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ORIGINAL ARTICLE

Prepulse Inhibition of Auditory Cortical Responses in the Caudolateral Superior Temporal Gyrus in *Macaca mulatta*

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Abstract Prepulse inhibition (PPI) refers to a decreased response to a startling stimulus when another weaker stimulus precedes it. Most PPI studies have focused on the physiological startle reflex and fewer have reported the PPI of cortical responses. We recorded local field potentials (LFPs) in four monkeys and investigated whether the PPI of auditory cortical responses (alpha, beta, and gamma oscillations and evoked potentials) can be demonstrated in the caudolateral belt of the superior temporal gyrus (STGcb). We also investigated whether the presence of a conspecific, which draws attention away from the auditory stimuli, affects the PPI of auditory cortical responses. The PPI paradigm consisted of Pulse-only and Prepulse + Pulse trials that were presented randomly while the monkey was alone (ALONE) and while another monkey was present in the same room (ACCOMP). The LFPs to the Pulse were significantly suppressed by the Prepulse thus, demonstrating PPI of cortical responses in the STGcb. The PPI-related inhibition of the N1 amplitude of the evoked responses and cortical oscillations to the Pulse were not affected by the presence of a conspecific. In contrast, gamma oscillations and the amplitude of the N1 response to Pulse-only were suppressed in the ACCOMP condition compared to the ALONE condition. These findings demonstrate PPI in the monkey STGcb and suggest that the PPI of auditory cortical responses in the monkey STGcb is a pre-attentive inhibitory process that is independent of attentional modulation.

Keywords Prepulse inhibition · Superior temporal gyrus · Local field potential

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Introduction

Prepulse inhibition (PPI) is a neurophysiological phenomenon in which a weaker stimulus (prepulse) suppresses the reaction of an organism to a subsequent strong stimulus (pulse) [1]. During this process, the sensory information is forward-masked so that an individual can focus on the most salient aspects of the sensory environment [2, 3]. Most studies on PPI have focused on such physiological measures as the eye-blink reflex in humans and whole-body flinching in rodents, while only a few studies [4, 5] have shed light on the PPI of auditory cortical processing. PPI has also been used to investigate the biology of some neuropsychiatric disorders [2, 3, 6, 7]. Studies on humans using electroencephalography (EEG) suggest that several components of the auditory evoked potentials (P50, N1, P2, and P3) exhibit PPI [8]. The amplitude of the N1 response to the Pulse-only stimulus is correlated positively with both the N1 amplitude of the prepulse-evoked response and with the degree of PPI [4]. Also alpha-, theta-, and gamma-band oscillatory activity exhibits PPI in humans [5]. PPI of oscillations may reflect either reduced activity within the higher-order cortical areas or the cortical areas might receive already reduced input from the midbrain [9]. Due to the suggested clinical relevance of the PPI in neuropsychiatric disorders [2, 3, 6, 7], there is a need to further

investigate the neuronal mechanisms underlying the PPI of cortical responses. The startle reflex of the PPI has been extensively studied in rat models and, more recently, the neuronal mechanisms of PPI have been investigated in humans using magnetoencephalography (MEG) and EEG [4, 5, 8, 9]. Non-human primate models have been successfully used to study human brain functions and to model brain disorders [10, 11]. However, to the best of our knowledge, there are no earlier reports on PPI of cortical responses in non-human primates, although these animals might provide a valuable model in which to investigate the neuronal underpinnings and neurochemical background of PPI. The purpose of the present study was to determine whether the PPI of cortical responses can be established in a monkey model by recording intracortical responses to auditory stimuli with a PPI paradigm. The recordings were performed in non-anesthetized animals that were not trained to perform any tasks, a set-up that can also be applied to human subjects who are not able or willing to follow instructions.

The primary and secondary auditory cortices, along with higher-level cortical areas, have been suggested to be involved in attention and perception-dependent processes [12]. In primates, the primary auditory cortex in the superior temporal gyrus (STG) is surrounded by several interconnected areas, the belt and parabelt fields [13–15]. Cortical processing of auditory information in nonhuman primates is organized hierarchically in primary auditory, lateral belt, and parabelt cortices of the STG [15–17]. The caudolateral belt area in the STG (STGcb), posterior to the primary auditory cortex, is involved in the processing of auditory space and the localization of sounds [18–20]. Previous studies have shed light on the functions of the STGcb in monkeys [14, 21, 22]. This area receives multimodal sensory inputs [23], suggesting that the modulation of auditory processing by distraction from multiple sensory modalities probably occurs in the STGcb.

PPI is commonly considered to represent sensorimotor gating, which is a pre-attentional inhibitory process [24]. Physical, innate emotional, or cognitive states can modify PPI through activity in cortical and subcortical structures. PPI of the startle reflex increases when human participants are instructed to attend to the Prepulse [25]. Similarly, PPI of cortical processing can be modulated by directing attention to the Prepulse [25], or by drugs [26]. It has been shown that attention to the Prepulse or to the Pulse stimuli increases the PPI of cortical oscillations depending on the length of the Prepulse-Pulse interval [25].

In the current study, we recorded local field potentials (LFPs) in the STGcb in four monkeys when the monkey was alone (ALONE) and when it was accompanied by another monkey (ACCOMP). We hypothesized that the cortical responses to a strong auditory stimulus in the STGcb are suppressed by a preceding weaker auditory stimulus in a manner similar to the PPI of the startle reflex. We also hypothesized that the presence of a conspecific, which draws attention away from the auditory stimulation, suppresses auditory processing in the STGcb but does not affect the PPI of cortical responses, as PPI is considered to be a pre-attentive process [2–4].

Materials and Methods

Animals

Four adult male rhesus monkeys (*Macaca mulatta*, 7.2 kg–10.6 kg) participated in this study. The monkeys were selected from four different social male groups raised in the Kunming Primate Center of the Chinese Academy of Sciences. They had not had any contact with each other prior to the experiment and were thus unfamiliar with each other. They were housed individually in cages in different rooms.

All experiments were approved by the Internal Review Board at Kunming Institute of Zoology, Chinese Academy of Sciences, and all experimental procedures were in compliance with National Institutes of Health Guidelines for the Care and Use of Laboratory Animals (NIH Guidelines).

Surgical Preparation and Electrode Implantation

The electrode implants were stereotrodes, comprising two Teflon-coated platinum-iridium alloy wires (wire diameter, 50 μm) in a carrier silicon tube. The length of the two wires was unequal with a difference of ~1 mm at the tip. The tip impedance was 50 kΩ–100 kΩ at 10 Hz.

A magnetic resonance imaging (MRI) guided stereotaxic method developed in our laboratory [27] was used to accurately implant the stereotrodes. First, a pre-MRI surgery was performed on the monkey anesthetized with hydrochloric acidulated ketamine (10 mg/kg, i.m.) and maintained with sodium pentobarbital (20 mg/kg, i.m.). Rigid glass tubes were anchored stereotaxically on the skull of the monkey and were used as external markers in MRI (Fig. 1A). The tubes were filled with vitamin AD oil to provide a bright signal in the MR image and thereby to serve as reference points in both the MR image and the skull of the monkey to determine the implantation coordinates for the electrodes. After anchoring of the glass tubes, the monkey underwent structural MRI of the brain (GE Healthcare, Signa Excite Twinspeed 1.5 T, Chicago, IL) with the following parameters: slice thickness = 1 mm; spacing between slices = 1.3 mm; repetition time (TR) = 4000 ms; echo time (TE) = 99.96 ms.

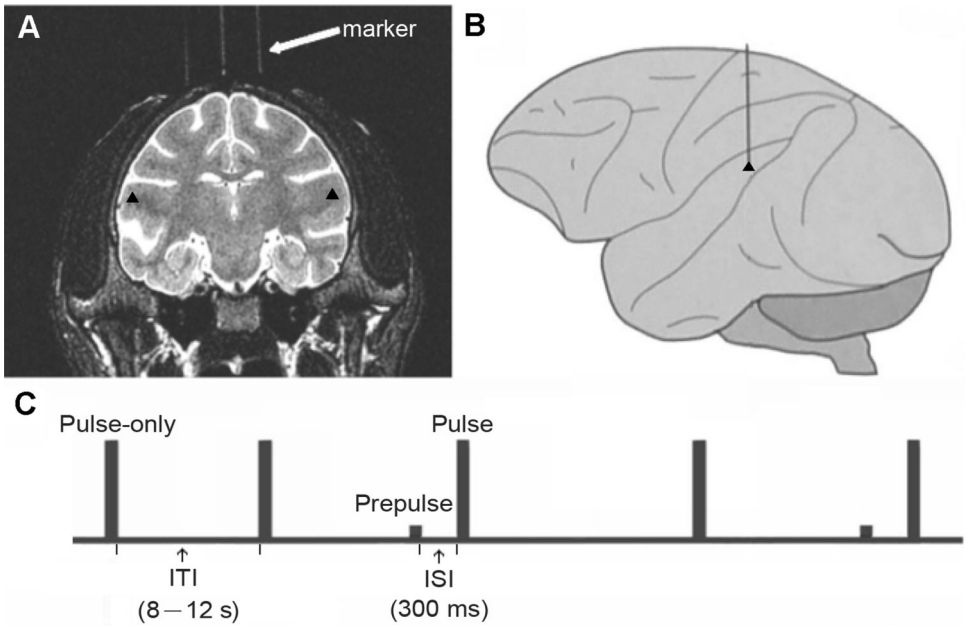

Fig. 1 **A** and **B** A coronary MRI slice from monkey M3 (**A**) and a schematic brain (**B**) showing the caudolateral belt of the STG (STGcb) recording sites (black triangles). In **A** the arrow indicates one of the external markers that were anchored stereotaxically on the skull of the monkey. **C** A schema of the presentation of the Pulse-only and Prepulse + Pulse stimuli. The amplitude of the Prepulse was 10% of that of the Pulse and Pulse-only. ITI, inter-trial interval; ISI, interstimulus interval.

The implantation coordinates of the target areas were calculated based on the stereotaxic locations of the tubes on the skull, on the location of the target area in the MR image, and on an anatomical atlas of the monkey brain [28]. Using this method, stereotrodes can be implanted successfully into brain targets with an error <1 mm [27] (Fig. 1A).

For the implantation surgery, the monkey was anesthetized as for the pre-MRI surgery. After fixing the head on the stereotaxic apparatus, the electrodes were implanted bilaterally into an area on the posterior surface of the superior temporal gyrus corresponding to the STGcb [29] for this study, and into 13 other brain areas for other studies according to the target coordinates that were calculated using the stereotaxic method described above (Fig. 1A, B). During the surgery, a chamber was anchored by screws and dental cement on the skull to fix the head to the primate chair during the electrophysiological recordings and connecting the stereotrodes to the recording equipment. Five additional screws implanted in the skull ~1 cm from the chamber, two screws to the left, two to the right side and one in the midline in front of the chamber, were connected by wires and used as a reference.

After a recovery period of about one month, the monkey was familiarized with the recording room and accustomed to sitting in a primate chair; this training took about one week.

During the LFP recordings, the monkeys sat quietly in the primate chair and listened to the auditory stimuli. In ACCOMP, the monkey was accompanied by another monkey, also sitting in a primate chair, but no vocal communication between the monkeys was recorded.

Auditory Stimulation

Auditory stimuli were presented from two loudspeakers (Edifier, Beijing, China) located on the left and right sides of the primate chair. The experiment was a PPI paradigm and consisted of Pulse-only trials and Prepulse + Pulse trials presented in random order. Both the Pulse and the Prepulse were tones with a frequency of 1000 Hz and duration of 10 ms. The interstimulus interval between the tones in the Prepulse + Pulse trial was 300 ms. The amplitude of the Pulse in the Prepulse + Pulse trials was the same as the amplitude in the Pulse-only trials. The amplitudes of the stimuli were set by the Psychtoolbox2 (http://psychtoolbox.org/) in MATLAB (Natick, MA) so that the amplitude of the Prepulse was 10% of the amplitude of the Pulse (Fig.1C). The volume of the loudspeakers was adjusted so that the Prepulse was easily audible at the level of the primate chair. The two types of trials were presented in a random order 50 times in one block. The interval between the trials varied randomly in the range of 8 s–12 s, in 1-s steps. The background noise in the recording room was ~45 dB. The peak intensity of the Prepulse was 70 dB SPL. The peak intensity of the Pulse-only and Pulse was 110 dB SPL measured at the location of the primate chair. The intracortical recordings were conducted under two conditions (ALONE and ACCOMP). In the ALONE condition, the monkey sat alone in the recording room and listened to the stimuli passively. In the ACCOMP condition, the monkey, while listening to the stimuli, was accompanied by another monkey (one of the three other animals in the study) that was sitting facing it in another primate chair at a distance of 2 meters. A video

camera monitored the monkeys' behavior and recorded any vocalizations. The second monkey was brought into the recording room a few minutes before the start of the recordings in the ACCOMP condition. During each day for 6 consecutive days, each monkey, except Monkey 1, completed one block either in the ALONE or in the ACCOMP condition so that in total three blocks were recorded in both conditions. The order of the six blocks was randomized. Monkey 1 completed 2 blocks (one block in ALONE and one block in ACCOMP) every day as the recordings were noisier and required the removal of more contaminated epochs than the data from other monkeys.

Data Collection and Analysis

Signals from the stereotrodes were amplified, bandpass filtered (0.01 Hz–120 Hz), and digitized (sampling frequency, 1000 Hz) using an amplifier (Symtop, Beijing, China) controlled by a program written by the staff of our lab. Data were saved for off-line analysis.

For data analysis, we used a custom MATLAB (Natick, MA) code and the FieldTrip toolbox [30]. To improve the signal-to-noise ratio, the signals were averaged from the two electrodes of one stereotrode. Trials contaminated by artifacts (eye or other movements and muscle artifacts) were first manually rejected using visual inspection. Then, a built-in function in FieldTrip toolbox (http://fieldtrip.Fcdonders.nl) was used to detect and reject artifacts automatically.

Data were segmented from −1000 ms to 1000 ms with respect to each stimulus onset for analysis of the event-related potentials and for spectral estimation. From the event-related potentials, we analyzed the N1 component to Pulse-only and to Pulse. The PPI of cortical evoked potentials was investigated for the N1 response amplitude [4] as the P1 and N2 responses to the Pulse were very weak. N1 was defined as the first negative deflection within a time window of 0 ms–70 ms after the tone onset (Pulse-only or Pulse). The amplitude of N1 was defined as the maximum absolute value within this time window. The amplitude was then normalized (Pulse-only: $Amp_{Pulse\text{-}only}$, Pulse: Amp_{Pulse}) by dividing the N1 amplitude by the standard deviation of the baseline values within the time window of −100 ms to 0 ms.

Time–frequency representations (TFRs) were computed using plain Morlet wavelets for lower frequencies (0.01 Hz–30 Hz) and multi-tapered wavelets for higher frequencies (30 Hz–120 Hz). The LFP power within the frequency range of interest across both conditions was normalized to the average power within that range in a 100-ms window before stimulus onset. Normalized alpha power was averaged over 9 Hz–14 Hz, beta power over 15 Hz–25 Hz [31] and gamma power over 30 Hz–120 Hz. The peak values of the normalized power of the alpha, beta, and gamma oscillations to Pulse-only ($Pow_{Pulse\text{-}only}$) or Pulse (Pow_{Pulse}) were used in statistical analysis.

PPI of Cortical Responses

To investigate whether the cortical responses to the Pulse were suppressed by the Prepulse, i.e. whether there was PPI of the cortical responses, we compared the N1 Amp_{Pulse} with the N1 $Amp_{Pulse\text{-}only}$ using two-way repeated-measures factorial ANOVA (hemisphere, stimulus). As explained above, the N1 amplitude was measured using the 0-baseline level from −100 ms to 0 ms. It is possible, however, that the N1 response to the Pulse (Amp_{Pulse}) was shifted relative to the 0-baseline level due to the late response to the Prepulse and, if so, the shift may have affected the calculation of PPI. We therefore also calculated the PPI using the relative N1 – P1 peak-to-peak amplitude to the Pulse, i.e. $|N1 - P1|_{Pulse}/|N1_{Pulse\text{-}only}|$, and to the Pulse-only, i.e. $|N1 - P1|_{Pulse\text{-}only}/|N1_{Pulse\text{-}only}|$. We then compared the relative N1 – P1 peak-to-peak amplitude to the Pulse with the corresponding amplitude to the Pulse-only using two-way (stimulus, hemisphere) repeated-measures factorial ANOVA.

To investigate whether the oscillations to the Pulse were suppressed by the Prepulse, we compared the $Pow_{Pulse\text{-}only}$ with the corresponding Pow_{Pulse} using two-way repeated-measures factorial ANOVA (hemisphere, stimulus) separately for the alpha, beta, and gamma oscillations.

Effect of Condition on PPI

To investigate whether condition (ALONE, ACCOMP) affected the PPI of cortical evoked potentials (N1), we calculated the percentage of PPI (%PPI) of N1 using the following formula: $\%PPI = 100\% \times (Amp_{Pulse\text{-}only} - Amp_{Pulse})/Amp_{Pulse\text{-}only}$ to test the effect of condition on evoked responses. The %PPI in ALONE and ACCOMP were compared using two-way (hemisphere, condition) repeated-measures factorial ANOVA.

In order to investigate whether condition (ALONE, ACCOMP) affected the PPI of the oscillations, we calculated the percentage of power change (%power change) using the following formula: $\%power\ change = 100\% \times (Pow_{Pulse\text{-}only} - Pow_{Pulse})/Pow_{Pulse\text{-}only}$, separately for the alpha, beta, and gamma oscillations in both the ALONE and ACCOMP conditions. The %power change in ALONE and ACCOMP were compared using two-way (hemisphere, condition) repeated-measures factorial ANOVA.

Effect of Condition on Pulse-only- and Pulse-Evoked Cortical Responses

To study the effect of condition on the N1 component of the evoked responses to Pulse and Pulse-only, the Amp_{Pulse} and $Amp_{Pulse\text{-}only}$ in the ALONE condition were compared to the corresponding values in the ACCOMP condition using two-way (hemisphere, condition) repeated-measures factorial ANOVA.

To study the effect of condition on the oscillations, we compared the $Pow_{Pulse\text{-}only}$ and Pow_{Pulse} of the alpha, beta, and gamma oscillations in the ALONE condition with the corresponding values in the ACCOMP condition and analyzed the results statistically using two-way (hemisphere, condition) repeated-measures factorial ANOVA.

In all statistical tests, $P < 0.05$ was considered a statistically significant result.

Results

PPI of Cortical Responses

The Prepulse suppressed the auditory evoked responses to the Pulse (Fig. 2A, B). The N1 Amp_{Pulse} was suppressed by the Prepulse, compared to the N1 $Amp_{Pulse\text{-}only}$ ($F_{(1,\ 3)} = 6.906$, $P = 0.039$). The control analysis confirmed the PPI by showing that the relative N1 – P1 peak-to-peak amplitude of the Pulse response, compared to the corresponding amplitude of the Pulse-alone, was suppressed by the Prepulse (Stimulus: $F_{(1,\ 3)} = 22.557$, $P = 0.003$) (Fig. 2C), thus confirming PPI.

The Pow_{Pulse} of gamma (Fig. 2D) and beta (Fig. 2E) oscillations were suppressed by the Prepulse compared to the $Pow_{Pulse\text{-}only}$ (gamma: $F_{(1,\ 3)} = 16.293$, $P = 0.027$; beta: $F_{(1,3)} = 14.853$, $P = 0.031$). The Pow_{Pulse} of alpha oscillations was not suppressed compared to $Pow_{Pulse\text{-}only}$ ($F_{(1,\ 3)} = 7.529$, $P = 0.071$).

Effect of Condition on PPI of Cortical Responses

The averaged evoked potentials to Prepulse + Pulse stimuli in both the ALONE and ACCOMP conditions are shown in Fig. 3A. Condition had no significant effect on the PPI of the cortical responses. Neither the %PPI of the N1 ($F_{(1,\ 3)} = 2.049$, $P = 0.248$) (Fig. 3B) nor the %power change of the gamma and beta power differed statistically between the two conditions (gamma: $F_{(1,\ 3)} = 0.769$, $P = 0.414$; beta: $F_{(1,\ 3)} = 0.890$, $P = 0.069$) (Fig. 3C, D).

There was no effect of hemisphere on cortical responses and oscillations (all P-values > 0.05).

Effect of Condition on Pulse-only- and Pulse-Evoked Cortical Responses

Condition (ALONE, ACCOMP) had a significant effect on the N1 amplitude to the Pulse-only as the $Amp_{Pulse\text{-}only}$ was lower in the ACCOMP than in the ALONE condition ($F_{(1,\ 3)} = 70.459$, $P = 0.004$) (Fig. 4). The differences in TRF between the conditions [(TFR in ACCOMP – TFR in ALONE)/TFR in ALONE], and the normalized power of the high-frequency (30 Hz–120 Hz) gamma responses to Pulse-only in the four monkeys are shown in Fig. 5. Two-way (hemisphere, condition) repeated-measures factorial ANOVA of $Pow_{Pulse\text{-}only}$ showed that the difference in the gamma-band power between the two conditions was significant ($F_{(1,\ 3)} = 30.188$, $P = 0.012$). Time–frequency analysis of the low-frequency oscillations (alpha 9 Hz–14 Hz; beta 15 Hz–25 Hz) showed that the difference of TFRs [(TFR in ACCOMP – TFR in ALONE)/TFR in ALONE] was not consistent in the four monkeys (Fig. 6). There was no difference between the two conditions in the alpha ($F_{(1,\ 3)} = 0.028$, $P = 0.878$) and beta range oscillations ($F_{(1,\ 3)} = 0.006$, $P = 0.945$).

Condition (ALONE, ACCOMP) had no effect on the N1 amplitude to the Pulse ($F_{(1,\ 3)} = 0.615$, $P = 0.490$).

Two-way ANOVA on Pow_{Pulse} of gamma oscillations showed no difference between the two conditions ($F_{(1,\ 3)} = 4.446$, $P = 0.126$) (Fig. 7). The results of time–frequency analysis of the low-frequency oscillations to Pulse showed that there was no difference between the two conditions in the alpha ($F_{(1,\ 3)} = 2.760$, $P = 0.195$) and beta ranges ($F_{(1,\ 3)} = 0$, $P = 0.994$) (Fig. 8).

There was no effect of hemisphere on cortical responses to Pulse-only and Pulse (all P-values > 0.05).

Discussion

Our results showed a significant PPI of auditory evoked responses in the STGcb. A weaker auditory tone (Prepulse) preceding a loud tone (Pulse) suppressed the evoked N1 response and gamma and beta oscillations to the latter stimulus. Furthermore, the presence of a conspecific diminished the N1 response and attenuated the gamma oscillations to the Pulse-only, but did not affect the PPI, suggesting that the PPI of the auditory cortical responses in the STGcb reflects a pre-attentive process.

In the STGcb, cortical responses to the Pulse were attenuated by the preceding weaker tone, Prepulse, in a manner similar to the PPI of the startle response. The N1 amplitude was significantly suppressed by the Prepulse, which is consistent with earlier human EEG/MEG studies [4, 8]. A cortical response peaking at $\sim$130 ms has been recorded in the human EEG/MEG when any change occurs

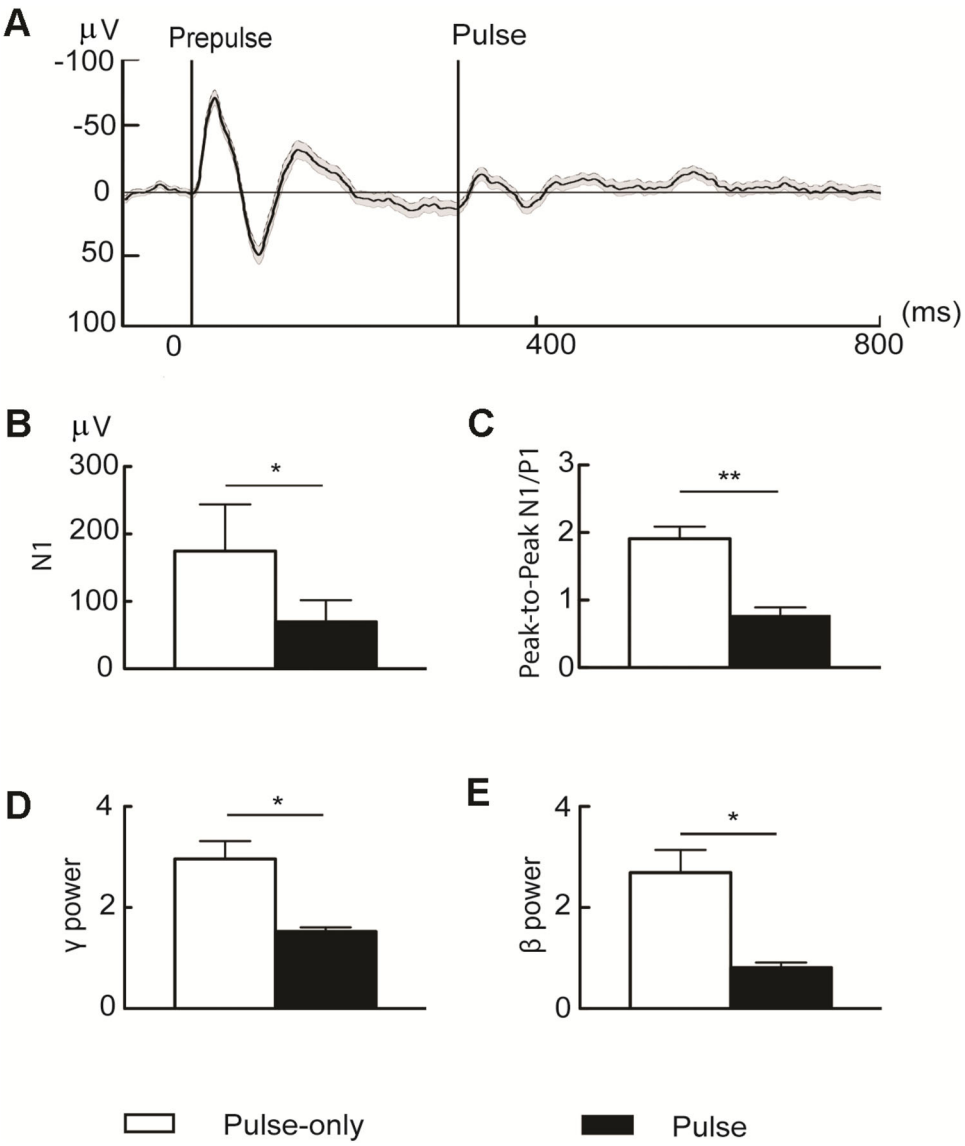

Fig. 2 Cortical responses to the Pulse were suppressed by the Prepulse. **A** Averaged evoked response to the Prepulse + Pulse stimuli in one monkey (M4). The shaded area shows the standard error of the mean (SEM) across all trials (n = 135). **B** N1 of the response to the Pulse (|N1|) was suppressed by the Prepulse compared to Pulse-only. **C** The relative N1 – P1 peak-to-peak amplitude (Peak-to-Peak N1/P1) of the Pulse response was suppressed by the Prepulse. **D** and **E** The normalized gamma (γ) (**D**) and beta (β) (**E**) power responses to the Pulse were significantly suppressed by the Prepulse compared to the corresponding normalized power responses to Pulse-only. *$P < 0.05$, **$P < 0.01$, repeated-measures factorial ANOVA; vertical bars, SEM.

in a train of sounds [4]. Therefore, the N1 amplitude is a useful indicator of the suppression in PPI. The PPI of cortical responses and that of the startle reflex may share some mechanisms/characteristics. The extent of PPI depends on the duration and intensity of the preceding stimulus [2, 4, 7].

In our study, the gamma responses to the Pulse were suppressed by the Prepulse. This finding is in line with previous studies in humans that also showed significant suppression of gamma oscillations by the Prepulse [5, 9]. The phase-locked, stable gamma oscillations occur at about 100 ms and 300 ms after sensory stimulation [32]. They are thought to play a role in perception, attention, memory, and language processing [33]. In the present study, suppression of gamma oscillations to the Pulse by the Prepulse occurred within 100 ms after the Pulse. The gamma oscillations occurring near 100 ms after auditory stimulation have been suggested to have a sensory origin, with a close relationship to the middle-latency auditory evoked response, and independent of the cognitive task [34, 35].

We also found that the beta oscillations to the Pulse were suppressed by the Prepulse. The beta-band oscillations have been suggested to mediate auditory sensory gating in humans [36, 37]. Phase-locked beta oscillations have a longer temporal delay than gamma oscillations and are associated with encoding and consolidating sensory information [38]. The beta oscillations, which index the neural process associated with the strength of sensory gating [36, 39], may be involved in the higher-level neural processing of sensory information and reflect the feedback to lower-order cortices [40, 41]. A recent study investigated the organization of interareal synchronization in the gamma- and beta-frequency bands in the primate visual system [42]. The authors suggested that the beta-frequency band might mediate

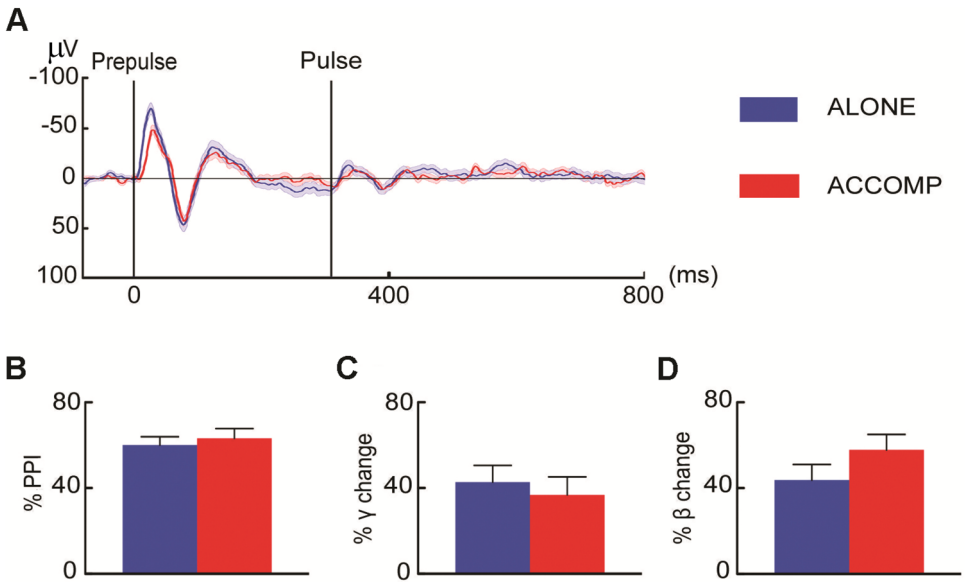

Fig. 3 The presence of a conspecific did not significantly affect the PPI of responses in the STGcb. **A** Averaged evoked potentials to Prepulse + Pulse in ALONE and ACCOMP conditions in one monkey (M4). Shaded areas, standard error of the mean across trials ($n = 135$ in ALONE, $n = 133$ in ACCOMP). **B–D** Percentage of PPI (%PPI) of the N1 amplitude (**B**), percentage of gamma power change (%γ change) (**C**), and percentage of beta power change (%β change) (**D**) in ALONE and ACCOMP conditions.

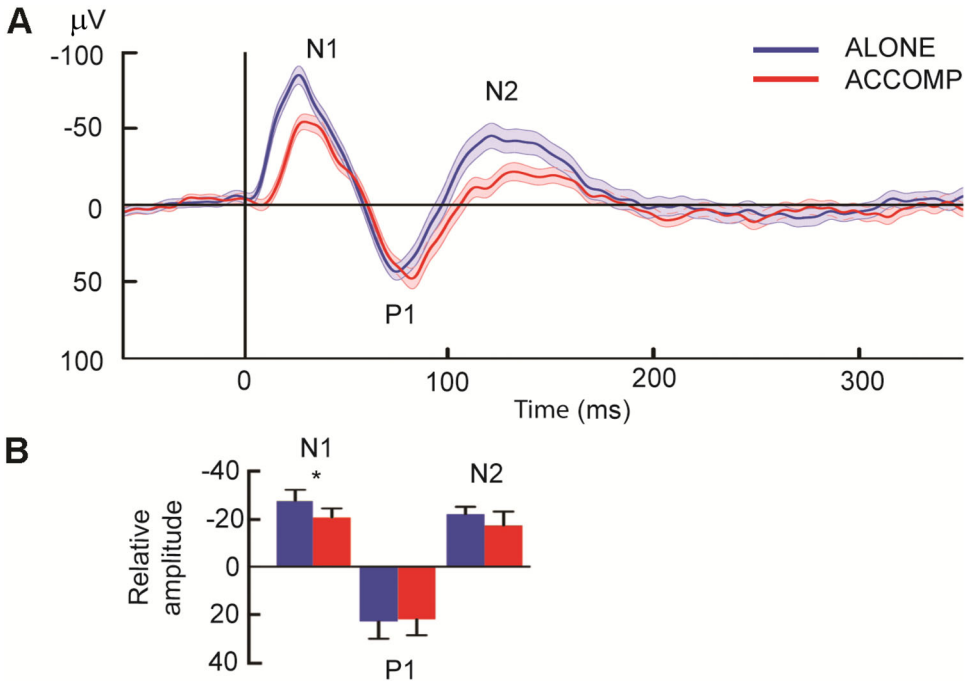

Fig. 4 The presence of a conspecific suppressed the N1 evoked response to Pulse-only in STGcb. **A** Averaged auditory evoked responses to Pulse-only in one monkey (M4). Shaded area, standard error of the mean (SEM) across all trials ($n = 155$ in ALONE, $n = 151$ in ACCOMP). **B** Relative amplitude of N1 in the STGcb (averaged over the four monkeys) was lower in the ACCOMP condition than in the ALONE condition. $^*P < 0.05$; vertical bars, SEM.

feedback influences to lower-level visual areas and thus be involved in top-down regulation of cognitive processing, whereas the gamma-frequency band might mediate feedforward influences from lower to higher areas.

We did not find PPI in the alpha-band oscillations. An earlier study on humans using EEG, however, reported that the alpha-band power exhibits PPI in cortical responses at the central and temporal recording locations [5]. Some differences between these two studies (other than the species difference) may explain the discrepant results in the alpha band oscillations. In our study, the Prepulse – Pulse interval was 300 ms, whereas Kedzior *et al*. studied shorter intervals that ranged from 0 ms to 240 ms [5]. Furthermore, our recordings were restricted to the STGcb area, suggesting that this area is not the origin of the alpha band modulation reported by Kedzior *et al*. [5].

The decreased cortical response to the Pulse when it is preceded by a Prepulse has been explained by neural mechanisms related to short-term plasticity [43–45]. It has been suggested that the diminished response to the Pulse is due to a decrease in the release probability of excitatory neurotransmitters from afferent axon terminals and to the release of gamma-aminobutyric acid (GABA) from the terminals of inhibitory interneurons [46–51]. Previous

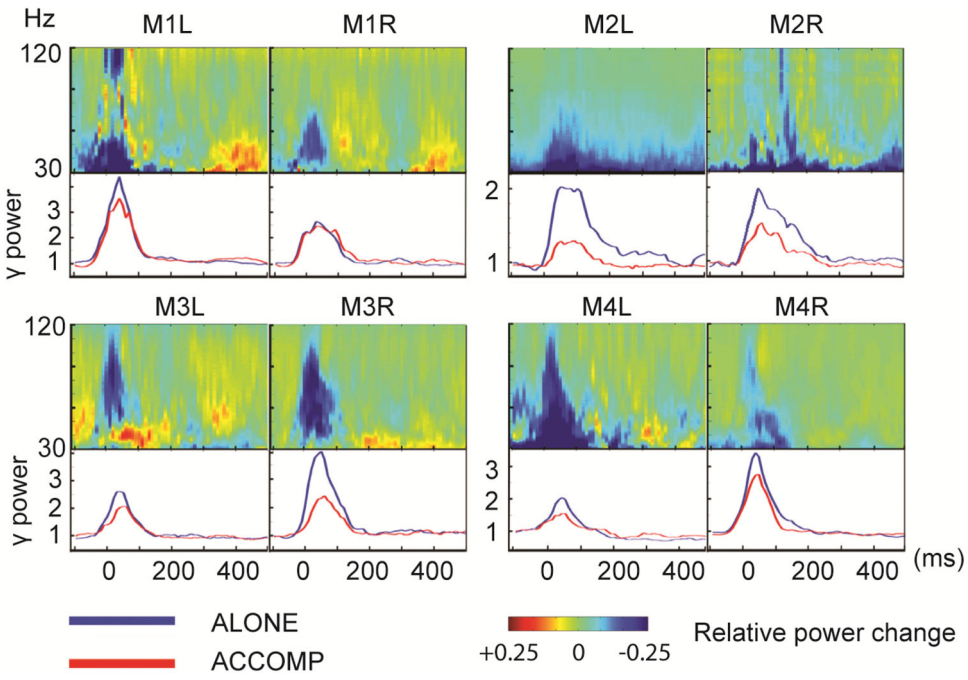

Fig. 5 The presence of a conspecific decreased high-frequency (30 Hz–120 Hz) gamma (γ) oscillations to the Pulse-only in bilateral STGcb. The relative change of the time-frequency representations between the conditions (ACCOMP – ALONE)/ALONE (upper graphs in each panel), and the normalized gamma power in the two conditions (lower graphs in each panel) are shown for the four monkeys. M, monkey; L, left; R, right.

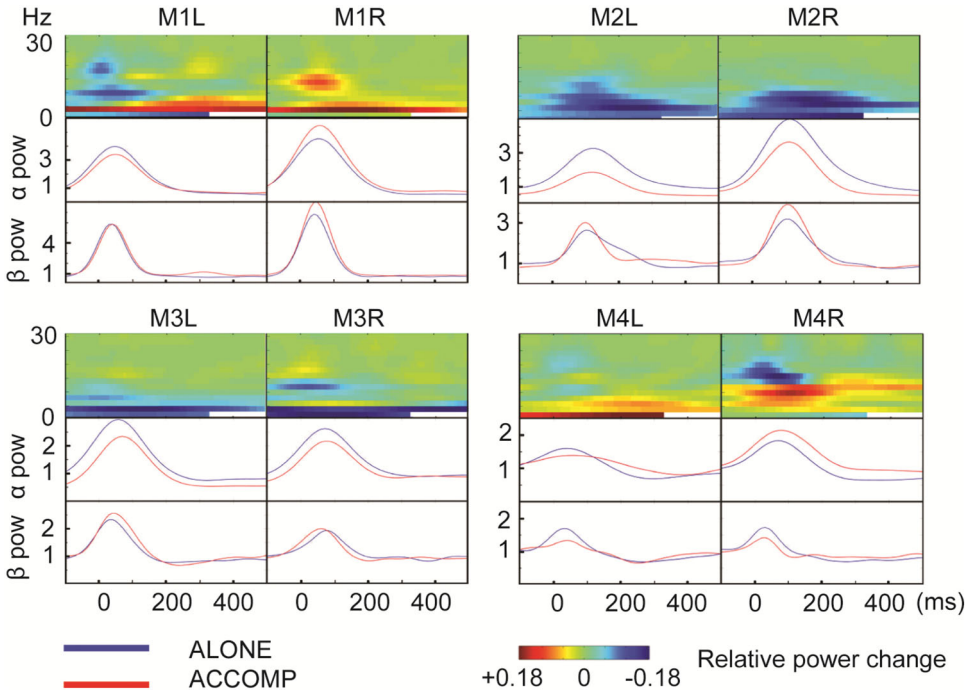

Fig. 6 The presence of a conspecific did not significantly affect the low-frequency (0.1 Hz–30 Hz) alpha (α) and beta (β) oscillations to the Pulse-only in bilateral STGcb. The relative change of the time-frequency representations between the two conditions (ACCOMP – ALONE)/ALONE (upper graphs in each panel), normalized alpha power (middle graphs in each panel) and normalized beta power (lower graphs in each panel) in the two conditions are shown for the four monkeys. M, monkey; L, left; R, right.

studies have shown that both the release of excitatory neurotransmitters and GABA release are correlated with the strength of the LFP [52–54].

The presence of a conspecific as an exogenous distraction in the environment did not affect the PPI of the cortical responses (N1 response and gamma and beta oscillations). In earlier studies on PPI in humans, selective attention to the Prepulse or Pulse was shown to increase the amplitude of the startle response [55], suggesting that attention modulates the PPI of the startle response. In the current study, we assumed that the presence of a conspecific would draw attention away from the auditory stimulation towards the other monkey. This assumption was correct as the responses to the auditory stimulus (Pulse-only) in ACCOMP were suppressed compared to those recorded while the monkey was alone. However, the presence of another monkey did not affect the PPI of the cortical responses to the Pulse stimulus. Unless the Prepulse always

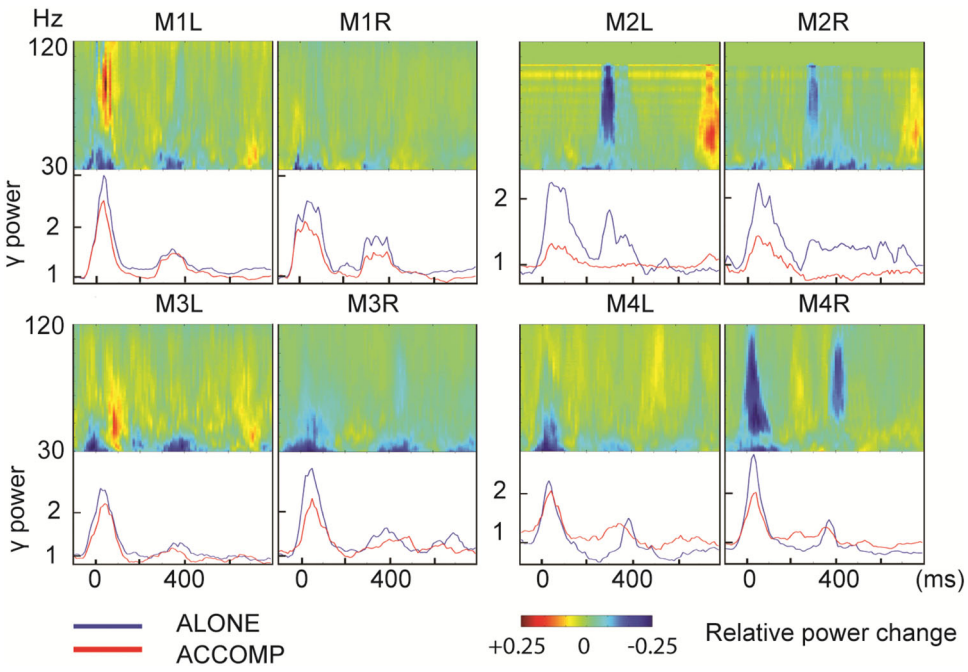

Fig. 7 The presence of a conspecific did not significantly affect the high-frequency (30 Hz–120 Hz) gamma (γ) power responses to the Pulse in the STGcb. The relative change of the time–frequency representations between conditions (ACCOMP – ALONE)/ALONE (upper graphs in each panel), and the normalized gamma power in the two conditions (lower graphs in each panel) are shown for the four monkeys. M, monkey; L, left; R, right.

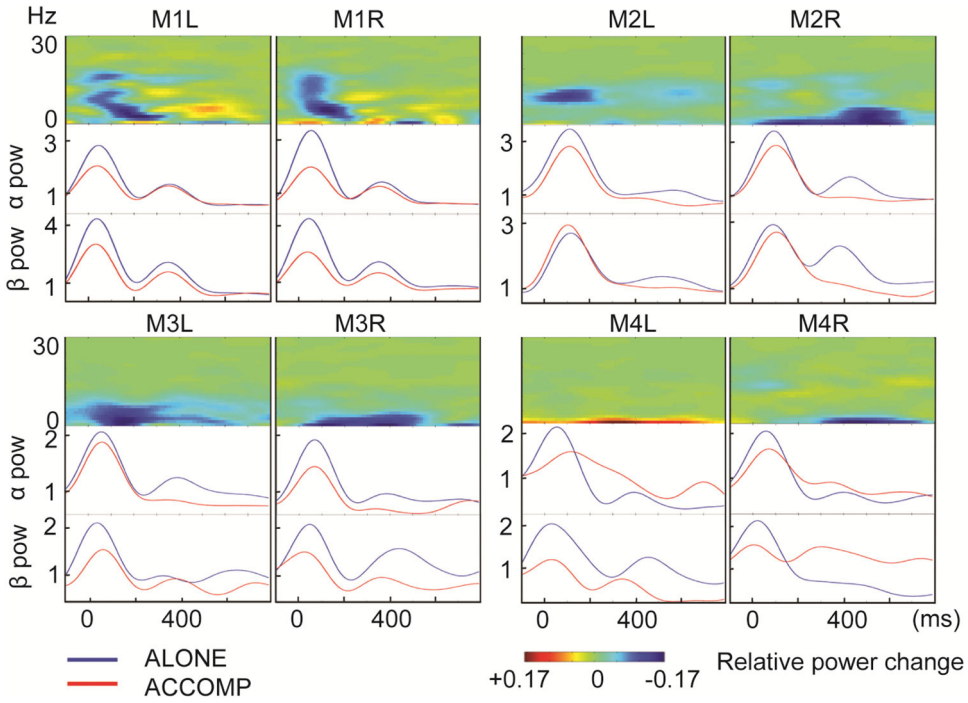

Fig. 8 The presence of a conspecific did not significantly affect the low-frequency (0.1 Hz–30 Hz) alpha (α) and beta (β) power responses to Pulse in the STGcb. The relative change of the time-frequency representations between the two conditions (ACCOMP – ALONE)/ALONE (upper graphs in each panel), normalized alpha power (middle graphs in each panel), and normalized beta power (lower graphs in each panel) in the two conditions are shown for the four monkeys. M, monkey; L, left; R, right.

captured attention, even in the presence of an attention-attracting conspecific, this finding supports the suggestion that, in monkeys, the PPI is an early pre-attentional process [3, 4] independent of exogenous attentional modulation.

Although the PPI of cortical responses was not affected by the presence of a conspecific, the response to the Pulse-only stimulus was significantly suppressed in the ACCOMP condition. When the monkey was facing another monkey sitting in a primate chair, the gamma activity in the STGcb in response to the Pulse-only stimulus was suppressed with respect to the stimulus onset. Several earlier studies have suggested that gamma oscillations reflect neural activity related to sensory processing [56–58], attention, and memory [59]. Attended stimuli trigger stronger gamma-band responses than unattended stimuli [60, 61], and the amplification of gamma-band activity by attention is not unique to unimodal perception [62]. The finding in the current study that the presence of a conspecific suppressed the gamma

oscillations in the STGcb likely reflects the top-down attentional modulation of STGcb activity. The results of the time-frequency analysis of low-frequency (alpha and beta) oscillations showed that the difference in power between the two conditions (ALONE and ACCOMP) was not significant. The properties of the low-frequency oscillations depend on the cortical area and the cortical layer from where they are recorded, and on the stimulus modality used in the experiment [63]. Auditory-evoked alpha oscillations in the auditory pathways and other brain areas are related to the activity in thalamo-cortical circuits [33, 64, 65]. Elevated beta oscillatory activity has been found during the processing of novel auditory stimuli [36, 37]. However, in the current study, neither alpha nor beta oscillations were affected by the presence of a conspecific.

The relative amplitude of the N1 component of the responses evoked by the Pulse-alone stimulus also decreased significantly in the ACCOMP compared to the ALONE condition among the four monkeys. In an earlier study, the N1 component in response to auditory stimuli in a passive listening condition was shown to be significantly smaller than in an active listening condition [66]. The N1-suppression effect could be caused by a switch of attention away from the task-irrelevant auditory stimulation [67, 68]. Some studies have shown that an increase in the LFP amplitude is correlated with neurons becoming synchronously entrained to take part in cooperative network activity [69–72]. A possible mechanism explaining the suppression of gamma oscillation and the N1 amplitude to the Pulse-alone in the ACCOMP, compared to the ALONE, is that auditory processing is suppressed by inhibitory postsynaptic inputs, and thus the number of neurons participating synchronously in the network activity is reduced [71, 72].

The present study showed that a weak auditory Prepulse suppressed the N1 amplitude of the LFPs and attenuated the gamma and beta oscillations in the STGcb in response to a strong Pulse. The results suggested that the PPI of cortical responses recorded in the monkey STGcb is independent of attentional modulation, since the presence of a conspecific did not affect the PPI.

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