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# Auditory processing deficits are sometimes necessary and sometimes sufficient for language difficulties in children: Evidence from mild to moderate sensorineural hearing loss

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ABSTRACT

There is a general consensus that many children and adults with dyslexia and/or specific language impairment display deficits in auditory processing. However, *how* these deficits are related to developmental disorders of language is uncertain, and at least four categories of model have been proposed: single distal cause models, risk factor models, association models, and consequence models. This study used children with mild to moderate sensorineural hearing loss (MMHL) to investigate the link between auditory processing deficits and language disorders. We examined the auditory processing and language skills of 46, 8–16 year-old children with MMHL and 44 age-matched typically developing controls. Auditory processing abilities were assessed using child-friendly psychophysical techniques in order to obtain discrimination thresholds. Stimuli incorporated three different timescales (μs, ms, s) and three different levels of complexity (simple nonspeech tones, complex nonspeech sounds, speech sounds), and tasks required discrimination of frequency or amplitude cues. Language abilities were assessed using a battery of standardised assessments of phonological processing, reading, vocabulary, and grammar. We found evidence that three different auditory processing abilities showed different relationships with language: Deficits in a general auditory processing component were necessary but not sufficient for language difficulties, and were consistent with a risk factor model; Deficits in slow-rate amplitude modulation (envelope) detection were sufficient but not necessary for language difficulties, and were consistent with either a single distal cause or a consequence model; And deficits in the discrimination of a single speech contrast (/bɑ/ vs /dɑ/) were neither necessary nor sufficient for language difficulties, and were consistent with an association model. Our findings suggest that different auditory processing deficits may constitute distinct and independent routes to the development of language difficulties in children.



## 1. Introduction

Specific Language Impairment (SLI)[1] and developmental dyslexia (hereafter *dyslexia*) are developmental disorders of language and communication that are estimated to affect 7–10% of the population

*Abbreviations:* AMD, amplitude modulation detection; AP, auditory processing; cAMD, complex amplitude modulation detection; cFMD, complex frequency modulation detection; DLD, Developmental Language Disorder; F2, F2 formant frequency modulation detection; FD, frequency discrimination; FMD, frequency modulation detection; MMHL, mild to moderate sensorineural hearing loss; RT, rise time; SLI, Specific Language Impairment; SNHL, sensorineural hearing loss; SP, speech processing.

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[1] Note that the term *Developmental Language Disorder* (DLD) has recently been proposed by Bishop, Snowling, Thompson, Greenhalgh, and the CATALISE-2 consortium (2016) as a replacement term for SLI, in an attempt to build some consensus in the field on issues of terminology and identification. Whilst we advocate this approach, we nonetheless continue to use the term *SLI* here to reflect (a) the terminology that has been used to date in the theories we are describing, and (b) the fact that many of the studies cited herewith involved children who showed a discrepancy between their verbal and nonverbal ability (a mismatch that is not a requirement for the diagnosis of DLD).

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(Snowling, 2000; Tomblin, Smith, & Zhang, 1997). They are diagnosed when a child experiences extreme delays and/or deviancies in acquiring oral language (SLI), and written language (dyslexia), despite having normal nonverbal ability, an absence of physical, sensory, neurological, or emotional deficits, and adequate opportunity to learn (World Health Organisation, 2010). There is now considerable evidence for an overlap between SLI and dyslexia, both in terms of co-occurrence (McArthur, Hogben, Edwards, Heath, & Mengler, 2000) and, potentially, shared aetiology (Catts, Adlof, Hogan, & Weismer, 2005; Ramus, Marshall, Rosen, & van der Lely, 2013). However, despite many decades of research, scientists still do not agree on what causes these disorders.

There are now several influential theories which attribute both SLI and dyslexia to difficulties in *auditory processing*, which are expressed as deficits in the discrimination and processing of non-speech sounds (e.g. Goswami, 2011; Tallal, 2004). These theories differ in terms of the sorts of auditory processing deficits that are proposed to underlie SLI and dyslexia. However, they are united in the premise that one or both disorders are caused by a single distal causal factor (unrelated to linguistic function) which leads, via impaired speech perception, to deficits in phonological processing. In turn, these deficits in phonological processing are proposed to be causally linked to difficulties in the acquisition of oral and/or written language and so to the development of SLI and/or dyslexia (see Fig. 1A).

A considerable body of evidence shows that many children and adults with SLI and/or dyslexia do exhibit deficits in auditory processing (for reviews, see Bishop, 2007; Hämäläinen, Salminen, & Leppanen, 2013). However, auditory processing theories nonetheless remain controversial, and have been the subject of much debate in the literature (for reviews, see Protopapas, 2014; Rosen, 2003). One area of controversy concerns the sorts of auditory processing deficits that are thought to underlie SLI and/or dyslexia. Here, there are at least two factors that lack consensus. First, researchers do not agree on the precise timescale on which the proposed deficits operate. According to the *rate-processing constraint hypothesis* (e.g. Tallal, 2004), children with SLI and a subset of children with dyslexia have a deficit in processing sounds over tens of milliseconds. This is argued to prevent the fine-grained analysis that is required to represent acoustic differences at the level of the phoneme (e.g. rapid transitions that distinguish English voiced stop consonants such as ɪbaɪʊ ɪdaɪʊ and ɪgaɪʘ. Evidence for deficits in auditory processing over this timescale in individuals with SLI and/or dyslexia has typically been garnered using stimuli that are brief (<75 ms) or rapidly presented (with inter-stimulus intervals < 200 ms), or that are modulated in frequency or amplitude over intermediate rates (~10–40 Hz) (SLI: Tallal & Piercy, 1973a, 1973b, 1974, 1975; Dyslexia: for a review, see Hämäläinen et al., 2013). In contrast, the more recent *temporal sampling framework* hypothesis (Goswami, 2011) proposed that both dyslexia and SLI are caused by a deficit in the detection of slower rates of modulation (i.e. hundreds of milliseconds to seconds), which is argued to primarily affect the perception of speech rhythm and stress. Tasks that have highlighted deficits over this timescale have typically involved the detection of differences in the abruptness of the rise in amplitude at the onset of a sound (so called *rise-times*), and in the detection of slow (~2–4 Hz) rates of amplitude modulation, as well as beat detection and production

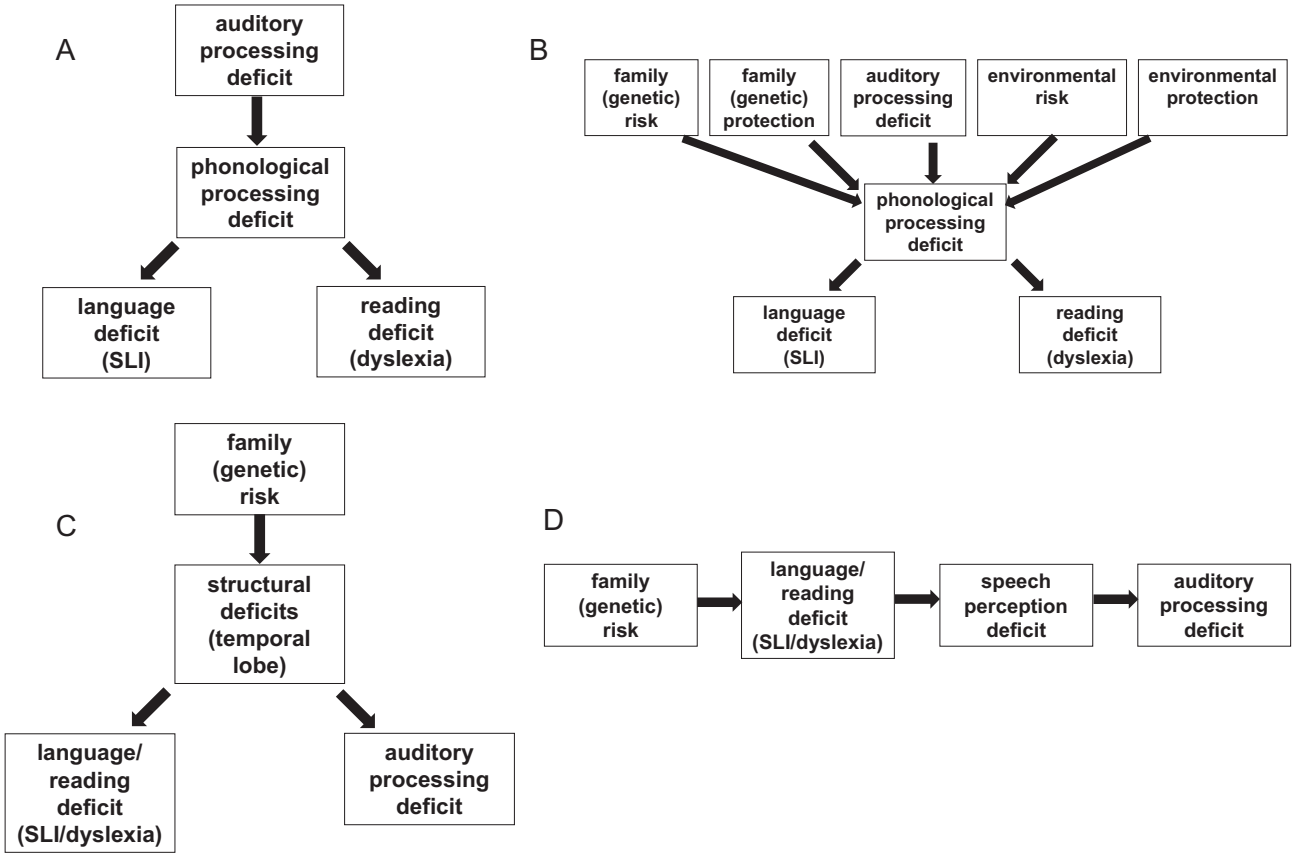


**Fig. 1.** Hypothesised relations between auditory processing and language abilities. (A) Single distal cause models (e.g. Goswami, 2011; Tallal, 2004) propose that deficits in auditory processing are the primary causal factor in the genesis of developmental disorders of language (SLI and dyslexia). (B) Risk factor models (e.g. Bishop, 2003a, 2003b; Pennington, 2006) propose that deficits in auditory processing may be one of a number of risk factors leading to the development of SLI and/or dyslexia. (C) Association models (e.g. Protopapas, 2014) propose that deficits in auditory processing are associated with but not causal to SLI and/or dyslexia. (D) Consequence models (e.g. Bishop et al., 2012) propose that deficits in auditory processing are a consequence of SLI and/or dyslexia.