

Trait anxiety is associated with reduced coupling between physiological dynamics and adaptive learning

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Article

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Abstract

Despite the societal costs of anxiety disorders, their underlying mechanisms remain debated. Traditional accounts suggest that anxiety is characterized by exaggerated responses to safety signals, whereas newer theories propose that it reflects deficits in uncertainty reduction. Whether these deficits are also evident at the psychophysiological level remains unexplored. Using data from 12 Pavlovian fear-conditioning studies ($N = 1,101$), we examined the relationship between skin conductance response (SCR) patterns and both threat-learning and uncertainty-reduction strategies and whether anxiety-related traits influenced this coupling. SCR patterns corresponded more closely with uncertainty-reduction than threat-learning strategies, supporting the view that SCRs index decreases in uncertainty. Importantly, trait anxiety was associated with weaker coupling between SCRs and both strategies. Further analyses revealed an asymmetric pattern: while individuals low in anxiety demonstrated greater coupling flexibility, individuals high in anxiety showed consistently reduced correspondence, suggesting that anxiety may involve constrained coordination between bodily dynamics and adaptive learning to changing environments.

Introduction

Anxiety disorders represent one of the most pressing public health challenges of our time, imposing substantial personal and societal burdens worldwide^{1,2}. Over recent decades, psychophysiological and neuroscientific research has made major efforts to elucidate the mechanisms underlying the development and maintenance of these disorders, which are characterized by excessive fearful and avoidant responses^{3,4}. Collectively, these advances have positioned Pavlovian fear conditioning as one of the most established and influential paradigms for investigating the neurobiological underpinnings of fear- and anxiety-related disorders^{3,5,6}.

In this paradigm, two initially neutral cues are differentially paired with an aversive unconditioned stimulus (US, e.g., an electric shock). One cue is consistently paired with the US (conditioned stimulus, CS+), whereas the other remains unpaired (CS-). It is suggested that, after a few learning trials, associative learning processes enable the CS + to elicit conditioned fear responses, reflected in elevated reports of fear and a characteristic physiological pattern across participants, such as enhanced skin conductance responses (SCRs), startle blink response, or heart rate deceleration^{3,6,7}. The same associative mechanisms that enable the acquisition of conditioned fear are thought to contribute to the formation of maladaptive fears that characterize anxiety symptomatology, thereby supporting the use of behavioral, psychophysiological and neuroimaging correlates of Pavlovian fear conditioning to investigate mechanisms underlying anxiety^{3,6,8}.

However, efforts to identify a psychophysiological *fear-response profile* that may characterize anxious individuals and thus inform about the underlying neurobiological underpinnings of anxiety, have yielded inconsistent, and at times contradictory, results^{9–13}. Whereas some studies observed a positive association between anxiety-related traits and differential psychophysiological responding (i.e., greater fear discrimination, indexed by stronger reactivity towards the CS+ compared to the CS-^{14,15}), others have reported the opposite pattern^{9,12} or no association^{13,16}. Converging meta-analytic evidence further indicates small, if any, differences in fear discrimination between anxiety patients and healthy individuals across psychophysiological indices of Pavlovian fear learning^{17–19} but a consistent heightened psychophysiological reactivity to both CSs in patients with anxiety disorders²⁰. Collectively, these findings provide only limited support for an association between anxiety and disrupted psychophysiological fear-learning responses, thereby, highlighting the need to consider alternative hypotheses regarding the etiology of anxiety^{21–24}.

Emerging accounts propose that a core feature of anxiety may lie in an impaired ability to adapt to dynamically changing environments^{24–26}. In such environments, some aspects are predictable, whereas others remain uncertain and subject to change. Adaptive learning allows individuals to reduce this uncertainty by constructing and updating internal beliefs about environmental contingencies based on incoming evidence^{27,28}. Heightened anxiety may be associated with impairments in these belief-updating processes^{21,29}. From this perspective, anxiety may not primarily reflect deficits in fear learning, but rather inefficient updating of beliefs about changing environmental contingencies²⁶. As a result, anxious individuals may persist in outdated or inaccurate beliefs, leading to suboptimal learning. Consistent with this account, recent studies have demonstrated that elevated trait anxiety is associated with deficits in reducing uncertainty in operant learning tasks^{21,25,26,29–31}.

It remains largely unexplored, however, whether similar mechanisms are reflected in psychophysiological responses²⁵. This is particularly relevant given contemporary models of psychopathology underscoring the importance of dynamic physiological reactions for understanding the development and maintenance of psychiatric conditions^{4,24,32,33}. The psychological symptomatology characteristic of psychiatric disorders is, almost ineludibly, accompanied by physiological unbalances. These models emphasize the need to efficiently coordinate physiological resources in anticipation of environmental demands (i.e., allostasis) as a regulating strategy for adaptation, positioning physiological dysregulation at the core of psychological disorders^{24,34}. In continuously changing environments, optimal adaptation should result in a close coupling between changes in environmental contingency and physiological responses, ensuring a balanced homeostatic state. However, when a mismatch between physiological reactions and environmental demands occurs, the organism may enter a dysregulated state. Over time, such uncoupling may contribute to persistent physiological alterations fostering the onset or maintenance of psychiatric symptoms, including anxious tendencies^{24,35}.

Investigating psychophysiological dynamics in changing environments may therefore provide critical insights into how the synchronization between bodily signals and changes in environmental contingencies is altered in anxiety. Furthermore, it may help clarify how disrupted predictive regulation of bodily states contribute to the emergence or maintenance of anxiety symptomatology.

The present study therefore investigated the association between anxiety-related traits and the coupling of psychophysiological dynamics, as measured by SCRs, and belief updating (i.e., operationalized using Bayesian probabilities) in Pavlovian fear-conditioning paradigms, aggregating data from 12 studies (10 samples; $N = 1,101$). Pavlovian paradigms provide a well-controlled framework for examining environmental contingency updating, as participants must iteratively update their predictions about the CSs in light of incoming evidence. Critically, these paradigms allow for the emergence of different learning strategies that align with etiological accounts of anxiety outlined above³⁶. One strategy emphasizes threat learning, whereby initially ambiguous CSs progressively acquire predictive value as signaling either threat (CS+) or safety (CS-). An alternative strategy centers on uncertainty reduction, in which CSs are initially maximally uncertain with respect to the unconditioned stimulus (US), and learning primarily reflects the gradual resolution of this uncertainty across trials. Physiologically, threat learning strategies predict that at the beginning of the acquisition phase, SCR to CS + and CS- would be similar due to unclear contingencies and would progressively differ as gathered evidence enables the distinction between the threat and safety cue. On the other hand, uncertainty learning strategies predict that in the initial stages of the acquisition phase, both CSs would evoke similar SCR responses given their maximal uncertainty with respect to the US. As evidence accumulates across trials, uncertainty should progressively decrease for both CSs, resulting in a gradual attenuation of SCR while maintaining similar responses to CS + and CS- throughout the acquisition phase.

In this study, we first quantified the trial-by-trial coupling between SCR dynamics and model-derived indices of threat learning and uncertainty reduction to characterize the predominant learning strategy expressed across studies and individuals³⁶. We then examined whether individual differences in trait anxiety, as measured with the trait scale of the State-Trait Anxiety Inventory³⁷, were associated with systematic alterations in the coupling between psychophysiological responses and environmental dynamics within the aggregated sample.

Results

Threat–safety discrimination across samples.

To show that datasets reproduced the canonical pattern observed in Pavlovian fear-conditioning paradigms (i.e., enhanced autonomic responding to CS+ cues relative to CS- cues) we first fitted linear mixed-effects models within each study.

Across studies, we observed robust evidence for differential conditioning effects. In 9 of the 12 studies (75%), Bayesian hypothesis testing provided evidence for the main effect of *CS type*, indicating larger SCRs to CS+ than to CS- (Fig. 1A; Suppl. Section S1). In addition, 9 of the studies (75%) showed decisive evidence for a main effect of *Trial as a continuous variable*, reflecting a general attenuation of SCR amplitude over the course of acquisition, with lower responses in later compared to earlier trials (Fig. 1B; Suppl. Section S1).

By contrast, evidence for a *CS type* × *Trial* interaction was limited. Only two studies (Studies 5 and 9; 17%) demonstrated support for an interaction effect, whereas the remaining 10 studies yielded anecdotal-to-moderate evidence favoring the absence of interaction effects (Fig. 1C; Table S1). Together, these findings indicate reliable differential conditioning and habituation effects across studies—replicating hallmark findings—but provide limited support for systematic differences in the temporal dynamic of responses to the CS + and CS-.

Decisive evidence for a coupling between SCR and uncertainty reduction but not threat learning strategies.

To address which learning strategy (i.e., threat learning or uncertainty reduction) was preferentially reflected in SCR dynamics, we quantified the correspondence between trial-by-trial SCR patterns and model-derived learning trajectories using representational similarity analysis (RSA). Unlike univariate methods, RSA is a well-suited approach to bring more insights into the representational structure of high-order dynamic processes and enables the assessment of their correspondence with representations of behavioral, autonomic, and brain-related responses^{38–41}.

For each participant, we constructed representational similarity matrices (RSMs) capturing trial-by-trial similarities in SCR and averaged these matrices across participants for each study (Suppl. Section S2). We then generated model-based RSMs reflecting predictions from threat learning and uncertainty reduction strategies (Suppl. Section S3-S7).

The threat learning model encoded changes in CS–US contingencies. It predicted that early in acquisition, responses to CS + and CS- would be similar due to ambiguous contingencies but would progressively diverge as evidence accumulated, with contingency strength increasing for CS + and decreasing for CS-. Trial-wise updates were computed using both forward ($P_{US|CS}$) and backward ($P_{CS|US}$) Bayesian probabilities (Suppl. Section S3). The forward model asks the question *given this CS, how likely is a threat to occur?* This model captures anticipatory predictions about the upcoming threat. In contrast, the backward model is conceptualized as *given the gathered*

evidence about a threat occurring (or not), what does this tell me about the CS? This model captures how participants update their beliefs about CS–threat relationships based on previous encounters. For instance, in a given CS+ trial, beliefs about the CS+/US relationship will be based on the accumulated evidence from preceding trials (both CS + and CS-). At the end of the current CS+ trial (after the US has been presented, or not), the new evidence will be used to update the $P_{CS|US}$. This update will be then considered in the upcoming trial. Estimating both models allowed us to test whether physiological responses primarily reflected the prediction of future threat (forward model) or learning about threat contingencies from past outcomes (backward model).

By contrast, the uncertainty reduction model captured the gradual decrease of uncertainty across trials. It predicted that both CS types would initially be maximally uncertain with respect to the US, with certainty increasing as evidence accumulated, irrespective of whether the outcome signaled threat or safety. Operationally, uncertainty reduction was quantified as the absolute deviation from maximal uncertainty ($P = 0.5$) in the conditional probabilities $P_{US|CS}$ and $P_{CS|US}$ on each trial (Suppl. Section S3). As with the threat model, we distinguished between forward ($UncP_{US|CS}$) and backward ($UncP_{CS|US}$) perspectives. The forward model represents the question *given this CS, how certain am I about what will happen next?* capturing anticipatory certainty about upcoming outcomes. In contrast, the backward model asks, *given what previously happened, how certain am I about what this CS means?* – this model reflects how certainty about CS–US relationships is updated after observing the outcome.

Across studies, only three datasets (25%) showed evidence for an association between SCR similarity structure and the backward threat learning model (Fig. 2A; Suppl. Sections S2, S4), while five studies (42%) showed evidence in favor of the forward threat learning model (Fig. 2B; Suppl. Section, S2, S5). In contrast, both backward and forward uncertainty reduction models showed evidence of correspondence with SCR dynamics in most of the studies (10 [83%] and 9 [75%] studies, respectively; Fig. 2C; Suppl. Section S2, S6, S7). Notably, the association with the backward uncertainty reduction model was particularly robust. Comparing the evidence for a correspondence between SCR and each model across studies (as indexed by the $\log[BF_{10}]$) further corroborated the differential effects between threat learning and uncertainty reduction strategies ($F_{1,83,20,18}=10.95$, $p = .001$, $\eta^2=0.49$, $BF_{10}>100$). Follow-up planned comparisons revealed that this effect was primarily driven by stronger evidence in favor of the backward uncertainty reduction model relative to the other three models (Fig. 2E; Suppl. Section S2). The same pattern of results was observed when considering each participant's similarity indices between SCR and the four models ($F_{1,47,1372.49}=117.95$, $p < .001$, $\eta^2=0.11$, $BF_{10}>100$; Fig. 2F; Suppl. Section S2)

Taken together, these findings indicate that trial-by-trial fluctuations in SCRs during Pavlovian conditioning are more strongly coupled with uncertainty reduction than with threat learning strategies. Importantly, the strongest association emerged for the backward uncertainty reduction model, suggesting that autonomic responses are particularly linked to how participants update their beliefs after an outcome—specifically, learning what caused the US or its absence. Physiological dynamics therefore appear to reflect learning from outcomes to refine cue–outcome relationships, and thus reduce uncertainty, rather than anticipatory estimates of threat or certainty. This interpretation aligns with prior work indicating that SCRs are particularly sensitive to learning-related changes in uncertainty, rather than to threat expectancy alone^{31,39,42,43}.

Reduced coupling between SCR dynamics and both threat-learning and uncertainty-reduction strategies is associated with higher trait anxiety.

We next examined whether the degree to which physiological dynamics aligned with model-derived strategies was modulated by individual differences in anxiety traits. To this end, we performed an unsupervised cluster analysis

using as input features each participant's similarity indices between SCR patterns and the overall best-fitting model (the backward uncertainty reduction model) as well as its complementary model, the backward threat learning model.

The analysis identified three distinct clusters of participants (Fig. 3A; Suppl. Section S8). Cluster 1 ($N = 321$; 34%) comprised individuals whose SCR dynamics showed strong coupling with the uncertainty reduction model (Fig. 4A), alongside weak coupling with the threat learning model. Cluster 2 ($N = 486$; 52%) included participants showing weak coupling with both models (Fig. 3A, 4B). Finally, Cluster 3 ($N = 125$; 13%) consisted of individuals whose SCR dynamics showed stronger correspondence with the threat learning model but weak correspondence with the uncertainty reduction model (Fig. 3A, 4C).

Comparisons across clusters revealed systematic differences in trait anxiety. Participants in Clusters 1 ($M = 36.82$) and 3 ($M = 35.93$) exhibited comparable levels of trait anxiety ($t_{209.82} = 0.97$, $p = 0.32$, $d = 0.07$, $BF_{10} = 0.19$). However, both groups reported lower anxiety than participants in Cluster 2 ($M = 38.4$; Cluster 1 vs. Cluster 2: $t_{552.36} = -3.05$, $p = .006$, $d = 0.22$, $BF_{10} = 8.04$; Cluster 2 vs. Cluster 3: $t_{182.28} = 3.39$, $p < 0.001$, $d = 0.34$, $BF_{10} = 18.81$; Fig. 3b). These results suggest that individuals whose physiological responses were more weakly coupled with both learning models were associated with elevated trait anxiety scores. These results were substantially consistent when evaluated using a leave-one-out cross-validation approach (see Suppl. Section S10). This pattern was further supported by analyses of categorical anxiety levels (see Suppl. Section S9).

Although uncertainty reduction emerged as the predominant strategy reflected in SCR dynamics at the group level, participants showed substantial variability in the SCR-learning strategy correspondence. Importantly, these differences were meaningfully related to anxiety tendencies: individuals whose physiological responses were more strongly aligned with either model were associated with lower trait anxiety.

These findings may help reconcile discrepancies with previous work. Prior studies of physiological responding in Pavlovian conditioning have typically relied on aggregated SCR measures, often computed as mean responses across CS+ and CS- trials. Such approaches may obscure trial-by-trial variability that contains critical information about underlying learning dynamics. In contrast, the present approach leverages inter-trial variability in physiological signals to derive richer response patterns.

To directly test whether dynamic response patterns provide greater sensitivity to individual differences than conventional average-based indices, we repeated the unsupervised clustering analysis using the common discrimination index (defined as CS+ minus CS- responses over the whole acquisition phase) as the input variable. This analysis again yielded three clusters (Cluster 1 = 204, Cluster 2 = 323, Cluster 3 = 436) that clearly distinguished participants with high, medium, and low discrimination (Fig. 3C). However, in contrast to the model-based clustering, these groups did not significantly differ in anxiety tendencies ($ps > 0.12$, $BF_{10} < 0.98$; Fig. 3D).

Together, these results indicate that trial-by-trial physiological response patterns and their correspondence with learning strategies provide more informative markers of anxiety-related variability than traditional average-based measures of conditioned responding, pinpointing the importance of considering response patterns instead of averaging scores to identify individual differences in how physiological representations adapt to dynamic environments^{38,44}.

Anxiety-related differences in SCR-model coupling follow an asymmetric pattern.

In a subsequent step, we used inter-subject RSA (is-RSA) to further examine the association between trait anxiety and the SCR-learning model coupling. This approach allowed us to determine whether anxiety-related differences in SCR-model correspondence followed a linear (symmetric) pattern across the trait anxiety dimension, or whether they were asymmetric—that is, whether the structure of this relationship differed across anxiety levels.

To this end, we constructed two is-RSMs capturing pairwise similarities between participants in the degree of correspondence between SCR patterns and the backward threat learning (Fig. 4D, E; upper part of the diagonal) and uncertainty reduction models (Fig. 4F, G; upper part of the diagonal). We then generated two additional is-RSMs based on participants' similarities in anxiety scores, reflecting two competing hypotheses about how anxiety might structure physiological-learning model coupling.

The first model assumed a symmetric relationship across the anxiety dimension, predicting that participants with similar anxiety scores would show similar levels of SCR-learning model correspondence (Fig. 4D, F; lower part of the diagonal). The second model captured an asymmetric relationship, predicting that participants with low anxiety would show greater heterogeneity in SCR-learning model coupling, whereas individuals with higher anxiety would become increasingly similar to one another (Fig. 4E, G; lower part of the diagonal).

We then compared anxiety-based is-RSMs with is-RSMs describing SCR-learning model correspondence. The symmetric anxiety model showed no positive association with any learning model ($\rho_{\text{threat}} = -0.03$, $\log(\text{BF}_{10}) < -100$; $\rho_{\text{uncertainty}} = -0.02$, $\log(\text{BF}_{10}) < -100$), providing strong evidence against a linear relationship across the anxiety continuum. In contrast, the asymmetric anxiety model showed positive associations with SCR-model correspondence for both the threat learning ($\rho_{\text{threat}} = 0.063$, $\log(\text{BF}_{10}) = 7.43$) and the uncertainty reduction model ($\rho_{\text{uncertainty}} = 0.057$, $\log(\text{BF}_{10}) = 20.38$). This indicates that among individuals with higher trait anxiety, the relationship between SCR dynamics and learning strategies was more consistent across participants (Fig. 4H).

Taken together, these results suggest that the association between trait anxiety and psychophysiological learning dynamics is not uniform across the anxiety spectrum. Rather, it follows an asymmetric pattern: individuals with lower anxiety exhibit more heterogeneous patterns of SCR-learning model correspondence, whereas individuals with higher anxiety display more homogeneous physiological-learning model correspondence. This pattern suggests that physiological responses among highly anxious individuals tend to converge toward a more consistent mode of uncoupling with environmental contingencies.

Discussion

The present study investigated how skin conductance response (SCR) dynamics during Pavlovian fear conditioning relate to learning strategies and trait anxiety. By aggregating data across 12 studies (10 samples; $N = 1,101$) and by combining trial-by-trial physiological representations with Bayesian estimates of threat learning and uncertainty reduction, we identified three main findings. First, trial-by-trial fluctuations in SCR were more strongly coupled with models of uncertainty reduction than with models of threat learning. Second, individuals who exhibited weaker coupling between physiological dynamics and both learning strategies also reported higher trait anxiety scores. Third, this relationship followed an asymmetric pattern across the anxiety dimension, such that individuals with higher anxiety showed more homogeneous patterns of uncoupling between psychophysiological patterns and learning models, whereas individuals with lower anxiety exhibited greater physiological flexibility. Together, these findings suggest that physiological responses in Pavlovian fear learning paradigms are best understood in relation

to uncertainty-related belief updating processes, and that uncoupling between physiological responses and environmental dynamics may represent an important feature of anxiety.

The current study leveraged the advantages of structurally representing trial-by-trial physiological dynamics to characterize the learning mechanisms underlying bodily reactivity^{38–40}. We showed that the structural representation of SCR responses corresponded more closely to models of uncertainty reduction than to models of threat learning. Importantly, by comparing forward and backward Bayesian probabilities, we observed that the strongest correspondence emerged for the backward uncertainty strategy. Within the context of Pavlovian fear conditioning, autonomic responses may thus reflect how people learn and update their certainty about what causes the US (or its absence), rather than simply anticipating how certain or uncertain an outcome will be.

These results also suggest that SCRs in Pavlovian fear conditioning may not simply reflect the anticipation of threat^{6,36}. Instead, they appear to index dynamic updates in beliefs about environmental contingencies. This interpretation is consistent with previous work showing that SCR signals are particularly sensitive to learning-related uncertainty^{31,42,43}. Computational and neurobiological accounts distinguish between several forms of uncertainty^{21,25,26,36,43,45}: Irreducible uncertainty refers to variability arising from stochastic noise in the relationship between stimuli and outcomes; estimated uncertainty reflects incomplete knowledge about stimulus–outcome associations; and unexpected uncertainty refers to uncertainty about which environmental context governs stimulus–outcome contingencies. The uncertainty model examined in the present study most closely corresponds to estimated uncertainty, as early learning trials in Pavlovian conditioning paradigms are characterized by ambiguity about the causal structure of the environment, which is progressively reduced as evidence accumulates.

Despite uncertainty learning emerging as the predominant account at the group level, we observed substantial variability in the extent to which SCR dynamics aligned with threat learning or uncertainty reduction models. This indicates that physiological patterns across individuals were not governed by a single learning strategy. More importantly, this variability was systematically related to anxiety-related traits. The trial-by-trial representational approach revealed that higher scores in trait anxiety were associated with reduced coupling between SCR dynamics and both threat learning and uncertainty reduction models. Crucially, conventional aggregated SCR indices did not differentiate individuals based on anxiety levels, suggesting that the degree to which physiological signals align with learning processes may provide a more informative marker of anxiety-related differences than indices of CS discrimination³⁶.

Inter-subject structural representations further revealed that the relationship between anxiety and physiological–learning coupling followed an asymmetric pattern across the anxiety continuum. Individuals with lower anxiety exhibited more heterogeneous patterns of physiological responses, whereas individuals with higher anxiety showed more homogeneous patterns of uncoupling. This asymmetric organization suggests that anxiety may constrain the range of physiological–learning dynamics expressed across individuals. One possible interpretation is that heightened anxiety is associated with a more consistent uncoupling between physiological responding and adaptive learning of environmental contingencies, whereas individuals with lower anxiety may flexibly deploy multiple learning strategies, resulting in greater variability in how physiological signals track environmental contingencies among these individuals.

These results offer novel insight into mechanisms underlying anxious symptomatology. A factor that may contribute to the development and maintenance of anxiety is an impaired ability to efficiently adapt to continuously changing environments^{24–26}. Emerging proposals suggest that such deficits may be reflected in difficulties

synchronizing physiological needs with dynamic environmental contingencies, giving rise to discrepancies between expected and actual bodily signals^{24,32,35,46}. This mismatch may lead to heightened sensitivity to bodily signals and increased monitoring of physiological sensations^{47–49}. Over time, this pattern could contribute to persistent physiological symptoms (e.g., fatigue, muscle tension, sleep disturbances) that are part of the clinical anxiety profile. The present findings link anxiety tendencies to reduced coupling between physiological dynamics and adaptive learning strategies, thereby supporting the view that anxiety may involve altered physiological adaptation to changing environments.

Our findings may also have important implications for the development of clinical interventions that target the integration and regulation of bodily information to alleviate psychiatric symptomatology. In this regards, recent evidence suggests that enhancing interoceptive abilities^{47,50} or improving bodily regulation⁵¹—thereby favoring body synchronization to environmental changes⁵²—may represent promising avenues for reducing anxiety-related symptoms.

The observation that individuals with higher anxiety exhibited weaker coupling between physiological responses and learning dynamics may also help reconcile longstanding inconsistencies in the fear conditioning literature^{17,18}. Rather than challenging the usefulness of Pavlovian paradigms for studying anxiety^{3,53}, the present findings suggest a shift in perspective. Fear conditioning tasks may be particularly valuable not only as models for identifying reduced safe responses per se, but also as experimental frameworks for examining the synchronization between adaptive learning strategies and bodily responses. Although the present study focused on SCR, extending this approach to other physiological measures commonly recorded in conditioning paradigms—such as startle eye blink reflex or heart rate^{31,54–56}—may help determine whether disruptions in body-environmental dynamics coupling associated with anxiety are modality-specific or reflect a broader physiological phenomenon.

Despite the novelty of the current findings, some considerations should be addressed in future research. We used a data aggregation approach to overcome the limitations of limited sample sizes; however, the current samples consisted primarily of community participants, most of whom were students. Future research should therefore replicate and extend these findings in clinical samples.

In summary, the present findings demonstrate that autonomic dynamics during fear conditioning are more closely linked to uncertainty learning than to threat discrimination and that the alignment between physiological signals and learning about environmental changes varies systematically with anxiety tendencies. These results highlight the importance of studying physiological responses in relation to learning dynamics and suggest that disruptions in bodily synchronization with environmental changes may represent a key mechanism underlying anxiety-related variability. This perspective may open new avenues for intervention protocols targeting the integration and regulation of bodily signals.

Methods

Studies 1a and 1b

Participants

The sample consisted of 51 students from the University of Potsdam who took part in the study in exchange of course credits. All participants had normal or corrected-to-normal vision. Each individual provided written informed

consent for a protocol on social perception approved by the ethics committee of the University of Potsdam, adhering to the principles of the Declaration of Helsinki. Participants could participate in the study if they were physically and mentally healthy and between the ages of 18 and 50. Exclusion criteria included impaired hearing or color vision, excessive smoking (more than ten cigarettes a day), being a competitive athlete, suffering from any neurological or mental disorders, brain surgery, undergoing chronic or acute medication, or having a history of migraine and/or epilepsy. 3 participants were excluded due to technical issues, leaving a total of 48 participants for the final analysis (37 female, 11 male; $M_{\text{age}} = 24.04$, $SD_{\text{age}} = 4.47$).

Table 3

Summary of the 10 aggregated samples. Log: log-transformed;CDA: continuous decomposition analysis; TTP: through-to-peak method; Max: Maximum; RC: range-corrected.

Study	Initial Sample	Final Sample	N valid data for RSA	Contingency CS+	N_{trial} (CS+/CS-)	SCR transformation method	STAI-T scores	Reference
<i>Study 1a</i>	51	38	38	100%	10/10	Log(CDA)	Yes	Unpublished data
<i>Study 1b</i>	51	38	38	60%	10/10	Log(CDA)	Yes	Unpublished data
<i>Study 2</i>	126	110	105	75%	12/12	Log(TTP)	Yes	Wendt et al. (2020) ⁵⁷
<i>Study 3</i>	128	117	111	50%	16/16	Log(TTP)	Yes	Blaser et al. (2023) ⁵⁸
<i>Study 4</i>	162	157	143	100%	12/12	Log(TTP)	Yes	Neubert et al. (2017); Wendt et al. (2015); Wendt, Neubert et al. (2015) ^{59–61}
<i>Study 5</i>	66	61	61	100%	9/9	Log(TTP)	Yes	Klingelhofer, Morris, & Lonsdorf (2022) ⁶²
<i>Study 6</i>	29	27	27	50%	30/30	Log(CDA)	No	Xia et al., 2021 ⁶³
<i>Study 7a</i>	120	107	107	100%	14/14	Log(TTP)	No	Klingelhofer et al. (2022a) ⁶⁴
<i>Study 7b</i>	120	71	68	100%	14/14	Log(TTP)	No	Klingelhofer et al. (2022b) ⁶⁴
<i>Study 8</i>	133	120	119	75%	8/8	Log(Max – baseline)	Yes	Mertens et al. (2021) ⁶⁵
<i>Study 9</i>	70	66	65	50%	10/10	RC(log(TTP-baseline))	Yes	Andres et al. (2024) ⁶⁶
<i>Study 10</i>	96	88	88	66%	9/9	Log(Max – baseline)	No	Wong & Beckers (2021) ⁶⁷
	1101	962	932					

Procedure

For the differential Pavlovian fear conditioning paradigm, four different geometrical forms were used as conditioned stimuli: a pink square, a yellow circle, a green triangle, and a blue pentagon. Participants underwent two different differential conditioning paradigms in two consecutive blocks. In one block, the CS + was reinforced in every trial (100% contingency; Study 1a). In the other block, the CS + was reinforced in 60% of the trials (Study 1b). In both blocks, the CS- was never paired with the shock. Each stimulus was used as CS + or CS- in any of the blocks. Both stimulus-block assignments were counterbalanced across participants. Visual stimuli were displayed on a black background in the center of the computer screen, each with a duration of 8 seconds. The intertrial intervals (ITI) varied in length between 7 and 11 seconds. The task was programmed with Presentation software (Version 20.3, Neurobehavioral Systems, Inc.). Additionally, an acoustic startle probe was presented in every trial. The startle probe was a 95 dB, 50 ms burst of white noise with a near-instantaneous rise time. The sound was administered in both ears using Audio Technica ATH-PRO700 MK2 headphones.

Each block consisted of 20 trials (10 CS+, 10 CS-). CSs presentation was pseudorandomized with no more than two consecutive presentations of the same CS. The order of the blocks was counterbalanced across participants. An electro-tactile stimulus of 1ms duration was used as unconditioned stimulus (US). The intensity was individually adjusted for each participant before fear acquisition to a level judged to be "very unpleasant but not painful". The US was administered 50 ms before stimulus offset. The stimulation electrode was placed on the inside of the right leg, approximately 3 cm above the ankle. Electrical stimulation was controlled using a DS7A Constant Current Stimulator (Digitimer, Hertfordshire, UK).

Trait anxiety was assessed with the German version of the State-Trait Anxiety Inventory (STAI³⁷). The trait scale of this self-assessment method assesses anxiety as a personal trait, which is persistent over time and different situations. High scores indicate a predisposition to react with anxiety in stressful situations. The scale includes 20 self-statements (e.g. "I wish I could be as happy as others seem to be"), and participants are asked to report how they feel in general. Answers are given on a 4-point Likert scale with the answer options *almost never*, *sometimes*, *often*, and *almost always*.

Physiological Measures.

SCR. electrodermal activity (EDA), was registered using a BIOPAC MP160 amplifier system and AcqKnowledge 5.0.2 software (BIOPAC Systems, Inc., Goleta, CA, USA). The EDA was sampled at a rate of 2,000 Hz and recorded using 2 shielded 8 mm Ag-AgCl sensors applied on the thenar and hypothenar eminence of the palmar surface of the participant's left hand⁶⁸. Skin conductance responses (SCRs) were analyzed using Continuous Decomposition Analysis in Ledalab Version 3.4.9 (Benedek & Kaernbach, 2010). The skin conductance data were downsampled to a resolution of 50 Hz and parameters of the impulse response function (IRF) used to decompose the SC were optimized using four sets of initial values (see for details on Continuous Decomposition Analysis⁶⁹). The response window was set from 1 to 4 s after stimulus onset. Prior to performing the analysis, a visual inspection of the data was carried out to detect possible anomalies during recording.

Participants were considered SCR non-responders if they had more than two thirds of zero responses (responses lower than .01 μ S) to the US ($N_{US} = 16$; as recommended by⁷⁰). 10 non-responders were detected and excluded for the SCR analyses, leaving a final sample of 38 participants.

Study 2

Participants

A total of 126 healthy participants (age range: 15–35 years) were recruited from the University of Greifswald. Participants were invited to take part in the study if they did not fulfill any of the following exclusion criteria: impaired hearing or color vision, excessive smoking habits (more than 10 cigarettes a day), being a competitive athlete, self-reported diagnosis of a neurological or mental disorder, regular intake of medication that affects the cardiovascular or central nervous system, neurodermatitis or pregnancy. 6 participants were excluded from the analysis due to technical issues or at their own request. The final sample consisted of 120 participants (60 female, 60 male; see for details⁵⁷).

Procedure

Two faces with neutral expressions were used as CSs. Each CS was presented 12 times in a pseudorandomized order, with no more than two consecutive trials of the same CS. Each CS was presented during 7 sec followed by an ITI that varied between 11 and 18 sec. In 75% of the CS+, the US was presented 50 ms prior to the CS offset. The CS- was never paired with the US. The US was a 10 ms train of 500 Hz single electrical pulses (1 ms; S48K; Grass Instruments, West Warwick, RI), administered to the left ankle, and individually adjusted to a level that was “highly unpleasant, but not painful”. Moreover, bursts of white noise (duration of 50 ms duration, 95 dB volume) were used as acoustic startle probes. Bursts were delivered binaurally through headphones 4.5 or 5.5 seconds after each CS onset and 11–13 sec after cue offset (see for details⁵⁷).

Trait anxiety was assessed with the German version of the STAI³⁷.

Physiological Measures.

SCR. EDA was recorded from the hypothenar eminence of the palmar surface of the participant’s right hand, using a Coulbourn S71-22 with a constant 0.5 V across two Ag/AgCl standard electrodes (8 mm diameter; Gelimed) filled with electrolyte medium. The analog signal was sampled continuously at a rate of 10 Hz. SCRs were scored from trough-to-peak as the first increase starting between 0.9 and 4.0 s after cue onset (first interval response; minimal response criterion = 0.04 μ S). SCRs were log-transformed to compensate for skewed distributions. Data from 9 participants could not be analyzed due to technical problems. Moreover, data from 1 participant was excluded due to showing no responses to the US in more than 66% of the trials, leaving a total of 110 participants with valid SCR data.

Study 3

Participants

The total sample consisted of 128 healthy participants (age range 18–40 years) recruited at both University of Potsdam and University of Greifswald (see for details, ⁵⁸). Exclusion criteria included having a body mass index lower than 18.5 kg/m² or higher than 30 kg/m², cardiovascular, neurological, or respiratory diseases, impaired hearing or color vision, claustrophobia, pregnancy, and the use of medications that alter the normal functions of the autonomic nervous system.

Procedure

A blue square and an orange circle were used as CSs. The geometrical forms were presented on a black background for 6 sec with ITIs ranging between 14 and 16 sec. A total of 16 CSs were presented (8 CS+, 8 CS-). In 50% of the CS+ trials, an electro-tactile stimulus for 1 ms duration (US) was presented 50 ms prior to CS offset. The CS- was never accompanied by an electro-tactile stimulus. The intensity of the electric shock was individually adjusted to be “very unpleasant but not painful”. The stimulation electrode was placed on the inside of the right leg, approximately 3 cm above the ankle. Bursts of white noise (duration of 50 ms duration, 95 dB volume) were used as startle acoustic startle probes, delivered binaurally through headphones (Potsdam: Audio-Technica ATH-PRO700MK2, Greifswald: AKG K 66). Startle probes were administered 4.5 or 5.5 sec after each CS onset and in half of the ITI, 7.5 seconds after CS offset (see for details ⁵⁸).

Trait anxiety was assessed with the German version of the STAI³⁷.

Physiological Measures.

SCR. EDA was recorded using the EDA100C module of a BIOPAC MP160 amplifier system with a constant 0.5 V across two Ag/AgCl standard electrodes (8 mm diameter; Easycap GmbH) filled with electrolyte medium that were attached to the hypothenar muscles. The EDA data were sampled at 2000 HZ and passed through a 10 Hz low-pass filter. Data from 1 participant could not be analyzed due to technical problems. Moreover, 10 participants were identified as non-responders (no SCRs in more than 66% US trials), leaving a total of 117 participants with valid SCR data. SCRs were scored from trough-to-peak as the first increase starting between 0.9 and 4.0 s after cue onset (minimal response criterion = 0.04 μ S), using Ledalab Version 3.4.9⁶⁹. SCRs were log-transformed to compensate for skewed distributions.

Study 4

Participants

Study 4 consisted of 162 (114 female, 58 male, age range = 19–36 years) healthy participants recruited at the University of Greifswald. Exclusion criteria were mental or neurological disorders, regular intake of medication affecting the central nervous system, color blindness, loss of hearing, and being of non-Caucasian descent. Participants' current and lifetime mental health was verified by way of the Structured Clinical Interview for DSM-IV (see for details,^{59–61}).

Procedure

Geometrical shapes (blue pentagon, pink triangle, yellow trapezoid, and black star^{60,61}), or insects (a butterfly and a spider⁵⁹) were used as visual conditioned stimuli. The US consisted of a 10 ms train of 500 Hz single electrical pulse applied to the participants' left ankle. The intensity of the US was individually calibrated to be perceived as highly unpleasant, but not painful. During the fear conditioning task, geometrical shapes were presented in pairs (e.g., AX+ or BX-). The AX+ stimulus was always accompanied by the US whereas the AX- was never paired with the electro-tactile stimulus. The US was presented 50 ms prior to CS offset. A total of 24 CS pairs were presented (12 AX+, 12 BX-) on a black screen for 7 sec with ITIs varying between 11 and 23 sec. The order of the CS presentation was pseudorandomized with no more than two consecutive stimuli of the same condition. The acoustic startle probe was 95 dB(A) white noise presented binaurally 5.5 or 6.5 s after every cue onset and 6 times during the ITIs.

Trait anxiety was assessed with the German version of the STAI³⁷.

Physiological Measures.

SCR. EDA was recorded from the hypothenar eminence of the palmar surface of the participant's right hand. A Coulbourn S71-22 provided a constant 0.5 V across two Ag/AgCl standard electrodes (8 mm diameter; Gelimed) filled with electrolyte medium. The analog signal was sampled continuously at a rate of 10 Hz. SCRs were scored from trough-to-peak as the first increase starting between 0.9 and 4.0 s after cue onset (minimal response criterion = 0.04 μ S). SCRs were log-transformed to compensate for skewed distributions. Participants were excluded for SCR analysis, if they showed no SCRs in more than 66% US trials. 5 participants were identified as non-responders, leaving a total of 157 participants for SCR analysis.

Study 5

Participants

A total of 66 participants were recruited at the University Medical Center of Hamburg (44 female, 22 male; age range: 18–40 years). Participants were selected to take part in the study if they did not report any of the following exclusion criteria: current or regular intake of medication (exception: oral contraceptives), substance abuse, neurological, psychiatric or chronic diseases, experienced trauma, age under 18 or over 40 years, or no fluent German language. 3 participants had to be excluded due to technical problems (see for details⁶²).

Procedure

Two white snow fractals were used as CSs and were presented for 8 sec in the center of the screen on a gray background with an ITI ranging from 10 to 13 sec. The CS+ was always paired with the US (100% contingency rate), whereas the CS- was never associated with it. Stimulus assignment to the CS+ and CS- conditions was counterbalanced across participants. The US, which consisted of a train of three 2ms electro-tactile square-waves (inter stimulus interval, ISI: 50 ms; Digitimer DS7A) administered on the back of the right hand, was calibrated to be unpleasant, but still tolerable. The US was presented 200 ms prior to CS+ offset. A total of 18 CSs were presented (9 CS+, 9 CS-), the CS presentation was pseudorandomized, with the condition that the CS was presented more than two times consecutively. The startle probes consisted of a 95dB white noise burst presented simultaneously on both ears via headphones. Startle probes were administered 6 or 8s after stimulus offset in one third the ITI-onset, and 6 or 7s after CS-onset in 6 out of the 9 CS trials (see for details⁶²).

Trait anxiety was assessed with the German version of the STAI³⁷.

Psychophysiological measures

SCR. EDA was recorded using BIOPAC MP 100 amplifier (BIOPAC Systems, Inc., Goleta California, USA) from the palm of the left hand with two Ag/AgCl-sensors located on the hypothenar muscles. A 1 Hz lowpass filter and a gain of 5 μ Ω were applied. Data were sampled at 1000 Hz. SCR in response to the CSs was operationalized as the onset of the first response within 0.9 to 4 s after stimulus onset from trough to peak. 2 participants were identified as non-responders (i.e., showing no SCR in more than 66% of the US trials) and thus excluded from SCR analysis. SCRs under 0.0 μ S or an absence of it within the defined time window were classified as a zero-responses. Data with recording artifacts or excessive baseline activity were treated as missing data points. The SCR data was square root transformed to reduce skewness, and thereafter z-scored (see for details⁶²).

Study 6.

A total of 29 healthy unmedicated participants with normal or corrected-to-normal vision recruited from general population (17 females and 12 males, $M_{\text{age}} = 25.3$ $SD_{\text{age}} = 3.7$) (see details⁶³). All participants provided written informed consent, and the study was conducted in accordance with the Declaration of Helsinki and approved by the governmental research ethics committee (Kantonale Ethikkommission Zürich: KEK-ZH-2013-0118).

Procedure

Two different full-screen colorful patterns (equal brightness, contrast, and spatial frequency) were used as CS stimuli. The assignment of each pattern to the CS conditions was counterbalanced across participants. The CS + was paired with the US in half of the trials (50% contingency rate) while the CS- was never paired with the US. The CS was presented during 4 sec (ITIs randomly varied between 7 and 11 sec) and followed (when presented) by the US, which consisted of a 500-ms train of 250 square electric pulses set. The intensity of US was estimated in a two-phase procedure. Participants underwent a staircase testing phase to determine the pain threshold by delivering a series of gradually strengthened shocks from unperceivable to clearly painful level. Thereafter a random test of 14 stimuli below this value were administered and participants were instructed to rate them on a scale from 0% (no sensation) to 100% (clearly painful). The final intensity used during experiments corresponded to 85% of the initial pain threshold. During the first acquisition phase, a total of 60 trials were presented (30 CS + and 30 CS-). Pattern-CS assignment was randomized across participants. During the experimental task, participants' heads were positioned on a chin rest in front of the monitor with a distance of 700 mm from head to monitor.

Psychophysiological measures.

SCR. EDA was recorded from the thenar/hypothenar of non-dominant hand by two 8-mm disk Ag/AgCl cup electrodes (EL258, Biopac Systems Inc., Goleta, CA) with an SCR coupler/amplifier (V71-23, Coulbourn Instruments). The openly available raw EDA data was used to extract SCRs. The CDA in Ledalab Version 3.4.9⁶⁹ was employed to extract the SCRs (i.e., same procedure as for Studies 1a and 1b). Data quality was reassessed in the current study. After reevaluation, 2 participants (compared to 3 in the original study) were excluded due to artifact or showing no response to the US⁶³.

Studies 7 a and b

Participants

The original sample consisted of 120 participants recruited via phone interview. Participants were invited to participate if they were healthy individuals between 18 and 50 years of age and did not fulfill any of the following exclusion criteria: having a history of childhood trauma, claustrophobia, cardiac pacemaker, non-MR-compatible metal implants, brain surgery, left handedness, participation in pharmacological studies within the past 2 weeks, medication (except for oral contraceptives), internal medical disorders, chronic pain, neurological disorders, psychiatric disorders, metabolic disorders, acute infections, complications with anesthesia in the past and pregnancy. All participants had normal or corrected-to-normal vision. Participants underwent a differential conditioning task at two different time points (approximately six months apart(see for details⁶⁴). Data from the first time point was coded as *Study 7a* and data from the second timepoint was coded as *Study 7b*.

Procedure

Two snow fractals centered on the screen with a gray background were used as CS stimuli. The CS + was always paired with the US (100% contingency rate), whereas the CS- was never paired with the US. The condition assigned

to each snow fractal was counterbalanced across participants. CSs were presented for 6 to 8 sec. The US followed CS + 0.2 sec before stimulus offset. The US was a train of three 2 ms electro-tactile rectangular pulses with an inter-pulse interval of 50 ms administered to the back of the right hand. The US was individually calibrated to a level that was unpleasant but still tolerable. A total of 28 trials were presented (14 CS+, 14 CS-). Trial order was pseudorandomized with no more than two consecutive trials of the same condition.

Psychophysiological measures

SCR. EDA was acquired continuously during each phase of conditioning using a BIOPAC MP 100 amplifier (BIOPAC Systems, Inc, Goleta, CA, USA) and Spike 2 software (Cambridge Electronic Design, Cambridge, UK). For analog to digital conversion, a CED2502-SA was used. Two Ag/AgCl-sensor recording electrodes (diameter = 55 mm) were attached to the palm of the left hand on the distal and proximal hypothenar. A 10 Hz lowpass filter and a gain of 50 were applied. Data were recorded at 1000 Hz and later downsampled to 10 Hz. Subsequently, SCRs were scored semi-manually using the custom-made computer program *EDA View* (developed by Prof. Dr. Matthias Gamer, University of Würzburg). SCRs to the CSs were quantified from the onset of the first response within 0.9 to 3.5 s after stimulus onset based on the through-to-peak method. SCRs confounded by recording artifacts due to technical reasons, such as electrode detachment or responses moving beyond the sampling window, were discarded and scored as missing values. SCRs smaller than 0.01 μ S were defined as zero responses. In Study 7a, 13 participants were removed from SCR analysis due to artifacts or because they were categorized as non-responders (non-responders were defined as participants with zero responses to the US in more than two-thirds [i.e., more than 9 out of 14] of US acquisition trials; see for details⁶⁴), leaving a final sample of 107 participants. In Study 7b, 49 participants were excluded from the analysis due to dropouts, recording issues or being identified as non-responders. The final sample of Study 7b was 71. For the current analysis, we used log-transformed SCRs ⁶⁴.

Study 8

Participants were invited to take part in the study if Dutch was their first language; had good (corrected) hearing and vision; took no medication use which can impair attention, reaction time, memory, or concentration; did not suffer from psychiatric disorder in the last two years; were not under treatment with a psychiatrist or psychologist currently or in the past two years; were not pregnancy; and had no current or past serious neurological or medical conditions (such as epilepsy or heart disease). The number of participants recruited was determined by the quality of the data until the predefined sample of 120 participants with good data quality (e.g., no artifactual data, no recording problems, no lack of contingency awareness) was achieved. A total of 133 participants were recruited, with 13 being excluded due to the abovementioned reasons (see for details⁶⁵). The final sample consisted of 37 men and 83 women with an average age of 22.63 years ($SD_{age} = 2.76$). All participants provided informed consent prior to participation and the procedure of this study was approved by the Ethics Review Board of the Faculty of Social Sciences of Utrecht University (reference code: FETC16-054).

Procedure

Words were used as CS stimuli. Words, which were related to variations in size (i.e., mini, enormous), were presented in the middle of the screen with font style 'Arial' and font size 36. The duration of the CSs was 8s. During the acquisition phase, a total of 16 trials were presented (8 CS+, 8 CS-). In six of the eight trials, the US followed immediately after the CS+ (75% contingency), while the CS- was never paired with the US. A 500 ms electric shock was used as the US. The US was delivered to the dominant hand, using a Digitimer DS7a device). The shock intensity was individually adjusted to ensure that it was uncomfortable but not painful. Startle probes were

presented 7s after CS onset and during the ITI. Inter-probe intervals ranged between at 19 and 23s. Startle was elicited by a 95 dB white noise probe for 50 ms.

The Dutch version of the STAI, trait version (STAI-DY-2³⁷) was used to assess trait anxiety levels.

Physiological Measures

SCR. EDA was measured using two BioSemi GSR electrodes (BioSemi, Amsterdam, the Netherlands) filled with conductive gel and attached on the non-dominant hand of the participant. The skin conductance signal was first downsampled to 10 Hz using BrainVision Analyzer software (Brain Products, Munich, Germany). Responses were calculated as the difference between the mean SCR for the 2 s preceding CS onset and the highest value recorded during the full 7 s CS-US interval. A response threshold of 0.02 μ S was applied for the SCRs (Boucsein et al., 2012). Responses below this cut-off were replaced with 0. The log-transformed SCR was used for the analysis.

Participants were excluded, if their data was of poor quality, if they did not show any discernible SCRS towards the US administration or if they had more than 50% unusable startle response datapoints. Excluded participants were replaced by new participants to maintain the targeted sample size (N = 120).

Study 9

Participants

70 healthy male participants were recruited for this study. Participants were invited to participate if they did not fulfill any of the following exclusion criteria: contraindications of L-DOPA intake, current physiological, neurological, or psychiatric disorders, excessive consumption of nicotine (more than 10 cigarettes a day), alcohol (more than 15 glasses of beer or wine per week), or cannabis (more than 1 joint per month), participation in other pharmacological studies, and skin conductance non-responding (see for details, ⁶⁶). The experiment was approved by the local ethics committee (Ethikkommission der Landesärztekammer, Rhineland-Palatinate, Germany, reference code: 837.561.17 (11395)) and was conducted in accordance with the Declaration of Helsinki.

Procedure

Black geometric forms (a square and a rhombus) superimposed on three gray-scaled photos (living room, kitchen, and sleeping room) were used as CS stimuli. The assignment of the geometric form to the CS condition was counterbalanced across participants. During the acquisition phase, the context picture remained on the screen throughout the task. A total of 20 trials were presented (10 CS+ and 10 CS-). In five out of 10 CS+ trials, the US was presented 50 ms prior to CS+ offset (50% contingency rate). The US consisted of a painful electrical stimulation of three square-wave pulses of 2 ms (50 ms inter-stimulus interval) generated with a DS7A electrical stimulator (Digitimer, Weybridge) and delivered to the ankle. The US intensity was individually adjusted to a level rated as “maximally painful but still tolerable”. CSs were presented for 4.5 s. Inter-trial intervals (ITIs) lasted 17, 18, or 19 s. Trial order was randomized in such a way that not more than two trials of the same type were consecutively presented (see for details, ⁶⁶).

The STAI, trait version was employed to assess trait anxiety levels ³⁷.

Physiological Measures

SCR. EDA was recorded from the thenar and hypothenar of the non-dominant hand using self-adhesive Ag/AgCl electrodes prefilled with an isotonic electrolyte medium and the Biopac MP150 with EDA100C device (EL-507,

BIOPAC® Systems Inc., Goleta, California, USA). SCRs were visually scored offline in a time window from 0.9 sec to 4 sec after CS onset. The phasic amplitude of SCRs was calculated by subtracting the tonic skin conductance level (SCL) from the subsequent peak, using a custom-made analysis script. SCRs with amplitudes smaller than 0.02µs were scored as zero and remained in the analysis. If more than 75% of trials during an experimental session were scored as zero, data of this participant during that session was regarded as invalid and excluded from SCR. Hence, in total, data from 4 participants were excluded, leaving a total of 66 participants with available SCR data during acquisition. To normalize distributions, data was log-transformed and range-corrected for each participant, using the data from the acquisition phase

Study 10

The sample consisted of psychology undergraduate students from KU Leuven. A total of 98 participants took part in the study. 8 participants were excluded for not showing CS-US contingency awareness, leaving a final sample of 90 participants (see for details, ⁶⁷). All participants provided consent prior to participation and this study was approved by the KU Leuven Social and Societal Ethics Committee (G201709860).

Procedure

Twelve grayscale images from birds and mammals depicted on a black background were used as CS stimuli. The task was divided into 3 blocks. In each block, three different exemplars of one category (e.g., mammals) served as CS+ and three exemplars of the other category acted as CS-. Each block consisted of 6 trials (3 CS+, 3CS-; each exemplar presented once). The CS+ were reinforced with a contingency rate of 67% (2 out of 3 trials). A 2-ms electro-tactile stimulus generated with a Digitimer DS7A stimulator was employed as US. The intensity of the US was individually calibrated to be felt as “definitely uncomfortable but not painful”. The CS- was never paired with the US. The image category (e.g., birds, mammals)-stimulus condition (CS+, CS-) assignment was counterbalanced across participants. CS was presented for 8 sec. The startle probe, a 40-ms white noise of 100 dB delivered via headphones, was presented 7 sec after stimulus onset. Additionally, 9 startle probes were presented in absence of any CS. ITIs ranged from 10 to 15 sec. The stimulus order presentation was pseudo-randomized to avoid that the same trial type was presented more than twice in a row.

The Dutch version of the Depression Anxiety Stress Scale-21⁷¹.

Physiological Measures

SCR. EDA was measured via two disposable Ag/AgCl electrodes attached to the palm of the non-dominant hand, at a sampling rate of 1000 Hz. For each trial SCRs were obtained by subtracting the averaged skin conductance level during 2 s prior to CS onset from the maximum response within 7 s after CS onset. Negative SCRs were scored as zero. All SCRs were then square-root transformed to reduce skewness.

Analysis

Effects of CS type, Trial and their interaction on skin conductance response.

To assess whether datasets reproduced the expected pattern observed in Pavlovian fear-conditioning paradigms characterized by enhanced autonomic responding to CS+ cues relative to CS- cues, we fitted linear mixed-effects

models within each study. Models included fixed effects of *CS type* (CS+ versus CS-), *Trial* (continuous), and their interaction. Participants intercepts were modeled as random effects (Figure S1 for visual depiction of each study).

To quantify the strength of evidence for CS type, Trial, and interaction effects, we adopted a Bayesian model comparison framework⁷². This approach evaluates the relative predictive adequacy of competing models and quantifies the extent to which the observed data support one model over another. Specifically, we computed logarithmic Bayes factors (BF_{10}) of the effects of interest by comparing the full model against a corresponding null model in which the relevant effect was omitted. Higher $\log(BF_{10})$ values therefore indicate stronger evidence in favor of the alternative hypothesis relative to the null model.

Representational Similarity Analysis for the correspondence between SCR and models of threat learning and uncertainty reduction.

We conducted representational similarity analysis (RSA) to assess the correspondence between the representational structure of SCR and models of threat and uncertainty reduction in each study separately.

Computation of Representational Similarity Matrices (RSMs) of learning strategies and SCR.

The threat learning model encoded changing CS–US contingencies. It predicted that early in acquisition, responses to CS + and CS- would be similar due to ambiguous contingencies but would progressively diverge as evidence accumulated—contingency strength increasing for CS + and decreasing for CS-. Trial-wise updates were computed using both forward ($P_{US|CS}$) and backward ($P_{CS|US}$) Bayesian probabilities, a probability of $P = 0.5$ was used as prior in both models:

$$\text{Forward model : } P(US|CS) = \frac{P(US) P(CS|US)}{P(CS)}$$

$$\text{Backward model : } P(CS|US) = \frac{P(CS) P(US|CS)}{P(US)}$$

By contrast, the uncertainty reduction model captured the gradual reduction of uncertainty across trials. It predicted that both CS types would initially be maximally uncertain with respect to the US, with certainty increasing as evidence grew—irrespective of whether the outcome signaled threat or safety. Operationally, uncertainty was quantified as the absolute deviation of the conditional probabilities $P_{US|CS}$ and $P_{CS|US}$ on each trial from the maximal uncertainty ($P = 0.5$).

Trial-by-trial probabilities for threat and uncertainty reduction were estimated based on the experimental trial structure whenever this information was available (Studies 1–4 and 6). For studies in which trial order or contingency information was unavailable in open repositories, learning trajectories were approximated using averaged probabilities derived from a paradigm with identical or closely matching CS–US contingencies. For example, model estimates for Study 5 were adapted from Study 4, as both employed a CS+/US contingency of 100%.

To construct physiological representational similarity matrices (RSMs), we used the SCR metric available in each study (e.g., log-transformed mean phasic driver or log-transformed trough-to-peak amplitude). The construction of physiological and model-based RSMs followed a two-step procedure.

First, for each participant, a physiological RSM was generated by ordering trials according to CS type (CS+, CS-) and trial number. Pairwise trial similarity was computed as the maximum value of the dataset minus the absolute difference between the two trials, yielding a trial-by-trial similarity structure. Model-based RSMs were constructed using the same procedure applied to the model-derived trial-wise probabilities whenever the necessary information was available (Suppl. Section S4-S7; see for comprehensive example, Suppl. Section S3).

Second, study-level physiological- and model-based RSMs were obtained by averaging participant-level RSMs within each study. In studies where individual-level data of the trial-by-trial probabilities from the learning models were unavailable (due to the absence of the true trial sequence), a single RSM was computed based on the adapted model probabilities described above.

Comparison between physiological and model-based similarity structures

To quantify the correspondence between physiological dynamics and model-derived learning trajectories, averaged physiological RSMs were compared with the model-based RSMs using Spearman rank correlations, implemented with the *stats* package in R^{38,40}.

Evidence for the presence or absence of positive associations was assessed within a Bayesian framework. Specifically, Bayes factors for linear correlations were computed using the *BayesFactor* package⁷³ in R environment (v. 4.3.3), with a null distribution derived from 1,000 random label permutations. This approach allowed us to quantify the relative evidence for the null hypothesis (no positive association) versus the alternative hypothesis (a positive association) between physiological and model-derived similarity structures.

Finally, logarithmic Bayes factors ($\log[\text{BF}_{10}]$) obtained for each study and model correspondence were compared using frequentist and Bayesian analysis of variance (MANOVA). Significant effects were followed by frequentist and Bayesian paired *t*-tests, and effect sizes were quantified using Cohen's *d*.

Additionally, we extracted participant-level similarity indices between SCR-based and model-based representational similarity matrices (RSMs), using Spearman's correlation. Whenever trial-level information was available, individualized model-based RSMs were generated according to each participant's trial sequence. When such information was unavailable (due to the absence of the true trial sequence), the adapted model-based RSM derived from the study design was applied to all participants within that study. Similarity scores between SCR and models for each participant were compared using frequentist and Bayesian analysis of variance (MANOVA). Significant effects were followed by frequentist and Bayesian paired *t*-tests, and effect sizes were quantified using Cohen's *d*.

Association between SCR-model correspondence and anxiety-related traits. To assess whether anxiety tendencies modulate the correspondence between physiological dynamics and learning strategies, we conducted an unsupervised cluster analysis using, as input features, each participant's similarity indices between SCR dynamics and the best-fitting model (the backward uncertainty reduction model) as well as its complementary model, the backward threat learning model. The optimal number of clusters was determined using the elbow criterion. Specifically, the clustering algorithm was run across a range of 20 potential cluster solutions, and the within-cluster sum of squares (WCSS) was computed for each solution. WCSS values were plotted as a function of cluster number, and the optimal solution was identified at the point where the reduction in WCSS began to plateau, indicating diminishing returns from additional clusters (Suppl. Section S8).

To assess whether traditional discrimination metrics (SCR difference between averaged CS + and CS- trials) capture similar individual differences, the same clustering procedure was repeated using the discrimination score. Prior to clustering, the CS discrimination index was scaled between 0 and 1 within each study (i.e., using the function *scale* from the package *base* in R) to ensure comparability across datasets.

Finally, clusters were compared on trait anxiety scores (STAI-T) using Bonferroni-corrected pairwise frequentists and Bayesian *t*-tests.

Moreover, leave-one-out cross-validation analysis was performed to assess the robustness of the results. To this end, clusters were subsequently compared using all samples except one ($N_{\text{included samples}} = 7$; Suppl. Section S10).

Inter-subject Representational Similarity Analysis to test symmetric and asymmetric associations with anxiety.

Finally, we examined whether the relationship between anxiety and the coupling between SCR dynamics and computational learning models followed a linear (symmetric) pattern across the anxiety dimension or instead exhibited an asymmetric structure. To address this question, we conducted an inter-subject RSA (is-RSA). Whereas trial-based RSA quantifies similarity across trials to construct representational matrices, is-RSA operates on similarities between participants.

For this analysis, two types of inter-subject representational similarity matrices (is-RSMs) were constructed. First, we generated is-RSMs reflecting similarities across participants in the degree of correspondence between SCR dynamics and the two models used for the cluster analysis: the backward threat learning model and the backward uncertainty reduction model. Second, we constructed two is-RSMs based on participants' trait anxiety scores (STAI-T), each reflecting a different theoretical structure of the anxiety dimension. The first anxiety model assumed a symmetric relationship across the anxiety continuum, predicting that participants with similar STAI-T scores would show similar physiological–computational coupling, whereas participants with more divergent scores would appear more dissimilar. The second model captured an asymmetric relationship, predicting greater differences among individuals with low anxiety and increasing similarity among individuals with higher anxiety.

To construct the four is-RSMs, participants were first ordered according to their STAI-T scores. Pairwise similarity scores were then calculated between all participants. For the symmetric anxiety model and the SCR–learning model RSMs, similarity values were defined as the maximum value of the aggregated sample minus the absolute difference between two participants' scores. For the asymmetric anxiety model, similarity between participants were computed as follows:

$$\text{Asymmetric Anxiety Model: } \textit{Similarity} = \frac{\text{mean}(V_{Ti}, V_{Tj})}{N_{\text{participants}}}$$

V_{Ti} represents the score of participant *i*. V_{Tj} denotes the score of participant *j*. $N_{\text{participants}}$ indicates the number of participants.

Associations between the anxiety-based is-RSMs and the SCR–learning model correspondence RSMs were assessed within a Bayesian framework to quantify evidence in favor of the null hypothesis (no positive association) versus the alternative hypothesis (a positive association). Bayes factors for linear correlations used null distribution generated from 1,000 random label permutations.

Declarations

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Figures

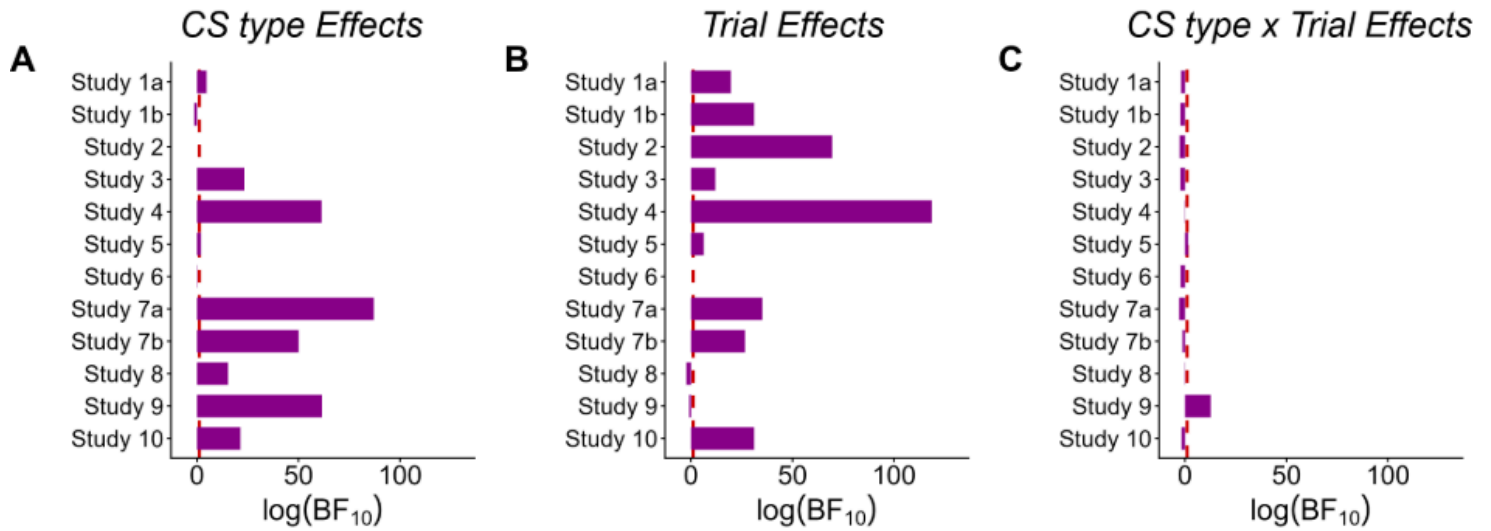


Figure 1

Consistent CS type and Trial effects across studies. (A-C) Bayesian evidence in favor of CS type (A), Trial (B) and CS type x Trial effects for each study. Dotted vertical red lines indicate the threshold above which evidence for an effect is considered ($\log(BF_{10}) = 1.03$).

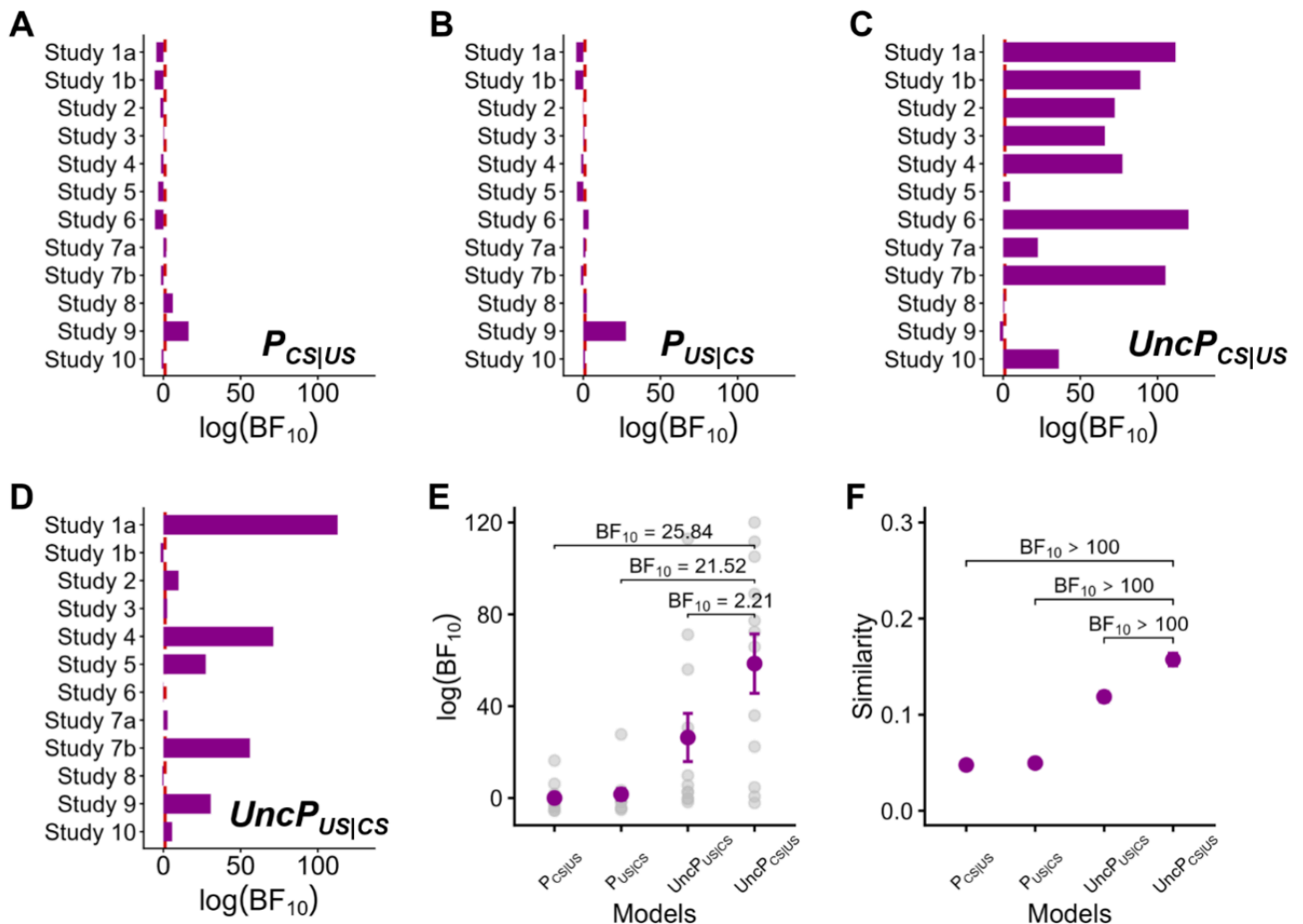


Figure 2

Consistent evidence for an association between SCR patterns and backward models of uncertainty reduction.

Bayesian evidence in favor of a correspondence between SCR and (A) backward ($P_{CS|US}$), and (B) forward ($P_{US|CS}$) models of threat learning, and (C) backward ($UncP_{CS|US}$) and (D) forward ($UncP_{US|CS}$) models of uncertainty reduction in each study. Dotted vertical red lines indicate the threshold above which evidence for an effect is considered ($\log(BF_{10}) = 1.03$). (E-F) Evidence for SCR-learning model correspondence compared across (E) studies and participants (F). Backward models of uncertainty reduction show the strongest evidence for a positive correspondence with SCR patterns.

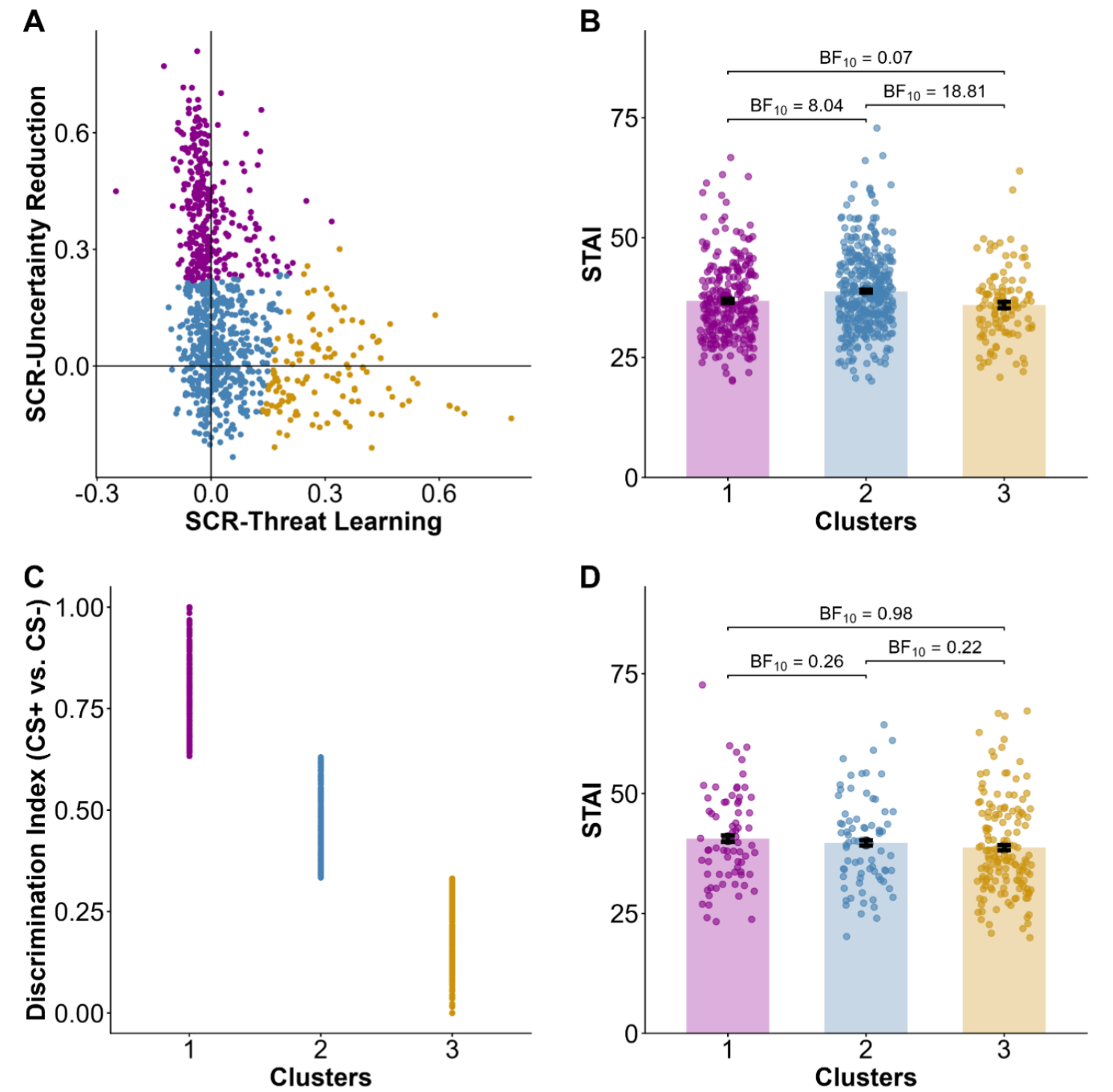


Figure 3

Participants with lower SCR-model coupling report higher anxiety tendencies. (A) Scatterplot of backward SCR-uncertainty reduction and SCR-threat learning correspondence from all participants ($n=932$). Participants are color-coded based on the cluster they were assigned to. (B) Average anxiety tendency scores across clusters based on SCR-model correspondences. Participants on Cluster 1 and 3 (stronger coupling with uncertainty reduction and threat learning strategies, respectively) showed lower anxiety tendencies than participants from Cluster 2 (i.e., weaker coupling). (C) cluster analysis, using the traditionally employed discrimination index (i.e., mean differences between CS+ and CS-; $n=962$). (D) Average anxiety tendency scores across clusters based on the discrimination index. No differences emerged between clusters.

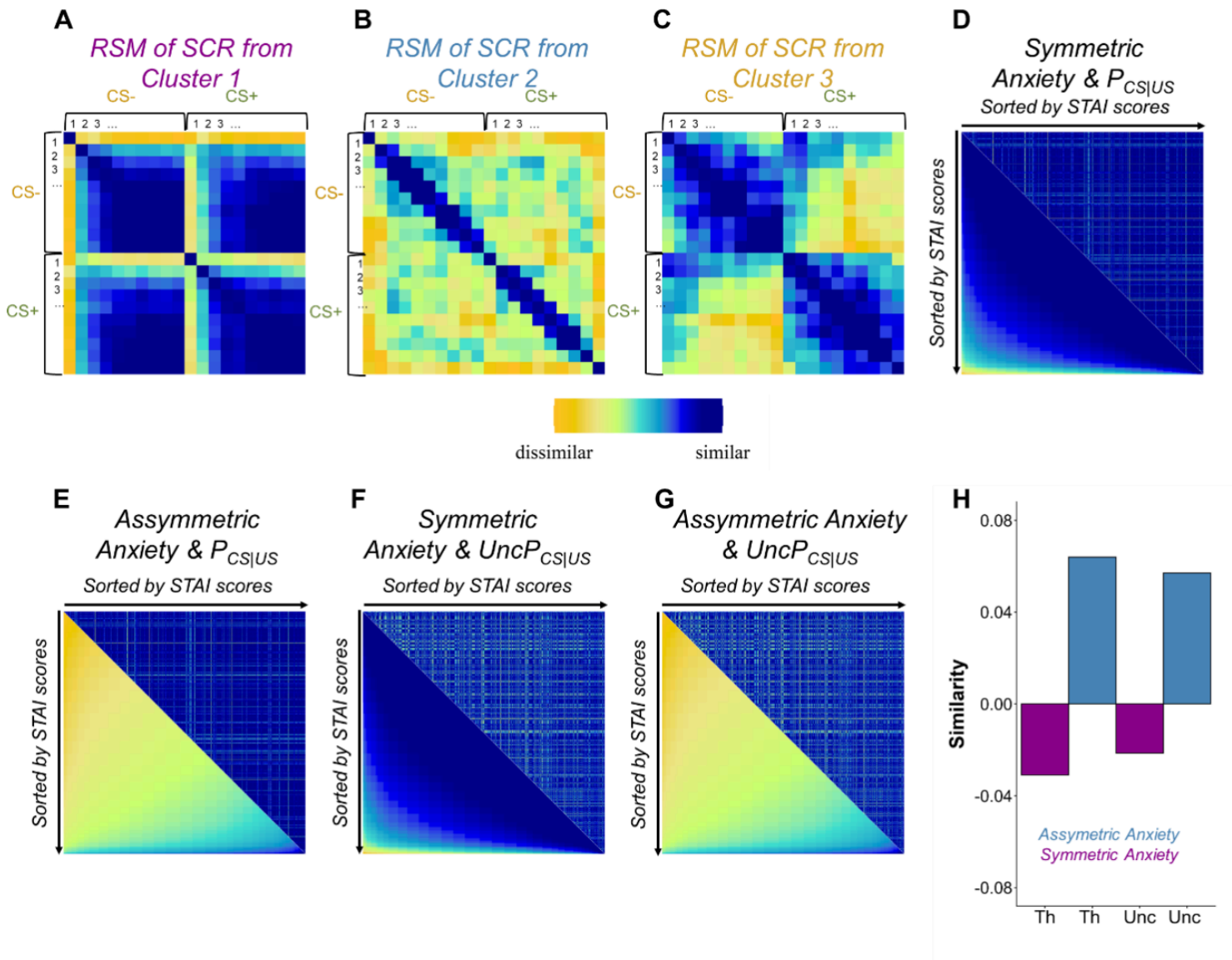


Figure 4

Anxiety-related differences in SCR-model coupling follow an asymmetric pattern. (A-C) Representational similarity matrices (RSMs) of (A) participants from Cluster 1, showing a strong correspondence with the uncertainty reduction model, (B) participants from Cluster 2, showing low correspondence with any of the models, and (C) participants from Cluster 3, showing a strong correspondence with the threat learning model. (D-G) Inter-subject

RSMs of the correspondence between SCR and threat learning (D, E; upper part of the diagonal), and uncertainty reduction models (F, G; upper part of the diagonal), as well as RSMs of symmetric and (D, F; lower part of the diagonal) asymmetric (E, G; lower part of the diagonal) models of anxiety. (H) Similarity indices between SCR-model correspondence and models of anxiety, showing evidence for a positive association with asymmetric, but not symmetric, levels of anxiety. Th= backward threat learning model; Unc= backward uncertainty reduction model.

Supplementary Files

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