivity/specificity are indicated for each NDP: ADHD (0.52/0.95), ASD: (0.71/0.95), DCD: (0.28/0.95), ASP: (0.62/0.93) (Larson et al., 2010).

2.3  |  Data analyses

Descriptive statistics of NDPs and dyslexia were calculated for all 1,688 twins. In order to evaluate the internal consistency of the SDS, Cronbach’s $\alpha$ was calculated on all the items. The prevalence of dyslexia was ascertained by using the diagnostic cut-off score established by Parra and Wass (2016).

The following describes how genetic influence on dyslexia (heritability) was estimated. First, probands (i.e., all twins with dyslexia) were identified ($n = 128$). When the co-twin of a proband also has dyslexia the pair is
‘concordant,’ otherwise it is ‘discordant.’ Then, the proband-wise concordance rates were calculated in order to approximate the influence of genes on dyslexia. Concordance rates function as a risk index that can be used to estimate genetic liability for psychiatric disorders and medical diseases (Plomin, DeFries, Knopik, & Neiderhiser, 2013). The proband-wise concordance estimates the likelihood of being affected at the individual level (2C/C + D; C: number of concordant pairs, D: number of discordant pairs). Identical twins (MZ) share 100% of their segregating alleles, while fraternal twins (DZ) on average share 50% of their segregating alleles. A greater concordance rate for dyslexia among MZ twins compared to DZ twins indicates the presence of genetic effects. By calculating the concordance rates in MZ and DZ twins it is possible to determine the relative importance of genes and environment. To estimate the heritability (h²) of dyslexia, Falconer’s formula was used: h² = 2(rMZ – rDZ), where h² is an estimate of narrow-sense heritability (the proportion of phenotypic variance explained by additive genetic effects), and rMZ and rDZ are the within-pair correlations for MZ and DZ twins, respectively (Plomin et al., 2013).

In order to investigate the phenotypical overlap between dyslexia and the other NDPs, the prevalence of NDPs (any NDP, at least two NDPs, at least three NDPs and all four NDPs regarded separately) was compared between the dyslexia group and the non-dyslexia group. Chi-square tests of independence were used to explore if the proportion of NDPs differed significantly between groups. Next, we utilized the dimensional scores of all seven A-TAC subscales (concentration and attention, impulsiveness and activity, language, flexibility, social interaction, motor control and perception) and performed Mann–Whitney U tests in order to examine whether cases with dyslexia but no clinical NDPs still displayed elevated NDPs traits compared with skilled readers.

Finally, a binary logistic regression analysis was applied to explore whether dyslexia as a dichotomous dependent variable can be predicted by specific neurodevelopmental dimensions. The independent variables were the dimensional scores of all seven A-TAC subscales. Twins of a pair are correlated to each other, that is, their observations are not independent. Therefore, before choosing a regression model, a generalized estimating equations (GEE) model was used in order to account for the correlated observations between twins of a pair, treating twin pairs as clusters. For the GEE model a binomial distribution with a logit link and a robust sandwich estimator were used in order to adjust the calculation of standard errors. The results of the GEE model were very similar to the ones from the logistic regression, implying that the correlation between twins did not have a significant impact on the regression parameters. For simplicity, the binary logistic regression model is reported below.

## RESULTS

### 3.1 Characteristics of the SDS

The SDS had excellent internal consistency with Cronbach’s α of .90. This result is very similar to the previously reported internal consistency of α = .87 in 137 nine to ten year-old primary school students (Parra & Wass, 2016). The distribution was highly skewed (skewness = 3.21) with a minimum value of 0 and maximum value of 14 (Figure 1). The mean score in the full sample was M = 1.74 (SD = 3.19). Using the cut-off value of eight, suspected dyslexia was identified in 128 individuals, yielding a population prevalence of 7.6% (95% CI, 6.4–9.0). More males (9.2%) than females (6.0%) were affected (ratio 1.5:1).

### 3.2 Heritability

The proband-wise concordance rates of categorically defined dyslexia were 54% for MZ twins and 30% for DZ twins, indicating the presence of genetic effects. Twin correlations for categorically defined dyslexia were rMZ = 0.51 and rDZ = 0.25, yielding a narrow-sense heritability estimate of h² = 0.52.
In the total sample, the prevalence of NDPs (defined as the presence of at least one of four non-dyslexia NDPs) was 7%. Table 1 presents the prevalence of NDPs depending on the presence of dyslexic difficulties. Chi-square tests of independence indicated highly significant differences in the proportion of NDPs between the dyslexia and the non-dyslexia group (all $p < .001$). As can be seen in the table, the prevalence of NDPs was higher among children with dyslexia compared to children without dyslexia by a factor of more than eight. Within the dyslexia group, somewhat
less than 40% of the children had at least one co-existing NDP. Among all co-existing NDPs in children with dyslexic problems, ADHD and sensory perception problems were the most common ones.

The above analyses revealed that around 40% of the children with dyslexia evidenced at least one NDP. But does that mean that the remaining 60% were completely free from quantitative symptoms of NDPs? Next, we compared children with and without dyslexia, including only children who were below the cut-off for any NDP from both groups. In these analyses we capitalized on quantitative scores in each subdomain of the A-TAC scores. Mann–Whitney U tests revealed significantly higher symptoms in the dyslexia group in five NDP dimensions: problems with concentration and attention, impulsiveness and activity, language, social interaction and perception (all \( p \leq .002 \), Table 2).

### 3.4 NDP dimensions as predictors for dyslexia

A binary logistic regression was performed to assess the impact of co-existing NDP dimensions on the likelihood that children would present with dyslexia. The model contained all seven NDPs dimensions/A-TAC subscales as independent variables, in order to gain a more specific view of their associations with dyslexia when considered collectively. Tests to see if the data met the assumption of collinearity indicated that multicollinearity was not a concern (VIF = 1.3–2.1).

The model was statistically significant, \( \chi^2 \) (7, \( N = 1688 \)) = 230.04, \( p < .001 \), indicating that it was able to distinguish between individuals with and without dyslexia based on the NDP symptom dimensions. As shown in Table 3, four of the independent variables made a unique statistically significant contribution to the model. The numerically strongest predictor of dyslexia was the language dimension with an OR of 1.70. This value indicates that when the score on the language problems dimension increases with 1 point, the odds of having dyslexia increases by 70%. Also, concentration and attention problems (OR = 1.59) as well as atypical sensory perception (OR = 1.35) significantly predicted dyslexia status. The OR for flexibility problems was significantly less than 1 (0.56), indicating that more flexibility problems were associated with a lower risk of dyslexia in the multivariate analysis.

#### TABLE 2 Comparing symptom dimensions of NDPs in children with and without suspected dyslexia where cases with above threshold NDPs were excluded in both groups

| NDPs symptom dimensions               | Dyslexia group \( n = 80 \) M (SD) | No dyslexia group \( n = 1,489 \) M (SD) | Mann Whitney U test | Possible range |
|----------------------------------------|------------------------------------|----------------------------------------|---------------------|---------------|
| Concentration and attention (ADHD)    | 2.77 (2.26)                        | 0.92 (1.35)                            | −8.79 < .001        | 0–9           |
| Impulsiveness and activity (ADHD)     | 1.93 (1.85)                        | 1.02 (1.51)                            | −5.25 < .001        | 0–10          |
| Language (ASD)                        | 0.68 (0.88)                        | 0.22 (0.46)                            | −6.78 < .001        | 0–6           |
| Flexibility (ASD)                     | 0.32 (0.64)                        | 0.28 (0.58)                            | −0.59 .553          | 0–5           |
| Social interaction (ASD)              | 0.46 (0.61)                        | 0.27 (0.51)                            | −3.84 < .001        | 0–6           |
| Motor control (DCD)                   | 0.04 (0.13)                        | 0.02 (0.11)                            | −1.10 .271          | 0–1           |
| Perception (ASP)                      | 0.40 (0.61)                        | 0.22 (0.45)                            | −3.10 .002          | 0–5           |

Abbreviations: ADHD, Attention-Deficit/Hyperactivity Disorder; Any NDP, at least one or more out of four NDPs; ASD, Autism Spectrum Disorder; ASP, Atypical Sensory Perception; DCD, Developmental Coordination Disorder; NDPs, neurodevelopmental problems.
DISCUSSION

According to the definition and cut-off adopted, the prevalence of suspected dyslexia in the studied group was about 8%, which is in line with previous estimates in international and Swedish literature (SBU, 2014; Siegel, 2006; Snowling, 2013). The heritability for dyslexia and reading was estimated at 52%, which also matches previous findings where dyslexia had been assessed with test scores rather than parent ratings (Grigorenko, 2004; Olson & Byrne, 2005; Pennington & Olson, 2005). This result confirms that genes play an important role in dyslexia as assessed with the SDS, as it has been shown in prior dyslexia research (Olson & Byrne, 2005; Pennington & Olson, 2005).

The most important contribution of the current study was the reported high rate of other NDPs in children with dyslexia compared to non-dyslexic readers (by a factor of more than eight). In the dyslexia group, almost 40% of children had at least one additional NDP compared to around 5% in children without dyslexia. This overall conclusion is in line with previous studies that typically used smaller clinical samples and focused on dyslexia and one or a two other NDP groups or dimensions (Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). It is important to note that the prevalence of NDPs is likely to be underestimated considering that we opted to utilize the high diagnostic cut-off for conservative reasons. Had the more lenient (subclinic) cut-off to NDPs status been chosen, an even greater proportion of the dyslexia group would have been flagged as affected. And indeed, further analyses of quantitative NDPs traits showed that subclinical NPD scores were elevated also in children with dyslexia who did not fulfil full criteria for any additional NDP. To our knowledge, there are no previous studies that in such comprehensive manner have explored the presence of several NDPs, defined both categorically and continuously, in dyslexia.

These findings have clear implications for dyslexia research. For many years, research on dyslexia has focused on what is purported as ‘pure’ cases, that is children, who, presumably, only have this one type of ‘specific’ difficulty. Even today, it seems common in dyslexia research to not report on any comorbid conditions at all, or, alternatively, to attempt, more or less ambitiously, to recruit ‘pure cases.’ An increasing number of researchers, however, have questioned the viability of this practice in view of the massive overlap with other NDPs (Russell et al., 2015). For instance, Hulme and Snowling (2013) state in a broader context regarding neurodevelopmental co-occurrence that in some case–control studies:

“Attempts are made to select ‘pure’ clinical cases that do not show signs of comorbid disorders. In practice, however, even when this is attempted it is only ever likely to be partly successful. Furthermore, if comorbidities between different disorders are as pervasive as recent evidence suggests, the selection of ‘pure’ cases may be practically impossible (too many tests would need to be given to each child), perhaps theoretically impossible (excluding all comorbidities will result in no ‘pure’ cases

### Table 3

| NDP symptom dimensions                     | OR  | 95% CI (OR)   | Wald $\chi^2$ (1) | p    |
|-------------------------------------------|-----|---------------|-------------------|------|
| Concentration and attention (ADHD)        | 1.59| 1.41, 1.78    | 60.95             | <.001|
| Impulsiveness and activity (ADHD)         | 0.98| 0.87, 1.09    | 0.21              | .643 |
| Language (ASD)                            | 1.70| 1.30, 2.22    | 14.82             | <.001|
| Flexibility (ASD)                         | 0.56| 0.41, 0.77    | 13.12             | <.001|
| Social interaction (ASD)                  | 1.13| 0.84, 1.52    | 0.64              | .422 |
| Motor control (DCD)                       | 1.47| 0.65, 3.36    | 0.85              | .356 |
| Perception (ASP)                          | 1.35| 1.01, 1.79    | 4.13              | .042 |

Abbreviations: ADHD, Attention-Deficit/Hyperactivity Disorder; ASD, Autism Spectrum Disorder; ASP: atypical sensory perception; DCD, Developmental Coordination Disorder; NDPs, neurodevelopmental problems; OR: odds ratio.
to study), and if it can be done at all such an approach will result in the selection of children who are unrepresentative of the group of children […] we set out to study” (Hulme & Snowling, 2013). At the very minimum, research on dyslexia should be clear as to if, and if so, how, participants were screened for other common NDPs (and what the outcomes of this screening were), in order to better understand the nature and representativeness of the included study samples. According to Hulme and Snowling (2013), the outcome of such assessments should not primarily be used to exclude ‘non-pure cases,’ but rather be used to explore if, and to what extent, the pattern of ‘comorbid’ problems and disorders affect the results of interest.

The findings of the present study also have potential implications for educational and clinical support. When a group of symptoms overlaps between categorical diagnoses it creates a multifaceted condition. It can often be presumed that the higher number of observed ‘atypical’ traits, the worse the clinical prognosis is for an individual. Consequently, children whose behavioural difficulties overlap will need a lot more individual adjustments from the perspective of all the strengths and weaknesses. In our experience, students with multiple diagnoses, such as ADHD and dyslexia, may need much additional support in coping with reading problems way up in higher education.

Difficulties with reading affect many aspects of life including mental health and may persist into adulthood (Ascherman & Shaftel, 2017; Boyes, Leitao, Claessen, Badcock, & Nayton, 2016; Morgan, Farkas, & Wu, 2012). The outcome might likely become more problematic when dyslexia is accompanied by other co-existing neurodevelopmental impairments, and there is indeed some evidence that the combination of dyslexia with oral language problems and ADHD are linked with poorer mental health outcomes (e.g., lower academic and general self-concept) compared with cases who only had difficulties in one domain (McArthur, Castles, Kohnen, & Banales, 2016). Coming back to the example of Hanna, the current study confirms that neurodevelopmental ‘comorbidity’ in dyslexia is common, and not exceptional, although NDPs do not necessarily reach the level of the diagnostic threshold. In order to better understand multifaceted conditions and plan interventions for children like Hanna, an interdisciplinary informed approach is likely needed, where the interventions at schools, clinics and at home should be of a comprehensive nature. Consequently, there may be a need to move from mutually exclusive diagnostic categories to behavioural profiles when trying to understand and help children who struggle with reading.

Among all the co-existing NDPs in children with dyslexic problems, ADHD and ASP were the most common ones. The co-occurrence of dyslexia and ADHD was highly expected (Kaplan et al., 2001; Peterson & Pennington, 2015; Willcutt et al., 2010). Moreover, the results of the logistic regression confirmed a large body of prior research showing that the inattentive dimension of ADHD is more clearly associated with dyslexia than is the hyperactivity/impulsivity dimension (Carroll, Maughan, Goodman, & Meltzer, 2005; Pham, 2016; Willcutt & Pennington, 2000). The existence of “comorbid” problems in the area of sensory perception was a rather more novel finding. While single results have suggested that sensory perception impairments in children with dyslexia may complicate the reading process due to deficits in auditory and/or visual domains (Jednoróg et al., 2014; Perrachione et al., 2016; Zoubrietzyk et al., 2014), much controversy surrounds this issue (Goswami, 2015). Critically, the current study is one of very few that relied on parental ratings in screening for ASP in dyslexia (Armstrong & Nicolson, 2018). Our results suggest that ASP in dyslexia is not only present in psychophysical lab test performances (Jednoróg et al., 2014), but also present in everyday life and indeed evident for parents, which is a finding of potentially great clinical significance. Moreover, the current study is unique in showing the association between atypical sensory processing and dyslexia in a multivariate analysis that also included several other NDP dimensions. Indeed, ASP is regarded to be a persistent symptom in ASD (Tomchek & Dunn, 2007) and common in ADHD (Little, Dean, Tomchek, & Dunn, 2018), and the current study suggests that it also seems to be uniquely linked to dyslexia. Of course, nothing is known about any causal relations here.

Also DCD—that is gross motor clumsiness and poor fine motor skills—was observed more often in children with dyslexic problems, although this association did not persist in the multivariate logistic regression model. The existence of motor abnormalities and delays has been observed both in children with dyslexia, ASD and ADHD (Dewey et al., 2000; Kaplan et al., 2001; Mascheretti et al., 2017). Also, balance and coordination problems in children with NDPs, including
dyslexia, have previously been linked with atypical functioning in cerebellar circuits (Nicolson & Fawcett, 2007; Stoodley, 2016). Whether motor problems have a causal role in reading development is a highly controversial topic. One body of research has conceptualized motor problems as rather unspecific markers of atypical brain development, with no obvious mechanistic association to reading (Ramus, 2003). This latter theory seems to be a parsimonious explanation for our pattern of results. At the same time, motor delays are often overlooked in clinical assessments and therapy is rarely offered. Therefore, it can be important to study and assess the co-occurrence of motor impairments in children with dyslexia in order to ensure proper understanding and help for motor and coordination problems.

Another salient finding was that certain behavioural traits or problem areas within various NDPs may act as particularly clear statistical predictors of dyslexia: (ASD-related) oral language problems and (ADHD-related) inattention was especially closely associated with dyslexia. However, the relationship between NDPs and dyslexia appears to be complex, especially in the case of ASD. Circa 12% of children with dyslexia presented with co-existing symptoms indicative of an ASD. Studies exploring the co-occurrence of ASD and literacy impairments suggest that genes contributing to general language skills may be shared among ASD and dyslexia (Eicher & Gruen, 2015, but see also Galluusi et al., 2020). Since ASD is a complex condition, large variations of behavioural strengths and weaknesses are very often observed. Indeed, reading skills in ASD may vary from poor (i.e., dyslexia) to exceptionally strong (i.e., hyperlexia) (Hulme & Snowling, 2013; White et al., 2006). In the current study, we found that in the multivariate model, increasing scores on the inflexibility subscale of the ASD module decreased the odds of having dyslexia, whereas the language problems typically associated with ASD increased them. This novel result points toward a complex pattern of overlap that needs to be confirmed and specified in future research. It is reasonable that such research might help to explain apparently paradoxical results from previous studies that both dyslexic (Åsberg & Dahlgren Sandberg, 2012; White et al., 2006) and hyperlexic traits are overrepresented in ASD (Åsberg Johnels et al., 2019; Grigorenko et al., 2002; Ostrolenk et al., 2017). We specifically propose the following hypothesis for future investigation: the autism phenotype consists of several related but distinct subdomains (Happé & Ronald, 2008), where some behavioural traits may in fact have a compensatory function for word reading acquisition in children (e.g., close attention to details, extreme adherence to routines, strong and restricted interests), whereas other dimensions (e.g., language problems) are detrimental to word reading and spelling performance.

4.1 Limitations

Our study has at least six potential limitations. First, data were collected through parent-report over the telephone, which may have less validity than reading tests from direct assessment. On the other hand, it can be argued that parents have the best possible insight in their children's development and their observation is clearly of a great value. In addition, the instruments used here have all been validated against established clinical measures and evaluations, and also here we reported on psychometric properties and other characteristics of the dyslexia scale. Second, dyslexic difficulties and NDPs were detected using the high diagnostic cut-offs, which runs the risk of some children with difficulties not having been identified. Nonetheless, using a high cut-off provides a higher certainty of identifying only the individuals with actual reading and spelling problems and other NDPs, which was considered to be of higher priority. Still, given that we were not able to directly assess the individuals classified with dyslexia, we refer to them as having suspected dyslexia. Third, and relatedly, the validity of the dyslexia “label” itself is a contentious topic (Elliott & Grigorenko, 2014). Here we follow a rather pragmatic approach by viewing dyslexia as synonymous with considerable difficulties in word reading and spelling development not associated with intellectual disability, or with severe hearing or visual impairments (see method section for our inclusion and exclusion criteria; cf. also, Snowling et al., 2020). Fourth, the A-TAC modules assessing motor difficulties (DCD) and sensory perception difficulties contain very few items. The DCD module, in particular, albeit having demonstrated construct validity, consists of only one item, making it difficult to nuance the nature of the motor problems. Even so, the module seemed useful as the prevalence of DCD was significantly (ca. 9 times) higher in dyslexic readers (14.8%) compared to non-dyslexic readers (1.7%) in our study. As a complementary analysis we also reran the logistic regression without DCD/motor...
problems, and the results remained the same. Regarding the 3-item perception/ASP scale, though covering both perceptual and sensory problems, it has been argued not to be sensitive to sensory under-responsivity or able to differentiate specific sensory profiles (Taylor et al., 2018). Clearly, a greater insight into atypical sensory reactivity in dyslexia is warranted. Fifth, while a growing body of research has implicated that the existence of overlapping symptoms is often due to partly shared etiologies (Lundström et al., 2015; Pettersson, Anckarsäter, Gillberg, & Lichtenstein, 2013), the current study did not examine the etiological bases of the overlap between dyslexia and other NDPs. However, the findings of the current study will be important for guiding future research, and our data collection is still ongoing in order to conduct such analyses with a sufficient number of participants. Sixth, and finally, one might ask to what extent do the current findings generalize to non-twin samples. While twinning per se comes with risks of complications at birth (Zuppa, Maragliano, Scapillati, Crescimbini, & Tortorolo, 2001), the generalizability to singletons is reasonably high, as the prevalence of NDPs is not, or only slightly, elevated in twins compared to non-twins (Croen, Grether, & Selvin, 2002; Hultman, Sparén, & Cnattingius, 2002).

5 | CONCLUSION

Summarizing our results, they confirm that dyslexia is partly heritable and that the co-occurrence of NDPs in children with dyslexia is very common, especially when also NDP traits are considered. Genetic factors can be an important piece of the puzzle for understanding the complexity of dyslexia and its prevailing overlap with other NDPs. A novel finding is that dyslexic children, apart from ADHD and DCD, may also show symptoms of atypical sensory processing and certain ASD symptoms, although the latter association appears to be complex indeed.

The awareness of neurodevelopmental co-existence should lead to enhanced collaboration among different specialties. Ultimately, we argue that assessments should identify each child's specific functional difficulties and strengths in order to provide tailored understanding and instruction.

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ENDNOTES

1 Since the term comorbidity often presumes independent etiologies it is not always a proper term to describe neurodevelopmental symptom overlap (Gillberg, 2010; Kaplan, Dewey, Crawford, & Wilson, 2001). We therefore mostly use terms such as co-occurrence and co-existence. The term comorbidity is put within quotes whenever it is used.

2 The spelling test (Järpsten & Taube, 2013) is a 20-word dictation task (Cronbach’s $\alpha = .76$) aimed at assessing the ability to spell a target word presented in a sentence context. Regarding the nature of dyslexia in Swedish readers, it has been observed that while word reading accuracy develops with time quite adequately in many dyslexic readers of (semi-)transparent orthographies, yet spelling and phonological decoding and recoding difficulties often persist (SBU, 2014; Wolff & Lundberg, 2003).

3 The SDS has been used also in a parallel clinical study of dyslexia by our research group (Sundqvist et al, in preparation). In that study, the scale distinguished without overlap between children with and without diagnosed dyslexia, and correlated strongly with measures of word and nonword reading, pointing further towards construct validity.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are with institutional approvals available from the corresponding author upon reasonable request.
Gialluisi, A., Andlauer, T. F., Mirza-Schreiber, N., Moll, K., Becker, J., Hoffmann, P., ... Tóth, D. (2020). Genome-wide association study reveals new insights into the heritability and genetic correlates of developmental dyslexia. Molecular Psychiatry, 1–14.

Gilger, J. W., & Kaplan, B. J. (2001). Atypical brain development: A conceptual framework for understanding developmental learning disabilities. Developmental Neuropsychology, 20(2), 465–481.

Gillberg, C. (2010). The ESSENCE in child psychiatry: Early symptomatic syndromes eliciting neurodevelopmental clinical examinations. Research in Developmental Disabilities, 31(6), 1543–1551.

Gillberg, C., & Billstedt, E. (2000). Autism and Asperger syndrome: Coexistence with other clinical disorders. Acta Psychiatrica Scandinavica, 102(5), 321–330.

Gori, S., Seitz, A. R., Ronconi, L., Franceschini, S., & Facoetti, A. (2016). Multiple causal links between magnocellular–dorsal pathway deficit and developmental dyslexia. Cerebral Cortex, 26(11), 4356–4369.

Goswami, U. (2015). Sensory theories of developmental dyslexia: Three challenges for research. Nature Reviews Neuroscience, 16(1), 43–54.

Grigorenko, E. L. (2004). Genetic bases of developmental dyslexia: A capsule review of heritability estimates. Enfance, 56(3), 273–288.

Grigorenko, E. L., Klin, A., Pauls, D. L., Senft, R., Hooper, C., & Volkmar, F. (2002). A descriptive study of hyperlexia in a clinically referred sample of children with developmental delays. Journal of Autism and Developmental Disorders, 32(1), 3–12.

Hansson, S. L., Svanströmörjvall, A., Rastam, M., Gillberg, C., Gillberg, C., & Anckarsäter, H. (2005). Psychiatric telephone interview with parents for screening of childhood autism–tics, attention-deficit hyperactivity disorder and other comorbidities (A-TAC): Preliminary reliability and validity. The British Journal of Psychiatry, 187(3), 262–267.

Happé, F., & Ronald, A. (2008). The ‘fractionable autism triad’: A review of evidence from behavioural, genetic, cognitive and neural research. Neuropsychology Review, 18(4), 287–304.

Hulme, C., & Snowling, M. J. (2013). Developmental disorders of language learning and cognition. Hoboken, NJ: John Wiley & Sons.

Hultman, C. M., Sparén, P., & Cnattingius, S. (2002). Perinatal risk factors for infantile autism. Epidemiology, 13(4), 417–423.

Järpenst, B., & Taube, K. (2013). DLS handledning: För skolär 2 och 3. Stockholm: Hogrefe Psychologiförlaget.

Jednoróg, K., Gawron, N., Marchewka, A., Heim, S., & Grabowska, A. (2014). Cognitive subtypes of dyslexia are characterized by distinct patterns of grey matter volume. Brain Structure and Function, 219(5), 1697–1707.

Kadesjö, B., Janols, L.-O., Korkman, M., Mickelsson, K., Strand, G., Trillingsgaard, A., & Gillberg, C. (2004). The FTF (five to fifteen): The development of a parent questionnaire for the assessment of ADHD and comorbid conditions. European Child & Adolescent Psychiatry, 13(3), iii3–iii13.

Kaplan, B. J., Dewey, D. M., Crawford, S. G., & Wilson, B. N. (2001). The term comorbidity is of questionable value in reference to developmental disorders: Data and theory. Journal of Learning Disabilities, 34(6), 555–565.

Larson, T., Anckarsäter, H., Gillberg, C., Ståhlberg, O., Carlström, E., Kadesjö, B., ... Gillberg, C. (2010). The autism-tics, AD/HD and other comorbidities inventory (A-TAC): Further validation of a telephone interview for epidemiological research. BMC Psychiatry, 10(1), 1.

Larson, T., Lundström, S., Nilsson, T., Selinus, E. N., Råstam, M., Lichtenstein, P., ... Kerekes, N. (2013). Predictive properties of the A-TAC inventory when screening for childhood-onset neurodevelopmental problems in a population-based sample. BMC Psychiatry, 13(1), 233.

Little, L. M., Dean, E., Tomchek, S., & Dunn, W. (2018). Sensory processing patterns in autism, attention deficit hyperactivity disorder, and typical development. Physical & Occupational Therapy in Pediatrics, 38(3), 243–254.

Lundberg, I., & Håkanson, T. (1989). Phonemic deficits: A core symptom of developmental dyslexia? The Irish Journal of Psychology, 10(4), 579–592.

Lundström, S., Reichenberg, A., Melke, J., Råstam, M., Kerekes, N., Lichtenstein, P., ... Anckarsäter, H. (2015). Autism spectrum disorders and coexisting disorders in a nationwide Swedish twin study. Journal of Child Psychology and Psychiatry, 56(6), 702–710.

Mårland, C., Lichtenstein, P., DeIlInnocenti, A., Larson, T., Råstam, M., Anckarsäter, H., ... Lundström, S. (2017). The autism-tics, ADHD and other comorbidities inventory (A-TAC): Previous and predictive validity. BMC Psychiatry, 17(1), 403.

Mascheretti, S., De Luca, A., Trezzi, V., Peruzzo, D., Nordio, A., Marino, C., & Arrigoni, F. (2017). Neurogenetics of developmental dyslexia: From genes to behavior through brain neuroimaging and cognitive and sensorial mechanisms. Translational Psychiatry, 7(1), e987.

McArthur, G., Castles, A., Kohen, S., & Banales, E. (2016). Low self-concept in poor readers: Prevalence, heterogeneity, and risk. PeerJ, 4, e2669.

Miniscalco, C., Nygren, G., Hagberg, B., Kadesjö, B., & Gillberg, C. (2006). Neuropsychiatric and neurodevelopmental outcome of children at age 6 and 7 years who screened positive for language problems at 30 months. Developmental Medicine and Child Neurology, 48(5), 361–366.
Willcutt, E. G., Betjemann, R. S., McGrath, L. M., Chhabildas, N. A., Olson, R. K., DeFries, J. C., & Pennington, B. F. (2010). Etiology and neuropsychology of comorbidity between RD and ADHD: The case for multiple-deficit models. Cortex, 46(10), 1345–1361.
Willcutt, E. G., Boada, R., Riddle, M. W., Chhabildas, N., DeFries, J. C., & Pennington, B. F. (2011). Colorado learning difficulties questionnaire: Validation of a parent-report screening measure. Psychological Assessment, 23(3), 778–791.
Willcutt, E. G., & Pennington, B. F. (2000). Comorbidity of reading disability and attention-deficit/hyperactivity disorder: Differences by gender and subtype. Journal of Learning Disabilities, 33(2), 179–191.
Willcutt, E. G., Pennington, B. F., Olson, R. K., Chhabildas, N., & Hulslander, J. (2005). Neuropsychological analyses of comorbidity between reading disability and attention deficit hyperactivity disorder: In search of the common deficit. Developmental Neuropsychology, 27(1), 35–78.
Wolf, M., & Bowers, P. G. (2000). Naming-speed processes and developmental reading disabilities: An introduction to the special issue on the double-deficit hypothesis. Journal of Learning Disabilities, 33(4), 322–324.
Wolff, U., & Lundberg, I. (2003). A technique for group screening of dyslexia among adults. Annals of Dyslexia, 53(1), 324–339.
World Health Organization. (1992). ICD-10 Classifications of Mental and Behavioural Disorder: Clinical Descriptions and Diagnostic Guidelines.
Zoubrintzky, R., Bielle, F., & Valdois, S. (2014). New insights on developmental dyslexia subtypes: Heterogeneity of mixed reading profiles. PLoS One, 9(6), e99337.
Zuppa, A. A., Maragliano, G., Scapillati, M. E., Crescimbini, B., & Tortorolo, G. (2001). Neonatal outcome of spontaneous and assisted twin pregnancies. European Journal of Obstetrics & Gynecology and Reproductive Biology, 95(1), 68–72.

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