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Diminished pain sensitivity mediates the relationship between psychopathic traits and reduced learning from pain

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Individuals with elevated psychopathic traits exhibit decision-making deficits linked to a failure to learn from negative outcomes. We investigated how reduced pain sensitivity affects reinforcement-based decision-making in individuals with varying levels of psychopathic traits, as measured by the Self-Report Psychopathy Scale-Short Form. Using computational modelling, we estimated the latent cognitive processes in a community non-offender sample (n = 111) that completed a task with choices leading to painful and non-painful outcomes. Higher psychopathic traits were associated with reduced pain sensitivity and disturbances in reinforcement learning from painful outcomes. In a Structural Equation Model, a superordinate psychopathy factor was associated with a faster return to original stimulus-outcome associations as pain tolerance increased. This provides evidence directly linking reduced pain sensitivity and learning from painful outcomes with elevated psychopathic traits. Our results offer insights into the computational mechanisms of maladaptive decision-making in psychopathy and antisocial behavior.

Healthy functioning in daily life requires us to constantly make decisions. In general, we tend to make choices that lead to favorable outcomes and cause a minimum amount of distress to ourselves and others. However, individuals with elevated psychopathic traits routinely make decisions that harm them and/or those around them, seemingly unable to learn sufficiently from past mistakes and adapt behavior accordingly[1–3]. Psychopathy is a personality construct typified by disturbances in the interpersonal (pathological lying and manipulativeness) and affective domains (shallow affect, lack of guilt), combined with antisociality (poor behavioral control, criminal versatility) and a tendency to lead an erratic lifestyle (impulsivity)[4]. These four domains are believed to reflect 4 facets embedded into a superordinate factor that captures the general construct of psychopathy[5,6]. While the prevalence and severity of psychopathic personality traits are highest among offender populations[7,8], research shows that psychopathy is a dimensional construct that can be assessed reliably in the general community[9–11].

Given the increased predisposition of those with elevated psychopathic traits to engage in aggressive, violent behavior[12,13], there has been great interest in understanding why they make such poor choices and how these tendencies are acquired. Reinforcement learning (RL) provides a framework that explains how people learn from reinforcers—such as rewards and punishments—and adapt their behavior to acquire as many rewards as possible while concurrently avoiding punishments[14]. This is essentially an optimization problem that relies on the ability to predict future outcomes. An effective reinforcement learner can maintain representations of stimulus–outcome (S–O) and action–outcome (A–O) associations, or contingencies, keeping track of which prior actions led to reward and which resulted in punishment. Deficits in the RL mechanisms would explain why individuals with elevated psychopathic traits cannot learn from their past mistakes (i.e., putting them at higher risk of offending and reoffending[15]).

In recent years, experimental research has focused on exploring which RL mechanisms may be impaired with higher psychopathic traits and how they might lead to poor decision-making[16–19]. In general, individuals with psychopathic traits show diminished value updating when learning from negative outcomes[20], and youths with disruptive behavior disorders

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demonstrate reduced use of expected value information within prefrontal regions during value-based learning[21]. Reduced sensitivity to punishment[22–24] could result in individuals experiencing negative outcomes as less aversive, and thus learning less from them[16,25,26]. At the same time, hypersensitivity to reward might contribute to an inability to inhibit reward-seeking behavior[19,27,28]. Such findings indicate that, at the behavioral level, both punishment hyposensitivity and reward hypersensitivity could explain the 'response perseveration' deficit found in psychopathy—the tendency to stick with a specific behavior even as it leads to increasingly more negative consequences[19,29,30]. However, incorporating such insights into neurobiological models that capture the mechanism behind altered RL and decision-making in relation to psychopathy has been a challenge.

Perhaps the most versatile of such models, the integrated emotion system (IES) model[31], proposes that the maladaptive reinforcement-based decision-making in psychopathy can be attributed to a failure to monitor changes in associations between events and outcomes. From a computational perspective, this is often referred to as volatility tracking [32–34]. Successful RL in volatile environments depends on how sensitive an individual is to reversals of contingencies (i.e., when a certain stimulus is no longer associated with a reward). The RL deficits in psychopathy are observed in the context of reversal learning[17,18], suggesting individuals with elevated psychopathic traits might be less sensitive to volatility. From a computational perspective, when a reversal occurs we experience greater uncertainty about the learned contingencies (i.e., our beliefs about the S–O association become less precise). Bayesian accounts of RL propose that the current level of uncertainty impacts the learning rate[32], and that reducing uncertainty is essential for successful learning. Consequently, research demonstrates that individuals with elevated psychopathic traits struggle with reducing their uncertainty when estimating the likelihood of changes occurring and the rate at which such changes occur[16,35]. Information about the volatility of the environment also needs to be incorporated relatively quickly so the agent can update their predictions about the new contingencies[33,34]. Failure to do so or slower updating would result in an individual sticking to their original beliefs. In the context of psychopathy, this represent a cognitive process driving the well-documented response perseveration deficit[19].

Increased uncertainty in predictions about reward contingencies, diminished reversal sensitivity, and response perseveration represent maladaptive processes that could explain why those with higher psychopathic traits struggle to learn effectively from reinforcers. In general, as one role of RL is to maximize reward while minimizing punishment, more salient reinforcers should have a more pronounced impact on the learning rate. A more salient punishment, such as pain, would lead to faster learning to motivate future actions that prevent harmful outcomes[36]. In fact, painful outcomes increase the motivational significance of errors to help avoid making the same errors in the future[37]. Additionally, pain can act as a primary teaching signal to speed up learning[38]. Within RL and decision-making paradigms, pain has also been found to impact the trade-off between exploiting known options or exploring new ones[39]. In the context of psychopathy, however, very little is known about the mechanisms of RL when choices can lead to painful outcomes. Psychopathic traits have been associated with higher pain tolerance and pain thresholds[40–42]. Therefore, we can expect that individuals with elevated psychopathic traits are less influenced by the prospect of pain and would show reduced learning from painful feedback. However, it is unclear which computational RL mechanisms would be affected by a reduced pain sensitivity with increasing levels of psychopathic traits.

To disentangle the different mechanisms underlying impaired RL with elevated psychopathic traits, we utilized a computational model, the hierarchical Gaussian filter (HGF[33,34]). The HGF provides a mathematical framework for learning about contingencies in a volatile environment and allows us to quantify the extent to which learning processes are affected. While standard reinforcement learning models such as the Rescorla-Wagner model assume that the individual's learning rate is fixed[43], the HGF model allows for the estimation of a dynamic learning rate. This makes the HGF particularly well-suited for modeling learning in volatile environments. The Bayesian inference process approximated by the HGF update equations[44,45] is also currently considered to be the optimal way to update beliefs in uncertain conditions[46]. The HGF allows for quantification of "belief resetting" (i.e., parameter φ), which determines the speed with which individuals return to their original beliefs about reward contingencies. This belief resetting essentially represents a cognitive process leading to the response perseveration deficit reported in psychopathy[19]. Additionally, we can quantify the sensitivity to reversals in contingencies (i.e., parameter ω)[18,31]. Using the HGF model, we can also estimate how precise individuals are in their initial predictions before learning occurs (i.e., the initial uncertainty about contingencies at the beginning of the task) similarly to previous work on psychopathy[16].

The overarching goal of the present study was to quantify the latent cognitive processes involved in learning from neutral vs. salient reinforcers to make simple choices in relation to levels of psychopathic traits. At the general level, we hypothesized that learning would differ depending on the type of reinforcers individuals learn from. To this end, we contrasted the cognitive computational parameters underlying learning in one condition where outcomes were monetary gains and losses (i.e., non-painful punishments) with the parameters in a second condition where outcomes were personalized rewards and painful punishments. Given the evidence for learning impairments in psychopathy, we also hypothesized that psychopathic traits would be associated with deficits in the RL processes. More specifically, based on findings of reduced pain sensitivity with increasing psychopathic traits and the proposed salience of pain as a reinforcer, we expected more pronounced RL deficits in the pain condition. To test this second hypothesis, we additionally incorporated an exploration readiness parameter β that represents the tendency to explore new options versus choosing in a more deterministic fashion. Since this tendency is robustly influenced by pain[39], we expected the exploration readiness in the pain condition would also be linked to the level of psychopathic traits. Finally, we investigated whether aberrant RL learning is associated with elevated levels of psychopathic traits through the mediating role of experimental and self-reported pain sensitivity. If so, it would point to a mechanism through which the pain processing impairs decision-making in psychopathy.

Methods

We recruited 111 healthy participants (age = 29.67 ± 9.32 [mean ± standard deviation], 88% right-handed), including women ($n = 61$), men ($n = 47$), and non-binary ($n = 2$), with no history of psychiatric illness, neurological or chronic pain conditions via social media and the electronic Radboud research participation system. The data of 5 participants were excluded due to equipment failure (2 cases), random responding (1 case), and outliers in the estimated computational parameters (3 cases). The study was approved by the Ethics Committee of the Faculty of Social Sciences, Radboud University (code ECSW-2020-120), and all participants gave informed consent. The study was not preregistered. Self-report data on participant gender was collected, but no data on race and/or ethnicity was obtained. Participants provided written informed consent prior to taking part in the study. Compensation for participation was €10 per hour, and participants received a small bonus, contingent on performance (~€6 euro and a personalized reward, following the procedure described below). The sample size was determined based on previous work with similar paradigms (effect size = 0.30, power = 0.90)[16,47]. Additionally, we employed a Bayesian SEM as it can offer increased power compared to traditional SEMs[47], particularly in relatively small samples (i.e., at $n > 50$[48]).

Questionnaires

Psychopathic traits were assessed with the self-report psychopathy-short form (SRP-SF[6,49]), a questionnaire with 29 items that yields a total psychopathy score and facet scores for the Interpersonal, Affective, Lifestyle, and Antisocial traits. Self-reported pain sensitivity was measured with the pain sensitivity questionnaire (PSQ), a short self-rating instrument that evaluates the individual's perception to a range of physical stimuli that can be encountered in daily life[50]. Higher scores on the PSQ denote a higher self-reported sensitivity to various types of pain.

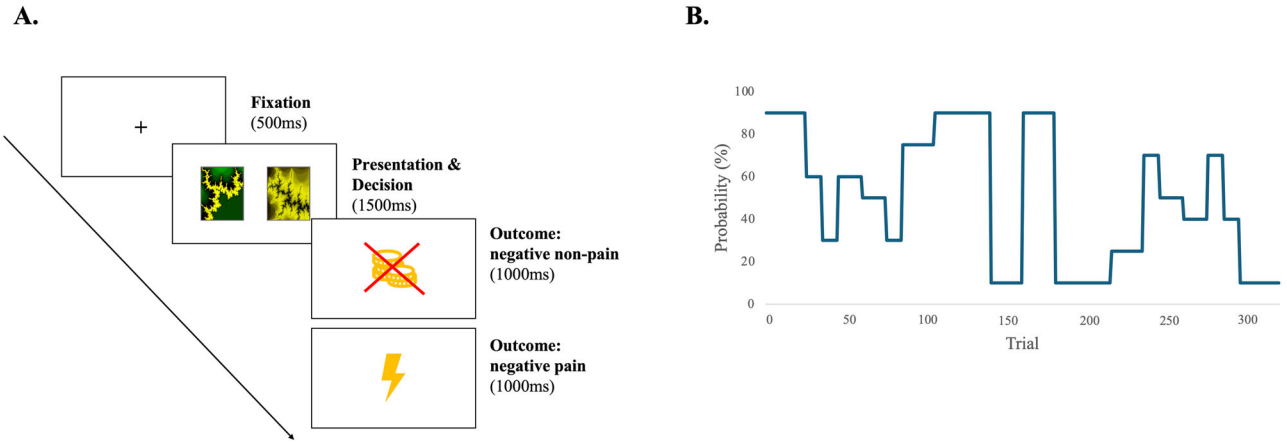

Fig. 1 | Reinforcement learning task and the employed reinforcement schedule **A** Task design: the stimuli were presented on screen for a maximum of 1.5 s. The feedback was shown for 1 s. **B** Reinforcement Schedule: The blue line depicts the reward likelihood of the green fractal. The first 160 trials were part of the non-pain condition (coin/crossed-out coin), whereas trials 161–320 were from the pain condition (naturalistic reward/shock).

Experimental pain assessment

Additionally, we collected sensory detection thresholds, pain thresholds, and pain tolerance (defined as 7 on a 10-point numerical rating scale [NRS]) via a stepwise quantitative sensory testing (QST) procedure using the Digitimer DS7A, a Food and Drug Administration (FDA) approved device for experimental and clinical settings[51]. The pain assessment of participants was sex-balanced and conducted by two trained researchers (a man and a woman). The electrode was placed on the inside of the non-dominant hand, halfway between the wrist and the elbow. The same location was used for the delivery of the painful stimulus during the experimental task.

To obtain sensation detection and pain thresholds, we delivered electrical shocks in increments of 0.1 mA. The participant was asked to first provide a verbal report if they experience "any sensation, even if it's a small sensation" and the intensity at which such a report was provided was recorded as the sensation detection threshold. Upon verbal confirmation, the intensity was reduced by one step until a "no" report was obtained, then increased incrementally until another "yes" was obtained. The average intensity across the three "yes" points was used as the threshold value. We used the same procedure to obtain pain thresholds, only altering the instructions slightly ("any pricking, burning, stinging, aching or painful sensation").

To measure pain tolerance, we increased the electrical current in steps of 0.2 mA, starting at 0 mA. Participants were instructed to verbally rate how painful the stimulation felt using a scale from 0 to 10 (0 = "a sensation, but not painful", 10 = "the most intense pain you can imagine"). The intensity of the current at a 7 NRS rating was recorded as the subjective pain tolerance, and no further increases were implemented. In cases when the tolerance was not reached due to the machine's limitation (maximum intensity 9.99 mA), the maximum intensity was used instead. For 13 out of the 106 participants, the tolerance level was not reached due to the described hardware limitations. In light of this, we also collected a measure of "pain tolerance intensity", the subjective NRS rating given at the maximum delivered stimulation.

Instructions for the pain assessment were delivered in English, but in cases where the participant's command of English was limited, the descriptions were supplemented with Dutch words: "pricking (prikkend), burning (brandend), stinging (stekend), aching (zeurend), or painful (pijnlijk) sensation".

Reinforcement learning task

A reinforcement-based probabilistic decision-making task (Fig. 1) was adapted from prior research to study simple binary choices[52]. On each trial, participants had to choose one of two stimuli (a green fractal card or a yellow fractal card). The stimuli were kept the same throughout the task, but their position was counter-balanced. The reinforcement schedule was the same for all participants (Fig. 1B), with the reward probabilities of the two fractals adding to 1 (i.e., $p(\text{yellow}) = 1 - p(\text{green})$). The task comprised two conditions: a non-pain condition where outcomes were monetary gains and losses (€0.10 and −€0.10, respectively) and a pain condition where outcomes were personalized rewards or valence-matched electric shocks at the tolerance level[53]. The order of conditions was kept the same for all participants, with the non-pain condition preceding the pain condition as it represented standard RL and can, therefore, be used as a baseline.

To determine the personalized reward, we asked participants to rate a list of potential participation rewards from 1 (=least preferred) to 10 (=most preferred). This was done prior to the reinforcement task or the experimental pain assessment, immediately after participants provided informed consent. Participants were informed that the item they rated as 7 on the 10-point NRS scale would be used as a reward on the task and that the amount of rewards (both monetary and personalized) would depend on their performance. The reward list was available in both English and Dutch. The monetary value and the size of all rewards on the list were roughly the same (~€2 each). The full list of available personalized rewards can be found in the Supplementary Fig. S1.

Computational model

We estimated latent cognitive computations with the HGF model[33,34]. Based on findings of response perseveration in psychopathy[19,29,30], we selected a model with a mean-reverting RL process which assumes that participants' beliefs about outcome likelihoods reset toward their initial expectations (which represents a potential process leading to a perseveration tendency). The model estimates individual differences in prior uncertainty about the reward contingencies ($\sigma^{(0)}$), sensitivity to reversals in the contingencies (ω), and belief resetting speed (φ).

The 3-level HGF model assumes that participants infer the true states in the world x_1–x_3 (corresponding to outcome, likelihood of change of outcome contingencies, and rate of change of outcome contingencies, respectively)[33,34]. As these true states are hidden, participants generate beliefs about them. On level 1 (x_1), beliefs μ_1 correspond to the outcome (win/loss). On level 2 (x_2), this information is used to infer the trial-by-trial beliefs about likelihood of change in reward contingencies (environmental volatility) μ_2. Finally, individuals also generate beliefs μ_3 about the rate/speed of change in outcome contingencies x_3. In the current model implementation, we only estimated beliefs at the second level μ_2, which depend on the prior uncertainty $\sigma^{(0)}$, the reversal sensitivity ω and the belief resetting speed φ. This perceptual model is combined with a softmax response model quantifying (inverse) exploration readiness: how closely actions match beliefs. Higher (more positive) values of ω denote reduced sensitivity to reversals, higher values of $\sigma^{(0)}$ more uncertainty about the likelihood of contingency changes,

higher φ—a greater tendency to stick to the prior beliefs $\mu_2^{(0)}$, and lower β—a stronger tendency to explore new options. Note that all four computational parameters have been studied and validated before ($\sigma(0)$ and φ in[46], ω in[35]; φ and β in[54]). The full update equations can be found in the Supplement.

Statistical analyses

Behavioral data analysis. Accuracy (percentage of choices of the stimulus with the higher reward probability), win-stay, and lose-switch rates were compared with Bayesian paired samples *t*-tests in Jamovi[55], with the Bayes Factor (BF) computed with default flat priors for the alternative hypothesis reported. BF > 100 represents decisive evidence, BF between 10 and 30—strong evidence, and BF between 3 and 10—substantial evidence for H_i[56]. 2We first assessed whether behavioral performance measures were associated with psychopathic traits, using a combination of frequentist non-parametric Spearman's rank correlations in R[55] and zero-order Bayesian correlations with default priors in Mplus[57]. Non-parametric Spearman's rank correlations were used due to the non-normal distribution of the psychopathic traits' data. However, since the task performance measures represent aggregates of behavior (mean accuracy, win-stay, lose-shift), we also conducted a post-hoc analysis on the trial-wise choices made by participants. A Bayesian Generalized Linear Mixed Model was fit on the behavioral choice using the bmrs package in R[58–61]. We regressed the choice to stay or switch from the previously chosen option (0 = stay, 1 = switch) on the four psychopathic traits, controlling for trial number, condition, and the outcome of the previous trial. Interactions between the psychopathic traits, conditions, and outcomes were also included. The random effects structure contained a by-subject intercept and slopes for trial, condition, and outcome. This model was fit with a Bernoulli distribution and default priors. Four Markov Chains were used, with 3000 iterations (the first half discarded as burn-in) each. Convergence was assessed visually via trace plots and by looking at the R-hat values of the posterior estimates (R-hat values should be <1.05). Model fit was determined based on the predictive posterior plots.

Pain data analysis. We assessed whether the psychopathic traits were associated with electrical pain threshold, tolerance, and tolerance intensity, and PSQ scores using non-parametric Spearman's rank correlations in R[62] and Bayesian zero-order correlations with default priors in Mplus[57]. We additionally looked at the correlations between the psychopathy subscale and total scores with our measures of experimental pain sensitivity (electrical pain threshold, tolerance, tolerance intensity) and self-reported pain sensitivity as measured with the PSQ. Due to the highly skewed nature of the pain tolerance intensity data, we conducted Kendall tau-b Bayesian correlations using default priors in Jamovi[63] in place of the zero-order Bayesian correlations in Mplus. The dual-analysis approach was undertaken to ensure the robustness of the results. For the Bayesian correlations in Mplus, we estimated the coefficients and corresponding 95% credibility intervals (CIs) with a Bayesian estimator (PX1) based on Markov Chain Monte Carlo (MCMC) sampling with four Markov chains and 75,000 iterations (first half discarded as burn-in training trials) using default priors. Model fit was determined based on the posterior predictive *p*-value (PPP-value), which should approach 0.5, and the posterior predictive check using χ^2 testing (the 95% CIs of the χ^2 test should include 0)[16,57,64]. The 95% CI of the non-parametric frequentist correlations were estimated using the bootstrap method with 9999 resamples. A result was considered statistically significant only when *both* the Bayesian and frequentist approaches yielded converging results[35,65].

Computational model selection. We compared the three-level mean-reverting HGF model described above (i.e., the main model) with reduced HGF models (M1–M4) and a Rescorla-Wagner model (RW). M1 assumes individual differences in learning are driven *only* by reversal sensitivity ω and uncertainty in original beliefs $\sigma^{(0)}$; M2 estimates individual differences in learning based on two parameters, reversal sensitivity ω and belief resetting φ; M3 represents the main model and assumes individuals differ in all three parameters (reversal sensitivity ω, uncertainty $\sigma^{(0)}$ and belief resetting φ). Additionally, as the pain condition always followed the non-pain one, an additional model (M4) was built for the pain trials only. M4 is identical to M3, but the modal of the subjects' posterior estimates in the non-pain condition was used as the new prior for the three parameters. This allowed us to account for the carry-over of beliefs across the two conditions. Finally, we also fitted an RW model where learning is governed by a single learning rate parameter α. The models were compared using Bayesian model selection (BMS)[66], as implemented in the Statistical Parametric Mapping 12 toolbox (https://www.fil.ion.ucl.ac.uk/spm/), using the optimal Bayes observer posteriors as priors. All models were fitted separately for the pain and non-pain conditions using the HGF toolbox (https://tnu.ethz.ch/tapas) and the optimal Bayes parameters as priors (except for M4). The full procedure is detailed in the Supplement, with the prior means and variances of the competing models in Supplement Tables S1 and S2.

After the winning model was selected with the BMS procedure, we performed 100 simulations per participant using the individual parameter estimates from the model. We correlated the simulated and original choices and visually inspected whether the true choice trajectories resembled the choice trajectories simulated with the model. Next, we fit the same model to the simulated data to assess parameter recovery by evaluating the correlations between the original (true) and recovered parameters. Finally, to determine if the model was also a realistic model for the data, we also used joint Bayesian zero-order and frequentist non-parametric correlations to evaluate the relationships between the estimated parameters and task performance (accuracy, win-switch, lose-stay).

Analysis of computational modeling results. After extracting the estimated computational parameters for both conditions, we used Bayesian t-tests in Jamovi with default priors to assess for differences in the learning processes between conditions to answer the first hypothesis (regardless of psychopathic traits). Following that, we evaluated the zero-order Bayesian and frequentist non-parametric Spearman's rank correlations between the learning parameters and psychopathic traits using the approach detailed above (see "Pain data analysis"). We then fit a series of structural equation models (SEMs) in Mplus to evaluate the paths leading from the computational parameters to the psychopathic traits, accounting for the role of pain. To test whether psychopathy would be associated with reduced pain sensitivity[40–42], lower reversal sensitivity[35], higher prior uncertainty in beliefs (similarly to findings of increased task-related belief uncertainty[16]), or higher belief resetting, we built two sets of SEMs: one with the individual psychopathy factors, and one with a latent psychopathy factor, onto which the four traits were loaded[35,67]. This allowed us to assess the paths leading from the learning parameters to both the individual psychopathic traits and psychopathy as a construct (accounting for the shared variance between the traits). The SEMs were fit for the pain and non-pain parameters separately to test for the effect of the condition. For the pain SEM model, we also regressed a latent pain factor on the learning parameters and used it as a predictor of psychopathy (or psychopathic traits). The latent pain factor comprised the experimental pain tolerance and the self-reported pain sensitivity (i.e., PSQ scores). We also tested a model where the pain measurements (electrical pain threshold, electrical pain tolerance, and self-reported pain sensitivity) were loaded onto the latent factor, producing almost identical results but with a worse model fit. Due to the highly skewed nature of the pain tolerance intensity and concerns over possible ceiling effects, it was not included in the SEM analysis. For all SEMs, we tested both indirect and direct paths.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Results

Behavioral task performance results

Participants were able to learn the reward contingencies well, with no credible evidence for differences in accuracy between the non-pain ($m = 70.2 \pm 10.0\%$) and pain conditions ($m = 68.8 \pm 11.5\%$, BF = 0.33). Win-stay rates did not differ significantly between the non-pain ($m = 51.7 \pm 11.0\%$) and pain conditions ($m = 52.4 \pm 10.6\%$, BF = 0.15). There was also no credible evidence for the difference between lose-shift rates in the non-pain ($m = 11.1 \pm 7.1\%$) and the pain condition ($m = 10.4 \pm 6.7\%$, BF = 0.30) (Fig. 2).

No significant correlations were found between the task performance measures (accuracy, win-stay, lose-shift) and the four psychopathic traits (see Supplementary Table S4). The Bayesian generalized linear mixed model (GLMM), however, revealed an interaction between affective traits and the outcome on previous trials in predicting switching behavior. The interaction was evaluated with a post-hoc analysis using the emmeans package in R[49]. More specifically, elevated Affective traits were associated with a greater tendency to switch after a previous win, $b = 0.39$, 95% CI [0.10–0.67] when controlling for the effect of the outcome on the previous trial, condition, and the trial number. The full GLMM results can be found in the Supplement (Table S6, Fig. S2).

Pain data results

Electrical pain threshold, electrical pain tolerance, and self-reported pain sensitivity (i.e., as measured with the PSQ) scores showed moderate-to-high correlations with each other, but the electrical pain tolerance intensity was only associated with electrical pain threshold and electrical pain tolerance measures (Table 1).

While all four psychopathic traits correlated positively with pain tolerance in the Bayesian analyses, only the association between the Lifestyle facet and experimental pain tolerance was statistically significant in the non-parametric analyses ($r_{Bayes} = 0.27$, 95% CI [0.18–0.34], $r_{np} = 0.25$, 95% CI [0.05–0.42]). The Lifestyle facet also correlated with reduced pain tolerance intensity across both statistical families ($r_{Bayes} = -0.20$, 95% CI [−0.07 to −0.32], $r_{np} = -0.24$, 95% CI [−0.01 to −0.43]), as did the Antisocial facet ($r_{Bayes} = -0.24$, 95% CI [−0.11 to −0.37], $r_{np} = -0.28$, 95% CI [−0.08 to −0.45]), and the Affective facet ($r_{Bayes} = -0.18$, 95% CI [−0.05 to −0.30], $r_{np} = -0.22$, 95% CI [−0.01 to −0.40]), The full correlations are reported in Supplementary Table S5.

Computational model selection

The BMS procedure identified the three-level mean-reverting HGF model as the best fitting model to the data (Fig. 3A) (M3 for the non-pain data and M4 for the pain data). The pattern of simulated choices closely tracked that of the real participants' behavior (Fig. 3B). The parameters of the winning model were also well recovered, with correlations ranging from 0.84 for $\sigma_2^{(0)}$ on the pain trials to 0.97 for φ on the non-pain trials (Fig. 3C). The Bayesian zero-order and non-parametric correlations also demonstrated sensible associations between the estimated parameters and the behavior (accuracy, win-switch, lose-shift) that elicited them, supporting the ecological validity of the computational model (Supplementary Table S3).

Computational modeling results

The Bayesian *t*-tests showed credible evidence for differences in the learning parameters across conditions. Individuals displayed higher uncertainty in their original beliefs about contingencies in pain ($m = 0.27 \pm 0.13$) compared to the non-pain condition ($m = 0.21 \pm 0.12$, BF = 17.0); higher belief resetting in pain ($m = 0.32 \pm 0.17$) than the non-pain condition ($m = 0.25 \pm 0.17$, BF > 100); decreased sensitivity to reversals (represented by more negative ω values) in pain ($m = -6.67 \pm 1.07$) compared to the non-pain condition ($m = -6.08 \pm 1.31$, BF > 100); and reduced exploration readiness (representing more stochastic responding) in pain ($m = 54.3 \pm 32.09$) relative to the non-pain condition ($m = 33.3 \pm 12.25$, BF > 100) (Fig. 4).

The correlations that retained significance across both analyses included the associations between Interpersonal traits and the original beliefs' uncertainty ($\sigma^{(0)}$) in the pain condition ($r_{Bayes} = 0.22$, 95% CI [0.02–0.40], $r_{np} = 0.27$, 95% CI [0.08–0.43]), and Affective traits and original beliefs' uncertainty ($\sigma^{(0)}$) in the pain condition ($r_{Bayes} = 0.21$, 95% CI [0.02–0.39], $r_{np} = 0.26$, 95% CI [0.05–0.44]).

The SEMs where the learning parameters in the non-pain condition were regressed onto the four psychopathic traits demonstrated an excellent model fit, PPP = 0.43, χ^2 95% CI [−20.83 to 25.5], but there was no evidence for unique associations between the psychopathic traits and the learning parameters. Similar results were obtained when the facet scores were loaded on a latent psychopathy factor (PPP = 0.53, χ^2 95% CI −20.91 to 17.98]).

For the pain condition, the model assessing the relationships between the learning parameters, the latent pain factor, and individual psychopathic traits also yielded an excellent fit (PPP = 0.51, χ^2 95% CI [−26.28 to 24.43]) (see Fig. 5). Lower pain sensitivity as measured by the latent pain factor,

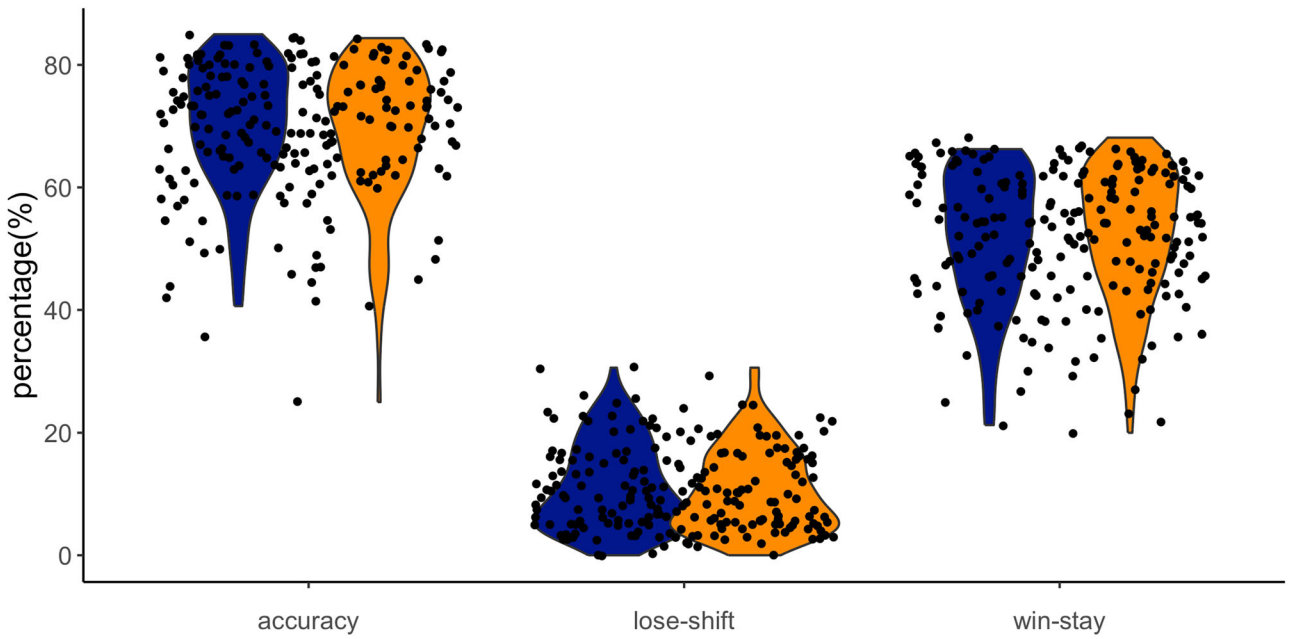

Fig. 2 | Task performance comparison across the non-pain (blue) and pain (orange) condition. Caption: Model-free task performance (accuracy, lose-shift, win-stay rates) across the two conditions (blue = non-pain, orange = pain), $n = 106$ participants.

Table 1 | Correlations between the pain measures

	Electrical pain tolerance	Self-reported pain sensitivity (PSQ score)	Electrical pain tolerance intensity (NRS score)
Electrical pain threshold	r_{Bayes} = **0.35*** [0.17–0.51] r_{np} = **0.33*** [0.14–0.49]	r_{Bayes} = **−0.21***, [−0.35 to −0.06] r_{np} = **−0.28***, [−0.45 to −0.08]	r_{Bayes} = **−0.21***, [−0.39 to −0.02] r_{np} = **−0.28*** [-.44 to −0.09]
Electrical pain tolerance		r_{Bayes} = **−0.31*** [−0.32 to −0.21] r_{np} = **−0.49*** [−0.65 to −0.30]	r_{Bayes} = **−0.49*** [−0.63 to −0.33] r_{np} = **−0.54*** [−0.66 to −0.42]
Self-reported pain sensitivity (PSQ score)			r_{Bayes} = 0.17, [−0.03 to 0.35] r_{np} = 0.19, [−0.05 to 0.38]

Note. Significance is denoted based on the 95% CIs which must not contain 0, and denoted with an asterisk and in bold. Abbreviations: *PSQ* pain sensitivity questionnaire, r_{Bayes} correlation coefficient for the zero-order Bayesian correlations, r_{np} correlation coefficient for the non-parametric Spearman's rank correlations (with bootstrapped 95% confidence intervals using 9999 resamples).

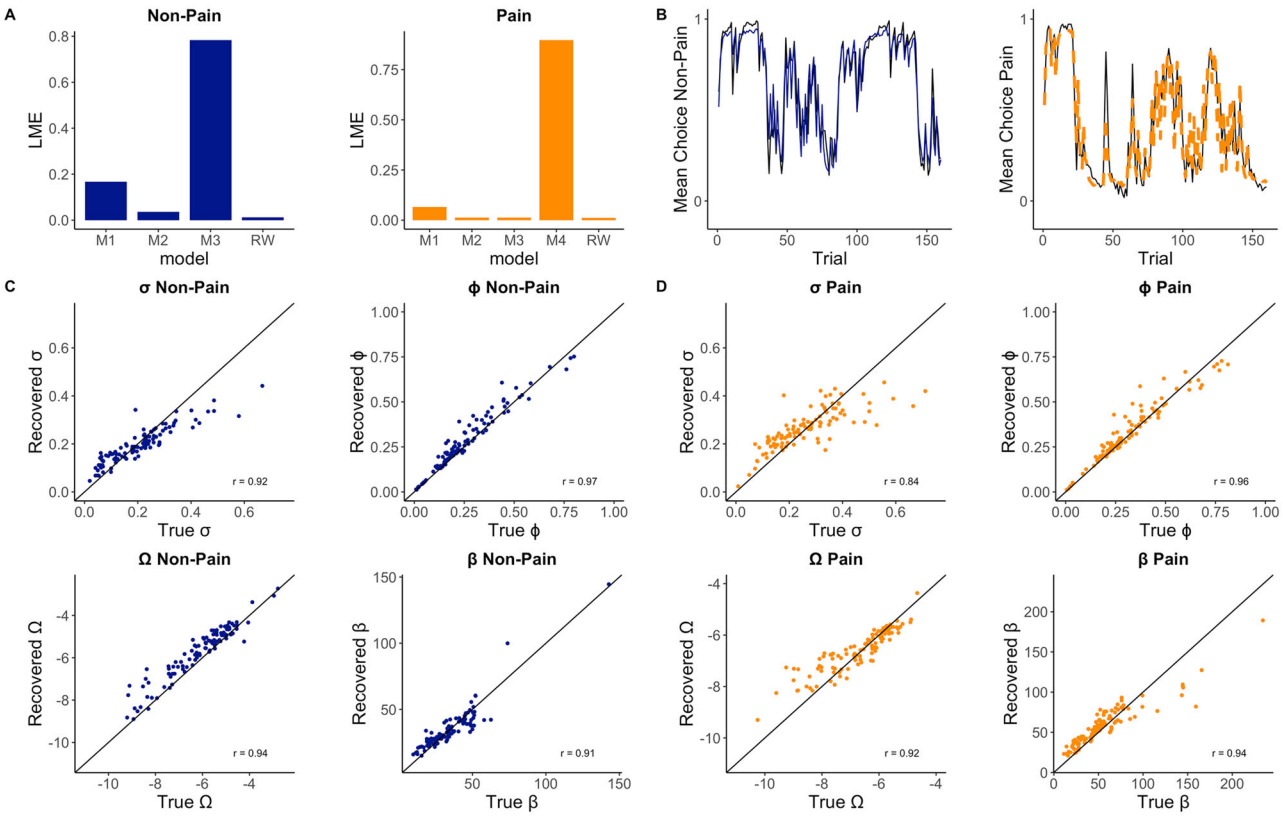

Fig. 3 | Model comparison and recovery of the winning model (mean-reverting HGF with uncertainty in original beliefs, reversal sensitivity, and belief resetting). A Bayesian model comparison for the two conditions. The winning perceptual M3 model for the non-pain data includes reversal sensitivity ω, uncertainty in original beliefs $\sigma(0)$, and belief resetting φ. The winning perceptual M4 model for the pain condition includes the same parameters but with their priors informed by the posterior estimates from the non-pain condition. **B** Choice trajectories: the original choices (averaged across participants; black) are closely tracked by the simulated choices (averaged across simulations and across participants) in the non-pain (left; blue) and pain (right; orange and dashed) conditions. **C** Correlations between real and recovered parameters in the non-pain condition: uncertainty in original beliefs $\sigma(0)$ ($r = 0.92$, BF > 100), belief resetting φ ($r = 0.97$, BF > 100), reversal sensitivity ω ($r = 0.94$, BF > 100), and exploration readiness β ($r = 0.91$, BF > 100). **D** Correlation between real and recovered parameters in the pain condition: uncertainty in original beliefs $\sigma(0)$ ($r = 0.84$, BF > 100), belief resetting φ ($r = 0.96$, BF > 100), reversal sensitivity ω ($r = 0.92$, BF > 100), and exploration readiness β ($r = 0.94$, BF > 100). $N = 103$ participants.

predicted higher latent psychopathy scores, $\beta = 0.31$, 95% [0.06–0.58]. Additionally, the path from belief resetting φ to the latent pain factor was also significant, $\beta = 0.27$, 95% [0.05–0.48], suggesting an association between lower pain sensitivity and faster belief resetting. The indirect path from the learning parameters to the latent psychopathy factor through pain was also significant, $\beta = 0.08$, 95% [0.004–0.22]. The path model where the individual psychopathic traits were regressed on the learning parameters and the latent pain factor did not converge.

Discussion

The overarching goal of the present study was to investigate the relationships between psychopathic traits and the latent cognitive processes involved in RL in a non-offender sample. First, we found that the latent parameters describing learning in pain and non-pain condition differed significantly, supporting our hypothesis that learning differs depending on the type of reinforcer individuals learn from. Concerning the second research aim, we uncovered impairments in the latent cognitive processes subserving binary decision-making. In particular, the superordinate psychopathy factor was associated with a tendency to disregard evidence and stick to initial (i.e., pre-learning) expectations in the pain condition. Third, this maladaptive tendency was specifically mediated by an insensitivity to pain at higher levels of psychopathic traits. While not explicitly part of our a priori hypotheses, additional analyses also indicated that higher Affective traits predicted a greater tendency to switch

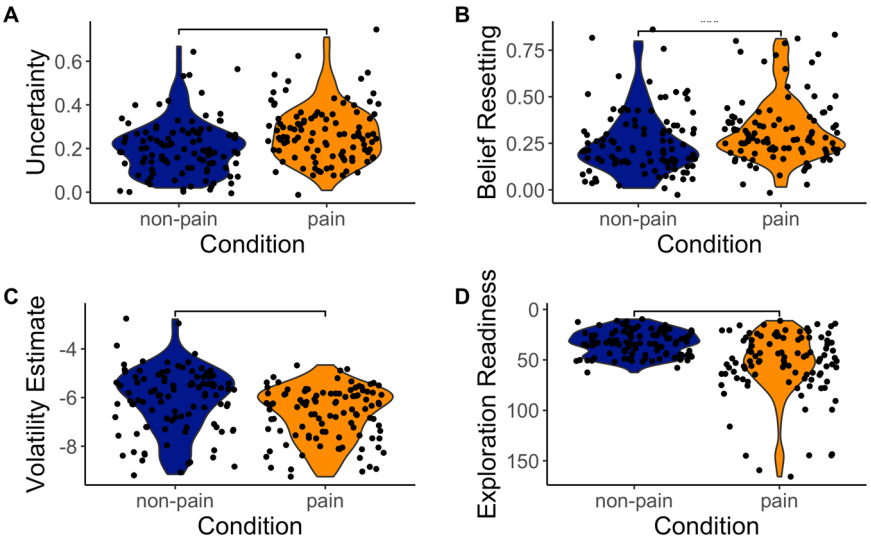

Fig. 4 | Comparison of estimated parameters across non-pain (blue) and pain (orange) trials. **A** Prior uncertainty in beliefs $\sigma(0)$ in the pain vs. non-pain condition. **B** Belief resetting φ across conditions. **C** Reversal sensitivity ω (more negative values denote reduced sensitivity) across conditions. **D** Exploration readiness β (higher values denote decreased exploration readiness) across conditions. Significance level denoted by an asterisk (***BF > 100), $n = 103$ participants.

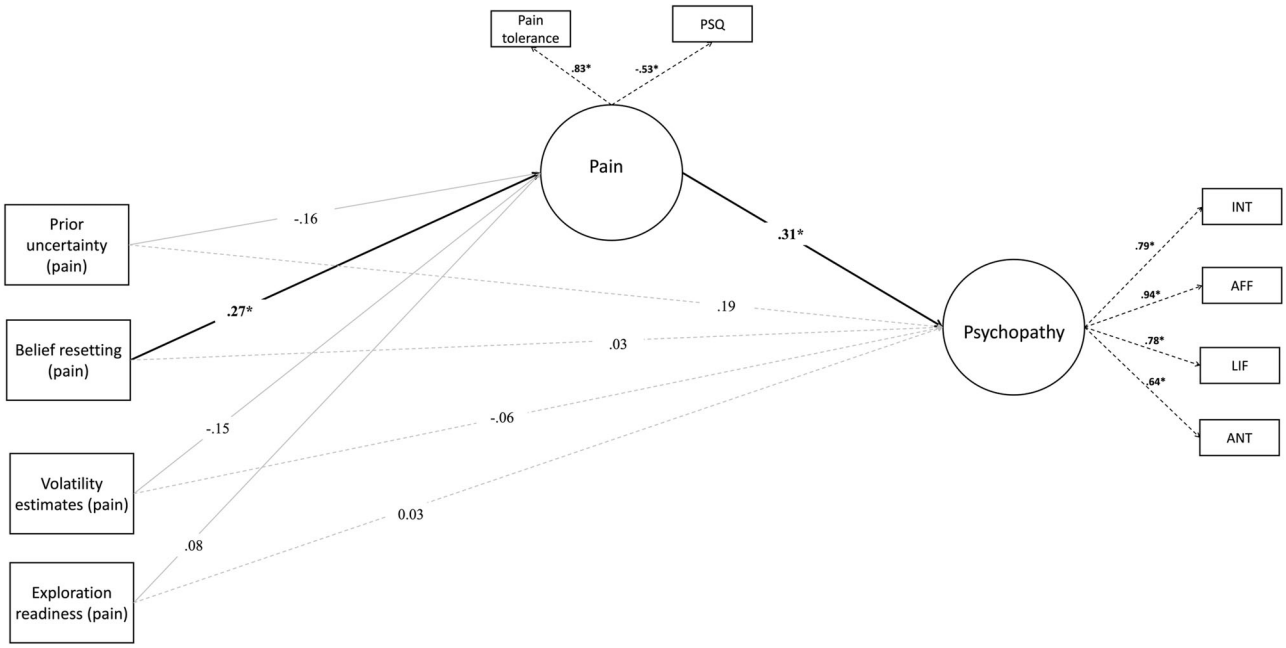

Fig. 5 | Structural Equation Model depicting the linear relationships between the latent psychopathy factor and the learning parameters via pain sensitivity. Solid lines denote the regression coefficients, while factor loadings are represented by a black dashed line. The direct path c' is denoted by a gray dashed line. Bold numbers with asterisks mark the significant associations between variables (95% Bayesian Credible Interval not including 0), $n = 103$ participants. INT interpersonal traits, AFF affective traits, LIF lifestyle traits, ANT antisocial traits, PSQ pain sensitivity questionnaire

after a winning choice regardless of the type of reinforcer (personalized or naturalistic reward).

The computational modeling approach we undertook revealed that the model best explaining binary decision-making accounted for individual differences in the uncertainty individuals have about the original outcome likelihoods, their sensitivity to reversals in the outcome likelihoods, and a belief resetting process which quantified how quickly they revert to their original beliefs. Partially in line with our predictions, we also found that psychopathic traits were associated with aberrations in the learning mechanisms only in the pain condition. More specifically, elevated Interpersonal and Affective traits correlated with increased uncertainty in original beliefs about outcome likelihoods in the pain condition. We could not find support for the hypothesis that exploration readiness in the pain condition would be associated with psychopathic traits. Crucially, however, we demonstrated that reduced pain sensitivity mediated the relationship between the superordinate psychopathy factor and increased belief resetting, representing a return to original beliefs about outcome contingencies in the pain condition.

Our findings of reduced pain sensitivity with elevated psychopathic traits support prior findings of higher pressure pain thresholds[68] and tolerance[40,42,69], as well as reduced fear of pain[40,70]. In the present study, the Bayesian zero-order correlations demonstrated associations between pain threshold, tolerance, and tolerance intensity, and virtually all four psychopathy facets. However, across the two statistical families we employed, only the correlations between the Lifestyle traits and pain tolerance, as well as the correlations between the pain tolerance intensity and Affective, Lifestyle, and Antisocial traits, remained significant. Nonetheless, as most of the research on the topic has been conducted in the pressure pain domain, our results also demonstrate an association between higher psychopathic traits and reduced electrical pain sensitivity.

A reduced pain sensitivity could lead to potentially dangerous situations being perceived as less threatening[71], likely resulting in more risky behavior. Individuals with higher Meanness psychopathic traits (such as irresponsibility and callousness) rate imagined scenes of self-inflicted harm as less painful, suggesting they underestimate the danger associated with the depicted injury[69]. Similarly, the Lifestyle facet, which covers excessive engagement in dangerous behaviors, negatively predicts the level of distress experienced at the prospect of pain[64]. Traits related to low threat sensitivity have been linked to both higher pain tolerance[42] and lower reported fear of pain, pain anxiety, and pain catastrophizing[40,70]. Thus, pain insensitivity with increasing levels of psychopathy might influence decision-making when faced with potentially painful outcomes.

In fact, we found impairments in RL with elevated psychopathic traits, specifically in the pain but not in the non-pain condition. Correlational results, for instance, showed that stronger Interpersonal and Affective traits correlated with a higher uncertainty about outcome likelihoods in the pain condition. The free-energy principle proposes that reducing cognitive uncertainty is essential for learning[72,73], therefore, larger initial uncertainty would lead to less successful RL. Behaviorally, more uncertain beliefs at the beginning of the task (i.e., the initial beliefs) were correlated with lower win-stay rates, representing a tendency to abandon winning choices. The higher uncertainty might bias the initial beliefs (and their updating during learning) and make ambiguous situations or outcomes be interpreted in a more pessimistic fashion, leading to less-than-optimal decision-making.

While this is the first study to demonstrate an increase in uncertainty about *initial* beliefs with higher psychopathic traits, previous studies have found increasingly uncertain representations about contingency changes with higher psychopathic[35] and higher callous-unemotional traits (which map onto the affective facet in adult populations[48])[16]. It should be noted that, while Interpersonal and Affective facets were associated with higher original belief uncertainty across conditions, only the correlations for the pain condition retained significance across the two statistical families we used. One explanation could be that, as the pain condition always followed the non-pain one, individuals with higher Interpersonal or Affective traits tend to perceive their own learning as less effective as the task progresses even though that was not reflected in their performance (i.e., neither facet was associated with reduced accuracy). Alternatively, they might exhibit a more profound impairment of the cognitive mechanism forming event-outcome representations when learning from painful outcomes. Since correlation analysis is limited in terms of the explanations it can provide, more complex relationships can be modeled with SEMs.

The SEM findings indeed revealed impairments in the RL from pain but in relation to increased belief resetting. The increased tendency to return to initial beliefs about reward contingencies was positively associated with a latent psychopathy factor. Importantly, the relationship was mediated by reduced pain sensitivity. In practice, the increased belief resetting represents a perseveration strategy where incoming trial-by-trial evidence is disregarded in favor of the initial beliefs. In the current computational framework, this effectively represents a return to maximum uncertainty about the reward contingencies. These observed alterations in reversal learning echo previous work on deficient threat and pain conditioning in psychopathy[16,74]. More specifically, the faster belief resetting represents a maladaptive perseveration strategy, well-documented in relation to elevated psychopathic traits[19,29,30,75]. This builds upon prior work that suggests psychopathy is associated with a general deficit in adapting behavior as event-outcome associations change[17,18,76–78]. Thus, beyond confirming the established deficits in reversal learning, our results specify the computational processes behind the reversal learning impairments in individuals with stronger psychopathic traits. They also highlight the role of different reinforcers in learning[54] and indicate a mechanism through which pain insensitivity may disrupt RL.

We should note that we only observed alterations in the pain condition. One explanation could be that learning from pain fundamentally differs from learning from non-painful outcomes. In general, painful outcomes result in greater amygdala activation[79] as well as different event-related potentials (ERPs) compared to monetary losses or other non-painful outcomes[80]. ERPs to painful feedback also peak earlier, suggesting faster processing, possibly due to the higher salience of the naturalistic punishment[80]. However, combined, our results point to an important implication: that painful punishments might not be salient enough for individuals with psychopathic traits to learn from. In general, pain sensitivity comprises two components: the intensity of pain and its unpleasantness. While the intensity is thought to reflect the sensory-discriminative aspect of pain, pain's unpleasantness is related to aspects that motivate individuals to reduce or stop the pain (affective-motivational aspect)[81]. Importantly, both of these processes can be affected by factors going beyond biological differences. The biopsychosocial model of pain proposes that social and psychological factors, such as expectations and personality traits, affect the experience of pain[82,83]. Tolerance levels, in particular, are thought to be related more to the affective-motivational aspect[84,85], suggesting that individuals with elevated psychopathic traits might be *less* bothered by the unpleasantness of pain. At the same time, however, the negative correlations between Affective, Lifestyle and Antisocial traits and our measure of pain intensity at the tolerance level also indicate individuals high in those traits might also have reduced sensory sensitivity to experimental pain. Taken together, these results suggest that people with elevated levels of psychopathic traits might feel less intense pain from a standardized stimulus and/or perceive the pain as less unpleasant, compared to people low in those traits. This notion is additionally supported by findings of reductions in pain-related ERPs with higher levels of psychopathic traits[36].

These insights align with theoretical frameworks of how pain affects behavioral adaptation. The RL framework of pain conceptualizes it as a behavioral control signal that minimizes current but also potential harm[38]. Pain sensitivity is endogenously fine-tuned to maximize the value of pain as a learning signal so individuals can balance the acquisition of new information while minimizing threat[38]. Therefore, if the painful outcomes do not elicit the same sensation of unpleasantness that should urge the individual to choose a course of action that reduces pain, then they will not be sufficiently aversive enough to motivate a behavioral adaptation. Beyond demonstrating reduced learning due to reduced pain sensitivity, our findings also shed light on the specific RL mechanism disrupted by the reduced aversion of the painful reinforcer. Because individuals with psychopathic traits are relatively pain-insensitive, they ignore the painful negative outcomes and adhere to their initial beliefs because the pain is not sufficiently motivating to drive incorporating those outcomes.

While pain is thought to represent a complex interplay of biological, psychological, and social factors, pain sensitivity and reduced RL might share a common biological substrate. The affective-motivational aspect of pain is encoded in the anterior cingulate cortex (ACC[86,87]), an area also involved in reversal[87] and associative learning[88]. Previous work within the same computational framework has found that the ACC is critical for maintaining representations of uncertainty and environmental volatility[89], and encoding prediction errors about contingencies[90]. However, neither of these studies measured psychopathic traits or accounted for perseveration in original beliefs. In this study, we found an association between psychopathy and increased belief resetting, specifically in the pain condition and through the role of reduced pain sensitivity, which could point to a shared neural mechanism that is impaired in psychopathy, possibly linked to the well-documented aberrations in ACC activity[91]. Given these findings, future research should explore the role of pain as a behavior control signal, ideally by directly collecting pain ratings of subjective intensity and unpleasantness.

Limitations

It should be noted that our results are based on individuals from the general population. While we had a good distribution of scores on the psychopathic traits, comparable to other studies[9,10], studying the concepts of interest in a population with more extreme psychopathic traits (e.g., offenders) would allow for an even more in-depth assessment of the learning deficits. Additionally, we only employed a computational and behavioral approach to investigate the relationship of interest. Given the findings of diminished pain-related ERPs with higher psychopathic traits[36], exploring learning from

pain with electrophysiological methods could elucidate the mechanisms at play. Furthermore, the shared neural underpinnings of reversal learning and affective pain processing in the ACC should be explored in more detail, ideally with functional brain imaging.

Another point to consider is that, as the painful negative outcome was always paired with a subjective reward (to ensure outcomes had a matched valence), we cannot directly disentangle the effects of reward and punishment sensitivity on learning. In general, psychopathy is associated with reduced punishment sensitivity and reduced punishment learning[23,26]. The observed effects can, therefore, be driven by a pull towards the reward, a push away from the punishment, or a combination of both. Given the salience of the painful outcomes in the literature[37,56], it is highly likely that the aberrations in learning processes can be attributed to a dampened effect of punishment (see ref. 25). However, future research should aim to disentangle the two mechanisms and isolate the specific role of pain on RL in people with higher psychopathic traits. The fixed order of conditions also presents a further limitation. Practice effects with the task might explain some of the differences between the non-pain and pain conditions, given that the pain condition always followed the non-pain one. Thus, we recommend accounting for possible practice and order effects to gain a deeper understanding of the specific impact pain has on RL.

Lastly, it should be noted that, while the computational parameters explaining learning in the two conditions differed, there was no significant difference between the behavioral task performance measures (accuracy, win-stay, lose-switch) between conditions. These results appear surprising at first, especially since the latent mechanisms are estimated based on the behavior. One explanation could be related to the more informed priors we adopted in modeling the pain condition data. It is, therefore, possible that the difference in parameters could be attributed to the pain model representing a better fit than the non-pain model. However, another interpretation is that, while the behavior was comparable across conditions, the contribution of the various mechanisms differed depending on the type of reinforcer individuals learned from. In a sense, the same level of accuracy (or rates of switching behavior) can be achieved through different levels of involvement of the underlying cognitive processes, the combination of which could represent unique strategies adopted in each condition. This suggests that while observed behavior might remain consistent, the mechanisms driving it vary, further highlighting the importance of considering the latent cognitive processes when studying learning.

Conclusions

In conclusion, adaptive decision-making requires weighing in outcomes associated with different actions but also learning about change in these associations. We demonstrated impairment in a computational mechanism underlying RL that leads individuals with elevated psychopathic traits to persevere with their original beliefs, resulting in a more exploitative decision-making style. The mediating role of reduced pain sensitivity in this process brings together findings of affective, pain, and reversal learning deficits in psychopathy and is crucial for our understanding of antisocial behavior. Individual differences in pain processing may contribute to the development of antisocial behavior[92], and empathy and empathic responding might also be reliant on intact RL[93]. The present results suggest a mechanism through which physical pain insensitivity contributes to maladaptive decision-making and prevents those with psychopathic traits from effectively learning from their past mistakes.

Data availability

The data described in this study are openly available at https://osf.io/dxrh5/?view_only=df20b9cf26474b9ba3d74f236f1867b5.

Code availability

The code for the computational model described in this paper is freely and publicly available at: https://github.com/translationalneuromodeling/tapas/blob/master/HGF/. The code used for the analysis of the data is openly available at https://osf.io/dxrh5/?view_only=df20b9cf26474b9ba3d74f236f1867b5.

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References

1. Anderson, N. E. & Kiehl, K. A. Psychopathy: developmental perspectives and their implications for treatment. *Restor. Neurol. Neurosci.* **32**, 103–117 (2014).
2. Hosker-Field, A. M., Molnar, D. S. & Book, A. S. Psychopathy and risk taking: examining the role of risk perception. *Personal. Individ Differ.* **91**, 123–132 (2016).
3. Snowden, R. J., Smith, C. & Gray, N. S. Risk taking and the triarchic model of psychopathy. *J. Clin. Exp. Neuropsychol.* **39**, 988–1001 (2017).
4. Hare, R. D. Psychopathy: a clinical and forensic overview. *Psychiatr. Clin. North Am.* **29**, 709–724 (2006).
5. Neumann, C. S., Hare, R. D. & Newman, J. P. The super-ordinate nature of the psychopathy checklist-revised. *J. Personal. Disord.* **21**, 102–117 (2007).
6. Neumann, C. S., Schmitt, D. S., Carter, R., Embley, I. & Hare, R. D. Psychopathic traits in females and males across the globe: psychopathic traits. *Behav. Sci. Law* **30**, 557–574 (2012).
7. Brazil, I. A., Van Dongen, J. D. M., Maes, J. H. R., Mars, R. B. & Baskin-Sommers, A. R. Classification and treatment of antisocial individuals: from behavior to biocognition. *Neurosci. Biobehav. Rev.* **91**, 259–277 (2018).
8. De Brito, S. A. et al. Psychopathy. *Nat. Rev. Dis. Prim.* **7**, 49 (2021).
9. Gordts, S., Uzieblo, K., Neumann, C., Van den Bussche, E. & Rossi, G. Validity of the Self-Report Psychopathy Scales (SRP-III Full and Short Versions) in a community sample. *Assessment* **24**, 308–325 (2017).
10. Sanz-García, A., Gesteira, C., Sanz, J. & García-Vera, M. P. Prevalence of psychopathy in the general adult population: a systematic review and meta-analysis. *Front. Psychol.* **12**, 661044 (2021).
11. Seara-Cardoso, A., Queirós, A., Fernandes, E., Coutinho, J. & Neumann, C. Psychometric properties and construct validity of the Short Version of the Self-Report Psychopathy Scale in a Southern European Sample. *J. Pers. Assess.* **102**, 457–468 (2020).
12. Leistico, A. M. R., Salekin, R. T., DeCoster, J. & Rogers, R. A large-scale meta-analysis relating the Hare measures of psychopathy to antisocial conduct. *Law Hum. Behav.* **32**, 28–45 (2008).
13. Neumann, C. S. & Hare, R. D. Psychopathic traits in a large community sample: links to violence, alcohol use, and intelligence. *J. Consult. Clin. Psychol.* **76**, 893–899 (2008).
14. Maia, T. V. Reinforcement learning, conditioning, and the brain: successes and challenges. *Cogn. Affect. Behav. Neurosci.* **9**, 343–364 (2009).
15. Hemphill, J. F., Hare, R. D. & Wong, S. Psychopathy and recidivism: a review. *Leg. Criminol. Psychol.* **3**, 139–170 (1998).
16. Brazil, I. A., Mathys, C. D., Popma, A., Hoppenbrouwers, S. S. & Cohn, M. D. Representational uncertainty in the brain during threat conditioning and the link with psychopathic traits. *Biol. Psychiatry Cogn. Neurosci. Neuroimaging* **2**, 689–695 (2017).
17. Budhani, S., Richell, R. A. & Blair, R. J. R. Impaired reversal but intact acquisition: probabilistic response reversal deficits in adult individuals with psychopathy. *J. Abnorm. Psychol.* **115**, 552–558 (2006).
18. Budhani, S. & Blair, R. J. R. Response reversal and children with psychopathic tendencies: success is a function of salience of contingency change. *J. Child Psychol. Psychiatry* **46**, 972–981 (2005).
19. Newman, J. P., Patterson, C. M. & Kosson, D. S. Response perseveration in psychopaths. *J. Abnorm. Psychol.* **96**, 145–148 (1987).

20. Oba, T., Katahira, K. & Ohira, H. The effect of reduced learning ability on avoidance in psychopathy: a computational approach. *Front. Psychol.* **10**, 2432 (2019).
21. White, S. F. et al. Disrupted expected value and prediction error signaling in youths with disruptive behavior disorders during a passive avoidance task. *Am. J. Psychiatry* **170**, 315–323 (2013).
22. Hare, R. D. Psychopathy and physiological activity during anticipation of an aversive stimulus in a distraction paradigm. *Psychophysiology* **19**, 266–271 (1982).
23. Hare, R. D., Frazelle, J. & Cox, D. N. Psychopathy and physiological responses to threat of an aversive stimulus. *Psychophysiology* **15**, 165–172 (1978).
24. Patrick, C. J. Emotion and psychopathy: startling new insights. *Psychophysiology* **31**, 319–330 (1994).
25. Elster, E. M. et al. Impaired punishment learning in conduct disorder. *J. Am. Acad. Child Adolesc. Psychiatry* 2023 Jul; S089085672300374X.
26. von Borries, A. K. L. et al. Neural correlates of error-related learning deficits in individuals with psychopathy. *Psychol. Med.* **40**, 1559–1568 (2010).
27. Morgan, J. E., Bowen, K. L., Moore, S. C. & Van Goozen, S. H. M. The relationship between reward and punishment sensitivity and antisocial behavior in male adolescents. *Personal. Individ Differ.* **63**, 122–127 (2014).
28. Murray, L., Waller, R. & Hyde, L. W. A systematic review examining the link between psychopathic personality traits, antisocial behavior, and neural reactivity during reward and loss processing. *Personal. Disord. Theory Res Treat.* **9**, 497–509 (2018).
29. Ribes-Guardiola, P., Poy, R., Segarra, P., Branchadell, V. & Moltó, J. Response perseveration and the triarchic model of psychopathy in an undergraduate sample. *Personal. Disord. Theory Res Treat.* **11**, 54–62 (2020).
30. Yang, Y., Raine, A., Colletti, P., Toga, A. W. & Narr, K. L. Abnormal structural correlates of response perseveration in individuals with psychopathy. *J. Neuropsychiatry Clin. Neurosci.* **23**, 107–110 (2011).
31. Blair, R. J. R. The roles of orbital frontal cortex in the modulation of antisocial behavior. *Brain Cogn.* **55**, 198–208 (2004).
32. Behrens, T. E. J., Woolrich, M. W., Walton, M. E. & Rushworth, M. F. S. Learning the value of information in an uncertain world. *Nat. Neurosci.* **10**, 1214–1221 (2007).
33. Mathys, C., Daunizeau, J., Friston, K. J. & Stephan, K. E. A Bayesian foundation for individual learning under uncertainty. *Front. Hum. Neurosci.* **5**, 39 (2011).
34. Mathys, C. D. et al. Uncertainty in perception and the Hierarchical Gaussian Filter. *Front. Hum. Neurosci.* **8**, 825 (2014).
35. Buades-Rotger, M., Smeijers, D., Gallardo-Pujol, D., Krämer, U. M., & Brazil, I. Acquisition of Hostile Expectation Biases in an online sample [Internet]. (Radboud University, 2023) https://data.donders.ru.nl/collections/di/dcc/DSC_2020.00142_320 (cited 2023 Nov 11).
36. Van Heck, C. H. et al. Pain processing in a social context and the link with psychopathic personality traits—an event-related potential study. *Front. Behav. Neurosci.* **11**, 180 (2017).
37. Willems, A. L., Jelinčić, V., Vlaeyen, J. W. S., von Leupoldt, A. & Torta, D. M. Is it a painful error? The effect of unpredictability and intensity of punishment on the error-related negativity, and somatosensory evoked potentials. *Biol. Psychol.* **165**, 108177 (2021).
38. Seymour, B. Pain: a precision signal for reinforcement learning and control. *Neuron* **101**, 1029–1041 (2019).
39. Krypotos, A. M., Crombez, G., Alves, M., Claes, N. & Vlaeyen, J. W. S. The exploration–exploitation dilemma in pain: an experimental investigation. *Pain* **163**, e215–e233 (2022).
40. Brislin, S. J., Buchman-Schmitt, J. M., Joiner, T. E. & Patrick, C. J. Do unto others"? Distinct psychopathy facets predict reduced perception and tolerance of pain. *Personal. Disord. Theory Res. Treat.* **7**, 240–246 (2016).
41. Fedora, O. & Reddon, J. R. Psychopathic and nonpsychopathic inmates differ from normal controls in tolerance levels of electrical stimulation. *J. Clin. Psychol.* **49**, 326–331 (1993).
42. Miller, J. D., Rausher, S., Hyatt, C. S., Maples, J. & Zeichner, A. Examining the relations among pain tolerance, psychopathic traits, and violent and nonviolent antisocial behavior. *J. Abnorm. Psychol.* **123**, 205–213 (2014).
43. Rescorla R. A., Wagner A. R. A Theory of pavlovian conditioning: variations in the effectiveness of reinforcement and nonreinforcement. In (eds Black A. H., Prokasy W. F.) In: *Classical Conditioning II: Current Research and Theory*. (Appleton-Century-Crofts, New York, 1972). pp. 64–99.
44. Friston, K. A theory of cortical responses. *Philos. Trans. R. Soc. B Biol. Sci.* **360**, 815–836 (2005).
45. Rao, R. P. N. & Ballard, D. H. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nat. Neurosci.* **2**, 79–87 (1999).
46. Adams, R. A., Napier, G., Roiser, J. P., Mathys, C. & Gilleen, J. Attractor-like Dynamics in Belief Updating in Schizophrenia. *J. Neurosci.* **38**, 9471–9485 (2018).
47. Brazil, I. A. et al. RB. Psychopathy-related traits and the use of reward and social information: a computational approach. *Front. Psychol.* http://journal.frontiersin.org/article/10.3389/fpsyg.2013.00952/abstract (2013).
48. Sideridis, G., Simos, P., Papanicolaou, A. & Fletcher, J. Using structural equation modeling to assess functional connectivity in the brain: power and sample size considerations. *Educ. Psychol. Meas.* **74**, 733–758 (2014).
49. Length R. emmeans: Estimated Marginal Means, aka Least-Squares Means. https://CRAN.R-project.org/package=emmeans (2023).
50. Ruscheweyh, R., Marziniak, M., Stumpenhorst, F., Reinholz, J. & Knecht, S. Pain sensitivity can be assessed by self-rating: development and validation of the pain sensitivity questionnaire. *Pain* **146**, 65–74 (2009).
51. FeldmanHall, O. et al. What we say and what we do: the relationship between real and hypothetical moral choices. *Cognition* **123**, 434–441 (2012).
52. Tomiello, S., Schöbi, D., Weber, L. A. E., Iglesias, S., Stephan, K. E. Dopaminergic and cholinergic modulation of reward learning: a computational trial-wise EEG analysis. *OHBM* https://www.tnu.ethz.ch/fileadmin/user_upload/OHBM19_Poster_STomiello.pdf (2019).
53. Glimmerveen, J. C., Maes, J. H. R., Bulten, E., Scheper, I. & Brazil, I. A. So what'cha want? The impact of individualised rewards on associative learning in psychopathic offenders. *Cortex* **149**, 44–58 (2022).
54. Diaconescu, A. O., Wellstein, K. V., Kasper, L., Mathys, C. & Stephan, K. E. Hierarchical Bayesian models of social inference for probing persecutory delusional ideation. *J. Abnorm Psychol.* **129**, 556–569 (2020).
55. The jamovi project. (2022).
56. Jeffreys H. *Theory of Probability*. 3rd ed. (Oxford: Oxford University Press, 1961).
57. Muthén L. K. & Muthén B. O. *Mplus: Statistical Analysis with Latent Variables: User's Guide*. (Los Angeles, CA, 2017).
58. Bürkner P. C. Bayesian Item Response Modeling in R with brms and Stan. Available from: https://arxiv.org/abs/1905.09501 (2019).
59. Bürkner P. C. Bayesian Item Response Modeling in *R* with **brms** and *Stan*. *J. Stat. Softw*. https://www.jstatsoft.org/v100/i05/ (2021).
60. Bürkner P. C. **brms**: An *R* Package for Bayesian Multilevel Models Using *Stan*. *J. Stat. Softw*. http://www.jstatsoft.org/v80/i01/ (2017).
61. Bürkner, P. C. Advanced Bayesian multilevel modeling with the R package brms. *R. J.* **10**, 395 (2018).
62. RStudio Team. RStudio: Integrated Development for R. *RStudio*. (PBC, Boston, MA, 2020). http://www.rstudio.com.
63. The Jamovi Project. Jamovi (Version 1.2). https://www.jamovi.org (2020).

64. Brazil, I. A., Atanassova, D. V. & Oosterman, J. M. Own pain distress mediates the link between the lifestyle facet of psychopathy and estimates of pain distress in others. *Front. Behav. Neurosci.* **16**, 824697 (2022).
65. Driessen, J. M. A. et al. A comparison of latent profiles in antisocial male offenders. *J. Crim. Justice* **57**, 47–55 (2018).
66. Stephan, K. E., Penny, W. D., Daunizeau, J., Moran, R. J. & Friston, K. J. Bayesian model selection for group studies. *NeuroImage* **46**, 1004–1017 (2009).
67. Hoppenbrouwers, S. S., Neumann, C. S., Lewis, J. & Johansson, P. A latent variable analysis of the Psychopathy Checklist–Revised and behavioral inhibition system/behavioral activation system factors in North American and Swedish offenders. *Personal. Disord. Theory Res. Treat.* **6**, 251–260 (2015).
68. Marcoux, L. A. et al. Feeling but not caring: empathic alteration in narcissistic men with high psychopathic traits. *Psychiatry Res. Neuroimaging* **224**, 341–348 (2014).
69. Brislin, S. J., Perkins, E. R., Ribes-Guardiola, P., Patrick, C. J. & Foell, J. Pain processing and antisocial behavior: a multimodal investigation of the roles of boldness and meanness. *Personal. Disord. Theory Res. Treat.* **13**, 685–696 (2022).
70. Durand, G. & Plata, E. M. The effects of psychopathic traits on fear of pain, anxiety, and stress. *Personal. Individ Differ.* **119**, 198–203 (2017).
71. Hoppenbrouwers, S. S., Bulten, B. H. & Brazil, I. A. Parsing fear: a reassessment of the evidence for fear deficits in psychopathy. *Psychol. Bull.* **142**, 573–600 (2016).
72. Friston, K. The free-energy principle: a unified brain theory? *Nat. Rev. Neurosci.* **11**, 127–138 (2010).
73. Frick, P. J. & Ray, J. V. Evaluating callous-unemotional traits as a personality construct. *J. Pers.* **83**, 710–722 (2015).
74. Paulhus D. L., Neumann C. S., Hare R. D. *Manual for the Hare Self-Report Psychopathy Scale*. (Multi-Health Systems, Toronto, ON, 2016).
75. Moltó, J., Poy, R., Segarra, P., Pastor, M. C. & Montañés, S. Response perseveration in psychopaths: Interpersonal/affective or social deviance traits? *J. Abnorm. Psychol.* **116**, 632–637 (2007).
76. Brazil, I. et al. Reversal deficits in individuals with psychopathy in explicit but not implicit learning conditions. *J. Psychiatry Neurosci.* **38**, E13–E20 (2013).
77. Brazil, I. A. et al. Early and late components of error monitoring in violent offenders with psychopathy. *Biol. Psychiatry* **65**, 137–143 (2009).
78. Newman, J. P., Patterson, C. M., Howland, E. W. & Nichols, S. L. Passive avoidance in psychopaths: the effects of reward. *Personal. Individ. Differ.* **11**, 1101–1114 (1990).
79. Delgado M. R., Jou R. L., Phelps E. A. Neural systems underlying aversive conditioning in humans with primary and secondary reinforcers. *Front. Neurosci.* http://journal.frontiersin.org/article/10.3389/fnins.2011.00071/abstract (2011).
80. Mulligan, E. M. & Hajcak, G. The electrocortical response to rewarding and aversive feedback: the reward positivity does not reflect salience in simple gambling tasks. *Int. J. Psychophysiol.* **132**, 262–267 (2018).
81. Talbot, K., Madden, V. J., Jones, S. L. & Moseley, G. L. The sensory and affective components of pain: are they differentially modifiable dimensions or inseparable aspects of a unitary experience? A systematic review. *Br. J. Anaesth.* **123**, e263–e272 (2019).
82. Loeser, J. D. & Melzack, R. Pain: an overview. *Lancet* **353**, 1607–1609 (1999).
83. Atanassova, D. V., Madariaga, V. I., Oosterman, J. M. & Brazil, I. A. Unpacking the relationship between Big Five personality traits and experimental pain: a systematic review and meta-analysis. *Neurosci. Biobehav. Rev.* **163**, 105786 (2024).
84. Greenspan J. D. Pain in humans, thresholds. In: (eds Gebhart G. F., Schmidt R. F.). *Encyclopedia of Pain.* (Springer Berlin Heidelberg, Berlin, Heidelberg, 2013). http://link.springer.com/10.1007/978-3-642-28753-4_3100 (cited 2024 Jan 16). pp. 2658–2660.
85. Fuchs, P. N., Peng, Y. B., Boyette-Davis, J. A. & Uhelski, M. L. The anterior cingulate cortex and pain processing. *Front. Integr. Neurosci.* **8**, 35 (2014).
86. Xiao, X. & Zhang, Y. Q. A new perspective on the anterior cingulate cortex and affective pain. *Neurosci. Biobehav Rev.* **90**, 200–211 (2018).
87. Rushworth, M. F. S., Behrens, T. E. J., Rudebeck, P. H. & Walton, M. E. Contrasting roles for cingulate and orbitofrontal cortex in decisions and social behaviour. *Trends Cogn. Sci.* **11**, 168–176 (2007).
88. Blair, R. J. R. Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Dev. Psychopathol.* **17**, 865–891 (2005).
89. Hein, T. P. et al. Anterior cingulate and medial prefrontal cortex oscillations underlie learning alterations in trait anxiety in humans. *Commun. Biol.* **6**, 271 (2023).
90. Iglesias, S. et al. Hierarchical prediction errors in midbrain and basal forebrain during sensory learning. *Neuron* **80**, 519–530 (2013).
91. Koenigs M. The role of prefrontal cortex in psychopathy. *Rev. Neurosci.* https://www.degruyter.com/document/doi/10.1515/revneuro-2012-0036/html (2012).
92. Niel, K. A., Hunnicutt-Ferguson, K., Reidy, D. E., Martinez, M. A. & Zeichner, A. Relationship of pain tolerance with human aggression. *Psychol. Rep.* **101**, 141–144 (2007).
93. Shamay-Tsoory, S. G. & Hertz, U. Adaptive empathy: a model for learning empathic responses in response to feedback. *Perspect. Psychol. Sci.* **17**, 1008–1023 (2022).

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Author contributions

D.V.A., J.M.O., and I.A.B. conceived the presented idea. D.V.A. designed the experiment. D.V.A. and V.I.M. collected the data. D.V.A. performed the computations and analysis with support from I.A.B., C.M., and A.O.D. C.M. and A.O.D. verified the analytical methods and contributed to the interpretation of the results. D.V.A. wrote the paper with input from all authors. All authors provided critical feedback and helped shape the research, analysis, and paper.

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