

Auditory sensory memory and language abilities in former late talkers: A mismatch negativity study

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Abstract

The present study investigated whether (a) a reduced duration of auditory sensory memory is found in late talking children and (b) whether deficits of sensory memory are linked to persistent difficulties in language acquisition. Former late talkers and children without delayed language development were examined at the age of 4 years and 7 months using mismatch negativity (MMN) with interstimulus intervals (ISIs) of 500 ms and 2000 ms. Additionally, short-term memory, language skills, and nonverbal intelligence were assessed. MMN mean amplitude was reduced for the ISI of 2000 ms in former late talking children both with and without persistent language deficits. In summary, our findings suggest that late talkers are characterized by a reduced duration of auditory sensory memory. However, deficits in auditory sensory memory are not sufficient for persistent language difficulties and may be compensated for by some children.

Descriptors: Auditory sensory memory, Late talker, Mismatch negativity, MMN, Specific language impairment, SLI

Language delay in the absence of other medical conditions is found in approximately 10%–20% of 2-year-olds (Klee et al., 1998; Rescorla & Alley, 2001) who are referred to as “late talkers” (LTs; Horwitz et al., 2003; Rescorla, 1989). According to several studies (e.g., Miniscalco, Westerlund, & Lohmander, 2005; Rice, Taylor, & Zubrick, 2008), language delay is a risk factor for specific language impairment (SLI). For example, Dale, Price, Bishop, and Plomin (2003) examined 8,386 twins (LTs: $n = 802$; non-LTs: $n = 7,584$) and reported that 40.2% of LTs had language difficulties at the age of 4 years in contrast to 8.5% in normally developing children.

Children with SLI have a higher risk of developing socio-emotional problems. For example, they show lower achievements in school in a broad range of subjects including mathematics (Snowling, Adams, Bishop, & Stothard, 2001). Moreover, later in adulthood a twofold increase in the incidence of psychiatric disorders, such as dissocial behavior and anxiety disorders was found (Beitchman et al., 2001). Therefore, it seems important to investigate the underlying neurophysiological mechanisms contributing to the development of SLI in order to enable and enhance possibilities for early intervention.

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Deficiencies in auditory short-term memory are among the postulated causes of SLI. In a number of studies, auditory short-term memory deficits were reported in children with SLI (Montgomery, 2003) and dyslexia (Jeffries & Everatt, 2004; Smith-Spark & Fisk, 2007). These deficits are markers of SLI and are assumed to be predictive of language development in these children (Botting & Conti-Ramsden, 2001; Conti-Ramsden & Hesketh, 2003; Gathercole & Baddeley, 1990). In contrast, there is a lack of knowledge regarding the neurobiological basis of late talking.

The relationship between SLI and auditory short-term memory has been interpreted using Baddeley and Hitch's (1974) working memory model (e.g., Montgomery, 2003). This model proposes a multicomponent capacity-limited system that comprises a “phonological loop” for verbal information processing and a “visuospatial sketch pad” for processing visual information. The “central executive” coordinates and integrates both subsystems. Deficiencies in auditory short-term memory found in SLI are explained by reductions in both the storage capacity of the phonological loop and the encoding speed of language input. Such deficits are assumed to lead to difficulties establishing phonological representations, consequently impacting vocabulary acquisition and the establishment of grammatical rules (Baddeley, Gathercole, & Papagno, 1998). A limitation of Baddeley's model however, is that the initial steps of information processing are not well described.

In comparison, Cowan's (1988, 1995) model specifies the reception and storage of sensory information in greater detail. According to this model, incoming sensory information is consecutively integrated within a sensory store for the purpose

of resolving component features. This first part of the sensory store is designed to briefly (200–400 ms) hold large amounts of data. From this system the sensory information is transferred to a second, longer-lasting division of the sensory memory store, where it is kept available for further processing in working memory. Information is suggested to decay from this second part after a period of about 10–20 s. Auditory sensory memory is presumed to operate automatically and preattentively. It is hypothesized that a reduced duration of sensory memory could be the neurophysiological background of disturbed language acquisition in children with SLI (Barry et al., 2008).

Auditory short-term memory is commonly assessed with behavioral tasks in which subjects are typically asked to verbally repeat sequences of tones, syllables, words, or numbers of increasing length. Difficulties successfully completing these tasks have been attributed not only to working memory deficits but also to language difficulties (Barry et al., 2008). Because repetition accuracy depends on lexical and sublexical properties, the repetition of nonwords is a powerful tool to identify children with language impairments (Coady & Evans, 2008), but less appropriate for the evaluation of short-term memory capacities in subjects with language deficits. Moreover, repetition tasks demand immediate responses from subjects, and thus results are affected by attention and motivation. For these reasons, behavioral tasks are not ideal for young children and subjects with language difficulties.

An objective method for assessing auditory sensory memory is the event-related potential (ERP) known as mismatch negativity (MMN; Näätänen, 2003). MMN is generally obtained in an acoustic oddball paradigm, in which rare deviant sounds are presented within a stream of reoccurring standard sounds. The MMN operates at the sensory memory level and reflects an automatic preattentive process of comparisons between acoustic stimuli. Thus, the MMN is observed regardless of attention to the stimuli (e.g., Näätänen, Paavilainen, Tiitinen, Jiang, & Alho, 1993). It is assumed that regular aspects of consecutively presented standards form a memory trace in the sensory store and that violation of these regularities by deviants induces an MMN (Näätänen & Winkler, 1999).

MMN is used in basic and clinical research to determine auditory discrimination accuracy and the duration of sensory memory. Discrimination accuracy is generally investigated in oddball paradigms with constant and relatively short stimulus offset-to-onset intervals (interstimulus interval, ISI). In children with SLI, diminished MMN amplitudes have been repeatedly reported for speech-sound stimuli, but less consequently for tone stimuli. These results suggest that children with SLI have discrimination deficiencies specific to speech sounds (e.g., Bishop, 2007; Shafer, Morr, Datta, Kurtzberg, & Schwartz, 2005; Uwer, Albrecht, & von Suchodoletz, 2002).

To determine the duration of sensory memory, ISIs of different lengths are used. MMN is only found when the memory trace of the standard stimulus has not yet decayed from sensory memory. Therefore, sensory memory duration can be examined by varying the ISIs. It is thought that investigating the lifetime of the memory trace using MMN probes the second phase of sensory memory storage described by Cowan (Näätänen, Jacobsen, & Winkler, 2005).

Several studies have used MMN experiments with variable ISIs to probe the duration of auditory sensory memory in healthy children and adults. In newborns, a prominent MMN was found after a stimulus delay of 0.7 s, but not after 1.4 s (Cheour et al.,

2002). Glass, Sachse, and von Suchodoletz (2008a, 2008b) found memory traces between 1 and 2 s in 2- and 3-year-olds, greater than 2 s in 4-year-olds, and between 3 and 5 s in 6-year-olds. Gomes et al. (1999) investigated the duration of auditory sensory memory in school-age children and adults (age groups: 6–7, 8–10, 11–12, and 22–38 years) and obtained a robust MMN at an ISI of 1 s in all age groups. An MMN for the ISI of 8 s was found only in the groups with subjects older than 10 years. In healthy adults an MMN was detected up to an ISI of approximately 10 s (Böttcher-Gandor & Ullsperger, 1992; Sams, Hari, Rif, & Knuutila, 1993). In summary, the duration of the auditory sensory memory trace demonstrates a maturational development from approximately 0.7 s in newborns to at least 10 s in adults.

Only a few studies have addressed the question of whether there is evidence for a diminished duration of auditory sensory memory in clinical samples. The lifetime of a memory trace in the sensory store has been reported to be reduced in patients with chronic alcoholism (Grau, Polo, Yago, Gual, & Escera, 2001; Zhang, Cohen, Porjesz, & Begleiter, 2001) and Alzheimer's disease (Engeland, Mahoney, Mohr, Iivitsky, & Knott, 2002; Pekkonen, Jousmaki, Kononen, Reinikainen, & Partanen, 1994). The findings suggest that MMN can objectively identify sensory memory deficits in patients with memory impairments.

To our knowledge, in children, auditory sensory memory duration has only been investigated in CATCH syndrome (Cheour et al., 1997) and oral clefts (Ceponienė et al., 1999). Both studies reported shorter auditory sensory memory duration in comparison to healthy peers and attributed this deficit causally to the children's language impairments. Therefore, children with other language acquisition disturbances, such as SLI, might exhibit similar deficits.

To our knowledge, only one MMN study has investigated auditory sensory memory duration in SLI (Barry et al., 2008). In this study, parents of children with SLI were compared to parents with typically developing children using ISIs of 800 ms and 3000 ms. Reduced MMN was found for the 3000-ms ISI in parents of language-impaired children. This result was independent of the parents' language abilities. The authors therefore postulated a shortened lifetime for auditory sensory memory traces in parents of children with SLI, providing evidence for persistent and heritable auditory sensory memory deficits.

Taken together, the results of previous MMN studies show that the duration of auditory traces in the sensory memory store is limited, that this limitation is age dependent, and that the duration is reduced in patients with memory or language impairments as well as in parents of children with SLI. Moreover, a deficient auditory sensory memory seems to be persistent because of its assumed heritability.

To our knowledge, no study has examined the auditory sensory memory of children at risk for SLI and its meaning for the persistence of language disabilities. For this reason the present study addresses the question of whether former LTs show a sensory memory deficit in the auditory modality. If a deficient auditory sensory memory is linked to persistent difficulties in language acquisition, this deficit should be found in LTs with persisting language disabilities but not in LTs with resolved language problems, so-called late bloomers. Additionally, we analyzed neuropsychological memory scores and correlations between MMN and neuropsychological memory data in an exploratory manner between groups.

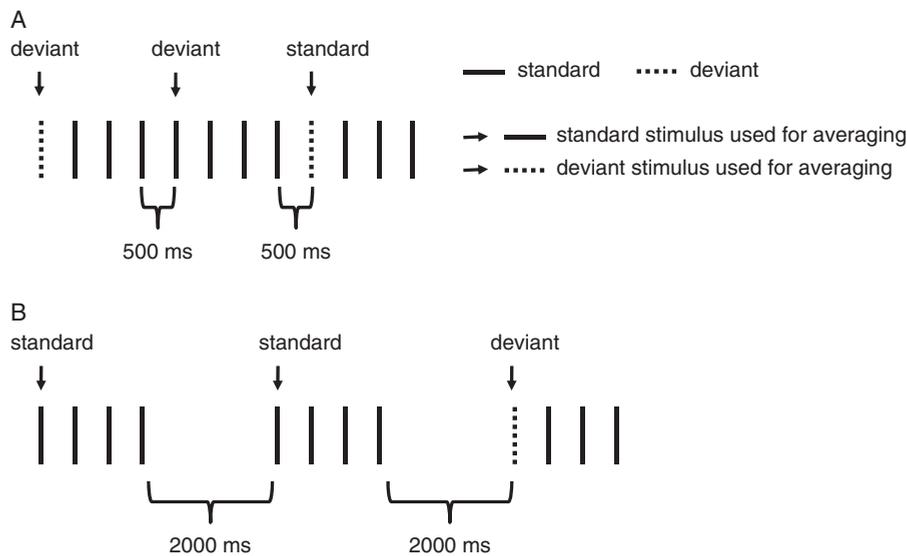


Figure 1. Oddball paradigm for probing the duration of auditory sensory memory with 500-ms (A) and 2000-ms (B) interstimulus interval condition.

In detail we used MMN to examine auditory sensory memory with two ISI durations (500 ms and 2000 ms) inserted between trains of four tones (Figure 1). We hypothesized a reduced MMN in LTs in comparison to control children for the longer ISI condition only. Additionally, if intact sensory memory is essential for normal language development, we should find no difference in mean MMN amplitude between late bloomers and control children.

Methods

Sample

Seventy-one German-speaking children participated in the study at the age of 4 years and 7 months ($M = 55.04 \pm 0.26$ months). All children took part in a longitudinal study beginning at 2;1 years of age, with follow-ups at 3;1 and 4;7 years.

To recruit children with and without language delay, we used birth announcements to contact parents of 2-year-old children (for details, see Sachse & von Suchodoletz, 2008). Children were classified as LTs at 2 years of age via a parent questionnaire (Elternfragebogen fuer die Frueherkennung von Risikokindern, ELFRA; Grimm & Doil, 2002), a German version of the McArthur Communicative Development Inventories (CDI, Toddler Form; Fenson, Dale, & Reznick, 1993), and a standardized language test (Sprachentwicklungstest fuer 2-jaehrige Kinder, SETK-2; Grimm, 2000) composed of two receptive and two productive language subtests. Children with poor results in ELFRA-2 (vocabulary < 50 words or vocabulary between 50 and 79 words and deficient morpho-syntactic abilities) as well as in SETK-2 (z -score [$M = 0$; $SD = 1$] ≤ -1.5 at least in one subtest) were classified as LTs ($n = 60$). Children with normal results in ELFRA-2 (vocabulary > 80 words and normal morpho-syntactic abilities) and SETK-2 (z -score > -1 in all subtests) were defined as control children ($n = 47$). Children with results between these two classifications were not included in the analysis, with the aim to construct two clearly defined groups.

Information about developmental milestones, medical history (complications during pregnancy or birth, prematurity, chronic

disorders, history of otitis media or other ear disorders), and socioeconomic characteristics were obtained by having the parents complete a questionnaire. There were no critical incidents reported for all of the participating children.

Forty-six LTs (77%) and 40 (85%) control children were reassessed at the age of 4 years and 7 months. Children with abnormal otoacoustic emission results due to common colds or other unspecific reasons at the time of measurement were excluded from analysis ($n = 8$). Other children were excluded because they refused to participate in the auditory screening ($n = 3$) or the ERP recording ($n = 3$) and because of emigration into a country with a foreign language ($n = 1$). This resulted in a total inclusion of 37 LTs (62%) and 34 (72%) control children.

All children had a normal nonverbal intelligence score (Snijders-Oomen nonverbal intelligence test: $IQ \geq 80$), normal hearing abilities (measured by otoacoustic emission screening or audiometry), and normal results on otoacoustic emission screening at least for one ear at the time of electroencephalogram (EEG) recording.

We additionally classified the LTs at the age of 4;7 years into late bloomers and non-late bloomers (z -score > -1 in all language tests vs. ≤ -1 in at least one language score including subtests sentence comprehension, sentence repetition, plural creation, and expressive vocabulary). Twenty-one of 37 (57%) LTs met the late bloomer criteria and 30 of 34 (8%) control children had language abilities within or beyond the normal range (z -score > -1) in all language tests ("language category"; see Table 1).

The characteristics of the sample are shown in Table 1. The groups (LTs vs. control children) differed in their frequencies for language category (normal vs. impaired: $\chi^2 = 8.68$, $p < .01$). Significant differences were also found for nonverbal intelligence ($T = 3.46$, $p < .01$) and language abilities (sentence comprehension: $T = 2.32$, $p < .05$; plural creation: $T = 3.83$, $p < .01$; sentence repetition: $T = 3.94$, $p < .01$; expressive vocabulary: $T = 3.93$, $p < .01$). No differences were observed for gender ($\chi^2 = 1.03$, $p > .1$) and handedness ($\chi^2 = 5.69$, $p > .05$) frequencies.

All parents gave their written informed consent for their children to participate in the study. The study was approved by the

Table 1. Characteristics of Late Talkers (LTs) Divided into Late Bloomers (LBs) versus Non-LBs and Control Children at the Age of 4;7 Years

	LTs (n = 37)	LB (n = 21)	Non-LB (n = 16)	Controls (n = 34)	LTs vs. Controls	
					χ^2	p
General characteristics (n)						
Boys/girls	25/12	15/6	10/6	19/15	1.03	.31
Handedness (right/left/ambidextrous)	27/2/8	16/2/3	11/0/5	30/3/1	6.09	.06
Language category ($SD > -1/SD \leq -1$)	21/16	21/0	0/16	30/4	8.68	.00
					<i>T</i>	<i>p</i>
IQ (<i>M</i> , <i>SD</i>)						
Non-verbal intelligence ^a	102.32 ± 11.16	104.76 ± 10.91	99.13 ± 11.0	110.53 ± 8.5	3.46	.00
Language (raw data: <i>M</i> , <i>SD</i>)						
Sentence comprehension ^b	10.05 ± 2.74	11.1 ± 1.92	8.69 ± 3.1	11.41 ± 2.12	2.32	.02
Plural creation ^b	21.68 ± 5.47	23.71 ± 2.95	19.00 ± 6.84	26.32 ± 4.70	3.83	.00
Sentence repetition ^b	76.22 ± 16.19	86.48 ± 9.36	62.75 ± 13.08	91.15 ± 15.73	3.94	.00
Expressive vocabulary ^c	16.03 ± 2.49	16.81 ± 1.86	15.00 ± 2.88	18.32 ± 2.43	3.93	.00

^aFour subtests of the Snijders-Oomen nonverbal intelligence test (Tellegen et al., 1998).

^bSETK 3-5 (Grimm, 2001).

^cK-ABC (Melchers & Preuss, 1991).

ethics commission of the medical faculty of the University of Munich (LMU Munich).

Stimuli and Procedure

Neuropsychological and EEG data were assessed on two consecutive days at the age of 4;7 years.

Neuropsychological assessment. Language abilities were assessed via standardized language tests comprising expressive vocabulary (Kaufman Assessment Battery for Children, K-ABC; Melchers & Preuss, 1991), grammar production, and comprehension (Sprachentwicklungstest fuer 3- bis 5-jaehrige Kinder, SETK 3-5; Grimm, 2001). Grammar production was quantified by sentence repetition and plural creation. The latter ability is more complex in German than in English because there is a larger range of plural forms in German. Grammar comprehension was assessed by means of sentence comprehension. Here, the children were required to carry out verbal instructions.

Short-term memory was measured using a nonword repetition task (NRT; subtest of SETK 3-5) and the subtest “word order” of the K-ABC. For the latter subtest, children listened to word sequences of increasing length; after each sequence children pointed to the corresponding pictures in the same order.

Handedness was evaluated using a preference inventory based on the Edinburgh Handedness Inventory (Oldfield, 1971). The children were asked to demonstrate how they would carry out everyday activities: to bring someone a book, to comb one’s hair, to hammer, to switch on the light, and to throw a ball. Nonverbal intelligence scores were calculated by four subtests of the Snijders-Oomen nonverbal intelligence test (Tellegen, Winkel, Wijnberg-Williams, & Laros, 1998) at the age of 3 years.

Neurophysiological assessment. Duration of auditory sensory memory was assessed using an oddball paradigm with varying ISI conditions. MMN was investigated using standard tones of 1000 Hz and deviant tones of 1200 Hz (duration 100 ms, rise and fall time 10 ms). The same frequencies had been used in previous studies exploring auditory sensory memory duration in children and adults (Barry et al., 2008; Glass et al., 2008a, 2008b; Gomes

et al., 1999). Because of difficulties associated with ERP recording in young children, we used a time-saving oddball paradigm described by Grau, Escera, Yago, and Polo (1998; Figure 1). Stimuli were grouped in trains of four tones with an interval of 500 ms between the tones within the trains. The trains began with either the standard or the deviant stimulus in a pseudorandomized order, and all nonleading stimuli were standards only, resulting in 1400 standards and 200 deviants (7:1) for each condition. The experiment was divided into four blocks of 400 stimuli each, with a 4 s break between blocks.

We expected that LTs would exhibit a MMN comparable to those of controls for shorter ISI conditions, and therefore we chose a control condition with an ISI of 500 ms. This assumption was based on previous findings showing that even newborns generate an MMN with ISIs of 700 ms (Cheour et al., 2002). For the experimental condition, we employed an ISI of 2000 ms. For normally developing 4-year-old children, it was shown that auditory information remains in sensory memory for longer than 2 s (Glass et al., 2008b). Accordingly, we considered an ISI of 2000 ms as the critical duration of sensory memory. Stimuli from the control (ISI: 500 ms) and the experimental condition (ISI: 2000 ms) were presented in separate blocks in a balanced order.

During the EEG recording, children were seated in an upright child’s seat and were shown a silent video. The child’s guardian remained in the testing room and silently completed questionnaires. Stimuli were presented through a loudspeaker placed in front of the child (distance: 2.3 m; sound pressure level: 74 dB). The total duration of the experiment was 42 min.

EEG Recording

The EEG was recorded using 20 Ag/AgCl sintered electrodes attached to an elastic electrode cap (Easy Cap, Herrsching, Germany). Electrodes were placed according to the International 10–20 System (Jasper, 1958). The horizontal electrooculogram (HEOG) was recorded from electrodes placed at the outer canthus of each eye. For vertical electrooculogram (VEOG) Fp2 and one electrode placed under the eye were used. One child refused the electrode placement underneath the eye, and therefore only

Fp2 was used for elimination of vertical eye artifacts for this child. The EEG electrodes were referenced to the right mastoid during the recording. Data acquisition was carried out using a BrainAmp system (Brain Products, Gilching, Germany). The online bandpass filter was set to 0.16 and 30 Hz (sampling rate: 250 Hz; impedances at the beginning of measurements: $< 5 \text{ k}\Omega$).

Data were analyzed off-line using Vision Analyzer. First, the scalp EEG was high-pass (0.8 Hz) and low-pass (20 Hz) filtered. Artifact correction was done in two steps. First, an independent component analyses (ICA) was conducted (Kalyakin, Gonzalez, Karkkainen, & Lyytinen, 2008) and eye movement and muscle artifacts were removed. Second, resting artifacts were rejected after re-referencing to linked mastoids by an amplitude criterion of $\pm 80 \text{ mV}$ for all central and frontal electrodes. Finally, the data were segmented (-100 to 600 ms) and averaged. Segmentation resulted in a mean number of 192 ± 6 epochs (range: 177–198) for the control children and a mean number of 194 ± 3 epochs (range 185–198) for the LTs. The mean number of epochs did not differ between groups (Mann-Whitney U test, $Z = -1.25$, $p > .2$).

Data Analysis

ERP. Event-related responses were averaged using the first tone of the trains in order to ensure that the number (200) and relative position of standards and deviants were comparable. MMN was obtained by subtracting standard from deviant-evoked responses for each ISI condition. The MMN was prevalent over frontal electrodes, and therefore F3, Fz, and F4 were used for further analyses. A frontal MMN maximum was also described in a previous study with 4–5.5-year-old children (Martin, Shafer, Morr, Kreuzer, & Kurtzberg 2003). Mean amplitudes of the MMN were calculated to quantify the MMN response. The time window for the mean amplitude was chosen based on running t tests (against zero) from the evoked responses of the combined group (LT and control group) for each ISI condition separately ($p < .05$ at ≥ 4 consecutive data points). The

Table 2. Time Window of Significant Differences between Standard and Deviant Responses in the Combined Group

ISI (ms)	n	F3	Fz	F4
500	71	124–272	120–260	128–260
2000	71	84–148	92–148	92–156

Note: ISI: interstimulus interval. Running t test: $p < .05$ at ≥ 4 consecutive data points.

resulting time window covered all intervals of significant differences in any of the three frontal electrodes (Table 2).

According to this procedure, the MMN time windows were 120 to 272 ms after stimulus onset for the 500-ms ISI control condition and 84 to 156 ms for the 2000-ms experimental condition (see gray areas of Figures 2 and 3).

Statistical analysis. Statistical analysis of the ERP and neuropsychological data was performed using analysis of variance (ANOVA). Main effects and interactions were calculated for the between-subject factors group (LTs vs. control children) and language category (normal vs. impaired) to control for differences in language abilities between groups. For the ERPs, mean amplitudes of F3, Fz, and F4 were averaged. Additionally, the within-subject factor ISI (500 ms vs. 2000 ms) was part of the ERP analysis. In the case of significant interactions, follow-up analyses were conducted. Finally, because nonverbal intelligence differed between groups (see Table 1), this score was subsequently implemented as a covariate (analysis of covariance, ANCOVA).

The NRT score was determined only for children without articulation difficulties (LTs = 23; control children = 28), because incorrect NRT responses may have arisen because of poor articulation rather than limited short-term memory capacity.

Pearson correlations were calculated between MMN (2000 ms ISI) and the short-term memory measures word order and NRT to examine the relationship between neuropsychological

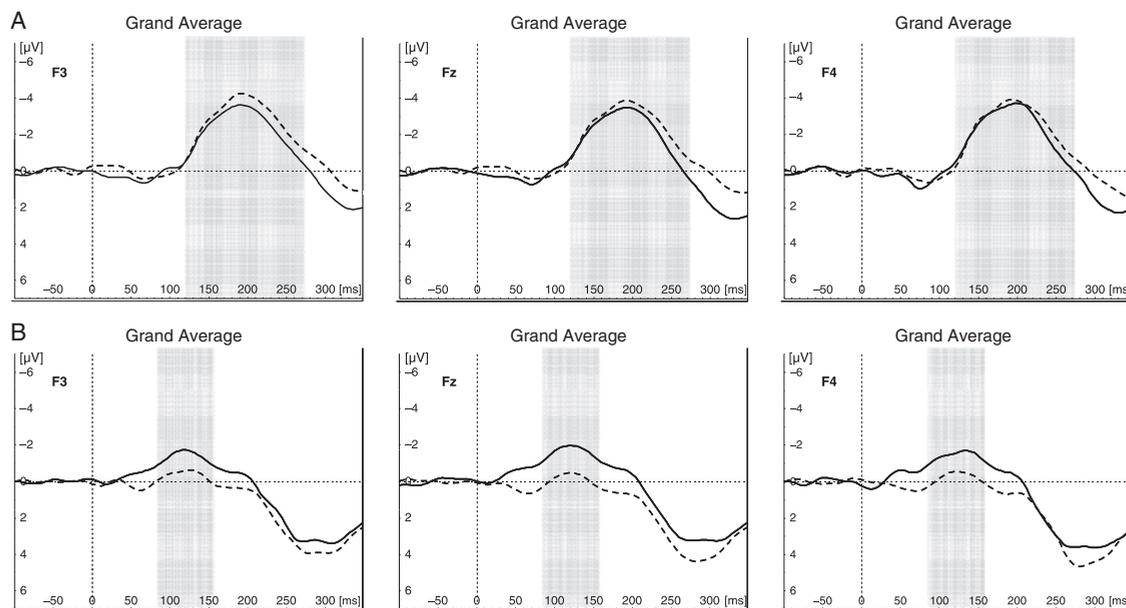


Figure 2. Mismatch negativity (MMN) as a function of interstimulus interval (ISI). MMN in the 500-ms control (A) and 2000-ms experimental (B) ISI condition for control children (solid lines) and late talkers (dashed lines). The gray area illustrates the interval of the mean amplitude.

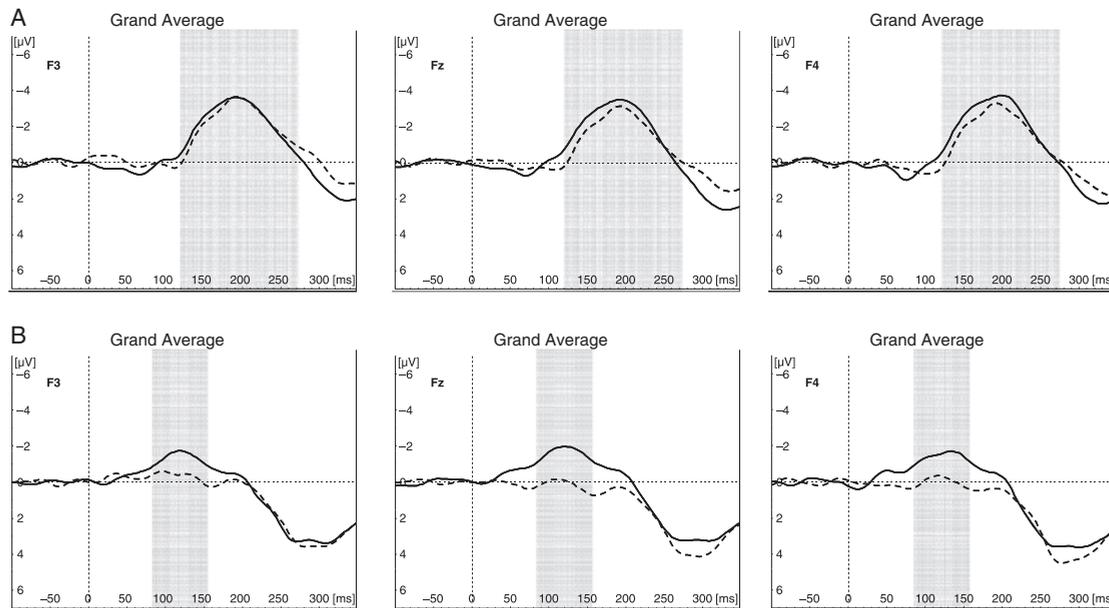


Figure 3. Mismatch negativity (MMN) as a function of interstimulus interval (ISI). MMN in the 500-ms control (A) and 2000-ms experimental (B) ISI condition for control children (solid lines) and late bloomers (dashed lines). The gray area illustrates the interval of the mean amplitude.

and neurophysiological memory parameters.

Because neuropsychological scores depend on various cognitive abilities, the neuropsychological data were analyzed with principal component analysis (PCA; orthogonal transformation varimax solution) in order to distinguish memory abilities. Therefore, each test score and the mean amplitude of the MMN in the experimental condition (ISI: 2000 ms) was *z*-transformed. Missing data were substituted by means. Only factors with an eigenvalue greater than 1 were extracted.

Significant effects are reported for $p < .05$.

Results

Descriptive data for MMN mean amplitudes and neuropsychological memory performance are listed in Table 3.

Behavioral Results

ANOVAs were performed for the neuropsychological memory scores word order and NRT with the between-subject factors group (LTs vs. control children) and language category (normal vs. impaired).

No main effects or interactions were found for word order.

The NRT analysis revealed a main effect for language category, $F(1,47) = 20.41$, $p < .01$, because children with average language abilities achieved better NRT scores. In addition, the interaction between language category and group was significant, $F(1,47) = 6.88$, $p = .01$. To explore this interaction further, *t* tests for independent samples were conducted separately for LTs and controls, resulting in a significant effect for the control group, $T(26) = 4.97$, $p < .01$, but not for the LTs, $T(21) = 1.44$, $p > .1$.

Including nonverbal intelligence as a covariate did not alter the significance of the results.

MMN Results

An ANOVA for mean MMN amplitude was performed with the between-subject factors group (LTs vs. control children) and language category (normal vs. impaired) and the within-subject factor ISI (500 ms vs. 2000 ms).

Main effects for the between-subject factors group and language category were not found, but the within-subject factor ISI was significant, $F(1,67) = 7.73$, $p < .01$, with higher amplitudes in

Table 3. Means and Standard Deviations for Mean Amplitude of Mismatch Negativity (MMN) and Neuropsychological Memory Tests for Late Talkers (LTs) Divided into Late Bloomers (LBs) versus Non-LBs and Control Children

	Group			
	LTs ($n = 37$)	LB ($n = 21$)	Non-LB ($n = 16$)	Controls ($n = 34$)
MMN				
ISI: 500 ms	-2.73 ± 3.25	-1.97 ± 3.25	$-.373 \pm 3.05$	-2.33 ± 2.53
ISI: 2000 ms	$-.27 \pm 1.56$	$-.09 \pm 1.6$	$-.52 \pm 1.52$	-1.49 ± 2.48
Neuropsychological tests				
Word order	6.27 ± 1.97	6.71 ± 1.87	5.69 ± 1.99	7.41 ± 2.34
NRT ^a	9.09 ± 3.01	9.73 ± 2.28	7.88 ± 3.94	12.25 ± 3.16

Note: ISI: inter-stimulus-interval; NRT: nonword repetition task.

^aLTs: $n = 23$ (LB: $n = 15$, non-LB: $n = 8$), control children: $n = 28$.

Table 4. Analysis of Mismatch Negativity (MMN) Mean Amplitudes for Interstimulus Interval (ISI, 500 ms, 2000 ms), Group (Late Talkers, Controls) and Language Category (Normal, Impaired), $N = 71$, $df = 1$

Source	Mean amplitude (μV)		
	<i>F</i>	<i>MSE</i>	<i>p</i>
ISI	7.73	37.05	.01
Group	0.02	0.13	.90
Language category	0.13	0.99	.72
ISI \times Group	6.06	29.05	.02
ISI \times Language category	0.06	0.27	.82
Language category \times Group	2.44	17.49	.12
Language category \times Group \times ISI	2.00	15.56	.16

the 500-ms ISI condition. A significant interaction was detected for ISI \times Group, $F(1,67) = 6.06$, $p < .05$, in accordance with our hypothesis (Table 4).

This interaction resulted from differences between control children and LTs in the 2000-ms ISI condition, $F(1,68) = 6.81$, $p < .05$, but not in the 500-ms ISI condition, $F(1,68) = 0.54$, $p > .4$ (Figure 2). This discrepancy described above was also significant for late bloomers compared to control children in the 2000-ms ISI condition, $F(1,52) = 4.56$, $p < .05$, but not in the 500-ms ISI condition, $F(1,52) = 0.02$, $p > 0.8$ (Figure 3).

Entering nonverbal IQ as a covariate did not alter the significance of the interaction between ISI and group, $F(1,66) = 6.85$, $p < .05$.

Correlations

To examine the relationship between neuropsychological and neurophysiological memory parameters, Pearson correlations were obtained. A significant correlation was observed between word order and MMN (2000-ms ISI condition; $r = -.24$, $p < .05$). Here, high test scores were associated with larger MMN amplitudes (signed negative). The correlation between MMN and NRT did not reach significance ($r = -.09$, $p > .5$).

PCA performed on the neuropsychological and neurophysiological scores yielded two factors with an eigenvalue > 1 . Each test measure was sorted into a two-dimensional vector space (Figure 4). Both identified factors accounted for 59.4% of the variance.

Late Bloomers versus Non-Late Bloomers

LTs who performed well in four language tests at the age of 4;7 years were classified as late bloomers ($n = 21$), whereas the remaining children showed persistent language deficits and were categorized as non-late bloomers ($n = 16$). Both groups differ in all language scores (t test, $p < .05$). No group differences were found for nonverbal intelligence and memory achievements (NRT and the K-ABC's word order subtest; t test, $p > .1$). The two groups did not differ in terms of handedness and sex (chi-squared test, $p > .2$).

Additionally, MMN mean amplitude differences for both ISI conditions were not observed (t test, 500 ms: $p > .1$; 2000 ms: $p > .4$). Finally, a logistic binary regression analysis showed that none of the variables measured at the age of 2 years, including sex, handedness, intelligence, and receptive and productive language abilities, could predict the outcome of late bloomers or non-late bloomers at the age of 4;7 years.

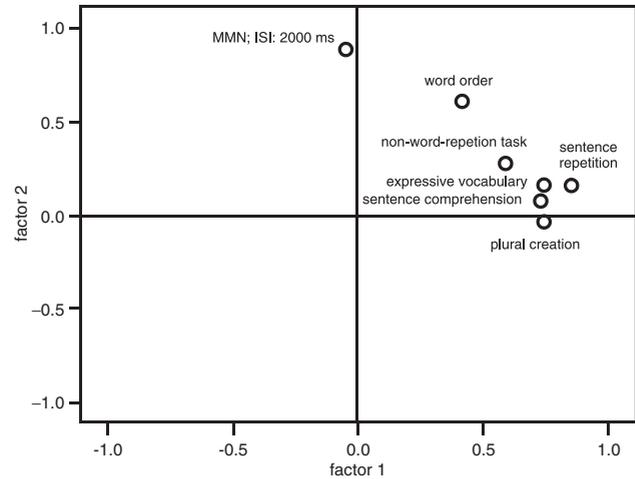


Figure 4. Neuropsychological measurements and mean amplitude of mismatch negativity (MMN) of the experimental condition (interstimulus interval of 2000 ms) illustrated by two main factors of a principal component analysis.

Discussion

The goals of this study were to investigate whether LTs show auditory short-term memory deficits, measured by MMN and neuropsychological tests, and to determine if such deficits are related to language impairment of LTs at preschool age.

Neurophysiological Findings

As hypothesized, MMN responses between LTs and control children did not differ in the 500-ms ISI control condition. These results imply that former LTs can normally discriminate attentively between the tones of 1000 and 1200 Hz, and information in the auditory sensory memory store was still available after 500 ms. In contrast, prominent MMN was observed in the 2000-ms ISI condition for the control children only, suggesting that the auditory sensory memory trace had decayed before 2000 ms in LTs. These findings cannot be explained by differences in intelligence, handedness, or sex and suggest that LTs are characterized by a persistent shortened duration of auditory sensory memory.

Unexpectedly, MMN (2000-ms ISI condition) was also reduced in late bloomers compared to the control group and not only in the children with persistent language deficits. No significant differences were observed between late bloomers and non-late bloomers in both MMN conditions and in all reported neuropsychological tests with the exception of language achievements.

Shortened sensory memory duration measured via MMN has been described for children with CATCH syndrome (Cheour et al., 1997) and oral clefts (Ceponienė et al., 1999). The authors assumed that a deficient auditory sensory memory could be causally associated with disturbed language and cognitive development in these children. Our results do not support this assumption, as MMN was reduced in the 2000-ms ISI condition even in late bloomers who had normal language abilities (Figure 3). Moreover, mean MMN amplitudes did not distinguish late bloomers from non-late bloomers.

Our outcome argues for a deficient auditory sensory memory in LTs that is not predictive for persistent language difficulties

and is in accordance with recent findings (Barry et al., 2008). Barry et al. examined parents with typically developing children and parents with children affected by SLI. They observed MMN attenuation for ISIs of 3000 ms but not for 800 ms in parents of children with SLI. An analysis of actual language impairment in the parents (self-report or direct test) revealed no effect for SLI.

On the one hand, our results suggest that the duration of auditory sensory memory is not causally associated with further language development in LTs. On the other hand, a higher frequency of children with SLI is consistently found in former LTs (Dale et al., 2003). Two explanatory issues for the relationship between LTs, auditory sensory memory, and SLI can be drawn. First, one could assume that a common factor exists behind memory achievements and SLI that is causally linked to both and that might also be moderated by other factors. For example, a genetic predisposition might be the common factor, which could lead to both reduced duration of auditory sensory memory and SLI. The second interpretation is that there is a causal relationship between auditory sensory memory and language acquisition. All LTs demonstrate deficient auditory sensory memory, but some children (late bloomers) can compensate for this deficit. However, the specific mechanisms that enable some LTs to compensate for sensory memory deficits are unclear.

Neuropsychological Findings

In contrast to sensory memory deficits found in the ERP data, no abnormalities were found in former LTs for neuropsychological short-term memory dimensions. The NRT yielded a significant group effect, which could be explained by different language abilities in both groups. All reported neuropsychological results were not affected by nonverbal intelligence scores. The observed relationship between NRT results and language abilities is in line with the observation that the NRT is sensitive for identifying children with language impairment (Coady & Evans, 2008). Moreover, it underlines the argument that the NRT is not suitable for measuring pure memory capacity because it depends heavily on language abilities (Barry et al., 2008).

Neuropsychological data are generally confounded with other cognitive processes besides language. The PCA yielded two factors, which can be interpreted as a language factor and an auditory short-term memory factor. This interpretation is underlined by the distribution of the test scores (Figure 4). The MMN mean amplitude accounts for one factor, which can arguably be interpreted as a factor representing auditory short-term memory. Language test scores form the language factor.

The word order test score lies between the two factors, whereas NRT clusters closer to the language factor. This is reflected by a moderate correlation between MMN mean amplitude (ISI: 2000 ms) and word order and no correlation between MMN mean amplitude and NRT. One possibility for the lack of correlation between NRT and MMN may be the diminished sample size. However, Barry and co-workers (2008) also found a lack of correlation between MMN and NRT.

A further reason why the neuropsychological memory data failed to confirm the ERP data could be because of the different underlying memory processes. It is assumed that MMN measurements with different ISI durations reflect how long auditory information is passively held in the sensory memory store, whereas neuropsychological data provide an indication of the capacity of working memory. It is therefore reasonable that the MMN findings and the results of the neuropsychological tests word order and NRT do not remarkably correlate.

Limitation

In our study we chose an ISI of 2000 ms as the experimental condition because normal developing children at the age of 4;7 years have an auditory sensory memory duration of at least 2000 ms (Glass et al., 2008b). However, it is conceivable that the duration of auditory short-term memory increases in late bloomers more than in non-late bloomers. Should differences in the duration of auditory sensory memory within the time window from 0.5 s to 2 s between these groups exist, we would not be able to detect them with this paradigm.

Conclusion

In summary, a deficient auditory sensory memory is related to late talking but is not sufficient for the development of SLI at 4;7 years of age. This is shown primarily from the results of the late bloomers, who exhibited a reduced MMN much like the LTs for the longer ISI condition, but did not show the subsequent language impairments. The reduced duration of auditory short-term memory is most likely not reflected in the neuropsychological memory performance due to task-specific language requirements or due to the taxing of possibly different memory components. Therefore, a reduced duration of auditory sensory memory is, similar to late talking, a risk but not a predictive factor for SLI. To date, more exploratory neuroscientific work is needed to detect the probable moderator variables that could explain why some LTs become late bloomers whereas others continue to have persistent language difficulties.

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