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RESEARCH REPORT

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Adaptation patterns and their associations with mismatch negativity: An electroencephalogram (EEG) study with controlled expectations

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Abstract

Adaptation refers to the decreased neural response that occurs after repeated exposure to a stimulus. While many electroencephalogram (EEG) studies have investigated adaptation by using either single or multiple repetitions, the adaptation patterns under controlled expectations manifested in the two main auditory components, N1 and P2, are still largely unknown. Additionally, although multiple repetitions are commonly used in mismatch negativity (MMN) experiments, it is unclear how adaptation at different time windows contributes to this phenomenon. In this study, we conducted an EEG experiment with 37 healthy adults using a random stimulus arrangement and extended tone sequences to control expectations. We tracked the amplitudes of the N1 and P2 components across the first 10 tones to examine adaptation patterns. Our findings revealed an L-shaped adaptation pattern characterised by a significant decrease in N1 amplitude after the first repetition (N1 initial adaptation), followed by a continuous, linear increase in P2 amplitude after the first repetition (P2 subsequent adaptation), possibly indicating model adjustment. Regression analysis demonstrated that the peak amplitudes of both the N1 initial adaptation and the P2 subsequent adaptation significantly accounted for variance in MMN amplitude. These results suggest distinct adaptation patterns for multiple repetitions across different components and indicate that the MMN reflects a combination of two processes: the initial adaptation in the N1 and a continuous model adjustment effect in the P2. Understanding these processes separately could have implications for models of cognitive processing and clinical disorders.

Abbreviations: A1, primary auditory cortex; ANOVA, analysis of variance; DV, dependent variable; DCM, dynamic causal modelling; EEG, electroencephalogram; ERP, event-related potential; GFP, global field power; ICA, independent component analysis; IV, independent variable; ISI, inter-stimulus interval; MMN, mismatch negativity; RMS, root mean square; RP, repetition positivity; SEM, standard error of the means; SOA, stimulus-onset asynchrony; SPL, sound pressure level; TANOVA, topographic analysis of variance.

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KEYWORDS
adaptation, electroencephalogram (EEG), expectation, mismatch negativity (MMN), repetition positivity (RP)

1 | INTRODUCTION

1.1 | Adaptation

Repeated presentation of the same stimulus typically leads to reduced neural activation, a phenomenon known by various terms such as neural adaptation (Grill-Spector et al., 2006; Ringo, 1996), repetition suppression (Desimone, 1996; Grill-Spector et al., 2006), sensory gating (Boutros & Belger, 1999; Freedman et al., 1987), habituation (Bourbon et al., 1987; Loveless, 1983; cf. Barry et al., 1992) and refractoriness (Berry & Meister, 1998; Budd et al., 1998; Rosburg & Mager, 2021), among others. In the present study, we refer to this phenomenon as adaptation. Adaptation occurs across various stimuli in both visual and auditory domains, such as faces (see Schweinberger & Neumann, 2016 for a review), symbols (e.g. Soltész & Szűcs, 2014) and tones (e.g. Todorovic & de Lange, 2012).

Adaptation can be measured by a decrease in the amplitude of the event-related potential (ERP) components recorded via electroencephalography (EEG) from the scalp. In the auditory modality, ERP components, including N1 and P2, are typically measured at fronto-central electrodes. The N1 component is characterised by a negative deflection occurring roughly between 60 and 160 ms (Woods, 1995), whereas the P2 component is a positive deflection peaking around 150–250 ms (Crowley & Colrain, 2004). These components are distributed over fronto-central areas and exhibit a polarity reversal over inferior posterior electrodes when measured against an average reference (Crowley & Colrain, 2004; Fogarty et al., 2020; Näätänen & Picton, 1987; Woods, 1995).

The auditory N1 and P2 adaptations, characterised by a decrement in amplitude, were found in a vast number of studies using pairs of tones or long sequences of tones (e.g. N1: Bourbon et al., 1987; Boutros et al., 1999; Budd et al., 1998; Lagemann et al., 2012; Näätänen & Picton, 1987; Rosburg, 2004; Rosburg & Mager, 2021; Rosburg et al., 2006; N1 and P2: Hari et al., 1982; Herrmann et al., 2016; Peter et al., 2019; Polich, 1986; Rosburg et al., 2022; Rosburg et al., 2010; Sambeth et al., 2004). Regarding the adaptation pattern, most studies have observed an initial N1 decrease, which stabilises after the second or third sound in a stimulus sequence, with no further decrease for subsequent sounds (e.g. Barry et al., 1992; Bourbon et al., 1987; Boutros et al., 1999; Budd et al., 1998; Lagemann et al., 2012; Rosburg, 2004; c.f. Öhman & Lader, 1972). An initial amplitude decrease has also been observed in the P2 in some studies (Rosburg et al., 2010; Rosburg & Sörös, 2016).

1.2 | Adaptation and MMN

In addition to adaptation, another key aspect of the present study is the mismatch negativity (MMN). First described by Näätänen et al. (1978), the MMN typically occurs when a deviant stimulus (hereafter referred to as *deviant*) with different properties (e.g. frequency, intensity, duration, etc.) is presented within a sequence of repeated standard stimuli (hereafter *standards*). Extensively studied in the auditory domain, the MMN is measured by subtracting the ERP response to standards, which primarily appear near the end of a stimulus sequence, from that of deviants (Garrido, Kilner, Stephan, & Friston, 2009). The MMN typically peaks around 100–250 ms from the point of deviation (Kujala & Näätänen, 2001). The MMN is usually maximal at fronto-central areas and exhibits a polarity reversal at the mastoids (Kujala & Näätänen, 2001), while its neural source is localised in temporal and frontal areas (Alho, 1995). Importantly, attention is not required to elicit the MMN, as it is associated with pre-attentive processing in the auditory domain (Näätänen et al., 2001).

Regarding the mechanism of MMN, two hypotheses were postulated mainly, namely the adaptation hypothesis (Jääskeläinen et al., 2004; May et al., 1999; May & Tiitinen, 2010) and the model-adjustment hypothesis (Näätänen et al., 2005; Näätänen & Alho, 1995; Näätänen & Winkler, 1999; Sussman & Winkler, 2001; Winkler et al., 1996). According to the adaptation hypothesis, the MMN arises from the attenuation and delay of the N1 component because of adaptation to the repetitive standard stimuli (May et al., 1999; May & Tiitinen, 2010). Consequently, the MMN does not reflect higher-level comparison processing or mismatch detection but rather signifies a release from stimulus-specific adaptation (Fishman, 2014). In contrast, the model-adjustment hypothesis (Näätänen et al., 2005; Näätänen & Alho, 1995; Näätänen & Winkler, 1999; Sussman & Winkler, 2001; Winkler et al., 1996) posits that the MMN represents the result of change detection between the

deviants and the memory trace formed by the standards. Initially, this hypothesis applied only to repeated sounds of the same standards, but it was later expanded to encompass the detection of regularity violation to explain the presence of the MMN in experiments using standard stimuli with predictable patterns or regularities. For example, sequences of tones with increasing frequencies, with deviants that disrupt the regularities, such as repeated or decreased tones (Winkler, 2007). The occurrence of the MMN in such experimental designs supported the model-adjustment hypothesis over the adaptation hypothesis, as no repeated sound could induce adaptation in the regularities (Garrido, Kilner, Stephan, & Friston, 2009).

While the aforementioned studies aimed to distinguish the two postulations of MMN mechanisms, it is important to note that they are not mutually exclusive. Indeed, a predictive coding framework could reconcile the adaptation hypothesis and the model-adjustment hypothesis (Carbajal & Malmierca, 2018; Friston, 2005; Garrido, Kilner, Stephan, & Friston, 2009; Winkler, 2007). This framework suggests that our brain extracts regularities from a sequence of stimuli and forms hypotheses about upcoming stimuli based on these regularities. If a deviant occurs, the MMN is elicited because a prediction error arises from the violation of the hypothesis regarding the stimulus and regularities by the deviant. The predictive coding framework incorporates the adaptation hypothesis by proposing that when standard stimuli can be predicted more precisely by the top-down process, less weight is assigned to bottom-up influences, resulting in stronger adaptation manifested by a weaker and delayed N1 (Garrido, Kilner, Stephan, & Friston, 2009). From the predictive coding perspective, adaptation and MMN can be viewed as microscopic and macroscopic correlates of the same deviance-detection process when the repetition rule is involved (Carbajal & Malmierca, 2018). However, the predictive coding framework aligns with the model-adjustment hypothesis in suggesting that the MMN arises from a comparison between predicted input based on memory traces and actual input. A mismatch between prediction and input generates a prediction error, and the prediction model has to be adjusted. Therefore, within the predictive coding framework, the MMN signifies failures to predict bottom-up input and to suppress prediction error (Garrido, Kilner, Stephan, & Friston, 2009).

Importantly, the formation of the MMN, which is measured by the amplitude of a difference waveform generated by subtracting the average response to standards from that to deviants, is not solely attributed to the more negative amplitude in the deviant trials. It also involves more positive (or less negative) amplitudes resulting from adaptation in the standard trials. For instance, if the amplitudes of the N1 and P2 components become less negative and more positive with repetitions, respectively, the MMN will be larger, assuming that the amplitude of the deviant remains unchanged. Conversely, the MMN will be smaller if the N1 and P2 amplitudes become more negative and less positive with repetitions. This underscores the relevance of repetition positivity (RP), an increase in a slow positive wave from 50 to 250 ms post-stimulus onset with stimulus repetition (Cooper et al., 2013; Recasens et al., 2015), to MMN.

RP plays a crucial role in supporting the predictive coding mechanism of MMN. It is formed by subtracting the ERPs of standard stimuli with fewer repetitions from those with more repetitions. Importantly, the RP is not confined to the N1 time window, which poses a challenge to the adaptation hypothesis of MMN. This suggests that the N1 adaptation effect alone can fully explain MMN (Haenschel et al., 2005). In the study by Haenschel et al. (2005), RP was observed early, starting from the P1 time window, and its amplitude increased with more repetitions of standards. The researchers also found that RP could account for a large proportion of the MMN (Haenschel et al., 2005). Based on these findings, the researchers proposed that RP serves as an ERP correlate of adaptation, a mechanism involved in memory trace formation in the primary auditory cortex (A1). According to the predictive coding account, RP serves as an index of prediction error suppression resulting from the congruence between sensory input and predicted input (Baldeweg, 2007). Notably, because RP corresponds to an increase in P2 and a decrease in N1, it appears contradictory to the conventional understanding of adaptation associated with a diminishing P2. In addition, despite its significance, research on RP remains relatively limited compared to studies on MMN. Thus, further investigations are needed to fully elucidate the relationships among RP, adaptation and MMN.

1.3 | Expectation control and the present study

Importantly, previous studies have suggested that stimulus expectations, which stem from statistical regularities in the environment (Todorovic et al., 2011), can influence components related to adaptation and MMN. For instance, studies have shown that the repetition of stimuli can lead to enhanced N1 amplitudes when participants expect the stimuli (Hari et al., 1979). Additionally, the consistent pairing of standards and deviants may induce perceptual grouping even with long stimulus onset asynchronies (SOAs), potentially affecting MMN amplitudes (Herholz et al., 2009). Explicit top-down

expectations have been found to diminish MMN responses (Chennu et al., 2013; Costa-Faidella, Grimm, et al., 2011; Lecaignard et al., 2021). Recent evidence has suggested that participant-generated stimulus expectations can modulate adaptation effects, even in the absence of attentional involvement (e.g. Barbosa & Kouider, 2018; Kuravi & Vogels, 2017; Todorovic et al., 2011). These findings highlight the role of top-down processing in shaping the adaptation effects.

Based on the findings mentioned above, controlling for stimulus expectations is crucial when investigating adaptation effects. However, experimental constraints may inadvertently modify expectations. For example, in typical MMN experiments, consecutive occurrences of deviants are often avoided, and a maximum number of tone repetitions is enforced. These constraints may lead participants to generate expectations regarding whether the next stimulus will be a deviant or standard, potentially influencing adaptation and MMN. To address this, the present study implemented an experimental paradigm with minimal constraints on stimulus arrangements to control subjective expectations by maintaining objective predictability. Specifically, stimuli were presented with a stable probability (85% standard, 15% deviant) and extended sequences of up to 30 standards, ensuring a pure measurement of adaptation unaffected by participant expectations.

The present study used *initial adaptation* to describe the amplitude decrease from the first to the second tones and *subsequent adaptation* to capture the decrease from the second to the final tones in each sequence of identical stimuli. Through an auditory experimental paradigm with controlled expectations, the present study addresses two primary research questions: (1) How do the patterns of initial and subsequent adaptation patterns manifest in the N1 and P2 components? (2) To what extent can these adaptation patterns elucidate the MMN? These two research questions were examined using an EEG experiment in healthy adults, coupled with adaptation pattern, correlation and regression analyses.

Regarding the first research question, we predicted that the N1 and P2 components would show reduced amplitudes in response to the initial tones, indicative of adaptation, in line with previous findings (Budd et al., 1998; Rosburg & Sörös, 2016). However, we anticipated potential increases in P2 amplitudes because of the influence of RP, suggesting a memory trace effect related to the model adjustment account (Haenschel et al., 2005).

Concerning the second research question, we predicted positive correlations between MMN and adaptation effects observed in the N1 component, as well as negative correlations between MMN and adaptation effects observed in the P2 component. Furthermore, we hypothesised that both initial and subsequent adaptation effects would be important predictors of the MMN, as suggested by the predictive coding account, which posits the involvement of both adaptation and the model adjustment mechanisms in MMN generation.

2 | MATERIALS AND METHODS

2.1 | Participants

We recruited 40 adults to participate in the experiment. Three participants were excluded because of excessive muscle artefacts or electric noise. Therefore, the data of the remaining 37 participants (*age range*: 19–26 years; *mean age*: 20.84 years; 19 females) were analysed. Two of them were left-handed, whereas the others were right-handed. All participants were undergraduate students studying at the Chinese University of Hong Kong (CUHK). Written consent was obtained from each participant before the experiment began. The participants were compensated either with course credits or cash. This study was approved by The Joint Chinese University of Hong Kong–New Territories East Cluster Clinical Research Ethics Committee (The Joint CUHK-NTEC CREC) (reference no.: 2019.048).

2.2 | Stimuli and procedure

The experiment was designed and implemented using E-Prime 3.0 (Psychology Software Tools, Pittsburgh, PA). Stimuli consisted of pure sinusoidal tones spanning seven different frequencies (500, 550, 600, 650, 700, 750 and 800 Hz). Each tone had a duration of 70 ms, including 5 ms rise and fall times. The stimulus onset asynchrony (SOA) was fixed at 650 ms. All tones were generated and scaled to a sound pressure level (SPL) of 70 dB using Praat (Boersma, 2001).

The present study employed a roving paradigm, where a deviant stimulus becomes the subsequent standard. This design allows the MMN to accurately reflect inherent differences between the two types of stimuli, without being influenced by extraneous factors such as frequency or duration. The roving paradigm has been utilised in previous studies (e.g. Costa-Faidella, Baldeweg, et al., 2011; Cowan et al., 1993; Garrido et al., 2008; Haenschel et al., 2005; Recasens et al., 2015). EEG data were collected using NetStation, employing a Net Amps 300 amplifier (Electrical Geodesics Inc.) and corresponding 128-channel nets with a reference electrode placed at Cz. The sampling rate was set to 500 Hz, and online filtering was applied with a high-pass cutoff of .1 Hz. Before

the experiment commenced, the impedance of each electrode was verified to be below 50 kΩ. Data acquisition occurred within a sound-attenuated laboratory environment. Prior to the experiment, the participants were informed that some sounds would be presented when they watched a silent movie, and they were asked to disregard these sounds. Also, the participants were instructed to minimise body movements during the experiment to reduce muscle artifacts.

In the present experiment, the first tone was randomly selected from among seven types of tones with varying frequencies. Subsequently, there was an 85% probability that the following tone (standard) would match the frequency of the preceding tone, while there was a 15% chance that the next tone (deviant) would be different. This criterion remained consistent throughout the experiment to control participants' expectations of the stimulus arrangement. However, an exception was made: if 30 tones were played consecutively, the next tone had to be a deviant. On average, this occurred 2.73 times ($SD = 1.59$ times; *range*: 0–5 times) across approximately 300 sequences. This restriction was implemented to ensure an adequate number of trials for analysis. Throughout the experiment, the participants watched a silent movie, 'Tom and Jerry', on a laptop while the tones were played through two speakers positioned 80 cm away from them. The experiment ended after 2000 tones had been presented, divided into two blocks with 1000 tones each. A brief pause occurred between the two blocks to allow the experimenter to check the impedance of all electrodes and adjust if necessary. The duration of the experiment was approximately 20 min. Figure 1 illustrates the experimental paradigm.

After the EEG experiment, the participants underwent a tone discrimination task using E-Prime 3.0 to confirm their tone discrimination ability. Details of this task are provided in the Supporting Information.

2.3 | EEG preprocessing

BrainVision Analyzer (version 2.2.1.8266; Brain Products, Gilching, Germany) was used for data preprocessing and waveform analyses. The data preprocessing steps for each participant began with segmentation, followed by filtering (.3-Hz high-pass with a slope of 24 dB/oct and 30 Hz low-pass with a slope of 48 dB/oct), bad channel selection, raw data inspection, independent component analysis (ICA) for corrections of eye blink and lateral eye movement artifacts using a restricted Infomax algorithm, bad channel interpolation with spherical splines, re-referencing to the average signals recorded from all electrodes, and artifact rejection (if an extreme voltage [$> +80$ or < -80 μV] was detected). A fixed delay of 18 ms was corrected because of the anti-aliasing filter of the amplifier (see the advisory notice about the timing affected by anti-alias filter effects used in Net Amps 300 amplifiers, 26 November 2014, EGI for more details). The epoch ranged from 168 ms pre-stimulus to 632 ms post-stimulus. Baseline correction was conducted based on pre-stimulus period data.

Based on these preprocessing steps, 24% of trials were rejected on average. For the adaptation and MMN analyses, only the final tones in the fourth to 30th positions were included, ensuring a balanced comparison between standards and deviants by maintaining a similar number of each. The average remaining trial numbers in the three conditions related to the adaptation effects, namely the deviants (first tones), the second tones and the final tones in the fourth to 30th positions, were 230 (*range*: 152–297),

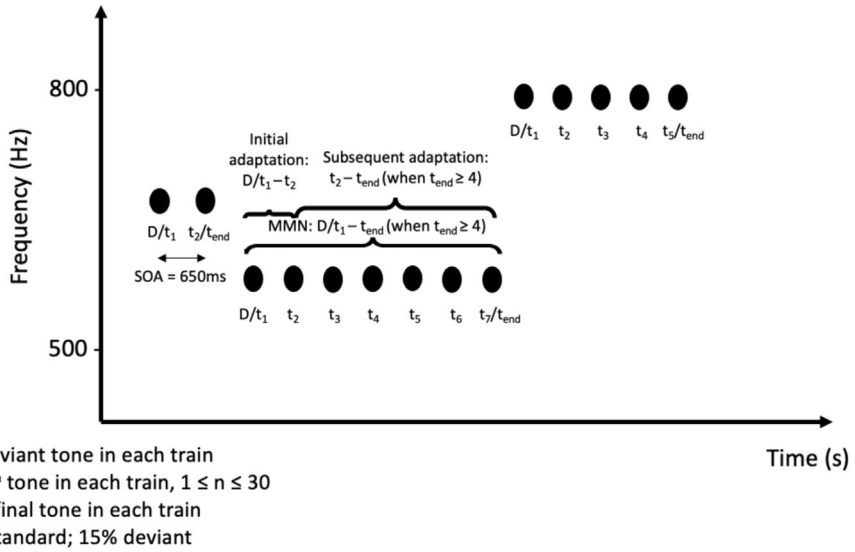

FIGURE 1 Roving paradigm with controlled expectations utilised in the present study, with the calculation methods for the amplitudes of initial adaptation, subsequent adaptation and MMN.

197 (*range*: 133–250) and 139 (*range*: 98–172), respectively. The average remaining trial numbers for the conditions of the third to 10th tones, which are relevant to the adaptation pattern analyses, are detailed in Table S1 in the Supporting Information.

2.4 | Data analyses

Based on previous literature concerning adaptation and MMN (e.g. Bühler et al., 2017; Jaffe-Dax et al., 2017; Jost et al., 2015), a set of 25 fronto-central electrodes were pooled for analyses: E19, E11, E4, E20, E12, E5, E118, E13, E6, E112, E10, E16, E18, E30, E7, E106, E105, E37, E31, E129, E80, E87, E55, E36 and E104 (see the top-left corner of Figures 3 or 4a for the electrode positions).

The time window for detecting the local peak amplitude of N1 was determined using the global field power (GFP) and global dissimilarity peaks from subsequent maps of the grand average data. GFP represents the root mean square (RMS) across the average-referenced electrode values or the standard deviation of all electrodes at a given time point (Murray et al., 2008), whereas global dissimilarity quantifies configuration differences between maps, irrespective of their strength (Lehmann & Skrandies, 1980; Murray et al., 2008). The GFP and global dissimilarity were generated from BrainVision Analyzer and Cartool (Brunet et al., 2011).

The N1 time windows for deviants, second tones and final tones were 70–218 ms, 76–118 ms and 76–140 ms, respectively. In contrast, as there was no distinct GFP peak in the P2, its time windows were determined by peaks between N1 and N2. The P2 time windows of the corresponding conditions were 120–348, 96–240, and 98–254 ms, respectively (see Figure S1 for illustrations of how these P2 time windows were defined).

In addition, the difference wave corresponding to the MMN was computed by subtracting the amplitudes of the final tones in the fourth to 30th positions from those of the deviants in the same fronto-central electrodes (deviants–final tones), with the time window defined based on the GFP and the global dissimilarity peaks as 76–196 ms. The local peak MMN amplitude was automatically identified using BrainVision Analyzer.

Similarly, for the adaptation effects, the local peak amplitude in each tone position and time window was automatically determined using BrainVision Analyzer. We defined the initial adaptation as the peak amplitude decrease from the deviants to the tones in the second position in the fronto-central electrodes (deviants–second tones). Nevertheless, the subsequent adaptation was defined as the subtraction of the peak amplitudes of the final tones in the fourth to 30th positions from those of the tones in the second position in the same electrodes (second tones–final tones). Figure 1 depicts the calculation methods of initial adaptation, subsequent adaptation and MMN.

The adaptation curves for the first 10 tones of N1 and P2 were first plotted based on the peak amplitudes in the corresponding time window. Given that the trial number might not be sufficient (< 50 trials on average) after the 10th tones to generate reliable results, we only focussed on the first 10 tones for the adaptation curves. Paired sample *t*-tests were conducted between each consecutive tone pair, with Holm–Bonferroni corrections (Holm, 1979) applied to control for multiple comparisons. Additionally, repeated-measures analysis of variance (ANOVA) was performed on the amplitude employing an a priori linear trend analysis across the first 10 tones in each stimulus sequence to elucidate the adaptation pattern observed in N1 and P2. To ascertain the presence of MMN, a one-sample *t*-test against zero was conducted for the amplitude of the difference wave.

Furthermore, the relationship between initial adaptation, subsequent adaptation effects, and MMN was examined through correlation and backward stepwise linear regression analyses. In the regression model, the independent variables (IVs) comprised N1 and P2 initial adaptations and subsequent adaptations, while MMN peak amplitude served as the dependent variable (DV). The elimination criterion for the backward regression was set at $P > .10$, meaning that in each step, the IV with the lowest partial correlation with the DV, meeting the criterion $P > .10$, was removed until all variables remaining in the model were $P < .10$. All statistical tests were performed using IBM SPSS Statistics Version 25 (IBM, Armonk, NY, USA).

3 | RESULTS

Figure 2 illustrates the adaptation patterns characterised by the peak amplitudes of N1 and P2 across the first 10 tones. The ERPs corresponding to the first 10 tone positions are presented in Figure 3. Details are described below.

3.1 | N1 adaptation

The adaptation curve for the first 10 tones, derived from the average N1 peak data, was plotted based on the peak amplitudes in the N1 time window. A steep decrease in N1 amplitude was observed between the first and second tones, followed by a plateau (Figure 2a). Paired-sample *t*-tests revealed that the N1 amplitude of the second tones

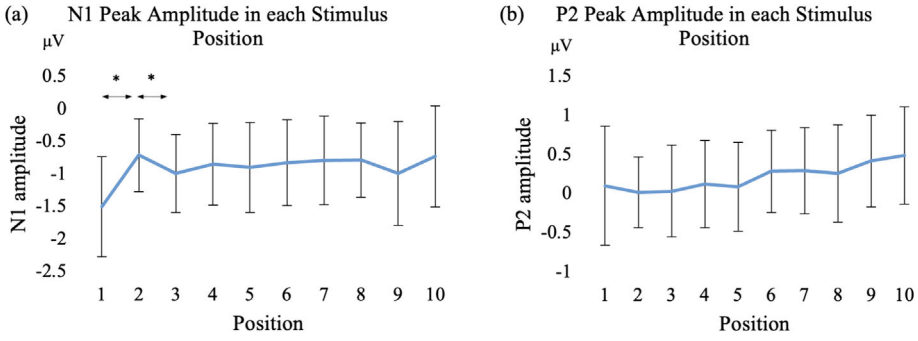

FIGURE 2 The adaptation curves based on the (a) N1 and (b) P2 peak amplitudes across the first 10 tones ($N = 37$). Error bars represent standard deviations. *Significant P-values based on paired-sample t-tests between consecutive tone pairs, adjusted using Holm–Bonferroni corrections.

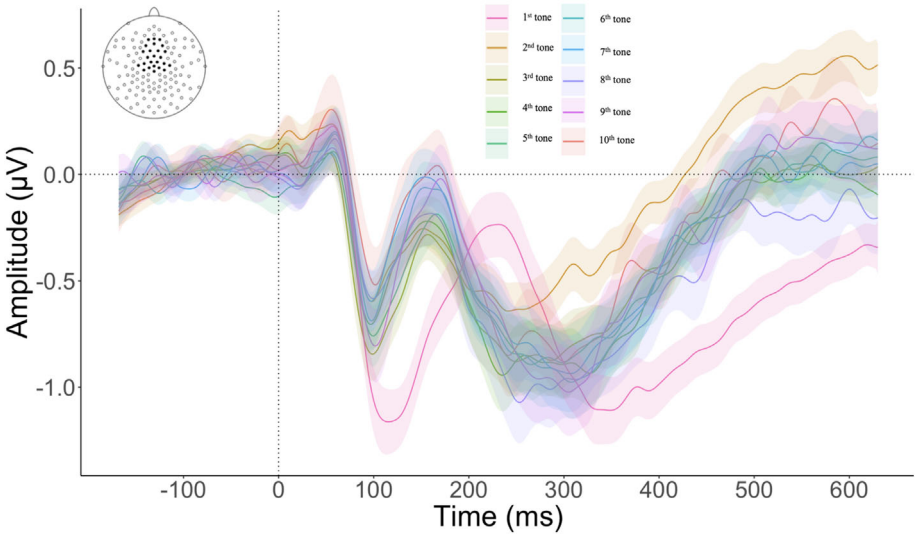

FIGURE 3 The ERPs of the first 10 tones indicated by different colours ($N = 37$). Data were pooled from 25 fronto-central electrodes (marked by black dots at the top-left corner). Shaded ribbons indicate standard errors of the means (SEMs).

($M = -.71$ μV, $SD = .56$ μV) was significantly less negative than that of the first tones ($M = -1.51$ μV, $SD = .77$ μV; $t_{36} = -6.68$, $P < .001$, $d = .72$). Additionally, there was a rebound effect, indicated by a significant increase in the N1 amplitude from the second to the third tones ($M = -.99$ μV, $SD = .60$ μV; $t_{36} = 3.91$, $P < .001$, $d = .43$). No other significant difference was observed in subsequent tone pairs (all $P > .007$ based on the Holm–Bonferroni correction).

Furthermore, a significant linear trend was evident across the first to 10th tones ($F_{1,36} = 17.06$, $P < .001$, $MSE = 4.40$, $\eta_p^2 = .32$). However, no significant linear trend was observed across the second to 10th tones ($F_{1,36} = .08$, $P = .78$, $MSE = .02$, $\eta_p^2 = .002$), suggesting that the initial amplitude decrement observed from the first to second tones did not continue across subsequent tones (Figure 2a).

3.2 | P2 adaptation

Similarly, the average peak amplitudes of the P2 were first extracted across the first 10 positions to construct adaptation curves. As depicted in Figure 2b, there was an increasing trend in P2 amplitude with the first 10 stimuli. Paired-sample t-tests revealed no significant differences between individual tone pairs after adjusting for Holm–Bonferroni corrections ($P > .006$). However, a significant linear trend was observed both from the first to 10th tones ($F_{1,36} = 26.90$, $P < .001$, $MSE = 7.43$, $\eta_p^2 = .43$) and from the second to 10th tones ($F_{1,36} = 33.28$, $P < .001$, $MSE = 7.64$, $\eta_p^2 = .48$). These findings indicate a continuous amplitude increase in the P2 time window, extending beyond the initial two tones (Figure 2b).

3.3 | MMN

A one-sample t-test was conducted to compare the average peak amplitude in the MMN time window of the difference wave (deviants minus final tones in the fourth to 30th positions) with zero. The analysis revealed that the amplitude was significantly lower than zero ($M = -1.01$ μV, $SD = .60$ μV; $t_{36} = -10.28$, $P < .001$), indicating the elicitation of MMN. Figure 4 illustrates the MMN-related difference wave and

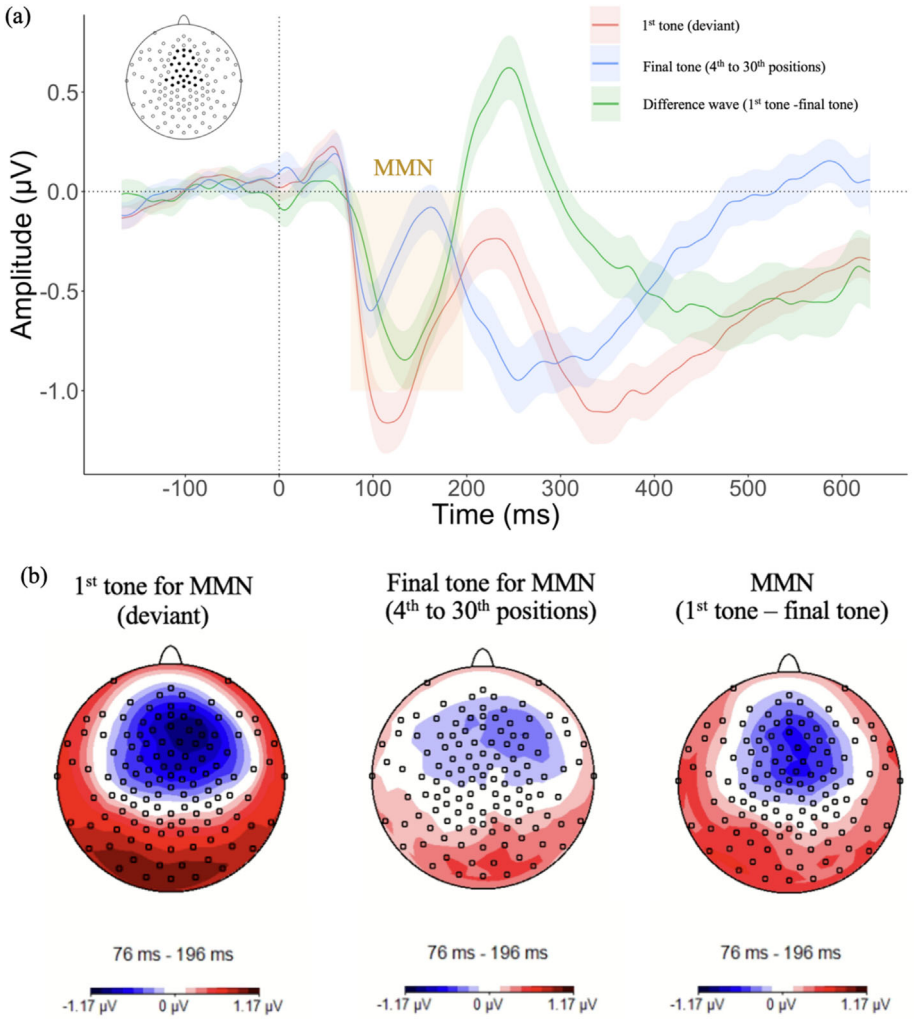

FIGURE 4 (a) Waveforms of deviants, last standards in fourth to 30th tones and the difference wave associated with MMN ($N = 37$). Data were pooled from 25 fronto-central electrodes (marked by black dots at the top-left corner). The average difference wave (green) was computed by subtracting the amplitudes in the final position of each sequence of stimuli (fourth position or after) from those in the deviants. Waveforms of deviants and the final tones are depicted in red and blue, respectively. Shaded ribbons indicate SEMs. The shaded region (yellow) denotes the MMN time window (76–196 ms). (b) Topography of MMN. From left to right, the topographic maps illustrate deviants, final tones in fourth to 30th positions and the corresponding MMN.

topography, characterised by fronto-central negativity and temporal positivity.

3.4 | Adaptation and MMN

Descriptive statistics of the amplitudes of the N1 and P2 initial adaptation and subsequent adaptation effects, along with the MMN, are provided in Table 1.

TABLE 1 Descriptive statistics of the peak amplitudes of MMN and adaptation in N1 and P2 ($N = 37$).

Variable	*Mean*	*SD*
MMN amplitude	−1.01	.60
N1 initial adaptation amplitude	−.79	.72
N1 subsequent adaptation amplitude	.06	.44
P2 initial adaptation amplitude	.08	.79
P2 subsequent adaptation amplitude	−.20	.41

3.4.1 | Correlation analyses

Table 2 displays the results of the Pearson correlation analysis of the variables. A positive and strong correlation between the amplitudes of MMN and N1 initial adaptation was observed ($r_{35} = .67$, $P < .001$). This indicates that the participants with a stronger N1 initial adaptation effect exhibited a larger MMN (see Figure 5a). No other significant correlations related to the MMN amplitude were identified (all $P > .008$ based on Holm–Bonferroni correction).

3.4.2 | Regression analyses

The relationship between adaptation and MMN was further examined through a backward stepwise regression to elucidate which adaptation effect contributes to the MMN and to what extent. All four adaptation variables measured by amplitudes were included in the regression model, with the MMN peak amplitude as the DV. Eventually, only N1 initial adaptation (β [standardised beta] = .72; $P < .001$) and P2 subsequent adaptation ($\beta = .33$; $P = .009$) emerged as significant predictors in the final model ($F_{2,34} = 20.50$;

TABLE 2 Pearson correlations between the peak amplitudes of MMN and adaptations in N1 and P2 ($N = 37$).

	1	2	3	4	5
1. MMN amplitude	-	.67*	−.07	.23	.22
2. N1 initial adaptation amplitude	-	-	−.47*	.51*	−.16
3. N1 subsequent adaptation amplitude	-	-	-	.24	.46*
4. P2 initial adaptation amplitude	-	-	-	-	−.32
5. P2 subsequent adaptation amplitude	-	-	-	-	-

*Significant *P*-values with Holm–Bonferroni corrections.

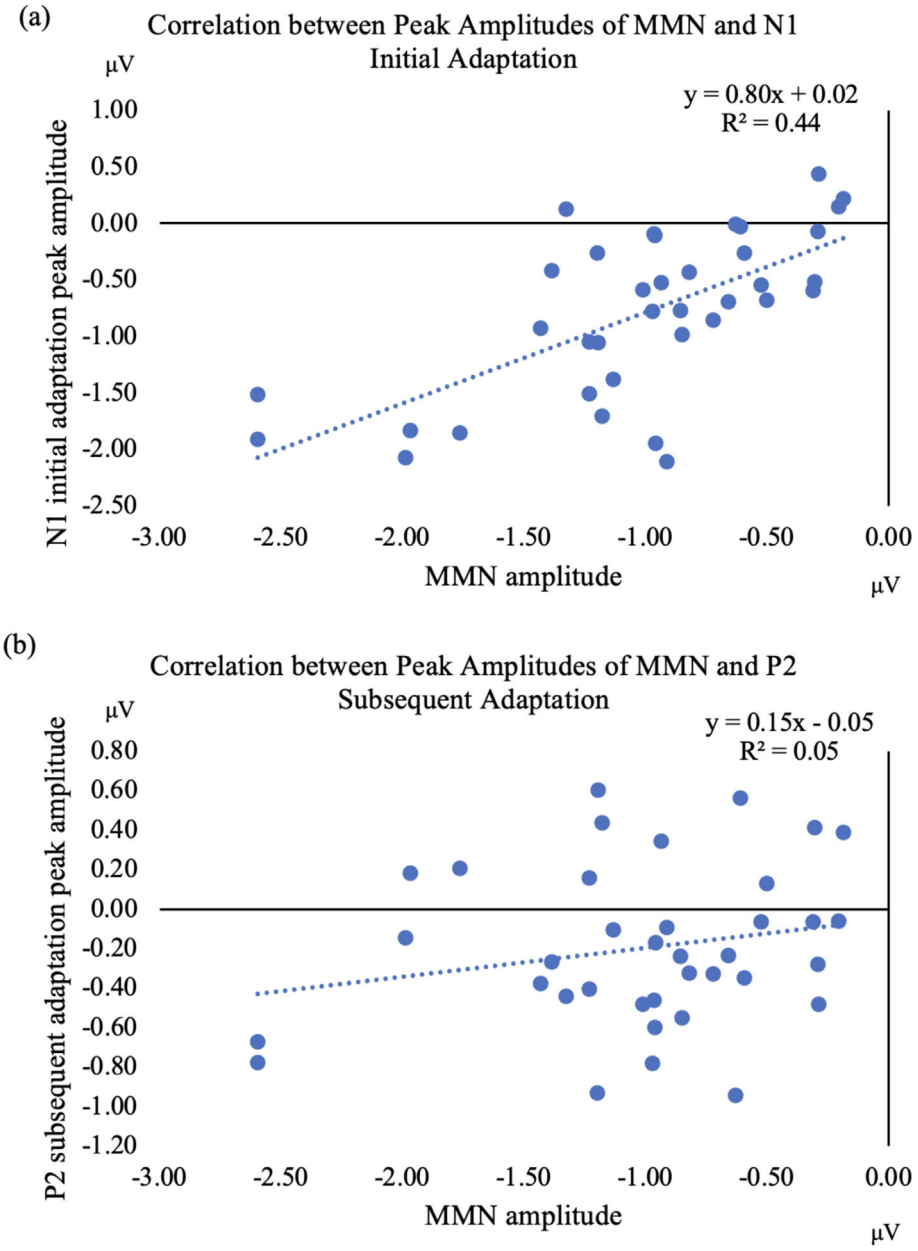

FIGURE 5 Scatterplots of significant correlations or regressions between MMN and the variables related to the adaptation effects, namely (a) N1 initial adaptation amplitude and (b) P2 subsequent adaptation amplitude. Each dot indicates a datum from each participant ($N = 37$).

$P < .001$; see Figure 5b for the correlation between the P2 subsequent adaptation effect and MMN). The squared semi-partial correlation, indicating the unique contribution of each independent variable to the dependent variable (Aloe & Becker, 2010), revealed that these two variables collectively explained over 50% of the MMN variance (49.98% and 10.43%, respectively). The standardised coefficients of each variable and the adjusted R^2 of the

initial and final models of the adaptation and MMN are presented in Table 3.

To ensure that the initial and subsequent adaptation effects did not influence each other, we conducted partial correlations. Specifically, we examined the partial correlation between the MMN and the N1 adaptation amplitudes while controlling for the N1 subsequent adaptation effect, as well as the partial correlation between the MMN and the P2 subsequent adaptation amplitudes while controlling for the P2 initial adaptation effect. These analyses yielded consistent results with the corresponding correlations reported earlier, indicating that the initial and subsequent adaptation effects relevant to the regression analysis were not confounded. Detailed results are presented in Table S2.

3.4.3 | Additional analyses

Although efforts were made to balance the trial numbers by including only the final standards (but not all standards) in the analyses, the trial numbers remained unequal among the deviant, second tone and final tone conditions. To address this issue, we conducted the same analyses with an equal number of trials in these three conditions, based on the trial number of the final tone condition (the condition with the least trial numbers) in each participant. The results were consistent with our previous findings, showing that N1 initial adaptation and P2 subsequent adaptation amplitudes predicted the MMN amplitude in the regression model. However, N1 subsequent adaptation also emerged as a significant predictor. Further details can be found in the Supporting Information.

3.4.4 | Topographic analysis

To assess potential differences in the topographic distributions of the two adaptation effects and infer distinct underlying neural sources (Michel & Murray, 2012), a topographic analysis of variance (TANOVA) was conducted using RAGU (Koenig et al., 2011). This analysis compared individual N1 initial adaptation (peak amplitudes of deviants–peak amplitudes of the second tones) and P2 subsequent adaptation (peak amplitudes of the second tones–peak amplitude of final tones in the fourth to 30th positions) with 5000 randomisation runs. The scalp topographies for N1 initial adaptation and P2 subsequent adaptation, normalised for strength (GFP = 1), were examined (see Figure 6; for the scalp topographies for N1 subsequent adaptation and P2 initial adaptation, see Figure S2).

The results indicated a significant difference in the topographic maps of N1 initial adaptation and P2 subsequent adaptation ($P < .05$), suggesting disparate topographic distributions and underlying sources for these two adaptation effects. Specifically, the gradients of the N1 initial adaptation effect exhibited a steep incline over left superior temporal regions, whereas the gradients of the P2 subsequent adaptation effect were steepest over bilateral frontal regions. These findings align with the notion of initial adaptation occurring in auditory cortex regions and the model reestablishment effect in bilateral frontal regions.

TABLE 3 Standardised coefficients and adjusted R^2 in the regression analysis of the amplitudes of adaptation effects and MMN ($N = 37$).

	Model	
Variable	**Initial**	**Final**
N1 initial adaptation amplitude	.83*	.72*
N1 subsequent adaptation amplitude	.20	-
P2 initial adaptation amplitude	−.06	-
P2 subsequent adaptation amplitude	.23	.33*
Adjusted R^2	.52	.52

*$P < .01$.

4 | DISCUSSION

Neural adaptation serves as a fundamental mechanism through which the brain utilises previous experiences with stimuli (e.g. Grill-Spector et al., 2006). While previous studies in the auditory domain have predominantly focussed on examining adaptation through stimulus pairs or series of repetitions, the present study delved deeper into the adaptation patterns of N1 and P2, comparing initial versus subsequent repetitions. Unlike previous MMN investigations that employed varied paradigms to differentiate the adaptation and model adjustment accounts of MMN (e.g. Jacobsen & Schröger, 2001), the present study investigated the interplay between MMN and adaptation patterns in N1 and P2. Crucially, expectations were controlled by presenting tones with fixed probabilities with extended sequences. The results unveiled adaptation effects within the N1 component during initial tone repetitions. Conversely, the P2 amplitude exhibited a linear increase with successive repetitions, suggestive of the model adjustment effect. Notably, MMN amplitudes were mainly influenced by the N1 adaptation effect induced by the initial tones and the continuous P2 adaptation effect elicited by subsequent tones. Further elucidation of these adaptation patterns is provided below.

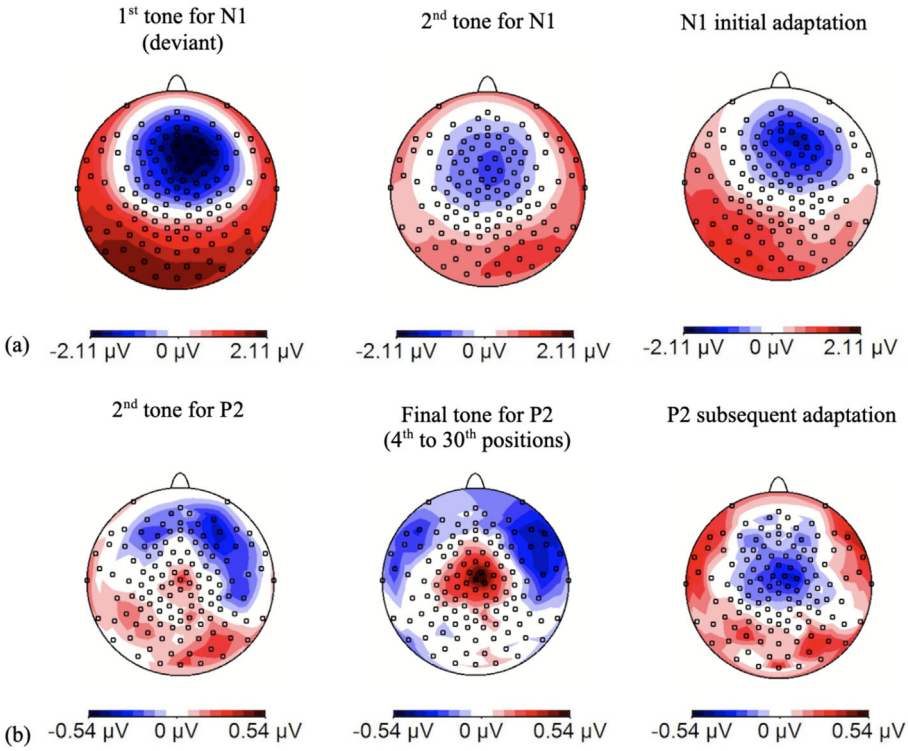

FIGURE 6 Scalp topographies based on the average individual peak amplitude ($N = 37$). (a) From left to right, the topographic maps of deviants, second tones and the ensuing initial adaptation effect in the N1. (b) From left to right, the topographic maps of second tones, final tones (fourth to 30th positions) and the ensuing subsequent adaptation effect in the P2.

4.1 | N1 adaptation at the initial stage

The pronounced amplitude decrease observed from the first tone to the second tone in the N1 adaptation effect aligns with our hypothesis and is consistent with findings from prior studies (e.g. Bourbon et al., 1987; Budd et al., 1998; Lagemann et al., 2012; Recasens et al., 2015; Rosburg, 2004; Rosburg et al., 2006). This finding suggests that N1 adaptation occurs quickly in response to simple tone stimuli presented in rapid succession when expectations are controlled. Nevertheless, these results may not generalise to conditions with longer interstimulus intervals (ISIs). Previous studies have shown that while the steep amplitude decrease is evident with short ISIs, a further reduction occurs with longer ISIs (e.g. Fruhstorfer et al., 1970; Özesmio et al., 2000; Sambeth et al., 2004; c.f. Bourbon et al., 1987; Budd et al., 1998; Rosburg et al., 2010). Moreover, a previous study has indicated that the primary source of the N1 response is the temporal lobe when employing a one-second ISI condition. However, with longer ISI, activity from brain regions beyond the auditory cortex becomes more prominent (Hari et al., 1982). Given that the present study employed a short ISI (580 ms), the observed steep decrease in amplitude may be ascribed to the ISI.

Additionally, a rebound was observed between the second and the third tones in the N1 adaptation curve (Figure 2a). This phenomenon, not anticipated by the adaptation account, may be linked to the findings from a previous study utilising dynamic causal modelling (DCM) to examine adaptation effects represented by the intrinsic connections in A1 (Garrido, Kilner, Kiebel, et al., 2009). Based on their interpretation, the initial decrease in intrinsic connectivity from the first tone to the second tone indicated reduced estimated precision of predictions triggered by the deviants, while the subsequent increase reflected gradual recovery because of learning. Hence, the rebound observed in the N1 adaptation effect between the second and third tones in our study could align with the notion proposed by Garrido, Kilner, Kiebel, et al. (2009): the adaptation effect in A1 may incorporate a predictive component, and the brain requires only a few repetitions to generate predictions. Importantly, the present finding extends this interpretation to situations where expectations are controlled, suggesting that the predictive component in the adaptation effect can be automatically generated.

4.2 | P2 adaptation: a model adjustment effect

The P2 amplitude displayed a continuous increase over the first 10 tones, contrary to our hypothesis and findings from previous studies where a steep decrease followed by a plateau was observed (Rosburg et al., 2010; Rosburg & Sörös, 2016). However, this continuous increase could be interpreted as RP, as seen in studies by Costa-Faidella,

Baldeweg, et al. (2011) and Costa-Faidella, Grimm, et al. (2011). Additionally, Recasens et al. (2015) reported repetition enhancement in a time window close to P2 (230–270 ms after stimulus onset).

Discrepancies in these findings may stem from differences in paradigms, such as variations in participant expectations and ISI. For instance, in studies where participants could easily predict the tone pattern (e.g. Rosburg & Sörös, 2016), a sharp decline followed by a plateau in P2 was observed. Conversely, when tone predictability was low, as seen in the present study and others (Costa-Faidella, Baldeweg, et al., 2011; Costa-Faidella, Grimm, et al., 2011; Recasens et al., 2015), a continuous increase was noted. Hence, expectations may play a role in modulating the adaptation pattern revealed by P2. Moreover, differences in the time allowed for recovery from adaptation may contribute to the discrepancies. Shorter ISIs used in previous studies (e.g. Costa-Faidella, Baldeweg, et al., 2011; Costa-Faidella, Grimm, et al., 2011; Recasens et al., 2015) may lead to continuous P2 increase, whereas longer ISIs (e.g. Rosburg et al., 2010; Rosburg & Sörös, 2016) may result in a sharp decrease followed by a plateau.

Furthermore, variations in experimental paradigms used across studies can also contribute to the inconsistencies. Studies by Costa-Faidella, Baldeweg, et al. (2011), Costa-Faidella, Grimm, et al. (2011) and Recasens et al. (2015), like the present one, employed a roving paradigm, potentially enhancing memory trace because of unpredictable tones. In contrast, paradigms with discontinuous stimulus sequences or predictable standard tones, such as the stimulus pair or traditional oddball paradigms, may require less memory trace strengthening (Cooper et al., 2013). This underscores the link between the RP and memory trace, particularly in paradigms like roving where sensory memory must be constantly updated (Cooper et al., 2013). As these postulations lack direct comparison, factors influencing the relationship between RP and adaptation warrant further investigation. Notably, the adaptation effect observed in the stimulus pair and traditional oddball paradigms may not generalise to roving paradigms or situations where expectations are controlled.

As previously discussed, the increase in P2 can be interpreted as an RP. Despite the negative frontal-central component observed in the topography of the P2 subsequent adaptation (Figure 6b), this was because of the reverse calculation method of the adaptation effect (second tone–final tone instead of final tone–second tone). Nonetheless, the RP found in the present study exhibited a delayed onset compared to a previous study by Haenschel et al. (2005), wherein they compared RP across two, six, and 36 standard repetitions in healthy adults. Their findings indicated that RP, occurring roughly from 50 to 250 ms post-stimulus, was larger in conditions with greater standard repetitions, with RP contributing predominantly to the MMN. Hence, they suggested that RP serves as an ERP correlate of adaptation, a mechanism that facilitates memory trace formation in the A1.

However, it remained uncertain whether the heightened RP and MMN observed after 36 standard repetitions in Haenschel et al. (2005) were attributable to an extended memory trace or an enhanced precision in participants' expectation. After a certain duration, the participants might readily expect the appearance of a deviant following 36 standard repetitions if none had occurred at the second and sixth positions. In contrast, in the present study, where the participants could not predict the occurrence of deviants, RP, characterised by a continuous amplitude increase across the stimuli, was still evident. However, it manifested with a later onset within the P2 time window, compared to the roughly 50 ms onset observed in the study by Haenschel et al. (2005).

While our findings do not challenge the interpretation made by Haenschel et al. (2005) that RP links to adaptation and memory trace formation, the present findings suggest that the latency of RP may be influenced by the precision of the prediction. Specifically, a delayed onset may manifest when prediction precision diminishes. Conversely, an earlier onset of RP may occur because of a heightened precision in the prediction model, as in Haenschel et al. (2005). However, this postulation remains speculative and warrants future research, given the parameter discrepancies between the two studies, as discussed below.

The differences in experimental parameters between the two studies, including intensity, pitch, and ISI, might lead to the weaker N1 and P2 components found in the current study. Admittedly, while the P2 amplitudes in our study exhibited a positive-going trend, they did not reach positive values as observed in Haenschel et al. (2005), where P2 exhibited stronger amplitudes. One plausible explanation is the difference in stimulus intensity; Haenschel et al. used stimuli with a higher intensity (80 dB) compared to our study (70 dB). Past research indicates that increased stimulus intensity often leads to larger N1 and P2 amplitudes (Adler & Adler, 1989; see Crowley & Colrain, 2004, for a review). Moreover, Haenschel et al. (2005) used more high-frequency stimuli with a boarder range on average (100 to 5000 Hz), compared to our study's lower frequency and narrower range (500 to 800 Hz). Studies have shown that as frequency increases, N1 and P2 amplitudes decrease (Wunderlich & Cone-Wesson, 2001), which could explain the weaker N1 and P2 observed in our study.

Regarding ISI, although it is known to positively affect N1 and P2 amplitudes, it is unlikely to be the primary cause of the discrepancy between our study and Haenschel et al. (2005). Our study used a longer ISI (580 ms) compared to Haenschel et al. (2005) (300 ms). In addition, they employed a between-train interval of 500 ms, whereas we played the sounds continuously. Although the ISI was longer in the present study, our study did not exhibit larger N1 and P2 amplitudes, as one might expect if ISI was a significant factor. More importantly, the disparity in ISI between the two studies was minimal. Previous research has indicated that for each 10-fold rise in ISI, N1 or P2 amplitudes increase by approximately 5.6 μV (Crowley & Colrain, 2004). Hence, the weaker amplitudes observed in our study are mainly attributed to differences in stimulus intensity and pitch.

4.3 | Adaptation and MMN

According to the regression analysis, both N1 initial adaptation and P2 subsequent adaptation amplitudes significantly predicted the MMN, explaining 49.98% and 10.43% of the MMN variance, respectively. While the effect of N1 initial adaptation aligns with the adaptation hypothesis of MMN, the contribution of P2 subsequent adaptation amplitudes suggests that this hypothesis alone may not suffice, as it proposes that the N1 adaptation alone explains MMN. Furthermore, a pure adaptation mechanism struggles to explain the N1 rebound effect. Thus, while the present study underscores the role of adaptation in MMN, it does not dismiss model adjustment or predictive coding accounts. Instead, our main findings support a predictive coding account of MMN, consistent with prior research (e.g. Alain et al., 1999; Herholz et al., 2009; Symonds et al., 2017; Wacongne et al., 2011).

Notably, MMN appears to comprise two distinct processes related to the RP: One involving the less negative N1 inclination during the first repetition, indicative of a purer adaptation effect, and the other involving the P2 subsequent adaptation with a positive deflection, reflecting memory trace formation required by model adjustment. In addition, the N1 initial adaptation effect possibly indicates the extent of model adjustment following the emergence of deviants, whereas MMN quantifies the degree of error detected by the model. Hence, the positive correlation between N1 initial adaptation and MMN suggests that the participants who exhibited heightened sensitivity to the tone changes also had the most precise models. In addition, the increasing P2 amplitude across repetitions aligns with the predictive coding account, which posits that prediction error decreases as top-down predictions match bottom-up inputs (Friston, 2005). This explanation integrates both adaptation and model adjustment accounts (Garrido et al., 2008). Importantly, the present study demonstrates that the predictive coding account holds even when the expectations are controlled, as evidenced by the adaptation findings and the distinct topographies of N1 initial adaptation and P2 subsequent adaptation.

Our regression findings underscore the importance of considering different components and time windows when examining the connection between adaptation and MMN. Specifically, while the N1 initial adaptation contributes to the MMN, the subsequent adaptation of the P2 predicts it. These results suggest that future studies investigating adaptation or its association with MMN should examine both N1 and P2 components. Focussing solely on one component may hinder the discovery of comprehensive adaptation effects. Notably, isolating the N1 and P2 processes could provide insights into the relationship between adaptation and MMN.

4.4 | Implications, limitations and future studies

This study holds theoretical significance because it elucidates the relationship among stimulus repetition, adaptation and MMN. While previous studies often examined the adaptation and MMN separately (e.g. Budd et al., 1998; Haenschel et al., 2005), this study sheds light on their relationship by tracing the adaptation pattern across a sequence of trials and distinguishing between initial and subsequent adaptation effects. Importantly, while Haenschel et al. (2005) found a relationship between RP and MMN, our study more specifically pinpointed the roles of initial N1 adaptation and subsequent P2 adaptation (or RP effect) in MMN. Furthermore, the practical implications are noteworthy as understanding these adaptation patterns and the mechanisms underlying MMN could potentially enhance the effectiveness of clinical applications. For instance, the diagnosis of dyslexia may benefit from utilising adaptation patterns and MMN, given previous studies indicating weaker adaptation and smaller MMN in dyslexic individuals, compared with healthy controls (e.g. Baldeweg et al., 1999; Jaffe-Dax et al., 2017).

It is important to note that the adaptation effect and MMN can be influenced by various factors, including stimulus characteristics such as tone frequency difference (e.g. Butler, 1968), tone duration (e.g. Lanting et al., 2013), number of repetitions (e.g. Baldeweg, 2007) and ISI (e.g. Budd et al., 1998; Herrmann et al., 2016; Lanting et al., 2013; Pereira et al., 2014). Additionally,

the participant-related variables such as expectations and attention also play a role (e.g. Costa-Faidella, Baldeweg, et al., 2011; Hari et al., 1979; Herholz et al., 2009; Todorovic et al., 2011). While the present study did not manipulate some of these variables, it is worth noting that almost all of these factors remained constant throughout the experiment, minimising potential bias in the results. However, future studies could examine how these factors may modulate the MMN and adaptation findings.

A limitation of the present study is that the contribution of the N1 initial adaptation to MMN might be overestimated. Previous research has indicated that pitch differences between standards and deviants can result in contamination of the MMN by N1 response recovery, attributed to the frequency specificity of some N1 generators (Butler, 1968, 1972). Therefore, the observed association between the initial N1 adaptation and MMN amplitude might be partly contaminated because of the overlapping time window of these two components. A related issue is the overlapping activity between the P2 component in the deviant condition and the P3a component in the difference wave. While P3a is not the focus of the present study, it was elicited because of the different amplitudes and latencies of the P2 component between the deviant and final tone conditions (Figure 4a). Although the time range of the P2 component in the deviant condition differs from that of the MMN, our main focus is on the relationships between adaptation effects and MMN, which do not necessarily have to occur within the same time ranges. While the present study sheds light on the contributions of N1 initial adaptation and P2 subsequent adaptation to MMN, future studies should consider adopting more sophisticated experimental designs and analysis methods to obtain a more precise estimate of the relationships between adaptations and MMN.

Another limitation is that we only examined frequency differences, leaving it unclear whether the observed adaptation patterns and the relationship between adaptation effects and MMN can be generalised to deviants with other features, such as intensity, duration and abstract pattern. However, based on previous findings, we would expect adaptation to play a less prominent role in MMN when the repetition rule is not involved (Carbajal & Malmierca, 2018). In extreme cases like sound omission, adaptation should not occur because silence does not activate new sensory neurons (Prete et al., 2022). A recent study found that the MMN response was elicited in cases of unexpected sound omission (Prete et al., 2022). This finding supports the predictive coding model, as it implies that the brain predicts not only sounds but also silence. Furthermore, future studies should consider including a control condition where the same tones as the standards in the experimental condition are presented but embedded within different tones to avoid adaptation. This control condition would help distinguish the adaptation and prediction error components (Carbajal & Malmierca, 2018). Overall, more research is needed to examine how the relationship between adaptation and MMN is modulated by different stimulus features, particularly when expectations are controlled.

5 | CONCLUSION

To conclude, the present study utilised a roving paradigm with controlled expectations and revealed diverse adaptation patterns within the N1 and P2 time windows. Both N1 initial adaptation and P2 subsequent adaptation (the RP) significantly contributed to MMN. Thus, while N1 initial adaptation plays a role in MMN, it alone cannot fully explain MMN as suggested by the adaptation hypothesis without considering the RP. Theoretically, our findings demonstrate that under a paradigm with expectations controlled, the precision of prediction remains relevant to adaptation effects reflected by N1 amplitude and RP. Practically, the distinct adaptation patterns in N1 and P2 components and their discrete relationships to MMN highlight the importance of isolating the two MMN processes represented by N1 and P2 in future studies investigating adaptation and MMN.

AUTHOR CONTRIBUTIONS

Conceptualisation: Brian W. L. Wong, Urs Maurer. *Methodology*: Brian W. L. Wong, Urs Maurer. *Investigation*: Brian W. L. Wong. *Software*: Brian W. L. Wong. *Formal analysis*: Brian W. L. Wong, Urs Maurer. *Writing—original draft preparation*: Brian W. L. Wong. *Writing—review and editing*: Brian W. L. Wong, Shuting Huo and Urs Maurer. *Funding acquisition*: Urs Maurer. *Resources*: Urs Maurer. *Supervision*: Urs Maurer.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/ejn.16546.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in OSF at https://doi.org/10.17605/OSF.IO/3XYFC

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