Coherence of Gamma-Band EEG Activity as a Basis for Associative Threat Learning

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Abstract

The ability to predict events from external or internal cues is vital for survival in dynamic environments. Seminal work by Miltner et al. (1999) suggested that coherence in the cortical gamma-band forms the basis for the integration of information between sensory cortices during associative learning. Grounded in Hebb's cell-assembly theory, these findings inspired extensive research on learning and memory, but results have been contradictory. Therefore, this registered report aims to replicate and extend the initial findings using a high-powered, multi-site study. Healthy young adults will be exposed to visual stimuli signaling the delivery or omission of an aversive electrotactile stimulus while electroencephalography is recorded. Successfully replicating the observed increase in gamma-band (37 – 43 Hz) power and coherence between sensors covering the occipital and primary/secondary somatosensory cortices for paired versus unpaired cues would provide robust support for the proposed role of gamma activity in associative learning. Extending these analyses, we anticipate that paired cues will elicit stronger visual alpha suppression (8 – 12 Hz), indicative of enhanced attentional engagement.

Keywords: associative learning, threat, gamma, coherence, replication

Introduction

Associative learning refers to the process of establishing connections between events that occur simultaneously or in close temporal proximity[1,2]. Since associative learning has been widely observed across species, it is considered a fundamental principle by which organisms encode and store information for the prediction about the future based on past experience[3], thereby securing adaptive behavior in changing environments. Neuroscientific and psychological research into associative learning contributed to the discovery of neural plasticity[4], which describes the brain's ability to flexibly reorganize its structure and function in response to experience. This discovery has been fundamental to understanding the neural processes underlying learning and memory. In 1999, Miltner and colleagues published an influential study that reported a potential neural correlate of associative learning processes in the human brain. Specifically, they recorded electroencephalogram (EEG) activity from 16 participants while exposing them to red and green room-illuminating lights. During the acquisition phase, one of the colors (conditioned stimulus; CS+) was paired with a painful electrical stimulus (unconditioned stimulus; US), while the other color (CS-) was never followed by the US. Examining the time-varying characteristics of neural oscillations, Miltner et al. (1999) identified heightened activity in the gamma-band (37 – 43 Hz) at occipital and parietal electrode sites during CS+ compared to CS- trials. Furthermore, the authors observed an increase in gamma-band coherence between visual and somatosensory areas for CS+ trials that were confined to the acquisition phase. This suggests that synchronicity in the gamma-band between specific cortical sites plays a crucial role in supporting inter-regional communication, which facilitates the formation of associative memories.

Following this seminal study, the association between gamma-band synchronization and neural plasticity has received additional support, particularly from animal research[5,6]: In general, the main function of gamma oscillations (~ 40 - 120 Hz) has been related to the transient formation of cell assemblies[7-9]. Accordingly, gamma oscillations have been observed in almost every brain region, including those relevant for associative learning processes, such as the neocortex, hippocampus, and amygdala[9-13], even though it is likely that different sub-bands of gamma activity vary in their origin and function[14,15]. Early theories proposed that cell assemblies selective for basic perceptual features are dynamically integrated through synchronization in the gamma-band in order to form representations of perceptual objects[16-21]. Likewise, the coherence of gamma-band activity has been suggested for playing a key role in facilitating communication both within and between cortical areas, fostering the emergence of various higher cognitive functions[22-24]. For example, Headley and Weinberger [6] demonstrated

that the strength of gamma oscillations (40 – 120 Hz) in the rodent auditory cortex not only predicted the acquisition of associative memory but also the magnitude of neural plasticity responsible for learning-induced changes in auditory receptive fields. Going beyond the scope of locally synchronized cell assemblies, it has been hypothesized that long-range interneurons are crucial for synchronizing gamma activity across different brain regions[7], possibly allowing an integration of sensory events. The basolateral and central nuclei of the amygdala have been proposed to be a neural hub that integrates sensory events occurring in close temporal proximity, as they receive sensory inputs from various brain regions e.g., sensory cortices and thalamus[27]. Thus, it is not surprising that gamma oscillations have been observed in the amygdaloid basolateral nucleus[10,13], where they might be involved in driving neural plasticity during learning. Indeed, learning-induced increases of synchronized gamma activity (35 – 45 Hz) have been found between the basolateral amygdala and rhinal cortex[28], as well as between striatal and basolateral amygdaloid regions[29]. Furthermore, it has been suggested that gamma oscillations in the basolateral amygdala might be involved in the long-term maintenance of conditioned fear responses, resulting from learning the association between a CS+ and an aversive US (i.e., fear conditioning). By examining local field potentials during a fear conditioning task in rodents, Courtin et al. demonstrated interindividual variations in basolateral gamma activity (30 – 80 Hz) during extinction training, which were found to be predictive of individual levels of post-extinction spontaneous fear recovery[30].

However, applying findings from animal studies to human research is not straightforward, as macroscopic measurements of neural activity using scalp EEG in humans are confined to cortical regions and less precise in identifying the exact sources of gamma oscillations compared to the intracranial recordings typically used in animal research. Additionally, differences in brain size and structure may influence the spatial spread and synchronization of gamma activity across cortical areas. Nevertheless, recent studies suggested that gamma activity is also associated with higher-order cognitive functions in humans. For example, heightened gamma activity and coherence in the human brain have been shown to predict performance across various types of memory tasks[25,26]. Focusing on the role of gamma oscillations during associative learning in humans, Keil et al. observed an early (60 – 90 ms) stimulus-locked increase in upper beta/lower gamma-band activity (18 – 35 Hz) in response to cues predicting an aversive picture (CS+) compared to cues predicting a neutral picture (CS-)[31]. During later time intervals (350 – 420 ms), there was an additional increase in induced gamma-band activity (55 – 80 Hz) that was, however, comparable between aversive and neutral conditioned stimuli. Further, a recent MEG study demonstrated an increase in

neuromagnetic gamma-band activity (peaking between 30 – 40 Hz) in response to a visual CS+ predicting an aversive noise compared to a visual CS- that was never paired with the noise during the acquisition phase[32]. Consistent with the notion of short-term plasticity changes during associative learning, this increase was most pronounced over visual cortices.

Despite the pioneering work by Miltner and colleagues[33], however, additional evidence supporting not only increased gamma activity, but also gamma-driven synchronicity between visual and somatosensory cortices during threat learning remains elusive. For instance, Pirazzini, et al. [34] performed a Granger Causality analysis on cortical source estimated EEG data obtained from a fear conditioning paradigm. The authors observed an increase in theta connectivity from the cingulate cortex to other cortical regions and a suppression of alpha activity in left posterior frontal and parietal cortices when presenting the threat-predicting stimulus. However, there were no statistically significant differences in gamma-band activity comparing threat vs. safety cues. The lack of successful replications of increased synchronicity between visual and somatosensory cortices during associative learning is also complicated by the fact that the original authors' were not able to reproduce their findings using other methods for estimating neural coherence, such as cross-spectral analysis based on Fourier transform or wavelet analysis[33].

Furthermore, neural activity in the visual cortices measured by scalp-EEG is dominated by alpha oscillations (~ 8 – 12 Hz) – a frequency band that has been linked to inhibitory control over neural populations[35,36], and, consequently, is suppressed during states of heightened perceptual processing indicating attentional or motivational engagement[37]. Indeed, previous studies have demonstrated functional coupling between the phase of alpha and the amplitude of gamma oscillations[38-40], suggesting an inverse relationship between alpha- and gamma-frequency activity. Consistent with findings of increased gamma activity during associative learning, a decrease of alpha power has been found for CS+ compared to CS-, indicating enhanced attentional engagement with the threat-predicting stimulus[41-44]. More recently, a similar decrease of alpha power (8 – 14 Hz) for threat-predicting compared to neutral stimuli has also been found over somato-motor cortices[45], suggesting that changes in alpha power may also be relevant for the integration of visual and somatosensory information during associative threat learning.

To elucidate the roles of gamma and alpha activity in associative threat learning, this Registered Report proposes a replication of the study conducted by Miltner et al. The specific choice to replicate this study is justified by its substantial impact on the field as evidenced by more than 1,000 citations since its publication (1,139 citations as of 21 February 2025).

Replicating this study is long overdue, as the original research was based on a relatively small sample of only 16 participants. Furthermore, the effect of increased gamma-synchronicity between visual and somatosensory cortices during threat learning did not reach statistical significance ($p = 0.06$) based on conventional thresholds and could only be demonstrated with very specific processing parameters and analysis algorithms, thus raising questions about the validity and generalizability of the findings in the original study. The authors observed enhanced activity in the gamma-band (37 – 43 Hz) over occipital and parietal electrode sites during CS+ compared to CS- trials. Furthermore, there was an increase in gamma-band coherence between visual and somatosensory areas for CS+ compared to CS- trials during the acquisition phase. Our present study aims to replicate these gamma-band effects with a statistical power to detect an effect size that is half of that reported in the original study. Consequently, we expect stronger visual gamma activity (H1) and increased gamma-band coherence (H2) during threat-related compared to safety cues. Expanding on the original findings, we also expect a stronger suppression of visual alpha activity (H3) for CS+ compared to CS- trials (see Table 1).

Table 1. Design table

Question	**Hypotheses**	**Sampling Plan (N, power analyses)**	**Analysis Plan**	**Interpretation given to different outcomes**
1. Does increased activity in the gamma-band relate to enhanced perceptual processing during associative learning?	H1: Larger gamma power-spectral densities (PSD) for CS+ compared to CS- trials during associative learning	N = 360, Cohens d = 0.263 Alpha = 1% Power = 99%	For all hypotheses we use an analytical procedure similar to the original study. In detail, we will rely on time-varying multivariate autoregression (AR) based on Kalman filtering to extract instantaneous coherence scores between sensors and power spectral densities for each frequency (Gamma: 37 – 43 Hz; Alpha: 8 – 12 Hz) and sample point. All measures will be submitted to paired-samples t-tests pooled across study sites.	Non-confirmatory findings would question the results of the original study. Given the substantially larger sample size and multi-site nature of the current study, such results would suggest that the original findings may have been influenced by the small sample size or specific methods (e.g., intracutaneous stimulation) used in the initial study. In addition, for all hypotheses, non-confirmatory outcomes could indicate that gamma coherence, gamma activity or alpha suppression are no reliable neural markers of associative learning, prompting a reevaluation of their roles in this process. Furthermore, if greater gamma power and stronger alpha suppression are observed for CS+ compared to CS− trials, but without a corresponding increase in gamma coherence between
2. Is increased coherence in the gamma-band a neural correlate of associative learning?	H2: Stronger gamma coherence between visual and somatosensory areas for CS+ compared to CS- trials	N = 360, Cohens d = 0.263 Alpha = 1% Power = 99%		
3. Can alpha suppression in posterior areas index attentional engagement with threat-	H3: Stronger alpha suppression for CS+ compared to CS- trials during associative learning	N = 360, Cohens d = 0.375 Alpha = 1% Power > 99%		

predicting stimuli?				visual and somatosensory areas, the results may be attributed to heightened perceptual processing of aversive stimuli rather than associative learning mechanisms.

Methods

The following publication represents a Stage 1 Registered Report. All study materials and preliminary code for data processing are available in a project repository hosted on the Open Science Framework (OSF; https://osf.io/km5jv/). This OSF repository serves as a central project hub, linking to anonymized raw EEG data which will be publicly accessible on OpenNeuro (https://openneuro.org/), and to data processing and analysis code which will be posted on GitHub (https://github.com/yannikstegmann/Miltner99_replication). This replication endeavor was inspired by the #EEGManyLabs project[46] and constitutes an international collaboration involving different labs across multiple independent and geographically diverse study sites across Italy, Hong Kong, Germany, and The Netherlands. At the time of the initial submission of this Stage 1 report, nine replicating labs are engaged, each utilizing different models of amplifiers and electrodes for EEG recording. The EEG systems employed include BioSemi (BioSemi, Amsterdam, Netherlands), Electrical Geodesics Inc. (Magstim EGI, Eugene, OR, USA), NeurOne Tesla (Bittium Biosignals Ltd., Finland), and actiCHamp/BrainAmp amplifier systems (Brain Products GmbH, Gilching, Germany). Comprehensive information regarding each site, including equipment and electrode specifications, site-specific protocols, and participant recruitment procedures, is presented in Table 2. Ethics approval was obtained from the Ethics Committee of the University of Würzburg (GZEK 2025-14). All other participating replicating lab will obtain approval from their respective local institutional review board/ethics committee to conduct the study and share anonymized raw data. It is expected that each replicating lab will collect EEG data from a minimum of 40 participants, resulting in a cumulative total of at least 360 participants across all participating labs before exclusions (see next section for further details on the power analysis and participant exclusion criteria).

Table 2. *Overview of EEG set-up and recording details at each replicating lab*

Participating University	**Amplifier System**	**Electrode/Cap Model, Number EEG**	**Sampling Rate**	**Reference**	**Stimulus Presentation, Language**	**Electrical stimulation delivery system**

University of Augsburg, Germany	Brain Products actiCHamp	32-channel, actiCap slim/snap	500 Hz	FCz	Neurobs Presentation, German	Digitimer DS5
University of Bologna, Italy	Brain Products BrainAmp DC	64-channel, Easycap Fast n Easy Electrodes	1000 Hz	FCz	Psychopy, Italian	Digitimer DS7A
University of Hamburg, Germany	Brain Products BrainAmp DC	61-channel, EasyCap Multitrodes	1000 Hz	between AF3 and Fz	Neurobs Presentation, German	Digitimer DS7A
University of Hildesheim, Germany	Brain Products BrainAmp DC	32-channel, actiCAP slim/snap	1000 Hz	FCz	Neurobs Presentation, German	Digitimer DS7A
University of Hong Kong, China	ANT Neuro eego mylab	64-channel, Waveguard net	500 Hz	Cz	Neurobs Presentation, English	BIOPAC STIMISOC (MP160 and STM100C)
University of Potsdam, Germany	EGI Net Amps 300	129-channel, HydroCel Geodesic Sensor Net	500 Hz	Cz	Neurobs Presentation, German	Digitimer DS7A
University of Regensburg, Germany	Bittium NeuroOne Tesla	32-channel, EasyCap Multitrodes	1000 Hz	FCz	Neurobs Presentation, German	Digitimer DS7A
University of Rotterdam, The Netherlands	Active Two Biosemi	64-channel, elastic cap including CMS and DRL	512 Hz		Neurobs Presentation, English	Digitimer DS7A
University of Würzburg, Germany	EGI Net Amps 300	129-channel, HydroCel Geodesic Sensor Net	500 Hz	Cz	Neurobs Presentation, German	Digitimer DS7A

Participants

In the original study, 16 student volunteers (9 female, between 22 and 36 years old) participated in a differential associative threat learning and extinction task[33]. In our replication study, participants will be recruited from undergraduate courses or the community and provide written informed consent prior to participation and to the posting of anonymized raw data. Participants must meet the following inclusion criteria: they must be over 18 years old and right-handed according to the Edinburgh Handedness Inventory. The exclusion criteria are the use of pain medication within 24 hours before the experiment, having any self-reported acute or chronic conditions, including pain-related, cardiovascular, neurological, or mental disorders, and being currently pregnant. Participants will be compensated for their time with course credits or financially according to local policies for the remuneration of participants. The present study aims at collecting data from at least $n = 320$ participants. This number is derived from a power analysis based on statistics from the original study.

The goal for our replications is to reach 99 % power with an alpha level of .01, calculated using an effect size as large as half of the effect size reported in the original study.

For the main finding of increased gamma coherence, Miltner et al. (1999) reported results, equaling an effect size of Cohen's $d = 0.525$. We assumed the original effect size to be twice as large as it might have been if the original study had been highly powered. This assumption is supported by work showing that the effect size in pre-registered studies is about half the size of that in studies without pre-registration[47], and by the results of large-scale replication efforts[48]. Therefore, to mitigate potential overestimation of the true effect size, we determined that the sample size should allow for detecting 50% of the original effect size with a power of 99% on an alpha level of .01. A power analysis for an effect size of Cohen's $d = 0.263$ in a repeated-measures *t*-test revealed a sample size of at least $n = 317$ participants, thus a total sample size of $n = 320$ complete datasets will be distributed across the participating replication sites. To ensure this, we aim for a cumulative sample size of $n = 360$ to account for an anticipated drop-out rate of approximately 10% (~36 participants) due to poor EEG signal quality (see exclusion criteria below). Crucially, this sample size also fulfills the criteria of 99% power on a 1% alpha level for detecting an effect size of $d = 0.375$, which equals half of the roughly estimated average effect size for alpha suppression observed in differential fear conditioning studies. All power analyses were conducted using R (version 4.2.2) with the *pwr* package (version 1.3-0).

In addition, as part of the #EEGManyLabs Resting State spin-off project[46], the labs will also collect resting state EEG data and personality measures (https://osf.io/sp3ck/). The analysis of both EEG and personality data is not within the scope of the current study, but the personality measures can be used in further exploratory analyses. To facilitate this, participating labs will gather 8 minutes of resting state EEG, and participants will complete questionnaires (translated into the local language where applicable), including the Karolinska Sleepiness Scale (KSS)[50], the Positive and Negative Affect Schedule (PANAS)[51], and the State Trait Anxiety Inventory Trait Version (STAI-T)[52]. Also, we will administer the Edinburgh Handedness Inventory (EHI)[53], the Behavioral Inhibition and Approach System Scales (BIS-BAS)[54], the Center for Epidemiologic Studies Depression Scale[55], the Intolerance of Uncertainty Scale[56], the Pain Sensitivity Questionnaire[57] and the Short Version of the Big Five Inventory[58] questionnaires to participants.

Experimental Paradigm

After resting state EEG recording and completion of self-report measures, participants will complete a differential associative learning and extinction task, that closely follows the protocol of the original study. The acquisition phase consists of 60 CS+ and 60 CS- trials (120

trials in total), which are shown as black filled symbols (diamond and square shapes) on a medium-grey background for a fixed duration of 3,000 ms. In contrast to the original study, we have chosen not to use red and green colors illuminating the laboratory as conditioned stimuli due to their inherent signaling properties[59]. The conditions are presented in random order using an intertrial-interval (ITI) of 3,000 – 4,000 ms, during which only a white fixation cross on a medium-grey background is presented. The allocation of the diamond and square shape to CS+ and CS- conditions, respectively, is counterbalanced between participants separately per site. Each CS+ is followed by the application of a US (10 ms electrical stimulation) at the offset of the visual stimulus presentation (reinforcement-rate = 100%). Also, in contrast to the original work that used electrical stimuli administered intracutaneously to the mid-volar tip of the third digit, we will deliver aversive stimulation via surface electrodes attached to the ventral side of the participant's lower arm in the current study. This adjustment ensures consistency with contemporary research standards in the fear conditioning literature [60,61] Prior to the experiment, the US intensity will be individually adjusted to a painful but tolerable sensation[62]. In detail, participants will receive two series of increasing and decreasing intensities until they reach a level of 6 on a scale from 0 (= no sensation) over 4 (= minimally painful) to 10 (= worst pain imaginable). The application of the US will occur on either the left or right lower arm, with the side being counterbalanced across participants.

The extinction phase is identical to the acquisition in terms of timing. However, each CS is only presented 40 times and no US is delivered. Before acquisition, as well as after the first half and at the end of each phase, participants will be asked to rate the CS+ and CS- regarding valence ("How do you rate this stimulus?") on a scale from -4 = "very unpleasant" to 4 = "very pleasant". Similarly, pain intensity und unpleasantness ratings of the US will be collected. The experimental paradigm will be presented using Presentation (Neurobehavioral Systems Inc., 2021), or PsychoPy[63]. Each study site will use a task adapted to the local language.

EEG Data Processing

Offline, EEG data will be imported into EEGLAB[64] and processed according to a pipeline similar to the original study. However, as many important details on the original pipeline could not be extracted from the manuscript or unequivocally clarified in discussion with the original authors, our pipeline follows the original study as closely as possible. Furthermore, we incorporated additional procedures for identifying and removing EEG artifacts, which were not addressed in the original study. See Table 3 for a summary of the

processing pipeline and the deviations from the original study. The final code necessary for data processing at each of the sites will be posted online at the submission of the Stage 2 report.

Continuous EEG will be filtered offline using a Hamming windowed FIR high-pass filter (1 Hz cutoff frequency, -6 dB). The EEG data will then be re-referenced to average reference, while retaining the original reference electrode. To remove line noise and identify and reject bad channels, the PREP pipeline[65] with default parameters will be applied to the data. The data will then be down-sampled to 200 Hz, before stimulus-locked epochs will be extracted using a time window from -250 ms to 3000 ms relative to stimulus onset. Unlike the original study, which used time windows from -250 ms to 4000 ms, we chose to omit the interval between 3000 ms and 4000 ms to eliminate artifacts in the EEG signal caused by the electrical stimulation. Notably, this last second was also not included in the original study's inferential statistical analyses. Independent component analysis (ICA) and the MARA algorithm[66,67] will be used to automatically identify and remove artifacts, such as eye-movements and muscle artifacts. Finally, rejected bad channels are subjected to a spherical spline interpolation. Trials with remaining artifacts will be identified and excluded if they deviate by more than 3.29 standard deviations from the trimmed normalized means in terms of joint probability, kurtosis, and frequency spectrum[68]. Current source densities (CSDs) will be calculated from the artifact-free trials.

The CSD transformed data will then be submitted to a time-varying multivariate autoregression (AR) based on Kalman filtering to extract instantaneous coherence scores between sensors for each frequency and sample point. As there is no working implementation of the original algorithm and it is described in insufficient detail, we use an algorithm described in Milde et al.[69] and implemented in the Dynet-Toolbox[70]. Similar to the original study, a model order of $p = 16$ and a default adaptation constant of $c = 0.03$ will be used. Time-varying instantaneous (undirected) coherence will then be calculated from the AR prediction error matrix[71] across trials for the entire time window in a frequency range between 1 and 45 Hz separated for each participant, condition, and pair of sensors of interest.

The gamma-band of interest is defined as 37 – 43 Hz over sensors covering occipital (Oz, O1, O2, Pz), somatosensory association (CPz, CP1, CP2, CP3, CP4), and primary somatosensory (Cz, C1, C2, C3, C4, FCz, FC1, FC2, FC3, FC4) areas. Similar to the original study, gamma coherence will be analyzed in a time-window between 2,750 – 3,000 ms after cue onset [33]. For somatosensory areas, analyses are restricted to sensors on the midline and contralateral to the stimulated side. Similarly, gamma and alpha abundance is calculated as

the parametric power-spectral-densities (PSDs) extracted from spectral representations of the AR weights[69]. Gamma abundance will be analyzed in a time-window between 2,000 – 3,000 ms post cue onset[33]. The alpha-band of interest is defined as 8 – 12 Hz over parieto-occipital areas (Oz, O1, O2, Pz) in a time-window between 500 – 1,000 ms after cue onset[43].

Table 3. Summary of the EEG processing pipelines of the original and the replication study.

Preprocessing Step	**Miltner et al. (1999)**	**Current Study**
Filter	Data were recorded with a 10 s time constant ~ .016 Hz high-pass filter	No online filters, 1 Hz offline high-pass filter
Re-referencing	Linked ears	Average reference
Segmentation	Stimulus-locked -250 to 4000 ms	Stimulus-locked -250 to 3000 ms
Line Noise Removal	NA	PREP-Pipeline
Bad Channel Identification	NA	
Ocular Artifact Correction	Linear regression	ICA and MARA
Artifact Rejection	NA	
Bad Channel Interpolation	NA	Spherical spline interpolation
Bad Trial Identification and Exclusion	NA	Outlier criterion of $z > 3.29$ regarding joint probability, kurtosis, and spectrum
CSD transformation	Spline flexibility: NA Head radius: NA Smoothing constant: NA	Spline flexibility: $m = 4$ Head radius: $r = 10$ Smoothing constant: $\lambda = 10^{-5}$
Coherence analysis using a time-varying autoregressive (AR) modelling of multivariate time series based on Kalman filtering		
Model order	$p = 16$	$p = 16$
Adaption constant	NA	$c = 0.03$
Trial handling	Analysis on single trial level and then averaged across trials	Trials as an additional multivariate dimension
Calculation of coherence	Based on the AR prediction error matrix	Based on the AR prediction error matrix
Frequency of interest	Gamma: 37 – 43 Hz	Gamma: 37 – 43 Hz

Transformation	Fisher-Z transform	Fisher-Z transform
Power-spectral-densities (PSD) scoring	NA	Based on AR weights
PSD baseline-correction	NA	%-change relative to baseline

Note: NA = no information from the original authors available; PREP = standardized preprocessing pipeline for large-scale EEG analysis[65]; MARA = Multiple Artifact Rejection Algorithm[66,67].

Statistical analysis

Data quality and participant exclusion criteria.

In the original study, no participants have been excluded. In the current study, participants will be excluded from the analysis, if fewer than 66 % of trials remain after artifact rejection. Note, that excluded participants do not count towards the sample size and data collection will be continued until at least 320 complete datasets have been recorded.

Inferential statistics.

The primary effect of interest in the original study was the observed increase in gamma-band coherence between visual and somatosensory areas during presentations of the threat-signaling stimulus (CS+) compared to the safety stimulus (CS-) in the acquisition phase. To analyze this, we will aggregate the samples over each study site and employ paired-samples t-tests to examine the Fisher-Z-standardized coherence measures derived from autoregressive modeling between CS+ and CS-. An α level of .01 will be used as the threshold for statistical significance. Furthermore, we will utilize paired-samples t-tests to assess differences in gamma and alpha power spectral densities (PSDs) between CS+ and CS- during both the acquisition and extinction phases separately. We expect to observe significantly (H1) larger gamma PSD (H2) larger gamma coherence scores, and (H3) stronger alpha suppression for CS+ compared to CS- trials during the acquisition phase across replicating labs. Each hypothesis will be considered confirmed if a statistically significant estimate ($p < .01$) across replicating labs is observed and if the effect is in the expected direction. To quantify differences between study sites, we will compute effect sizes (Cohen's *dz*) for each individual lab and hypothesis separately and then combine all datasets in a random-effects meta-analysis (with labs as a random effect) using the REML estimator for random-effects variance. Employing a random-effects meta-analysis will address and help in estimating the effect of heterogeneity in hardware and samples between labs. We will report distribution of the

weighted effect sizes, their 95% confidence intervals, and their heterogeneity (τ^2). The metafor package[72] for R will be used for the meta-analyses.

Secondary Analysis

Controlling for spurious synchronicity due to volume conductance.

Volume conductance (VC) refers to the passive spread of electrical activity through brain tissue and other matter in the head[73], leading to signal mixing across electrode sites. As a result, coherence measures between two sensors may be influenced by shared volume-conducted activity rather than neural interactions between sites[74]. This issue is particularly relevant for EEG-based connectivity analyses, where spatially close electrodes may show artificially high coherence due to VC rather than true neural synchronicity. The impact of VC was not explicitly addressed in the original study. While the authors mitigated this issue by calculating current source densities, and thus reducing the impact of VC[73], it cannot be eliminated entirely[75]. As a result, the findings of the original study may still be partially influenced by spurious synchrony. To assess whether the observed coherence in the current study reflects true functional connectivity rather than volume conduction, we will compare coherence measures between ipsilateral and contralateral connections of somatosensory and visual regions relative to the side of electrical stimulation (left vs. right arm). The rationale is that VC should equally affect sensors at both ipsilateral and contralateral sites due to their comparable distances from the source. However, if increased coherence is driven by associative learning rather than VC, we would expect this effect to be more pronounced at sites contralateral to the electrical stimulation. To test this, we will conduct an additional analysis of coherence scores using a 2 (cue: CS+ vs. CS−) × 2 (site: ipsilateral vs. contralateral) ANOVA. We predict significantly greater coherence at contralateral compared to ipsilateral sites during CS+ presentations, providing evidence that the observed synchrony reflects functionally relevant neural interactions rather than volume conduction artifacts.

Alternative time-frequency (wavelet) analysis.

To assess the robustness of our findings, EEG data will undergo time-frequency analysis using Morlet wavelets in addition to the time-varying multivariate AR approach described above. Accordingly, the preprocessing pipeline will be optimized for wavelet analysis. EEG data will be down-sampled to 500 Hz, and stimulus-locked epochs will be extracted from −1000 ms to 3000 ms relative to stimulus onset. The extended baseline window and higher sampling rate will enhance frequency resolution and minimize edge artifacts. The remainder of the preprocessing pipeline will remain consistent with the primary analysis. Wavelet

transformation will be performed using Morlet wavelets with a coefficient of m = 10, spanning a frequency range of 2–45 Hz. The resulting time-frequency representations will be averaged by condition before applying baseline normalization using a −800 to −200 ms window relative to cue onset. Power spectral densities (PSDs) will be extracted within the same frequency bands and regions of interest as defined in the primary analysis. However, the last 50 ms will be discarded to mitigate edge artifacts at the end of each trial. Additionally, the wavelet analysis results will be used to compute two alternative measures of synchrony: 1) Similar to the primary analysis, we will calculate coherence coefficients to assess phase synchrony between each sensor pair of interest. 2) We will compute imaginary coherence by extracting the imaginary part of the complex coherency coefficients and averaging them across trials. Imaginary coherence has gained interest in recent years as it discards interactions occurring at a phase difference of 0° or 180°, making it less susceptible to volume conduction effects [76,77]. However, this advantage comes at the cost of excluding true interactions that occur at near-zero phase differences, which may be relevant to the current study.

Heart rate analysis.

To gather supplementary evidence of associative learning on the autonomic response level, we will exploratively analyze changes in heart rate in response to the cue onsets[78]. The electrocardiogram will be recorded with a sampling rate of 1000 Hz from three adhesive Ag/AgCl electrodes, placed underneath the right clavicle, as well as the left and right costal arch. Using R (version 4.2.2), a 2 Hz high-pass filter will be applied to the ECG data to remove slow signal drifts. R-waves will be detected using a semi-automatic method and then converted into heart rate (in beats per minute, bpm). Changes in heart rate will then be separately averaged for each condition into 500 ms bins for the time window from 0 ms to 3000 ms relative to stimulus onset. The last 500 ms prior to cue onset will serve as baseline. Heart rate changes will be analyzed using a 2 (cue: CS+ vs. CS−) × 6 (bin) ANOVA. It is important to note, however, that the experiment was specifically designed for EEG recordings and that the stimulus presentation of 3 s and ITIs of 3–4 s may only capture the first acceleration phase of the heart rate response.

Temporal dynamics of learning.

To track changes in associative learning indices over time, we will perform an extended analysis by including trial-bin as an additional within-subjects factor across all measured variables. Trials will be grouped into bins of 10 per condition, resulting in six blocks for the acquisition phase and four for the extinction phase.

Timeline and Reproducibility

We expect to submit the Stage 2 report with accompanying open data and materials within 2.5 years of the Stage 1 manuscript receiving an in-principle acceptance. We will ensure reproducibility of findings by sharing anonymized data (including raw EEG data), data processing code, code for statistical analysis, and all study materials (e.g., study protocol, experimental paradigm). If there are deviations from the Stage 1 report, they will be documented in the Stage 2 report.

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