Neuroscience Letters 587 (2015) 102–106

Contents lists available at ScienceDirect

Neuroscience Letters

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

Research article

Maturation of mismatch negativity and P3a response across adolescence

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HIGHLIGHTS

- The MMN did not change reliably across adolescence.
- The amplitude and latency of P3a reduced across adolescence.
- The ability to orient towards an unexpected sound continues to develop across adolescence.

ARTICLE INFO

Article history:
Received 27 May 2014
Received in revised form
24 September 2014
Accepted 18 December 2014
Available online 23 December 2014

Keywords:
Adolescence
Maturation
Mismatch negativity
P3a

ABSTRACT

Adolescents experience significant changes in various physiological and psychological domains due to changes in their brain's structure and function. A lot is known about structural changes in the brain across adolescence. However, less research has investigated changes in brain function during this period. In this study, we tracked the maturation of the auditory mismatch negativity (MMN) and P3a brain responses – both posited as neural indices of auditory discrimination – in 90 adolescents aged 10–18 years. We found that P3a mean amplitude and latency decreased significantly across adolescence, but there was no reliable change in the MMN. These results suggest that neural processes associated with passive auditory processing continue to develop well into adolescence.

1. Introduction

Of all the stages in a human's life, adolescence is arguably the most dramatic. Most obvious to the outside world are the physical changes that occur as a person transitions from a child to an adult. Less obvious to the outside world, but obvious to the individual, are marked shifts in various psychological domains such as emotions, personality and cognition [1]. These latter changes result from large changes in an adolescent's brain structure and function [2].

A considerable amount of research has investigated the significant developments in brain structure across adolescence [1–4]. In contrast, the development of the brain's function across adolescence has received far less attention. Event-related potential (ERPs), which reflect the pattern of electric activity produced by brain cells in response to a sensory stimulus or event, is an efficient non-invasive tool to measure various brain functions. To date, ERPs have been used to investigate functional changes in the brain across adolscence in relation to "high-level" cognitive functions such as attention [5,6], working memory and language [7,8]. ERPs have also been used to look at changes in "low-level" cognitive functions such as the encoding of simple speech and non-speech sounds (sound detection and orientation) [9,10]. To our knowledge, only a few studies have used ERPs to measure changes in neuronal functions that fall between these high-level and low-level cognitive processes [11,12]. In this study, we help to fill this gap by using the auditory mismatch negativity (MMN) and P3a ERPs to investigate the development of auditory memory and attention, sound discrimination and novel sound detection across adoloscence.

The auditory MMN and auditory P3a can be measured by subtracting an ERP elicited by a frequently occuring "standard" sound from an ERP elicited by an infrequent "deviant" sound. In adults, the MMN typically presents as a negative response found 100–250 ms after the onset of a sound, while the P3a is a positive response found between 200 and 300 ms. The cognitive processes that the MMN and P3a are supposed to represent are not yet fully understood. It has been proposed that the MMN represents pre-attentive sen-

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http://dx.doi.org/10.1016/j.neulet.2014.12.041
0304-3940/

sory memory [13], auditory discrimination [14], and redirection of focussed attention [14]. Two cortical sources have been propsed for the generation of MMN. One lies in bilateral supratemporal auditory cortices, which is related to pre attentive auditory processing (stimulus specific). The other is located in the frontal cortex either in inferior frontal and middle frontal cortex, which is related to the redirection of the attention during presentation of deviant sounds [13–16].

It has been suggested that the P3a reflects activation of an attentional switching mechanism or an orienting response to either an novel or unexpected (deviant) sound [19]. Since both the MMN and P3a are represented by the difference between a (larger) ERP to a rare deviant sounds and a (smaller) ERP to frequent standard sounds, it seems reasonsable to assume that both reflect auditory memory, discrimination, and attention to some degree. Since P3a is involved in attentional switching mechanisms, it has been suggested that its neural generators lie in the dorsolateral prefrontal cortex, the inferior parietal cortex, and the inferior temporal cortex [17–20].

To our knowledge, only three studies have examined the development of the MMN or P3a across adolescence. All these studies used tones as stimuli in a passive listening paradigm (i.e., participants did not pay overt attention to the stimuli). Bishop et al. [11] measured the mean MMN amplitude and latency in three age groups: children (7–12 years; $N = 30$), adolescents (13–16 years; $N = 23$) and adults (32–56 years; $N = 32$). They reported that MMN amplitude increased and latency decreased from childhood to adulthood. Oades et al. [12] measured both the MMN and P3a in eleven 10–14 year old and eleven 14–17 year old in a three-tone oddball paradigm. They found an increase in MMN amplitude and a decrease in MMN latency across age. Interestingly, they also found an increase in P3a amplitude with age when P3a was measured from deviant and target ERPs, but a decrease in P3a amplitude with age when P3a was measured from difference waveforms (i.e., when ERPs to standard stimuli were subtracted ERPs to deviant stimuli or to target stimuli). Finally, Fuchigami et al. [21] looked at the development of P3a latency between 4–21 years, and reported no change in P3a latency across adolescent participants of their study. Unfortunately, this study did not consider P3a amplitude. Considered together, the outcomes of these few studies suggest that the MMN may increase in amplitude; decrease in latency and P3a amplitude may decrease with age. The changes in P3a latency across adolescence are currently obscure due to limited or contradictory data.

Given our limited knowledge about changes in the MMN and P3a across adolescence, the aim of the current study was to examine the development of both these ERP components in typically developing listeners aged from 10–18 years at both the group level (i.e., compared between age groups) and individual level (compared across individuals with different ages). To this end, we built upon the strengths of the previous studies in four ways. First, like all these studies, we presented tones in a passive listening paradigm to elicit the MMN and P3a, which minimizes confounding effects of overt attention [22]. Second, like Oades et al., but unlike Bishop et al. and Fuchigami et al., we tested both the MMN and P3a in the same participants. Third, like Bishop et al. ($N = 53$) but unlike Fuchigami et al. ($N = 36$) and Oades et al. ($N = 22$), we recruited a relatively large number of adolescents for our sample ($N = 90$). Finally, unlike any of the previous studies, we used fine-grained age groups of just one year (e.g., 10–11 years, 11–12 years) to track the development of the MMN and P3a across adolescence. Based on the existing, albeit limited, evidence we predicted that the MMN would increase in amplitude, would decrease in latency. We further predict that P3a measured from difference waveforms would decrease in amplitude across adolescence. We could not predict changes in P3a latency due to contradictory outcomes from existing studies [12,21]. The present study aims to provide a more accurate description of P3a development across adolescence by grouping large numbers of adolescents into 1-year age groups.

2. Materials and methods

2.1. Participants

A total of 100 individuals (40 females), aged 10–25 years, were divided into nine age groups: 10–11 ($N = 14$), 11–12 ($N = 10$), 12–13 ($N = 14$), 13–14 ($N = 12$), 14–15 ($N = 10$), 15–16 ($N = 11$), 16–17 ($N = 9$), 17–18 ($N = 10$) and 22–25 ($N = 10$; adults). All participants had normal hearing bilaterally (≤15 dB HL) for 500 Hz, 1000 Hz and 2000 Hz pure-tones as assessed using a screening audiometer. Participants were also assessed on the matrices subtest of the Kaufman Brief Intelligence test (KBIT) [23], and had non-verbal IQ scores in at least the average range. The range of the KBIT standard scores was 85–133. Thus, the adolescents had average (i.e., 85–115, $N = 72$) or above-average (i.e., >116, $N = 18$) KBIT scores. Participants reported no history of psychological, physiological, or learning problems as confirmed by their parents.

2.2. Stimuli

The experimental stimuli consisted of four blocks of pure tones. Two blocks used 1000 Hz tones as a frequent 'standard' stimulus ($p = 85$) and 1200 Hz tones as an infrequent 'deviant' stimulus ($p = 15$). In the remaining two blocks the stimuli were reversed, with a 1200 Hz tone as standard stimulus and a 1000 Hz tone as deviant stimulus. The order of presentation of the two blocks was counterbalanced between participants.

Each block contained 333 stimuli that were presented binaurally via headphones at 80 dBSPL. The stimuli were separated by a jittered stimulus-onset asynchrony (SOA) of 0.9–1.1 s to minimize the confounding effect of ERP artifacts related to anticipation of a stimulus and overt attention [22]. The stimuli had a stimulus duration of 175 ms in order to (1) meet the standard guidelines to record MMN [24], and (2) facilitate adequate comparison with previous studies of maturation of MMN and P3a in adolescents [11,12,21].

2.3. Recording

Participants were seated on a comfortable chair and asked to focus their attention on a silent movie of their choice. The scalp of each participant was combed to reduce the time needed to bring the electrode impedances down to <5 kohm [25]. The 30 Ag–AgCl electrodes were positioned in a cap according to the international 10–20 system (Quik-Cap). The vertical and horizontal eye movements were also recorded with left mastoid serving as online reference. The continuous EEG was sampled at 1000 Hz with an online band-pass filter from 0.05 to 100 Hz.

2.4. Data analyses

A standard ocular reduction algorithm [26], which uses a regression procedure that correlates variance in EEG activity with vertical EOG activity, was used to remove the eye blinks from the EEG data. The EEG data were then re-referenced to the average of both the mastoids. The EEG activity was band-pass filtered (0.1 Hz high pass and 30 Hz low pass; 12 dB-per-octave roll-off) with a finite impulse response filter (FIR) and divided into 500 ms epochs with a 100 ms pre-stimulus baseline interval for baseline correction, after which all epochs with a voltage change exceeding ±150 μV were removed. All accepted epochs generated by 1000 Hz standard tones (97.7%; 544–564 epochs) and 1000 Hz deviant tones (95.7%; 89–97 epochs) were averaged together to produce a standard ERP (maximum