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Pitch discrimination without awareness in congenital amusia: Evidence from event-related potentials

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A B S T R A C T

Congenital amusia is a lifelong disorder characterized by a difficulty in perceiving and producing music despite normal intelligence and hearing. Behavioral data have indicated that it originates from a deficit in fine-grained pitch discrimination, and is expressed by the absence of a P3b event-related brain response for pitch differences smaller than a semitone and a bigger N2b–P3b brain response for large pitch differences as compared to controls. However, it is still unclear why the amusic brain overreacts to large pitch changes. Furthermore, another electrophysiological study indicates that the amusic brain can respond to changes in melodies as small as a quarter-tone, without awareness, by exhibiting a normal mismatch negativity (MMN) brain response. Here, we re-examine the event-related N2b–P3b components with the aim to clarify the cause of the larger amplitude observed by Peretz, Brattico, and Tervaniemi (2005), by experimentally matching the number of deviants presented to the controls according to the number of deviants detected by amusics. We also re-examine the MMN component as well as the N1 in an acoustical context to investigate further the pitch discrimination deficit underlying congenital amusia. In two separate conditions, namely ignore and attend, we measured the MMN, the N1, the N2b and the P3b to tones that deviated by an eight of a tone (25 cents) or whole tone (200 cents) from a repeated standard tone. The results show a normal MMN, a seemingly normal N1, a normal P3b for the 200 cents pitch deviance, and no P3b for the small 25 cents pitch differences in amusics. These results indicate that the amusic brain responds to small pitch differences at a pre-attentive level of perception, but is unable to detect consciously those same pitch deviances at a later attentive level. The results are consistent with previous MRI and fMRI studies indicating that the auditory cortex of amusic individuals is functioning normally.

1. Introduction

Humans appear to be born musical, but some individuals are unable to enjoy and produce music despite normal exposure to it. This condition is now known as congenital amusia, a lifelong developmental disorder (Peretz, 2001), that has been estimated to affect about 4% of the population.

Recent studies have demonstrated that congenital amusia can be explained by a deficit in fine-grained pitch discrimination. In a study particularly relevant to the present one, Hyde and Peretz (2004) presented an oddball paradigm composed of sequences of five 100 ms long isochronous identical tones with an inter-stimulus interval of 250 ms, in which the fourth tone differed or not from the other tones of the sequence by one of five pitch differences, ranging from 25 to 300 cents (100 cents = 1 semitone). Amusics and their matched controls had to indicate whether they detected or not a change. The results showed that typically, amusics cannot detect pitch variations smaller than a semitone despite normal hearing. In further brain imaging studies, the music deficit underlying congenital amusia has been related to brain anomalies in white and grey matter of the auditory cortex, the inferior frontal cortex, as well as in the arcuate fasciculus (Hyde, Zatorre, Griffiths, Lerch, & Peretz, 2006; Hyde et al., 2007; Loui, Alsop, & Schlaug, 2009), and is expressed by an abnormal connectivity between the auditory cortex and the inferior frontal cortex (Hyde, Zatorre, & Peretz, 2011). Abnormal brain electrical activity in amusics has also been observed by Peretz et al. (2005), who presented the same stimuli as used by Hyde and Peretz (2004) while recording brain evoked potentials. They observed the absence of a normal P3b event-related potential (ERP) component to small pitch changes as well as a larger N2b and P3b component in response to pitch differences that were greater than a semitone, which they interpreted as an indication that the brain anomaly underlying congenital amusia may lie outside the auditory cortex along the auditory pathway. In another study, Peretz, Brattico, Jarvenpaa, and Tervaniemi (2009) presented amusics and controls with melodies in

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which they had to determine whether it contained a pitch anomaly. The results showed that amusics exhibited a normal mismatch negativity (MMN) ERP component but no normal P3b in response to the melodic incongruities. These results suggested that the amusic brain can detect small pitch anomalies without awareness.

The MMN, N2b, and P3b ERP components reflect pitch-dependent variations in brain activity at various levels of processing along the auditory pathways. Namely, the auditory MMN is taken to reflect the preconscious processing of changes, for example in frequency or intensity, in a continuous stream of identical sounds, and is associated with brain activity in the auditory cortex which may involve frontal regions as well (Naatanen & Winkler, 1999). The auditory N2b reflects deviance detection and is only observed when attention is dedicated to the targets (Folstein & Van Petten, 2008). The N2b is ascribed to activity of the supratemporal auditory cortex (e.g., Bruneau & Gomot, 1998). The P3b is also associated with the conscious discrimination of task-relevant targets in a repeating sequence of distractors and is typically associated with multiple generators over the parietal and cingulate cortex as well as in prefrontal regions (e.g., Linden, 2005).

Although ERP and brain imaging data appear to converge in attributing the neural anomaly underlying congenital amusia to an impoverished connectivity between the auditory cortex and the inferior frontal cortex, it is still unclear why the amusic brain overreacted to large, well-detected, pitch changes in the study of Peretz et al. (2005). One could argue that the recruitment of larger populations of neurons may be needed in amusics' brains, resulting in a greater N2b–P3b response. On the other hand, the N2b and P3b are ERP components known to vary in amplitude as a function of the probability of occurrence of the pitch changes (Donchin, 1981; Donchin, Miller, & Farwell, 1986; Naatanen & Picton, 1986), namely by showing greater amplitude as the pitch change frequency of occurrence decreases. In the Peretz et al.'s (2005) study, amusic subjects did not perceive pitch changes as frequently as controls, and this difference changed the relative probability of perceived pitch changes. The larger N2b–P3b response observed in amusics to large pitch changes, relative to controls, may thus have reflected the increased rarity of perceived changes in the context of the experimental session. If so, the larger N2b–P3b response observed in amusics should not be interpreted to reflect physiological differences to large pitch changes, *per se*, relative to controls, but rather as an indirect reflection of their inability to perceive the small pitch changes.

The goal of this study was to evaluate brain electrical responses to pitch changes in a single experiment by measuring preattentive and attentive brain responses for small pitch changes in amusics and controls. Furthermore, we sought to clarify the cause of the greater N2b–P3b previously observed in amusics in response to large pitch changes. This goal was achieved by controlling the frequency of occurrence of perceived pitch changes using a matching procedure that equated this frequency across amusics and controls. We predicted that amusics would show a normal MMN and a normal P3b for the large pitch changes, but an abnormal P3b to small pitch differences.

2. Method

2.1. Participants

Ten amusic participants and ten controls matched for age, gender, education, and musical experience participated to the study. The participants were considered amusic or not on the basis of their scores on the Montreal Battery of Evaluation of Amusia (MBEA; Peretz, Champod, & Hyde, 2003). The MBEA is composed of 6 tests (180 trials) that evaluate various music processing components. The participants were considered amusic if their global MBEA score fell two standard deviations or more below the mean global score of the control group. Both groups had normal hearing relative to their age as determined by an audiometric test (around 40 dB HL at 1000 Hz), and no participant had previous neurological or psychiatric history. The characteristics of the participants are presented in Table 1. The protocol was vetted by an institutional committee at Université de Montréal.

2.2. Materials and procedure

2.2.1. Auditory stimulation

The auditory stimuli were piano notes created with specialized software (Sibelius 5.0, exported as wave files using KontaktPlayer 2, from Native Instrument, running the Sibelius Sounds Essentials samples). The wave files had a duration of 100 ms (rise and fall of 10 ms) and were normalized in energy with Adobe Audition V3.0 at 44.1 kHz in 16-bit format. The sounds were used in two conditions in which a frequent standard tone was played at a pitch level of C6 (1047 Hz). The rare deviant tones were lower or higher in pitch than the standard tone by 25 cents (1032 or 1062 Hz) or 200 cents (933 or 1175 Hz; 100 cents corresponds to one semitone). In the ignore condition, the participants were instructed to ignore the auditory stimulation and to watch a self-selected movie presented with subtitles with the sound track turned off. The sequence of tones contained 2736 standard tones (probability of occurrence = .9) and 76 tones of each of the four pitch deviances (probability of occurrence = .025) for a total of 3040 sounds presented at a fixed inter-tones interval of 400 ms (SOA = 500 ms) in two blocks, allowing for a short pause in-between, and lasting 25 min in total. The presentation of the sounds was pseudo-randomized so that every deviant tone was preceded by at least four standard tones. In this experiment, we did not use deviants as standards and vice versa because we used five different deviants; replacing standards by each deviant would have been too long for the participants, who are relatively old.

The attend condition was always presented after the ignore condition, to avoid conscious switches of attention to pitch differences. Indeed, the attend condition made participants aware of the nature of the auditory stimulation and of the size of the pitch changes, in particular. In the attend condition, participants were presented with sequences of five tones, using the same sounds as in the ignore condition, with an inter-tone interval of 250 ms (SOA of 350 ms)[1] and a pause of two seconds between each sequence. Standard sequences were composed of five standard tones. Deviant sequences comprised 3 standards followed by a deviant and a standard. The pitch of the deviant fourth tone was displaced upward or downward by 25 or 200 cents. The standard and deviant sequences were randomly mixed. The participants were presented with a total of 240 sequences. For the amusics, there were 120 standard sequences and 30 of each of the four deviant sequences. For the control subjects, the number of deviant sequences was matched to the number of deviants correctly detected by the amusics. Because

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[1] The experimental paradigms differed slightly in each condition because we wanted to maximize the quality of the brain response recordings and allow comparison with previous data. In the attend condition, the inter-tone interval was chosen to match the one used in the previous study by Peretz et al. (2005) because our main goal was to clarify the origins of the greater N2 and P3 ERP components observed in amusics in responses to large pitch changes in 2005. Unfortunately, these parameters are not well suited to study the MMN. In fact, the MMN is best recorded with longer inter-tone intervals (around 400 ms or more; Kujala, Tervaniemi, & Schröger, 2007) and more sounds before a deviant are needed in order to observe a reliable MMN (Duncan et al., 2009). Nevertheless, these adaptations to the paradigm are not expected to influence the result, as suggested by the similar results of a follow up experiment in which the inter-tone interval was matched between the attend and the ignore conditions (Mignault Goulet, Moreau, Robitaille, & Peretz, 2012).

Table 1
Characteristics of the participants, global percentage score on the Montreal Battery of Evaluation of Amusia (MBEA) with standard deviations calculated relative to the controls' mean score in parentheses.

	Age (yrs)	Gender	Education (yrs)	Musical experience (yrs)	MBEA (%)
Amusics					
A-01	65	M	19	1	51.33 (−8.7)
A-02	66	F	14	2	68 (−4.8)
A-03*	66	F	21	4	70 (−4.4)
A-04	68	F	15	2	68 (−4.8)
A-05*	64	M	14	1	60 (−6.7)
A-06	62	F	20	1	72.67 (−3.8)
A-07*	67	M	19	1	55.33 (−7.8)
A-08	58	F	19	3	59.33 (−6.9)
A-09	59	M	19	1	63 (−6)
A-10	72	M	15	1	66.67 (−5.1)
M	64.7	–	17.5	1.7	63.4 (−5.9)
Controls					
C-01	68	M	16	1	86.67 (−0.5)
C-02	64	F	18	2	94 (1.2)
C-03*	64	F	19	2	90.67 (0.4)
C-04	68	F	15	4	90 (0.3)
C-05*	65	M	16	3	93 (1)
C-06	63	F	15	1	93 (1)
C-07*	67	M	13	1	81 (−1.8)
C-08	59	F	16	3	88.67 (0.0)
C-09	65	M	12	1	87.78 (−0.2)
C-10	72	M	16	2	83 (−1.3)
M	65.5	5F	15.6	2.1	88.8 (4.3)
t-Tests	n.s.	–	n.s.	n.s.	$p < .001$

* Participants who were excluded in the analyses of the P3b. Significance levels on corresponding t-tests; 'n.s.' refers to a non-significant difference ($p > .05$).

controls do not always detect 100% of the deviant sounds presented (i.e., they miss, on average, 12% of the 25 cents changes and 2% of the 200 cents changes), two deviant sequences of 25 cents and one sequence of 200 cents pitch deviant were systematically added to the matched number of sequences presented to the controls (see Table 2). The total number of sequences presented (240) remained unchanged. The task lasted 19 min for both controls and amusics. Each control was yoked to a particular amusic. For example, if amusic #1 correctly detected all 200 cents deviants (60 trials) and 19 of the 25 cents deviants (out of 60 trials), control #1 was presented with 60 +1 200 cents sequence, 19 +2 25 cents sequences and 158 standard sequences, thus matching the frequency of detected changes for the yoked amusic.

Table 2
Average number of sounds presented and detected by seven amusics and their seven yoked controls in the Attend condition for the standard tone, the 25 and the 200 cents pitch differences. False alarms correspond to a "yes, there was a change" for standard sequences (which contain no change). Standard deviations are in parentheses.

	Amusics ($N = 7$)	Controls ($N = 7$)
25 Cents upward		
Presented	30 (0.0)	10.6 (8.4)
Detected	9.1 (7.2)	9.9 (8.3)
25 Cents downward		
Presented	30 (0.0)	7.1 (4.1)
Detected	6.7 (4.7)	5.7 (3.3)
200 cents upward		
Presented	30 (0.0)	30 (0.0)
Detected	29.1 (2.3)	29.4 (1.1)
200 Cents downward		
Presented	30 (0.0)	30 (0.0)
Detected	28.6 (3.4)	29.4 (0.8)
Standard		
Presented*	120 (0.0)	154.6 (24.4)
Detected	114.1 (10.8)	147.6 (24.0)
False alarms	5.57 (10.8)	7.0 (8.6)
Total change detection	79.4 (13.5)	81.3 (12.0)

* The different rate of presentation for the standard sequences between amusics and controls reflects the matching procedure to maintain *probabilities* of occurrence of deviants equivalent.

The participants were informed of the position in the sequence where a change could occur. They were instructed to press a button when they perceived a different pitch in the fourth tone and to press a different button when they perceived no difference. There was no feedback. Feedback was only given during the practice trials, which comprised one standard sequence and each of the four deviant sequences presented in a random order. Participants could repeat the practice trials as often as they wished, but they did not exceed two blocks of practice trials. Participants were asked to blink between the sequences, to focus on their hands, and to remain relaxed. Throughout the testing session, the sounds were presented binaurally through headphones and the intensity level was adjusted to each individual's hearing level for an average intensity level of presentation of 70 dB SPL. The whole experimental session, including the ignore and the attend condition, lasted approximately 40 min, following the electrode installation which lasted about 30 min.

2.2.2. EEG recording parameters and analysis

The subjects were seated in an electrically shielded and sound-attenuated chamber. The electroencephalogram (EEG) was recorded (bandpass, 0.05–70 Hz; sampling rate, 256 Hz; impedance <5 KΩ) via a Neuroscan amplifier (Neuroscan SynAmps2, Compumedics, El Paso, TX) from 66 electrodes at the standard 10–10 scalp sites referenced online to the tip of the nose. Bipolar electrode pairs monitored horizontal and vertical electrooculograms (EOG). Offline, the EEG data were corrected for eye movement (Semlitsch, Anderer, Schuster, & Presslich, 1986), and the signal was filtered (bandpass, 0.05–30 Hz, 24 dB/octave). An artifact rejection was conducted on all EEG channels except for the EOG (criteria of ±100 μV; average rejection rate of 6.32% and 4.76% in amusics and controls, respectively; artifact rejection was similar across conditions). The data were then divided into epochs of 600 ms including a 100 ms pre-stimulus interval for baseline correction and re-referenced to the averaged mastoids. In both conditions (ignore and attend), separate averages were computed for the standard tones and for pitch deviances (25 cents and 200 cents). Type 1 errors associated with inhomogeneity of variance were controlled by decreasing the degrees of freedom using the Greenhouse–Geisser epsilon. The original degrees of freedom are reported for all statistical analyses.

3. Results

The results are first presented in the ignore condition, in which the MMN component was analyzed, and then in the attend condition, in which the auditory P3b component was analyzed. The auditory N1 component recorded in both the ignore and the attend conditions will be presented in the last section.

3.1. Ignore condition

Before subtracting the standard ERP waveform to those of the deviants in order to compute the MMN, the standard waveforms were compared in amusics and controls to ensure that both groups shared similar subtraction baselines. An independent *t*-test run on the measures of amplitude of the standard waveforms, calculated as the mean amplitude over a window ranging from 140 ms to 300 ms and corresponding to the detection window of the MMN, yielded no significant difference ($t(18) = .84$, $p > .41$). Therefore, the analyses were performed on the subtracted waveforms. The MMN amplitudes were quantified by computing the mean amplitude over a 40 ms window centered at the grand average peak latency detected within a time window of 140–300 ms, and the latency was measured as the time point of peak amplitude. The results are reported for electrode Fz where the MMN amplitude was the largest (Fig. 1).

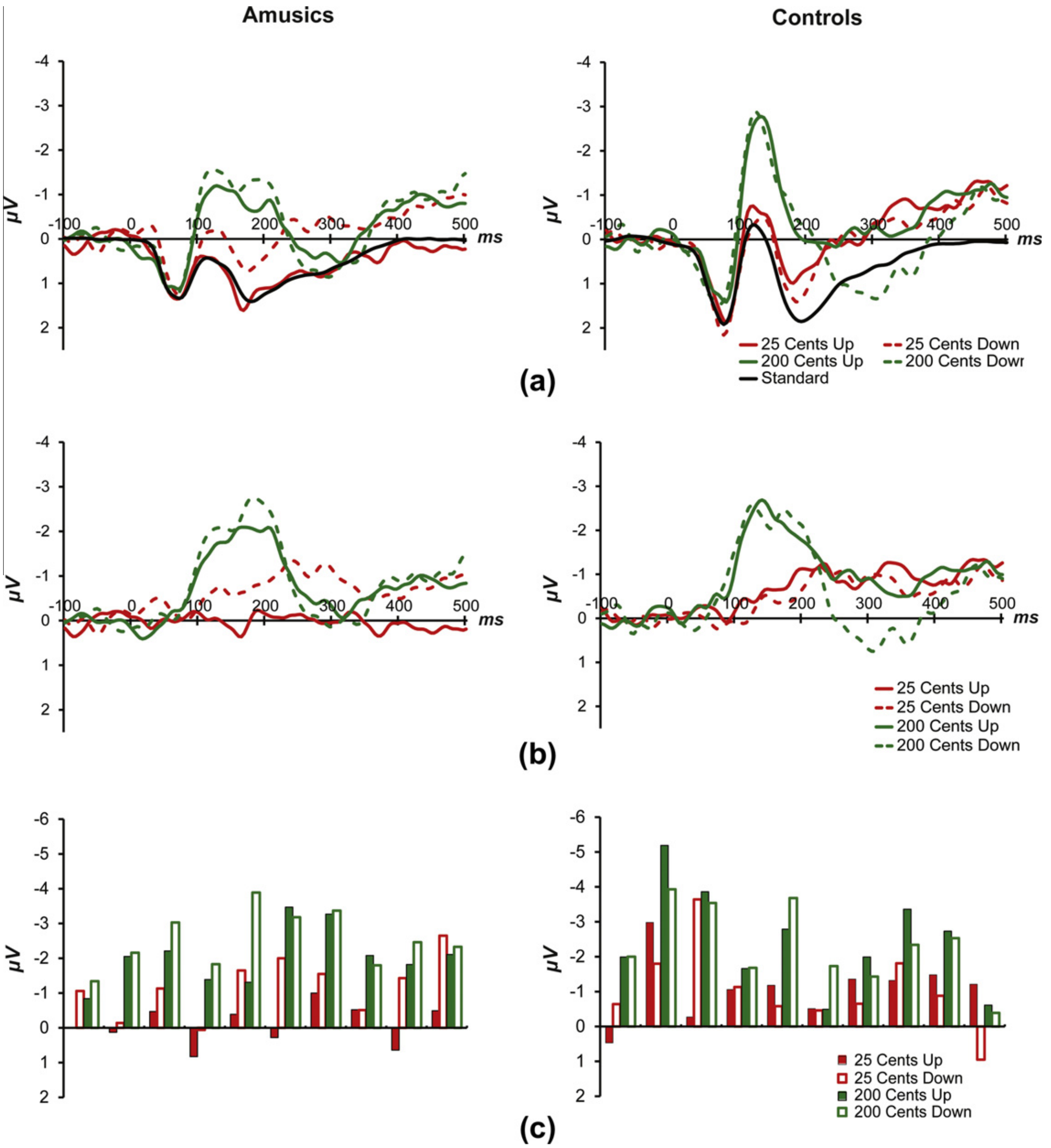

Fig. 1. (a) Grand average ERP waves obtained for the standard sound and the 25 and 200 cents pitch changes (upward and downward) for the amusic and control subjects at Fz. (b) MMN difference waves (deviant minus standard) for de 25 cents and 200 cents pitch changes (upward and downward) for amusics and controls at Fz. (c) Individual MMN amplitudes for the 25 and 200 cents pitch differences (upward and downward) for amusics and controls. Dashed lines indicate a downward pitch change, solid lines an upward pitch change and the standard in their respective colors (green for 200 cents; red for 25 cents and black for the standard). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

As can be seen in Fig. 1, amusics and controls obtained similar MMNs. Except for the upward 25 cents pitch deviance which was not significantly different from zero in amusics ($t(9) = .54$, $p > .6$), all the other MMNs differed from zero. The ANOVA yielded a significant interaction of pitch direction by group with $F(1,18) = 7.13$, $p < .02$. As can be seen in Fig. 1c, eight amusics out of ten have a MMN for the 25 cents downward pitch difference and five amusics out of ten also show a MMN for the upward 25 cents deviance, showing that the majority of the amusics do respond to the 25 cents pitch difference and preferentially to the downward 25 cents pitch difference. As expected, a main effect of the size of the pitch difference (25 vs. 200 cents) was found, with $F(1,18) = 97.25$, $p < .0001$, confirming that the MMN for the 25 cents pitch deviance was smaller in amplitude than the MMN for the 200 cents deviance. The ANOVA on peak-latency measures only revealed an effect of the size of the pitch difference, $F(1,18) = 73.06$, $p < .0001$, reflecting the later peak for the 25 cents MMN than for the 200 cents MMN[2].

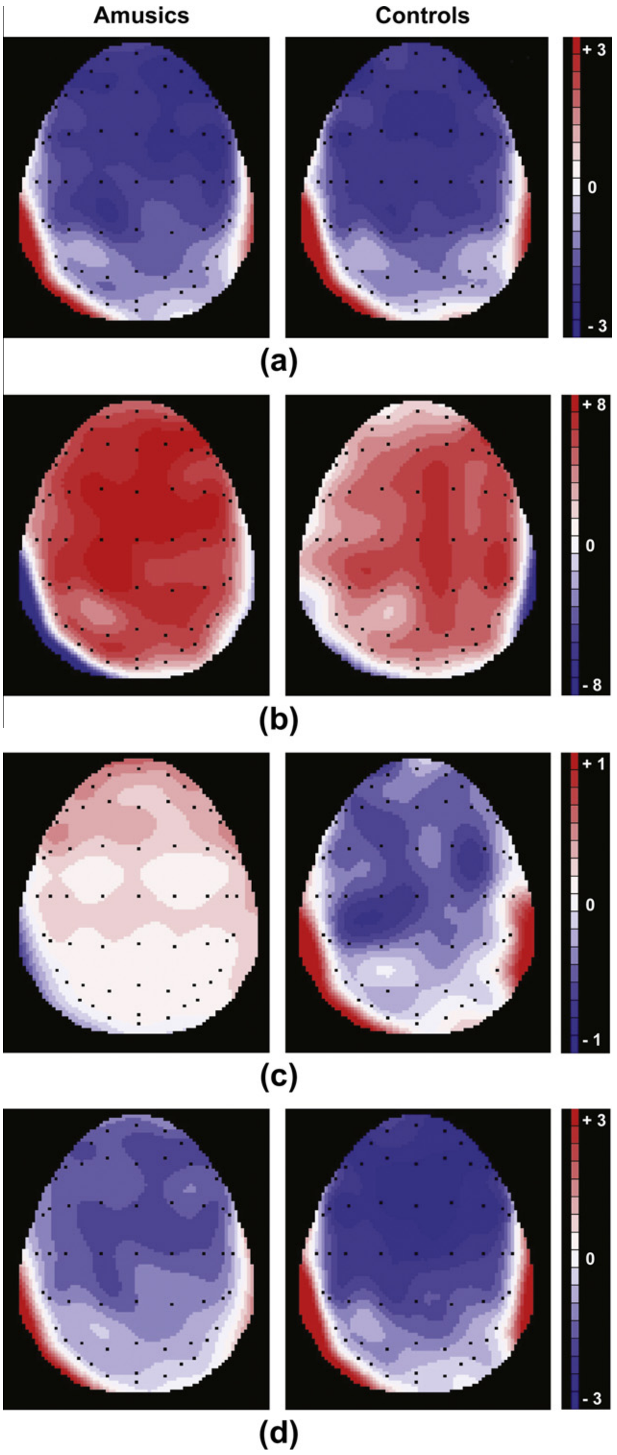

Fig. 2. (a) Scalp voltage maps for the 200 pitch difference in MMN; (b) for the P3b; (c) for the N1 in the ignore condition; (d) for the N1 in the attend condition.

The MMN scalp topographies are depicted in Fig. 2a and were analyzed for the 200 cents pitch deviance.[3] Originally, the EEG was recorded using 66 electrodes, but in the topographical analyses, we chose the eight electrodes of the midline for the anteriority effect, and four fronto-central electrodes, where the MMN amplitude was the largest, in each hemisphere for the laterality effect. Both groups showed a clear anteriority effect by obtaining larger MMN towards the pooled frontal electrodes (FPz, Fz, FCz, and Cz; $M = -2.21$) compared to the pooled posterior electrodes (CPz, Pz, Poz, and Oz; $M = -1.13$) for both pitch directions, $F(1,18) = 74.97$, $p < .0001$. No laterality effect was observed between the pooled left hemisphere electrodes (F4, F6, FC4, and FC6) and the pooled right hemisphere electrodes (F3, F5, FC3, and FC5). In the results presented here, we used a mean mastoid reference in order to capture both sides of the electrical fields produced by the MMN generators at the sites of analysis (e.g., Fz). However, separate analyses that maintained the nose reference revealed a clear inversion of the MMN between vertex electrodes (e.g., Fz) and the mastoids.

3.2. Attend condition

Amusics detected few 25 cents deviant tones (Table 2). Thus, the amplitude of the P3b, calculated as an average over a window ranging from 300 to 500 ms and computed on the correct responses, was variable. To reduce this variability, we identified outliers, before running the analysis of variance, according to the following procedure. The amplitude of the P3b of each participant was compared to the group mean amplitude. The participant whose P3b amplitude was either the biggest or the smallest and thus was the furthest away from the group mean in microvolts was temporarily excluded and the mean and standard deviation were re-calculated for the remaining participants. If the temporarily excluded participant differed from the new mean by more than 3.5 standard deviations, it was considered an outlier and permanently excluded. The procedure was run iteratively as long as outliers were found. The outlier rejection criterion varies as a function of group size, with standard deviations needed to reject a participant increasing as the number of remaining participants decreases (see Van Selst & Jolicoeur, 1994, for a computational simulation). This procedure led to the rejection of two amusics (A-03 and A-07 in Table 1) and one control (C-05). The behavioral data of these discarded participants were not atypical since their performance did not exceed two standard deviations from the mean of the group and thus could not explain the atypical brain waves observed here. Because these two amusics and the control participants were experimentally matched for the frequency of detection of the 25 cents deviants to other participants, we only analyzed the behavioral data and the P3b obtained in the data of seven amusics and seven matched controls.

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[2] The MMN obtained in the 14 participants (7 amusics and 7 controls) considered for the P3b analyses were similar to those obtained with the entire sample. The ANOVA yielded a significant interaction of pitch direction by group, with $F(1,12) = 6.33$, $p < .03$. A main effect of the size of the pitch difference (25 vs. 100 cents) was also found, with $F(1,12) = 62.62$, $p < .0001$. All other effects were not significant, all $ps > .07$.

[3] The MMN scalp topographies were analyzed in both groups for the 200 cents pitch deviance only because the MMN obtained for the 25 cents condition was not present in all subjects.

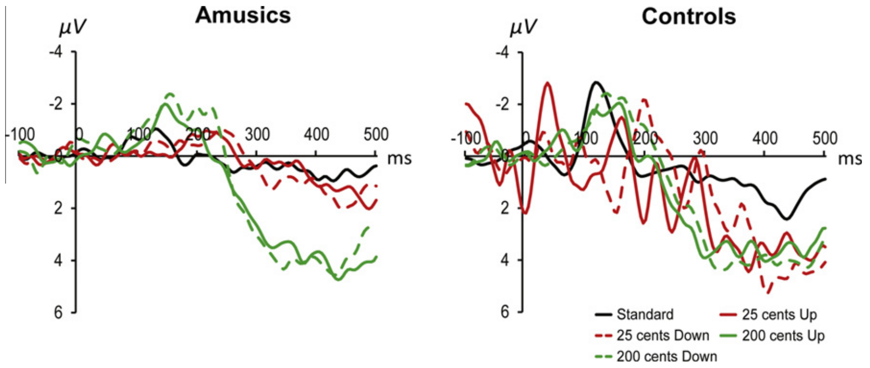

Fig. 3. Grand average ERP waves obtained in the pitch change detection task in the attend condition for the standard tone as well as the 25 and 200 cents pitch differences (upward and downward) in the amusics and the controls at Cz.

3.2.1. Behavioral results

The behavioral results confirmed that the seven amusics had lower detection accuracy for the 25 cents pitch changes (average percentage of correct response = 26.4%), compared to their yoked controls (91.1%), $t(12) = 8.56$, $p < .0001$. In contrast, the 200 cents pitch deviance was detected with equal accuracy, 96.2% and 97.9% for the amusics and controls, respectively, $t(12) = .46$, $p > .65$. This pattern was also reflected in analyses of hits minus false alarms, which produced an interaction between the size of the pitch difference (25 vs. 100 cents) and group in an ANOVA, $F(1,12) = 53.14$, $p < .0001$. The results did not vary as a function of pitch direction, $F(1,12) = 2.52$, $p > .13$.

3.2.2. P3b

The P3b data obtained in the attend condition were computed on the correct responses and reported at the electrode Cz where the amplitudes were the largest (Fig. 3). The following analyses will only focus on the P3b component because no N2b ERP component was observed in amusics and controls. In controls, the P3b amplitude obtained for upward and downward pitch changes did not vary (all $F < 1$) nor did it vary for the 200 cents in amusics ($p > .95$). Therefore, upward and downward pitch differences were averaged together in order to generate a single P3b waveform for the 25 cents and 200 cents pitch deviations.

As can be seen in Fig. 3, there was no P3b for the 25 cents pitch changes in amusics, as revealed by a non-significant *t*-test against zero conducted on the measures of amplitude, $t(6) = .52$, $p > .62$, while the P3b significantly differed from zero in controls with $t(6) = 3.16$, $p < .05$. The ANOVA run on the measures of amplitude revealed a significant interaction between size of the pitch difference and group, $F(2,21) = 4.17$, $p < .03$. The analysis of the simple effects with unilateral independent *t*-tests confirmed that the amusics differed from controls for the 25 cents pitch changes, with $t(12) = 2.79$, $p < .02$, but not for the 200 cents pitch difference, $t(12) = .16$, $p > .44$, nor for the standard tone, $t(12) = .63$ $p > .27$. The mean peak latency of the P3b for the 200 cents changes did not differ between groups, $t(12) = .66$, $p > .52$. The P3b amplitude results have been analyzed in relation to the behavioral data but we could not find any significant correlation (all $ps > .05$).

The scalp topographies of the P3b for the 200 cents pitch deviance are depicted in Fig. 2b and revealed no significant lateralization effect between the pooled left hemisphere electrodes (C3, C5, CP3 and CP5) and the pooled right hemisphere electrodes (C4, C6, CP4, and CP6; $F < 1$), nor any significant anteriority effect between the pooled frontal electrodes (FPz, Fz, FCz, and Cz) and the pooled posterior electrodes (CPz, Pz, POz, and Oz; $F < 1$).

3.3. N1

The analysis of N1, for the entire sample ($N = 20$; Fig. 4),[4] was based on the first 1080 standard tones presented during the ignore condition to equate the number of standard tones analyzed in the attend condition for comparison. To avoid contamination of the ERP wave by the deviant tones, the standard tones following a deviant tone were rejected from the analysis. The N1 was computed at Cz, where it was the largest, as the mean amplitude over a 40 ms window centered at the grand average peak, detected between 90 and 130 ms after tone onset, and the latency was measured as the time point of peak amplitude. As shown in Fig. 4, amusics seem to show a smaller N1 than controls but this difference was not statistically significant, $t(18) = 1.27$, $p > .22$; nor on latencies, $t(18) = 1.48$, $p > .16$.

In order to examine the N1 attenuation over time, the data were divided in three parts by dividing the data in three blocks containing the same number of sounds. The ANOVA yielded no significant interaction ($p > .72$), nor any significant main effect of group ($p > .20$), but a significant main effect of block, with $F(2,36) = 5.75$, $p > .007$, indicating that in both amusics and controls, N1 amplitude decreased over time, with a significantly smaller amplitude in the second ($p < .01$) and the third blocks ($p < .05$) as compared to the first block.

In the attend condition, the N1 was also analyzed for the entire sample ($N = 20$; Fig. 4) and revealed no difference between groups on amplitude, $t(18) = 1.43$, $p > .17$), or latency, $t(18) = .94$, $p > .36$). As observed in the ignore condition, the attenuation of N1 over the blocks was also found here, $F(2,36) = 8.47$, $p < .001$. The interaction between block and Group was not significant ($F = (2,36) = .02$; $p > .97$).

As seen in Fig. 4, the N1 appeared to be larger in the attend than in the ignore condition. An ANOVA was run on the amplitude of the N1 with condition (attend and ignore) as a within-subject factor and group (amusics and controls) as a between-subjects factor. The analysis yielded no significant interaction ($p > .69$), nor any significant effect of group ($p > .16$), but a significant main effect of condition was found with $F(1,18) = 95.00$, $p < .0001$, revealing that the N1 amplitude was larger in the attend condition ($M = -1.86$; $SD = 1.21$) than in the ignore condition ($M = -.04$; $SD = 1.08$).

The N1 scalp topographies for the ignore and the attend conditions are depicted in Fig. 2c and d respectively. They appeared central with no significant laterality effect in the attend ($F < 1$) or

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[4] As for the MMN, the N1 of the 6 excluded participants in the P3b analysis was normal and thus was kept in the analyses.

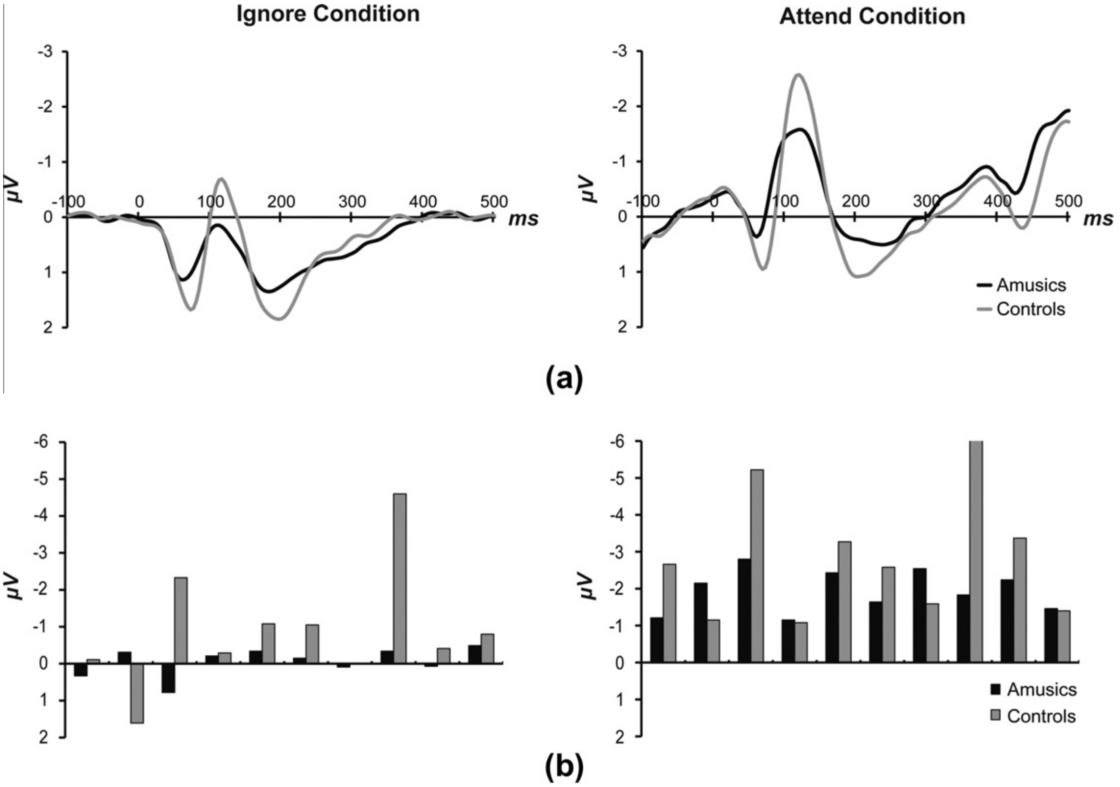

Fig. 4. Grand average ERP waves obtained for the first 1080 standard tones in the ignore condition (left panel) and the attend condition (right panel) for the amusics and the controls at Cz.

ignore condition ($F < 1$) in both groups, but it was slightly more anterior in the attend condition, $F(1,18) = 12.21$, $p < .003$.

4. Discussion and conclusion

As found in earlier studies (Hyde & Peretz, 2004; Peretz et al., 2005), the present study shows that congenital amusia is associated to a deficit in discrimination of pitch differences smaller than a semitone. Importantly, this deficit is visible not only in behavior but also in a specific pattern of brain electrical activity that brings a better understanding of the neurodynamic functioning of amusics' pitch change perception. When a pitch deviance occurs in a stream of identical isochronous sounds, the amusic brain seems to react appropriately at early unconscious levels of processing, by eliciting a normal MMN. This finding of perception without awareness falls in line with a previous study in which amusics exhibit a normal MMN in a melodic context (Peretz et al., 2009). Normal perception without awareness is also consistent with the finding that amusics can implicitly learn statistical regularities in tonal sequences (Omigie & Stewart, 2011 but see Peretz, Saffran, Schön, & Gosselin, 2012 for negative results) and implicitly process subtle differences in harmonic structure (Tillman et al., 2012). However, as the information travels along the auditory pathways, crucial neuronal activity appears to be lost so that small pitch changes do not reach the higher conscious levels of processing, as suggested here by the absence of the P3b for small pitch deviances. This pitch perception deficit cannot be explained by a lack of attentional resources or a general unresponsiveness to pitch variations because pitch changes greater than a semitone are correctly detected and generate a normal P3b brain response in amusics.

More importantly, the present study shows that the larger P3b response observed by Peretz et al. (2005) in amusics as compared to normal controls most likely results from an inequality in the probabilities of *perceived* pitch changes across groups. Here, we matched the frequency of *perceived* events between the amusics and the controls, by taking into account the lower probability of detection of small pitch changes by amusics. However, the matching procedure may have disadvantaged the controls not only because there was less room for showing "unconscious pitch processing effects" in the attend condition but also because there were very few trials that contained a 25 cents pitch change. Nevertheless, in these difficult conditions, controls showed a reliable P3b. Thus, when frequency of detection is taken into account and controlled for, the P3b response to large pitch changes no longer differs between amusics and controls. One could argue that the data in the attend condition may have been noisier and more variable because there were less standard and deviant sounds as compared to the ignore condition. Nevertheless, a reliable P3b was obtained for the 25 cents deviants in controls, suggesting enough sensitivity of the paradigm. Yet, amusics who *perceived* as many 25 cents changes as controls did not show any evidence of a P3b. It remains to be tested whether amusics with repeated exposure and training may detect fine-grained pitch differences better and exhibit a near-normal P3b. This should be the goal of further testing, especially in children when the brain is more plastic (Mignault Goulet, Moreau, Robitaille, & Peretz, 2012).

In conclusion, the present study brought light on the mechanisms of perception underlying the fine-grained pitch deficit at the origin of congenital amusia by showing that small pitch changes have an effect on brain activity at early unconscious stages of processing, but do not reach higher levels of conscious processing. To the extent that changes in the amplitude of brain responses could be considered as evidence that stimulus differences were registered, and in some sense perceived, the present results may constitute another example of perception without awareness of pitch deviations by the amusic brain (see Loui, Guenther, Mathys, & Schlaug, 2008; Peretz et al., 2009, for similar findings).

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