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Neurobehavioural Effects of Fear Conditioning: Linking Freezing Behaviour to Prefrontal EEG Patterns

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Abstract: Stress has become an unavoidable part of daily life, shaping both behavioural and neural responses. This study investigated how fear conditioning influences defensive behaviour in rats, integrating standardised behavioural paradigms with prefrontal EEG monitoring. Twenty male Sprague Dawley rats were evaluated pre- and post-conditioning using Open Field, Elevated Plus Maze, Forced Swim, and Novel Object Recognition tasks. Fear conditioning produced a significant increase in anxiety and depression indicators, with conditioned rats spending less time in the open arms of the maze and more time immobile during forced swim testing. No significant differences emerged in exploratory or cognitive performance. EEG analyses revealed no global differences in prefrontal cortical activity between pre- and post-conditioning states. However, significant increases across all frequency bands were observed during freezing episodes, indicating a state-dependent modulation of prefrontal activity linked to defensive mechanisms. These findings suggest that aversive conditioning selectively impacts emotional and defensive domains rather than cognitive performance, with freezing behaviour reflecting a neurophysiological signature of adaptive coping strategies.

Keywords: stress; fear conditioning; freezing behaviour; EEG; prefrontal cortex; defensive mechanisms; deception.

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1. Introduction

Fear conditioning is an experimental paradigm used in non-human animal research to investigate the behavioural and neural mechanisms underlying threat processing and emotional regulation. Fear conditioning means the exposure to aversive stimuli to elicits a coordinated set of defensive responses, both autonomic and behavioural, that are evolutionarily conserved across mammalian species (Fanselow, 1994; LeDoux, 2000). These responses are regulated by an intricate mechanism of limbic and cortical regions, particularly the amygdala and prefrontal cortex, which are responsible for detecting, evaluating, and responding to threat (McNaughton & Gray, 2000; Pessoa, 2017).

In rodents, fear is conditioned typically through increased anxiety- and depression-like behaviours, as well as through defensive reactions such as freezing (Tovote et al., 2015). Widely used for fear conditioning in rodents are the following behavioural tests, such as the Open Field Test, Elevated Plus Maze, Forced Swim Test, and Novel Object Recognition (Porsolt et al., 1977; Carobrez & Bertoglio, 2005; Ennaceur & Delacour, 1988). These paradigms offer valuable results regarding the exploratory drive, emotional reactivity, and cognitive function, allowing for a multidimensional evaluation of the effects of fear and stress exposure.

Many cortical and subcortical regions are involved in the mechanism of fear conditioning and foremost among them prefrontal cortex seems to integrate sensory input, emotional evaluation, and action selection (Borkar & Fadok, 2021; Sandi & Haller, 2015). According to studies (Bienertova-Vasku et al., 2020; Mochizuki et al., 2025; Widman et al., 2019), performed on both animal and human subjects, aversive learning can modulate neural oscillations linked to attention, threat monitoring, and behavioural inhibition.

These oscillatory dynamics are frequently state-dependent, emerging most prominently during discrete defensive behaviours such as freezing, rather than as global shifts in neural activity (Zinebi et al., 2002; McEwen, 2007; Tovote et al., 2015).

The present study aimed to examine how fear conditioning affects emotional and defensive behaviour in rats, with a particular focus on state-dependent prefrontal activation during freezing. Using standardised behavioural tests (Open Field, Elevated Plus Maze, Forced Swim, Novel Object Recognition) and EEG recordings over the prefrontal cortex, we assessed how aversive stimulation modulates anxiety, depression, cognitive performance, and neural oscillatory patterns. By linking behavioural outcomes with neural activity, this study aimed to elucidate how fear selectively engages emotional networks rather than producing global alterations in cortical states.

2. Methodology

2.1. Animals

Twenty male Sprague Dawley rats (approximately 350 g at the time of testing) were used in this study. The animals were obtained at one month of age and allowed to acclimatise for two weeks prior to experimentation, in order to minimise stress associated with relocation and handling. They were housed individually in standardised cages (435 × 290 × 150 mm) under controlled environmental conditions (temperature 23–24 °C, relative humidity 30–60%, 12 h light/dark cycle). Food and water were provided ad libitum. All experimental procedures complied with EU Directive 2010/63/EU for the protection of animals used for scientific purposes, as well as with national legislation on animal welfare. The protocol was reviewed and approved by the Research Ethics Commission of the University of Bucharest (Decision no. 85/10.11.2022).

The group allocation (experimental $n = 15$, control $n = 5$) was chosen to prioritise statistical power for the study's primary within-subject comparisons (pre- vs post-conditioning), while limiting the total number of animals used in accordance with the 3Rs principle. Power calculations (two-tailed, $\alpha = 0.05$) indicate that, for paired (within-subject) comparisons, a sample of 15 animals provides acceptable power (≈ 0.82) to detect large effects (Cohen's $d \approx 0.8$) and very high power for larger effects ($d \geq 1.0$). The small control group ($n = 5$) was retained to provide a baseline estimate

of test–retest variability and to detect very large between-group differences, but the study was not powered to detect small or moderate between-group effects. This allocation thus focuses resources on the most informative contrasts (within-subject changes induced by conditioning), while minimising animal usage. We acknowledge the limited power for independent between-group tests and report effect sizes and confidence intervals for all comparisons to facilitate interpretation.

2.2. Fear Conditioning Protocol

Animals were randomly assigned to an experimental group (n = 15) or a control group (n = 5). This allocation minimised animal use while maintaining adequate statistical power. The experimental group underwent a five-day conditioning protocol (5 min/day), during which a visual stimulus (~100 lux) was consistently paired with an auditory stimulus (~80 dB), thereby establishing the visual cue as a conditioned aversive signal. Control animals were handled under identical conditions but were not exposed to paired stimuli. This procedure was designed to model naturalistic threat exposure and facilitate the emergence of defensive behaviours, such as freezing.

2.3. Behavioural Testing

All animals underwent a behavioural assessment before (T1) and after (T2) conditioning. Exploratory behaviour was evaluated using the Open Field Test (OFT), anxiety-like responses with the Elevated Plus Maze (EPM), depressive-like behaviour with the Forced Swim Test (FST), and recognition memory with the Novel Object Recognition (NOR) task. These paradigms provided an integrated behavioural profile of locomotion, emotional reactivity, and cognitive performance. During post-conditioning sessions, freezing behaviour was also recorded as a defensive response index. All tests were performed between 09:00 and 14:00 in a controlled environment, and the device was cleaned with 70% ethanol between trials.

2.4. EEG Recording and Signal Processing

Electrophysiological activity was recorded using an OpenBCI 4-Channel Ganglion Biosensing Board. Signals were continuously recorded during baseline and post-conditioning sessions. EEG data were preprocessed for artifact removal and analysed offline. Data sampling was performed at a frequency of 200 Hz. The board has a hardware band-pass filter with a lower cut-off frequency of 0.3 Hz. The surface EEG electrode system is of our own design and construction, containing two active exploratory electrodes for the bilateral frontal regions, a reference electrode placed midsagittally at the level of the binaural line, and the active ground electrode also placed midsagittally, posterior to the reference electrode. The placement of the electrodes was carried out taking into account the dimensions, anatomical conformation of the brain in the adult rat, and the bony landmarks on the surface of the cranial cavity, based on the serial section plates from the stereotaxic atlas of George Paxinos and Charles Watson: *The Rat Brain in Stereotaxic Coordinates*, 5th edition, Elsevier 2005. The scalp contact electrodes were made of a special silver sponge-type material used for wound dressing (TheraBond Antimicrobial Barrier Systems, 3DAC-48, Choice Therapeutics Inc.), with an elemental silver content of 15% (w/w). A printed microcircuit measuring 20 mm x 10 mm was designed and manufactured, containing on the lower side (in contact with the scalp) 4 electrode bonding pads and on the upper side 4 contact areas with a short cable for transmitting EEG signals to a connector on the main shielded cable for interfacing with the Ganglion EEG system. To further attenuate electrical interference noise from the power supply, a software notch filter set at 50 Hz and a band-pass filter with a passband of 1-50 Hz were used in the OpenBCI software, with which EEG monitoring and recording were performed. Power spectral density was computed using Fast Fourier Transform (FFT) for standard frequency bands (delta, theta, alpha, beta, gamma). Freezing and non-freezing epochs were segmented for state-dependent comparisons.

2.5. Statistical Analysis

Behavioural and EEG data were analysed using JASP and Python. Paired-samples t-tests were used to assess within-group differences (T1 vs. T2), and independent-samples t-tests were applied for between-group and state-dependent contrasts (freezing vs. non-freezing). The significance threshold was set at $p < 0.05$. Effect sizes and 95% confidence intervals were reported where appropriate.

3. Results

3.1. Fear Conditioning Increases Emotional Reactivity Without Affecting Cognitive Performance

Fear conditioning is known to modulate emotional and cognitive domains, often reflected in changes in exploratory, affective, and memory-related behaviours. To evaluate these effects, animals underwent behavioural testing using the Elevated Plus Maze, Forced Swim Test, Open Field Test, and Novel Object Recognition task.

In the Elevated Plus Maze (EPM), conditioned rats showed a marked reduction in the time spent in the open arms following conditioning (EPM1 M = 29.40, SD = 13.59; EPM2 M = 16.97, SD = 8.07), indicating heightened anxiety levels ($p = 0.00$, 95% CI [5.74, 19.12]) (see Figure 1). This behavioural profile is consistent with previous studies showing that exposure to aversive stimuli robustly enhances anxiety-like behaviour through activation of limbic–prefrontal networks (Borkar & Fadok, 2021; Zinebi et al., 2002). In contrast, the control group showed no significant differences between pre- and post-testing (EPM1 M = 10.5, SD = 6.25; EPM2 M = 11.14, SD = 2.17; $p = 0.81$, 95% CI [-8.80, 7.32]) (see Figure 2).

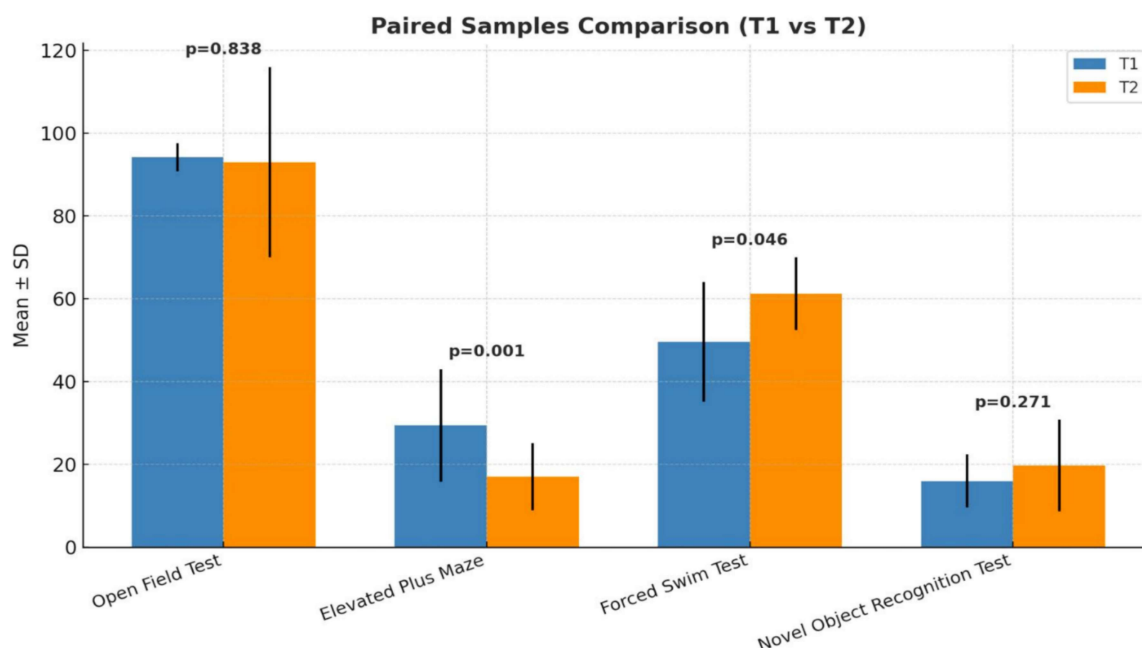


Figure 1. Behavioural test results for the experimental group (T1 = pre-conditioning; T2 = post-conditioning). Data represent performance in the Elevated Plus Maze, Forced Swim Test, Open Field Test, and Novel Object Recognition task for the experimental group across the two testing sessions. Statistical significance was assessed using paired-samples t-tests.

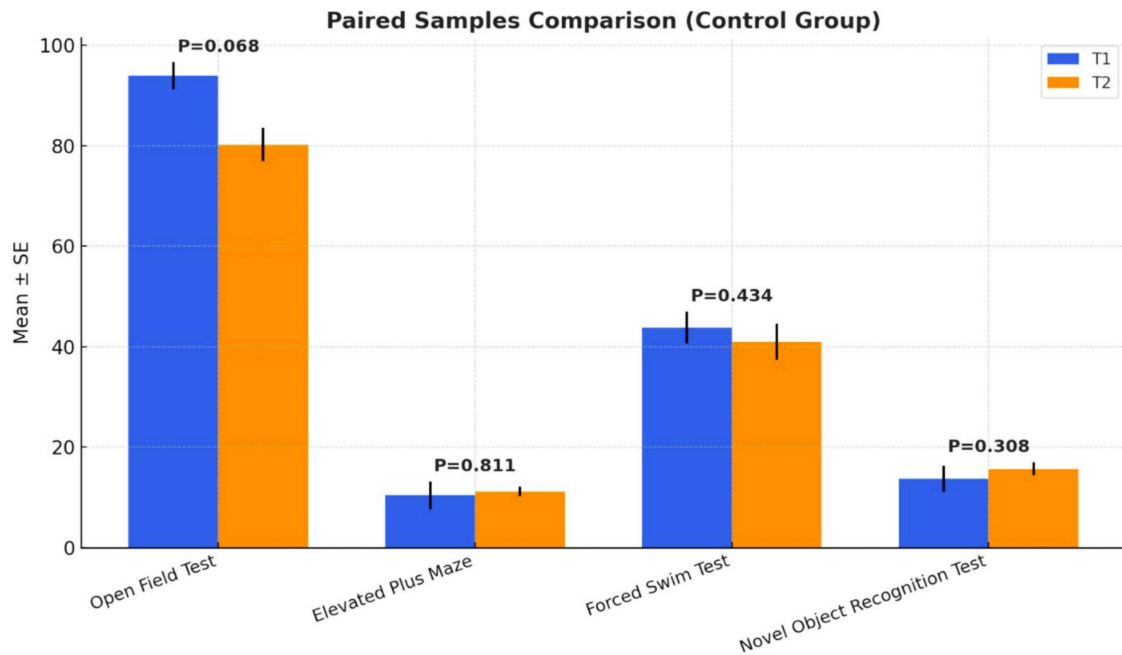


Figure 2. Behavioural test results for the control group (T1 = first testing session; T2 = second testing session). Data represent performance in the Elevated Plus Maze, Forced Swim Test, Open Field Test, and Novel Object Recognition task for the control group across the two testing sessions. Statistical significance was assessed using paired-samples *t*-tests.

Similarly, the Forced Swim Test (FST) revealed an increase in immobility time in conditioned rats (FST1 $M = 49.6$, $SD = 14.4$; FST2 $M = 51.14$, $SD = 8.80$), reflecting enhanced depressive-like behaviour ($p = 0.04$, 95% CI $[-22.87, -0.22]$). No significant changes were observed in the control group (FST1 $M = 43.8$, $SD = 7.05$; FST2 $M = 40.98$, $SD = 7.99$; $p = 0.43$, 95% CI $[-6.18, 11.82]$). These findings align with literature describing how chronic or unpredictable stressors can increase passive coping strategies and depressive-like states in rodents (Bienertova-Vasku et al., 2020; McEwen, 2007).

By contrast, no significant effects were found in the Open Field Test (OFT) or the Novel Object Recognition (NOR) task. This suggests that exploratory behaviour and cognitive performance remained stable following aversive stimulation. Our findings on this selective modulation of emotional—but not cognitive—domain are consistent with other studies showing that acute stress primarily alters emotional reactivity, while cognitive functions may remain intact or require prolonged exposure to stress to be affected (Sandi & Haller, 2015; Widman et al., 2019).

Based on these results, fear conditioning increased anxiety- and depression-like behaviours in rats without altering exploratory or cognitive performance (see Figures 1 and 2). Therefore, the study findings suggest that aversive learning primarily activates emotional defence systems while leaving higher-order cognitive processing intact.

3.2. Prefrontal EEG Activity During Aversive Stimulation and Defensive Freezing

In order to investigate the neural processes associated with the behavioural effects of fear conditioning, we analysed the prefrontal EEG activity across several conditions, with a focus on broadband oscillatory power (delta, theta, alpha, beta and gamma). Comparisons were made between unstimulated and post-stimulation periods, between the left and right prefrontal hemispheres, and between freezing and non-freezing behavioural states.

No significant differences were found in broadband power when comparing the unstimulated and post-stimulation conditions in the experimental group. *P*-values ranged from 0.16 to 0.35 across all frequency bands, showing that the aversive stimulus did not induce a detectable global change in prefrontal oscillatory activity (see Figure 3). This absence of widespread cortical change is

consistent with previous research indicating that acute fear conditioning may not consistently result in generalised EEG alterations, but rather in state-dependent or regionally specific modulation (Mochizuki et al., 2025).

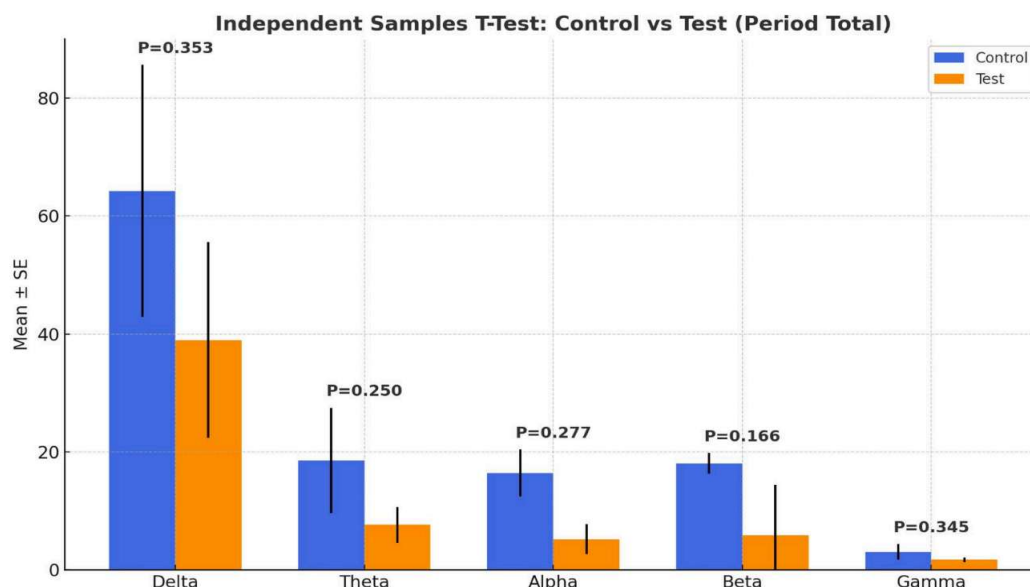


Figure 3. Prefrontal EEG broadband power (delta, theta, alpha, beta, gamma) before vs after aversive stimulation in the experimental group. Comparisons were made between the unstimulated and post-stimulation conditions; statistical differences were commuted with independent-samples t-tests.

Similarly, analysis of left versus right prefrontal hemispheres in the post-stimulation condition revealed no significant differences, with p-values ranging from 0.36 to 0.90 for delta, theta, alpha, beta, and gamma bands (Figure 4). This absence of lateralised activity suggests a bilateral engagement of the prefrontal cortex under aversive conditions, consistent with previous evidence indicating symmetrical prefrontal involvement in stress-related processing (Widman et al., 2019).

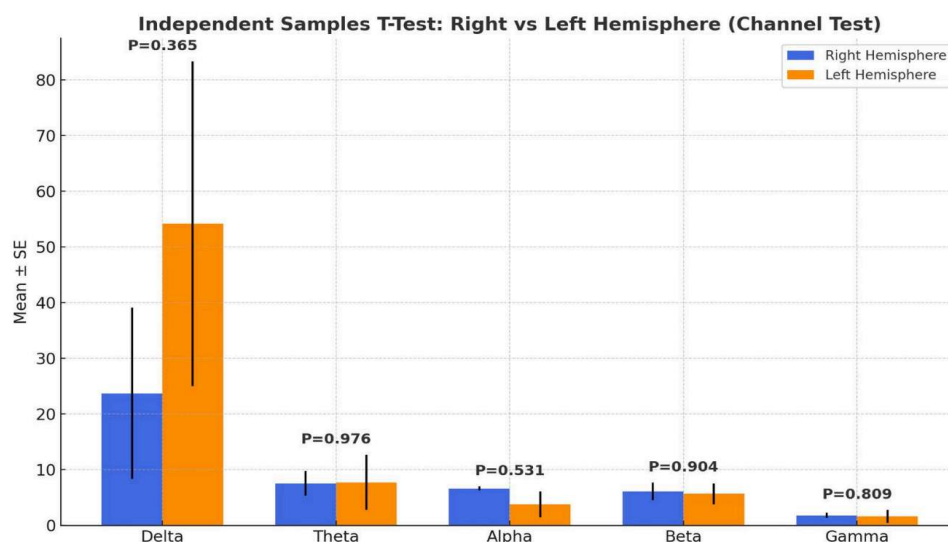


Figure 4. Prefrontal EEG broadband power (delta, theta, alpha, beta, gamma) in left vs right hemispheres after aversive stimulation. Comparisons were made between left PFC and right PFC within the post-stimulation condition; statistical differences were commuted with independent-samples t-tests.

In contrast, when EEG activity during freezing and non-freezing behavioural states was compared, significant differences emerged across all frequency bands. Broadband power increased during freezing episodes, with p-values and confidence intervals as follows: Delta ($p = 0.04$, 95% CI

$[-121.75, -2.20]$), theta ($p = 0.02$, 95% CI $[-25.19, -1.88]$), alpha ($p = 0.04$, 95% CI $[-18.02, -0.45]$), beta ($p = 0.03$, 95% CI $[-14.32, -0.69]$) and gamma ($p = 0.04$, 95% CI $[-2.98, -0.066]$) (see Figure 5). This pattern is consistent with earlier research suggesting that defensive behavioural states, like freezing, are linked to short-lived but significant increases in activity in the prefrontal cortex. These increases show how the brain's fear and threat-monitoring circuits are engaged (Borkar & Fadok, 2021; Mochizuki et al., 2025).

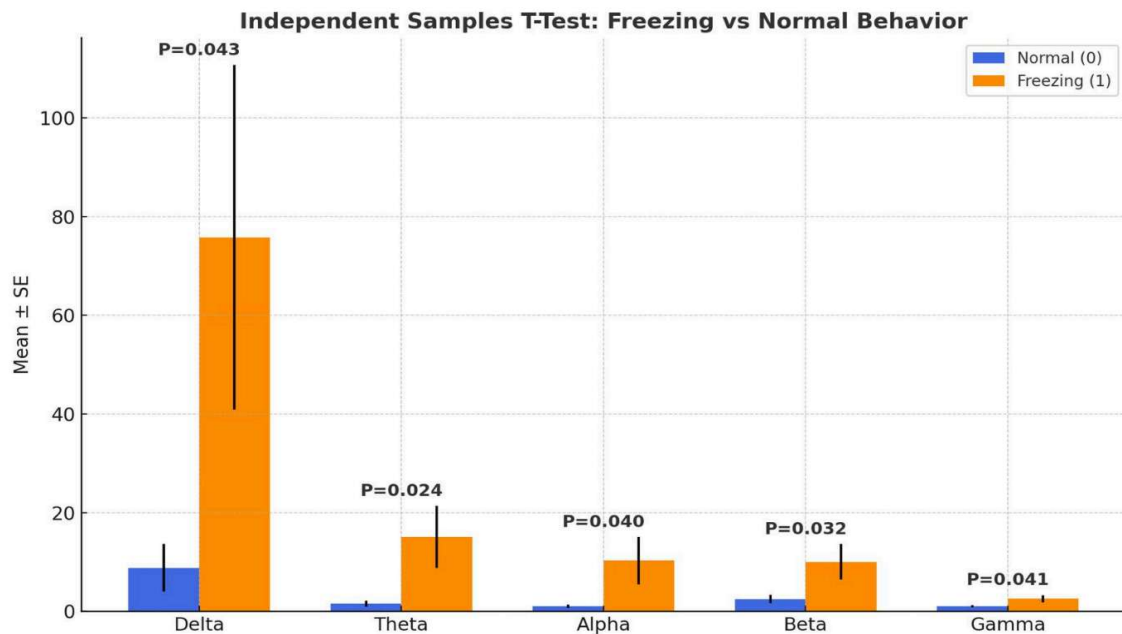


Figure 5. Prefrontal EEG broadband power (delta, theta, alpha, beta, gamma) during freezing vs normal behaviour. Comparisons are between freezing and non-freezing epochs; statistical differences were evaluated with independent-samples *t*-tests

Together, these results show a clear contrast between global neural measures and state-dependent modulation: while no differences were observed at the global or hemispheric level, freezing episodes were associated with significant broadband increases in prefrontal EEG power. This finding is consistent with literature highlighting the functional specificity of defensive behavioural states and their capacity to recruit emotional regulatory networks more strongly than baseline fear exposure alone (Sandi & Haller, 2015; Widman et al., 2019).

4. Discussions

The present findings show that fear conditioning induced robust emotional and defensive behavioural changes, without global or lateralised shifts in prefrontal EEG activity. Instead, increased prefrontal broadband power was observed selectively during freezing episodes, suggesting state-dependent modulation of cortical networks. This pattern is consistent with the view that defensive behaviours reflect transient engagement of prefrontal circuits, rather than sustained background activation.

Behaviourally, fear conditioning led to increased anxiety- and depression-like behaviours, as reflected in the Elevated Plus Maze and Forced Swim Test. These results are in line with previous evidence showing that aversive learning amplifies emotional reactivity while sparing exploratory and cognitive domains (Bienertova-Vasku et al., 2020; McEwen, 2007; Sandi & Haller, 2015). The absence of significant changes in Open Field and Novel Object Recognition performance supports the notion that acute fear predominantly targets emotional systems, leaving cognitive performance relatively preserved (Gray & McNaughton, 2000; Pessoa, 2017).

EEG analyses revealed no significant global differences between pre- and post-stimulation conditions, and no lateralised activity across hemispheres. These findings suggest that prefrontal engagement following fear conditioning is not expressed as a diffuse oscillatory shift. Instead, increased oscillatory power emerged specifically during freezing episodes, consistent across delta, theta, alpha, beta, and gamma bands. This observation aligns with previous studies reporting state-dependent prefrontal activation during defensive behaviours, reflecting the involvement of top-down regulatory networks in threat evaluation and action selection (Borkar & Fadok, 2021; Mochizuki et al., 2025; Tovote et al., 2015).

Within this framework, freezing can be viewed as a transient state in which the PFC is recruited to evaluate threat, gate amygdala-driven defensive output, and bias downstream motor or visceral responses. The broadband escalation we report across delta-gamma bands may, therefore, reflect the transient engagement of top-down evaluative processes and the increased coupling required for rapid PFC-amygdala communication during defensive states (LeDoux, 2000; Tovote et al., 2015; Sandi & Haller, 2015). Rather than indicating a global upregulation of cortical tone, these episodic prefrontal signatures are likely to index functional interactions that enable threat monitoring, decision-making about coping strategies, and preparation or inhibition of action (e.g., immobility) when escape is not advantageous.

Taken together, the current findings indicate that fear conditioning does not induce sustained alterations in prefrontal activity at rest, but selectively modulates neural activity during behavioural states associated with defensive strategies. This distinction underscores the importance of examining brain-behaviour coupling rather than relying solely on baseline measures. It also supports theoretical models in which the prefrontal cortex dynamically coordinates emotional and defensive responses in interaction with limbic circuits (LeDoux, 2000; Pessoa, 2017; Widman et al., 2019).

Several limitations should be acknowledged. First, the relatively small sample size may limit the statistical power to detect subtler effects. Second, the aversive conditioning protocol used visual-auditory pairing rather than electrical shocks, which may have elicited a milder stress response compared to classical paradigms. Third, EEG recordings were restricted to the prefrontal cortex; future studies should include additional regions, such as the amygdala and hippocampus, to better capture network-level dynamics. Finally, the study focused on acute effects; longitudinal designs could provide further insights into how repeated fear exposure impacts long-term plasticity and emotional regulation.

Our design prioritised within-subject sensitivity to detect changes induced by fear conditioning, while minimising the total number of animals. The experimental group size ($n = 15$) affords good power to detect large pre-post effects (e.g., Cohen's $d \approx 0.8$; power ≈ 0.82), but the small control group ($n = 5$) limits power to detect between-group differences unless effects are very large ($d > 1.5$). Accordingly, non-significant between-group comparisons should be interpreted cautiously. Future work could increase the control sample, use a crossover or repeated-measures design to improve power, or leverage historical control data to reduce the number of newly required animals.

5. Conclusions

This study demonstrates that fear conditioning in rats produces robust emotional and defensive behavioural changes, with state-dependent rather than global cortical effects. Freezing episodes were associated with increased broadband prefrontal oscillatory activity, highlighting the role of the prefrontal cortex in coordinating defensive strategies. These results emphasise the importance of integrating behavioural and neural measures to better understand the dynamics of fear responses. Future research should expand the spatial resolution of neural recordings and investigate the temporal evolution of these effects during prolonged exposure to aversive stimuli.

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