**Manuscript accepted for publication in Journal of Speech, Language, and Hearing Research**

# Noise equally degrades central auditory processing in 2- and 4-year-old children

**Niemitalo-Haapola Elina [1,2,*], Haapala Sini[2,3], Kujala Teija[4], Raappana Antti[5,6], Kujala Tiia[5,7], & Jansson-Verkasalo Eira[3]**

[1] Child Language Research Center, Faculty of Humanities, University of Oulu, Oulu, Finland

[2] Clinical neurophysiology, Oulu University Hospital, Oulu, Finland

[3] Department of Psychology and Speech-Language Pathology, University of Turku, Turku, Finland

[4] Cognitive Brain Research Unit, Institute of Behavioural Sciences, University of Helsinki, Helsinki, Finland

[5] PEDEGO Research Unit, University of Oulu, Oulu, Finland

[6]Department of Otorhinolaryngology - Head and Neck Surgery, Institute of Clinical Medicine, Oulu University Hospital, Oulu, Finland

[7] Medical Research Center Oulu, Oulu, Finland

* Corresponding author (e-mail: elina.niemitalo-haapola@oulu.fi)

## Abstract

**Purpose:** The aim of this study was to investigate developmental and noise-induced changes in central auditory processing indexed by event-related potentials in typically developing children.

**Method:** P1, N2, N4, and mismatch negativities (MMNs) were recorded for standard syllables and consonant, frequency, intensity, vowel, and vowel duration changes in silent and noisy conditions in the same fourteen children at the ages of 2 and 4 years.

**Results:** Developmentally, the P1 and N2 latencies decreased and the N2, N4, and MMN amplitudes increased. The amplitude changes were strongest at frontal electrodes. At both ages, background noise

decreased the P1 amplitude, increased the N2 amplitude, and shortened the N4 latency. The noise-induced amplitude changes of P1, N2, and N4 were strongest frontally. Furthermore, background noise degraded the MMN. At both ages MMN was significantly elicited only by the consonant change and at the age of 4 years additionally by the vowel duration change during noise.

**Conclusions:** Developmental changes indexing maturation of central auditory processing were found from every response studied. Noise degraded sound encoding and echoic memory as well as impaired auditory discrimination at both ages. The older children were as vulnerable to the impact of noise as the younger children.

## Introduction

Proper auditory input is essential for language acquisition (Bishop, 2014; Jansson-Verkasalo et al., 2010; Kuhl, 2010). However, young children are often exposed to high background noise levels (McAllister, Granqvist, Sjölander, & Sundberg, 2009) contaminating the auditory signal with harmful effects on language and cognitive development (for a review, see Evans, 2006). Measured noise levels in children's daily environments vary from 72 dB(A) (Shield & Dockrell, 2004) to 82.6 dB(A) $L_{eq}$ (McAllister et al., 2009) depending on protocols used for their respective investigations.

It is well established that background noise is detrimental in various ways to human wellbeing (Evans, 2006). First of all, long-term exposure to high-level noise or even transient high-energy noise can cause acoustic trauma to the inner ear resulting in hearing problems or tinnitus (Langguth, Kreuzer, Kleinjung, & De Ridder, 2013). In addition, even moderate noise levels, not deleterious to hearing, may influence health. Noise increases systolic blood-pressure and alters stress-hormone levels (for a review, see Hohmann et al., 2013), thus interfering with normal bodily functions.

In addition to these direct effects on hearing and physiology, noise also has effects on cognitive skills (Evans, 2006). It degrades memory (Hygge, Boman, & Enmarker, 2003; Söderlund, Sikström, Loftesnes, & Sonuga-Barke, 2010) and interferes with attention functions (Hygge et al., 2003). The effects of noise on language skills and speech production are also evident. Noise hampers vocabulary growth (Riley & McGregor, 2012), degrades speech perception (Gifford, Bacon, & Williams, 2007; Prodi, Visentin, & Feletti, 2013), and causes auditory memory problems (Marrone, Alt, DeDe, Olson, & Shehorn, 2015). Furthermore, noise may delay reading-skill acquisition (Evans, 2006).

Noise has diverse effects on different populations. For example, younger children were found to be more affected by noise than older children (Bradley & Sato, 2008; Newman, 2011), and the elderly compared to their younger adult counterparts (Wong et al., 2009). Furthermore, bi- or trilingual children (Tabri, Chacra, & Pring, 2011) and children experiencing difficulties in learning (Warrier, Johnson, Hayes, Nicol, & Kraus, 2004) or language (Wible, Nicol, & Kraus, 2005) are more sensitive to the harmful effects of noise than typically developing monolingual children.

Neurally, noise degrades central auditory processing (CAP; Kujala & Brattico, 2009), which is fundamental for language acquisition (Kuhl, 2010). CAP denotes the neural processes used for handling auditory information (ASHA Technical Report, 2005). It includes mechanisms needed for auditory discrimination, pattern recognition, and auditory performance during competing acoustic signals, as well as the neurobiological activity engaged in those processes (ASHA Technical Report, 2005). One means to study the effects of noise on CAP is to use event-related potentials (ERPs), which provide millisecond-level information on the electrophysiological events in CAP. ERPs are extracted from an electroencephalogram (EEG) with which it is possible to non-invasively study stimulus-specific processing (Luck, 2005) even in participants with limited co-operational skills.

ERPs are time-locked to external stimuli and the timing and magnitude of ERP components reflect the processing speed and efficiency of CAP, respectively.

In children, sounds elicit obligatory P1, N2, and N4 ERPs in children. The P1 response, reflecting basic sound encoding, has its maximum around 100 ms after stimulus onset (Čeponienė, Rinne, & Näätänen, 2002). The N2 and N4 responses, in turn, reflect higher-order processing (Čeponienė, Alku, Westerfield, Torki, & Townsend, 2005) and forming of memory representations of sounds (Anderson, Chandrasekaran, Yi, & Kraus, 2010; Choudhury & Benasich, 2011). The N2 peaks around 250 ms after stimulus onset in children and is followed by the N4. The obligatory responses are generated at the thalamo-cortical pathway (Ponton, Eggermont, Kwong, & Don, 2000). All these auditory obligatory responses have a fronto-central scalp distribution in children.

Changes in the repetitive aspects of sound stream (e.g., an occasional change from vowel /a/ to vowel /o/) elicit the mismatch negativity (MMN) indexing pre-attentive auditory discrimination and sensory memory (Näätänen, Gaillard, & Mäntysalo, 1978; for a review see Näätänen, Kujala, & Winkler, 2011). The latency of MMN for syllable stimuli is also suggested to reflect language skills in children (Paquette et al., 2015), while the amplitude of MMN correlates with behavioral discrimination skills (Amenedo & Escera, 2000; Kujala & Näätänen, 2010; Kujala, Tervaniemi, & Schröger, 2007). The MMN is elicited at 200–300 ms after stimulus change onset in young children (Kushnerenko, Čeponienė, Balan, Fellman, & Näätänen, 2002; Putkinen, Niinikuru, Lipsanen, Tervaniemi, & Huotilainen, 2012). The main MMN generators are located at the supra-temporal cortices and right frontal lobe (Näätänen & Kreegipuu, 2012). As with the obligatory responses, the auditory MMNs also have a fronto-central scalp distribution (Kujala et al., 2007).

The obligatory responses and the MMN have different developmental trajectories. Responses in infants, children, and adults have different morphology, timing, magnitude, and amplitude distribution (e.g. Glass, Sachse, & von Suchodoletz, 2008; Jing & Benasich, 2006; Kujala et al., 2007; Ponton et al., 2000; Sussman et al., 2008). The developmental trajectories are well reported from birth to 2-years of age and again from school-age onwards, but there are very few reports on the developmental changes of ERPs from 2 to 4 years (Wunderlich, Cone-Wesson, & Shepherd, 2006).

The P1 latency decreases developmentally from early childhood towards adulthood, which was suggested to reflect the maturation of the auditory cortex (Jing & Benasich, 2006; Paquette et al., 2015; Ponton et al., 2000; Sharma, Kraus, McGee, & Nicol, 1997; Sussman et al., 2008). The results on the developmental trajectory of the P1 amplitude in childhood are diverse. The P1 magnitude either was reported to be stable early in the childhood, from 3 to 36 months (Paquette et al., 2015), from 6 to 48 months (Choudhury & Benasich, 2011), from 4 to 9 years (Čeponienė et al., 2002), or maximally at the age of 10–11 months, then decreasing by 24 months (Jing & Benasich, 2006) and from 5 or 8 years to adulthood (Ponton et al., 2000; Sussman et al. 2008, respectively). Similarly to the P1 latency, the N2 latency decreases developmentally from infancy towards adulthood (Choudhury & Benasich, 2011; Jing & Benasich, 2006; Sharma et al., 1997). The N2 amplitude is largest in infancy. From 9 months of age its amplitude decreases to 24 months (Jing & Benasich, 2006) and from 4 years to adulthood (Čeponienė et al., 2002). The N4 response for syllable stimuli has scarcely been studied in young children and its developmental trajectories are unknown. For tone stimuli, the latency of N4 decreases from 3 to 48 months (Choudhury & Benasich, 2011) while the amplitude of N4 increases from 3 to 16 months and then decreases by 24 months (Jing & Benasich, 2006).

The MMN is elicited very early on, even in fetuses (Huotilainen et al., 2005). During early childhood the MMN morphology changes substantially. A positive polarity has been reported in most infant studies, which then changes to a negative MMN during the early months (Choudhury & Benasich, 2011; He, Hotson, & Trainor, 2009; Kushnerenko et al., 2002; Paquette et al., 2015). During development, MMN latency becomes shorter (Morr, Shafer, Kreuzer, & Kurtzberg, 2002). However, the latency of MMN for syllable change was found to be stable for syllable stimuli between 12 to 36 months (Paquette et al., 2015). The amplitude of the MMN for tone stimuli is developmentally stable from toddlers to preschoolers (from 3 to 44 months; Morr et al., 2002; from 2 to 6 years; Glass et al., 2008). The amplitude distribution of MMN shifts from the parietal area towards anterior scalp areas from 3 to 24 months (Jing & Benasich, 2006).

Our previous study suggested that background noise influences CAP of 2-year-old children in several ways (Niemitalo-Haapola, Haapala, Jansson-Verkasalo, & Kujala, 2015). We found that the P1 amplitude decreased and the N2 amplitude increased during noise. Furthermore, the MMN for consonant, frequency, and vowel duration changes decreased and the MMN for intensity and vowel changes was abolished. However, the influence of noise on neural processes at the ages of 2 and 4 years as well as the developmental trajectories of CAP during these years has so far scarcely been studied. Noise might be particularly harmful for the CAP in toddlers because their language system is less mature than that of older children. Thus, their neural representations might be less stable and less noise tolerating than in older children. Therefore, the aim of the present study was to evaluate the effects of background noise on CAP at the ages of 2 and 4 years. This study also allows us to determine the development of the P1, N2, N4, and MMNs. We expected to find detrimental effects of noise on both sound encoding and auditory discrimination. In addition, we assumed that the impact of noise on CAP would be smaller in older than younger children.

## Methods

### Participants

The participants of this follow-up study were 14 children (six girls, eight boys) who were studied twice, at the age of 2 and 4 years. The participants are a sub-group from the studies reporting the eligibility of the multi-feature paradigm in investigating CAP (Niemitalo-Haapola et al., 2013) and the effects of background noise on CAP at the age of 2 years (Niemitalo-Haapola et al., 2015).

Families with potential participants were recruited via internet mailing lists, in parent-child meetings, and with public advertisements. This resulted in 22 volunteering families with 22–26-month-old children (mean age 24 months). The inclusion criteria were: typical development, monolingualism, born from typical pregnancy, and no language or severe mental problems (for example schizophrenia) in the family. In addition, the children were allowed to have a maximum of two diagnosed acute otitis media episodes before study participation at the age of 2 years. All of the participants came from monolingual Finnish families, were born full-term with typical birth-weight, and were developing typically. All aspects of participant development were followed in family clinics, which is a standard practice in Finland. In the written questionnaire, parents reported no concerns about the participant's cognitive (e.g., communication), motor, or physiological development and no language, literacy, or severe mental problems (e.g., depression) in the family.

At age of 2 years and prior to the first EEG registration, typical language development was confirmed using the Finnish version of the Comprehension Scale of Reynell Developmental Language Scales III (Kortesmaa, Heimonen, Merkoski, Warma, & Varpela, 2001), the Finnish version of MacArthur Developmental Inventory (Lyytinen, 1999), and by clinical assessment of a speech sample acquired

in a semi-structured play situation. Before the EEG registrations, an otolaryngologist confirmed that the children had clinically healthy ears. In addition, six children passed transient otoacoustic emissions screening (TEOAE, non-linear click sequence 1.5 to 4.5 kHz, 73 dB SPL, fail/pass, MADSEN AccuScreen® pro, GN Otometrics, Taastrup, Denmark). However, this examination could not be conducted on all children due to lack of child co-operation. The parents of these children reported no concerns about the hearing of their child who had also passed the neonatal otoacoustic emissions screening.

At the age of 4 years, before the second EEG registration, language skills were screened again using the Finnish version of Reynell Developmental Language Scales III (Kortesmaa et al., 2001), Finnish version of the Illinois Test of Psycholinguistic Abilities Auditory Sequential Memory tasks (Kuusinen & Blåfield, 1974), Finnish version of Boston Naming Test (Laine, Koivuselkä-Sallinen, Hänninen, & Niemi, 1997), and by clinical assessment of a speech sample acquired in a semi-structured play situation. An otolaryngologist again examined the children to ensure clinically healthy ears. All returning participants passed the TEOAE screening (MADSEN AccuScreen® pro) and hearing screening (15 dB hearing level; 0.5 kHz, 1 kHz, 2 kHz, and 4 kHz frequencies).

Three children from the 22 volunteers were withdrawn or excluded from the study at the age of 2 years: two children had to be excluded because of a substantial amount of alfa-activity contaminating data and one had to be withdrawn because of an acute otitis media at the time of EEG-registration. At the age of 4 years, an additional four children were withdrawn: one child was over-aged, one refused to continue, one had moved away from the region, and one did not co-operate with the hearing screening and reliable hearing status could not be achieved. In addition, the language assessments showed problems with speech production in one child at the age of 4 and therefore his data was excluded from both samples. Thus, data from 14 children (six girls, eight boys) who participated both

at the ages 2 and 4 years (mean 24 months and 50 months, respectively) was analyzed and reported in this article.

The study was conducted in accordance with the Declaration of Helsinki and the Ethical Committee of Northern Ostrobothnia Hospital District approved the study. The parents gave written informed consent before participation when the children were 2-years old and again before participation at the age of 4 years. The children gave oral informed assent at the age of 4. The families received 15 euros to compensate for traveling cost to the EEG recordings at the age of 2 years and the children received a toy for their time in the EEG recordings at the age of 2 years.

**Stimulation and EEG registration**

The multi-feature MMN paradigm (Näätänen, Pakarinen, Rinne, & Takegata, 2004), including five deviant types, was used because it allows the presentation of multiple deviant types in a short time (Näätänen et al., 2004). It is also more speech-like than the odd-ball paradigm (Kujala et al., 2007; Lovio et al., 2009; Sorokin, Alku, & Kujala, 2010) and diminishes neural refractoriness effects (Kujala et al., 2007). In the multi-feature paradigm standards and deviants alternate and only one feature of the deviant changes at a time. Standards and the unchanged deviant features reinforce the memory representation of the common features of the stimuli (for example frequency or phoneme identity).

In our study, the repetitive standard stimulus (50% of all stimuli) was syllable /ke:/ in half of the stimulus sequences and syllable /pi:/ in the other half (Figure 1). The duration of the standard stimuli was 170 ms (consonant 12 ms and vowel 158 ms) with a 101 Hz fundamental frequency. The five deviant types (consonant, frequency, intensity, and vowel with bi-directional changes, and vowel

duration decrement) resemble speech-sound features typical in Finnish (details of deviant syllables are seen in Table 1). Bi-directional changes were used to control phoneme specificity effects (Lovio et al., 2009), to reduce the neural refractoriness effects of the ERPs (Kujala et al., 2007), and to avoid increment/decrement effects (Lovio et al., 2009). The mono-directional vowel duration decrement was used because of intangible interpretation of the increment-induced MMN (Jacobsen & Schröger, 2003; Putkinen et al., 2012). Our semisynthetic stimuli were produced with the Semisynthetic Speech Generation method (Alku, Tiitinen, & Näätänen, 1999; Sorokin et al., 2010). It enables the manipulation of naturally produced vowels based on an artificial vocal tract model. After the modification of the duration, intensity, and fundamental frequency of glottal flow, the plosive /k/ or /p/ was added to a vowel in order to create a plosive-vowel syllable stimulus with desired characteristics. The stimuli were presented with approximately 75 dB sound-pressure level.

(Table 1 approximately here)

(Figure 1 approximately here)

Each stimulus sequence began with ten repetitive standards (/ke:/ or /pi:/) and had in total 540 stimuli, which were presented so that the standard alternated with the deviant stimuli with a 670 ms stimulus onset asynchrony (Figure 1). The duration of one stimulus sequence was six minutes. The deviant type which preceded a standard stimulus never immediately followed it. In addition to standards and deviants, non-linguistic novel sounds were embedded in the paradigm, the responses for which will be reported in another publication.

The effects of the background noise were studied by presenting background noise in half of the stimulus blocks so that these blocks alternated with the silent ones. The noise was artificial, non-semantic cafeteria noise including babble and other cafeteria sounds presented with a 55 dB SPL (20

dB signal to noise ratio). It was chosen because it resembles the real-life auditory environment of children. In addition, the babble noise was shown to interfere with CAP in adults more than wide-band or traffic noise (Kozou et al., 2005). Depending on the participant's co-operation, six to eight stimulus sequences were presented in total.

The EEG registration and analysis software, hardware, and environment were identical for both age groups (Niemitalo-Haapola et al., 2013, 2015). The EEG-registration booth was sound-damped and electrically shielded. An experienced EEG technician was in charge of the registration and was assisted by two speech and language therapists. For stimulus presentation, the Presentation 13.0 program (Neurobehavioral Systems, CA) and two loudspeakers (Genelec 6010A, Iisalmi, Finland) were used. Headphones were not used because the combination of an electro-cap and headphones might have been intolerable for the 2-year-old participants. The loudspeakers were located 40 degrees to the left and right and 130 cm in front of the participant and were focused towards the participant.

Thirty-two Ag-AgCl active electrodes (Acti-CAP, Brain Products GmbH, Gilching, Germany) with an integrated active shield were used together with the Brain Vision recorder 1.10 (Brain Products GmbH). The electrodes were connected to the electro-cap according to a 10 to 20 system (Klem, Lüders, Jasper, & Elger, 1999). The impedance during the EEG registration was kept under 20 kohm. In addition, bipolar electrodes below the outer canthus of the left eye and above the outer canthus of the right eye were used to register eye movements for ocular artefact rejection. During the EEG registration, online band-pass filtering (0.16 -1000 Hz) with 5 kHz sampling rate was used (BrainVision Brainamp, Brain Products GmbH). To ensure children's co-operation, an FCz electrode was used as the online reference instead of the potentially weakly tolerated nose tip electrode.

During the stimulus presentation children played with silent toys, stickers, books, activity books, or watched muted video. The child and the EEG were constantly monitored during the EEG registration. When necessary, breaks were held and snacks were offered to the participant and the parent. Electrode contacts were inspected and adjusted after breaks and when needed.

**EEG analysis**

The EEG analysis was performed off-line with the Brain Vision Analyzer 2.0 (Brain Products Gmbh). For each participant, the EEG sequences were combined by the listening condition (silent and noisy) separately in both ages. Next, the data from seven electrode locations (Fp1, Fp2, PO9, PO10, O1, Oz, and O2) were rejected because of muscle artifacts. The rest of the electrodes were used for EEG analysis and visual examination of data. The online 5 kHz sampling rate was digitally down-sampled to 250 Hz for off-line analysis. In order to evade aliasing and non-cranial artifacts (Luck, 2005), the signal was digitally off-line filtered (band-pass from 0.5 to 45 Hz, 24 dB/octave, zero phase shift filter). The average of the mastoid values were used as the off-line re-reference to provide better MMN signal to noise ratio (Kujala et al., 2007). After that, the ocular correction was carried out with independent component analysis and data epochs with over ±150 µV amplitudes at any electrodes, with low activity (1.5 µV in 100 ms time window) or with voltage gradient (50 µV/ms), were withdrawn. Then, the off-line band-pass filter (1 to 20 Hz, 48 dB/octave, zero phase shift filter) was applied to diminish noise in the EEG data. We used digital zero phase shift filters because they do not produce phase conversion (Cook & Miller, 1992).

After artefact rejection and filtering, the data was divided into 600 ms segments (from -100 ms to 500 ms from the stimulus onset) and clustered based on the stimulus type (standard, consonant change, frequency change, intensity change, vowel change, and vowel decrement) in both age groups and

conditions separately. Ten standards in the beginning of each EEG-sequence and standards following novel sounds were excluded from the clustering. The clusters were baseline corrected according to the 100 ms pre-stimulus time window and averaged based on the stimulus type.

The P1, N2, N4 peaks and MMN were separately visually detected from the standard grand average wave (P1, N2, and N4 responses) and from the deviant minus standard difference wave (MMN) at both ages and conditions by using the most representative electrode (Table 2). The age and component specific representative electrodes were selected based on previous literature (Kujala et al., 2007; Ponton et al., 2000) and visual inspection of the data. Namely, the scalp distributions of the different ERPs vary and also the age influences the distributions (Ponton et al., 2000). Therefore, to detect each response the most reliably, the representative electrode, showing the maximum amplitude, was used. Table 2 presents the time windows used for peak detection. The peak latencies were identified from the representative electrode in the selected time window (Table 2). The mean amplitudes with a ±20 ms integration time window centered at the peak at the representative electrode were measured at electrodes F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 for each response in both ages and conditions. In addition, the amplitude distributions were evaluated visually from 25 electrodes.

(Table 2 approximately here)

**Statistical analysis**

The numeric data from nine electrodes (F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4) were exported and statistically analyzed with Statistica 10. First, the existence of every response was determined with a *t*-test by comparing the amplitudes from the representative electrodes to zero. The analysis was continued with repeated-measures ANOVA (within-subject factors age, condition, anterior-posterior

electrode location (AP), left-right electrode location (LR), and deviant type) which was used to investigate the effects of age and background noise on every response. The latency and amplitude scalp distribution comparisons were carried out only between responses that were significantly different from zero. If necessary, Greenhouse-Geisser correction was used. For the source detection and effect size determination, Fisher's least significant difference post hoc test and partial eta squared ($\eta_p^2$) were calculated, respectively.

## Results

### Developmental changes in obligatory responses

In the silent condition, the obligatory P1, N2, and N4 responses significantly differed from zero at the ages of 2 and 4 years (Table 3, Figure 2). From 2 to 4 years, the P1 and N2 latencies significantly decreased [for P1 $F(1, 13) = 103.13$, $p < 0.001$, $\eta_p^2 = 0.89$, post hoc $p < 0.001$; for N2 $F(1, 13) = 4.59$, $p = 0.052$, $\eta_p^2 = 0.26$, post hoc $p = 0.053$]. The N4 latency increased from 2 to 4 years, but the change was not statistically significant.

(Table 3 approximately here)

(Figure 2 approximately here)

No developmental changes were found in the P1 amplitude (Figures 2 and 3, Table 3). However, there was a significant anterior-posterior x age interaction [$F(1, 18) = 4.52$, $p = 0.038$, $\eta_p^2 = 0.26$, Supplement 1]. At the age of 2 years, the P1 amplitude was the strongest at the frontal and central electrodes (post hoc $p < 0.001$), while at the age of 4 years the P1 was the strongest at the frontal

electrodes (post hoc $p \leq 0.001$). At both ages the P1 response was the smallest at the parietal electrodes (post hoc $p < 0.001$).

(Figure 3 approximately here)

The N2 amplitude increased developmentally, being larger at the age of 4 years than at the age of 2 years [$F(1, 13) = 11.70$, $p = 0.005$, $\eta_p^2 = 0.47$, post hoc $p = 0.005$; Figures 2 and 3, Table 3]. In addition, the anterior-posterior distribution as well as left-right distribution of the N2 response changed [age x AP interaction $F(1, 15) = 18.26$, $p = 0.001$, $\eta_p^2 = 0.58$; age x LR interaction $F(2, 26) = 3.87$, $p = 0.03$, $\eta_p^2 = 0.23$; Figure 2, Supplement 1]. At the age of 2 years the N2 response was the strongest at the central electrodes (post hoc $p \leq 0.001$), whereas at the age of 4 years it was the strongest at both frontal and central electrodes (post hoc $p < 0.001$). At the age of 2 years the N2 response was stronger laterally than at the midline (post hoc $p = 0.048$) and at the age of 4 years it was the strongest on the left hemisphere (post hoc $p \leq 0.001$).

The N4 amplitude also increased developmentally, being larger at the age of 4 than 2 years [$F(1, 13) = 20.60$, $p = 0.001$, $\eta_p^2 = 0.61$, post hoc $p = 0.001$; Figures 2 and 3, Table 3]. The N4 scalp distribution changed developmentally [age x AP interaction $F(1, 17) = 13.08$, $p = 0.001$, $\eta_p^2 = 0.50$; Figure 2, Supplement 1]. At the age of 2 years, the N4 response was the strongest at frontal and central electrodes (post hoc $p < 0.001$), while at the age of 4 years the N4 response was the strongest at frontal electrodes (post hoc $p \leq 0.002$). At both ages the N4 response was the smallest at the parietal electrodes (post hoc $p \leq 0.019$).

**Noise-induced changes in obligatory responses**

Noise had several effects on the obligatory responses. However, no condition x age interactions were found for the timing, magnitude, or distribution of the P1, N2, and N4 indicating that the effects of noise on the obligatory responses were similar at both ages.

In the noisy condition, the obligatory P1, N2, and N4 responses significantly differed from zero at both ages. Noise had no significant effects on the P1 and N2 latencies (Figure 2, Table 3), whereas the N4 latency was shorter in the noisy than silent condition [$F(1, 13) = 24.83$, $p < 0.001$, $\eta_p^2 = 0.66$, post hoc $p < 0.001$].

At both ages, the P1 amplitude was smaller in the noisy than in the silent condition [$F(1, 13) = 59.31$, $p < 0.001$, $\eta_p^2 = 0.82$, post hoc $p < 0.001$; Figures 2 and 3, Table 3]. Noise affected also the anterior-posterior distribution [condition x AP interaction $F(1, 17) = 19.13$, $p < 0.001$, $\eta_p^2 = 0.60$, Supplement 1]. In the silent condition the P1 response was the strongest at the frontal electrodes (post hoc $p \leq 0.03$) and the smallest at the parietal electrodes (post hoc $p < 0.001$), whereas in the noisy condition the P1 response was the strongest at frontal and central electrodes (post hoc $p < 0.001$).

In contrast to the P1 amplitude, the N2 amplitude was stronger in the noisy than silent condition at both ages [$F(1, 13) = 34.42$, $p < 0.001$, $\eta_p^2 = 0.73$, post hoc $p < 0.001$; Figures 2 and 3, Table 3]. Noise affected also the anterior-posterior distribution of the N2 response [condition x AP interaction $F(2, 26) = 15.58$, $p < 0.001$, $\eta_p^2 = 0.55$, Supplement 1]. In the silent condition, the N2 response was the strongest at central electrodes (post hoc $p < 0.001$), whereas in the noisy condition the N2 response was the strongest at frontal and central electrodes (post hoc $p < 0.001$).

For the N4, no influence of noise was found on the amplitude, but the amplitude distribution changed by noise [condition x AP interaction $F(2, 26) = 7.35$, $p = 0.003$, $\eta_p^2 = 0.36$; Figures 2 and 3,

Supplement 1]. In the silent condition, the N4 amplitude was the strongest at the frontal and central electrodes (post hoc $p < 0.001$), whereas in the noisy condition, it was the strongest at frontal electrodes (post hoc $p \leq 0.002$).

**Developmental changes in the MMN responses**

The MMNs were significant for all deviant types at both ages in the silent condition (Figure 4, Table 3). The MMN latencies decreased from 2 to 4 years of age, but the differences were not statistically significant.

(Figure 4 approximately here)

The MMN amplitudes increased developmentally [$F(1, 13) = 5.16$, $p = 0.041$, $\eta_p^2 = 0.28$, post hoc $p = 0.041$; Figure 4], but there were no significant age x deviant type interactions, suggesting similar developmental trajectories for the deviant types. In addition, developmental changes were found in the MMN amplitude distribution [age x AP interaction $F(2, 26) = 8.42$, $p = 0.002$, $\eta_p^2 = 0.39$; Figure 5, Supplement 2]. At the age of 2 years, the MMNs were the strongest at the central electrodes and the smallest at the parietal electrodes (post hoc $p = 0.007$). At the age of 4 years, the MMNs were the strongest frontally (post hoc $p \leq 0.027$). The MMN changed developmentally also in the left-right dimension [age x LR interaction $F(2, 26) = 3{,}86$, $p = 0.030$, $\eta_p^2 = 0.23$]. At the age of 2 years, the MMN distribution was even between the left hemisphere, midline, and the right hemisphere. At the age of 4 years, the MMNs were the strongest at midline and the right hemisphere (post hoc $p = 0.030$).

(Figure 5 approximately here)

**Noise-induced changes in the MMN responses**

Only the consonant deviant elicited a significant MMN response in the noise condition at both ages. At the age of 4, there was also a significant MMN response in noise condition to the vowel duration deviant (Table 3). When including MMNs for all deviant types in the analysis, the MMN amplitudes were overall smaller in the noisy condition than in the silent condition [$F(1, 13) = 33.81$, $p < 0.001$, $\eta_p^2 = 0.72$, post hoc $p < 0.001$; Figure 4]. However, no condition x age interactions were found on the MMN amplitudes, suggesting similar effects of noise on the MMNs at both ages.

Because the MMN was significantly elicited in both conditions only by the consonant change in both ages and by the duration change in 4-year old children, latency and distribution analyses could be carried out only for these MMNs. Noise had no significant influence on the latency, amplitude or scalp distribution of the MMN elicited by the consonant change or on the latency of the MMN elicited by the vowel duration change (Supplement 2). The amplitude of MMN for vowel duration change was smaller in the noisy than silent condition [$F(1, 13) = 41.05$, $p < 0.001$, $\eta_p^2 = 0.76$, post hoc $p < 0.001$; Table 3]. In addition, noise significantly influenced the anterior-posterior x left-right distribution of the MMN elicited by the vowel duration changes [condition x AP x LR interaction $F(2, 32) = 3.19$, $p = 0.044$, $\eta_p^2 = 0.20$]. The MMN for the vowel duration change was the strongest fronto-centrally in the silent condition (post hoc $p < 0.001$), and frontally in the noisy condition (post hoc $p \leq 0.038$). In addition, in the silent condition, it was stronger at Cz than C3 (post hoc $p = 0.038$) and in the noisy condition the smallest at Cz and the strongest at C3 (post hoc $p = 0.018$).

**Discussion**

The aim of our study was to evaluate changes in CAP between different listening conditions and during development by investigating the alterations in ERP latencies, amplitudes, and scalp distributions. We expected developmental changes and detrimental effects of noise on speech-sound encoding and preattentive auditory discrimination in both age groups. In addition, we hypothesized that the changes would be smaller in older children with more matured and thus possibly more noise tolerating CAP than in the younger children. We found that speech-sound encoding and preattentive auditory discrimination change during development and due to noise in typically developing children. In addition, noise influenced in multiple ways central auditory processing of both the 2- and 4-year-old children.

**Developmental changes**

We found several developmental changes in ERPs reflecting the maturation of CAP between the ages of 2 and 4 years. Our main findings were decreased P1 and N2 latencies indexing maturational changes in neural timing, increased N2, N4, and MMN amplitudes reflecting changes in activation strength, and altered scalp distributions due to the strongest amplitude changes at frontal electrodes.

In our study, the timing of P1 and N2 changed developmentally while the N4 and MMN responses showed no significant latency changes. The latency of P1, indexing sound encoding, diminished as was expected based on previous findings in small children (Jing & Benasich, 2006; Paquette et al., 2015; Ponton et al., 2000; Sharma et al., 1997), and the suggestion that the P1 latency is a biological marker of auditory pathway maturity (Sharma et al., 1997). The N2 latency, indicating also forming of memory representation of sounds (Anderson et al., 2010; Choudhury & Benasich, 2011), was diminished developmentally as expected (Choudhury & Benasich, 2011; Jing & Benasich, 2006; Paquette et al., 2015; Sharma et al., 1997). The latency of N4 increased but not statistically

significantly. The MMN latencies were also altered, consistent with the findings of Glass et al. (2008) in 2- to 6-year-old children, but this change did not reach statistical significance.

The P1, N2, and N4 amplitudes had different developmental trajectories in our study, the P1 amplitude being stable and both N2 and N4 increasing from 2 to 4 years. Our finding of developmentally stable P1 amplitude in young children is in line with previous studies, which showed no significant P1 amplitude changes in childhood from 3 to 36 months (Paquette et al., 2015) or from 6 to 48 months (Choudhury & Benasich, 2011). After the age of 10 years, the P1 has been found to remarkably decrease towards adulthood (Ponton et al., 2000). The N2 amplitude, in turn, increased in our study from 2 to 4 years. In previous studies, the amplitude of the N2 for vowel stimuli was developmentally stable from infancy to school years (Shafer, Yu, & Warner, 2015) while the amplitude of N2 for tone stimuli was shown to decrease from 9 to 24 months (Jing & Benasich, 2006), as well as from 4 years to the adulthood, when N2 response no longer exists (Čeponienė, 2002). Thus, the developmental trajectory of the N2 response might be different for various types of stimuli. In addition, we found that the N4 amplitude increased from 2 to 4 years. Due to the lack of developmental studies focusing on this component, it is impossible to tell whether this result also applies to other types of stimuli than syllables used in our study and a more complete developmental trajectory of this response remains to be further clarified in future studies.

In the current study, the MMN amplitudes increased from 2 to 4 years, which might reflect developmental maturation of auditory discrimination as the MMN amplitude increment is connected with an improved accuracy in auditory discrimination (see Kujala and Näätänen, 2010, for a review). Our findings are in line with Paquette et al. (2015) showing an increment of MMN amplitudes for syllable stimuli from 12 to 36 months. However, the pattern for the development of the MMNs elicited by tone stimuli was reported to be different. Their amplitudes were found to decrease from 6 to 48

months (Choudhury & Benasich, 2011) and from 12 to 36 months (Jing & Benasich, 2006). These differences might be explained by the different maturational trajectories of MMN amplitudes for different sound types (Partanen et al., 2013).

Also the amplitude distribution of ERPs changed during development. Both obligatory responses and the MMNs increased frontally from 2 to 4 years consistent with previous studies (Ponton et al., 2000). This result might suggest maturational changes with increased activity in the frontal neural generators from 2 to 4 years.

**Noise-induced changes**

In the present study, noise altered CAP as reflected by the noise-induced changes of the ERPs. Noise had no major impact on the processing speed of sound encoding or auditory discrimination as suggested by the unaltered latencies during noise. However, those processes were degraded by noise at both ages as reflected by the diminished P1 amplitude, the increased N2 amplitude and the diminished MMNs. In addition, in contrast to our hypothesis, we found no major differences in the effects of noise on the CAP between the ages.

We found no noise-induced changes on the latencies of the P1, N2, and MMNs for consonant and vowel duration changes, whereas the latency of the N4 diminished during noise. Thus, noise had no major effect on the processing speed of early stages of sound encoding, memory representation forming, or auditory discrimination in children. Because the functional role of N4 is still poorly understood, the implications of its noise-induced latency shortening remains to be determined by future studies. Previous studies carried out in adults have found mixed results on noise-induced changes in MMN latencies, reporting decreased (Kozou et al., 2005), stable (Kozou et al., 2005) or

increased (Martin, Kurzberg, & Stapells, 1999; Muller-Gass, Marcoux, Logan, & Campbell, 2001) MMN latencies during noise. These differences might be due to different noise types (Kozou et al., 2005) or noise levels (Muller-Gass et al., 2001) used.

In the current study, the P1 amplitude diminished in noise conditions similarly at both ages, which is in line with the findings of Anderson et al. (2010). The P1 amplitude decrement by noise was previously connected with impaired pre-attentive speech-sound encoding (Anderson et al., 2010; Niemitalo-Haapola et al., 2015), which might partially result from the acoustical mixing of the target signals and noise. In our study, the N2 amplitude, in turn, was increased in noise conditions at both ages. An enhanced N2 amplitude in noise might suggest that more neural resources are needed for speech-sound representation forming during noise (Anderson et al., 2010; Niemitalo-Haapola et al., 2015).

Noise degraded cortical auditory discrimination in children, as reported previously in adults (Kozou et al., 2005; Martin et al., 1999; Muller-Gass et al., 2001; Shtyrov et al., 1998; for a review, see Kujala & Brattico, 2009). In children at both ages, the frequency, intensity, and vowel changes elicited no significant MMN responses in noise conditions, which suggests poorer discrimination of sound changes in the noisy than silent condition. Only the consonant change elicited the MMN in noise condition at both ages, with no noise-induced changes on its amplitude or scalp distribution. The consonants (/k/ or /p/) used in our study were short, only 12 ms, thus they might have been audible during the short decrements in noise levels. In addition, both /k/ and /p/ are plosives differing only by the articulation place, which might be behaviorally easier to discriminate than sounds differing in articulation manner (Kraus et al., 1996), also during noise (Hazan et al., 2013). Therefore, the consonant change from /k/ to /p/ and from /p/ to /k/ might have been detectable during noise. In addition to the MMN to consonant changes, the MMN to vowel duration changes was present in noise

condition at the age of 4, but its amplitude was diminished, as also previously reported in adults (Kozou et al., 2005). The persistence of the MMN to vowel duration change during noise could be expected because it was very robust in the silent condition, suggesting that this change was easy to discriminate. However, this MMN was absent in younger children during noise, which was the only noise-related difference between the two ages in our study. This finding gives some support to our hypothesis on the more vulnerable CAP of 2-year-old compared to 4-year-old children in noise. However, these results should be replicated and extended in future studies to make any firm conclusions. Overall, our results of different effects of noise on MMNs support previous findings of different auditory discrimination profiles of speech-sound features during noise (Hazan et al., 2013; Niemitalo-Haapola et al., 2015; Shetake et al., 2011).

Moreover, the distributions of the P1, N2, N4, and MMN for the vowel duration change were altered during noise as previously reported for vowel change elicited MMN in adults (Kozou et al., 2005). In our study, the noise-induced amplitude changes of these responses were the largest at the frontal electrode locations, suggesting noise-induced modulation of frontal neural generators (Ponton et al., 2000).

In the current follow-up study, we assumed that the impact of noise on CAP is smaller at the age of 4 years than at the age of 2 based on previous behavioral findings of weaker speech comprehension during noise at the age of 6 than at the ages of 8 or 11 years (Bradley & Sato, 2008). However, we found no major differences in the impact of noise on ERPs between these ages, with the exception of present MMN for the vowel duration changes at the age of 4. These results suggest that at the neural level, the impact of noise is to a large extent equally detrimental at both ages. The reason for the different findings in our and Bradley & Sato (2008) study might be that these studies tap different

processes; neural bottom-up preattentive vs. top-down attentive processes, respectively. Also the different ages of the participants in these two studies as well as the small participant group in the current study might have influenced the results.

**Limitations and future studies**

The size of the final participant group, 14 children, was relatively small, which must be taken into account while interpreting the results. Due to this, for example, some possible influences of age on CAP during noise might not have been detected in the current study. However, the children were their own references for developmental as well as for the listening condition comparisons, which, on the other hand, improved the reliability of the results. Also the age range of the participants was kept narrow in order to avoid extensive developmental intersubject variability.

The stimuli of the current study were presented through loudspeakers and, therefore, the stimuli might have variably entered the two ears during the EEG recordings due to head movements. This could potentially have influenced the lateralization of the ERP components the results on which should, therefore, be interpreted with caution. Furthermore, since the scalp-distribution analyses carried out in the current study can only provide limited information on the different neural sources, future studies with advanced source-modelling approaches are needed. Also in order to determine in more detail preattentive and attentive processing of speech during noise, future research including both behavioral and ERP testing in different acoustic conditions is needed.

In the future, also the effects of long-term noise exposure on children would be important to study. Investigation of children from different sub-urban areas with different background noise levels might provide more information on potentially harmful long-term noise-induced changes on CAP.

In addition, it should be determined whether it is possible to increase the noise tolerance of the auditory system with some more active means. For example, Parbery-Clark, Skoe, & Kraus (2009) have demonstrated more robust cABRs and ERPs during noise in people with musical education, suggesting that their auditory system is more tolerant to noise than that of musically untrained. Consistent with this, it has been shown that musical training modifies CAP in children (for a review, Kraus & Chandrasekaran, 2010). Thus, it would be worth of investigating further what kind of musical training has a protective effect against the deleterious effects of noise in young children.

**Clinical implications**

The well-functioning CAP, e.g. sound encoding and auditory discrimination, forms a basis for the development of language. Our results suggest that noise hampers encoding and memory representation formation for speech-sound features in children, as reflected by the P1 and N2 amplitude changes, as well as discrimination of speech-sound features, as reflected by the diminished MMN amplitudes. Thus, noise is a potential risk for language acquisition. The awareness of harmful effects of noise on CAP should be increased among professionals working with children, and the exposure to noise should be minimized in children's daily life. This means that acoustic issues should be considered when planning and renovating day-care centers and schools. For direct noise reduction, for example, kindergarten teachers should be instructed to use different pedagogical means, i.e. to divide children into small subgroups, in order to decrease noise originating from the activity.

Participants in our study were typically developing children. Previously, it has been shown that children with language problems are even more vulnerable to the negative effects of noise on CAP than typically developing children (Wible et al., 2005). Thus, the hampering effects of noise found in

our study might be even more pronounced in children with language problems and dyslexia, which already involve CAP dysfunctions. Therefore, in speech and language therapy, the evaluation and improvement of these children's real-life listening conditions should be an essential part of assessment and intervention.

## Conclusions

The present study shows developmental changes and unfavorable effects of background noise on CAP as indexed by ERPs within the same population of children studied at the ages of 2 and 4 years. From the age of 2 to 4 years, the P1 and N2 latency decreased, the N2, N4, and MMN amplitudes increased, and the scalp distribution of all these responses changed, suggesting several kinds of maturational changes of CAP. Background noise decreased the P1 amplitude, increased the N2 amplitude, decreased the N4 latency, diminished the MMN amplitudes, and altered scalp distributions of the P1, N2, N4, and MMN for the vowel duration change, reflecting altered sound encoding and memory representation formation as well as impaired auditory discrimination during noise. These potentially negative effects of noise on CAP were to a large extent similar at both ages. Our results together with previous findings in school-aged children (Anderson et al., 2010) and in adults (Kozou et al., 2005; Martin et al., 1999; Muller-Gass et al., 2001; Shtyrov et al., 1998) indicate that noise interferes with CAP, as indexed by altered ERPs, at different ages.

## Acknowledgements

The authors wish to thank all of the children who participated in the study and their families for their invaluable help. We also wish to thank EEG technicians Raija Remes and Sari Pehkonen and speech and language therapist Henna Häkli MA for their help with the data collection and A.B. of Semantix

Finland Oy for language proofreading the article. In addition, we wish to thank physicist Kalervo Suominen Phil. Lic., Pasi Lepola PhD, Tuomo Starck PhD, and technician Hannu Wäänänen for their technical support during the different phases of the study.

The study was supported by the Child Language Research Center (E.N.-H.), the Emil Aaltonen foundation (E.N.-H.), The Jenny and Antti Wihuri Foundation (E.N.-H.), Oulu University Fund (E.N.-H), The Finnish Ministry of Education and Culture, the Langnet Doctoral Programme (S.H.), and The Academy of Finland (Grant 276414; T. K.).

The authors declare no other conflict of interest.

## References

Alku, P., Tiitinen, H., & Näätänen, R. (1999). A method for generating natural-sounding speech stimuli for cognitive brain research. *Clinical Neurophysiology, 110*, 1329–1333.

Amenedo, E., & Escera, C. (2000). The accuracy of sound duration representation in the human brain determines the accuracy of behavioural perception. *European Journal of Neuroscience, 12*, 2570–2574.

American Speech-Language-Hearing Association. (2005). *(central) auditory processing disorders* [Technical Report]. Available from www.asha.org/policy. doi:10.1044/policy.TR2005-00043

Anderson, S., Chandrasekaran, B., Yi, H-G., & Kraus, N. (2010). Cortical-evoked potentials reflect speech-in-noise perception in children. *European Journal of Neuroscience, 32*, 1407-1413.

Bishop, D.V.M. (2014). *Uncommon understanding (Classic edition): Development and disorders of language comprehension in children.* Hove: Psychology Press.

Bradley, J. S., & Sato, H. (2008). The intelligibility of speech in elementary school classrooms. *Journal of Acoustical Society of America, 123,* 2078–2086.

Čeponienė, R., Alku, P., Westerfield, M., Torki, M., & Townsend, J. (2005). ERPs differentiate syllable and nonphonetic sound processing in children and adults. *Psychophysiology, 42,* 391–406.

Čeponienė, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, *113*, 870–882.

Choudhury, N., & Benasich, A. (2011). Maturation of auditory evoked potentials from 6 to 48 months: Prediction to 3 and 4 year language and cognitive abilities. *Clinical Neurophysiology, 122,* 320–338.

Cook, E. W. III, & Miller, G. A. (1992). Digital filtering: Background and tutorial for psychophysiologists. *Psychophysiology, 29*, 350–367.

Evans, G. W. (2006). Child development and the physical environment. *Annual Review of Psychology, 57,* 423–451.

Glass, E., Sachse, S., & von Suchodoletz, W. (2008). Development of auditory sensory memory from 2 to 6 years: an MMN study. *Journal of Neural Transmission, 115,* 1221–1229.

Gifford, R. H., Bacon, S. P., & Williams, E. J. (2007). An Examination of Speech Recognition in a Modulated Background and of Forward Masking in Younger and Older Listeners. *Journal of Speech, Language, and Hearing Research*, *50*, 857–864.

Hazan, V., Messaoud-Galusi, S., & Rosen, S. (2013). Effect of talker and intonation variability on speech perception in noise in children with dyslexia. *Journal of Speech, Language, and Hearing Research, 56*, 44–62.

He, C., Hotson, L., & Trainor, L. J. (2009). Maturation of cortical mismatch responses to occasional pitch change in early infancy: Effects of presentation rate and magnitude of change. *Neuropsychologia, 47*, 218–229.

Hohmann, C., Grabenhenrich, L., de Kluizenaar, Y., Tischer, C., Heinrich, J., Chen C.-M., Thijs, C., Nieuwenhuijsen, M., & Keil, T. (2013). Health effects of chronic noise exposure in pregnancy and childhood: A systematic review initiated by ENRIECO. *International Journal of Hygiene and Environmental Health, 216*, 217–229.

Huotilainen, M., Kujala, A., Hotakainen, M., Parkkonen, L., Taulu, S., Simola, J., …Näätänen, R.(2005). Short-term memory functions of the human fetus recorded with magnetoencephalography, *Neuroreport, 16*, 81–84.

Hygge, S., Boman, E., & Enmarker, I. (2003). The effects of road traffic noise and meaningful irrelevant speech on different memory systems. *Scandinavian Journal of Psychology, 44,* 13–21.

Jacobsen, T., & Schröger, E. (2003). Measuring duration mismatch negativity. *Clinical neurophysiology, 114*, 1133–1143.

Jansson-Verkasalo, E., Ruusuvirta, T., Huotilainen, M., Alku, P., Kushnerenko, E., Suominen, K.,…Hallman, M. (2010). Atypical perceptual narrowing in prematurely born infants is associated with compromised language acquisition at 2 years of age. *BMC Neuroscience.* 30, 11:88.

Jing, H., & Benasich, A. A. (2006). Brain responses to tonal changes in the first two years of life. *Brain & Development, 28*, 247–256.

Klem, G.H., Lüders, H. O., Jasper, H. H., & Elger, C. (1999). The ten-twenty electrode system of the International Federation. In G. Deuschl & A. Elsen (Eds.), Recommendations for the Practice of Clinical Neurophysiology: Guidelines of the International Federation of Clinical Physiology (EEG Suppl. 52) (pp. 3–6). Elsevier Science B.V.

Kortesmaa, M., Heimonen, K., Merikoski, H., Warma, M.-L., & Varpela, V. (2001). Finnish version of Reynell Developmental Language Scales III. Helsinki, Finland: Psykologien kustannus Oy.

Kozou, H., Kujala, T., Shtyrov, Y., Toppila, E., Starck, J., Alku, P., & Näätänen, R. (2005). The effect of different noise types on the speech and non-speech elicited mismatch negativity. *Hearing Research, 199,* 31–39.

Kraus, N., & Chandrasekaran B. (2010). Music training for the development of auditory skills. *Nature review Neuroscience*, 11, 599–605.

Kraus, N., McGee, T. J., Carrell, T.D., Zecker, S. G. Nicol, T. G., & Koch, D. B. (1996). Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science, 273*, 971–973.

Kuhl, P. (2010). Brain Mechanisms in Early Language Acquisition. *Neuron, 67,* 713-727.

Kujala, T., & Brattico, E. (2009). Detrimental noise effects on brain's speech functions. *Biological Psychology, 81,* 135–143.

Kujala, T., & Näätänen, R. (2010). The adaptive brain: A neurophysiological perspective. *Progress in Neurobiology, 91,* 55–67.

Kujala, T., Tervaniemi, M., & Schröger, E. (2007). The mismatch negativity in cognitive and clinical neuroscience: theoretical and methodological considerations. *Biological Psychology, 74,* 1–19.

Kushnerenko, E., Čeponienė, R., Balan, P., Fellman, V., & Näätänen, R. (2002). Maturation of the auditory change detection response in infants: a longitudinal ERP study. *Neuroreport, 13,* 1843–1848.

Kuusinen, J., & Blåfield, L. (1974). Finnish version of Illinois test of psycholinguistic abilities, Auditory sequential memory task. Helsinki, Finland: Psykologien kustannus Oy.

Lovio, R., Pakarinen, S., Huotilainen, M., Alku, P., Silvennoinen, S., Näätänen, R., & Kujala, T. (2009). Auditory discrimination profiles of speech sound changes in 6-year-old children as determined with multi-feature MMN paradigm. *Clinical Neurophysiology, 120,* 916–921.

Laine, M., Koivuselkä-Sallinen, P., Hänninen, R., & Niemi, J. (1997). Finnish version of Boston naming test. Helsinki, Finland: Psykologien kustannus Oy.

Langguth, B., Kreuzer, P. M., Kleinjung, T., & De Ridder, D. (2013) Tinnitus: causes and clinical management. *The Lancet Neurology, 12,* 920–930.

Luck, S. J. (2005). An Introduction to the Event-Related Potential Technique. Cambridge, MA: The MIT Press.

Lyytinen, P. (1999). Finnish version of MacArthur Communicative Development Inventories (MCDI). Jyväskylä, Finland: Niilo Mäki Institute.

Marrone, N., Alt, M., DeDe, G., Olson, S., & Shehorn, J. (2015). Effects of Steady-State Noise on Verbal Working Memory in Young Adults. *Journal of Speech, Language, & Hearing Research*, *58*, 1793-1804.

Martin, B. A., Kurzberg, D., & Stapells, D. R. (1999). The effects of decreased audibility produced by high-pass noise masking on N1 and the mismatch negativity to speech sounds /ba/ and /da/. *Journal of Speech, Language, and Hearing Research, 42*, 271–286.

McAllister, A.M., Granqvist, S., Sjölander, P., & Sundberg, J. (2009). Child voice and noise: A pilot study of noise in day cares and the effects on 10 children's voice quality according to perceptual evaluation. *Journal of Voice*, *23*, 587–593.

Morr, M. L., Shafer, V. L. Kreuzer, J. A., & Kurtzberg, D. (2002). Maturation of mismatch negativity in typically developing infants and preschool children. *Ear & Hearing*, *23*, 118–136.

Muller-Gass, A., Marcoux, A., Logan, J., & Campbell, K. B. (2001). The intensity of masking noise affects the mismatch negativity to speech sounds in human subjects. *Neuroscience Letters*, *299*, 197–200.

Newman, R. (2011). 2-year-olds' speech understanding in multitalker environments. *Infancy, 16*, 447–470.

Niemitalo-Haapola, E., Haapala, S., Jansson-Verkasalo, E., & Kujala, T. (2015). Background noise degrades central auditory processing in toddlers, *Ear and Hearing, 36,* 342–351.

Niemitalo-Haapola, E., Lapinlampi, S., Kujala, T., Alku. P., Kujala, T., Suominen, K., & Jansson-Verkasalo, E. (2013). Linguistic multi-feature paradigm as an eligible measure of central auditory processing and novelty detection in 2-year-old children. *Cognitive Neuroscience,* doi: 10.1080/17588928.2013.781146

Näätänen R., Gaillard, A. W. K., & Mäntysalo, S. (1978). Early selective attention effect on evoked potential reinterpreted. *Acta Psychologica, 42*, 313–29.

Näätänen, R., & Kreegipuu, K. (2012). The Mismatch Negativity (MMN). In S. J. Luck & E. S. Kappenman (Eds.), *The Oxford Handbook of Event-related Potential Components* (pp. 143–158). Oxford: Oxford University Press.

Näätänen, R., Kujala, T., & Winkler, I. (2011). Auditory processing that leads to conscious perception: a unique window to central auditory processing opened by the mismatch negativity and related responses. *Psychophysiology, 48,* 4–22.

Näätänen, R., Pakarinen, S., Rinne, T., & Takegata, R. (2004). The mismatch negativity (MMN): towards the optimal paradigm. *Clinical Neurophysiology, 115,* 140–144.

Paquette, N., Vannasing, P., Tremblay, J., Lefebvre, F., Raoy, M.-S., McKerral, M., …Gallagher, A. (2015). Early electrophysiological markers of atypical language processing in prematurely born infants. *Neuropsychologia, 79,* 21–32.

Parbery-Clark, A, Skoe, E., & Kraus, N. (2009). Musical experience limits the degradative effects of background noise on the neural processing of sounds. *Journal of Neuroscience, 29*, 14100–14107.

Partanen, E. Torppa, R., Pykäläinen, J., Kujala. T., & Huotilainen, M. (2013). Children's brain responses to sound changes in pseudo words in a multi-feature paradigm. *Clinical Neurophysiology, 124,* 1132–1138.

Ponton, C., Eggermont, J. J., Kwong, B., & Don, M. (2000). Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clinical Neurophysiology, 111,* 220–236.

Prodi, N., Visentin, C., & Feletti, A. (2013). On the perception of speech in primary school classrooms: Ranking of noise interference and of age influence. *Acoustical Society of America, 133,* 255–268.

Putkinen, V., Niinikuru, R., Lipsanen, J., Tervaniemi, M., & Huotilainen, M. (2012). Fast measurement of auditory event-related potential profiles in 2–3-year-olds. *Developmental Neuropsychology, 37,* 51–75.

Riley, K. G., & McGregor, K. K. (2012). Noise hampers children's expressive word learning. *Language, speech, and hearing services in schools, 43,* 325–337.

Shafer, V., Yu, Y. H., & Wagner. (2015). Maturation of cortical auditory evoked potentials (CAEPs) to speech recorded from frontocentral and temporal sites: Three months to eight years of age. *International Journal of Psychophysiology, 95,* 77–93.

Sharma, A., Kraus, N., McGee, T. J., & Nicol T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables, *Electroencephalography and Clinical Neurophysiology, 104,* 540–545.

Shield, B., & Dockrell, J. E. (2004). External and internal noise surveys of London primary schools. *Journal of Acoustical Society of America, 115,* 730–738.

Shtyrov, Y., Kujala, T., Ahveninen, J., Tervaniemi, M., Alku, P., Ilmoniemi, R., & Näätänen, R. (1998). Background acoustic noise and the hemispheric lateralization of speech processing in the human brain: magnetic mismatch negativity study. *Neuroscience Letters, 251,* 141–144.

Sorokin, A., Alku, P., & Kujala, T. (2010). Change and novelty detection in speech and non-speech sound streams. *Brain Research, 1327,* 77–90.

Sussman, E., Steinschneider, M., Gumenyuk, V., Grushko, J., & Lawson, K. (2008). The maturation of human evoked brain potentials to sounds presented at different stimulus rates. *Hearing Research, 236,* 61–79.

Söderlund, G. B. W., Sikström, S., Loftesnes, J. M., & Sonuga-Barke, E. J. (2010). The effects of background white noise on memory performance in inattentive school children. *Behavioral and Brain Functions*, 6:55.

Tabri, D., Chacra, K.M.S.A., & Pring, T. (2011). Speech perception in noise by monolinguagl, bilingual and trilingual listeners. *International Journal of Language & Communication Disorders*, *46*, 411–422.

Warrier, C. M., Johnson, K. L., Hayes, E. A., Nicol, T., & Kraus, N. (2004). Learning impaired children exhibit timing deficits and training-related improvements in auditory cortical responses to speech in noise. *Experimental Brain Research, 157*, 431–441.

Wible, B., Nicol, T., & Kraus, N. (2005). Correlation between brainstem and cortical auditory processes in normal and language-impaired children. *Brain, 128*, 417–423.

Wong, P. C. M., Jin, J. X., Gunasekera, G. M., Abel, R., Lee, E. R., & Dhar, S. (2009). Aging and cortical mechanisms of speech perception in noise. *Neuropsychologia*, *47*, 693–703.

Wunderlich, J. L., Cone-Wesson, B. K., & Shepherd, R. (2006). Maturation of CAEP in infants and children: A review. *Hearing Research*, *212*, 212–223.

Figure legends

Figure 1. A schematic presentation of the stimulus sequences used. In the beginning of the sequences, there were 10 standards, which were excluded from the data analysis. After those, 540 stimuli were presented so that the standard stimulus alternated with the different deviant stimuli. In addition to /ke:/ sequences, similar sequences with standard syllable /pi:/ were presented. Half of the sequences were presented in silent condition and the rest of them with additional cafeteria noise.

Figure 2. The grand average waves for standard stimuli at the ages of 2 and 4 years in the silent and noisy conditions. The developmental changes in the P1 and N2 latencies and the N2 and N4 amplitudes as well as the noise-induced changes in the N4 latencies and P1 and N2 amplitudes were statistically significant. The negativity is plotted upwards.

Figure 3. The anterior-posterior distribution of the P1, N2, and N4 responses at the ages of 2 and 4 years in the silent and noisy conditions. Negativity is plotted upwards. Vertical bars denote 0.95 confidence intervals.

Figure 4. The grand average waves for the standard responses (dashed line), deviant responses for the five deviant types (dotted line), and the deviant-standard difference waves (solid line) at the ages of 2 and 4 years at the representative electrodes in the silent and noisy conditions. The developmental increment and the noise-induced decrement of the MMN amplitudes are statistically significant. In order to detect the peak latency of each response as reliably as possible, the electrode where the maximum response was found was chosen for this purpose as a representative electrode. The time-window for the MMN peak detection is marked with squares, ns. denotes MMN amplitude not statistically differing from zero, and the negativity is plotted upwards.

Figure 5. The anterior-posterior distribution of the MMN averaged over the five deviant types at the ages of 2 and 4 years in the silent condition. The negativity is plotted upwards. Vertical bars denote 0.95 confidence intervals.