Who will develop dyslexia? Cognitive precursors in parents and children

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Children with dyslexia are characterized by slow and laborious reading. Dyslexia negatively affects educational and occupational attainment. Therefore, it is important to increase our understanding of the causes of dyslexia.

Studies comparing mono- and dizygotic twins have revealed that dyslexia is moderately to highly heritable. This also fits with the observation that dyslexia tends to run in families. Family-risk studies employ this fact. Such studies follow the development of at-risk children: children with a family history of dyslexia. After a few years of reading instruction, the at-risk children who have developed dyslexia can be identified. Subsequently, these children can be compared in retrospect to at-risk children without dyslexia and to typically developing children without family risk. Did the children with later dyslexia already demonstrate cognitive deficits before they started to learn to read? In this way family-risk studies enable the investigation of precursors of dyslexia.

Several models have been formulated in which dyslexia is attributed to a single cognitive deficit, but none of these models can readily explain the range of deficits associated with dyslexia. Pennington has developed a model in which developmental disorders (like dyslexia) are caused by a combination of underlying cognitive factors, which can be affected to different degrees. His model –the multiple deficit model– is depicted in Figure 1.1 in Chapter 1. Some of these cognitive deficits are specific and some are shared with other developmental disorders. The current thesis studied the specificity of precursors of reading ability by investigating whether they also predict arithmetic ability.

Inspired by the results of aetiological studies, the multiple deficit model includes genetic and environmental factors that influence the probability of developing a disorder; in this case, dyslexia. From this multifactorial aetiology it follows that dyslexia is not an all-or-none condition, but instead that the liability or risk of developing dyslexia is continuously distributed. In the present thesis it is argued that a family-risk study offers the opportunity to test this implication. Given that parents pass on their genes to their offspring and create their childhood environment, it is expected that reading skills of parents can be seen as indicators for children's liability to dyslexia.
In short, the aims of the current thesis are threefold:

1. To study children's cognitive precursors of dyslexia (and their specificity) before reading instruction begins;
2. To test whether the cognitive profile of parents is indicative of a child's liability or predisposition to dyslexia;
3. To contribute to testing, specifying, and extending the multiple deficit model.

As mentioned above, we employed a family-risk design to pursue these aims. The reported studies are part of the Dutch Dyslexia Programme. Children were considered at high risk if they had at least one parent and another family member with dyslexia. The parents of the low-risk children were both average to good readers. Two independent samples were followed, referred to as the Amsterdam (N = 79) and the national (N = 212) sample. In both samples the analyses consisted of, among others, comparing the following three groups: 1) at-risk children with dyslexia, 2) at-risk children without dyslexia, and 3) control children (i.e., low-risk children without dyslexia). Roughly one-third of the at-risk children developed dyslexia.

In the Amsterdam sample (Chapter 2) children were followed from kindergarten through Grade 5. The at-risk children who went on to have dyslexia had poor letter knowledge in kindergarten. In addition, they were impaired on a cognitive skill called rapid naming. Rapid naming is the ability to rapidly retrieve the name of a symbol from long-term memory, measured by naming a list of familiar items, like colours or pictures. The at-risk children without dyslexia performed better on letter knowledge and rapid naming, but still slightly below the level of controls. The same pattern was found for subsequent reading development. A key finding related to the reading difficulties of the parents with dyslexia: those of the affected children were more severely impaired than those of the unaffected children.

Chapters 3 to 5 are devoted to the national sample, reporting the development of 4 to 9 years of age. IQ was assessed at the age of 4 and preliteracy skills at the end of kindergarten. The investigated preliteracy skills included letter knowledge, rapid naming, and phonological awareness. Phonological awareness is the ability to detect and manipulate sounds in spoken words. In kindergarten this can be measured with items like “What are the sounds in ‘cat’?” Answer: “/k/ /a/ /t/”. Compared to controls, at-risk children
with later dyslexia were impaired across IQ and preliteracy tasks. The other at-risk children showed mild deficits in verbal IQ and phonological awareness, but not in nonverbal IQ, letter knowledge, and rapid naming. Relations between these precursors and later reading and arithmetic revealed that nonverbal IQ and rapid naming were related to both outcome measures, whereas the other precursors were more specific for reading. At-risk dyslexia children continued to show deficits in phonological awareness and rapid naming in second and third grade, alongside impairments in literacy and arithmetic. At this age, the at-risk no-dyslexia children had weaknesses in phonological awareness and literacy, although they scored within normal limits. Importantly, the finding in the Amsterdam sample regarding differences between the two at-risk groups in parental reading could be replicated and extended: also parental rapid naming differentiated the groups, as did literacy difficulties of the parent without dyslexia. In both the Amsterdam and the national sample there was no clear evidence of effects of home literacy environment on children’s reading outcome.

Chapter 6 gives an extensive summary of the findings and discusses theoretical implications. To return to the multiple deficit model, one of the predictions of this model is that the liability distribution for a given developmental disorder is continuous. Both the findings at the child-level and parent-level support this prediction. It is argued that cognitive skills of parents are an indicator of their offspring’s position on the liability continuum. Chapter 6 works towards specifying the multiple deficit model for the case of dyslexia and concludes by presenting an extended model: the intergenerational multiple deficit model (see Figure 6.1), which includes the effect of parental characteristics. Finally, recommendations for future research are given.