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ORIGINAL RESEARCH

Familial Dyslexia in a Large Swedish Family: A Whole Genome Linkage Scan

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Abstract There is a compelling body of evidence that developmental dyslexia runs in families and seems to be highly inheritable. Several investigations during the last two decades have shown possible locations of genes that might be involved in dyslexia, including regions of chromosomes 1, 2, 3, 6, 11, 13, 15 and 18. In addition, six candidate genes (KIAA0319, DYX1C1, DCDC2, ROBO1, MRPL19 and C2ORF3) seem to be related to dyslexia. The present study carried out a whole genome scan in a six-generation pedigree. In addition to literacy skills the assessment included cognitive skills and records concerning the history of reading and writing ability. Thirty-five percent were regarded as dyslexic in the family. A linkage analysis using both a quantitative and a qualitative approach has been performed. No evidence was obtained to support the hypothesis that the transmission of dyslexia in this pedigree is due to a highly penetrant major gene, and previous linkage findings were not replicated; however, power in this small study was not adequate to confirm linkage of genes with small to moderate effects. The results were discussed in relation to diagnostic procedures and sample characteristics.

Keywords Dyslexia · Heredity · Linkage · Chromosomes · Genes

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Introduction

Dyslexia is a specific reading and writing disability that affects approximately 5–10% of the population. The primary problem in dyslexia is a weakness in the rapid, fluent and accurate decoding of written words caused by a shortcoming in the phonological system (Høien and Lundberg 2001; Ramus and Szenkovits 2008; Stanovich and Siegel 1994). An abundance of research has demonstrated that it is highly inheritable (Barr and Couto 2007; DeFries and Gillis 1993; Gayán and Olson 2003; Hallgren 1950, 2003; Lubs et al. 1993; Fisher and Smith 2001; Grigorenko 2005; Smith 2007). Grigorenko (2001) reviewed eight family studies, including in all 516 families, and found a median value of 37% of reading problems in parents of children with reading difficulties. A study by Wadsworth et al. (2002) showed no parent-offspring correlations in adoptive families regarding reading performance in contrast to non-adoptive families. Many other studies give strong evidence for a genetic influence on reading and writing disabilities (for recent reviews see Olson 2007; Scerri and Schulte-Körne 2010; Wagner 2005).

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So far at least nine dyslexia susceptibility loci have been identified. Smith et al. (1983) were the first to report a linkage of dyslexia to a specific chromosome on region 15. After that, genetic loci for dyslexia have been suggested on chromosomes 1, 2, 3, 6, 11, 18 and X (Cardon et al. 1994; Grigorenko et al. 2001; Fagerheim et al. 1999; Hsiung et al. 2004; Nopola-Hemmi et al. 2001; Grigorenko et al. 1997; Petryshen et al. 2001; Fisher et al. 2002). In a recent study by Bates et al. (2007) suggestive support was found for linkage on chromosomes 4 and 17. Candidate genes for dyslexia have also been suggested (DYXICI, Taipale et al. 2003; ROBO1, Hannula-Jouppi et al. 2005; DCDC2, Meng et al. 2005; KIAA0319, Cope et al. 2005a; MRPL19 and C2ORF3, Anthoni et al. 2007). In general, dyslexia is most likely determined by a number of genes with small to moderate effects where the genes contribute both to a general and to an explicit phenotype (Smith 2007). However, highly penetrant genes have been found in other studies (Fagerheim et al. 1999).

Quite a few investigations have failed to replicate earlier genetic findings (e.g. Brkanac et al. 2007; de Kovel et al. 2008; Cope et al. 2005b; Field and Kaplan 1998; Petryshen et al. 2000; Schumacher et al. 2006; Svensson 2003) and reasons for failures have been discussed (Barr and Couto 2007; Fisher and DeFries 2002; Grigorenko 2005) focusing on three main issues: the ascertainment procedure, the choice of analytical method and the characteristics of the phenotype (Fisher and DeFries 2002; Grigorenko 2005; Grigorenko et al. 2006, 2007).

In an earlier study by Svensson (2003) we carried out linkage analyses, in an extended family with high incidence of dyslexia, on selected markers at candidate regions on chromosomes 2, 3, 6, 13, 15 and 18 in an attempt to replicate earlier findings by other researchers. We used both a quantitative and qualitative approach when analyzing data. However, none of the selected candidate regions could be replicated. The investigated regions, however, only covered a small fraction of the genome. Thus, there is a risk that we might have missed regions (Fisher and DeFries 2002).

The purpose of the present study was to complete a whole genome scan on the same family as in Svensson (2003), thereby increasing the possibility of finding genetic loci for dyslexia, and to search for a highly penetrant major gene.

Method

Participants

One multiplex family (n = 62) was recruited from a local reading and writing center (LUK) in a county in Sweden. The proband was tested at LUK and diagnosed as having severe reading and writing difficulties. We then tested a six-generation family (n = 62). The participants in the family were 36 men and 26 women, whose age varied between 7 and 92 years with an average age of 36 years. Sixteen adults and 6 children were regarded as dyslexic, and 10 persons were regarded as uncertain. The remaining members of the family (30) were considered to have normal reading skills. Thus, in this family 35% were defined as dyslexic.

The participants above 19 years (45) had in average been in school for almost 12 years which is representative of the educational level in Sweden. Furthermore, approximately 30% of the family members had educations on university level. SCB (Statistics Sweden 2009) reported recently that 23% of the Swedish population have studied at university level.

Assessment

The test battery in the present investigation is similar to that used in many other genetic studies concerning dyslexia (Grigorenko et al. 1997; Fagerheim et al. 1999; Fisher et al. 1999; Kaplan et al. 2002; Olson et al. 1989). In total, 10 reading and spelling tests (word recognition, oral word reading, letter chain, orthographic choice, phonological choice, pseudo-word reading, spoonerism, spelling, reading comprehension, listening comprehension) covering both phonological and orthographic decoding skills as well as a self-reported history of reading ability, years in school and short-term memory were included. Furthermore, data from a non-verbal intelligence test (Raven 1995) were available to exclude low extremes (below the 10th percentile) on this test. No one was excluded for that reason. The prevailing consensus view among dyslexia researchers is that phonological dysfunction is a core symptom or a marker of developmental dyslexia. For this reason, a carefully selected set of valid and reliable indicators of phonological functions was used. More information regarding the test battery is described elsewhere (Svensson 2003).

Procedure

Fifty-six of the participating family members completed tests in the assessment battery. For a few participants there were missing values of some of the tests. Three participants only completed a few of the tests. One of them was regarded as dyslexic on the basis of a history of reading and writing difficulties, and the other two were classified as uncertain. For 10–13-year-old the tests were adapted to their age. Two 9-year-old children completed some reading tests and an interview. Both of them were regarded as dyslexic. For one child below the age of 9 only personal records from the parents were available. Children below

the age of 7 were not included in the investigation. The total testing time for each individual amounted to nearly one and a half hours. Blood samples were collected from all the 62 participants.

The ethical committee of the University of Gothenburg approved this study (Dnr: 45-09, 2009-03-03).

Genotyping

To reduce genotyping costs DNA was isolated from whole blood from the 47 samples in the study that were considered most informative for linkage (see Fig. 1). For the genotyping, 250 ng of total genomic DNA was used for analysis on the GeneChip Human Mapping 10K Array Xba 142 2.0 (Affymetrix, Santa Clara, CA). The restriction enzyme Xba 1 (New England Biolabs) was used to digest the DNA, and an adapter was ligated to the fragmented DNA. Each sample was amplified using generic PCR primers. The amplified PCR products were fragmented and labeled according to the manufacturer's protocol. The DNA was hybridized to the GeneChip Human Mapping 10K Array at 48°C for 16–18 h. Washing, staining and scanning, using a GCS3000 scanner (Affymetrix) of the probe array, were performed according to the manufacturer's protocol. The analysis and allele calling of the genotype data were performed with the Affymetrix software Gene Chip Operating Software (GCOS9) (Affymetrix) and GeneChip DNA Analysis software (GDAS) (Affymetrix).

Criteria for dyslexia when using the qualitative approach

To be regarded as dyslexic the participants had to fail at least two tests of phonological ability together with having a positive history of reading problems. In addition, if there was no self-report of reading and writing disabilities, the participant had to fail on at least three phonological tests and two manifest tests of literacy such as word recognition and orthographic choice. The participants were regarded as having failed on a test if they performed below 1 SD compared to the norms on these tests.

The cut-off score of 1 SD has been used in several previous studies (van der Leij et al. 2001; Nopola-Hemmi et al. 2001; Schulte-Körne et al. 1998). For a discussion of the cut-off issue see Siegel (1999). In some uncertain cases the diagnosis was based on all the tests. The interview and the questionnaire were also used as a complement to either confirming or rejecting a dyslexic diagnosis, especially in uncertain cases and for children below 13 years. Those participants that failed on two to four tests were classified as uncertain cases if there were no reports of reading and writing disabilities.

Statistics

A parametric linkage analysis was performed, using a dominant model assuming a rare susceptibility allele (0.1%), reduced penetrance (90%) and a phenocopy rate of 3%. Allegro (Gudbjartsson et al. 2005) was used. Due to computation limits the family was split into two branches. The power was simulated and estimated to >95%. For the quantitative trait locus (QTL) analysis we focused the analysis on the four phenotypes that showed the highest heritability in the family. These were orthographic coding, spelling, non-word reading time and non-word reading error. A variance component analysis with school years as covariate was used as a QTL linkage method, as implemented in the Solar software (Almasy and Blangero 1998).

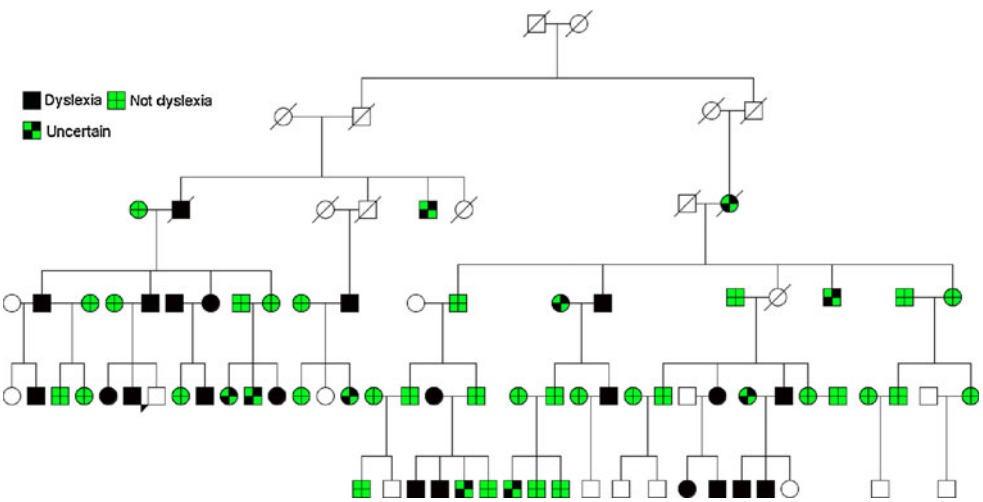

Fig. 1 A six-generation family with dyslexia

Results

Thirty-five percent were regarded as dyslexic and 16% were considered as uncertain cases in the family. Among the males 47% were regarded as dyslexic compared to 19% among the females.

The average genotype call rate was 95%. Mendelian errors were detected in 4% of the SNPs. These markers were removed. The results of the parametric linkage analysis on the dyslexia phenotype as well as the QTL analysis on the four phenotypes with the highest heritability are shown in Fig. 2. In addition to the presented results we ran a QTL analysis with all the other test variables and also a parametric linkage analysis with different settings, such as affected-only analysis (data not shown). Neither of the analyses revealed any regions of interest in the whole family or in any of the two branches.

Discussion

The share of dyslexics in this family is close to what has been reported in other family studies (Fagerheim et al. 1999; Nopola-Hemmi et al. 2001). None of the earlier reported regions for dyslexia could be replicated. This is not surprising since these regions are associated to genes with small to moderate effects. This single large pedigree has no power to detect such genes, but high power to detect the presumably major gene causing the phenotype pattern in the family. However, we did not find any new regions of interest either. Since the resolution is sufficient and the genotyping success rate acceptable, the reason for the failure may be in the phenotypes. A model with slightly reduced penetrance and a substantial phenocopy rate may have a few phenotypes that do not segregate as the genetic markers, but as the amount or errors grow larger the

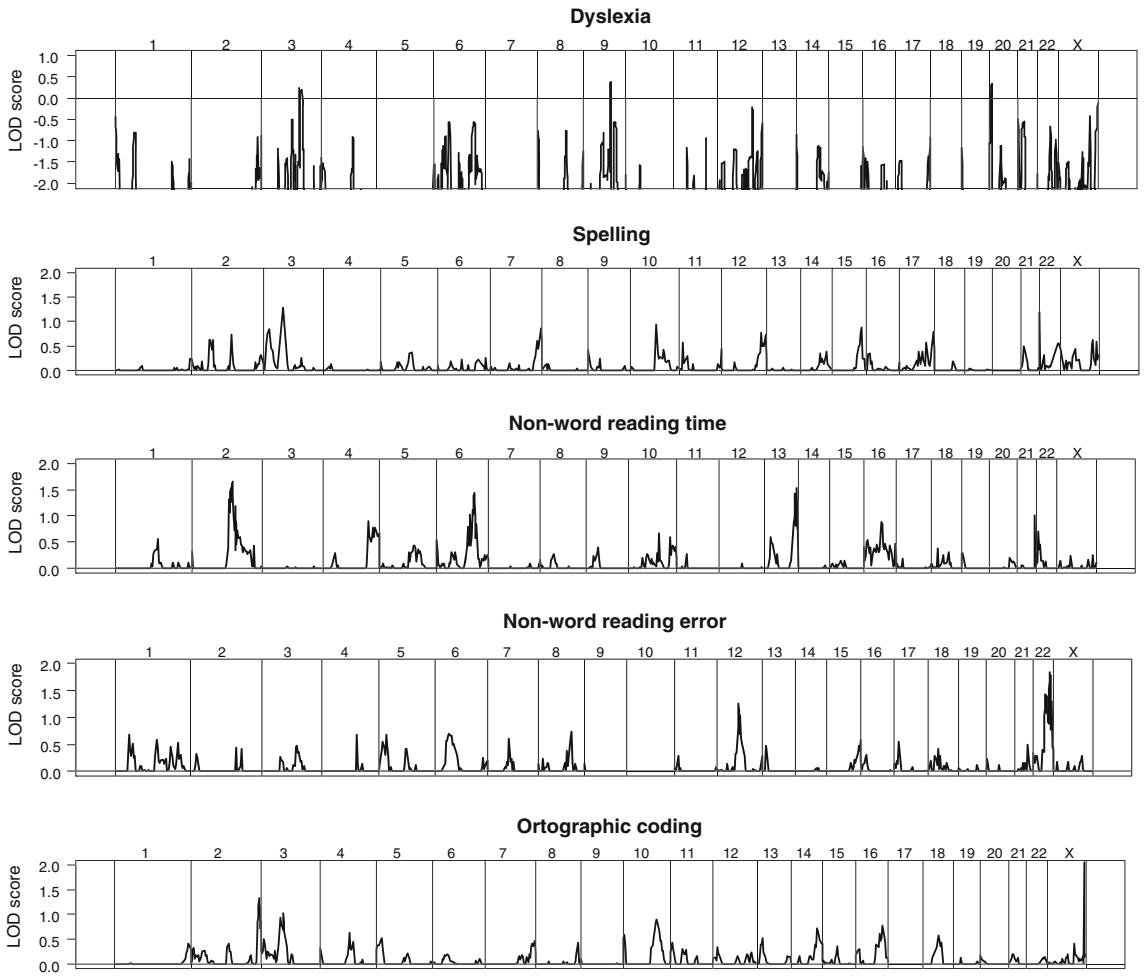

Fig. 2 Lod score plots for parametric linkage and QTL analysis

linkage signal will be destroyed. While the QTL analysis does not suffer from misclassification in the same way, the quantitative phenotypes do suffer from large variations which are not due to dyslexic genetics.

Given the high proportion of dyslexia in the family, it might be surprising that we did not find any genetic markers for dyslexia. However, null results are not exceptional in the domain of genetics and dyslexia. The following studies clearly illustrate that the results regarding genetic findings can be contradictory. In a study by Cope et al. (2005a) it was quite convincingly shown that DYX1c1 is not a susceptible gene for dyslexia. However, in an investigation by Brkanac et al. (2007) it was reported that in their sample the DYX1c1 gene most likely contributed to dyslexia. Furthermore, findings on 6p are the most reported candidate genes for dyslexia. Nevertheless, in studies by Zhou et al. (2008) and Brkanac et al. (2007) no evidence for loci on 6p was found.

In the most recent studies regarding genes and dyslexia researchers have dissected the dyslexia phenotype into several cognitive functions, e.g. phonological awareness, phonological coding and working memory. The task non-word repetition (designed to measure phonological memory) is often reported as one crucial measure when diagnosing dyslexia. In the study by Brkanac et al. (2007) the OTL approach was used, giving support for the phenotype non-word repetition on 4p and 12p. In contrast, Zhou et al. (2008) argued that the non-word repetition showed little phenotypic correlation with dyslexia or other quantitative traits even if there were linkage peaks on 11p and 15q.

The family in this investigation had a very evident dyslexia pedigree. Although a few subjects may be misclassified, the risk of facing genetic heterogeneity is small in this extended family compared to samples with many nuclear families. A plausible explanation of the null results in this study is the phenotype problem regarding both the cut-off procedure and the nature of the tests. We set the cut-off to 1 SD below means for test norms or comparison data). This cut-off might have been too high. Earlier investigations have reported that more severely affected samples tend to show stronger linkages (for a review see McGrath et al. 2006). However, to be regarded as dyslexic the participants had to report a history of reading and writing disabilities and to fail on at least two phonological tests. This cut-off seems to be an equivalent sharp criterion for dyslexia comparing to other studies (de Kovel et al. 2008; Wigg et al. 2004). There were quite a few that we considered as uncertain cases. They failed on several tests (but not on a sufficient number of tests) but reported no difficulties with the written language. We set up a more rigorous cut-off for those participants with no reports of reading and writing disabilities, i.e. the participants had to fail on at least five tests, but there is still a risk that we have diagnosed them as uncertain even though they might have belonged to the affected group. It is also possible that some of the uncertain were "garden-variety poor readers", i.e. they had reading and writing problems related to other factors than dyslexia. Other important factors to consider include age and years in school. It is not self-evident what norms should be used when classifying a 45-year-old person with 7 years in school.

There is no doubt about the importance of phonological ability in preschool children. However, the phonological ability seems not to have the same dignity in adults (Frost et al. 2005; Gathercole 2006; Uppstad and Tønnessen 2007; Snowling 2008) making the phenotype unstable across ages Furthermore, for some of the tests there were no norms available, especially not for the elderly participants. Thus, the characteristics of the tests and available norms are crucial when deciding the phenotype and might be one explanation behind the difficulty to find a sharp and "global" phenotype, not just in this investigation.

Grigorenko (2005) and Grigorenko et al. (2006) suggested that one way to overcome problems with the phenotype and genotype might be to combine linkage results and to merge samples from different investigations. In a forthcoming study we will merge our sample with others from different languages and orthographies. This will increase the sample size and enable higher comparability concerning tests, test settings and norms.

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