European Journal of Pain

ORIGINAL ARTICLE OPEN ACCESS

Contextual Modulation of Fronto-Central, Parietal, and Global Peak Alpha Frequency

Carolina Ceruti[1] | Laura Petrini[1] | Elia Valentini[2] | Michelle Bonvini[1] | Giulia Erica Aliotta[1] | Kristian Hennings[1] | Carsten Dahl Mørch[1] | Dennis Boye Larsen[1]

[1]Center for Neuroplasticity and Pain (CNAP), Faculty of Medicine, Department of Health Science and Technology, Aalborg University, Aalborg, Denmark | [2]Department of Psychology and Centre for Brain Science, University of Essex, Essex, UK

Correspondence: Dennis Boye Larsen (dbl@hst.aau.dk)

Received: 5 January 2026 | **Revised:** 10 September 2026 | **Accepted:** 10 September 2026

ABSTRACT

Background: Peak alpha frequency (PAF), the dominant frequency in the alpha band (8–12 Hz), has been proposed as a biomarker of pain sensitivity, but mixed findings may reflect broad sensory and cognitive influences on PAF. Operant conditioning can modulate pain perception and related brain activity, but its effect on PAF has not been explored. This study investigated if PAF was lower in the context of punishment compared to PAF in the context of reward and whether these contexts differentially modulate PAF when pain intensity is held constant.

Methods: Twenty-nine healthy participants performed an auditory task while receiving painful pressure stimuli within a reward and punishment context, achieved via feedback on response correctness followed by a consequential change in pressure (maintained pressure with relief and maintained pressure with increase). Electroencephalography was recorded continuously, and PAF was extracted from frontal, central, parietal, and global regions of interest.

Results: PAF was significantly lower after an incorrect response in the reward context, compared to a correct response in the punishment context, despite matched pain intensity. A more consistent finding was that PAF was lower following an incorrect response in the punishment context (increased pain) compared to a correct response in the same context (maintained pain).

Conclusions: These findings suggest that PAF is modulated by ongoing pain intensity within a fixed motivational context, while evidence for context-specific effects (reward vs. punishment) remains preliminary and warrants replication.

Significance Statement: This study introduces a paradigm in which task performance determined whether pressure pain increased or decreased, to test whether context shapes peak alpha frequency (PAF) independently of pain intensity. Lower PAF was observed when pain increased, suggesting that PAF tracks pain intensity when context is held constant. A preliminary, cross-context difference in PAF at matched pain intensities warrants replication in an adequately powered sample.

1 | Introduction

Chronic pain remains one of the largest contributors to years lived with disability (Mills et al. 2019), and current therapies do not sufficiently manage pain for all patients (Wang and Doan 2024). One reason may be that the neural processes that shape the pain experience are still unclear, which result in symptomatic rather than targeted treatment. Brain activity measures such as peak alpha frequency (PAF), that is, the frequency with maximum power in the alpha frequency band (8–12 Hz), reflect sensory processing (van Diepen and Mazaheri 2017; Foxe and Snyder 2011; Klimesch 2012). Slower PAF is associated with higher pain sensitivity in experimental pain models (Furman et al. 2018; Nir et al. 2010), chronic neurogenic pain (Sarnthein et al. 2006) and Complex Regional Pain Syndrome (Walton et al. 2010). PAF has therefore been suggested as a biomarker

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2026 The Author(s). *European Journal of Pain* published by John Wiley & Sons Ltd on behalf of European Pain Federation - EFIC ®.

of pain sensitivity (Furman et al. 2018). However, contrasting evidence has demonstrated that PAF is not specific to pain (Valentini et al. 2022) or pain sensitivity (May et al. 2025), but rather sensory experiences (Valentini et al. 2015) or psychological aspects (Schmidt et al. 2012) that involve more than just pain perception. Since experimental pain models only mimic certain features of clinical pain, they do not account for the context in which pain emerges and evolves. This may partly explain the discrepancies in PAF literature. An approach to study the context of pain is to use reward and punishment and how pain is experienced and maintained. Event-related potential studies demonstrated that chronic pain patients exhibit dysregulated reward processing as reflected by a reduction in anticipatory responses to reward compared to healthy controls (Baker et al. 2022). Fibromyalgia patients exhibit reduced attentional reward processing as compared to healthy controls (Park et al. 2025) and reduced reward circuitry activity, especially in the dopaminergic system (Loggia et al. 2014). These results indicate that chronic pain disturbs reward processing which may manifest as reduced reward sensitivity measured by feedback-related negativity and P300 (Ma et al. 2022). This, however, has never been investigated using PAF. Understanding the mechanisms by which reward context and motivational significance of pain-related feedback (Wu and Zhou 2009) influence PAF, may provide further evidence on the feasibility of PAF as a potential biomarker for the pain experience, especially when that pain experience is controlled.

This study aimed to investigate PAF in a reward and punishment context, and if the same level of pain would modulate PAF differently depending on the condition. We hypothesized that equal painful pressure would yield lower PAF in the context of punishment compared to PAF in the context of reward. The reward and punishment contexts were achieved via correctness feedback and direction of pressure stimulation (decrease or increase), without behavioural or subjective validation (manipulation check) that the conditions were experienced as rewarding or punishing.

2 | Methods

2.1 | Participants

Twenty-nine healthy Danish participants (mean age 26.25 ± 5.29; 18 women) were recruited through local notices and social media. Participants with chronic pain, neurological disorders (e.g., epilepsy), musculoskeletal issues, mental health conditions (such as depression), diabetes mellitus (risk of peripheral neuropathy), recent or current use of substances known to affect pain processing, hearing impairments, or pregnancy were excluded. All volunteers provided informed consent and were compensated 150 DKK per hour for their time in the lab. The local ethics committee approved the study (approval number N-20220014), and it was conducted in accordance with the Declaration of Helsinki.

2.2 | Experimental Design

The order of the starting conditions was randomized for each participant to either begin with the decrease in pressure pain (DP) or increase in pressure pain (IP) condition (see Figure 1; Consequence phase). Importantly, the gender balance starting in the DP or the IP condition was approximately similar ($\chi^2(1) = 0.12$, $p = 0.728$), removing gender imbalance as a confounder of starting order. The experiment consisted of three consecutive sessions, each separated by a 15-min break, with each session comprising four blocks of 10 trials, totalling 120 trials overall. The participants were always told which condition they were in for each block. Four familiarization trials were conducted to ensure that participants understood the procedures and rating scales. Additionally, the pressure was calibrated to elicit painful sensations at visual analogue scale (VAS) levels of 3, 5 (defined as pain threshold), and 7. Moreover, the noise level was adjusted based on the signal-to-noise ratio for the Hearing-in-Noise Task (HINT).

Throughout the experiment, electroencephalographic (EEG) signals were recorded as participants completed the HINT task concurrent with pressure pain stimulation (VAS5 was maintained throughout each trial and only changed during the consequence phase). The intensity of the subsequent pain stimulus was determined by performance: While pain intensity remained constant at VAS 5 during the HINT task, it was adjusted based on outcomes where the reward (correct answers) was a reduction in pressure stimulation in the DP condition and unchanged pressure stimulation in the IP condition, and the punishment (incorrect answers) was unchanged pressure stimulation in the DP condition and increased pressure stimulation in the IP condition. Auditory feedback was provided before the ensuing pressure stimulation to indicate whether the performance was correct or incorrect, and both reward and punishment stimulation lasted for 15 s (Figure 2).

2.3 | The Hearing-In-Noise-Test

The hearing task was done with Hearing-in-Noise-Test (HINT) (Nielsen and Dau 2011) which consists of several sentences made of five words mixed with white noise. The task was to listen, memorize, and repeat the sentences. A total of 250 randomized sentences were available, and participants were randomly exposed to 120 of these through earphones.

A correct answer was considered when all the five words of the sentences were repeated while an incorrect answer was considered if less than five words were repeated. The Signal-to-Noise ratio was calibrated at the beginning of every session to obtain a 50–50 rate between correct and incorrect answers.

2.3.1 | Signal-To-Noise Ratio Calibration

The SNR psychometric function was estimated and adjusted to 50% using the Psi-method (Kontsevich and Tyler 1999), as implemented in the Palamedes Toolbox (Prins and Kingdom 2018). Specifically, participants listened to 20 different HINT sentences presented at various noise levels and then repeated them. The experimenter recorded a 'yes' if the entire sentence was correctly repeated and a 'no' otherwise. The SNR was calibrated prior to each session.

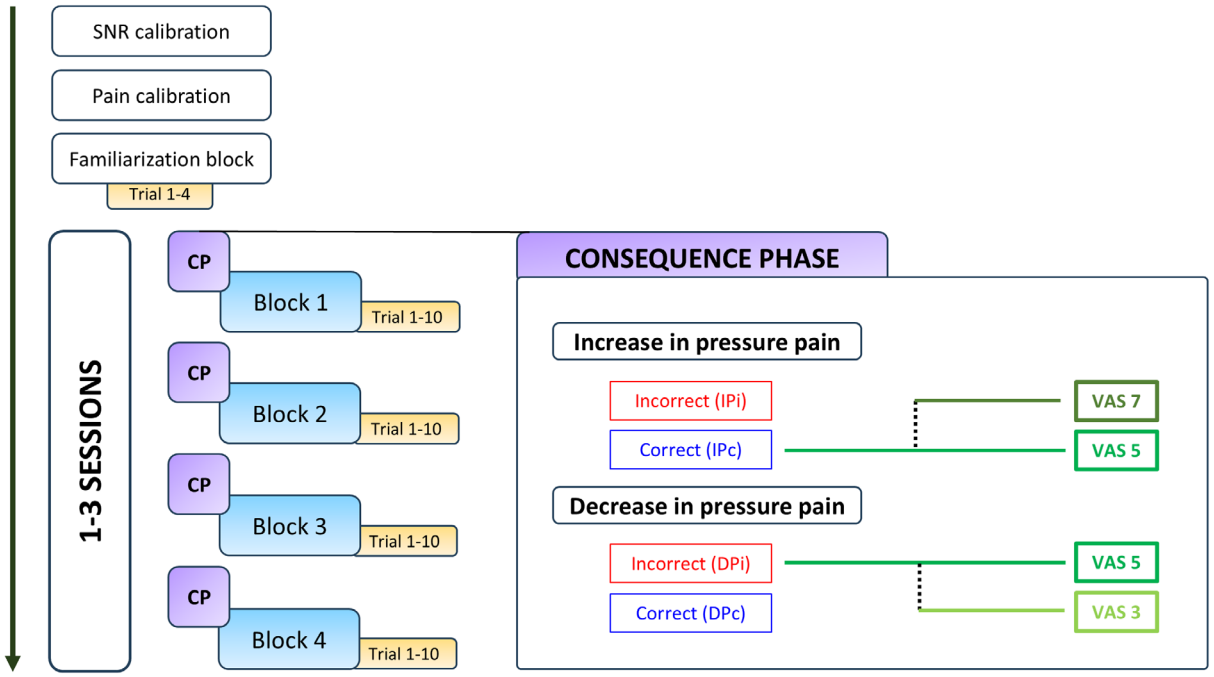


FIGURE 1 | Consequence phase: prior to each session, both the signal-to-noise ratio (SNR) and the pressure cuff intensity for painful stimulation were calibrated. The experiment consisted of 120 trials, divided into three sessions, each comprising four blocks of 10 trials. At the start of each block, participants were informed of the upcoming condition, either increase in pressure pain or decrease in pressure pain. During the Hearing-in-Noise Test (HINT) task, pressure pain stimulation was maintained at each participant's pain threshold (corresponding to a score of 5 on a visual analogue scale). The outcome of each trial fell into one of two conditions: (1) in the DP condition, participants received pain relief as a reward or no change in pressure as punishment; and (2) in the IP condition, no change in pressure served as the reward, while an increase in pressure constituted the punishment.

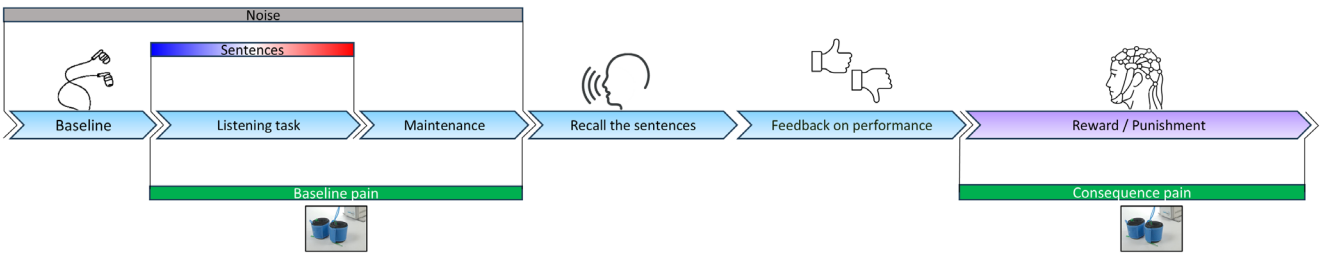


FIGURE 2 | Experimental design: each trial began with a baseline period during which participants were exposed to background noise. Following this, they engaged in a listening task involving HINT sentences composed of five words. Participants were required to memorize the sentence during a maintenance phase and then repeat it. Based on their performance, they received auditory feedback in the form of two distinct tones—one indicating a correct response and the other indicating an incorrect one. This feedback also determined whether they received a reward or a punishment.

2.4 | Tonic Pressure Pain

Tonic pressure stimulation was administered through a cuff algometer (a silicon high-pressure cuff, Nocitech Aps) positioned on the left calf at the gastrocnemius level. The cuff was connected to a computer that controlled its inflation rate and pressure. Participants continuously rated their pain using a hand-held dial and could press a button to immediately stop the cuff inflation if the pain became unbearable. The maximum pressure of the cuff was 100 kPa (760 mmHg). Participants used a VAS to rate their sensation: 0 indicating no sensation, 5 the pain threshold, and 10 the worst pain imaginable. Prior to the experiment, a calibration determined the pressure levels corresponding to VAS 3, 5, and 7. Initially, the pressure was set at VAS 5, and the operant target was adjustments to that pressure based on the experimental condition: in the DP condition, a correct response reduced the pressure to VAS 3 while an incorrect response maintained it at VAS 5; in the IP condition, a correct response kept the pressure at VAS 5, whereas an incorrect response increased it to VAS 7. Decreased and increased stimulation intensities were induced in 1 s and maintained for 15 s. Throughout the experiment, the pressure never exceeded VAS 7.

2.4.1 | Tonic Pressure Pain Calibration

The pressure increased at a rate of 1 kPa per second, and participants continuously rated their sensations using a digital visual analogue scale (VAS) ranging from 0 (no sensation at

all), 5 (pain threshold), to 10 (worst pain imaginable) (Graven-Nielsen et al. 2015). This procedure was repeated three times before each session, and the average ratings for VAS 3 (no pain), VAS 5 (pain threshold), and VAS 7 (pain) were calculated. Across the three sessions, there were no significant differences in pressure needed to elicit VAS3 (mean ± SD: 21.88 ± 9.54 kPa [S1]; 23.04 ± 11.92 kPa [S2]; 20.71 ± 8.68 kPa [S3], $F_{2,56} = 1.06$, $p = 0.35$), VAS5 (mean ± SD: 29.91 ± 11.63 kPa [S1]; 32.66 ± 14.73 kPa [S2]; 31.27 ± 11.58 kPa [S3], $F_{2,56} = 1.87$, $p = 0.16$), or VAS7 (mean ± SD: 40.05 ± 14.71 kPa [S1]; 42.36 ± 17.26 kPa [S2]; 41.24 ± 14.96 kPa [S3], $F_{2,56} = 0.78$, $p = 0.47$).

2.5 | Electroencephalographic Recording

The electroencephalographic (EEG) signal was recorded through 64 channels (g.Scarabeo) placed on the scalp of the subjects according to the 10–20 international system. Reference electrodes (A1 and A2) were placed on the earlobes, and the ground electrode was placed on AFz. The impedance of the electrodes was kept under 10 kΩ, and the signal was amplified and sampled at 1200 Hz (g.HIamp biosignal amplifier g.Tec medical engineering GmbH, Schiedlberg, Austria). To allow the correct extraction of the EEG epochs, several triggers including auditory signals on task performance and feedback, and cuff inflation, were used to enable the alignment between the EEG signal and the pressure stimulation.

2.6 | Data Analysis

2.6.1 | EEG Data Pre-Processing

The EEG data were pre-processed through EEGlab (Delorme and Makeig 2004) and Matlab (v.2021b) functions. Data were resampled at 512 Hz, and the powerline noise interference was removed with Cleanline (https://www.nitrc.org/projects/cleanline). The data were high-pass filtered at 1 Hz (IIR Butterworth filter, 8th order) and referenced to A1 and A2. Subsequently, an ICA decomposition using ICLabel (Pion-Tonachini et al. 2019) was performed and optimized by the 1-Hz high-pass filtering, following general recommendations (Klug and Gramann 2021). The ICA weights were transposed on the signal, band-pass filtered (IIR Butterworth filter, 8th order) between 0.1 and 40 Hz, and bad ICA components were removed (number of rejected components was not logged and is therefore not available).

EEG data were epoched using the trigger for the consequence pressure stimulation. The signal was extracted from −2 s pre-trigger of cuff inflation to 15 s post-trigger of cuff inflation and baseline corrected normalizing the signal (Groppe et al. 2009). This was done in the time-domain prior to power spectral estimation. The data were extracted from the frontal region (F3, Fz, F4), the central region (Cz, C3, C4), and the parietal region (Pz, P3, P4) electrodes (McLain et al. 2022), epoched from 1 s post trigger of cuff inflation and up to 15 s post-trigger and divided per condition (DP and IP) and per answer (correct and incorrect). Regions of interest (ROIs) were not defined a priori but followed guidelines from McLain et al. (2022).

2.6.2 | Peak-Alpha-Frequency

To quantify the individual peak alpha frequency (PAF), we computed the centre of gravity (CoG) within the alpha band (8–12 Hz) for each EEG channel and condition. Specifically, EEG data were epoched and stored in separate EEGLAB.set files per subject and condition. For each file, the EEG epochs were kept and used to compute power spectral density (PSD) per channel using Welch's method (Hamming window, 50% overlap, 512-point FFT). The PSDs were estimated for every epoch of 14 s. The CoG was then calculated for each channel as a weighted average of frequency bins within the alpha band, using the formula: $\mathrm{CoG} = \sum(f \times P[f]) / \sum P[f]$, where f is the frequency and $P(f)$ is the power at that frequency bin. This computation was repeated separately for each epoch within each condition and subject, yielding a single-trial PAF value per epoch (~40 epochs per condition per subject = 120 epochs across four conditions). Regional PAF summaries were also computed by averaging across predefined clusters of electrodes as listed in Section 2.6.1.

2.6.3 | Statistical Analysis

All statistical analyses were performed in Statistical Package for Social Sciences (SPSS, IBM, v.29). To investigate how the context of pain influences PAF, a series of mixed linear models were conducted for each of the three regions of interest (frontal, central, and parietal) as well as the global PAF. Single trial PAF was investigated with restricted maximum likelihood (REML) estimation (Satterthwaite method), with random intercepts and slopes for participants, which do not require equal cell sizes. Mean trial counts per condition were: DPc = 36.4 ± 5 (range 29–46), DPi = 23.6 ± 5 (range 14–31), IPc = 37.2 ± 5.4 (range 20–43), and IPi = 22.8 ± 5.4 (range 17–40). Repeated measures included conditions (decrease pain correct (DPc), decrease pain incorrect (DPi), increase pain correct (IPc), increase pain incorrect (IPi)) and epochs, and an autoregressive (AR1) covariance structure was specified to account for unequal covariance (Regis et al. 2022). Epochs were included as a covariate and time effect on PAF across trials. Alpha was set to 0.05 for statistical significance. All comparisons were between post-inflation conditions. Contrast comparisons were conducted against the reference condition (IPc) within each regional model, yielding three comparisons that were corrected for multiple testing using false discovery rate (FDR) (Benjamini and Hochberg 1995). Cohen's dz was computed from subject-level condition means and reflects the standardized within-subject effect size.

3 | Results

3.1 | Global PAF

The mixed linear model demonstrated significant main effects of condition ($F_{3,2047} = 3.160$, $p = 0.024$) and epochs ($F_{1,1459} = 3.907$, $p = 0.048$). There was a small decline in PAF across epochs over time (Estimate = −0.00048, $p = 0.048$). Pairwise comparisons for condition demonstrated significantly lower PAF for DPi compared to IPc (mean difference = 0.017 Hz, $p_{FDR} = 0.039$, $dz = -0.3$) and significantly lower for IPi compared to IPc (mean difference = 0.018 Hz, $p_{FDR} = 0.039$, $dz = -0.50$) (Figure 3). The

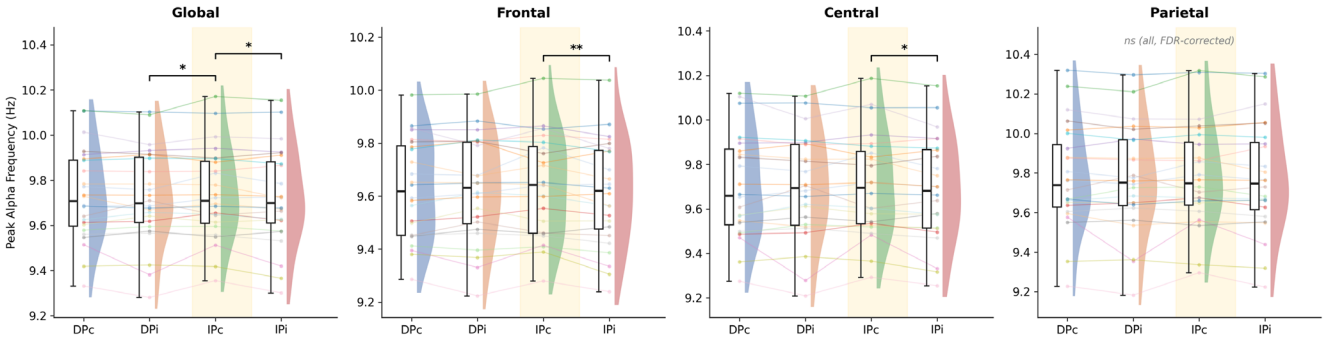


FIGURE 3 | Peak alpha frequency (PAF). Rainbow plot representation of the global, frontal, central, and parietal PAF distribution within each condition. Global PAF: the decrease pain incorrect (DPi) condition demonstrated significantly slower PAF compared to increase pain correct (IPc) and increase pain incorrect (IPi) was significantly slowed compared to IPc. *$p < 0.05$. Frontal PAF: the IPi (Increase pain incorrect) demonstrated significantly slower PAF compared to IPc (Increase pain correct). **$p < 0.01$. Central PAF: the IPi (Increase pain incorrect) condition demonstrated significantly slower PAF compared to IPc (Increase pain correct). *$p < 0.05$. Parietal PAF: no significant differences were found across conditions. Box represents the 25th and 75th percentile and the median with min/max whiskers. Dots are raw values for each participant. KDE distribution is shown as shapes.

model estimated a subject variance of 0.042 (standard error ±0.012), and residual variance for the repeated measures (epochs and condition) of 0.016. Within-subject variability was 0.116–0.127 Hz across conditions.

3.2 | Frontal PAF

The mixed linear model demonstrated a significant main effect of condition ($F_{3,2029} = 4.580$, $p = 0.003$) and epochs ($F_{1,1431} = 6.967$, $p = 0.008$). There was a small decline in PAF across epochs over time (Estimate = −0.001, $p = 0.008$). Pairwise comparisons for condition demonstrated significantly lower PAF for IPi compared to IPc (mean difference = 0.03 Hz, $p_{FDR} = 0.001$, $dz = -0.65$) (Figure 3) with a tendency for significance between IPc and DPi (mean difference = 0.017 Hz, $p_{FDR} = 0.078$, $dz = -0.23$). The model estimated a subject variance of 0.037 (standard error ±0.011), and residual variance for the repeated measures (epochs and condition) of 0.023. Within-subject variability was 0.141–0.149 Hz across conditions.

3.3 | Central PAF

The mixed linear model demonstrated a significant main effect of condition ($F_{3,2852} = 3.089$, $p = 0.026$) but not epochs ($F_{1,2852} = 3.127$, $p = 0.077$). Pairwise comparisons for condition demonstrated significantly lower PAF for IPi compared to IPc (mean difference = 0.025 Hz, $p_{FDR} = 0.024$, $dz = -0.48$) (Figure 3). The model estimated a subject variance of 0.054 (standard error ±0.016), and residual variance for the repeated measures (epochs and condition) of 0.024. Within-subject variability was 0.145–0.154 Hz across conditions.

3.4 | Parietal PAF

The mixed linear model did not demonstrate significant main effects of condition ($F_{3,2087} = 1.745$, $p = 0.156$) or epochs ($F_{1,1532} = 0.503$, $p = 0.479$). There was not a decline in PAF across epochs over time (Estimate = 0.0002, $p = 0.479$). The model estimated a subject variance of 0.069 (standard error ±0.021), and residual variance for the repeated measures (epochs and condition) of 0.024. Within-subject variability was 0.141–0.155 Hz across conditions.

4 | Discussion

The current study showed that global PAF modulation by painful pressure stimulation is, as hypothesized, dependent on the context in which the stimulation is delivered. We found lower PAF after an incorrect answer in the DP condition compared to a correct answer in the IP condition, despite the controlled pressure at VAS 5 in both conditions. This may indicate that PAF is not affected by pain perception alone but is rather an interplay between context and perception. Frontal and central PAF were similarly lower on incorrect versus correct IP trials. Conversely, parietal PAF was not affected by either condition or time. Substantial between-subject variance in PAF was clear across all regions. Global and frontal PAF showed small but significant declines over epochs, suggesting gradual cognitive fatigue or decreased attentional engagement, while this was not the case for central and parietal PAF.

4.1 | Lower PAF Is Dependent on the Context in Which the Painful Stimulation Is Delivered

This study, for the first time, demonstrated that lower global PAF was dependent on the context in which the same pressure pain intensity was experienced, even when accounting for individual variance in PAF. This finding adds to earlier findings that PAF is not specific to pain intensity per se (May et al. 2025), as lower PAF was contextually dependent on an incorrect answer in the DP condition, and mirrors the slowing seen after experimental pain (Furman et al. 2018; Millard et al. 2023). This may indicate that PAF reflects the sensory experience of pressure pain in the given context, rather than pain intensity per se, as the pressure intensity was kept at VAS5 (i.e., pain detection threshold) (Foxe and Snyder 2011; Klimesch 2012; Sarnthein et al. 2006). This builds on recent evidence that showed slowing of PAF to

both experimental pain and an unpleasant non-painful auditory stimulus (Valentini et al. 2022). One possible explanation is that the contextual situation of DPi was considered an actual punishment, which is supported by the finding that the relative estimate of change in the DPi-IPc (0.017 Hz) and IPi-IPc (0.018 Hz) comparisons were similar. Such contextual shifts in perception have earlier been demonstrated, where moderate pain was rated as pleasant when it followed stronger pain (relative relief), but unpleasant when compared to no pain (Leknes et al. 2013), which may partly explain the lower PAF in the current study where the unchanged pressure pain is evaluated in context of pain relief as the reward. This interpretation, however, is only one of several plausible contributors. First, no measures of valence or perceived reward or punishment was recorded. Incorrect trials are inherently associated with prediction errors that may drive alpha dynamics independently from reward or punishment (Recasens et al. 2018). However, alpha oscillations are thought to reflect expectancy to sensory feedback, while gamma oscillations reflect prediction error (Bauer et al. 2014), and post-stimulus alpha oscillations track sensory intensity rather than expectancy (Nickel et al. 2022). As DPi and IPc involved matched pain intensity, prediction error alone cannot fully explain this modulation. Second, outcome evaluation is tightly linked to the correct/incorrect conditions in the current study. This aligns with reinforcement learning and performance monitoring, and how action, outcome, and effort are associated (Holroyd and Coles 2002). When an error occurs, alpha oscillations are suppressed and predict post-error behaviour (Li et al. 2023). Whether this would also manifest as lower PAF remains to be investigated. Condition-block awareness likely also allowed participants to anticipate the trajectory of pressure changes within the condition, introducing an anticipatory component that cannot be dissociated from trial-level prediction error or outcome evaluation in the current design. Third, PAF has been proposed as a neural index of cognitive preparedness (Angelakis et al. 2004) and general performance may have contributed to the lower PAF observed. As an example, within-subject fluctuations in alpha frequency are associated with performance, where correct discrimination of sensory stimuli yield higher alpha frequency compared to incorrectly discriminated stimuli (Samaha and Postle 2015). In this context, lower PAF in the incorrect conditions may reflect a general reduction in cognitive resource engagement, independent of the contextual framework. Incorporating trial-by-trial measures of, for example, valence and cognitive effort, and simultaneous measures of task-state PAF would allow for disentangling the reward and punishment-based framework from prediction error, general performance monitoring and outcome evaluation. As the observed effect size estimate is small-to-medium, this finding is preliminary and in need of future replication in a prospectively powered study, that further includes resting-state PAF at baseline.

4.2 | Higher Pain Intensity Resulted in Lower PAF

The current study demonstrates lower global and fronto-central PAF in incorrect IP trials. Lower PAF with increasing pressure aligns with PAF as a trait-like predictor of pain sensitivity in healthy participants (Furman et al. 2018). Later work showed that PAF slows in response to ongoing tonic pain (Furman et al. 2019), which aligns with our finding from the incorrect IP trials. The current findings reflect short, tonic pressure pain and are not directly comparable to earlier state-like modulation of PAF in models of longer-term nerve-growth factor induced pain that closely aligns with prolonged musculoskeletal pain progression (Furman et al. 2019). Recent evidence questions if PAF is indeed a biomarker for pain intensity (May et al. 2025), or rather a trait-level marker of vulnerability to prolonged pain (Furman et al. 2019). The observed differences are modest, but this is expected, as meaningful differences in PAF, in relation to, for example, pain sensitivity, operate in a narrow window where large shifts are not expected (Furman et al. 2020). The global PAF modulation shown in the current study is likely related to fronto-central PAF as this ROI consistently showed lower PAF for incorrect trials in the IP condition. The absence of parietal findings may be informative rather than inconclusive, as the epoch-level model structure and within-subjects design provided sensitivity to detect medium-sized effects, if present. This is consistent with earlier evidence on resting-state EEG, where phasic pain slowed fronto-central PAF that became more globally distributed during prolonged pain (Furman et al. 2019). It could be speculated that the parietal null findings reflect the relatively short duration of tonic pressure pain used in this study and lack the broader topographical distribution of responses as seen in prolonged pain (Furman et al. 2019; McLain et al. 2022). Importantly, direct clinical translation is premature, as the trait-level biomarker literature is derived from resting-state EEG. The interpretation that lower task-state PAF during tonic pressure pain increases may reflect aversive pain processing, is speculative and contingent on further replication. Whether lower task-state PAF during contextual pain maps onto slower resting-state PAF is an important question for future work.

4.3 | Limitations

A key limitation is that EEG was recorded during painful stimulation, rather than true resting-state, so PAF reflects a pain-influenced brain state rather than a baseline, trait-like neural marker (Furman et al. 2020). Eyes open/eyes closed, and pain-free comparisons were not performed as our interest was relative PAF modulation across pain contexts. This limits generalizability to resting-state PAF findings but preserves valid within-subject comparisons, since pain intensity was matched across conditions, and are more likely to reflect the influence of cognitive context on cortical processing during pain (May et al. 2025; Valentini et al. 2022), but direct comparison with resting-state PAF literature is precluded. Laterality was not tested given model complexity relative to sample size, and evidence suggesting broad scalp distribution for PAF-pain associations (Furman et al. 2020), but should be investigated in future studies. A small monotonic drift across sessions, consistent with temporal/fatigue-related effects cannot explain condition differences, as (1) DP and IP blocks were counterbalanced; (2) incorrect and correct trials within the IP block were positioned at matched temporal positions; and (3) epochs were included as a covariate, partialling out linear time trends. However, task demand and sustained attention effects on PAF cannot be fully ruled out without an explicit resting-state control. The absence of manipulation checks (valence, perceived reward/punishment) limits our ability to rule out prediction error (Recasens et al. 2018) and cognitive modulation (Holroyd and Coles 2002). Future studies should include manipulation checks at trial-by-trial or block-level, to verify the intended

motivational context. Menstrual cycle was not recorded, and with a predominantly female sample, estradiol-dependent gamma-aminobutyric (GABA) modulation of PAF cannot be excluded but is unlikely since recordings were done on the same day, and the mixed-model random intercept.

While the sample size may limit statistical power to detect small effects, the use of linear mixed-effects models enhances robustness by accounting for inter-individual variability (Barr et al. 2013). A sensitivity power analysis (repeated measures model with $\alpha = 0.05$ and $\beta = 0.8$) indicated a conservative minimum detectable effect of $dz = 0.5$, and replication in larger samples is necessary for definite conclusions. Likewise, gender influences (Brötzner et al. 2014) could not be tested as the study had 80% power to detect a gender × condition interaction of 0.036–0.037 Hz that is substantially larger than the condition effects reported.

4.4 | Conclusion

PAF was lower during more intense pain even when motivational context was held constant, and showed a preliminary, cross-context difference at matched pain intensities. These findings contribute to understanding how task-state PAF is modulated during painful stimulation.

Author Contributions

This study was designed by C.C., L.P., and C.D.M. The experiment was set up by C.C. and K.H. and performed by C.C. The data were analysed by C.C., M.B., G.E.A., and D.B.L., and the results were critically examined by all authors. C.C. and D.B.L, had the primary role in preparing the manuscript, which was edited by E.V., L.P., M.B., G.E.A., and C.D.M. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Acknowledgements

The authors would like to express their gratitude to Eva Nedergaard Søholm and Sofia Hennings for helping in the laboratory during data collection.

Funding

Center for Neuroplasticity and Pain (CNAP) is supported by the Danish National Research Foundation (DNRF121).

Disclosure

AI (Claude.ai) was used for improving readability of the manuscript in certain paragraphs.

Conflicts of Interest

Kristian Hennings is the owner of LabBench ApS and kindly supported the study in the form of equipment and working hours in the lab. Carsten Dahl Mørch holds a non-controlling position in Inventor's Way ApS. All other authors have no conflicts of interest to declare.

References

Angelakis, E., J. F. Lubar, S. Stathopoulou, and J. Kounios. 2004. "Peak Alpha Frequency: An Electroencephalographic Measure of Cognitive Preparedness." *Clinical Neurophysiology* 115: 887–897.

Baker, A. K., L. C. Ericksen, V. Koppelmans, et al. 2022. "Altered Reward Processing and Sex Differences in Chronic Pain." *Frontiers in Neuroscience* 16: 889849.

Barr, D. J., R. Levy, C. Scheepers, and H. J. Tily. 2013. "Random Effects Structure for Confirmatory Hypothesis Testing: Keep It Maximal." *Journal of Memory and Language* 68: 255–278.

Bauer, M., M.-P. Stenner, K. J. Friston, and R. J. Dolan. 2014. "Attentional Modulation of Alpha/Beta and Gamma Oscillations Reflect Functionally Distinct Processes." *Journal of Neuroscience* 34: 16117–16125.

Benjamini, Y., and Y. Hochberg. 1995. "Controlling the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing." *Source: Journal of the Royal Statistical Society. Series B, Methodological* 57: 289–300.

Brötzner, C. P., W. Klimesch, M. Doppelmayr, A. Zauner, and H. H. Kerschbaum. 2014. "Resting State Alpha Frequency is Associated With Menstrual Cycle Phase, Estradiol and Use of Oral Contraceptives." *Brain Research* 1577: 36–44.

Delorme, A., and S. Makeig. 2004. "EEGLAB: An Open Source Toolbox for Analysis of Single-Trial EEG Dynamics Including Independent Component Analysis." *Journal of Neuroscience Methods* 134: 9–21.

Foxe, J. J., and A. C. Snyder. 2011. "The Role of Alpha-Band Brain Oscillations as a Sensory Suppression Mechanism During Selective Attention." *Frontiers in Psychology* 2: 154.

Furman, A. J., T. J. Meeker, J. C. Rietschel, et al. 2018. "Cerebral Peak Alpha Frequency Predicts Individual Differences in Pain Sensitivity." *NeuroImage* 167: 203–210.

Furman, A. J., M. Prokhorenko, M. L. Keaser, et al. 2020. "Sensorimotor Peak Alpha Frequency Is a Reliable Biomarker of Prolonged Pain Sensitivity." *Cerebral Cortex* 30: 6069–6082.

Furman, A. J., T. Thapa, S. J. Summers, et al. 2019. "Cerebral Peak Alpha Frequency Reflects Average Pain Severity in a Human Model of Sustained, Musculoskeletal Pain." *Journal of Neurophysiology* 122: 1784–1793.

Graven-Nielsen, T., H. Bjarke, S. Finocchietti, G. Handberg, and L. Arendt-nielsen. 2015. "Assessment of Musculoskeletal Pain Sensitivity and Temporal Summation by Cuff Pressure Algometry: A Reliability Study."

Groppe, D. M., S. Makeig, and M. Kutas. 2009. "Identifying Reliable Independent Components via Split-Half Comparisons." *NeuroImage* 45: 1199–1211.

Holroyd, C. B., and M. G. H. Coles. 2002. "The Neural Basis of Human Error Processing: Reinforcement Learning, Dopamine, and the Error-Related Negativity." *Psychological Review* 109: 679–709.

Klimesch, W. 2012. "Alpha-Band Oscillations, Attention, and Controlled Access to Stored Information." *Trends in Cognitive Sciences* 16: 606–617.

Klug, M., and K. Gramann. 2021. "Identifying Key Factors for Improving ICA-Based Decomposition of EEG Data in Mobile and Stationary Experiments." *European Journal of Neuroscience* 54: 8406–8420.

Kontsevich, L. L., and C. W. Tyler. 1999. "Bayesian Adaptive Estimation of Psychometric Slope and Threshold." *Vision Research* 39: 2729–2737.

Leknes, S., C. Berna, M. C. Lee, G. D. Snyder, G. Biele, and I. Tracey. 2013. "The Importance of Context: When Relative Relief Renders Pain Pleasant." *Pain* 154: 402–410.

Li, Q., S. Yin, J. Wang, et al. 2023. "Not All Errors Are Created Equal: Decoding the Error-Processing Mechanisms Using Alpha Oscillations." *Cerebral Cortex* 33: 8110–8121.

Loggia, M. L., C. Berna, J. Kim, et al. 2014. "Disrupted Brain Circuitry for Pain-Related Reward/Punishment in Fibromyalgia." *Arthritis and Rheumatology* 66: 203–212.

Ma, Q., W. Mao, and L. Hu. 2022. "Electrophysiological Evidence for the Effects of Pain on the Different Stages of Reward Evaluation Under a Purchasing Situation." *Frontiers in Psychology* 13: 1–9.

May, E. S., L. Tiemann, C. Gil Ávila, et al. 2025. "Assessing the Predictive Value of Peak Alpha Frequency for the Sensitivity to Pain." *Pain* 1: 1–15.

McLain, N. J., M. S. Yani, and J. J. Kutch. 2022. "Analytic Consistency and Neural Correlates of Peak Alpha Frequency in the Study of Pain." *Journal of Neuroscience Methods* 368: 109460.

Millard, S. K., A. K. I. Chiang, P. Humburg, et al. 2023. "Effects of Nicotine Compared to Placebo Gum on Sensitivity to Pain and Mediating Effects of Peak Alpha Frequency."

Mills, S. E. E., K. P. Nicolson, and B. H. Smith. 2019. "Chronic Pain: A Review of Its Epidemiology and Associated Factors in Population-Based Studies." *British Journal of Anaesthesia* 123: e273–e283.

Nickel, M. M., L. Tiemann, V. D. Hohn, et al. 2022. "Temporal–Spectral Signaling of Sensory Information and Expectations in the Cerebral Processing of Pain." *Proceedings of the National Academy of Sciences of the United States of America* 119: e2116616119.

Nielsen, J. B., and T. Dau. 2011. "The Danish Hearing in Noise Test." *International Journal of Audiology* 50: 202–208.

Nir, R. R., A. Sinai, E. Raz, E. Sprecher, and D. Yarnitsky. 2010. "Pain Assessment by Continuous EEG: Association Between Subjective Perception of Tonic Pain and Peak Frequency of Alpha Oscillations During Stimulation and at Rest." *Brain Research* 1344: 77–86.

Park, S. H., A. K. Baker, and K. T. Martucci. 2025. "Neural Correlates of Altered Reward-Driven Attention in Chronic Pain and Opioid Use." *Scientific Reports* 15: 14560.

Pion-Tonachini, L., K. Kreutz-Delgado, and S. Makeig. 2019. "ICLabel: An Automated Electroencephalographic Independent Component Classifier, Dataset, and Website." *NeuroImage* 198: 181–197.

Prins, N., and F. A. A. Kingdom. 2018. "Applying the Model-Comparison Approach to Test Specific Research Hypotheses in Psychophysical Research Using the Palamedes Toolbox." *Frontiers in Psychology* 9: 1–14.

Recasens, M., J. Gross, and P. J. Uhlhaas. 2018. "Low-Frequency Oscillatory Correlates of Auditory Predictive Processing in Cortical-Subcortical Networks: A MEG-Study." *Scientific Reports* 8: 14007.

Regis, M., P. Serra, and E. R. van den Heuvel. 2022. "Random Autoregressive Models: A Structured Overview." *Econometric Reviews* 41: 207–230.

Samaha, J., and B. R. Postle. 2015. "The Speed of Alpha-Band Oscillations Predicts the Temporal Resolution of Visual Perception." *Current Biology* 25: 2985–2990.

Sarnthein, J., J. Stern, C. Aufenberg, V. Rousson, and D. Jeanmonod. 2006. "Increased EEG Power and Slowed Dominant Frequency in Patients With Neurogenic Pain." *Brain* 129: 55–64.

Schmidt, S., J. R. Naranjo, C. Brenneisen, et al. 2012. "Pain Ratings, Psychological Functioning and Quantitative EEG in a Controlled Study of Chronic Back Pain Patients." *PLoS One* 7: e31138.

Valentini, E., S. Halder, D. McInnerney, J. Cooke, I. L. Gyimes, and V. Romei. 2022. "Assessing the Specificity of the Relationship Between Brain Alpha Oscillations and Tonic Pain." *NeuroImage* 255: 119143.

Valentini, E., K. Koch, V. Nicolardi, and S. M. Aglioti. 2015. "Mortality Salience Modulates Cortical Responses to Painful Somatosensory Stimulation: Evidence From Slow Wave and Delta Band Activity." *NeuroImage* 120: 12–24.

van Diepen, R. M., and A. Mazaheri. 2017. "Cross-Sensory Modulation of Alpha Oscillatory Activity: Suppression, Idling, and Default Resource Allocation." *European Journal of Neuroscience* 45: 1431–1438.

Walton, K. D., M. Dubois, and R. R. Llinás. 2010. "Abnormal Thalamocortical Activity in Patients With Complex Regional Pain Syndrome (CRPS) Type I." *Pain* 150: 41–51.

Wang, J., and L. V. Doan. 2024. "Clinical Pain Management: Current Practice and Recent Innovations in Research." *Cell Reports Medicine* 5: 101786.

Wu, Y., and X. Zhou. 2009. "The P300 and Reward Valence, Magnitude, and Expectancy in Outcome Evaluation." *Brain Research* 1286: 114–122.