Cognition 166 (2017) 139–151

Contents lists available at ScienceDirect

Cognition

journal homepage: www.elsevier.com/locate/COGNIT

Original articles

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

Lorna F. Halliday *, Outi Tuomainen, Stuart Rosen

*Speech, Hearing, and Phonetic Sciences, Division of Psychology and Language Sciences, University College London, Chandler House, 2 Wakefield Street, London WC1N 1PF, United Kingdom*

ARTICLE INFO

*Article history:*
Received 5 February 2015
Revised 26 April 2017
Accepted 30 April 2017
Available online 31 May 2017

*Keywords:*
Specific Language Impairment (SLI)
Dyslexia
Developmental Language Disorder (DLD)
Auditory processing
Mild to moderate hearing loss
Sensorineural hearing loss

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.

* Corresponding author.
*E-mail address:* l.halliday@ucl.ac.uk (L.F. Halliday).

[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).

http://dx.doi.org/10.1016/j.cognition.2017.04.014
0010-0277/

(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 /bɑ/, /dɑ/, and /gɑ/). 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

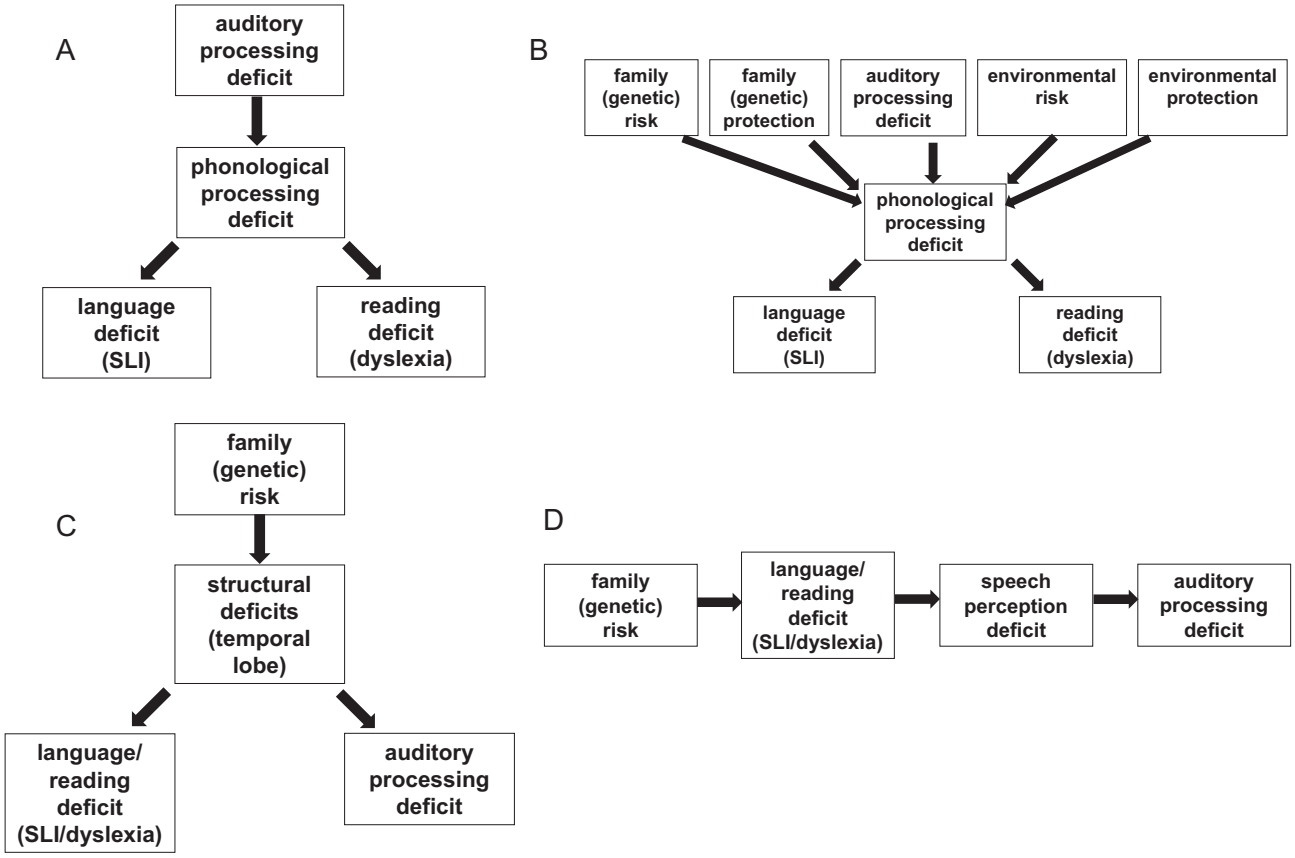


**Fig. 1.** Hypothesised relations between auditory processing and language abilities. (A) Single distal cause models (e.g. Goswami, 2011; Tallal, 2004) propose that deficits in auditory processing are the primary causal factor in the genesis of developmental disorders of language (SLI and dyslexia). (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.

(SLI: Corriveau & Goswami, 2009; Corriveau, Pasquini, & Goswami, 2007; Fraser, Goswami, & Conti-Ramsden, 2010; Dyslexia: for a review, see Hämäläinen et al., 2013). Recently, it has been argued that whilst a deficit in the temporal sampling of slow-rate modulations may be a primary impairment in dyslexia, children with SLI may instead have difficulties with faster rate information (Goswami et al., 2016). Finally, there is also an increasing body of evidence to suggest that many children and adults with SLI and/or dyslexia show deficits in discriminating differences in frequency in pure tones, even when the stimuli are steady-state (i.e. not modulated) and where stimuli are long (>75 ms) as well as short in duration (SLI: e.g. McArthur & Bishop, 2005; Dyslexia: for a review, see Hämäläinen et al., 2013). These deficits cannot be easily explained by either the rate-processing constraint hypothesis (Tallal, 2004) or the temporal sampling framework (Goswami, 2011), and have been attributed to deficits in phase-locking at the level of the brainstem or lower (*Magnocellular theory*; McAnally & Stein, 1996; see also Hornickel & Kraus, 2013; c. f. Amitay, Ahissar, & Nelken, 2002; Halliday & Bishop, 2006a; Hill, Bailey, Griffiths, & Snowling, 1999).

Aside from the question of the time scale over which the deficit operates, researchers also do not agree on whether the auditory deficits observed in SLI and dyslexia are general to all sounds or whether they are specific to speech. For instance, there are now several studies showing that some children with dyslexia (and indeed some with SLI) have difficulties processing synthetic speech sounds (e.g. consonant-vowels or vowels) but nonetheless show either no deficit or a less consistent deficit in processing nonspeech analogues of these contrasts which incorporate similar critical distinguishing acoustic features (Dyslexia: Mody, StuddertKennedy, & Brady, 1997; Rosen & Manganari, 2001; Serniclaes, Sprenger-Charolles, Carre, & Demonet, 2001; SLI: van der Lely, Rosen, & Adlard, 2004). This, along with other evidence, has led some researchers to argue that children with SLI and/or dyslexia have a specific deficit in speech-sound processing, that cannot be explained by the low-level deficits outlined above (SLI: Vandewalle, Boets, Ghesquiere, & Zink, 2012; Ziegler, Pech-Georgel, George, & Lorenzi, 2011; Dyslexia: Boets, Ghesquiere, van Wieringen, & Wouters, 2007; Mody et al., 1997; Schulte Korne, Deimel, Bartling, & Remschmidt, 1998; Ziegler, Pech-Georgel, George, & Lorenzi, 2009). Others have argued that a speech-specific deficit in SLI/dyslexia is more likely to arise under conditions of internal or external noise (Robertson, Joanisse, Desroches, & Ng, 2009; Ziegler et al., 2009) or due to the attentional demands of the task (Hazan, Messaoud-Galusi, Rosen, Nouwens, & Shakespeare, 2009; Messaoud-Galusi, Hazan, & Rosen, 2011). Alternatively, since the speech sounds that have been used in some studies have been acoustically more complex than their nonspeech analogues, it is possible that apparent deficits may arise due to an effect of acoustic complexity rather than as a result of impaired linguistic/phonological processing (Rosen & Manganari, 2001). However, contrary to this are findings that adults with dyslexia are impaired at categorizing both speech and nonspeech sounds that vary in terms of rapidly changing acoustic cues (i.e. temporal processing) when those sounds are matched for acoustic complexity (Vandermosten et al., 2010, 2011).

A further, but arguably key, area of controversy is whether or not these deficits actually *cause* the language difficulties that are characteristic of these disorders. Most published studies have focused on children or adults with pre-existing diagnoses of SLI and/or dyslexia, and so finding evidence for poor auditory processing abilities in these groups says little about the causal nature of the relationship. Nonetheless, in addition to single distal cause models, there are now at least three competing theories that might explain the observed association between auditory processing deficits and SLI and dyslexia. The first, termed the risk factor model (e.g. Bishop, 2006; Pennington, 2006), argues that no single deficit is either necessary or sufficient to lead to SLI or dyslexia, but that a number of genetic and/or environmental risk factors interact with a number of (genetic and/or environmental) protective factors to determine whether or not a given individual will go on to develop one or both of these disorders (see Fig. 1B). According to these models, auditory processing deficits may constitute one of a number of risk factors. Two more recent models argue that the auditory processing deficits seen in individuals with SLI and/or dyslexia could actually be down-stream consequence of these disorders, either resulting from the language deficits themselves (e.g. Bishop, Hardiman, & Barry, 2012, see Fig. 1D) or from differences in neural connectivity in these individuals that affects both language and auditory processing, but without a causal link between the two (Protopapas, 2014, see Fig. 1C).

Determining which of these models is correct is not a straightforward task. However, one way to test the theoretical relationship between deficits in auditory processing and developmental disorders of language is to examine the relationship between language development and deficits in auditory processing in other populations (i.e. aside from SLI or dyslexia). The current study aimed to do so by examining the co-occurrence of auditory processing and language deficits in children with mild to moderate sensorineural hearing loss (MMHL). By definition, these children do not have SLI or dyslexia, because current diagnostic criteria specifically rule out the diagnosis of hearing loss (World Health Organisation, 2010). Nevertheless, both children and adults with sensorineural hearing loss (SNHL) have been shown to have deficits in a wide range of basic auditory processing skills, albeit for potentially different reasons than children with SLI or dyslexia (for review, see Moore, 2007). These include, among other things, deficits in frequency discrimination, frequency and amplitude modulation detection, and speech discrimination and categorization (Moore, 2007), all of which have been implicated in SLI and/or dyslexia. This population therefore provides a natural experiment in which an over-representation of different auditory processing deficits allows the relationship with language outcomes to be examined. Specifically, if deficits on a given auditory processing construct are sufficient to cause oral and/or written language impairments, then language difficulties should be seen in all individuals with those auditory deficits, regardless of the origin of those deficits.

### 1.1. Predictions

Based on previous research, our assumptions were that, as a group, children with MMHL would show deficits on a range of different auditory processing abilities (Halliday & Bishop, 2005, 2006b; Rance, McKay, & Grayden, 2004) and these would be similar in presentation to those of children with dyslexia and/or SLI (Halliday & Bishop, 2005, 2006a, 2006b). For each of these auditory processing abilities, we aimed to examine their relationship with oral and written language. If single distal cause models (Fig. 1A) are correct, then (a) all children who have deficits in language should have deficits in a particular aspect of auditory processing (i.e. auditory processing deficits should be necessary to cause language impairments), and (b) all children who have deficits in that same aspect of auditory processing should have deficits in language (i.e. auditory processing deficits should be sufficient to cause language impairments). If risk factor models (Fig. 1B) are correct, then children who have deficits in auditory processing would have a greater likelihood of deficits in language than their peers, but not all children with deficits in auditory processing would show deficits in language. If the association model (Fig. 1C) is correct, then children with MMHL who show deficits in auditory processing would be no more likely than their peers to show deficits in lan-

guage. Finally, if the consequence model (Fig. 1D) is correct, then children who show deficits in language would be more likely than their peers to show deficits in auditory processing. We tested these predictions in two ways: First, by examining whether deficits in auditory processing were necessary or sufficient for language deficits in children with MMHL and controls; and, second, by examining whether children with MMHL who had poor auditory processing had poorer language abilities than those with normal auditory processing, and vice versa.

## 2. Methods

### 2.1. Participants

Two groups of children aged from 8 to 16 years were recruited: children with MMHL (MM group) and a chronological age-matched control group of typically developing children (CA group). All children were from monolingual English speaking backgrounds and all were required to achieve a minimum T-score of at least 40 (i.e. not more than 1 *SD* below the mean, and equivalent to an IQ-score of 85) on a test of nonverbal ability (see below). All children attended mainstream schools. Ethical approval was obtained from the UCL Research Ethics Committee, and informed written consent was obtained from the parent/guardian of each child.

#### 2.1.1. MM group

Fifty-seven children with a diagnosis of bilateral MMHL were recruited for the study, where SNHL was defined as a permanent hearing impairment which is caused by a deficit in the cochlea or auditory nerve (Moore, 2007). Participants were identified via Peripatetic Services in Local Educational Authorities across London and the South East of England. Children were required to communicate solely via the oral/aural modality (i.e. to not use sign language), and not have any other known additional needs. Children whose hearing loss was attributed to a known syndrome or neurological impairment including auditory neuropathy spectrum disorder were also excluded from the study. Those children who met these criteria were invited to UCL for screening. Hearing sensitivity at 250, 500, 1000, 2000, 4000 and 8000 Hz was measured using an Interacoustics AC33 audiometer. Mild hearing loss was defined as a better ear pure-tone average (PTA) threshold of 20–40 dB HL, and moderate hearing loss as a better ear PTA threshold of 41–70 dB HL, across 250–4000 Hz (British Society of Audiology, 2011). One child did not meet the criteria for MMHL, and was therefore excluded from the study. A further four did not achieve a nonverbal ability T-score of at least 40. Six children in this group dropped out of the study prior to completing all testing, and so their data were not included. This left a total sample size for this group of 46 (*M* age = 11.4 years, *SD* = 2.2; 27 boys, 19 girls; See Table 1). Nineteen of these children had a mild hearing loss, and 27 had a moderate loss. The age of confirmation of SNHL ranged from 2 months to 14 years (median = 57 months; *M* = 54 months; *SD* = 35.57), although all were believed to have had congenital SNHL. Forty-three had been prescribed a hearing aid in at least one ear, and none had any cochlear implants, as is normal for children with this level of SNHL. The age of hearing aid fitting ranged from 3 months to 15 years (median = 65 months; *M* = 63 months; *SD* = 39.60). Because the late age of confirmation of some of the children in our study raised the possibility that they had late-onset MMHL,[2] we ran all of the analyses reported here twice: First, including all children, and second, excluding those children whose MMHL was confirmed after 7 years of age (n = 6). The results did not change significantly after excluding those children with a late confirmation, and so the only results of the first analysis are reported here.

#### 2.1.2. CA group

Forty-four control children (*M* age = 11.5 years, *SD* = 2.1; 19 boys, 25 girls) with no known hearing loss, educational difficulties, or history of speech and language problems were recruited from primary and secondary schools located in the same geographical locations as those of the MM group (See Table 1). Children were individually matched in age to the MM group to within 6 months (±3 months). All children had PTA thresholds across octave frequencies 250–4000 Hz of less than 20 dB HL in both ears (British Society of Audiology, 2011), and obtained thresholds no higher than 25 dB HL at any particular frequency.

### 2.2. Procedure

Testing was carried out during two sessions, each lasting approximately 90 min, and separated by at least a week. Each child was tested individually by one of two experimenters at UCL. Children underwent a test battery of audiometric, psychophysical and psychometric assessments. The audiometry and auditory processing tasks were conducted in a double- walled sound attenuating booth, and the cognitive tests were completed in a quiet room. All of the children in the MM group who owned a hearing aid used amplification during the psychometric assessments. Psychophysical thresholds were measured in both aided and unaided conditions, counterbalanced between participants across the two sessions. Here we focus on the unaided thresholds.[3]

### 2.3. Auditory processing tests

Children completed a battery of seven auditory processing tests delivered via psychophysical procedures (Table 2 and Fig. 2). To test whether children with MMHL showed deficits in basic nonspeech processing, we included three tasks that involved the discrimination of simple nonspeech stimuli: frequency discrimination (FD), frequency modulation detection (FMD), and rise time (RT). Note that performance on all of these tasks has been shown to be impaired in children and/or adults with SLI and/or dyslexia (see Section 1). To test whether children with MMHL showed deficits in complex nonspeech processing, we included three tasks that involved the detection of modulations in the fundamental frequency ("complex FMD": cFMD), second formant ("F2 formant FMD": F2) and amplitude ("complex amplitude modulation detection": cAMD) of a complex nonspeech analogue. Finally, to test whether children with MMHL showed deficits in speech processing over and above deficits in acoustic complexity, we

[2] Note that late age of confirmation of SNHL does not necessarily imply late-onset. Many children with MMHL are diagnosed relatively late in childhood, either because they were born prior to the introduction of Universal Newborn Hearing Screening (UNHS), or because the current UNHS in England only detects levels of SNHL ≥ 40 dB (Wood, Sutton, & Davis, 2015).

[3] Unaided rather than aided thresholds were used in this study for two reasons. First, we believe them to be of greater ecological validity than aided ones. As already outlined, many children, including some with congenital SNHL, are diagnosed relatively late in childhood. Once they are diagnosed, there is often a delay before hearing aid fitting, and some children with the mildest levels of hearing loss do not even receive an aid. Even if and when hearing aids are provided, the type of device they use may change over time, and many children do not wear them as much as is recommended, if at all (Fitzpatrick, Durieux-Smith, & Whittingham, 2010; Walker et al., 2015). Together, these factors mean that many children with MMHL learn to process auditory information (including speech) without access to hearing aids. Second, modern hearing aids are designed to alter both the spectral and temporal characteristics of the auditory signal, in addition to amplifying the incoming sounds. This makes interpretation of aided thresholds difficult as they may underestimate or over-estimate the auditory processing abilities of their wearers, depending on the task. We would therefore anticipate that any relationships between auditory processing and language seen in children with MMHL would be strongest for unaided thresholds.

**Table 1**
Participant characteristics of the MM and CA groups.

| | MM (n = 46) | | CA (n = 44) | | Statistics | | | |
|---|---|---|---|---|---|---|---|---|
| | *M* | *SD* | *M* | *SD* | *t*(df) | *p* | Cohen's *d* | CI |
| Age (years) | 11.4 | 2.2 | 11.5 | 2.1 | $t(88) = 0.23$ | 0.821 | 0.05 | [−0.8, 1.0] |
| Nonverbal ability (T-Score) | 55.6 | 8.7 | 60.6 | 8.5 | $t(88) = 2.76$ | 0.007 | 0.58 | [1.4. 8.6] |
| Maternal education (age) | 19.3 | 2.6 | 20.5 | 2.9 | $t(83) = 1.88$ | 0.063 | 0.43 | [−0.1. 2.3] |
| PTA threshold better ear (dB) | 43.4 | 12.0 | 7.3 | 4.0 | $t(55.23) = -19.28$ | <0.001 | 4.06 | [−39.8, −32.3] |
| PTA threshold *M* (dB) | 46.0 | 11.9 | 8.8 | 4.1 | $t(56.30) = -19.89$ | <0.001 | 4.18 | [−40.9, −33.4] |

*Note.* Age = mean age of session 1 and 2; Nonverbal ability as assessed using the Block Design subtest of the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999; see Table 3); Maternal education = age (years) at which mother left full-time education; PTA = pure tone average. CI = confidence interval.

**Table 2**
Auditory processing test battery.

| Test | Standard | Target | Initial target value | Percept of target |
|---|---|---|---|---|
| Frequency discrimination (FD) | 1-kHz sinusoid | Higher frequency sinusoid | 1.5 kHz | Sounds higher in pitch |
| FM detection (FMD) | 1-kHz sinusoid | 1-kHz sinusoid frequency modulated at 40 Hz | 40-Hz deviation | Has a different and more complex timbre |
| Onset discrimination (Rise time; RT) | 1-kHz sinusoid with a 15 ms rise time and fixed 50 ms fall time | 1-kHz sinusoid with a longer rise time and fixed 50 ms fall time | 435 ms rise | Has a duller attack |
| Complex FMD (cFMD) | Complex harmonic sound | Complex harmonic sound modulated in f0 at 4 Hz around a centre frequency of 100 Hz | 16-Hz deviation | Has a 'wobble' in its pitch |
| F2 formant FMD (F2) | Complex harmonic sound | Complex harmonic sound modulated in F2 at 8 Hz | 200-Hz deviation | Has a wobble in its timbre |
| Complex amplitude modulation detection (cAMD) | Complex harmonic sound | Complex harmonic sound, amplitude modulated at 2 Hz | 80% AM depth | Has a varying loudness |
| Speech discrimination (speech) | Digitised /bɑ/ syllable spoken by a female speaker | More /dɑ/-like syllable morphed from the endpoint of the continuum using the programme STRAIGHT (Kawahara, Masuda-Katsuse, & de Cheveigne, 1999). | Digitised /bɑ/ syllable spoken by a female speaker | Sounds more /dɑ/-like |

*Note.* Stimuli were 500 ms in duration except for the speech sounds which were 175 ms. Stimuli were ramped on with a 15-ms linear ramp except for the target stimuli in the RT task, where the ramp varied. Stimuli along each continuum varied logarithmically, except for the speech task. Each continuum contained 100 stimuli except for FD which contained 28. For FMD and cFMD, the initial value given is the maximum deviation in frequency in one direction, so the excursion covers a frequency range twice that. All complex harmonic sounds were based on a steady-state synthetic neutral vowel (f0 = 100 Hz) with three formants (500, 1500 and 2500 Hz).

included a /bɑ/-/dɑ/ speech discrimination task ("Speech"). Only one speech processing task was included because we were primarily interested in assessing the role of (nonspeech) auditory processing in the development of language difficulties. Note that the test battery included discriminations that spanned three different timescales, from μs (FD, F2), to ms (FMD, cFMD) to seconds (RT, cAMD), and tasks that required the discrimination of spectral (FD, F2, FMD, cFMD) as well as temporal (RT, cAMD) information.

Tests were delivered via a child-friendly computer-game format with a touch-screen response, incorporating an adaptive, three-interval, three-alternative, forced-choice paradigm (odd-one-out), with a 500 ms inter-stimulus interval. Stimuli were presented free-field at a fixed sound pressure level of 70 dB SPL, via a single speaker that was positioned facing the child approximately one metre away from the child's head. Children received feedback regarding the accuracy of their responses and were given unlimited time to respond. A three-down one-up procedure was used to select, on each trial, the appropriate target sound from the continuum (tracking a performance level of 79.4% correct) (Levitt, 1971). This followed an initial one-down, one-up rule until the first reversal (Baker & Rosen, 2001). The step size decreased over the first three reversals and then remained constant. Tracks terminated after 50 trials, or after four reversals had been achieved at the final step size (whichever came first).

Each child typically completed two runs per task, with one run per session. However, children were asked to repeat a run if their threshold was at ceiling (CA group: 2 (0.3%); MM group: 7 (2.1%)) or if they had achieved an insufficient number of reversals (<4) at the final step size (see below; CA group: 7 (%); MM group: 3 (0.9%)). The threshold was the arithmetic mean of the last four reversals in response direction, expressed in stimulus number on the continuum. For the CA group, this was the mean of the two runs, where both runs were available. For the MM group, thresholds were from the unaided runs only. The order of tests was counter-balanced between children.

Each test was preceded by a series of five practice trials which contained discriminations that were deemed suprathreshold for adult listeners. Participants were required to obtain four out of five correct responses on the practice trials in order to proceed to the corresponding test, with three attempts at reaching this criterion. Very few practice trials were repeated (1.4% for the CA group, 6.5% for the MM group).

### 2.4. Psychometric assessments

The battery of psychometric assessments is shown in Table 3. Note that the battery included tests that have been shown to pose difficulties for children with dyslexia (word and nonword reading, nonword repetition) as well as SLI (nonword repetition, receptive and expressive vocabulary, receptive grammar, and recalling sentences). All tests were standardised based on UK norms, apart from nonword repetition (Korkman, Kirk, & Kemp, 1998), where UK norms were not available.

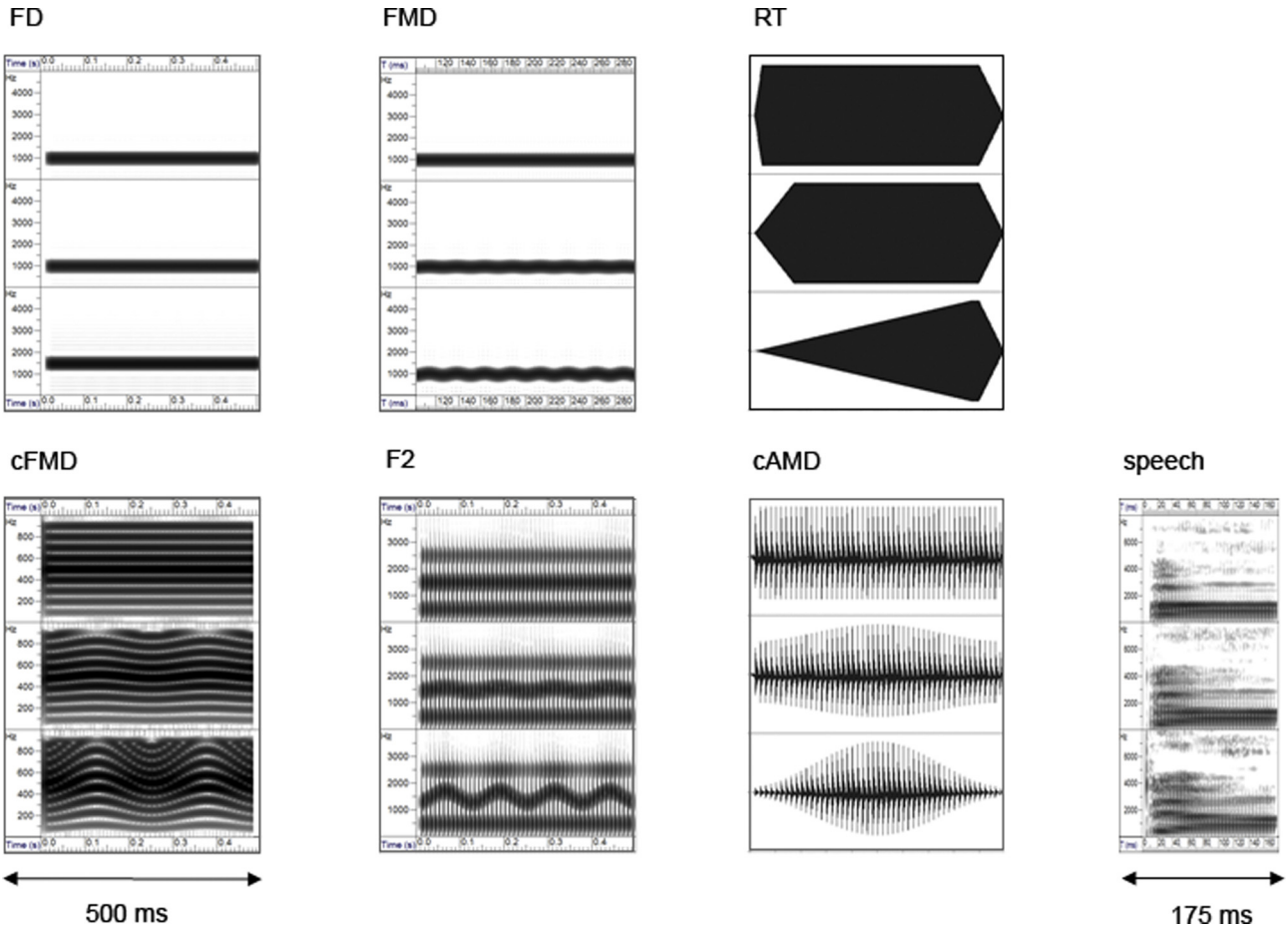


**Fig. 2.** Spectrograms and waveforms (RT, cAMD) depicting stimuli from each of the seven stimulus continua used in the auditory processing test battery. In each case, the top panel represents the standard stimulus, the bottom panel represents the comparison stimulus most different from the standard, and the middle panel represents an intermediate stimulus. Waveforms are shown for RT and cAMD because these two stimulus continua varied only in temporal envelope properties which are best seen in such a representation. The carrier for RT was a sinusoid so its spectrogram would look similar to the top panel of FD. The carrier for cAMD was a synthetic neutral vowel so the spectrogram for the standard stimulus was identical to the top panel of F2. Note the different frequency scales on the spectrograms for the different continua, chosen to display their properties most clearly.

### *2.5. Missing data*

Psychophysical thresholds were not obtained for one child in the MM group because she was unable to hear the stimuli clearly. Note that this child had the greatest level of hearing loss in the study (better ear PTA of 70 dB HL) and was not representative of the group as a whole. Psychophysical data were not obtained for one child in the CA group during the second session owing to time pressure. Thresholds for this child were calculated from the first session only. Psychophysical thresholds could not be obtained for nine tracks (two controls, seven MM) owing to ceiling effects even after repeated attempts. Three children in the MM group obtained fewer than four reversals in the final rule on one of their tracks, meaning that their data for those tests were excluded from the analysis.

### *2.6. Data processing*

In order to control for the effects of age in the auditory processing tests, scores for each child were converted to z-scores based on the standardised residuals from a linear regression of performance across age in the CA group only. These standardised residuals were then checked for normality. Data for all bar one of the auditory processing tests failed Kolmogorov-Smirnov tests of normality as a result of positive skew (the exception was the cAMD task which was normally distributed for both groups). Consequently, each of the non-normal data sets was log transformed (to base 10). This normalised the data in all but two of the data sets (cFMD for the CA group and FD for the MM group). Scores for each of the language assessments apart from nonword repetition were converted into z-scores with a mean of 0 and a *SD* of 1. For nonword repetition, z-scores were again calculated as they had been for the AP tests, because the norms for this test (a) are based on data from the USA, and (b) only go up to 12;11 (years; months). Kolmogorov-Smirnov tests showed that scores were non-normally distributed for the subtests of expressive vocabulary/word definitions (MM and CA groups), recalling sentences (MM group only), receptive grammar (MM and CA groups), and pseudoword decoding (CA group only). Given that (a) the majority of data sets met the assumptions of normality and (b) parametric statistics are relatively robust to violations of normality, the data for the language assessments were not transformed, and non-normal psychometric and psychophysical data were analysed using parametric statistics. Scores for each of the auditory processing and language tests were checked for extreme outliers (data points with values more than three times the difference between the 25th and the 75th percentile) and none were found. Consequently, all available data were included in the analyses.

In order to examine possible relationships between the auditory processing and language tasks, two factor analyses were conducted

**Table 3**
Psychometric assessment battery.

| Domain | Test | Subtest | Reference |
|---|---|---|---|
| Nonverbal ability | Wechsler Abbreviated Scale of Intelligence (WASI) | Block Design | Wechsler (1999) |
| Communication | Children's Communication Checklist-2nd Edition (CCC-2) | NA | Bishop (2003a) |
| Phonological input/output[a] | NEPSY | Repetition of Nonsense Words (*nonword repetition*) | Korkman et al. (1998) |
| Receptive vocabulary[a] | British Picture Vocabulary Scale – 3rd Edition (BPVS-3) | NA | Dunn, Dunn, Styles, and Sewell (2009) |
| Expressive vocabulary[a] | Clinical Evaluation of Language Fundamentals – 4th UK Edition (CELF-4 UK) | Expressive Vocabulary (8–9 year olds); Word Definitions (9+ years) | Semel, Wiig, and Secord (2006) |
| Receptive grammar[a] | Test for Reception of Grammar – 2nd Edition (TROG-2) | NA | Bishop (2003b) |
| Expressive grammar and memory[a] | CELF-4 UK | Recalling Sentences | Semel et al. (2006) |
| Word reading[a] | Wechsler Individual Achievement Test – 2nd UK Edition (WIAT-II UK) | Word Reading | Wechsler (2005) |
| Nonword reading[a] | WIAT-II UK | Pseudoword Decoding | Wechsler (2005) |

[a] *Note.* The seven language tests used to derive the Language component (see text).

to reduce the number of variables tested and to minimise the risk of multicollinearity between variables. The first factor analysis included the seven auditory processing tests. A principal component analysis (PCA) was conducted intended to be followed by Varimax rotation. The initial R-matrix containing all seven auditory processing tests identified two measures (cAMD and speech) for which fewer than 50% of correlations with the other tests were greater than 0.3 (see Supplementary Table 1). These tests were therefore excluded and the PCA re-run (Field, 2013). The final model included 82 participants who had data for all five variables. This analysis therefore had a participant-to-variable ratio of 16, comfortably meeting the usual criteria for factor analysis (Bryant & Yarnold, 1995; Hatcher, 1994). The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis, KMO = 0.76 (Hutcheson & Sofroniou, 1999). Extracted communalities were high (>0.45 for all of the variables) showing that the variables shared a substantial amount of variance. Only one factor had an eigenvalue > 1, and examination of the scree plot was consistent with the decision to retain a single factor. The unrotated component matrix is shown in Supplementary Table 2. The retained component was deemed to represent those capabilities general to auditory processing and was therefore named *auditory processing* (AP). The two tests removed from the PCA were retained and treated as independent tasks: *AMD* (cAMD task) and *speech processing* (*SP*; speech task). We decided to treat these tasks as independent (a) because of the strong theoretical link that has been made between deficits in amplitude modulation detection and reading ability in children with dyslexia (e.g. Goswami, 2011) and (b) because SNHL has been linked to spared amplitude modulation detection abilities in adults (Bacon & Gleitman, 1992; Bacon & Opie, 2002; Glasberg & Moore, 1989; Moore & Glasberg, 1986, 2001; Moore, Shailer, & Schooneveldt, 1992).

The second factor analysis included the seven language tests. The KMO statistic was 0.87. Extracted communalities were >0.5 for all of the variables. Only one factor had an eigenvalue of >1, and examination of the scree plot was consistent with this. The unrotated component matrix is shown in Supplementary Table 3. The retained component was named *Language*.

## 3. Results and discussion

### 3.1. Are deficits in auditory processing necessary or sufficient for language deficits?

In order to relate our data to the different models shown in Fig. 1, we first asked whether deficits in auditory processing were necessary or sufficient for language deficits in children with MMHL and/or controls. To this end, we examined the relationship between auditory processing (AP, AMD, and SP) and the Language component across groups. Fig. 3 displays these relationships. In order to identify those who performed poorly on the various tasks we followed Ramus et al. (2003), and defined poor performers as children who obtained an AP, AMD, SP, or Language component score that was more than 1.65 *SD* below the mean for the CA group, calculated after excluding those controls who fell below this criterion. This minimised the risk of the control mean and standard deviations being skewed by outliers, and identified those children whose scores were in the bottom 5% of those of the CA group (see Fig. 3). If auditory processing deficits were necessary for language impairments, then we would expect that all participants who showed poor scores on the Language component would perform poorly on AP, AMD, or SP. That is, that there should be no participants in the bottom left hand quadrants of Fig. 3 (poor language, normal/good auditory processing). On the other hand, if auditory processing deficits were sufficient for language problems, then we would expect that all participants who scored poorly on auditory processing would also score poorly on the Language component. That is, that there should be no participants in the upper right hand quadrants of Fig. 3 (poor auditory processing, normal/-good language).

As is evident from Fig. 3, the three different auditory processing abilities showed qualitatively different relationships with the Language component. For the AP component, six (16%) of the MM group and one (2%) of the CA group showed poor scores on the Language component but nonetheless normal/good AP thresholds. However, all but three (4%) of these cases were on the border of poor/normal performance. Conversely, for the same component, six (16%) of the MM group and five (11%) of the CA group showed the opposite relationship (i.e. poor AP but normal/good language). These results suggest that across the two groups, poor language scores were almost always associated with poor performance on the AP component (i.e. poor AP appeared necessary for poor language). However, poor performance on the AP component was not always associated with poor language in either group (i.e. poor AP was not sufficient for poor language).

For the AMD component, the opposite pattern was observed. In total, 22 (50%) of the MM group and one (2%) of the CA group showed poor scores on the Language component but nonetheless normal/good performance on the AMD task. In contrast, only two of each of the MM and CA groups (5%) showed elevated AMD thresholds and normal/good language, and these cases were typically borderline. This pattern of results suggests that poor performance on the AMD task was not necessarily linked to language

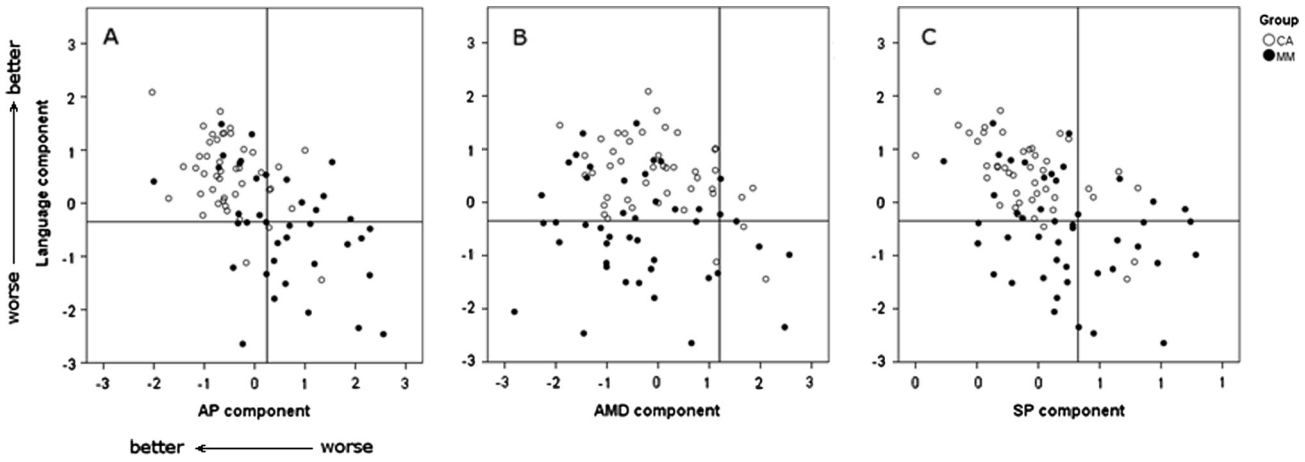


**Fig. 3.** Relationships between scores on the Language component and scores on (A) AP, (B) AMD, and (C) SP for the MM and CA groups. Cases below the horizontal line represent those participants whose scores on the Language component were in the bottom 5% of those of the CA group. Cases to the right of the vertical line represent those participants whose scores on AP, AMD, or SP were in the bottom 5% of those of the CA group.

difficulties, but that in the rare event that such deficits were observed, they were typically sufficient.

Finally, for SP, 16 (36%) of the MM group and one (2%) of the CA group showed poor scores on the Language component and normal/good scores on SP, whereas four in each of the two groups (9%) showed poor scores on SP and normal/good scores on the Language component. These findings suggest that deficits on the SP task were neither necessary nor sufficient for language problems in children with MMHL or in those with normal hearing.

### *3.2. Do children with poor auditory processing have poorer language than those with normal auditory processing?*

Our findings therefore suggest that none of the auditory processing abilities that we tested here were both necessary *and* sufficient for language difficulties in children with MMHL and controls. We then asked whether those children with MMHL who had poor auditory processing as a group showed poorer scores on the Language component (risk factor model). To answer this question, we first identified from Fig. 3 participants in the MM group who performed poorly on each of the three different auditory processing abilities. This led to the formation of six subgroups within the MM group: MM AP− (poor performers on the AP component; n = 21), MM AP+ (normal/good performers on the AP component; n = 17), MM AMD− (poor performers on AMD; n = 6), MM AMD+ (normal/good performers on AMD; n = 38), MM SP− (poor performers on SP; n = 15), and MM SP+ (normal/good performers on SP; n = 30).

To verify that the MM+ subgroups did indeed show normal/good auditory processing, we compared their performance to that of the MM- subgroups and the CA group on AP, AMD, and SP using univariate ANOVAs. For all three comparisons, there was a significant main effect of subgroup (AP: $F(2,79) = 57.84$, $p < 0.001$; $d = 10.41$; AMD: $F(2,85) = 19.77$, $p < 0.001$; $d = 1.08$; SP: $F(2,86) = 39.22$, $p < 0.001$; $d = 17.33$). Differences between subgroups were followed-up using post hoc Schéffe tests. These confirmed that, for AP and SP, the MM+ subgroups did not differ significantly from the CA group (AP: $p = 0.588$; SP: $p = 0.918$). For AMD, the MM AMD+ subgroup actually obtained significantly lower (better) thresholds than the CA group ($p = 0.005$). As expected, the MM- subgroups showed significantly higher (poorer) scores than both the MM+ subgroups and the CA group on all three auditory processing abilities (all $p$ values < 0.001). These findings therefore confirmed that the classification of the subgroups into normal/good (MM+) and poor (MM−) performers was valid.

The performance of the MM+, MM−, and CA subgroups on the Language component is shown in Fig. 4 (top row). Three univariate ANCOVAs with subgroup (CA versus MM+ versus MM−) as the between-groups factor and scores on the Language component as the dependent variable were conducted, with the $p$ value adjusted to 0.016 correct for multiple comparisons. Because nonverbal ability differed significantly between the MM and CA groups (see Table 1), we entered this as a covariate in the initial models. In addition, children in the MM AP− subgroup had significantly poorer PTA thresholds than children in the MM AP+ subgroup ($t(36) = -2.04$, $p = 0.049$, $d = 0.67$; although children in the MM AMD+/− and MM SP+/− subgroups did not differ in this way). Consequently, *M* PTA threshold was also entered as a covariate in the initial models. The final models contained only those main effects and covariates that contributed a significant amount to the variance. For all models, only the covariate nonverbal ability was significant. For all auditory processing abilities, there was a significant main effect of subgroup (AP: $F(2,78) = 12.98$, $p < 0.001$; $d = -5.26$; AMD: $F(2,84) = 11.46$, $p < 0.001$; $d = -6.51$; SP: $F(2,85) = 13.38$, $p < 0.001$; $d = -6.34$). Post-hoc analyses (Least Significant Difference (LSD)) showed that the MM AP− subgroup obtained significantly lower (poorer) scores on the Language component than both the MM AP+ subgroup ($p < 0.010$) and the CA group ($p < 0.001$). The MM AP+ subgroup did not differ significantly from the CA group on this measure ($p = 0.060$). For AMD, in contrast, the CA group obtained significantly higher (better) scores than both the MM AMD+ ($p < 0.001$) and the MM AMD− ($p = 0.012$) subgroups, with the latter two subgroups not differing significantly from each other ($p = 0.821$). Similarly, for SP, the CA group obtained a significantly higher score on the Language component than both the MM SP− subgroup and the MM SP+ subgroup (both $p$ values < 0.001), and the latter two subgroups did not differ significantly from each other ($p = 0.111$). Together, these analyses indicate that, for children with MMHL, those who showed poorer scores on the AP component also had poorer language than those with normal AP performance. However, scores on the AMD and SP tasks were of little or no use in predicting language outcomes.

### *3.3. Do children with poor language have poorer auditory processing than those with normal language?*

Finally, we asked whether those children with MMHL who had poor scores on the Language component as a group showed poorer auditory processing abilities (consequence model). To answer this question, we first divided the MM group into those who showed

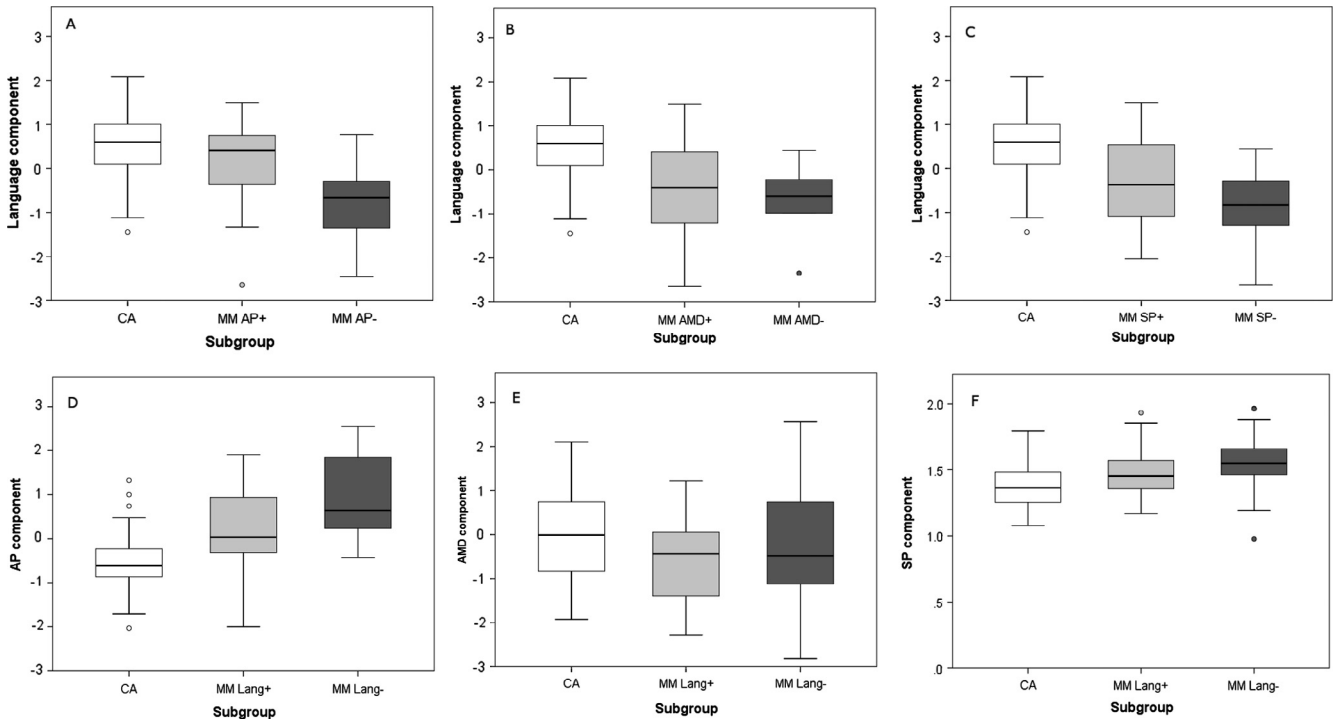


**Fig. 4.** Top row: Scores on the Language component for the CA group and the (A) MM AP+ and MM AP− subgroups, (B) MM AMD+ and MM AMD− subgroups, and (C) MM SP+ and MM SP− subgroups. Bottom row: Scores on (D) the AP component, (E) AMD, and (F) SP for the CA group and the MM Lang+ and MM Lang− subgroups.

poor performance on the Language component (MM Lang-; n = 28), versus those who showed normal/good performance (MM Lang+; n = 18) (see Fig. 3). A univariate ANCOVA (CA group versus MM Lang+ subgroup versus MM Lang− subgroup) with nonverbal ability as a covariate, confirmed a significant difference in Language component scores between groups ($F(2,86) = 43.57$, $p < 0.001$; $d = -8.56$). Post-hoc analyses (LSD) confirmed that, as expected, the MM Lang− subgroup obtained significantly lower (poorer) scores on the Language component than both the CA group and the MM Lang+ subgroup (both $p$ values < 0.001). The MM Lang+ subgroup did not differ significantly from the CA group ($p = 0.655$), confirming the normal performance of the MM Lang+ subgroup on this measure.

We then compared these subgroups to each other and to the CA group on the three auditory processing abilities (AP, AMD, and SP) (see Fig. 4, bottom row). A series of univariate ANCOVAs was conducted, with nonverbal ability entered as a covariate in the initial models, and alpha adjusted to $p < 0.016$ to control for multiple comparisons. As nonverbal ability was not a significant covariate in any of the models, the final models were univariate ANOVAs. For AP, there was a significant main effect of subgroup ($F(2,79) = 21.75$, $p < 0.001$; $d = 7.71$). Post-hoc Schéffe tests showed that the CA group obtained significantly lower (better) scores on the AP component than both the MM Lang+ and the MM Lang− subgroups ($p = 0.017$ and $p < 0.001$ respectively). In turn, the MM Lang+ subgroup obtained significantly lower AP scores than the MM Lang− subgroup ($p = 0.028$). There was also a significant main effect of subgroup for SP ($F(2,86) = 7.31$, $p = 0.001$; $d = 9.02$). Here, the CA group had significantly lower (better) SP thresholds than the MM Lang- subgroup ($p < 0.001$). However, the MM Lang+ subgroup did not differ from either the CA group ($p = 0.392$) or the MM Lang- subgroup ($p = 0.202$) on this measure. Finally, for AMD, there was no significant main effect of subgroup ($F(2,85) = 1.30$, $p = 0.279$; $d = -1.70$). These findings suggest that children with MMHL who had poor language skills showed poorer AP (but not AMD or SP) than those with normal language.

## 4. General discussion

### 4.1. Different relationships between different auditory deficits and language

The primary result of this study was that deficits in different auditory processing abilities showed different relationships with language abilities in children. Our PCA analysis indicated that the variance from five out of seven auditory processing tasks (frequency discrimination (FD), frequency modulation detection (FMD), rise time (RT), complex FMD (cFMD) and F2 formant FMD (F2)) loaded on a single, general, component, which we termed auditory processing (AP). We found that 12% of our sample showed normal language skills despite showing poor performance on this component. These findings appear to rule out single distal cause models for AP (Fig. 1A; e.g. Goswami, 2011; Tallal, 2004), which would predict a one-to-one relationship between auditory processing deficits and poor language outcomes. However, the fact that children with MMHL who had poor AP also had poorer language skills than those with normal AP suggests that there is nonetheless a relationship between AP and language. This seems to rule out association models (Fig. 1C; e.g. Protopapas, 2014) which would predict no relationship between the two variables in children with MMHL. Finally, the fact that children with MMHL who had normal language also showed poorer AP performance than controls argues against the notion that the AP deficits observed here were a consequence of the language difficulties observed in this group (Fig. 1D; Bishop et al., 2012). Our data for the AP component therefore align most closely to risk factor models, which predict that deficits in auditory processing should lead to an increased likelihood of language difficulties in the absence of a one-to-one relationship between the two (Fig. 1B; e.g. Bishop, 2006; Pennington, 2006; see also Plakas, van Zuijen, van Leeuwen, Thomson, & van der Leij, 2013).

In contrast, our complex amplitude modulation detection (AMD) and speech discrimination (SP) tasks were generally only

weakly correlated with the AP tasks and, consequently, their relations with language were considered separately. Here, a different relationship was observed between performance on the cAMD task and language. Our finding that deficits in AMD were almost always linked to poorer language appears to rule out both risk factor and association models. However, children with MMHL who had poor AMD skills did not as a group have poorer language abilities than those with normal/good AMD. Moreover, children with MMHL who had poor language skills did not show poorer AMD than those with normal language skills, thereby appearing to rule out consequence models. These findings suggest two alternative explanations. First, given that very few children with MMHL had poor AMD abilities (6 out of 46), it may be that deficits in AMD are sufficient for language difficulties, but we had insufficient power to detect this. Second, it may be that deficits in AP were so prevalent in children with MMHL that they blurred the potential link that would have ordinarily been observed between AMD and language. Both of these explanations are consistent with both the notion of single distal route and consequence models to explain the relationship between deficits in AMD and language. Either way, our findings suggest a more direct relationship between the detection and discrimination of the auditory envelope and language development in children.

Finally, performance on our speech task showed a more nuanced relationship with language skill. Poor performance on this task was not inevitably linked to poor language, and children with MMHL who showed impaired SP did not have poorer language skills than those with normal SP. These findings question the association between deficits in SP and poor language outcomes, thereby appearing to rule out both single distal cause and risk factor models. In addition, children with MMHL who showed deficits in language were no more likely than their peers to show deficits in SP. This finding therefore appears to rule out consequence models which would predict a consistent relationship between the two. We can therefore think of two, related, explanations for our findings. First, it may be that SP mediates language development via an association model. According to this model, children with MMHL who showed deficits in SP (for different reasons to those of children with SLI and/or dyslexia), would be no more likely than their peers to show deficits in language (which we observed). However, in a normally hearing population these deficits may be mediated by genetic risk factors and/or structural deficits in auditory cortex, affecting both SP and language outcomes (e.g. Protopapas, 2014). Second, it is possible that these abilities might be associated via a third variable, for example attention, which affects performance on both SP and language/literacy without a causal link between them (Halliday & Bishop, 2005; see also Papadopoulos, Georgiou, & Parrila, 2012).

### 4.2. Implications for theoretical models of SLI and/or dyslexia

At a broader level, our findings have implications for theoretical models that attempt to explain the observed association between deficits in auditory processing and developmental disorders of language and literacy. First, our findings indicate that there may be multiple routes that mediate the relationship between auditory processing deficits and language difficulties in children. As such, they rule out extreme versions of any single distal cause model – i.e. that there is one, single auditory processing deficit in childhood that leads to developmental oral or written language impairment. In this regard our conclusions are supported by empirical studies that have failed to attribute deficits in auditory processing in individuals with SLI and/or dyslexia to a single impairment or type of impairment (Amitay et al., 2002; Law, Vandermosten, Ghesquiere, & Wouters, 2014).

Second, our results suggest that not all auditory processing deficits are equal regarding their relationship with language. Deficits on a range of auditory processing tasks (including FD, FMD, and RT) may be associated with an increased risk of language impairments, but they do not appear to determine them. In contrast, deficits in the processing of slow-rate amplitude modulation may have a more detrimental link to language outcomes, although the direction of this relationship has yet to be determined. In this regard our findings lend at least some support to the temporal sampling framework hypothesis (Goswami, 2011), which proposed that deficits in the detection of slow rates of modulation (in the range of hundreds of milliseconds) lead to dyslexia and SLI. However, there are three caveats to this conclusion. First, the temporal sampling framework predicts that performance on both slow-rate AMD and RT tasks should go together. We did not find this, although performance on the cAMD and RT tasks was correlated across groups. Second, recent findings would predict that slow-rate AMD should be more associated with reading than language (Goswami et al., 2016). We did not find this either, although owing to the results of our PCA we did not specifically look. Finally, as a single distal cause model, the temporal sampling framework currently fails to incorporate the role of other auditory processing deficits in the aetiology of language impairments. This is surprising, given that even those studies that have been cited as evidence for this theory have reported poorer discrimination in individuals with dyslexia and/or SLI for frequency (Goswami, Fosker, Huss, Mead, & Szuecs, 2011; Goswami, Gerson, & Astruc, 2010; Goswami, Huss, Mead, Fosker, & Verney, 2013; Goswami et al., 2013; Leong, Hämäläinen, Soltesz, & Goswami, 2011; Wang, Huss, Hämäläinen, & Goswami, 2012), intensity (e.g. Fraser et al., 2010; c.f. Goswami, Huss et al., 2013; Goswami et al., 2010; Leong et al., 2011; Stefanics et al., 2011; Wang et al., 2012), and duration (e.g. Corriveau et al., 2007; Goswami, Huss et al., 2013; Goswami, Mead et al., 2013; Wang et al., 2012; c.f. Goswami et al., 2010) in addition to measures of RT discrimination, beat detection and/or AMD. The current study highlights the need to do so.

### 4.3. Clinical implications for children with MMHL

Our findings also have implications regarding the role of auditory processing in children with MMHL. We found evidence for deficits in auditory processing in 78% of the MM group. Poor performance was more frequently observed on tasks contributing to the AP component than on the speech or cAMD tasks, suggesting that envelope cues may be relatively well preserved in this group. At the same time, language deficits were more frequent in children with MMHL, with 61% of our sample performing in or below the bottom 5% of that of controls. Together, our findings suggest that deficits in AP may constitute a risk factor for the development of language problems in children with MMHL. Future studies are needed to ascertain this.

### 4.4. Potential limitations

Clearly, our findings cannot speak to the sorts of auditory processing deficits *actually* present in people with SLI and dyslexia, because we did not test participants with those disorders. Indeed, current diagnostic criteria for SLI and dyslexia specifically rule out the diagnosis of hearing loss (World Health Organisation, 2010). We therefore make no claims from this data about the sorts of auditory processing deficits (if any) that might be necessary to underlie language difficulties in children with SLI and/or dyslexia (c.f. White et al., 2006). Our findings do however illustrate the likely consequences (if any) on language development of having

an auditory processing deficit in childhood (i.e. whether deficits in auditory processing are sufficient for language impairments).

It is also clear that the reasons why many children with MMHL have deficits in auditory processing are likely to differ from why some children with SLI and/or dyslexia do. SNHL is largely peripheral in origin, and poor performance on auditory processing tests generally may be due to reductions in audibility as well as to damage to the cochlea and auditory nerve (Moore, 2007). In contrast, the auditory processing deficits proposed to underlie SLI and/or dyslexia have been attributed to higher centres of the auditory system (i.e. from the brainstem upwards; Giraud & Ramus, 2013; Goswami, 2011; Hornickel & Kraus, 2013; Tallal & Gaab, 2006). It is therefore possible that we would have seen a different pattern had we tested children with SLI and/or dyslexia (see Grube, Cooper, Kumar, Kelly, & Griffiths, 2014). Nevertheless, although the *origins* of MMHL are peripheral, we would expect knock-on effects on brain structure and function in these individuals, especially where the loss is congenital or acquired during childhood (Buran et al., 2014; see Cardon, Campbell, & Sharma, 2012, for a review). Moreover, although the causes of auditory processing deficits might be different in MMHL and dyslexia/SLI, the consequences should be the same (i.e. single distal cause models propose that it is the deficit in auditory discrimination that leads to language impairment, not the neural mechanism underlying the auditory deficit). We therefore believe that our extrapolation from children with MMHL to language impairments is a valid one.

A further potential limitation is that owing to the results of our PCA, we were unable to test the precise predictions of any specific auditory processing theory. For instance, the rate-processing constraint hypothesis (Tallal, 2004) predicts that language deficits should be associated with difficulties in the processing of sounds that are changing over tens of milliseconds (i.e. our F2 (8 Hz), FMD (40 Hz), and speech tasks, but not FD, RT, cFMD (4 Hz) or cAMD (2 Hz)). The temporal sampling framework hypothesis (Goswami, 2011) would predict our AMD and RT tasks to be most important; Magnocellular theory (Stein & Walsh, 1997) our tasks that relied on phase-locking (i.e. FD, FMD, and cFMD). Likewise, we were unable to directly compare the effect of acoustically complex (cFMD, F2, cAMD) versus noncomplex signals (FD, FMD, RT; e.g. Rosen & Manganari, 2001). However, we believe the largely data-driven approach we followed to be a strength of this paper.

It is unclear why our tasks grouped together the way they did. Three of the tasks that contributed to the AP component (FD, FMD, and cFMD) are likely to rely on the use of *temporal fine structure* (i.e. the rapid oscillations in an auditory signal whereby the rate is close to the centre frequency of the carrier; Rosen, 1992). Our RT and F2 tasks, in contrast, may have depended on sensitivity to amplitude modulation; RT is thought to measure detection of differences in the abruptness of the amplitude envelope onset (the *attack*), F2 sensitivity to amplitude modulation differences across frequency (*dynamic spectral structure*). Although each of these abilities has been linked to different aspects of speech perception (Rosen, 1992; Rosen & Iverson, 2007), there is no obvious perceptual reason why they would be expected to group together. The fact that they did here may suggest that performance on these tasks reflects some sort of general auditory processing ability or indeed some 'higher-level' cognitive ability (e.g. attention, working memory, nonverbal IQ). However, we are reassured that others have reported similar general groupings of auditory skill in other populations (Grube, Kumar, Cooper, Turton, & Griffiths, 2012).

Finally, as a correlational study, our results allow us only to speculate on causation. Therefore, whilst we found evidence for an *association* between deficits in various auditory processes and language, this does not indicate that one caused the other. There is a need for future studies to examine the predictive relationship between these auditory processes and language outcomes (and vice versa) over developmental time.

## 5. Conclusion

In summary, this study has shown that deficits in auditory processing appear to sometimes be necessary, and sometimes sufficient, for language problems in children, but they are not always necessary *and* sufficient. For children with MMHL, deficits in a general, AP component, appeared to be necessarily associated with poor language, but they were not sufficient. Deficits in the discrimination of slow-rate amplitude modulation were, in contrast, not necessary for language difficulties, but when they did occur they appeared to be sufficient. Finally, deficits in speech discrimination were neither necessary nor sufficient for poor language. We interpret these findings as evidence for the existence of multiple routes between auditory processing and language outcomes in children, each acting via independent mechanisms. Our findings therefore suggest that no single auditory deficit is likely to be solely responsible for the observed association with language deficits in children.

## Funding

This work was supported by an ESRC First Grants Award (RES-061-25-0440). The funding body had no involvement in the study design, in the collection, analysis and interpretation of data, in the writing of the report, or in the decision to submit the article for publication.

## Conflicts of interest

There are no conflicts of interest.

## Acknowledgements

The authors would like to thank Mike Coleman for developing the computer software, Steve Nevard for assistance with setting up the laboratory facilities, Kathleen McCarthy for recording some of the stimuli, and Páraic Scanlon for testing some of the participants. Thanks to Dorothy Bishop and Axelle Calcus for helpful comments on an earlier draft of this manuscript. The authors are especially grateful to all the children who participated, along with their parents, as well as the Local Educational Authorities and schools who assisted with recruitment.

## Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.cognition.2017.04.014.

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