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SHORT COMMUNICATION

The incidence of prematurity or low birth weight for gestational age among children with dyslexia

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Dyslexia is characterized as a specific deficit of reading acquisition. It is one of the most common learning disabilities, persists throughout life and can have a major impact on someone's socio-economic success in our knowledge society. Preterm birth is associated with an increased risk of developmental problems including reading disabilities later in life (1–3). Poor reading skills are reported in preterm borns after controlling for IQ, and in particular, they are characterized by slow reading (2,3). In the last half of gestation, major structural maturational events take place at a neural level, and even at 34 weeks of gestational age, the brain weight is only 65% of its term weight (4). Disruptions to brain development owing to being born preterm place individuals at risk of behavioural, educational and cognitive dysfunction (5), both in very preterm (<32 weeks of gestation) and moderately preterm (32–36 weeks of gestation) born children (1,5). In particular, Andrews et al. (6) revealed an association between being born preterm, white-matter disruptions and developing reading disabilities. Another group of at-risk infants are those with reduced foetal growth. Previous literature repeatedly reported that a low birth weight for gestational age (BWGA), indicating impaired foetal growth, is a negative determinant of these children's academic achievements, including their reading and spelling skills (7,8), although the literature is somewhat equivocal with some studies reporting no dysfluent reading in children with a low BWGA (9), or children catching up in reading achievements in their later childhood years (10).

Our goal was to examine whether born preterm or with low BWGA are risk factors for developing dyslexia at school age. If these are indeed risk factors, one might expect an elevated prevalence of prematurity or a lower BWGA within populations of children with dyslexia. Therefore, we addressed the question from a reversed perspective than that of the pertinent literature and tested the prevalence of reduced foetal growth and preterm in a large sample of children with dyslexia.

We collected data from a cohort of children who were referred to a nationwide clinical care centre for reading disorders in the Netherlands. To be included in the study, a child had to have specific and persistent reading problems at school and was attending grade 3–6 of primary education (age 7–12 years). Following these criteria, a total of 709 children were selected. Next, we divided the selected children into those with severe dyslexia and those with mild reading disabilities. Severe dyslexia was defined according to the criteria in the Dutch healthcare system, basically implying that all of the following three criteria were met: (i) reading was at least 1.5 SD below average or reading was at least 1 SD below average and spelling was at least 1.5 SD below average, (ii) performance on at least two of six administered phonological tasks was 1.5 SD below average, (iii) the child had shown a poor response to intervention provided at school. These criteria resulted in 452 children with severe dyslexia, and 257 children with specific reading disabilities who did not meet the criteria for severe dyslexia (hereafter referred to as mild dyslexia). Participant characteristics are presented in Table 1.

For all the children, data on birth weight and gestational age (based on recorded information of early ultrasound dating) were collected. Z scores of BWGA were calculated from Dutch reference standards for birth weight for gestational age (allowing for sex, parity and ethnicity) (11). Using this Z score as a proxy for foetal growth, we were able to analyse growth retardation as a continuous variable, allowing greater precision than the binary SGA score.

Table 1 Descriptive characteristics of the group with severe dyslexia and the group with mild dyslexia

	Severe dyslexia (n = 452) mean (SD)	Mild dyslexia (n = 276) mean (SD)
IQ*	102.55 (11.40)	106.60 (12.45)
SES	2348.30 (779.91)	2512.03 (978.51)
Age (year:month)	9:5 (1:4)	9:1 (1:0)
Sex (M/F)	276/176	169/107
Gestational Age (weeks)	39.52 (2.30)	39.51 (2.14)
<32	1.3%	1.1%
32–36	6.0%	5.8%
>37	92.7%	93.1%
Birth weight (g)	3451.38 (626.52)	3511.37 (662.43)
BWGA Z-score	0.30 (1.10)	0.41 (1.29)
SGA (%)	7.1%	7.8%
Reading Fluency (SS)	73.23 (4.91)	84.18 (11.19)
Spelling Accuracy (SS)	79.75 (9.81)	86.13 (12.28)
Spelling Rate (SS)	80.05 (11.06)	86.95 (11.81)

SES = socioeconomic status; BWGA = birthweight for gestational age; SGA = small (lowest 10%) for gestational age; SS = standard score ($M = 100$; SD = 15).
*Full-scale IQ score WISC-III.

Socio-economic status (SES) was operationalized as the average household income of subject's neighbourhood. All children took a battery of standardized measures of reading fluency (number of words read correctly within 1 min), spelling (speed and accuracy of spelling knowledge) and phonological abilities (speed and accuracy of both letter–speech sound matching and phoneme awareness and rapid naming of letters and numbers) (12). A priori calculation indicated a power of our statistical tests of at least 0.99 to detect significant effects [under the condition of $\alpha = 0.05$ and a small-to-moderate effect size (χ^2: w = 0.2, multiple regression: $f^2 = 0.10$, *t*-test: d = 0.20)] for the severe dyslexia group and a power of at least .91 for the mild dyslexia group.

Multiple regression analyses were conducted to examine the relation between gestational age (GA) and reading and spelling skills within our samples of disabled readers. In each regression, sex and SES were entered first to statistically control for their potential as a confounding variable. The results revealed that age and SES were not significant predictors of reading and spelling skills (all β between −0.10 and 0.14, all $p > 0.05$), except for a significant association between sex and spelling accuracy in the severe dyslexia group ($\beta = -0.18$, $p < 0.05$, males making more spelling errors). Entered next, gestational age was not predictive for reading fluency ($\beta = 0.03$, $p = 0.60$) or spelling accuracy ($\beta = -0.03$, $p = 0.63$) in the severe dyslexia group. GA was a significant predictor for spelling rate in this group ($\beta = 0.12$, $p < 0.05$), albeit with a very small effect size ($f^2 = 0.01$), indicating that a 6-week increase in GA results in a one-third SD increase in spelling rate. For those with mild dyslexia, no significant associations between gestational age and outcome variables were found (reading fluency: $\beta = 0.01$, $p = 0.85$; spelling accuracy: $\beta = -0.01$, $p = 0.91$; spelling rate: $\beta = 0.05$, $p = 0.47$). Additionally, a chi-square test was used to analyse whether gestational ages of children with reading disabilities were below the national average. In accordance with the national normative data, we divided gestational age into three groups: very preterm (<32 weeks), moderately preterm (32–36 weeks) and at term (>37 weeks). The results revealed that the distribution of gestational ages of children with severe dyslexia did not differ from the national population distribution (1.7% very preterm, 6.7% moderately preterm, 91.6% at term, respectively, in the Netherlands in 2001) (13), $\chi^2_2 = 0.29$, $p = 0.86$. The distribution of gestational ages of children with mild dyslexia did not differ from the national population distribution as well, $\chi^2_2 = 0.60$, $p = 0.74$.

Multiple regression analyses were conducted to examine the relation between BWGA and reading and spelling skills. Again, sex and SES were entered first to statistically control for their potential as a confounding variable. The results showed that after controlling for the effects of sex and SES, BWGA did not account for a significant amount of the variance in reading fluency ($\beta = 0.06$, $p = 0.32$), spelling accuracy ($\beta = 0.10$, $p = 0.07$) or spelling rate ($\beta = -0.02$, $p = 0.68$) in the severe dyslexia group. BWGA did also not explain a significant proportion of unique variance in the literacy performances of the mild dyslexia group (reading fluency: $\beta = 0.01$, $p = 0.86$; spelling accuracy: $\beta = 0.08$, $p = 0.29$; spelling rate: $\beta = -0.10$, $p = 0.17$). To test whether dyslexic children have a lower than average BWGA, we used one sample t-tests (one sided). The results revealed no reduced foetal growth in the severe dyslexia group ($t = 5.86$, $p = 1.00$) nor in the mild dyslexia group ($t = 5.32$, $p = 1.00$).

To our knowledge, this is the first report on the prevalence of children born preterm or with reduced foetal growth within a population of children with dyslexia. The results showed no higher prevalences of preterm or SGA borns in children with dyslexia than in the general population, despite the fact that we included large sample sizes and, thus, adequate power (>0.99 for severe dyslexia, >0.91 for mild dyslexia) to detect small-to-moderate effects.

As noted previously, several longitudinal studies on children born preterm and SGA revealed poorer reading skills later in life. Notably, besides these reading difficulties, poorer academic achievements in general, including arithmetic difficulties, poor executive functioning, as well as behavioural and attentional problems were also reported in these groups of children. In contrast, the present study examined two groups of children with a specific, singular reading deficit. We believe our study therefore contributes to the evidence based on the sequelae of prematurity or reduced foetal growth at birth, by indicating that being born preterm or with low BWGA and the associated vulnerabilities in brain development do not increase the risk of developing dyslexia later in life, but in contrast, appear to increase risks for poorer reading acquisition as part a broader complex of learning, attentional and/or behavioural disorders.

Neurological findings seem to be in line with this conclusion. Evidence on the neurological basis of dyslexia

provides strong indications of a specific neural deficit in the audio-visual integration of letters and speech sounds, localized in superior temporal brain regions (14). Being born preterm, on the other hand, may disrupt general neuronal organizational processes, such as growth in gyri, sulci, synapses and dendritic arborization occurring during the final weeks of gestation (4,15). This appears to make them more vulnerable to diffuse neurodevelopmental disorders, instead of a disorder with a more localized neurocognitive basis, as is dyslexia.

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