



# N1-P2 event-related potentials and perceived intensity are associated: The effects of a weak pre-stimulus and attentional load on processing of a subsequent intense stimulus

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## ABSTRACT

A weak stimulus presented immediately before a more intense one reduces both the N1-P2 cortical response and the perceived intensity of the intense stimulus. The former effect is referred to as cortical prepulse inhibition (PPI), the latter as prepulse inhibition of perceived stimulus intensity (PPIPSI). Both phenomena are used to study sensory gating in clinical and non-clinical populations, however little is known about their relationship. Here, we investigated 1) the possibility that cortical PPI and PPIPSI are associated, and 2) how they are affected by attentional load. Participants were tasked with comparing the intensity of an electric pulse presented alone versus one preceded 200 ms by a weaker electric prepulse (Experiment 1), or an acoustic pulse presented alone with one preceded 170 ms by a weaker acoustic prepulse (Experiment 2). A counting task (easy vs. hard) manipulating attentional load was included in Experiment 2. In both experiments, we observed a relationship between N1-P2 amplitude and perceived intensity, where greater cortical PPI was associated with a higher probability of perceiving the 'pulse with prepulse' as less intense. Moreover, higher attentional load decreased observations of PPIPSI but had no effect on N1-P2 amplitude. Based on the findings we propose that PPIPSI partially relies on the allocation of attentional resources towards monitoring cortical channels that process stimulus intensity characteristics such as the N1-P2 complex.

## 1. Introduction

When a weak stimulus is presented immediately before a stronger one, the motoric (e.g., startle and blink reflex) and cortical (electrophysiological) response to and perceived intensity of the stronger stimulus are reduced compared to presenting the strong stimulus alone (Graham, 1975; San-Martin et al., 2018; Swerdlow et al., 2005). These phenomena are broadly referred to as prepulse inhibitions (PPI) and are operational measures of sensorimotor and sensory gating. Sensory gating involves the cortical process that permits or suppresses the further processing of incoming stimuli. Although the underlying mechanisms and protocols to elicit both types of gating (motor and sensory) overlap, it is likely that these two phenomena are affected by different brain processes. Here, we were interested in understanding the processes which influence gating of conscious perception.

Startle PPI is a measure of motor gating: the presence of a weaker preceding stimulus (prepulse) reduces the blink reflex to an intense

stimulus (pulse; Blumenthal, 2015). It is observed shortly after the pulse at short gaps between prepulse and pulse onset (SOA). SOAs from 15 to 300 ms will elicit startle PPI, and SOAs of 60 – 120 ms are where inhibitory effects of the prepulse on the blink reflex are maximal (Blumenthal, 2015; Swerdlow et al., 2005). Because the blink reflex has a short onset latency, and the SOA required to elicit startle PPI is also very short, startle PPI is mostly informative about the lower-level gating mechanisms (Fendt et al., 2001). That is, the short latencies suggest that the blink reflex and startle PPI occur so quickly that there is little time for the involvement of higher-order processes with longer cortical loops. While explicitly instructing participants to pay attention to the prepulse does enhance startle PPI, these studies differ significantly from typical startle PPI experiments: they utilise longer SOAs (120 ms+) and a continuous prepulse (Ashare et al., 2007; Dawson et al., 1993; Elden & Flaten, 2002; Filion & Poje, 2003; Hawk et al., 2002; Heekeren et al., 2004; Poje & Filion, 2021). Typical studies of startle PPI use short SOAs (e.g., 60 ms) and discrete prepulses, settings which are not optimal for

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the observation of attentional enhancement of PPI (Poje & Fillion, 2021). Furthermore, evidence that gating of reflexes is driven by brainstem and midbrain structures supports the conceptualisation that startle PPI is mediated by lower-level gating mechanisms (for detailed reviews see, Azzopardi et al., 2018; Fendt et al., 2001). Combined with findings that startle PPI is observed in infants (Graham et al., 1981), sleeping adults (Silverstein et al., 1980) and even decorticated rats (Ison et al., 1991), the broader literature suggests that the gating of reflexes is largely driven by processes that are independent of attention. However, conscious perception requires the engagement of attentional processes (Noah & Mangun, 2020), and we are yet to understand the ways by which lower-level mechanisms may contribute to gating of conscious perception.

Sensory gating of perception has been proposed to be more directly measured by a related phenomenon known as prepulse inhibition of perceived stimulus intensity (PPIPSI; Swerdlow et al., 1999a, 1999b; Swerdlow et al., 2005). PPIPSI is measured by the reported reduction in the perceived intensity of the pulse stimulus when a prepulse is present (Swerdlow et al., 1999a, 1999b; Swerdlow et al., 2005). Meaning, participants are required to make relative judgments about the intensity of their experiences. Studies measuring startle PPI and PPIPSI simultaneously have reported strong positive correlations ( $r = .72 - .75$ ; Swerdlow et al., 1999a, 1999b; Swerdlow et al., 2005). These findings indicate that gating of perception (measured by PPIPSI) likely shares underlying mechanisms with gating of reflexes (Swerdlow et al., 2005). Subcortical signals (those activated by startle PPI) may propagate to perceptual areas of the cortex where perceived intensity is processed (Swerdlow et al., 2005). However, there is little direct evidence for this, and studies show that the time-course of startle PPI and PPIPSI activation differ (Favero et al., 2022; Swerdlow et al., 2005; Swerdlow et al., 2007). While startle PPI is observed at short SOAs (>15 ms), Swerdlow et al. (2005) found that for acoustic stimuli, the prepulse has no effect on perception at SOAs below 60 ms and maximally reduces perceived intensity at 120 ms. Moreover, in a previous study, we found that electrotactile PPIPSI requires SOAs of 160 – 600 ms (Favero et al., 2022). In line with the task requirements of PPIPSI, that participants direct their focus to the pulse stimuli (with vs. without prepulse) and provide an intensity rating or comparison, it has been proposed that the requirement of longer SOAs is because attentional mechanisms are required to experience sensory gating of perception (Favero et al., 2022; Swerdlow et al., 2005; Swerdlow et al., 2007). More specifically, longer times are required to direct attention to the sensory inputs which reflect the inhibitory effects of the prepulse. These data suggest that, although gating of perception may be partially shaped by subcortical processes, this is unlikely to be the complete picture – attention and perception are higher order processes and likely associated with or represented by cortical activity.

Although time-course data and task requirements provide some insight into the involvement of higher order processes in perceptual gating, stronger evidence of this may come from studies specifically measuring cortical activity such as an electroencephalogram (EEG). The presence of a prepulse is known to correspond with inhibited N1 and P2 event related potentials (ERP) at central and centroparietal areas in human subjects – reflecting what is known as cortical PPI (Dawson et al., 2004; Kedzior et al., 2007; San-Martin et al., 2018). Researchers have suggested that the N1 and P2 ERP reductions might be due to cortical encoding of the processes which contribute to PPIPSI (Swerdlow et al., 2007). Indeed, cortical PPI and PPIPSI share similar magnitude reductions and temporal sensitivities (Kedzior et al., 2006; Swerdlow et al., 2007). Furthermore, studies have evidenced the N1 and P2, or N1-P2 complex represent functionally relevant processes to PPIPSI, such as those involved in processing the physical characteristics of the stimulus (e.g., intensity; Annic et al., 2014; Garcia-Larrea et al., 1992). For example, N1 and P2 magnitude both increase as a function of stimulus intensity (Mulert et al., 2005; Paiva et al., 2016; Rosburg et al., 2008).

While the N1-P2 may represent encoding of stimulus intensity and

classification, the literature indicates that perceptual and decisional processes are not contained within this processing stage or time window, but that they provide specific feature traces, or possibly complete representations of a stimulus, which perceptual and decisional processes may access (Näätänen & Winkler, 1999). One possibility is that perceptual and decisional processes can access cortical representations of sensory stimuli via attentional mechanisms such as selective attention. Directing attention to the stimulus is said to facilitate conscious perception by bringing its cortical representation into focus (Näätänen & Winkler, 1999). The above findings suggest a relationship between cortical PPI and PPIPSI where inhibition of the N1 and P2 cortical responses, which represent encoding of stimulus features, contribute to the perceptual experience of reduced perceived intensity. Moreover, they indicate that this relationship is modulated by selective attention, which brings the inhibited cortical representation into conscious perception. Such a model is consistent with previous propositions that PPIPSI requires higher order (attentional and self-monitoring) mechanisms (Favero et al., 2022; Swerdlow et al., 2005). However, this relationship is yet to be investigated more directly, and while PPIPSI is observed in other sensory modalities (e.g., tactile), current propositions of its similarities with cortical PPI have only been based on acoustic data (Kedzior et al., 2007; Swerdlow et al., 2007).

Additionally, the extent to which N1 represents encoding of attentional processes is debated. According to Näätänen's (1992) model of auditory processing, N1 is considered a predominantly exogenous ERP, shaped by stimulus characteristics (e.g., particularly intensity) and representing encoding of its feature traces. In this model, N1 is pre-attentive (unaffected by and non-representative of attentional processing), and even if unattended, or not entering conscious perception, its contained information (stimulus trace or representation) is still encoded (Näätänen & Winkler, 1999). For these authors, attention is directed post-N1 via a perceptual mechanism. Alternatively, Muller-Gass and Campbell (2002) suggest N1 is endogenous and represents attentional allocation to a stimulus, whether task relevant or not. Indeed, selective attention has been found to modulate N1 and P2 cortical responses, enhancing their magnitude for attended as opposed to unattended stimuli (Tiitinen et al., 1993; Mishra & Hillyard, 2009), and increasing cortical PPI when directed towards the prepulse (Annic et al., 2014). Assessment of PPIPSI differs from these paradigms in that attention is directed toward each pulse-stimulus in a trial, thus even if a relationship was found between cortical PPI and PPIPSI, inferring at what stage attentional processes are recruited, and their effects would not be possible. However, given that attention is widely held to be a finite resource (Petersen & Posner, 2012; Swallow & Jiang, 2013), we can gain insight into the cortical mechanisms of PPIPSI by manipulating attentional load. If N1-P2 represents attentional allocation to a stimulus, presenting a secondary task which induces attentional load would likely modulate this cortical response, and the inhibitory effect of the prepulse. Conversely, if the N1-P2 response is unaffected by attentional load, this would align best with Näätänen and Winkler (1999), suggesting that N1-P2 is encoding of stimulus representation, and that attention is directed towards this channel at a later stage of processing via a perceptual mechanism which may result in PPIPSI.

In this study, we sought to investigate the relationship between gating of perception (PPIPSI) and the net neural response of the cortical system at the N1-P2 timeframe (~50–250 ms) in both tactile (Experiment 1) and acoustic (Experiment 2) modalities. The N1-P2 complex reflects the initial cortical processing of sensory stimuli, and the cortical responses of this complex provide an index of the net neural activity at a critical time for perception that can be compared by measuring ERPs with and without prepulses. To extend this further, in Experiment 2 we also manipulated attentional load, providing insight into the influence attentional processes have on PPIPSI and cortical PPI.

## 2. Methods

### 2.1. Participants

Participants were Curtin University undergraduate students who participated in exchange for course credit and volunteers. We ran an a priori power simulation based on pilot data from five participants. Our sample sizes were based on our prior study using the same protocol we employed in the current study (Favero et al., 2022), where we collected between 22 and 25 participants. This range is similar to that of a related study by San-Martin et al. (2018), which recruited 22 participants to examine cortical PPI. For Experiment 1, 26 participants (21 female) were recruited (age  $M(SD) = 21.79(4.46)$  years, range = 18 – 38 years). However, EEG data from two participants was not collected due to software error, resulting in a final sample of 24 participants (20 female), age  $M(SD) = 21.74(4.55)$ , range = 18 – 38. For Experiment 2, a separate sample of 24 participants (20 female) were recruited (age  $M(SD) = 24.04(7.02)$  years, range = 18 – 48 years). All participants self-reported having normal or corrected-to-normal vision, with no known neurological conditions or injuries that may affect their performance in the experiment. In accordance with the Declaration of Helsinki and with approval from the Curtin University human research ethics committee (Approval Code: HRE2018–0257) informed written consent was provided by all subjects prior to participation.

### 2.2. Pre-experiment procedures

In both experiments we used a within-participant repeated measures design. Consistent with our previous work (Favero et al., 2022), participants were seated at a desk 57 cm from a 24-inch BenQ LCD monitor (1920 × 1080 resolution; 120 Hz refresh rate), with both arms rested on the desk.

#### 2.2.1. Experiment 1 – Tactile

Two Digitimer STIMULATOR-DS7As – one to deliver the prepulse and one to deliver the pulse – were then attached to participants' left wrist around the ulnar styloid process, via four Kendall Covidien Ag-AgCl electrodes. Both stimulators were set to emit a single square wave prepulse or pulse with a duration of 2 ms. Perceptual thresholds (weakest identifiable stimulation) were then identified and documented via the following process: starting at 0.50 mA and decreased in increments of 0.10 mA until participants no longer reported feeling the stimulus, intensity was then increased using finer increments of 0.05 mA until first perceived again – this value was defined as their individual perceptual threshold. Once perceptual threshold was identified, the prepulse intensity was set by doubling this (e.g., 0.50 mA perceptual threshold = 1.0 mA experiment prepulse intensity). Pulse intensity was set by starting at perceptual threshold and rising in 1 mA increments until the pulse was reported to be “unpleasant, but not painful” by the participant. Descriptive statistics of participants perceptual thresholds, prepulse and pulse test intensities are provided below in Table 1.

#### 2.2.2. Experiment 2 – Acoustic

Acoustic stimuli were generated by the motherboard of the computer used to run the experiments and presented binaurally through stereophonic headphones (Model: HD25–1 II). The background noise was set

**Table 1**  
Means, Standard Deviations and Ranges for Stimuli Intensities in Experiment 1.

Stimulus	Experiment 1	
	<i>M (SD)</i>	Range
Threshold	0.35 (0.17)	0.1 – 0.8
Prepulse	0.70 (0.39)	0.2 – 1.6
Pulse	4.63 (2.54)	2.0 – 12.0

Note. Unit of measurement = milliamps (mA).

at 60dBa. The stimuli consisted of a brief white noise burst (50 ms duration with a rise and fall time < 1.5 ms). Consistent with the optimal intensity range reported by and cortical PPI studies by San-Martin et al. (2018), the pulse stimulus intensity was 100 dB and the prepulse intensity was 80 dB.

#### 2.2.3. Procedures - Experiment 1: tactile perceptual task

In each trial, participants were instructed to compare two pulses: the pulse-alone (control) against the ‘pulse with prepulse’ (PPIPSI condition). A 200 ms SOA was used for the PPIPSI condition. The task consisted of 90 trials (i.e., 90 comparisons of ‘pulse alone’ with ‘pulse with prepulse’ conditions). Stimulus conditions were randomised with an equal number of ‘pulse alone’ and ‘with prepulse’ first trials. To commence the experiment, a prompt stating “press any button to start” was displayed on the screen. After participants pressed a key, the delivery of the first stimulus was randomised to 1, 2 or 3 s. Within each trial, the time interval between the first stimulus (S1) and the second stimulus (S2) was randomised to either 3, 4 or 5 s. After both S1 and S2 were delivered, participants were prompted to select via mouse clicking: “which shock-stimulus was more intense (left-click = first stimulus; S1, right-click = second stimulus; S2 or felt the same = middle-click)?”. Participants were told there was no correct response and that we were purely interested in comparing their perception with brain responses. The commencement of the next trial was randomised to 1, 2 or 3 s after the mouse response, thus time between trials varied but was at least 1 s, but the effective window including response time was about 5 s. We opted for a comparison between pulse (with/without prepulse) conditions method as opposed to the visual analogue scale (VAS; assigning a numeric perceived intensity rating) for consistency with our previous study (Favero et al., 2022), and to avoid the possibility that our ethical requirement of non-painful stimuli intensities may result in participants selecting intensities below those that the VAS can record meaningful differences from (see Favero et al., 2022 and Swerdlow et al., 2005 for further detail).

#### 2.2.4. Procedures - Experiment 2: acoustic perceptual task and attentional load

In Experiment 2, participants completed the same perceptual comparison task as in Experiment 1, with an additional counting task to manipulate attentional load and the following stimulus interval differences. A SOA of 170 ms (interstimulus interval: 120 ms) was used for the PPIPSI condition informed by previous studies (San-Martin et al., 2018; Swerdlow et al., 2005). The experiment consisted of 60 trials, stimulus conditions were randomised with an equal number of ‘pulse alone’ and ‘with prepulse’ first trials. Attentional load was divided into two levels of task difficulty (easy and hard) and presented in blocks of 30. Block order was randomised between participants. To manipulate attentional load, numbers were presented in the centre of the screen for a duration of 1 s. In the easy task, these numbers were a series of 1 s (11 in total, same number of 1 s/numbers on each trial), and in the hard task numbers were pseudorandomised and ranged from – 9 to + 11. Participants were instructed to add these and report the sum via keyboard input after reporting their perception.

To commence the experiment, a prompt was displayed on screen stating, “press enter to start”, 1 s after the participant pressed the enter key the first number would appear, displayed for 1 s and then be replaced by a focus point (dot). Acoustic S1 was set to randomly deliver between 3.5 and 4.5 s into a trial, and S2 was set to be randomly delivered between 12.5 and 13.5 s into the trial. Thus, the interval between S1 and S2 was randomised between 8 and 10 s. Presentation of visual and acoustic stimuli were offset so as not to be presented at the same time. After the last number of the counting task had been presented a screen prompt for the perceptual comparison would appear: “which loud-stimulus was more intense (left-click = first stimulus; S1, right-click = second stimulus; S2 or felt the same = middle-click)?”. Following reporting of perception, participants were asked to report the

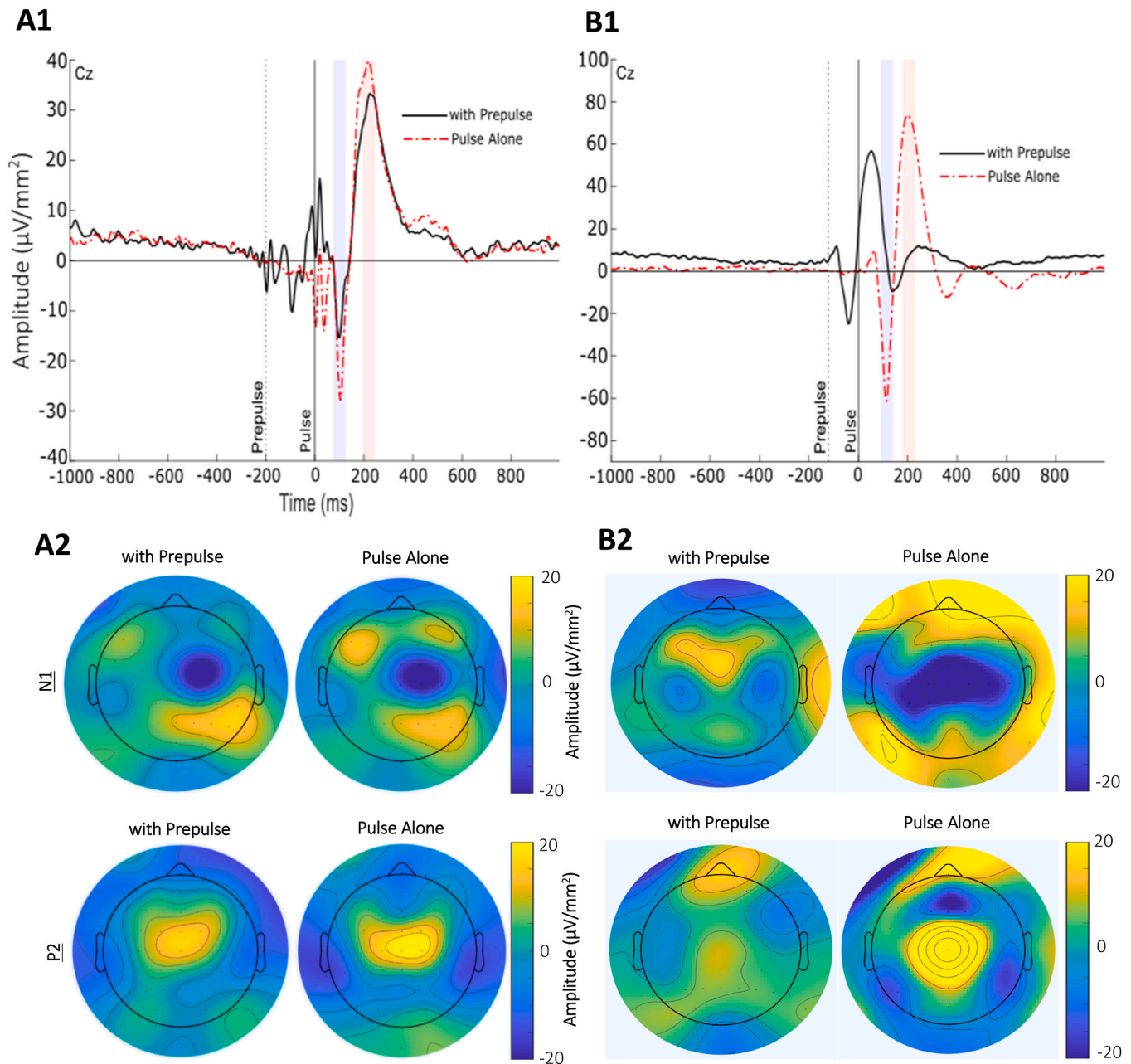
sum from the counting task via the number pad and press enter to record the response and start the next trial. Time between responding to the present trial and commencement of the next was also randomised to 1, 2 or 3 s post entering number response, thus time between trials varied but was at least 1 s.

### 2.3. EEG acquisition & pre-processing

EEG data were recorded continuously for the duration of the experiment. Data were acquired using a Biosemi ActiveTwo EEG system and ActiView (ver. 7.07) at a sampling rate of 1024 Hz with a 100 Hz low-pass online filter. Data were recorded from 64 scalp electrodes arranged according to the 10–5 system with additional electrodes placed

adjacent to the outer canthi of both eyes and on the left infraorbital region. For online referencing, the Biosemi EEG system uses active electrodes with common mode sense (CMS) and driven right leg (DRL) electrodes providing a reference relative to the amplifier reference voltage.

The EEG data were processed offline in MATLAB 2021a using EEGLAB (Delorme & Makeig, 2004) and SASICA plugins (Chaumon et al., 2015). The data were re-referenced to the average of the 64 scalp electrodes, filtered from 0.1 to 40 Hz using separate low- and high-pass filters using the ‘pop\_eegfiltnew’ function in EEGLAB and then down-sampled to 256 Hz. Two epochs were extracted per trial, time-locked to the presentation of the pulse for each stimulus condition. Epochs spanned from – 1000–1000 ms and amplitudes were baseline



**Fig. 1.** Waveforms, and Scalp Map Distributions for N1 and P2 of Both Conditions (with Prepulse vs Pulse Alone) in Experiment 1(A) and Experiment 2(B). Note. Grand-average ERP waveforms for each stimulus condition (with prepulse = black, pulse alone = red), in both Experiment 1 (A1) and Experiment 2 (B1), with shaded areas depicting N1 and P2 measurement intervals (blue = with prepulse, red = pulse alone). Experiment 1 (A2) and Experiment 2 (B2) topographical plots of the grand average waveform across ‘with prepulse’ and ‘pulse alone’ trials for N1 and P2 over an average of the measured time intervals. All amplitudes are CSD-transformed (µV/mm²).



corrected to a short pre-stimulus period prior to and avoiding the prepulse:  $-400$  to  $-200$  ms (Experiment 1) and  $-300$  to  $-200$  ms (Experiment 2). Independent Component Analysis was conducted and independent components (ICs) containing artifacts were manually identified with the guidance of SASICA and removed to correct for blinks, horizontal saccades and other artifacts. In Experiment 1, an average of 13.88 (SD = 4.69) ICs were removed. In Experiment 2, an average of 12.88 (SD = 6.06) ICs were removed. Note that the number of IC artifacts removed does not result in the removal of trials. However, trials containing voltages on analysed channels exceeding  $\pm 100$   $\mu\text{V}$  were excluded,  $M(\text{SD})_{\text{excluded}} = 3.21(8.22)$  trials (Experiment 1), and  $4.50(5.34)$  trials (Experiment 2).

Using algorithms described in Perrin et al. (1989) we applied a surface Laplacian filter (smoothing factor =  $1e^{-5}$ , order of Legendre polynomial = 10), which then transformed our data to reference-free current source density (CSD). CSD transformation reduces volume conduction effects in EEG sensor space and increases spatial resolution of the signal (Gevins et al., 1995; Kayser & Tenke, 2015). In our context, because each trial contained two temporally overlapping stimuli of interest (the pulse stimuli), the surface Laplacian was applied to prevent later ERPs to the first stimulus from masking early ERPs to the second stimulus. This process aimed to emphasise the signal we were interested in (N1/P2 complex). Voltages were measured at the central midline (Cz) based on previous cortical PPI studies of maximal inhibition (Abduljawad et al., 1999; Ford et al., 1999; San-Martin et al., 2018) as well as inspection of scalp density plots highlighting larger activation in these areas (see Fig. 1).

In both experiments, we focused on the peak-to-peak amplitude difference. Peaks were defined using the mean amplitude of a short time window (25 ms either side) surrounding the peak of N1 and P2 components. In Experiment 2, the SOA and stimulus intensities resulted in temporal overlap of prepulse and pulse ERPs in the ‘with prepulse’ condition. To resolve this, we used the peak amplitude timings of the ‘pulse alone’ condition for both conditions. That is, instead of focussing on the specific peaks of response to the pulse in the ‘with prepulse’ condition, which are morphed by the prepulse, we focussed on the net neural response of the system at the time frames corresponding to the N1 and P2 of the ‘pulse alone’ condition. The logic here is that the processing (N1 response) of the pulse in the ‘with prepulse’ condition is still happening at this time-point, only the processing of the prepulse is added on top. Thus, the net neural response at this time may represent key processing differences between the ‘pulse-alone’ and pulse ‘with prepulse’ which are associated with how the stimuli are perceived. For consistency we applied this method to both experiments. Therefore, we searched for the maximal peak on the grand-average data at Cz (between 50 and 170 for N1 and 140 – 300 ms for P2). The measured windows for Experiment 1 were, N1 (71 – 121 ms) and P2 (143 – 193 ms). For Experiment 2, the measured windows were N1 (91 – 141 ms) and P2 (179 – 229 ms).

## 2.4. Statistical analysis

### 2.4.1. Experiment 1 – PPIPSI and Cortical PPI (Tactile)

All statistical analyses were conducted using R software (v3.5.1; R Foundation for Statistical Computing, Vienna, Austria). We aimed to investigate whether a relationship existed between cortical PPI (N1-P2 magnitude reduction) and PPIPSI. Because an N1-P2 response is elicited by both pulse conditions in a single PPIPSI trial, to aid interpretation of their relationship with perceived intensity, we created a N1-P2 amplitude trial difference variable by subtracting the N1-P2 response to the ‘with prepulse’ condition from the N1-P2 response to the ‘pulse alone’ condition. We fit multinomial baseline logit models using the ‘mlogit’ function from the ‘mlogit’ package 0.8.5.1 (Elf, 2020) to model the predicted odds of participants perceived intensity choice (three levels: pulse alone more intense, pulse with prepulse more intense or felt the same) as a function of Cz N1-P2 amplitude difference on a given trial.

‘Pulse alone more intense’ was the reference category as it was the most frequent category and reflects the occurrence of PPIPSI, and participant ID was entered as the random factor to account for subject-level variation and repeated measurement.

### 2.4.2. Experiment 2 – PPIPSI, Cortical PPI, and attentional load (Acoustic)

Statistical analyses were conducted using the same software as Experiment 1. A final sample of 24 participants (20 female; age  $M(\text{SD}) = 24.04(7.02)$ , range = 18 – 48) were used for analyses. We conducted a trial-level multinomial baseline logit analysis to model the relationship between attentional load (levels: easy and hard), Cz N1-P2 amplitude difference, and perceived intensity responses, with participant ID as the random factor. Again, ‘Pulse alone more intense’ was used as the reference category as it was the most frequent category and reflects the occurrence of PPIPSI.

To investigate the possible effect of attentional load on Cz N1-P2 magnitude, we conducted a linear mixed model (LMM) analysis using a logistic regression model at the trial level. Attentional load (‘Block’) was set as the fixed effect predictor, and participant ID as the random factor. For this LMM we used the ‘gamljMixed’ function from the ‘gamlj’ package (Gallucci, 2019). To gain further insight into the effect (or lack thereof) attentional load has on N1-P2 magnitude, Bayesian analyses were conducted using the ‘generalTestBF’ function from the ‘BayesFactor’ package (v0.9.12; Morey et al., 2018).

## 3. Results

### 3.1. Experiment 1 – Tactile

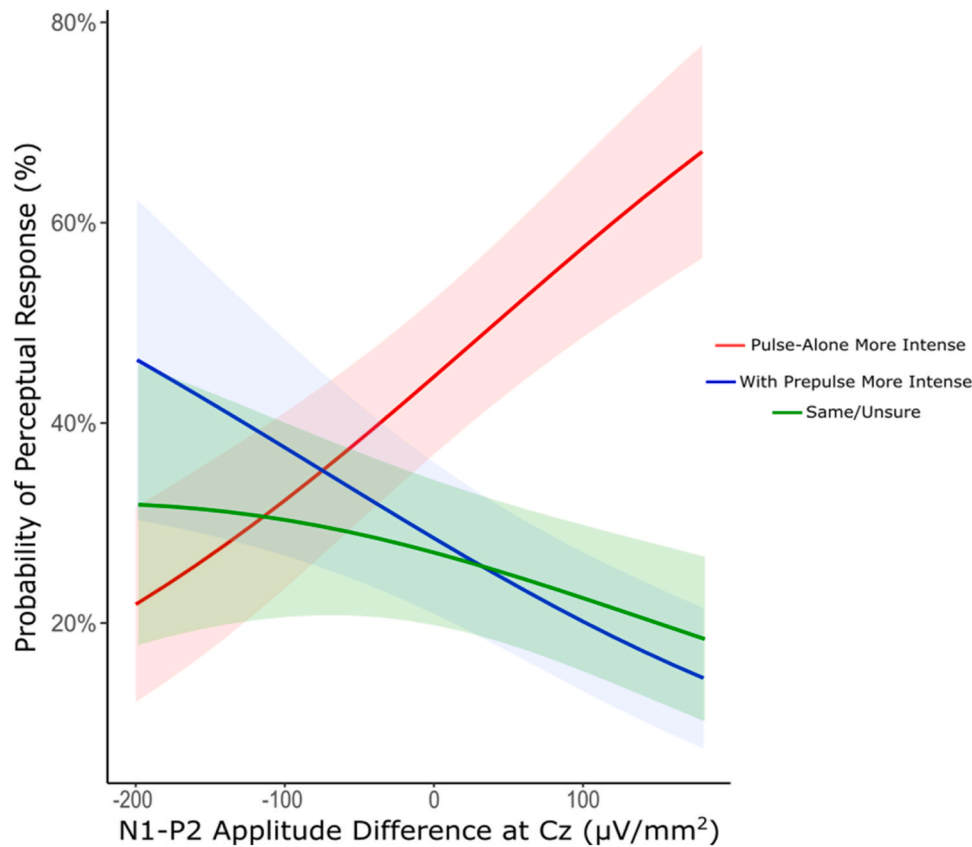
#### 3.1.1. Association between Cortical PPI and PPIPSI

In this experiment, the ‘pulse-alone’ condition was perceived more intense on 43.19% of trials (chance level = 33.33%), providing evidence of perceptual gating. The analysis that follows seeks to determine whether cortical responses can influence perception. The multinomial logit model revealed that Cz N1-P2 magnitude difference was a statistically significant predictor of perception in both baseline comparisons. In the first comparison, ‘with prepulse’ more intense vs ‘pulse alone’ more intense the estimate for Cz N1-P2 magnitude difference was  $-.0059$  (SE = 0.001,  $z = 4.40$ ,  $p < .001$ ; OR = 0.994, 95% CI [0.990, 0.999]), indicating that as Cz N1-P2 magnitude difference increases, the odds of perceiving ‘pulse alone’ more intense also increase. In the second comparison, ‘same’ vs ‘pulse alone’ more intense the estimate for Cz N1-P2 magnitude difference was  $-.0043$  (SE = 0.001,  $z = -3.35$ ,  $p < .001$ ; OR = 0.996, 95% CI [0.992, 0.999]). Similar to the first comparison, as Cz N1-P2 magnitude differences becomes more positive the odds of perceiving the ‘pulse alone’ more intense increase, and the odds of perceiving the pulse-stimuli as the same decrease. The pattern of results depicted in Fig. 2 shows that when the N1-P2 difference is more negative (meaning the N1-P2 response was larger in the ‘with prepulse’ condition) the probability of perceiving the ‘pulse with prepulse’ more intense is higher. Conversely, when the N1-P2 difference was more positive (meaning the N1-P2 response was larger in the ‘pulse alone’ condition) there is a higher probability of perceiving the ‘pulse-alone’ as more intense. These patterns are consistent with our hypothesis that inhibition of the N1-P2 response to the pulse, elicited by the prepulse, is associated with PPIPSI.

### 3.2. Experiment 2 – Acoustic

#### 3.2.1. Association between Cortical PPI and PPIPSI

Here, the ‘pulse-alone’ condition was perceived more intense on 51.60% of trials (chance level = 33.33%), evidencing perceptual gating. The analysis that follows seeks to determine whether cortical responses can influence perception. Results from the multinomial logistic regression evidenced that Cz N1-P2 magnitude difference was not a statistically significant predictor of perception in the first comparison, ‘with



**Fig. 2.** Predicted Probabilities and Standard Error (SE) for each Perceptual Response Option as a Function of Cz N1-P2 Magnitude Difference (CSD-transformed =  $\mu\text{V}/\text{mm}^2$ ). Note. The figure demonstrates that when N1-P2 magnitude difference is more negative, the probability of perceiving the ‘with prepulse’ condition more intense or perceiving them the ‘same’ is higher. Alternatively, the probability of perceiving the ‘pulse-alone’ more intense increases as the N1-P2 magnitude difference becomes more positive.

prepulse’ more intense vs ‘pulse alone’ more intense ( $z = -0.89$ ,  $p = 0.37$ ; OR = 0.999, 95% CI [0.996, 1.002]). However, Cz N1-P2 magnitude difference was a statistically significant predictor of perception between ‘same’ vs ‘pulse alone’ more intense, estimate =  $-0.005$  (SE =  $-0.001$ ,  $z = -3.84$ ,  $p < .001$ ; OR = 0.995, 95% CI [0.992, 0.999]). As depicted in Fig. 3, the results indicate a relationship whereby, the more positive the Cz N1-P2 magnitude difference, the higher the probability of perceiving the ‘pulse-alone’ more intense.

### 3.2.2. Attentional load influences the observation of PPIPSI

The multinomial logistic regression also found that attentional load (‘block’) was a statistically significant predictor of perception in both comparison equations. In the first comparison, ‘with prepulse’ more intense vs ‘pulse alone’ more intense the estimate effect of attentional load (‘block’) was 0.44 (SE = 0.135,  $z = 3.26$ ,  $p < .001$ , OR = 1.552, 95% CI [1.09, 2.22]). In the second equation, ‘same’ vs ‘pulse alone’ more intense, the estimate effect of attentional load (‘block’) was 0.676 (SE = 0.15,  $z = 4.61$ ,  $p < .001$ ; OR = 1.96, 95% CI [1.32, 2.74]). For both comparisons, the results indicate that the probability of observing PPIPSI (‘pulse-alone’ more intense perceptions) decreased with increasing attentional load.

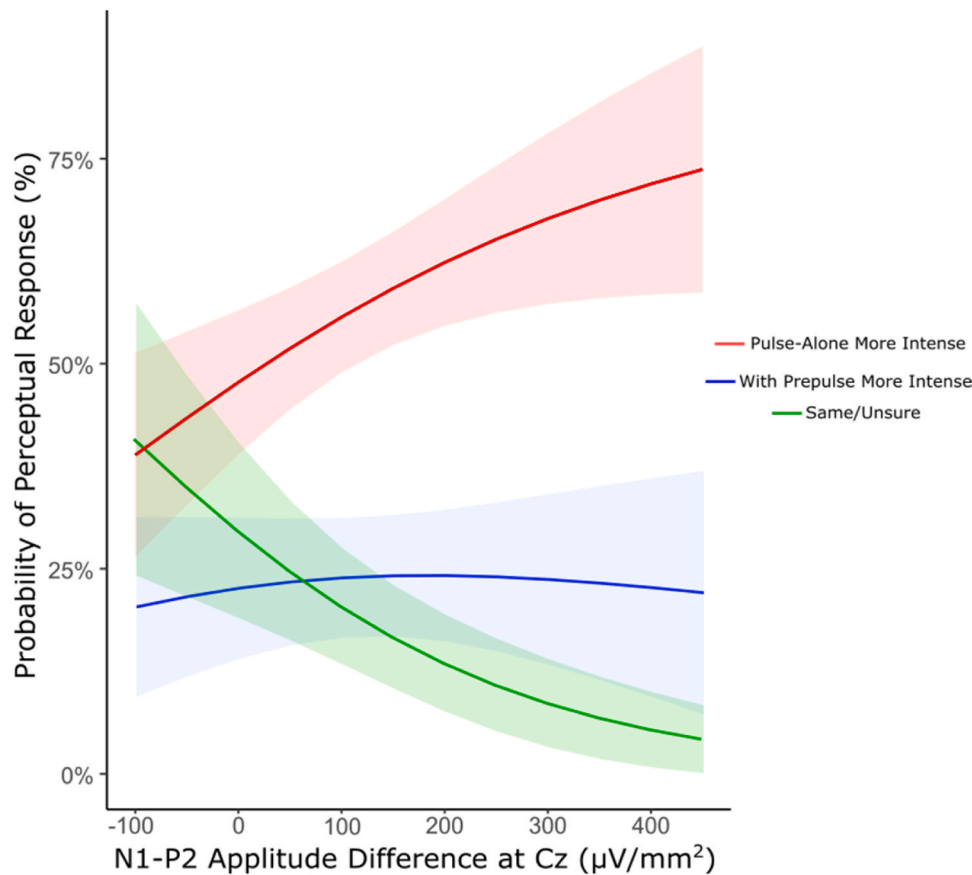
### 3.2.3. Effect of attentional load on Cz N1-P2 magnitude difference

The LMM analysis revealed no significant effects of attentional load on Cz N1-P2 amplitude difference ( $F(1, 1356) = 2.97$ ,  $p = 0.08$ ), with ‘Easy Block’ (M(SE) = 111.15 (11.50)), and ‘Hard Block’ (M(SE) = 105.90(11.47)). A follow-up Bayesian analysis to investigate the evidence for the null effect of attentional load on Cz N1-P2 amplitude difference produced a  $\text{BF}_{01}$  of 4.31, providing substantial evidence for the absence of an effect ( $H_0 = 4.31$  times more likely than  $H_1$ ; Jeffreys,

1961).

## 4. Discussion

In the present study we investigated the relationship between electrotactile (Experiment 1) and acoustic (Experiment 2) PPIPSI and cortical PPI, as measured by the ERP magnitudes during the N1-P2 complex timeframe. In other words, we examined how the magnitude of cortical suppression, reflected in the reduction of N1-P2 amplitudes to a pulse stimulus when preceded by a prepulse, relates to the perception of the pulse’s intensity. In Experiment 2, we also examined the influence attentional load has on cortical responses and perceived intensity. In both modalities we identified that on a given trial, the larger the N1-P2 response magnitude to a pulse-stimulus is, the higher the probability participants will perceive that stimulus as more intense. Specifically, the more effectively the prepulse inhibits N1-P2 response magnitude to the pulse (‘with prepulse condition’), the greater the difference will be from the N1-P2 to that same intensity pulse presented alone. As this difference increases, so too does the probability participants will perceive the ‘with prepulse’ less intense, demonstrating PPIPSI. These findings support previous suggestions that the processes captured in cortical PPI may contribute to the inhibition of conscious perception or perceived intensity seen in PPIPSI (Swerdlow et al., 2007). In Experiment 2, we extended our understanding of the influence attention has on cortical responses and perceived intensity. In more detail, we found that attentional load impedes the observation of PPIPSI but does not modulate the inhibitory effect a prepulse has on the N1-P2 response to a subsequent pulse (cortical PPI).



**Fig. 3.** Predicted Probabilities and Standard Error (SE) for each Perceptual Response Option as a Function of Cz N1-P2 Magnitude Difference (CSD-transformed =  $\mu\text{V}/\text{mm}^2$ ). Note. The figure demonstrates that when N1-P2 magnitude difference is more negative, the probability of perceiving both pulses the 'same' intensity is higher. Conversely, as the N1-P2 magnitude difference becomes increasingly positive, the probability of perceiving the 'pulse-alone' more intense also increases.

#### 4.1. The relationship between PPIPSI and Cortical PPI

Cortical PPI at central brain areas (e.g., Cz) has been suggested to represent attentional and encoding processes (Kedzior et al., 2007). Consistent with this, studies evidence that the N1 ERP represents processing of a stimulus' physical characteristics (e.g., intensity), while the P2 represents stimulus classification and decision-making processes (Annic et al., 2014; García-Larrea et al., 1992). Each of these event-related potentials have been hypothesised as reflecting processes that contribute to the phenomenon of PPIPSI (Favero et al., 2022; Swerdlow et al., 2007). Thus, the current findings of a relationship between the gating of these cortical responses (cortical PPI) and PPIPSI support both the functional representation of the N1-P2 complex and their involvement in gating at the perceptual level. In other words, the reduction in these cortical responses, which represent encoding of stimulus characteristics such as intensity, correlate with the reduction in perceived intensity that is PPIPSI. This suggests that the processes captured in the N1-P2 complex are reflecting key factors that affect the perceptual experience of PPIPSI.

#### 4.2. The influence of attention on PPIPSI, Cortical PPI, and their relationship

Paradigm assessments by and Favero et al. (2022) indicate that PPIPSI relies on attentional and self-monitoring mechanisms. This has been inferred from the task designs in studies of PPIPSI and time-course evidence. In PPIPSI experiments, participants are explicitly instructed to attend to the pulse stimulus to either assign an intensity rating (Swerdlow et al., 2005), or to compare and decide which pulse they perceive as more intense (with or without prepulse; Favero et al., 2022).

Therefore, studies of PPIPSI employ an active design involving directed attention to the pulse and self-monitoring of perceived intensity to make relative judgements. Additionally, PPIPSI peaks at longer SOAs than startle PPI, which does not require active engagement by participants (Graham et al., 1981; Ison et al., 1991; Silverstein et al., 1980). Acoustic PPIPSI is maximal at a SOA of 120 ms ( ), and electro-tactile PPIPSI emerges at SOAs of 160 ms+ (Favero et al., 2022). Consistent with these timings, attentional processes are said to be engaged around 120 ms after stimulus onset (Dawson et al., 1993). These findings led us to propose that PPIPSI is likely observed at longer SOAs than startle PPI because to perceive the effects of the prepulse on the subsequent pulse stimulus requires attention to be directed towards monitoring sensory inputs to the cortex. Moreover, that attention itself requires greater time, or a larger gap between prepulse onset and pulse onset to be sufficiently ready to monitor relevant sensory channels (Favero et al., 2022). The current study extends these propositions in two ways, first by providing evidence that attentional mechanisms are engaged in PPIPSI - manipulation of attentional load significantly affects the observation of PPIPSI. Specifically, when attentional load was higher, there was a significant increase in the number of trials where 'with prepulse' more intense and 'same' were perceived, and a reduction in 'pulse alone' more intense (PPIPSI) perceptions. Secondly, our findings provide evidence that attentional resources required for PPIPSI might be used to access stimulus traces or representations reflected within the N1-P2 cortical response.

We found a relationship between PPIPSI and N1-P2 magnitude that is consistent with the functional representation of the N1-P2 complex. These are the processing or encoding of a stimulus' physical characteristics (e.g. intensity), and stimulus classification (Annic et al., 2014; Kedzior et al., 2007; García-Larrea et al., 1992). Of course, it is possible

that the additional attentional load could have impacted the magnitude of the N1-P2 complex, rather than making it more challenging to monitor them as we hypothesized. Our finding that the N1-P2 magnitude was unaffected by attentional load, whereas PPIPSI was clearly impacted by our manipulation renders this explanation unlikely. In a cortical PPI study, [Annic et al. \(2014\)](#) observed an effect of stimulus-driven, and goal-directed attention to the prepulse on the N1 and P2 cortical responses. They found the N1 response to the pulse was inhibited more by stimulus-driven attention to the prepulse, while P2 was inhibited by goal-directed attention to the prepulse, when compared to a 'do not attend' condition. At first, our lack of an effect of cognitive load on the N1-P2 complex might seem contradictory to [Annic et al. \(2014\)](#) findings. However, differences in the designs of the two studies might explain this discrepancy. First, we manipulated attentional load, not goal or stimulus-driven attention. In addition, attention is directed towards the pulse in PPIPSI paradigms, while in [Annic et al. \(2014\)](#) attention was directed towards the prepulse, and the pulse was considered irrelevant. One possible synthesis of these differences is that [Annic et al. \(2014\)](#) investigated how allocation of attention to a prestimulus affects the cortical response to a subsequent, more intense stimulus; thus demonstrating that the inhibitory effect of a prepulse on the N1 and P2 cortical responses to a subsequent stimulus is enhanced by stimulus-driven and goal-directed attention to the prepulse. This indicates that attention driven by stimulus properties (stimulus-driven) and goal-directed attention to the prepulse influence PPI by modulating the pulse stimulus's sensory characteristics (e.g., intensity) and cognitive evaluation ([Annic et al., 2014](#)). In our experiment, we manipulated the availability of attentional resources to be allocated towards the pulse-stimuli (sensory inputs), appraising their characteristics (i.e., intensity) and evaluating these characteristics in comparison to each other. When attention is goal-directed or stimulus-driven by the prepulse, the cortical response to the pulse is further inhibited ([Annic et al., 2014](#)), but general manipulation of the availability of attentional resources does not affect the cortical response (as shown here in Experiment 2). These results suggest that attention to the prepulse enhances the inhibitory effects of the prepulse, but attentional load has little effect on the information that reaches the cortex. Conversely, attentional load appears to impair PPIPSI. This finding suggests that PPIPSI may partially rely on the allocation of attentional resources to monitor cortical responses so that accurate perceptual inferences can occur.

It may be argued that the attentional load tasks were not difficult enough to interfere with N1-P2 attentional processing, or that participants prioritised the perception task. To check this, we conducted a paired t-test to compare counting task performance between the Easy and Hard conditions. The results revealed that the average percentage of correct responses was significantly higher in the Easy Task ( $M = 91.42$ ,  $SD = 27.22$ ) compared to the Hard Task ( $M = 29.44$ ,  $SD = 45.60$ ),  $t(1439) = 46.80$ ,  $p < .001$ , 95% CI [0.60, 0.65]. The high percentage correct in the Easy Task indicates that participants were engaging with the attentional load task. It is also evident that participants performed worse in the Hard Task, suggesting it was sufficiently difficult to recruit more attentional resources. These data, combined with the finding that attentional load influenced PPIPSI observation, make these alternative explanations unlikely. The current findings are consistent with [Näätänen and Winkler's \(1999\)](#) model of auditory processing. The N1-P2 complex likely reflects pre-attentive encoding of a stimulus trace or representation. Furthermore, the link between cortical responses and conscious perception appears to be via the perceptual mechanisms directing attention towards cortical processes. This direction of attention towards the cortical responses then brings their contained information into conscious perception ([Näätänen and Winkler, 1999](#)).

#### 4.2.1. Prepulse as temporal cue

One further way attention may modulate PPIPSI is by the prepulse acting as a temporal cue for the timely allocation of attentional resources. [Annic et al. \(2014\)](#) demonstrated that a salient prepulse recruits

stimulus-driven attention, enhancing cortical PPI. Although we did not manipulate attention to the prepulse directly, it is likely that a prepulse of perceptible intensity, at an SOA which makes it perceivably distinct from the pulse, engages a degree of stimulus-driven attention. Consistent with this, we previously proposed that in combination with inhibiting processing of the pulse, in PPIPSI, the prepulse may facilitate directing attention towards monitoring inputs to the cortex ([Favero et al., 2022](#)). Our findings of a relationship between PPIPSI and cortical PPI, and the effect of attentional load support the proposition that attentional resources are required for more reliable observation of PPIPSI.

Experiments in other paradigms have demonstrated that temporal predictability facilitates the allocation of cognitive resources to task relevant processes ([Nobre & van Ede, 2018](#)). For example, in motor preparation experiments where a response to a target stimulus must be made as fast as possible, the introduction of a warning stimulus reduces reaction time (RT; [Alegria, 1975](#); [Müller-Gethmann et al., 2003](#)). This is said to occur because the resources used to hold motor responses in a high state of preparation are finite, only optimal 100 – 300 ms prior to target stimulus onset ([Alegria, 1975](#); [Müller-Gethmann et al., 2003](#)). Thus, presenting a warning signal approximately 200 ms prior to the target stimulus facilitates the optimal allocation of resources towards motor preparation, reducing reaction time. Moreover, when a movement is being prepared, suppression of corticospinal excitability occurs, known as preparatory suppression ([Hasbroucq et al., 1997](#)). However, under time constraints which limit movement preparation, preparatory inhibition does not occur, leading to greater response disruption by external stimuli ([McInnes et al., 2021](#)). Similarly, studies have evidenced that attentional orienting facilitated by a cue is most effective when presented 100 – 200 ms prior to a target ([Nakayama & MacKeben, 1989](#); [Egeth & Yantis, 1997](#)). In visual perception, anticipatory cues presented 100 ms before a target enhance visual target representations and delay interference by distractor stimuli on the target's cortical representation ([van Ede et al., 2018](#)). [van Ede et al. \(2018\)](#) interpret this enhanced target detection and minimised interference by distractor stimuli as indication that cued anticipation aids perception by enabling a 'protective temporal window' from distractor interferences ([van Ede et al., 2018](#)). The above findings from different research domains suggest a common mechanism may be involved. Consistent with [Brunia's \(1993\)](#) proposition that motor and attention processes use similar mechanisms, the attention allocated to perceiving the pulse (monitoring the sensory input channels) likely cannot stay in an optimal state the entirety of a trial in PPIPSI paradigms, because the timing of pulse presentations is uncertain. Therefore, the prepulse may (even if indirectly) facilitate the allocation of attention towards monitoring inputs to the cortex which process the pulse-stimulus. This may contribute to why PPIPSI requires longer SOAs than startle PPI, because the attentional shift towards monitoring sensory inputs aided by the prepulse requires greater time.

Here, we demonstrate that increased attentional load hinders PPIPSI. If the time-course of PPIPSI is modulated by the allocation of attentional resources to monitor inputs to the cortex, PPIPSI may be observable at shorter SOAs if attention can be allocated faster or more efficiently. This could be investigated by manipulating temporal predictability. Studies show that expectation assists the allocation of attention ([Zhao et al., 2013](#)), and selective attention aids perception by prioritising sensory inputs based on their salience or relevance to tasks aims ([Desimone & Duncan, 1995](#); [Nobre & van Ede, 2018](#); [Summerfield and de Lange, 2014](#)). Therefore, making the presentation of the prepulse and pulse stimuli temporally predictable (presented at the same time in each trial), might speed directed attention towards monitoring inputs to the cortex. We are not proposing that this is the mechanism by which the prepulse inhibits responses to the pulse, but that this is a way by which cortical changes induced by prepulses might enter consciousness more easily. Cortical PPI at central locations is elicited using SOAs of 50 – 140 ms ([San-Martin et al., 2018](#)). Based on our results, indicating that the inhibition of the N1-P2 cortical response is linked to perceived intensity,



and previous findings that larger cortical inhibition might be more prominent at shorter SOAs (San-Martin et al., 2018), we propose that the manipulation of temporal predictability using cues could enhance the observation of PPIPSI at shorter SOAs, matching that of cortical PPI.

#### 4.3. Similarities and differences between tactile and acoustic modalities

The general finding from the current experiments is that the relationship between N1-P2 magnitude and PPIPSI is observed for both tactile and acoustic stimuli. However, it must be highlighted that in the tactile modality (Experiment 1), N1-P2 magnitude differences were a significant predictor of perceptual responses at both levels of comparison ('pulse-alone more intense' vs 'with prepulse more intense' and 'pulse-alone more intense' vs 'same'). In contrast, in the acoustic modality, N1-P2 magnitude difference was a significant predictor of perceptual response only for the 'pulse-alone more intense' vs 'same' comparison in Experiment 2. Differences in results across experiments may be explained by differences in stimulus parameters (e.g. intensity), and influence of the additional task in Experiment 2. In Experiment 2, the influence of the attentional load task likely affected the relationship between N1-P2 magnitude difference and perceptual responses. This is evident from the increase in 'same' responses between high and low attentional load conditions. One explanation is that attentional load increases perceptual uncertainty resulting in participants selecting 'same/unsure'. Subsequently, weakening the relationship between N1-P2 magnitude difference and perceptual comparisons of 'pulse-alone more intense' and 'with prepulse more intense'. This possibility may be investigated further by reproducing Experiment 2 with a greater number of trials, and no manipulation of attentional load.

Previous studies suggest that startle PPI and PPIPSI are sensitive to prepulse and pulse intensity (Blumenthal et al., 2015; Swerdlow et al., 2005). Acoustic studies have found that both startle PPI and PPIPSI increase with increasing prepulse and pulse intensity, to a certain threshold, after which decrements are observed with further intensity increases (Swerdlow et al., 1999, 1999b; Swerdlow et al., 2007). In Experiment 1, we used parameters previously identified as optimal for electrocutaneous PPIPSI (Favero et al., 2022), and because electrocutaneous stimuli are more subjectively aversive than acoustic (Sperl et al., 2016) this entailed individualised intensity settings as opposed to predetermined ones. By contrast, in experiments 2 we used predetermined intensities for our stimuli, informed by maximal acoustic PPIPSI findings (Swerdlow et al., 2005; 2007). It is possible that the use of individualised intensities in Experiment 1 led to weaker overall stimulus intensities, particularly for the prepulse which was set to 2x perceptual threshold (M (SD) intensity = 0.70 mA (0.39) compared to an 80 dB prepulse in the acoustic experiments. Although translating stimulus intensity across modalities is difficult, the N1-P2 magnitudes were larger in the acoustic experiments, indicating the stimuli were physically more intense (Mulert et al., 2005; Paiva et al., 2016; Rosburg et al., 2008). Moreover, the acoustic prepulse appears to have had a greater inhibitory effect on the N1-P2 response to the pulse. Thus, it may be that the intensity of the acoustic stimuli was more effective in eliciting gating, but the link between cortical and perceptual gating is weakened – possibly due to the overlapping of prepulse and pulse N1-P2 signals caused by the higher intensity prepulse and shorter SOA.

#### 4.4. Limitations and considerations

Previous studies have identified sex dimorphism and menstrual cyclicity effects on baseline startle PPI measurements (Swerdlow et al., 1999, 1999b). As such, the predominantly female sample in the current study deserves consideration. While PPI and PPIPSI may share lower-level gating mechanisms, previous studies have shown that PPIPSI is not affected by sex differences or menstrual cyclicity (Swerdlow et al., 2005). Nonetheless, we found that in both experiments group proportion of PPIPSI trials and directional effects were qualitatively similar

between males and females: Experiment 1 (M(SD) proportion of PPIPSI trials for males = 0.57(0.42), and females = 0.57(0.40), and Experiment 2 (M(SD) males = 0.59(0.44), and females = 0.64(0.41). These results suggest the imbalanced sample is unlikely to have affected our findings.

Our application of a surface Laplacian filter which converts the EEG data to CSD should be considered when interpreting and comparing the current data with other studies. Surface Laplacian is a mathematical transformation applied to EEG surface potentials designed to mitigate EEG signals reference dependence and masking of smaller signals by volume conduction (Kayser & Tenke, 2015). The surface Laplacian enhances EEG signals' spatial resolution by dampening ERP components with broad spatial distributions, which would otherwise mask smaller transient signals in the EEG (Gevins et al., 1995; Nunez & Srinivasan, 2014). We applied this filter because each trial contained two stimuli of interest, and a concern was that the late ERP signals to the first stimulus—typically long with broad spatial distributions—could interfere with the early ERPs of the second stimulus, such as the N1/P2 complex which were of specific interest. Consequently, the surface Laplacian was used to emphasise the transient and focal N1/P2 complex by filtering away ERP components of less interest, particularly those with broad spatial distributions. Lastly, because the surface Laplacian is computed from signal differences which filter out lower frequency components, one further concern is that these transformed signals may become more susceptible to noise (Bradshaw & Wikswow, 2001). Noise, including recording artifacts, tends to be of high spatial frequencies and, therefore, by amplifying higher spatial frequencies, Laplacian transformation can increase noise representation in the signal (Bradshaw & Wikswow, 2001). However, noise added by the surface Laplacian filter is unlikely to be a major issue in the current study due to the high quality of our recorded data (Kayser & Tenke, 2015; see the waveforms and scalp maps for non-transformed data in the Supplementary Materials).

## 5. Conclusion

In the present study we investigated the relationship between cortical PPI and PPIPSI. We also examined the effect attention has on both by manipulating attentional load. We observed a relationship between N1-P2 magnitude difference and relative judgments of perceived intensity, which produced a pattern of results consistent across both electrocutaneous and acoustic modalities. When the N1-P2 magnitude was larger for a specific pulse-stimulus, the probability that pulse would be perceived more intense was higher. This means that when the prepulse effectively inhibited the N1-P2 response to the subsequent pulse (cortical PPI), the probability that participants would perceive the 'pulse-alone' more intense increased (demonstrating PPIPSI). Our findings are consistent with studies which attribute the N1-P2 response to processes of encoding stimulus characteristics such as intensity (Annic et al., 2014; García-Larrea et al., 1992; Mulert et al., 2005; Paiva et al., 2016; Rosburg et al., 2008). In addition, our results extend this evidence by demonstrating a link between N1-P2 gating, and gating of conscious perception within the PPI/PPIPSI paradigm. This suggests a relationship by which the processes captured in the N1-P2 response contribute to the perceptual experience of PPIPSI. We identified that increased attentional load impedes the observation of PPIPSI, providing evidence in support of propositions that perceptual gating involves the recruitment of attentional processes (Favero et al., 2022; Swerdlow et al., 2005). Moreover, the finding that attentional load affects PPIPSI, but not cortical PPI further characterises their relationship. It appears that what reaches the cortex (N1-P2) is largely free of attentional processes, but to consciously perceive that gating has occurred requires directing attention to processes captured in these cortical responses. That is, consistent with Näätänen and Winkler (1999), these cortical processes are likely preattentive and driven by stimuli features, after which attention is used to access this information and form a conscious perception. We conclude that PPIPSI may be observed at longer SOAs than startle PPI because, even if driven indirectly by the prepulse, directing attention to the

appropriate cortical processes requires more time. Given that cortical PPI is observed using shorter SOAs (50–80 ms; San-Martin et al., 2018), we propose that if attention can be directed to these processes faster or more optimally, the time-course of PPIPSI may too be shortened. A future study could investigate this by manipulating temporal predictability, which has been found to enhance the allocation of attentional resources and facilitate perception in other studies (Desimone & Duncan, 1995; Nobre & van Ede, 2018; Summerfield and de Lange, 2014; Zhao et al., 2013).

### Ethics approval

All procedures performed in this series of experiments were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by the Human Research Ethics Committee (EC00262) of Curtin University (HRE2018–0257).

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### Declaration of Competing Interest

The authors have no financial or proprietary interests in any material discussed in this article.

### Data availability

Data files and analysis scripts (R code) can be accessed at <https://doi.org/10.7910/DVN/A7BLOC>.

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### Declarations

#### Declaration of generative AI in scientific writing

During the preparation of this work the author(s) did not use generative AI during any part of the writing process.

### Consent to participate

Informed consent was obtained from all individual participants included in both experiments.

### Consent for publication

When receiving and providing informed consent, all participants consented to have their data de-identified (replaced with a code) for submission to the journal.

### Availability of data materials and code availability

Data files and analysis scripts (R code) can be accessed at <https://doi.org/10.7910/DVN/A7BLOC>.

### Open Practices Statement

The data and materials for all experiments are available at the request of the authors ([j.favero@postgrad.curtin.edu.au](mailto:j.favero@postgrad.curtin.edu.au) or [welber.marinovic@curtin.edu.au](mailto:welber.marinovic@curtin.edu.au)), and none of the experiments were preregistered.

### Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.biopsycho.2023.108711](https://doi.org/10.1016/j.biopsycho.2023.108711).

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