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Research Report

Sensory attenuation in the absence of movement: Differentiating motor action from sense of agency

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ABSTRACT

Sensory attenuation is the phenomenon that stimuli generated by willed motor actions elicit a smaller neurophysiological response than those generated by external sources. It has mostly been investigated in the auditory domain, by comparing ERPs evoked by self-initiated (*active* condition) and externally-generated (*passive* condition) sounds. The mechanistic basis of sensory attenuation has been argued to involve a duplicate of the motor command being used to predict sensory consequences of self-generated movements. An alternative possibility is that the effect is driven by between-condition differences in participants' sense of agency over the sound. In this paper, we disambiguated the effects of motor-action and sense of agency on sensory attenuation with a novel experimental paradigm. In Experiment 1, participants watched a moving, marked tickertape while EEG was recorded. In the *active* condition, participants chose whether to press a button by a certain mark on the tickertape. If a button-press had not occurred by the mark, then a tone would be played 1 s later. If the button was pressed prior to the mark, the tone was not played. In the *passive* condition, participants passively watched the animation, and were informed about whether a tone would be played on each trial. The design for Experiment 2 was identical, except that the contingencies were reversed (i.e., a button-press by the mark led to a tone). The results were consistent across the two experiments: while there were no differences in N1 amplitude between the *active* and *passive* conditions, the amplitude of the Tb component was suppressed in the *active* condition. The amplitude of the P2 component was enhanced in the *active* condition in both Experiments 1 and 2. These results suggest that motor-actions and sense of agency have differential effects on sensory attenuation to sounds and are indexed with different ERP components.

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1. Introduction

Sensory attenuation is the phenomenon that self-generated sensations feel less salient, and evoke a smaller neurophysiological response, than externally generated sensations, even when the evoking stimuli are physically identical (Hughes, Desantis, & Waszak, 2013). The neurophysiological aspect of sensory attenuation has been most commonly investigated in the auditory domain, by using EEG/MEG to compare the evoked response to self-initiated and externally-initiated sounds (Horváth, 2015; Schafer & Marcus, 1973). There is substantial evidence that certain components of the auditory-evoked potential are reduced in amplitude when participants listen to sounds initiated by their own motor actions, compared to when they passively listen to the same sound. This effect has been most consistently observed with the N1 component (Bäß, Jacobsen, & Schröger, 2008; Elijah, Le Pelley, & Whitford, 2018; Mifsud et al., 2016; Neszmélyi & Horváth, 2017; Oestreich et al., 2016; Pinheiro, Schwartze, Gutierrez, & Kotz, 2019; van Elk, Salomon, Kannape, & Blanke, 2014), but has also been identified with the Tb (SanMiguel, Todd, & Schröger, 2013; Saupe, Widmann, Trujillo-Barreto, & Schröger, 2013) and P2 components (Knolle, Schröger, Baess, & Kotz, 2012; Horváth & Burgyán, 2013).

The mechanistic basis of sensory attenuation has been argued to involve an internal forward model in which the brain uses a copy of the outgoing motor command ('efference copy') to make predictions ('corollary discharges') about the expected sensory consequences of self-initiated movements (Miall & Wolpert, 1996). Sensory attenuation has been conceptualized as a specific example of predictive coding, in which sensory predictions and sensory feedback are compared, and observed deviations (i.e., prediction errors) are used to update and improve the sensory predictions (Crapse & Sommer, 2008; Poulet & Hedwig, 2007; Schütz-Bosbach & Prinz, 2007; Straka, Simmers, & Chagnaud, 2018; Subramanian, Alers, & Sommer, 2019).

Sensory attenuation has often been assumed to result from the comparison between sensory predictions and sensory feedback in the internal forward model. This implies that sensory attenuation is dependent on the presence of the motor action by which the sensory predictions are generated (Bäß et al., 2008; Hughes et al., 2013). However, an alternative possibility is that the effect is driven by participants' sense of agency in the self-initiation condition. Sense of agency refers to *"the feeling of control over actions and their [sensory] consequences"* (Moore, 2016), or "*the experience of controlling one's own motor acts and, through them, the course of external events*" (Haggard, 2017). In a typical self-stimulation paradigm, the *active* condition consists of the participant repeatedly performing a motor action (e.g., a button-press) to elicit a sequence of sounds. In the *passive* condition, the same sequence of sounds is presented without the participant having to perform any motor action. A consequence of this design is the participant has control over the sounds in the *active* condition but not the *passive* condition. This raises an important question: to what extent is the sensory attenuation effect driven by the between-condition differences in sense of agency as opposed to the presence of the motor action *per se*? Rather than merely being a byproduct of comparator processes, as has been suggested (Synofzik, Vosgerau, & Newen, 2008), sense of agency may instead have a pivotal causal role in sensory attenuation.

Most previous studies of sensory attenuation have conflated participants' sense of agency with the motor action as the two co-occur in a typical self-stimulation paradigm. We attempted to dissociate these two factors by means of a novel experimental paradigm. In our paradigm, participants had to decide, on every trial, as to whether or not to press a button. This decision determined whether a sound would subsequently be presented after a significant delay. In Experiment 1, sounds were only played on trials in which participants did *not* press the button. In other words, participants had control over whether and when they heard the sounds, but their sense of agency did not result from a motor action. Sounds in these *active* blocks were compared to sounds presented in *passive* blocks, where participants did not perform actions on any trials. If the N1, Tb and/or P2 components are associated with a participant's sense of agency over the sounds – independent of the presence of a motor action – then this would manifest as differences in component amplitude between the *active* and *passive* blocks.

2. Method

2.1. *Experiment 1*

2.1.1. *Participants*

Forty-four undergraduate students from the University of New South Wales (Sydney, Australia) participated in the study in exchange for course credit. All participants gave written informed consent prior to the experiment. Two participants were removed from analysis due to insufficient number of artifact-free epochs (as described in EEG Recording and Analysis) leaving a final sample of $N = 42$ participants (mean age = 22 years, SD = 4.3, 21 females). Given our sample size of $N = 42$, this study could detect an effect size of $\eta_p^2 = .15$ at power = 80% with alpha = .05. The study was approved by the Human Research Ethics Advisory Panel (Psychology) at the University of New South Wales.

2.1.2. *Stimuli, materials, and procedure*

The audio stimulus was a sinusoid tone of frequency 1000 Hz (100 ms duration, with a 5 ms rise/fall time). Audio stimuli were sent to participants through Sennheiser HD 210 headphones. Audio input/output was controlled by a specially written MATLAB script using the Psychophysics Toolbox (Brainard, 1997). Participants made responses by pressing the space bar of a low-latency keyboard (DuckyShine 4, 1000 Hz report rate). Visual stimuli were displayed on a high-performance monitor (24-inch, BenQ XL2420T).

During each trial, participants observed a visual animation, which was adapted from the paradigm employed by Whitford et al. (2017) and Jack et al. (2019). The animation lasted for about 6 s. A schematic of the animation is presented in Fig. 1. The animation consisted of a central red fixation line that sat in the middle of a green horizontal bar, which we refer to as the 'ticker tape'. Participants were instructed to keep their eyes fixated on

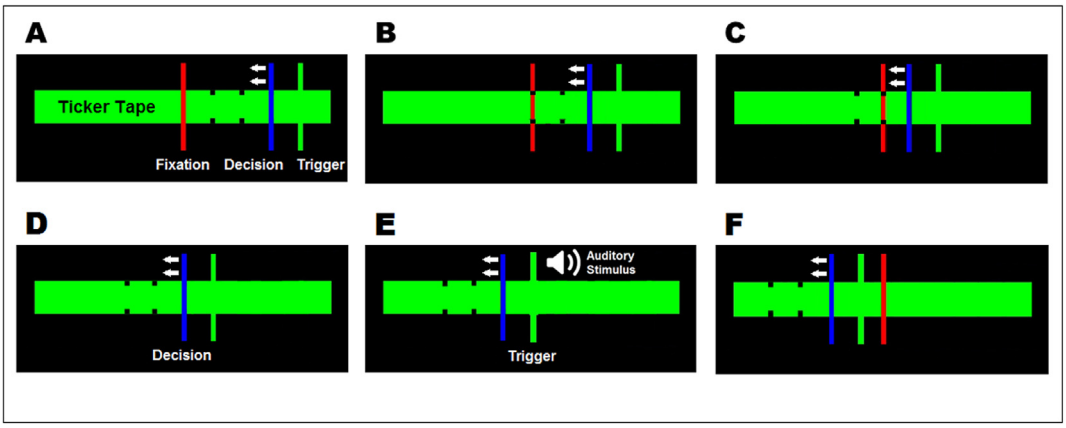

Fig. 1 – A schematic of the experimental protocol. In the *active* condition, participants were instructed to fixate their eyes on the central red fixation line (*Panel A*). After a 1 s delay, the blue decision line and the green trigger line moved slowly towards the central red fixation line at a rate of 6.5° per second (*Panel B–C*). On each trial, participants were told that they had to decide whether or not to press the space bar on the keyboard by the decision time (i.e., the time at which the decision line intersected the fixation line) (*Panel D*). In Experiment 1, participants were told that if they did not press the space bar by the decision time, this would cause the audio stimulus to be played at trigger time (i.e., the time at which the trigger line intersected the fixation line) (*Panel E*). Conversely, participants were told that if they did press the space bar by decision time, the audio stimulus would not play at the trigger time. In Experiment 2, this contingency was reversed. That is, if the participant did not press the space bar before the decision time, the audio stimulus was not played at the trigger time; conversely, if the participant did press the space bar before the decision time, this caused the audio stimulus to be played at the trigger time. In the *passive* condition of both experiments, participants observed the same animation but did not perform any motor actions. The audio stimulus was played on exactly half of the trials in the *passive* condition. Participants were informed at the start of each trial whether or not the audio stimulus would be played. *Passive* conditions were identical across both experiments.

the fixation line during the trial. There was also a blue 'decision line' and a green 'trigger line' located on the right side of the ticker tape. The trigger line was initially positioned on the far right hand side of the ticker tape; the decision line was positioned to the left of the trigger line (Fig. 1A).

Upon commencement of the trial, after a 1 sec delay, both the decision line and the trigger line started to move towards the fixation line at a constant rate of 6.5° per second. Approximately 3 sec after the lines started moving, the decision line intersected the fixation line. The trigger line intersected the fixation line 1 s later, i.e., approximately 4 sec after the lines initially started moving. The auditory stimulus was presented when the trigger line intersected with the fixation line (see Fig. 1). The lines continued to move for another 1 sec, before the animation concluded and the trial was completed.

There were two conditions in the experiment: the *active* condition and the *passive* condition. In the *active* condition, participants had the option of pressing the space bar on the keyboard at any time up until the point at which the decision line intersected the fixation line (hereon referred to as the 'decision time'). Participants were told that if they *did not* press the button by the decision time (Fig. 1D), this would cause the audio stimulus to be played at the point at which the trigger line intersected the fixation line (hereon referred to as the 'trigger time') (Fig. 1E). In contrast, the participant was told that if they *did* press the button before the decision time, the audio stimulus would not be played. In other words, the participant had complete control over whether they heard the audio stimulus on any given trial. Participants were asked to press the button on approximately half the trials, and to avoid conforming to any obvious pattern of responses. At the start of every trial, participants were reminded (by means of instructions on the screen) as to what their options were and what the consequences of those options were.

In the *passive* condition, participants watched the same animation as in the *active* condition but were not required to perform any action. The auditory stimulus was presented on 50% of trials (randomly selected) at the trigger time. At the start of every trial, participants were informed (by means of instructions on the screen) as to whether the audio stimulus would be played on that trial.

The experiment consisted of four *active* blocks and *four passive* blocks,[1] totaling eight blocks for the whole experiment.

[1] Although the conditions in current study are called the *active* and *passive* conditions, they differ significantly from those of a typical self-stimulation paradigm. In a typical self-stimulation paradigm (Schafer & Marcus, 1973), the *active* condition consists of a participant repeatedly performing a motor action (e.g., a button-press) in order to elicit a sequence of tones. In the *passive* condition, the same sequence of tones is presented without the participant having to perform any action. In the typical self-stimulation paradigm, tones immediately followed the button-press in the *active* condition. The *active* and *passive* conditions of the current study differ in that tones are not time-locked to a participant's decision to elicit the tone. Additionally, tones in the *active* and *passive* conditions were both triggered at exactly the same point of the animation (i.e., at the 'trigger time'). We retained the *active* and *passive* condition names given that the *active* condition of both the traditional and current experiments require participants' control while sounds in both *passive* conditions are out of the control of participants.

Each block consisted of 30 trials. The order of the blocks alternated between *active* and *passive* blocks, and the starting block was counterbalanced between participants.

2.1.3. EEG recording and analysis

EEG was recorded with a BioSemi ActiveTwo system from 64 Ag/EgCl active electrodes (P1, FPz, FP2, AF7, AF3, AFz, AF4, AF8, F7, F5,F3, F1, Fz, F2, F4, F6, F8, FT7, FC5, FC3, FC1, FCz, FC2, FC4, FC6, FT8,T7, C5, C3, C1, Cz, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPz, CP2, CP4,CP6, TP8, P9, P7, P5, P3, P1, Pz, P2, P4, P6, P8, P10, PO7, PO3, POz,PO4, PO8, O1, Oz, O2, Iz). In the BioSemi ActiveTwo system, the 'ground' electrode is replaced with two separate electrodes – an 'active' CMS (Common Mode Sense) electrode, and a 'passive' DRL (Driven Right Leg) electrode. These electrodes are arranged in a feedback loop which drives the average potential of the participant (i.e., the Common Mode voltage) as close as possible to the reference voltage (i.e., the 'zero') of the amplifier. See www.biosemi.com for more details. A vertical electrooculogram (EOG) was recorded by placing an electrode above and below the left eye; a horizontal EOG was recorded by placing an electrode on the outer canthus of each eye. Electrodes were also placed on each mastoid, and the nose. During data acquisition, the reference was composed of sites CMS and DRL, and the sampling rate was 2,048 Hz.

For data analyses, we re-referenced the EEG data offline to the nose electrode, as is common in studies investigating the components-of-interest, and necessary for extracting the Tb component (Näätänen & Picton, 1987; SanMiguel et al., 2013; Woods, 1995). Data were band-pass filtered from .1 to 30 Hz using a half-amplitude phase-shift-free Butterworth filter, then notch filtered (50 Hz) to remove mains artefact. The filtered data were segmented into 500 ms epochs, from −100 ms pre-stimulus to 400 ms post-stimulus. Only trials in which the auditory stimulus were played were analyzed. Epochs were baseline-corrected to the mean voltage from −100 to 0 ms. We corrected the epochs for eye blinks and movement artefacts using the technique described in Gratton, Coles, and Donchin (1983) and Miller, Gratton, and Yee (1988). We excluded all epochs with signals exceeding peak-to-peak amplitudes of 200 uV and had a maximal allowed voltage step of 50 uV/ms. We analysed the amplitude of the N1, Tb and P2 components of the auditory-evoked potential. Component amplitude was calculated as the average voltage within 30 ms time-window, the center of which was defined using the collapsed localiser approach (Luck & Gaspelin, 2017). The collapsed localiser approach is a technique whereby one first averages (or 'collapses') the ERP waveforms across all conditions for all participants. The components-of-interest (e.g., N1, Tb, P2) are identified on this 'collapsed' waveform, and a time-window is centred around these peaks, which is then used for the statistical analysis of the original (or 'uncollapsed') waveforms (Luck & Gaspelin, 2017).

For the N1 and P2 components, mean voltage in the analysis window was submitted to a 2 (Condition: *active, passive*) x 9 (Electrode) repeated-measures ANOVA. Electrodes of interest for the N1 component were the Fz, F1, F2, FCz, FC1, FC2, Cz, C1, and C2 electrodes. The electrodes of interest for the P2 component were the FCz FC1, FC2, Cz, C1, C2, CPz, CP1, and CP2 electrodes. The mean voltage of the Tb component in the analysis window was submitted to a 2 (Condition: *active, passive*) x 2 (Electrode: T7, T8) repeated-measures ANOVA. Electrodes for the Tb component (T7 and T8) were based on recommendations by Tonnquist-Uhlen et al. (2013) and SanMiguel et al. (2013).

2.1.4. Experiment 1 results

There was an average of 55.9 (SD = 11.6) useable epochs in the *active* condition and 53.6 (SD = 9.8) in the *passive* condition. If a participant pressed the button on exactly half the trials in the *active* condition, and if all epochs were artefact-free, there would be 60 useable epochs in the *active* condition. Similarly, if participants followed instructions perfectly, and if all epochs were artefact-free, there would be 60 useable epochs in the *passive* condition. Choice frequencies (i.e., the proportion of trials in which participants heard the sound) were obtained and compared across the active and passive conditions. Participants' choice frequencies were 62.29 sound trials (SD = 5.85) in the active condition and 59.31 sound trials (SD = 2.45) in the passive condition. The chi-square test yielded χ^2 (41, N = 42) = 22.55, p = .991.

2.1.4.1. N1. Fig. 2A shows the N1 component analysis elicited in the *active* and *passive* conditions. N1 was maximal at electrode FCz for both conditions and showed the expected fronto-central topography. The time-window for the N1 analysis was centered at 89.8 ms and extended from 74.8 to 104.8 ms. The main effect of Condition was not statistically significant, $F(1, 41) = .47$, $p = .497$, $\eta_p^2 = .01$. Similarly, the *Condition*×*Electrode* interaction was not statistically significant, $F(1, 41) = 1.26$, $p = .266$, $\eta_p^2 = .03$. The results indicate that N1 amplitude was not significantly different between the *active* and *passive* conditions.

2.1.4.2. Tb. Fig. 2B shows the Tb component analysis elicited in the *active* and *passive* conditions. Tb was maximal at electrodes T7 and T8 for both conditions and showed the expected temporal-lobe topography. The time-window for the Tb analysis was centered at 124.5 ms and extended from 109.5 to 139.5 ms. The repeated measures ANOVA yielded a significant main effect of *Condition*, $F(1, 41) = 4.74$, $p = .035$, $\eta_p^2 = .10$. The *Condition* × *Electrode* interaction was not significant, $F(1, 41) = 1.39$, $p = .246$, $\eta_p^2 = .03$. The results suggest that the Tb amplitude of the *active* condition was suppressed relative to the amplitude in the *passive* condition, and the effect was not driven by any one electrode.

2.1.4.3. P2. Fig. 2C shows the P2 component analysis elicited in the *active* and *passive* conditions. P2 was maximal at electrode Cz for both conditions and showed the expected central topography. The time-window for the P2 analysis was centered at 182.6 ms and extended from 167.6 to 197.6 ms. The main effect of Condition was significant, $F(1, 41) = 10.30$, $p = .003$, $\eta_p^2 = .20$. The *Condition*×*Electrode* interaction was not significant, $F(1, 41) = .42$, $p = .907$, $\eta_p^2 = .01$. The results suggest that the P2 amplitude of the active condition was enhanced relative to the amplitude in the passive condition, and the effect was not driven by any individual electrode.

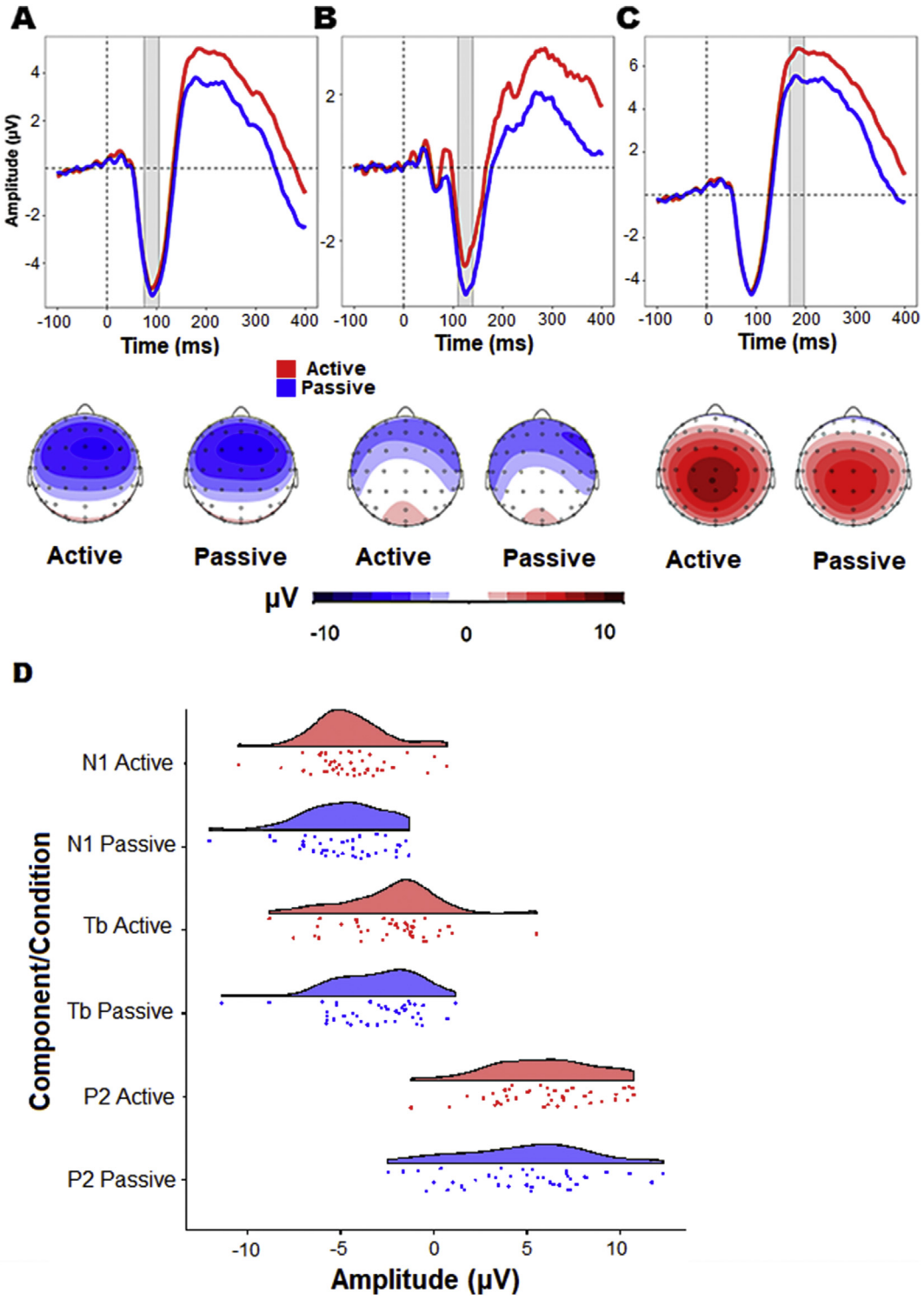

Fig. 2 – Experiment 1: Waveforms showing ERPs elicited by the *active* condition and the *passive* condition in addition to corresponding topographic mappings. (A) N1 component (Fz, FCz, Cz): 74.8–104.8 ms. (B) Tb component (T7, T8): 109.5–139.5 ms. (C) P2 component (FCz, Cz, CPz): 167.6–197.6 ms. (D) Raincloud graph (Allen, Poggiali, Whitaker, Marshall, & Kievit, 2019) containing density plots and scatter plots of mean amplitudes for the N1, Tb, and P2 components for the *active* and *passive* conditions.

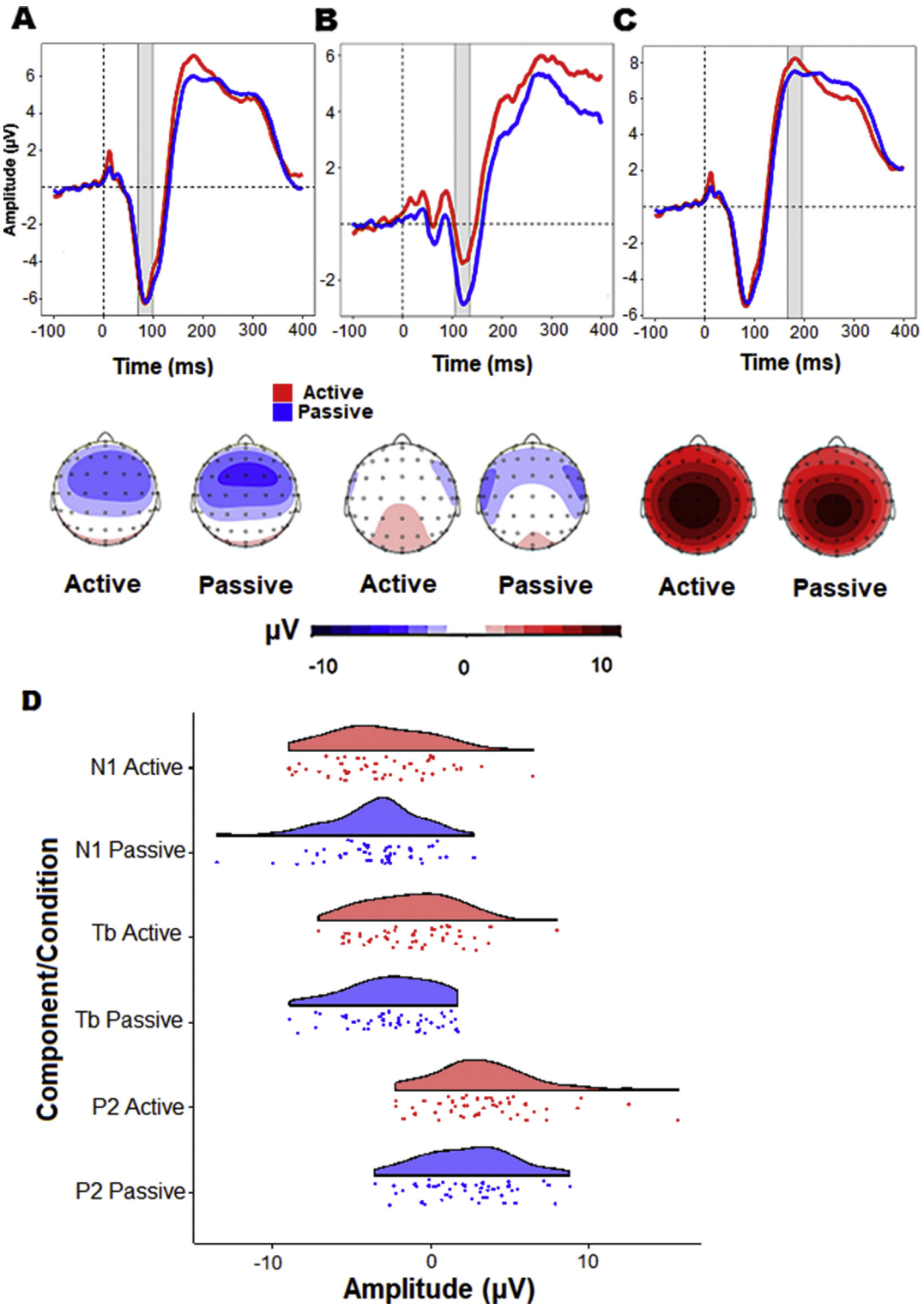

Fig. 3 – Experiment 2: Waveforms showing ERPs elicited by the *active* condition and the *passive* condition in addition to corresponding topographic mappings. (A) N1 component (Fz, FCz, Cz): 69–99 ms. (B) Tb component (T7, T8): 105.6–135.6 ms. (C) P2 component (FCz, Cz, CPz): 166.2–199.2 ms. (D) Raincloud graph (Allen et al., 2019) containing density plots and scatter plots of mean amplitudes for the N1, Tb, and P2 components for the *active* and *passive* conditions.

2.1.5. *Experiment 1 discussion*

In Experiment 1, participants were able to determine whether a sound was presented by means of a prior-made decision; specifically, if participants chose *not* to press the button by the decision time, this resulted in the sound being presented at the trigger time. The upshot of this was that participants had

complete control over the sound delivery, but this control was not a consequence of a motor action. The results revealed that while the amplitude of the N1 component did not differ between the *active* and *passive* conditions, the amplitudes of the Tb and P2 components did, with Tb suppressed in the *active* condition, and P2 enhanced in the *active* condition.

The aim of Experiment 2 was to determine whether the observed results were dependent on the participant's decision to hear the sound being indexed by a non-action (i.e., in which participants implemented their decision to hear a subsequent tone by choosing *not* performing a motor action), or whether the same results would be observed when the participants decision was indexed by a motor-action that was temporally distant (i.e., occurred well prior) to the sound.

2.2. *Experiment 2*

2.2.1. *Participants*

Forty-seven undergraduate students from the University of New South Wales (Sydney, Australia) participated in the study in exchange for course credit ($N = 47$). All participants gave written informed consent prior to the experiment. Participants' mean age was 20.3 years (SD = 5.6), and 29 of the participants were female. Given our sample size of n = 47, this study could detect an effect size of $\eta_p^2 = .18$ at power = 80% with alpha = .05. The study was approved by the Human Research Ethics Advisory Panel (Psychology) at the University of New South Wales.

2.2.2. *Stimuli, materials, and procedure*

The stimuli and materials were identical to Experiment 1. The only difference between the experiments was the action-effect contingency in the *active* condition. In Experiment 1, the audio stimulus was played if the participant elected not to press the button before the decision time. In Experiment 2, this contingency was reversed: the audio stimulus was played *only* if the participant pressed the button prior to the decision time. As in Experiment 1, the audio stimulus was played at the trigger time, which occurred 1 sec after the decision-time which, to reiterate, was the last possible time the participant could elect to press the button; trials in which the participant pressed the button after the decision-time were excluded.

2.2.3. *EEG recording and analysis*

The EEG recording and analysis were identical to Experiment 1.

2.2.4. *Experiment 2 results*

There was an average of 60.3 (SD = 7.2) useable epochs in the *active* condition and 57 (SD = 1.9) in the *passive* condition. Participants' choice frequencies in the active condition was 61.85 sound trials (SD = 6.31) compared to 59.06 sound trials in the passive condition (SD = 1.13). The chi-square test yielded χ^2 (46, N = 47) = 29.62, p = .971.

2.2.4.1. N1. Fig. 3A shows the N1 component analysis elicited in the *active* and *passive* conditions. N1 was maximal at electrode FCz for both conditions and showed the expected fronto-central topography. The time-window for the N1 analysis was centered at 84 ms and extended from 69 to 99 ms. The main effect of Condition was not statistically significant, $F(1, 46) = 1.18$, $p = .283$, $\eta_p^2 = .03$. The *ConditionxElectrode* interaction was also not statistically significant, $F(1, 46) = 1.53$, $p = .144$, $\eta_p^2 = .03$.

2.2.4.2. Tb. Fig. 3B show the Tb component analysis elicited in the *active* and *passive* conditions. Tb was maximal at electrodes T7 and T8 for both conditions and showed the expected temporal topography. The time-window for the Tb analysis was centered at 120.6 ms and extended from 105.6 to 135.6 ms. The repeated measures ANOVA yielded a significant main effect of *Condition*, $F(1, 46) = 11.12$, $p = .002$, $\eta_p^2 = .20$, as well as a significant *ConditionxElectrode* interaction, $F(1, 46) = 9.08$, $p = .004$, $\eta_p^2 = .20$.

2.2.4.3. P2. Fig. 3C shows the P2 component analysis elicited in the *active* and *passive* conditions. N1 was maximal at electrode Cz for both conditions and showed the expected central topography. The time-window for the P2 analysis was centered at 181.2 ms and extended from 166.2 to 196.2 ms. The repeated measures ANOVA yielded a significant main effect of Condition, $F(1, 46) = 7.21$, $p = .010$, $\eta_p^2 = .14$, as well as a significant *ConditionxElectrode* interaction, $F(1, 46) = 3.16$, $p = .002$, $\eta_p^2 = .06$. The results of this additional analysis suggest that the P2 in the active condition was enhanced relative to the passive condition, with the effect being driven by lateral electrodes.

See Fig. 4 for a summary of results for both experiments.

3. General discussion

In this study, we set out to investigate whether sensory attenuation (operationalized as suppression of the N1, Tb, and P2 components of the auditory-evoked potential) occurs when a participant has complete control over the occurrence of a sound – and thus a sense of agency over the sound – but does not perform a motor action (Experiment 1) or performs a motor action that is temporally distant to the sound (Experiment 2). The results were similar across experiments. In both experiments, the Tb component, but not the N1 component, was attenuated in the *active* condition relative to the *passive* condition. The P2 component was enhanced in the *active* condition relative to the *passive* condition for both Experiments 1 and 2. As summarized in Fig. 4, the results of these two experiments suggest that the effect of motor-based predictions on sensory attenuation can be dissociated from those associated with one's sense of agency *per se*, as they seemingly impact on different components of the auditory-evoked potential. Although we have used the label "sense of agency", we acknowledge that it is not possible to differentiate the effect of participants' 'sense of agency' from their 'agency *per se*' in the current experiment.

Our experimental design did not require us to control for motor-related differences in the *active* condition relative to the *passive* condition. Many iterations of the self-stimulation paradigm include a third *motor* condition wherein participants press a button that does not result in a sound. Typically, the ERP of this 'motor-only' condition is subtracted from the ERP of the *active* condition, resulting in an audio-only ERP that

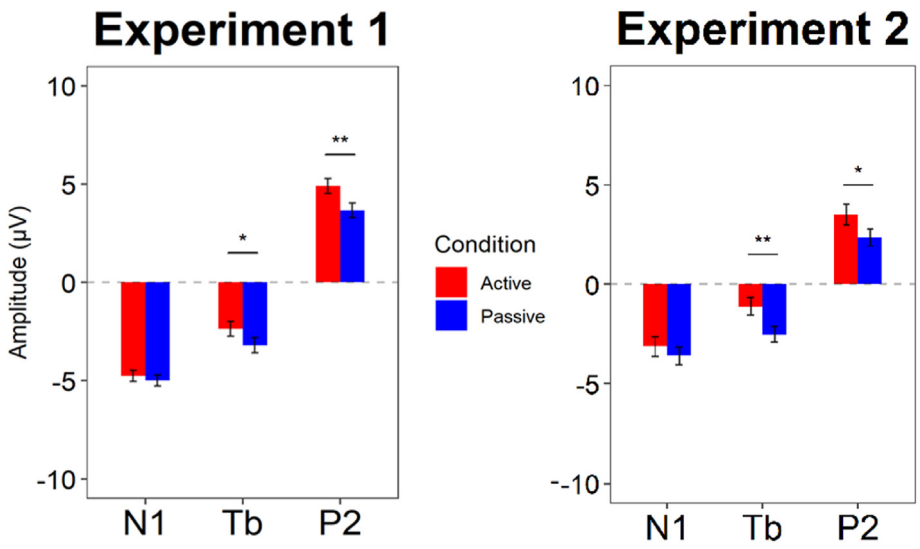

Fig. 4 – Bar graphs of Experiments 1 and 2 illustrating mean amplitudes for the N1, Tb, and P2 components for the *active* and *passive* condition. *Error bars show the standard error of paired differences (SEPD;* Pfister & Janczyk, 2013*).* Asterisks represent levels of significance (*p < .05; **p < .01).

is ostensibly motor-controlled. However, several arguments have been made that query the assumptions behind this subtraction (Horváth, Bíró, & Neszmélyi, 2018; Neszmélyi & Horváth, 2017, 2019). In Experiment 1 of the current study, it was a willed *inaction* that resulted in sounds. In Experiment 2, the time between the action and the sound was at least 1 s, and varied substantially between trials and participants, as the action was not time-locked to the sound. Our design also attempted to control for between-condition differences in temporal predictability and temporal control (see Hughes et al., 2013; Lange, 2011); the temporal onset of the tone was equally predictable and uncontrollable in both the *active* and *passive* conditions, as the tone only ever occurred at the time at which the trigger line and fixation line intersected.

In both experiments, we found no difference in N1 amplitude between the *active* and *passive* conditions. This stands in contrast to most previous self-stimulation studies that have observed smaller N1 amplitudes in the *active* condition relative to the *passive* condition (i.e., N1-suppression) (Baess, Horváth, Jacobsen, & Schröger, 2011; Bäß et al., 2008; Elijah et al., 2018; Mifsud et al., 2016; Neszmélyi & Horváth, 2017; Oestreich et al., 2016; Pinheiro et al., 2019; van Elk et al., 2014). The N1 is not a unitary component; in contrast, there are believed to be at least three obligatory sources for the N1 (Horváth, 2015; Näätänen & Picton, 1987). Given the observed scalp distribution and the long silent periods between trials (>10 sec on average), the present design may have tapped into the non-specific (i.e., modality free) N1 component (Davis & Zerlin, 1966; Hari, Kaila, Katila, Tuomisto, & Varpula, 1982; SanMiguel et al., 2013). N1 amplitude has been shown to index physical features of sound, notably auditory intensity (Adler & Adler, 1989; Beagley & Knight, 1967; Picton, Goodman, & Bryce, 1970). N1 suppression has been argued to reflect the sound of the *active* condition being processed as less intense than the *passive* condition, possibly through the action of efference copy/corollary discharge-related mechanisms (Hughes et al., 2013; Whitford, 2019). Previous research that manipulated the delay between action and effect found that delays longer than a few hundred milliseconds abolished the N1 suppression effect (Oestreich et al., 2016; Pinheiro et al., 2019; Whitford et al., 2011), suggesting that N1 suppression is dependent on the motor action occurring close-in-time to the resulting sound. The result of Experiments 1 and 2 corroborate this finding; the absence of N1-suppression in Experiment 1 suggests that a motor action is necessary for N1 suppression, while the absence of N1-suppresion in Experiment 2 suggests that a motor-action must occur close-in-time to the resulting sound if it is to elicit N1-suppression. Our design attempted to control for motor-related differences and temporal predictability and control, which left participants' sense of agency as one of the few remaining difference between conditions. These results suggest that a motor action that is approximately temporally coincident with the sound is necessary for N1-suppression to occur, and that having a sense of agency over the sound is not sufficient. It is important to note, however, that N1 amplitude can be influenced by factors other than the performance of willed motor actions. For example, several studies have shown that visual stimuli that are predictive of auditory events (such as an animation of a person clapping) can also result in a reduction in N1 amplitude (Libesman, Mannion, & Whitford, 2020; Stekelenburg & Vroomen, 2007; Vroomen & Stekelenburg, 2010).

As indexed by the non-significant chi-squared tests, we did not find much evidence to suggest that participants varied in their choice frequencies for either experiment (i.e., the proportion of trials in which participants opted to hear vs not hear the sound). However, if such a behavioural difference between conditions did in fact exist, then this would represent a potential confound that could underlie the apparent absence of N1-suppression in the two experiments.

The Tb component was suppressed in the *active* condition relative to the *passive* condition in both experiments. This suppression occurred in the absence of any motor action in

Experiment 1, and when the motor-action was temporally distant and not time-locked to the sound in Experiment 2. Taken together, these results suggest that in contrast to N1, the Tb component, is sensitive to manipulations in sense of agency (i.e., the ability to cause the sound to occur), but is not dependent on the occurrence of a co-incident motor action, and thus is likely not dependent on efference copy/corollary discharge-related mechanisms. The Tb suppression effects observed in both experiments may have also received contribution from the relatively longer periods of silence between trials. SanMiguel et al. (2013) and Horváth (2013) also reported Tb suppression effects with long inter-stimulus intervals (ISI) (3.2 sec and >4 sec, respectively). SanMiguel et al. (2013) assessed Tb suppression among different levels of ISIs (.8, 1.6 and 3.2s) but only reported Tb suppression effects with the longest ISIs.

The Tb component is believed to be generated within the secondary auditory cortex (Gallinat et al., 2002; Rihs et al., 2013; Tonnquist-Uhlen, Ponton, Eggermont, Kwong, & Don, 2003; Wolpaw & Penry, 1975), potentially in the vicinity of Wernicke's area (Alain, Woods, & Covarrubias, 1997). While the functional significance of the Tb component has not been definitively established, it has been implicated in auditory processing, particularly of 'high level' auditory stimuli including music and language (Azouz, kozou, Khalil, Abdou, & Sakr, 2014; Bruneau, Bonnet-Brilhault, Gomot, Adrien, & Barthélémy, 2003; Giard et al., 1994; Hämäläinen, Fosker, Szücs, & Goswami, 2011; Harpaz, Levkovitz, & Lavidor, 2009; Langers, Backes, & van Dijk, 2007; Tonnquist-Uhlen et al., 2003; Shahin, Bosnyak, Trainor, & Roberts, 2003).

In regard to the present study: the results of both experiments indicated that Tb amplitude was decreased when participants had control over whether they heard the sound. In other words, the Tb component appeared to index participants' sense of agency over the sound. Our sense of agency has been argued to arise when our motor actions result in predictable sensory consequences (Blakemore, Wolpert, & Frith, 2000; Blakemore, Wolpert, & Frith, 2002; Synofzik et al., 2008). Given this, it may seem odd to divorce sense of agency from our motor actions. However, there are instances in real life where one may be in control of events by virtue of inaction; the classic trolley problem in philosophy is a theoretical example. A real-world example would be when one decides not to intervene when a jar is about to fall off a table. It may be helpful to distinguish between the *feeling of agency* versus the *judgement of agency*, as outlined by Synofzik et al. (2008). The *feeling of agency* is simply the sense of agency someone experiences when they perform a motor action that is followed by a sensory event. This is what the literature typically refers to when discussing agency within the context of comparator models (Synofzik et al., 2008). The *judgement of agency*, on the other hand, requires an explicit cognitive judgement of one's agency, and does not rely on sensorimotor indicators. The sense of agency experienced by participants in Experiments 1 and 2 would more likely be that of the *judgement of agency*, and it is therefore possible that it is judgements of agency, as opposed to feelings of agency, that are indexed by Tb amplitude. This question has not (to our knowledge) been investigated previously and would be a worthwhile topic for future research. If the Tb component is found to index sense of agency (or even judgements of agency more specifically), it would be interesting to know whether Tb is specific to auditory stimuli, or whether analogous components can be elicited by manipulations of sense of agency in other sensory domains.

The P2 component was enhanced in the *active* condition relative to the *passive* condition in Experiments 1 and 2. Although its functional significance is not clear, the P2 component has been associated with attention and categorization processes (Crowley & Colrain, 2004; García-Larrea, Lukaszewicz, & Mauguiére, 1992; Lijffijt et al., 2009). Further evidence has also linked the P2 component to working memory processes (Duzcu, Özkurt, Mapelli, & Hohenberger, 2019; Finnigan, O'Connell, Cummins, Broughton, & Robertson, 2011; Lefebvre, Marchand, Eskes, & Connolly, 2005). Most studies investigating sensory attenuation have found suppression of the P2 component in the *active* condition relative to the *passive* condition (Knolle et al., 2012; Horváth & Burgyán, 2013; Timm, SanMiguel, Keil, Schröger, & Schönwiesner, 2014; Klaffehn, Baess, Kunde, & Pfister, 2019). However, the present study found P2 enhancement in the *active* condition. One potential reason for this inconsistency may be related to the long (>1 sec) and variable action-effect delays used in the present design. For example, Klaffehn et al. (2019) used a similar design (with a loading bar instead of a tickertape) in which there was a 750 ms delay between action and outcome. They observed no difference in P2 amplitude between the *active* and *passive* conditions; a result that is intermediate between the results of the present study (which had a longer action-effect delay and observed P2 enhancement) and most of the existing literature (which has had negligible-to-small action-effect delays and observed P2 suppression). Another potential factor is sense of agency over the sounds. For example, a previous study by Timm, Schönwiesner, Schröger, and SanMiguel (2016) demonstrated significantly larger P2 amplitudes when participants experienced agency over sounds than when they did not. These results might suggest that the P2 and Tb components are suitable candidates for investigation of the relationship between the sense of agency and sensory attenuation.

It is also worth noting that N1 and P2 suppression effects are likely caused by different factors. For example, lesions to the cerebellum (thought to be a key anatomy of sensory attenuation Knolle et al., 2012), differentially affect N1- and P2- suppression, as does the type of motor-action (e.g., hand-movement *vs* foot-movement) producing the sensory outcome (van Elk et al., 2014). Though sensory attenuation studies have typically observed both N1- and P2- suppression in the *active* condition, the pattern of results for the P2 component has been less consistent than that of the N1 component (Pinheiro et al., 2019). The results of the current study are consistent with previous research demonstrating

higher P2 component amplitudes when stimuli are considered task relevant within working memory (Getzmann, Wascher, & Schneider, 2017; Duzcu et al., 2019). For Experiment 1, sounds produced in the *active* condition might have contained a novel relevance by virtue of the fact that it was inaction that caused the sound, since inactions rarely result in sensory consequences in everyday life.

There are several studies that have used similar designs to the present set of experiments. Weller, Schwarz, Kunde, and Pfister (2020) used a similar action/nonaction paradigm to assess temporal binding, a phenomenon wherein a voluntary action and a subsequent sensory effect are perceived to be temporally compressed (Haggard, Clark, & Kalogeras, 2002). The temporal compression of action and effect has been interpreted as an implicit marker of the sense of agency. In Weller et al.'s (2020) second experiment, participants observed a rotating clock hand and were given the option to either press or not press a button in a given time frame. Both options produced distinct sounds. They then estimated the time between the point of action/inaction and sound onset. Weller et al. (2020) found that temporal binding effects existed even for inactions, thereby providing evidence that willed inactions can also result in a sense of agency. Their third experiment replicated the results of their second experiment but also controlled for temporal predictability. Here, participants used action/inaction to decide the direction that a pinball stimulus would fire a ball. When participants opted for inaction, a loading bar filled up, which was immediately followed by a clicking sound. After onset of the clicking sound, the ball would be fired from the pinball (which was paired with a ball launch sound). Participants were instructed to estimate the time interval between the clicking sound and the ball launch sound. Again, Weller et al. (2020) found a temporal binding effect for inactions, providing further evidence that willed inactions can result in a sense of agency. Participants in the third experiment also reported higher agency ratings for inaction compared to a baseline condition.

Another study by Klaffehn et al. (2019) assessed the role of sense of agency in the sensory attenuation effect. In this study, a loading bar was used to control for temporal predictability between the active and passive conditions. However, in contrast to the present study, they found evidence of N1 suppression effect for two of three electrodes (FCz and Cz) when a 750 ms delay was imposed between action and effect (similar to the present study in which the delay between action and effect was > 1s). One possible explanation for why our N1 results are inconsistent with those of Klaffehn et al. (2019) may be the differences in ISI between experiments, as this may have led to the N1 waveform being dominated by different subcomponents. Klaffehn et al.'s (2019) study had ISIs of <4 sec, meaning that N1 waveforms may have been dominated by a frontal or fronto-central distribution (Horváth, 2015; Vaughan & Ritter, 1970). In contrast, the present experiment included ISIs that were on average > 10 sec, meaning that the N1 was most likely dominated by the non-specific component. One possible future study to disentangle the inconsistent results may be to incorporate different ISIs within the same experiment, such as in the study of SanMiguel et al. (2013). Taken together, these results suggest that sensory attenuation may extend to action-effect pairings in which the participant has a sense of agency over a sensory outcome, but the action and outcome are not temporally coincident.

Several previous studies of sensory attenuation have linked the phenomenon with the characteristic abnormalities in agency that are often observed in patients with schizophrenia (e.g., Ford et al., 2001; Ford et al., 2007; Fletcher & Frith, 2009; Whitford, 2019). These models are often premised on the idea that sense of agency arises as a consequence of the same comparator processes that underlie sensory attenuation (Frith, Blakemore, & Wolpert, 2000). The alternative possibility is that sensory attenuation and sense of agency arise from distinct processes, and that schizophrenia is independently associated with deficits in both. By disambiguating the effects of motor action from sense of agency, our experimental paradigm may provide a platform for future studies aimed at disambiguating these competing possibilities, by testing whether schizophrenia patients show deficits in Tb suppression to controllable sounds arise as a consequence of willed inactions.

In conclusion, the results of this study suggest that motor-actions and sense of agency have differential effects on the evoked response to self-initiated sounds, and are indexed by different components of the auditory evoked potential. Specifically, while N1-suppression did not occur in the absence of a temporally coincident motor action, Tb-suppression did occur when participants could control whether or not a sound was presented by means of a willed inaction. This result suggests that the Tb component may index one's sense of agency over sensory events. Whether this role is limited to auditory events or extends to other sensory modalities is an open question, and may be a worthwhile question for future research.

Notes

No part of the study procedures or analyses was preregistered prior to the research being conducted. We report how we determined our sample size, all data exclusions (if any), all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study. Raw data and study materials are publicly available via the OSF: https://osf.io/dcwpg/.

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Author contribution

Nathan Han: Conceptualization; Formal analysis; Visualisation; Writing- Original Draft; Writing- Review & Editing; Methodology; Software; Investigation.

Bradley N. Jack: Conceptualization; Writing- Review & Editing; Methodology; Formal analysis; Writing- Review & Editing; Software.

Gethin Hughes: Conceptualization; Methodology; Writing-Review & Editing.

Ruth B. Elijah: Methodology; Writing-Review & Editing.

Thomas J. Whitford: Conceptualization; Writing- Review & Editing; Methodology; Resources; Funding acquisition; Supervision; Project administration.

Open practices

The study in this article earned Open Data and Open Materials badges for transparent practices. Materials and Data for this study can be found at https://osf.io/dcwpg/.

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