2024年度

大学院文学研究科博士課程後期3年の課程入学試験

(冬期・一般選抜) 問題

筆記試験 ______言語学 専攻分野

試験開始の合図があるまで、この問題冊子を開いてはいけない。

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筆記試験(

言語学 専攻分野)

問題 I. 以下の文章を読み、問 (i) ~ (iii) に答えなさい。

Individual Differences in Reading Development

To consider the nature and developmental course of dyslexia, it is important to highlight the resource demands of learning to read. Universally, reading is a process of mapping between the visual symbols on the page (orthography) and the spoken language. However, the nature of the symbols and the units of spoken language to which they connect differ across languages (e.g., Ziegler & Goswami, 2005). In an alphabetic language such as English, the foundation of literacy is a system of mappings between letters and phonemes (the smallest speech sounds of words) and a challenge for the child is to abstract the mapping principle (Byrne, 1998). In contrast, Chinese is nonalphabetic; the orthographic symbols are characters comprising semantic and phonetic radicals that correspond to morphemes (units of meaning). The semantic radical provides information about the meaning and the phonetic radical provides a cue to the pronunciation of the word (Shu & Anderson, 1997).

Orthographies also differ in their "transparency"—that is the regularity of the mappings between symbols and sounds. Among European languages, English has the most inconsistent writing system, embodying many exceptions; German and Dutch have relatively few inconsistencies and Finnish is the most consistent. In general, it has been shown that regular languages pose fewer challenges for the beginning learner than irregular languages (Seymour, 2005) but arguably, this is a simplistic view. Languages also differ in the way in which they convey the grammar in writing and differences in morphological structure may moderate the ease of learning (Seidenberg, 2011).

Nonetheless, it is clear that the specific demands of learning to read will differ across languages. Irrespective of this, the child needs to learn the symbol set and how that set maps to the language and this requires explicit awareness of the structure of the language. Once the basic mappings have been established, reading practice ("print exposure") is required to achieve reading fluency.

Among alphabetic languages it is well established that the predictors of individual differences in decoding (the skill that is impaired in dyslexia) are the same regardless of the transparency of the language: these are, letter knowledge, phoneme awareness, and rapid automatized naming (Caravolas et al., 2012; Ziegler et al., 2010a). However, reading development proceeds faster in the more transparent orthographies than in opaque orthographies (Caravolas et al., 2013). The process of learning is more protracted in languages that have large symbol sets and in nonalphabetic languages where mappings are to meaning rather than sound (Nag, Caravolas, & Snowling, 2011) (i). Nonetheless, a similar set of skills appear to predict progress in these languages—namely symbol knowledge, metalinguistic awareness, and rapid automatized naming; the corollary is that deficits in these skills compromise decoding in dyslexia.

Perceptual and Cognitive Deficits in Dyslexia

A major thrust of research on dyslexia has been to specify the underlying deficits that are candidate causes of the condition. Such impairments (that may be cognitive and/or have their origins in basic perceptual processes) mediate the impact of heritable, brain-based differences on behavior (Morton & Frith, 1995; Pennington, 2002; Ramus, 2003, 2004).

Within this approach, the predominant view for many years was that dyslexia could be traced to deficits within the phonological system of language (Melby-Lervåg, Lyster, & Hulme, 2012; Vellutino, Fletcher, Snowling, & Scanlon, 2004). As we have seen, phonological skills are critical foundations for learning to read in alphabetic systems. More generally, phonological deficits have been reported to characterize dyslexia in logographic Chinese (Hanley, 2005; Ho, Chan, Lee, Tsang, & Luan, 2004; Ho, Chan, Tsang, & Lee, 2002) and poor readers of alphasyllabic scripts (Nag & Snowling, 2011). However, a problem in assessing the causal status of phonological deficits is that performance on phonological tasks (such as phoneme awareness and nonword repetition) is influenced by reading skill (Morais & Kolinsky, 2005 for a review)(ii). It follows that deficits in these processes could be correlates (rather than causes) of poor reading. An advantage of family risk studies is that phonological processing can be measured before the onset of literacy. As we shall see, all family risk studies have assessed the development of phonological skills.

At a more fine-grained level, research has pursued the causes of the phonological deficits in dyslexia. In now classic work, Tallal (1980) proposed that dyslexia is caused by a problem with the rapid temporal processing of auditory information needed for the perception of speech sounds, leading to a cascade of difficulty from auditory processing through speech perception to phonological skills. Family risk studies are well placed to test such causal chains from the early stages of language development. Accordingly many of these studies draw on research suggesting deficits in speech perception in dyslexia (e.g., Adlard & Hazan, 1998; Nittrouer, 1999; Serniclaes, Van Heghe, Mousty, Carré, & Sprenger-Charolles, 2004; Ziegler, Pech-Georgel, George, & Lorenzi, 2009) or in basic auditory processing (Hämäläinen, Salminen, & Leppänen, 2012 for a review).

A separate line of investigation has focused on possible causes of difficulties with the letter-by-letter structure of words (orthographic deficits) in dyslexia. One theory is that spatial coding deficits affect ocular motor control (Boden & Giaschi, 2007; Kevan & Pammer, 2008; Vidyasagar & Pammer, 2010). Alternatively, problems in the system of visual attention could affect the left-to-right extraction of orthographic information critical for parsing letter strings before decoding (Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000; Valdois, Bosse, & Tainturier, 2004). In addition, there are modality—general theories that aim not to explain particular features of dyslexia but rather seek overarching explanations (e.g., Ahissar, Lubin, Putter-Katz, & Bani, 2006; Nicolson & Fawcett, 1990; Vicari, Marotta, Menghini, Molinari, & Petrosini, 2003). Whereas such theories may hold promise for understanding how dyslexia relates to other co-occurring disorders (comorbidity, e.g., Rochelle & Talcott, 2006) they do not explain why dyslexia can and sometimes does occur in the absence of any other cognitive deficits.

Nevertheless, as a long history of the search for subtypes of dyslexia attests, these causal hypotheses are not mutually exclusive and it is important to recognize that dyslexia is a heterogeneous condition (e.g., Ramus et al., 2003). As Pennington (2006) has argued, the etiology of complex disorders like dyslexia is multifactorial and involves the interactions of risk and protective factors. Longitudinal studies of children at family risk of dyslexia that follow children from early childhood to formal schooling can reveal the risk factors associated with a dyslexia outcome. In addition, because dyslexia is a dimensional disorder, the study of unaffected relatives can be informative in highlighting protective or compensatory factors that mitigate familial risks. Such risks (that could be biological processes or cognitive impairments) can be described as "endophenotypes" (Skuse, 2001).

According to Bearden and Freimer (2006), an endophenotype is a "marker" that is associated with the disorder in the population and expressed at a higher rate in unaffected relatives of probands than in the general population. Put another way, it is intermediate between the genotype and the phenotype and, importantly, the impact of such processes can be moderated or compensated for by areas of skill or through interventions. This particular characteristic of an endophenotype deserves mention in relation to comorbidities. When two neurodevelopmental disorders frequently cooccur it is probable that they have endophenotypes in common (Thapar & Rutter, 2015, for a

問題 I と II の回答は次ページ以降にまとめて記すこと。

review). Prospective family risk studies can identify putative endophenotypes of dyslexia (or subclinical features) that mark the presence of co-occurring disorders. For example, findings of family risk studies can elucidate relationships between difficulties with oral language observed in preschool and later written language disorders, in short, the comorbidity between dyslexia, specific language impairment (Bishop & Snowling, 2004; Pennington & Bishop, 2009, for reviews).

(Snowling, M. J., & Melby-Lervåg, M. 2016. Oral language deficits in familial dyslexia: A meta-analysis and review. *Psychological Bulletin*, 142, 498-545. を一部改編して抜粋)

- 問(i) 下線(i) はどういうことか。自分が知っている言語の例を挙げながら具体的に説明しなさい。
- 問 (ii) 下線 (ii) で指摘されている問題の背景には、どのような事情があるか。本文の記述に即して具体的に説明しなさい。
- 問 (iii) これから dislexia に関してどのような研究知見が求められるか。この文章の内容を踏まえて有用な実験研究案を示しなさい。

問題 II. 自分の関心ある言語事象について概説し、それを研究する意義、目的、方法、予測される結果を具体的に述べなさい。 その研究の特徴を、一般言語学および関連領域の文脈の中に位置づけながら説明しなさい。

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