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Revisiting the electrophysiological correlates of valence and expectancy in reward processing – A multi-lab replication

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ABSTRACT

Two event-related brain potential (ERP) components, the frontocentral feedback-related negativity (FRN) and the posterior P300, are key in feedback processing. The FRN typically exhibits greater amplitude in response to negative and unexpected outcomes, whereas the P300 is generally more pronounced for positive outcomes. In an influential ERP study, Hajcak et al., (2005) manipulated outcome valence and expectancy in a guessing task. They found the FRN was larger for negative outcomes regardless of expectancy, and the P300 larger for unexpected outcomes regardless of valence. These findings challenged the dominant Reinforcement Learning Theory of the ERN. We aimed to replicate these results within the #EEGManyLabs project (Pavlov et al., 2021) across thirteen labs. Our replication, including robustness tests, a PCA and Bayesian models, found that both FRN and P300 were significantly modulated by outcome valence and expectancy: FRN amplitudes (no-reward - reward) were largest for unexpected outcomes, and P300 amplitudes were largest for reward outcomes. These results were consistent across different methods and analyses. Although our findings only partially replicate the original study, they underscore the complexity of feedback processing and demonstrate how aspects of Reinforcement Learning Theory may apply to the P300 component, reinforcing the need for rigorous ERP research methodologies.
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1. Introduction

Performance monitoring is critical for detecting possible mismatches between goals and actions and, upon their detection, triggering specific remedial processes (Ullsperger, Fischer, Nigbur, & Endrass, 2014). This monitoring can be based either on internal cues, such as response errors, or external ones, such as unfavorable or negative evaluative feedback. A wealth of studies has used electroencephalographic (EEG) methods in humans and established the electrophysiological correlates of performance monitoring when it is based on internal or external cues (Ullsperger, Danielmeier, & Jocham, 2014). Regarding the latter process, two distinct and successive event-related potential (ERP) components have been identified as reliable markers of performance monitoring: the feedback-related negativity (FRN) (Gehring & Willoughby, 2002) and the P300 (Courchesne, Hillyard, & Courchesne, 1977). The FRN is a negative component recorded at fronto-central electrodes along the midline (most pronounced at electrodes Fz and FCz) that typically peaks around 250 msec after feedback onset. It is larger (i.e., more negative-going) for negative than positive feedback/outcomes (Miltner, Braun, & Coles, 1997). Following the FRN, the P300 component, or more specifically the P3b (Polich, 2007; Walentowska, Moors, Paul, & Pourtois, 2016), is elicited around 300–500 msec following feedback onset, and shows a more central/centro-parietal scalp distribution than the FRN (electrodes Cz and Pz). The P300 is larger (i.e., more positive-going) for unexpected/infrequent than expected/frequent events (Johnson & Donchin, 1980; Polich, 2007). The P300 is most often studied in the context of attention (Herrmann & Knight, 2001) and might reflect motivational processes involved during outcome and feedback processing (Huvermann, Bellebaum, & Peterburs, 2021; San Martín, 2012). Along these lines, these two ERP components likely reflect different aspects of information processing and/or a progressive accumulation of evidence of internal predictions endorsed by the participant during performance monitoring (Ullsperger, Danielmeier, & Jocham, 2014).

The influential Reinforcement Learning Theory of the ERN (ERN-RL) put forward by Holroyd and Coles (2002) proposed that the FRN and its response-based counterpart, the error-related negativity (ERN, Gehring, Goss, Coles, Meyer, & Donchin, 2018) is a scalp manifestation of neural activity originating from the (dorsal) ACC, which itself receives direct dopaminergic inputs from the basal ganglia, including the striatum. In this model (Holroyd & Coles, 2002; see also Nieuwenhuis, Holroyd, Mol, & Coles, 2004), the FRN reflects the detection of a discrepancy between the actual and the expected outcome (i.e., prediction error). Moreover, the FRN appears to be somewhat monotonically related to the size of the prediction error: the more unexpected an outcome is, the larger is the FRN (Holroyd, Krigolson, Baker, Lee, & Gibson, 2009; Weismüller & Bellebaum, 2016), although this

relationship might not be linear (Williams, Hassall, Trska, Holroyd, & Krigolson, 2017). Whether the feedback is utilitarian (e.g., incentive-related) or performance-related (e.g., informing about accuracy) is irrelevant, as this prediction error captured by the FRN is equally large for unexpected outcomes in both cases (Nieuwenhuis, 2004).

Using this framework, Hajcak, Holroyd, Moser, and Simons (2005) performed an EEG study in which they assessed amplitude changes of the FRN and P300 components as a function of both valence and expectancy. They used a guessing task (a.k.a. the Doors Task; see Holroyd et al., 2003) in which participants had to guess which of four presented doors hid a small monetary prize (.10$ reward). Importantly, prior to the choice, the probability to win (25%, 50%, or 75%) was announced to manipulate outcome expectancy. Results showed that the FRN did not differentiate between these three levels of expectancy, while the P300 increased as a function of unexpectedness [i.e., it was more pronounced for unexpected (25%) than neutral (50%) outcomes, and for neutral than expected (75%) outcomes]. These findings were found across two experiments in which expectancy was manipulated trial-wise ($N = 17$) and block-wise ($N = 12$), respectively.

In the following years, these findings received mixed support, and the extent to which the P300 is insensitive to valence and the FRN is insensitive to expectancy remains contested. Whereas various experiments and meta-analyses have consistently shown that the P300 increases with outcome unexpectedness (Stewardson & Sambrook, 2020), the effect of outcome valence on the P300 remains unclear. Some studies report similar results as Hajcak et al. (2005), i.e., no effect of outcome valence on the P300 component (Pfabigan, Alexopoulos, Bauer, & Sailer, 2011), yet others have shown effects in the opposite direction, i.e., positive outcomes elicited either larger or smaller P300 amplitudes (Glazer, Kelley, Pornpattananangkul, Mittal, & Nusslock, 2018; San Martín, 2012; Stewardson & Sambrook, 2020). To explain these discrepancies, methodological differences such as imbalanced stimulus frequencies, have sometimes been discussed (Stewardson & Sambrook, 2020). In comparison, the observed insensitivity of the FRN to expectancy has gained much more attention as this observation was at odds with the predictions of the ERN-RL theory (Holroyd & Coles, 2002; Walsh & Anderson, 2012) and inconsistent with previous empirical observations (Holroyd et al., 2003).

To reconcile the divergent findings, Hajcak et al. (2005) suggested that this signed prediction error effect conferred to the FRN was observed using trial-and-error learning tasks, as opposed to guessing tasks. Consistent with this interpretation, later ERP studies using learning-based tasks reported modulations of the FRN by expectancy (e.g., Ferdinand, Mecklinger, Kray, & Gehring, 2012; Gu et al., 2021; Holroyd et al., 2009; Warren & Holroyd, 2012), while expectancy modulations were only rarely found in guessing tasks (Gheza, Paul, & Pourtois, 2018; HajiHosseini, Rodríguez-Fornells, & Marco-Pallarés, 2012). The close coupling of choices, expectations, and the following outcomes could be at the core of this discrepancy (Hajcak, Moser, Holroyd, & Simons, 2007). Thus, while this finding for the FRN was surprising at first, subsequent studies and some meta-analyses confirmed that insensitivity (or lower sensitivity) of the FRN to expectancy could be common in contexts in which learning remains inherently limited, such as in guessing tasks (e.g., Guthrie, 1942; Sambrook, Roser, & Goslin, 2012).

This original study has engendered a large amount of ERP studies and theoretical models, which have often used similar guessing tasks, and characterized the electrophysiological correlates of reward processing during performance monitoring in various contexts and situations (see Glazer et al., 2018; San Martín, 2012; Walsh & Anderson, 2012). Moreover, following the publication of this study, several methodological and theoretical refinements have been proposed to explore reward-based feedback processing at the FRN level. Chief amongst these developments has been the recognition that variation in the FRN signal may be the product of a superimposed positive-going deflection, a so-called Reward Positivity (RewP; see Proudfit, 2015). When conceptualizing feedback-related ERPs as the difference between positive and negative outcomes, the component labels are interchangeable as this new perspective affects only the direction of the effects (i.e., for unexpected outcomes the component is more positive or more negative) (Krigolson, 2018; Proudfit, 2015). However, when looking at the condition-specific ERPs, this new perspective affects the sign of the prediction error. If the response to negative, "worse-than-expected", outcomes drives the effects, the FRN/RewP captures a negative prediction error. If the response to positive, "better-than-expected", outcomes drives the effects, the FRN/RewP captures a positive prediction error. While many attempts have been made to disentangle these different responses (Foti, Weinberg, Dien, & Hajcak, 2011; Gable, Paul, Pourtois, & Burgdorf, 2021; Gheza et al., 2018), the FRN/RewP probably captures both due to the underlying frequency responses (Bernat, Nelson, & Baskin-Sommers, 2015; Hoy, Steiner, & Knight, 2021). Nevertheless, this paradigm shift did not only move the focus towards positive (as opposed to negative) outcomes, but also contributed to important methodological discussions about how to best measure this early ERP component following feedback onset (Klawohn, Meyer, Weinberg, & Hajcak, 2020). Hence, it appears important to investigate if the sensitivity to expectedness is driven by the response to positive or negative outcomes.

The results of this study sparked numerous conceptual replications on the nature of the FRN/RewP and the P300 component across different tasks, motivational contexts, and in clinical and non-clinical populations. To date, the work has been cited over 620 times (Google Scholar in November 2024). Yet, despite this intense focus, there has been no direct replication of the original procedure, measures, and analyses. The goal of the present study was to undertake a multi-lab replication of Hajcak et al. (2005), using a trial-by-trial manipulation of both expectancy and valence. We intended to complement this direct replication with modern preprocessing and analytical approaches to test the robustness of the reported effects. Based on Hajcak et al. (2005), we hypothesized that:

1. The FRN/RewP will not vary with expectancy. More specifically, the amplitude of the FRN/RewP will not be

statistically different for expected, neutral, and unexpected outcomes.
2. The amplitude of the P300 will increase as a function of unexpectedness (i.e., unexpected > neutral > expected), irrespective of valence (reward versus no-reward).

Finally, if, in contrast to the original replication, but in line with the RL-Theory, we would find an effect of expectedness on FRN/RewP amplitudes, we would explore if this effect is driven by the response to reward or no-reward outcomes.

2. Methods

2.1. Statistical power and recruitment procedures

To guide a decision on sample size, the non-significant interaction of expectancy and location for the FRN/RewP component reported in Hajcak et al. (2005) was used. Not only is this the smallest reported effect, it is also the key theoretically relevant result. Unfortunately, the original paper did not report a complete set of statistical results ["$F(2,32) < 1$"), so estimates of the effect size of $\eta_p^2 = .059$[1]] were only a rough overestimation of the true effect size. Additionally, there was no meta-analytical evidence readily available for this effect to compare this estimate. While a meta-analysis by Sambrook and Goslin (2015) reported an effect size of $d = .71$ for expectancy modulation of the FRN/RewP (equal to calculated $\eta_p^2 = .11$), it is important to note that this was aggregated across mostly learning tasks, and it is reasonable [and also discussed by Sambrook and Goslin (2015)] to assume that the effect size could be smaller in guessing tasks. While this could be considered an upper bound of the FRN/RewP effect of expectancy during guessing tasks, we refrained from using this estimate to guide an *a-priori* sample size determination.

To circumvent these limitations, we opted for a sensitivity analysis. Based on available resources, each of the thirteen replicating labs will provide the data from 25 participants [excluding participants because of computer malfunction, drop out, technical problems, or insufficient clean data (see below)], resulting in a sample size of 325 participants across all labs. With such a sample size, a sensitivity analysis in MorePower (6.0.4. Campbell & Thompson, 2012) showed that the smallest effect size that can be reliably detected is $\eta_p^2 = .014$ ($\alpha = .02$, $1 - \beta = .90$, 3×3 interaction in repeated measures ANOVA). This allowed us to identify a much smaller effect than any individual study on this matter has been able to identify so far.

A similar rationale was applied to the non-significant valence effect on the P300 [$F(1,16) = < 1$, calculated $\eta_p^2 = .048$] and the non-significant interaction of valence and expectancy [$F(2,32) = 2.88$, $p > .09$, calculated $\eta_p^2 = .152$]. In comparison, the effect size of the expectancy modulation on the P300 was reported to be relatively large [$F(2,32) = 45.48$, $p < .001$, $\mathcal{E} = .82$, calculated $\eta_p^2 = .740$]. Even after dividing this effect size in half to correct for shrinkage effects commonly observed in replication studies [see Pavlov et al. (2021)], each individual lab had the statistical power to replicate this effect in the collected subsample ($n = 25$, $\alpha = .02$, $\eta_p^2 = .370$, $1 - \beta = .99$, main effect with 3 levels in repeated measures ANOVA).

In each replicating lab, participants were recruited via local advertisements or online recruitment systems. For their participation, they were reimbursed with 15 EUR/200 NOK or course credits. Additionally, each participant received a payout of their in-task wins of 5 EUR/17 AUD/50 NOK/5000 CLP. Participants were told that they could increase their payouts if they chose the "correct door". However, regardless of their choices the outcome was preprogrammed and unrelated to the choices made by the participants.

For each replicating lab ($n = 13$), the study was approved by the local or national ethical committee/Institutional Review Board [ANU (2022/859); Bond University (DA03365); German Psychological Society (DGPS) (PK-22-02-21); Ghent University (2022/14); Leiden University (2022-05-12-M.J.W. van der Molen-V2-3819); University of Bergen, Faculty of Psychology (2020/1926-28) & NSD (320122); UCM (CEC-UCM 54/2023); Erasmus University Rotterdam (ETH2223-0061)].

2.2. Procedure

The procedure followed the process employed in Experiment 1 in Hajcak et al. (2005) as closely as possible, and any departures from this were explicitly stated. Participants were tested individually in an EEG laboratory. Upon their arrival in the lab, they received a brief description of the experiment and provided informed consent. Then they were prepared for EEG recording and the EEG electrodes were attached. Participants were familiarized with the guessing task and the feedback using a practice block consisting of 40 trials (not included in the analysis). Afterwards, they completed 6 blocks of the guessing task, with each block comprising 40 trials (240 trials in total). Self-paced breaks were allowed in between blocks. Every other block, the experimenter entered the testing room to inform about the current winnings (which were presented on the screen), monitored the EEG signal, and kept participants alert.

As this project was part of a wider initiative on replicability in EEG (#EEGManyLabs), most of the laboratories in this replication also collected resting state data EEG data together with some personality measures (https://osf.io/sp3ck/) (Pavlov et al., 2021). Neither EEG nor personality data was analyzed in the current study but will be merged across sites as part of a future replication project to be reported elsewhere. For this purpose, participating labs recorded 8 min of resting state EEG and participants will be asked to fill in three brief questionnaires (using previously validated translations into the local language where possible) prior to the start of the guessing task for the present study. These include the Karolinska Sleepiness Scale (KSS; Åkerstedt & Gillberg, 1990), the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) and the State Trait Anxiety Inventory Trait Version (STAI-T; Spielberger, Gorsuch, & Lushene, 1970). After the guessing task, the labs recording

[1] For this and the following statistics, η_p^2 was calculated from the reported F values (Cohen, 1988; Lakens, 2013), when no F values were reported, we used $F = 1$.

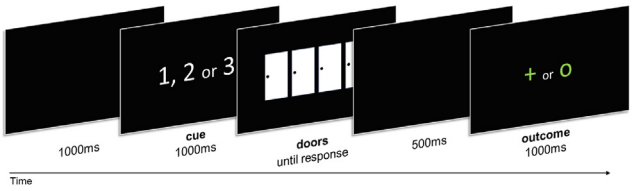

Fig. 1 – Trial structure. Each trial comprises three successive visual events: a cue (that informs about reward probability in the current trial), followed by the presentation of four doors (imperative stimulus; the participant is asked to pick one of them based on guessing), before the outcome (either reward or no-reward) is presented.

this additional data asked participants to fill in the Edinburgh Handedness Inventory (EHI; Oldfield, 1971), the Behavioral Inhibition and Approach System Scales (BIS-BAS; Carver & White, 1994), the Center for Epidemiologic Studies Depression Scale (Radloff, 1977), and the Short Version of the Big Five Inventory (Gerlitz & Schupp, 2005) questionnaires. In the labs that did not record this additional data (see Supplementary Table 7), only the guessing task was presented.[2]

Each trial started with a cue presented for 1000 msec in the center of the screen (see Fig. 1). The cue was presented as the number 1, 2, or 3, corresponding to a probability of winning of 25%, 50%, or 75% (i.e., how many of the four doors contained a prize). After this cue, four doors appeared in the center of the screen and the participant was asked to select one of them by pressing one of four predefined keys on the keyboard (exact keys varied across labs but correspond to four horizontally aligned keys pressed with the index and middle fingers of both hands, e.g., ZCBM for QWERTY keyboards, see Supplementary Table 7). Participants were asked to guess which door could contain a prize. The four doors stayed on screen until the response/choice. Then a blank screen ensued (500 msec), before the outcome was presented in green font for 1000 msec. The outcome was presented as a "+", indicating that a small monetary reward was attained (value is .04 EUR or .15 AUD or .4 NOK or 35 CLP), or as a "o", indicating that no-reward was attained. The trial ended with a 1000 msec blank screen used as inter-trial interval. Stimuli were presented in white on black background. Accordingly, in this task, reward motivation was promoted while no punishment motivation was involved.

There were six experimental conditions, corresponding to the combinations of cue and outcome: expected reward (i.e., "+" symbol following "3" used as cue, 60 trials), neutral reward (i.e., "+" symbol following "2" used as cue, 40 trials), unexpected reward ("+" symbol following "1" used as cue, 20 trials), expected no-reward (i.e., "o" symbol following "1" used as cue, 60 trials), neutral no-reward (i.e., "o" symbol following "2" used as cue, 40 trials), and unexpected no-reward (i.e., "o" symbol following "3" used as cue, 20 trials). Across all blocks, these 6 conditions were shown in random order.

Upon completion of the task, participants were asked to answer two questions related to the attention paid to the numerical cue prior to the doors and the outcome during the experiment. These were answered on a seven-point scale, ranging from "ignored it" to "paid close attention" by the corresponding numbers on the keyboard.

The whole experiment lasted approximately 1–1.5 h. The experiment was programmed using Presentation software (Neurobehavioral Systems, Inc., www.neurobs.com) and PsychoPy (Peirce, 2007) and translated into the local languages (English, Dutch, German, Norwegian, Spanish). Additional details on the used version of the experiment, the screen size, operating systems, used equipment etc. at each replicating lab are listed in the Supplementary Table 7.

2.3. *Neurophysiological recordings*

The replicating labs were using one of the following four EEG systems: (1) Biosemi Active 2; (2) BrainAmp DC, (3) BrainAmp actiCHamp Plus, (4) NeurOne Tesla. Using elastic caps, all labs recorded with either 32 or 64 channels positioned according to the extended 10/20 EEG system (Chatrian, Lettich, & Nelson, 1985). One to four of these 32/64 electrodes or one to four additional external electrodes were used to record electrooculogram (EOG), and two were placed on the left and right mastoids. One EOG electrode was attached below the left eye, additional electrodes were placed above the left eye and on the outer canthi of the two eyes in some labs. The EEG (and EOG) data was sampled at 512, 500, 1000 Hz (depending on the setup). Labs also varied in their use of active versus passive electrodes, and the applied online reference/ground (CMS/DRL, Cz, FCz, AFz). For details on each lab's set-up, see Supplementary Table 7.[3]

2.4. *Artifact removal and EEG preprocessing*

Data preprocessing closely followed the original study, including the following steps: activity recorded from Fz, Cz, and Pz and the additional external electrodes were: (i) re-referenced to Cz (the online-reference of the original study); (ii) filtered with a high-/low-pass filter of .05 and 35 Hz [the offline filter settings of the original study; EEGLAB defaults (Delorme & Makeig, 2004), transition bandwidth .05/8.75 Hz, passband edge .05/35 Hz, cutoff frequency (−6 dB) .025/39.38 Hz] (iii) down-sampled to 200/250/256 Hz as the original study recorded with a sampling rate of 200 Hz; (iv) segmented into epochs of interest (−500/+1500 msec around the onset of the outcome); (v) corrected for ocular artifacts (following Gratton, Coles, & Donchin, 1983, implemented into MATLAB);

[2] Since the recording of the additional data before the guessing task took less than 15 min, we did not expect that these differences would affect the results. Nevertheless, we accounted for inter-lab variance in our statistical analyses (see below).

[3] The new recordings deviate from the original study in a few notable points: amplifier setup (Grass Model 7D polygraph with Neurosoft Quik-caps), number of recording sites (9), sampling rate (200 Hz), as well as pre-processing software (VPM) and applied offline filters (bandpass .05–35 Hz).

(vi) re-referenced to the linked mastoids; (vii) cleaned of segments containing artifacts (25 msec of invariant analog data on any channel; voltage exceeding ±100 μV)[4]; (viii) low-pass filtered at 20 Hz using a FIR filter [eeglab defaults, transition bandwidth 5 Hz, passband edge 20 Hz, cutoff frequency (−6 dB) 22.5 Hz]; (ix) baseline corrected to −200 to 0 msec prior to outcome onset.

In addition to the use of a data preprocessing protocol that closely followed the one provided in the original study, the data was also preprocessed according to recent developments in psychophysiology, which allowed us to test the robustness of the results. Activity recorded from all EEG sensors was: (i) down-sampled to 500/512 Hz (if recorded with higher sampling rates); (ii) re-referenced to mastoids; (iii) high-pass filtered at .1 Hz using a FIR filter [eeglab defaults, transition bandwidth .1 Hz, passband edge .1 Hz, cutoff frequency (−6 dB) .05 Hz]; (iv) low-pass filtered at 40 Hz using a FIR filter [eeglab defaults, transition bandwidth 10 Hz, passband edge 40 Hz, cutoff frequency (−6 dB) 45 Hz]; (v) interpolated (spherically) if activity is invariant (>5 sec) or not correlated to other channels ($r < .8$); (vi) cleaned from bad segments identified by ASR (with burst criterion of 55 SD, ran on 1 Hz high-pass filtered data; segments flagged as bad are then removed from the unfiltered data); (vii) cleaned for ocular artifacts through an Independent Component Analysis (ICA, infomax, performed on 1 Hz high-pass filtered data, rank lowered by the number of interpolated channels, otherwise eeglab defaults; weights were then applied to the unfiltered data) and ICLabel based on the probability of being not a brain component [<30 %) but ocular artifacts (>70%)]; (viii) segmented into epochs of interest (−200/+800 msec around the onset of the outcome); (ix) baseline corrected to −200 to 0 msec prior to outcome onset; and (x) cleaned of bad segments [epochs deviating more than 3.29 SD (Tabachnick & Fidell, 2007) from trimmed normalized means with respect to joint probability, kurtosis or the spectrum].

2.5. Outlier handling

The original study did not mention the use of any particular outlier criterion, and therefore for the direct replication the data from all participants was included.

Nevertheless, to test the robustness of the results, we aimed to ensure good data quality in two ways: First, from all complete recordings, we excluded participants who had more than 75% of trials rejected (i.e., only 60 trials out of the 240 trials used). Second, we excluded participants who had less than 8 trials per condition as the FRN/RewP shows good internal consistency with at least 8 trials (Ethridge & Weinberg, 2018). Included trial number as well as standardized measurement error (Luck, Stewart, Simmons, & Rhemtulla, 2021) were calculated and reported to describe data quality across conditions (and across participating labs).

[4] The original study excluded data segments based on invariant data and/or A/D values exceeding the converter's minimum/maximum values. Since all replicating labs recorded with a different setup than the original study, we chose this cut-off instead.

To ensure that all participants paid attention to the numerical cues as well as the outcome, participants were excluded if they indicated in the attention ratings that they ignored the cue (i.e., answering with one or two on the seven-point scale).

2.6. Quantification of the ERPs

The FRN/RewP was quantified at Fz, Cz, and Pz as follows: First, a difference wave was created by subtracting the ERP observed for reward outcomes from the ERP observed for no-reward outcomes. This difference wave was computed separately for expected outcomes (expected no-reward minus expected reward), neutral outcomes (neutral no-reward minus neutral reward), and unexpected outcomes (unexpected no-reward minus unexpected reward). For each level of expectancy, the FRN/RewP was initially defined as the maximum negative amplitude of these difference waves within a window between 200 and 500 msec following outcome onset. This quantification procedure led to the peak of the FRN/RewP component to be misclassified with an average peak of 325 msec ($SD = 87$, $Range = 203–496$). In around 30% of cases, the FRN/RewP peak was identified *after* the P300 peak. We therefore repeated the analysis constraining the time window to end at the peak of the P300 component (if earlier than 500 msec after outcome onset). These results were mostly similar to the original quantification method. We report the results from the more appropriately scored FRN in the main text and highlight possible differences (where they arose) in the footnotes.

The P300 was scored at Pz as follows. Unlike the FRN/RewP, no difference wave was created. For each of the six conditions, the P300 was defined as the most positive peak in the ERP 200–600 msec following outcome onset.

In addition to this direct replication of the ERP components, we also scored the FRN/RewP and the P300 as mean amplitudes, since peak amplitude values are often more sensitive to high-frequency noise (Luck, 2014). Together with comparing different preprocessing of the data, this allowed us to test the robustness of the results. The FRN/RewP was scored following current recommendations as the mean amplitude 200–300 msec following outcome onset (Gheza et al., 2018; Krigolson, 2018; Proudfit, 2015; Sambrook & Goslin, 2015), while the P300 was scored as the mean amplitude 300–500 msec following outcome onset.

Moreover, since difference waves reduce some of the information helpful for follow-up tests, we additionally scored the FRN/RewP using the actual condition ERPs at Fz (for both peak and mean scoring).

Considering that the FRN/RewP and the P300 components occur in rapid succession, we additionally quantified the EEG components in terms of a principal component analysis (PCA) to ascertain possibly dissociable effects on these components and to disentangle them better using the ERP PCA Toolkit (EP Toolkit, version 2.80; Dien, 2010b). The individual ERPs (for each of the six conditions) from the preprocessing following current standards and after excluding outliers (see above) was used for this analysis. Considering the differences in the recording systems that were used, the individual ERPs were first standardized. Specifically, data was downsampled to a

common denominator (500 Hz) and only 56 electrodes which were common across most labs were used (8 labs, 224 participants).[5] The ERPs were then subjected to a recommended two-step sequential PCA (Spencer, Dien, & Donchin, 1999, 2001). If not further specified, all default values in the graphical interface were used. The procedure began with a temporal Promax rotation to capture the variance across the time points from the average ERP data, followed by a spatial Infomax (ICA) rotation to obtain the variance of the spatial distribution of the data across the common recording sites (Dien, 2010a). The number of factors retained in each step depended on the scree plot, such that only factors explaining more variance than identified in random data was included (similar to parallel testing, see Dien, 2012). From all temporospatial factor combinations, default windowing was applied to screen out factors explaining less than .5% variance. All remaining factors were reconstructed into voltage space, in which the voltage accounted for at the peak time point and channel were evaluated as ERP waveforms. Factors whose peak latencies and channels coincided (based on visual inspection) with the canonical scalp distribution and time course of the FRN/RewP (fronto-central, 200–300 msec) and P3 components (posterior-central, 300–500 msec) were tested.

2.7. Statistical analyses

The main focus of the analyses was (1) a direct replication of the approach applied in the original study using repeated measures analyses of variance (ANOVAs). However, we also tested the robustness of these effects (2) in multilevel models (MLMs), and (3) in a meta-analysis of our effects identified in each lab.

2.7.1. Direct replication through ANOVAs

The ERP amplitudes calculated from the preprocessing and quantification methods used in the original study were subjected to two ANOVAs. For the FRN/RewP, the peak amplitude values were analyzed using a 3 (Location) x 3 (Expectancy) ANOVA. For the P300, a 2 (Valence) x 3 (Expectancy) ANOVA was used. In case a sphericity violation was detected, Greenhouse–Geisser correction was applied to p values. The significance alpha level was set to .02.

Moreover, to test if the results for the FRN/RewP were driven by the response to reward outcomes or no-reward outcomes, we calculated a 2 (Valence) x 3 (Expectancy) ANOVA on the amplitudes extracted at Fz (where it was shown to be maximal in the original study) together with the corresponding post-hoc tests. The main analyses are complemented by a series of robustness analyses (see below and Table 1).

2.7.2. Robustness test through MLMs

To better account for variability across participants and laboratories, we fitted eight Bayesian multilevel linear models on the FRN/RewP and P300 amplitude values. These models were set up identically, but the dependent variable was extracted either after (1) "original" or "current standard" preprocessing pipelines, and (2) quantified as either "peak" scores (as in the original publication) or as "mean" scores (as a more robust measure of the ERP components). By crossing these analytical choices, we were able to assess the impact of these choices on the outcome and the robustness of the replication.

The models were specified as follows [in Wilkinson notation (Wilkinson & Rogers, 1973)]:

FRN/RewP_amplitudes = 1 + location * expectancy + (1 + location * expectancy | laboratory/participant).[6]

P300_amplitudes = 1 + valence * expectancy + (1 + valence * expectancy | laboratory/participant).

Robustness test 1. Amplitudes were extracted after the preprocessing of the original publication and defined as the maximum peak in the specified time window. This followed the analysis of the original publication most closely, while controlling for inter-lab variance.

Robustness test 2. Amplitudes were extracted after the preprocessing of the original publication and defined as the mean in the specified time window.

Robustness test 3. Amplitudes were extracted after the preprocessing according to current standards and defined as the maximum peak in the specified time window.

Robustness test 4. Amplitudes were extracted after the preprocessing according to current standards and defined as the mean in the specified time window.

We allowed intercepts and slopes to vary as a function of participant and laboratory, to model varying effects on amplitude peak (or mean) originating from different laboratory setups and individual characteristics (e.g., skull thickness, hair). As a likelihood function, we chose a Gaussian distribution.

An important aspect of Bayesian analysis is the choice of priors (e.g., Natarajan & Kass, 2000). Given the unknown susceptibility of the electrophysiological signal to inter-individual differences in relation to the predictors of interest, we placed a weakly informative prior on intercepts and slopes: a normal distribution with $\mu = 0$ and $\sigma = 10$. Since we had no prior knowledge regarding the other model parameters (e.g., standard deviation of laboratory or participant), we kept the software default weakly informative priors.

Models were fitted in *R* using the *brms* package (Bürkner, 2018), which employed the probabilistic programming language *Stan* (Carpenter et al., 2017) to implement a Markov chain Monte Carlo (MCMC) algorithm (Hoffman, 2014) to estimate posterior distributions of the parameters of interest. We started sampling by using 4 MCMC chains with 4000 iterations (2000 warm-up) and no thinning. In case of non-convergence, we increased the number of iterations by 500 until convergence was reached or a maximum of 8000 iterations per chain. Model convergence was assessed as follows: (i) visual inspection of trace plots, rank plots, and graphical posterior predictive checks (Gabry, Simpson, Vehtari, Betancourt, & Gelman, 2019); (ii) Gelman-Rubin $\widehat{R}$ statistic (Gelman & Shalizi, 2013) between 1 and 1.05 (see also

[5] Restricting the analyses to only common channels across all thirteen labs resulted in a dramatically lower number of channels (19). Hence, we chose to include those channels present in most labs as a tradeoff between sample size and channel number.

[6] Additionally, we reported the following model in the supplement: FRN/RewP_amplitudes_at_Fz = 1 + valence * expectancy + (1 + valence * expectancy | laboratory/participant). This additional analysis helped to identify if the response to reward outcomes or no-reward outcomes was driving the effect.

Table 1 – Overview of analyses and reported results.

	Hajcak et al.	Direct Replication	Robustness Test 1	Robustness Test 2	Robustness Test 3	Robustness Test 4	Robustness Test 5	Robustness Test 6
Pre-processing	Original	Original	Original	Original	Current standard	Current standard	Original	Current standard
Outlier Handling	None	None	None	None	Applied	Applied	None	Applied
Quantification of ERPs	Peak	Peak	Peak	Mean	Peak	Mean	Peak	PCA
Statistical Test	ANOVA	ANOVA	MLM	MLM	MLM	MLM	Meta-Analysis	ANOVA
N FRN	17	307	307	360	297	328	13/307	230
N P300	17	360	360	360	323	328	13/360	230
FRN replication								
Expectancy	Not sign. $\eta p^2 < .08$[b]	Sign. $\eta_p^2 = .08$ [.05, .13]	++	FZ + PZ –	++	FZ + PZ –	Sign. $r = .32$ [.22, .42]	(1/1) sign. ♦
Location	Sign. $\eta_p^2 = .34$[a]	Sign. $\eta_p^2 = .34$ [.28, .39]	++	++	++	++	Sign. $r = .60$ [.52, .66]	*n.r.*
Location x expectancy	Not sign. $\eta_p^2 < .02$[b]	Sign. $\eta_p^2 = .02$ [.01, .04]	*n.a.*	*n.a.*	*n.a.*	*n.a.*	Sign. $r = .16$ [.05, .27]	*n.r.*
P300 replication								
Valence	Not sign. $\eta_p^2 < .06$[b]	Sign. $\eta_p^2 = .32$ [.24, .39]	++	++	++	++	Sign. $r = .59$ [.49, .68]	(2/3) sign.
Expectancy	Sign. $\eta_p^2 = .74$[a]	Sign. $\eta_p^2 = .37$ [.32, .42]	++	++	+++	++	Sign. $r = .63$ [.56, .69]	(2/3) sign.
Valence x expectancy	Not sign. $\eta_p^2 = .15$[a]	Not sign. $\eta_p^2 = \leq .001$ [≤.001, .02]	*n.a.*	*n.a.*	*n.a.*	*n.a.*	Not sign. $r = .07$ [–.04, .17]	Not sign.

Note: For the original results, the direct replication and the meta-analysis (Robustness Test 5), the entries show the effect sizes along the applied analysis (η_p^2, r) together with their 95% CI. For the Bayesian statistics (Robustness test 1–4), the ++/– is a descriptive summary of the positive/negative evidence for H1 across the relevant paired comparisons to approximate the main effects. For the PCA (Robustness Test 6), the number of components capturing the respective ERP and showing that significant effect is reported.
N refers to sample size of the analysis.
n.r. refers to not relevant: PCA includes spatial components and need to be considered as such.
n.a. refers to not applicable: Robustnesstests 1–4 were carried out using paired comparisons using Bayesian MLMs.
♦ Unlike in the original study, the PCA included the two factors outcome expectancy and valence, which showed a significant interaction.
[a] As the original study did not report an effect size, these are deduced from the reported F-statistics and *p*-values.
[b] For non-significant effects, no exact statistics were reported and these values reflect the largest effect size compatible with those.

Nalborczyk, Batailler, Loevenbruck, Vilain, & Bürkner, 2019). Goodness-of-fit was assessed via Bayesian R^2 (Gelman, Goodrich, Gabry, & Vehtari, 2019).

Posterior distributions of the model parameters were summarized using the mean and 95% credible interval (CI). Differences between conditions were calculated by computing the difference between posterior distributions of the respective conditions and summarized as above.

The existence of an effect was ascertained using the MAP-Based *p*-Value (*pMAP*), a Bayesian equivalent of the frequentist *p*-value (Mills, 2018). This index represents the odds of the posterior distribution of the parameter of interest against the point null hypothesis $H_0 = 0$ and, mathematically, corresponds to the density value at 0 divided by the density at the Maximum A Posteriori (MAP) (see also Makowski, Ben-Shachar, Chen, & Lüdecke, 2019). Following the current arbitrary *p*-value convention for Registered Reports in Cortex, we considered an effect statistically significant if $pMAP < .02$.

Two caveats of the *pMAP* should be noted here (Makowski et al., 2019). First, just like the frequentist *p*-value, *pMAP* allows us to assess the *presence* of an effect, not its *magnitude* or *practical importance*. Second, *pMAP* is sensitive only to the amount of evidence for the *alternative hypothesis* H_1, but it is *not* useful when assessing the amount of evidence in favor of the *null hypothesis* H_0. In our case, $pMAP < .02$ would suggest that the effect is statistically significant. However, $pMAP > .02$ would not allow us to conclude that the effect does not exist, only uncertainty about its existence (absence of evidence rather than evidence of absence).

To address these issues and increase the informativeness of our results, we additionally computed Bayes factors [BFs (Jeffreys, 1998; Kass & Raftery, 1995; Morey, Romeijn, & Rouder, 2016)]. BFs indicate *"the extent to which the data sway our relative belief from one hypothesis to the other"* (Etz & Vandekerckhove, 2018, p. 10). BFs were calculated as a Savage–Dickey density ratio (Dickey & Lientz, 1970; Wagenmakers, Lodewyckx, Kuriyal, & Grasman, 2010), i.e., comparing the marginal likelihoods of the alternative model against a model in which the tested parameter (i.e., the posterior distribution of condition differences) has been restricted to the point-null. We descriptively qualified BFs according to the arbitrary convention proposed by Kass and Raftery (1995): (i) $BF_{10} = 1$: *no* evidence in favor of H_1; (ii) $1 < BF_{10} < 3$: *weak* evidence in favor of H_1; (iii) $3 < BF_{10} < 20$: *positive* evidence in favor of H_1; (iv) $20 < BF_{10} < 150$: *strong* evidence in favor of H_1; (v) $BF_{10} > 150$: *very strong* evidence in favor of H_1. The reciprocal of BF_{10} (i.e., $BF_{01} = 1/BF_{10}$) indicated the corresponding evidence in favor of H_0.

As outlined, in our Bayesian multilevel models, we focused on estimating the posterior distributions of the parameters of interest rather than directly analyzing main effects and interactions. This approach provided a more nuanced understanding of the data by offering credible intervals for each parameter. As the model estimates the differences between specific conditions and their associated uncertainty, it is not designed to explicitly isolate and test interaction effects in the conventional sense.

2.7.3. *Meta-analysis (robustness test 5)*

Even though each replicating lab only had the statistical power to test the effect of expectancy on the P300, the data of each lab was separately subjected to the same ANOVAs described above (2.7.1). Then, a random effects meta-analysis was run where the effect sizes of valence (for the P300) or electrode (for the FRN/RewP), expectancy, and their interaction gathered in each replicating lab were combined. Following the method utilized previously in other large-scale replication projects (Ebersole, Mathur, & Baranski, 2020; Open Science Collaboration, 2015), as implemented in the *esc* package for R (Lüdecke, 2019), we converted partial eta squared to correlation coefficients. Given that eta represents a non-directional effect size, we established directionality by fitting linear regression models, analogous to ANOVAs, and derived the sign of the regression coefficients for each effect of interest. We utilized Fisher's z-transformed correlation coefficients, adjusted by the signs from the linear regressions, from each laboratory in our meta-analyses. The back-transformed correlation coefficients are presented and depicted in forest and funnel plots. The *metafor* package (Viechtbauer, 2010) for R was used for the meta-analysis.

2.7.4. *Temporospatial principal component analysis (PCA) (robustness test 6)*

The PCA factors were analyzed using the statistics function of the EP toolkit using all default parameters. The implemented ANOVAs are robust against violations of statistical assumptions. It included the following features: (i) trimmed means (cutting the outer quartiles) and winsorized covariances that protect against outliers; (ii) a bootstrapping routine (499,999 simulations, ran 11 times) that estimated the population distribution instead of assuming the normality of this distribution; and (iii) a Welch–James approximate degrees-of-freedom statistic that did not assume homogeneity of error variance (Dien, 2010b). The robust 2 × 3 repeated-measures ANOVA included the within-subject factors Valence and Expectancy. The *p*-value was adjusted with the Bonferroni correction for multiple comparisons. Follow-up tests for significant interactions were reported. In case the interaction effect needed a better characterization of its source, the EP Toolkit implements a Dunn-Šidák post-hoc test.

The PCA identified 31 temporal factors x 5 spatial factors based on the Scree plot, generating a total of 155 temporospatial factor combinations. Using an automated windowing step, the factors were further sifted through a predetermined minimum .5% threshold for accounted variance. The remaining PCA factors after the windowing step were then visually inspected for further analysis. Factors that only resembled the FRN/RewP and P300 components, based on canonical time course and scalp topography, were subjected to the robust ANOVA test.

Similar to the results from the main analyses above, we expected for the factor corresponding to the FRN/RewP a significant main effect for valence (more factor negativity for no-reward outcomes), but no effect of expectancy or their interaction. In contrast, for the factor corresponding to the P300 component, we expected a significant effect of expectancy

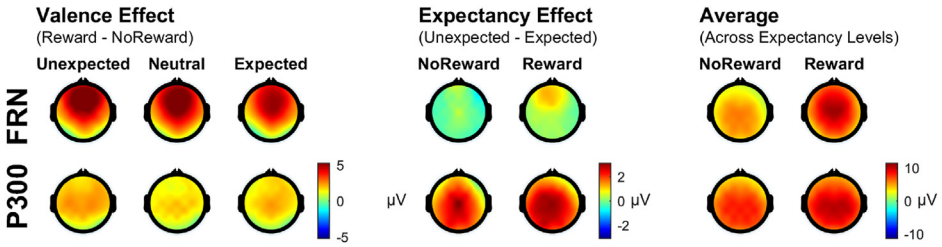

Fig. 2 – Topographical Plots of Valence and Expectancy Effects for FRN/RewP and P300 components. The FRN/RewP and P300 components were defined as the average amplitude in the 200–300 msec and 300–500 msec interval after outcome onset, respectively (preprocessing according to current standards as original preprocessing included only three channels).

(more factor positivity for unexpected outcomes), but no effect of valence or their interaction.

2.8. *Evaluation of the replication and robustness of effects*

The replication's success was mainly evaluated in the light of the outcomes of the ANOVAs (see 2.7.1) above: The FRN/RewP results were considered to be replicated successfully if the ANOVA showed a significant main effect of position (Fz > Pz), but no significant effect of expectancy or the interaction of expectancy and position. The P300 results were considered to be replicated successfully if the ANOVA showed a significant main effect of expectancy (unexpected > expected), but no significant effect of valence or the interaction of expectancy and position.

However, going beyond the mere replication of the original study, we provided preliminary robustness tests by comparing these results to the outcomes of the MLMs [see (2.7.2.) above] and a PCA [see (2.7.4) above]. If the MLMs and the PCA provided evidence for a similar pattern of results as (2.7.1), the effect was considered not only to be replicated but robust and, to some extent, independent of analytical choices. If the direct replication failed, i.e., significant effects were detected where none were expected, or expected effects did not reach significance, the MLMs were particularly important to conclude if the effects are present or not. If the pattern diverged across the robustness tests, possible sources of these discrepancies were discussed (with regard to preprocessing choices and/or quantification of the ERPs). Finally, the results of the MLM, (Robustness Test 1) were compared to the meta-analysis [see (2.7.3) above].

2.9. *Analysis of ratings*

The descriptive statistics for the subjective ratings pertaining to the attention paid to the cue and the feedback were reported (see Hajcak et al., 2005).

2.10. *Sharing of data and code*

Pre-processing steps were carried out using EEGLAB 2022.0 (Delorme & Makeig, 2004) implemented in MATLAB 2019, while statistical analyses were carried out in R (R-Core-Team, 2019). All experimental procedures, pre-processing scripts, analytical analyses are shared openly via the Open Science Framework (OSF, https://osf.io/2w9gy). All collected data will be made available online through GIN (https://gin.g-node.org/EEGManyLabs/EEGManyLabs_Replication_HajcakHolroyd2005). This study is a registered report, the preregistered stage 1 manuscript can be accessed at https://osf.io/db4rs.

3. Results

The results of the direct replication as well as all robustness tests are summarized in Table 1.

3.1. *Participants*

In total, 370 participants were tested across the thirteen labs ($M = 28.46$, $SD = 4.39$, *Range* = 21–37). All participants gave written informed consent. Sixty-six percent were women. Across all labs, 4 recordings were incomplete (e.g., computer failing, fainting of participants, battery issues, recording issues) and 5 participants were excluded since data from the mastoids were too noisy. For the original pre-processing, 2 participants had less than one trial after data cleaning. For the preprocessing according to current standards, 14 participants had less than eight trials after data cleaning. FRN/RewP or P300 peaks could not be detected in at least one condition for 66 participants (for the additional analyses where reward and no-reward outcomes were analyzed separately, this number increased to 108). Nineteen participants were excluded from analysis since they reported to not have paid attention to the cue. The final number of participants can be found in Table 1.

3.2. *Direct replication through ANOVAs*

The FRN/RewP component showed the expected frontocentral distribution peaking on average around 270 msec after feedback onset ($SD = 41$, *Range* = 203–495). The P300 component showed the expected central distribution peaking on average around 355 msec after feedback onset ($SD = 72$, *Range* = 203–598), see Fig. 2.

The direct replication, using the original preprocessing and peak values, revealed for the FRN/RewP component significant main effects of Expectancy (Table 2, row 1) and Location (Table 2, row 2) and an interaction between these two factors (Table 2, row 2). The FRN/RewP was largest for unexpected outcomes

Table 2 – Statistics of the direct replication.

	Effect	F	df_1	df_2	p	η_p^2	95 % CI η_p^2
FRN/RewP component at Fz, Cz, Pz							
1	Expectancy	28.34	1.79	546.6	≤ .001	.08	.05, .13
2	Location	154.16	1.50	460.17	≤ .001	.34	.28, .39
3	Expectancy × location	6.71	2.65	811.27	≤ .001	.02	.01, .04
FRN/RewP component at Fz							
4	Valence	514.64	1	262	≤ .001	.66	.60, .71
5	Expectancy	6.09	1.72	451.22	.004	.02	≤.01, .05
6	Valence × expectancy	10.70	1.81	474.82	≤ .001	.04	.01, .07
P300 component at Pz							
7	Valence	167.73	1	359	≤ .001	.32	.24, .39
8	Expectancy	215.18	1.74	625.04	≤ .001	.37	.32, .42
9	Valence × expectancy	1.60	1.72	617.4	.207	≤.01	≤.01, .02

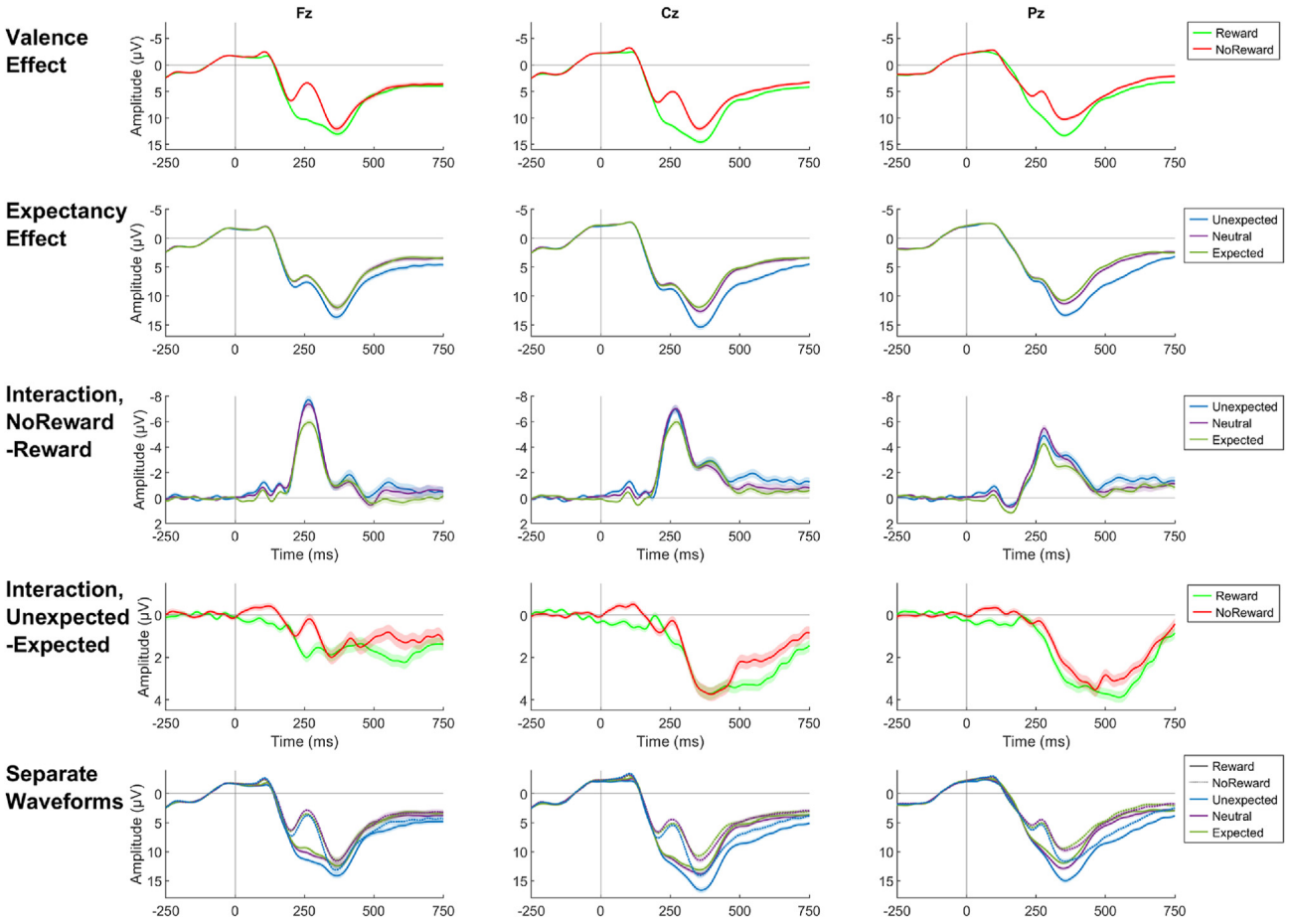

Fig. 3 – ERP Plots using the preprocessing following the original preprocessing at electrode sites Fz, Cz, and Pz, separately for the different conditions. Shaded Areas represent + - SEM.

($M_{Unexpected} = -9.65$ μV, $sd = 6.46$ *vs* $M_{Neutral} = -9.03$ μV, $sd = 5.6$ *vs* $M_{Expected} = -7.58$ μV, $sd = 4.57$) and at electrode Fz ($M_{CZ} = -9.38$ μV, $sd = 5.66$ *vs* $M_{FZ} = -9.62$ μV, $sd = 5.64$ *vs* $M_{PZ} = -7.27$ μV, $sd = 5.4$). The difference between unexpected and expected outcome was largest at Fz ($M_{CZ} = -1.79$ μV, $sd = 6.27$ *vs* $M_{FZ} = -2.75$ μV, $sd = 6.28$ *vs* $M_{PZ} = -1.81$ μV, $sd = 5.91$), see Figs. 2–4.[7]

To better understand the impact of expectancy and valence, we re-ran our analyses of the FRN/RewP component at Fz, treating reward and no-reward outcomes as separate

[7] When using the pre-registered quantification time window following the original study, similar results were obtained: Location [$F_{1.49,536.69} = 141.36$, $p \leq .001$, $\eta_p^2 = .28$, 95% CI (.23, .33)], Expectancy [$F_{1.79,641.32} = 44.11$, $p \leq .001$, $\eta_p^2 = .11$, 95% CI (.07, .15)], Interaction [$F_{2.8,1005.85} = 5.03$, $p = .002$, $\eta_p^2 = .01$, 95% CI (≤.001, .03)].

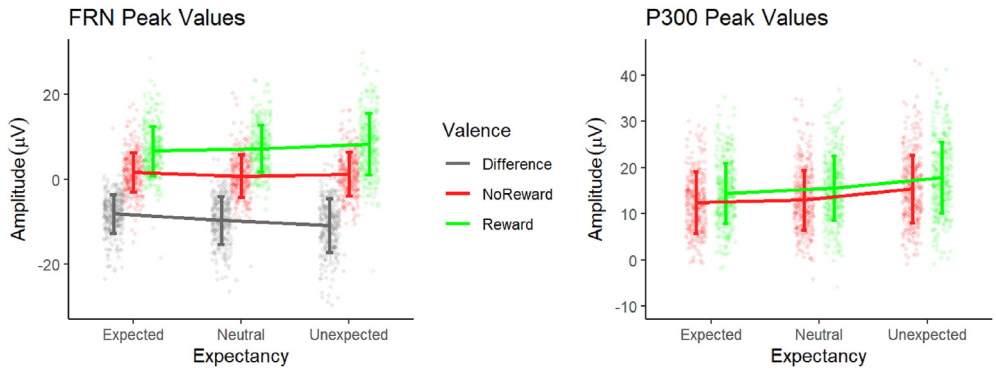

Fig. 4 – Mean ERP Values (+- SD) with individual data points for the direct replication, separately for each Expectancy and Valence level. The FRN component is shown at Fz, while the P300 component is shown at Pz.

conditions (i.e., without creating a difference wave prior to statistical analysis). This approach confirmed a significant main effect of Valence (Table 2, row 4), with more positive FRN/RewP amplitudes for reward compared to no-reward outcomes (M_{Reward} = 7.4 μV, sd = 6.27 *vs* $M_{NoReward}$ = 1.18 μV, sd = 4.97). The main effect of Expectancy was also significant (Table 2, row 5), with more positive values for unexpected compared to expected outcomes ($M_{Unexpected}$ = 4.67 μV, sd = 7.22 *vs* $M_{Neutral}$ = 3.88 μV, sd = 6.2 *vs* $M_{Expected}$ = 4.09 μV, sd = 5.81). Additionally, the interaction between Valence and Expectancy was significant (Table 2, row 6), see Fig. 4. The difference between unexpected and expected outcomes was largest (most positive) and only significant for reward outcomes ($M_{NoReward}$ = −.34 μV, sd = 4.46, p = .18, d = −.08 *vs* M_{Reward} = 1.25 μV, sd = 4.84, p < .001, d = .26).[8]

For the P300 component, the main effect of Valence was significant (Table 2, row 7), as was the main effect of Expectancy (Table 2, row 8). However, the interaction between these two factors was not significant (Table 2, row 9). P300 values were largest for reward compared to no-reward outcomes (M_{Reward} = 16 μV, sd = 7.21 *vs* $M_{NoReward}$ = 13.64 μV, sd = 6.93), and for unexpected compared to expected outcomes ($M_{Unexpected}$ = 16.66 μV, sd = 7.59 *vs* $M_{Neutral}$ = 14.29 μV, sd = 6.86 *vs* $M_{Expected}$ = 13.51 μV, sd = 6.65), see Figs. 3 and 4.

3.3. *Bayesian MLMs (robustness test 1–4)*

As we aimed to replicate a null effect, we included Bayesian statistics to allow testing for the absence of specific effects (i.e., Valence for P300, Expectancy for FRN). Moreover, to better control for unknown Lab effects in this multi-lab sample, we carried out these analyses using multilevel linear models. The results of the different Bayesian MLMs aligned with the results of the direct replication based on ANOVAs (see above). These robustness tests varied the preprocessing (original versus current standard) as well as the quantification method (peak versus mean amplitude). The detailed BF are reported in Tables 3 and 4. For more details, all comparisons, and parameters, please consult the supplementary section 8.3.

For the FRN/RewP component, we found positive evidence for the effect of Location across all preprocessing and quantification methods (Table 3, row 4/5, robustness test 1–4). In comparison, the interaction between Expectancy and Location was dependent on the quantification choice: When using a peak amplitude as quantification, there was positive to strong evidence for an effect of Expectancy at all electrodes (Table 3, row 1/2, robustness test 1/3). When using the mean amplitude as quantification (Table 3, row 1/2, robustness test 2/4), the Expectancy effect was only weakly supported at Fz (weak evidence for H_1), but not at Pz (positive evidence for H_0), suggesting that this effect could be robustly detected at electrode Fz, but not at Pz.

When we assessed the FRN/RewP component separately for reward and no-reward outcomes, we found strong evidence for the expected main effect of Valence, which was robustly found across all preprocessing and quantification methods (Table 3, row 7/8, robustness test 1–4). Regarding Expectancy, the type of quantification method used actually influenced the results: When using a mean quantification, we found positive evidence for an effect of Expectancy for reward outcomes (Table 3, row 5, robustness test 2/4), but not for no-reward outcomes (positive evidence for H_0, Table 3, row 6, robustness test 2/4). However, when using a peak quantification, the results were dependent on the preprocessing methods: For the original preprocessing, there was positive evidence for an effect of Expectancy for reward outcomes (Table 3, row 5, robustness test 1), but not for no-reward outcomes (weak evidence for H_0, Table 3, row 6, robustness test 1). In contrast, for the preprocessing according to current standards, the opposite pattern emerged: there was weak evidence against an effect of Expectancy for reward outcomes (Table 3, row 5, robustness test 3), but positive evidence for an Expectancy effect for no-reward outcomes (Table 3, row 6, robustness test 3). These results suggest that the mean quantification is probably better suited than the peak scoring to capture a robust effect of Expectancy for reward outcomes, given that they often do not elicit a clear peak (see last panel in Fig. 3).

[8] When using the pre-registered time window for quantifying the peak, the interaction was not significant: Valence [$F_{1,342}$ = 309.47, $p \leq .001$, η_p^2 = .48, 95% CI (.4, .54)], Expectancy [$F_{1.9,650.98}$ = 5.32, p = .006, η_p^2 = .02, 95% CI (≤.001, .04)], Interaction [$F_{1.9,650.1}$ = 1.92, p = .149, η_p^2 = .01, 95% CI (≤.001, .02)]. Since determining a negative peak for reward outcomes can be difficult, using a mean window approach could provide a solution to score this component. However, using this alternative scoring method, the results were similar, see Supplementary Table 3.

Table 3 – Bayes factor analysis for the different robustness tests and FRN/RewP component.

			Original		Current Standards	
			Peak (RobTest 1)	Mean (RobTest 2)	Peak (RobTest 3)	Mean (RobTest 4)
1	*Expectancy at Fz*	Unexpected Diff Fz - Expected Diff Fz	<.001* BF = 12.5 ++	.004* BF = 1.91 +	<.001* BF = 23.15 +++	.004* BF = 1.42 +
2	*Expectancy at Pz*	Unexpected Diff Pz - Expected Diff Pz	<.001* BF = 5.4 ++	.506 BF = −3.17 - -	<.001* BF = 5.98 ++	.811 BF = −3.74 - -
3	*Location for Unexpected*	Unexpected Diff Fz - Unexpected Diff Pz	<.001* BF = 13.19 ++	<.001* BF = 20.2 +++	<.001* BF = 15.79 ++	<.001* BF = 15.61 ++
4	*Location for Expected*	Expected Diff Fz - Expected Diff Pz	<.001* BF = 7.67 ++	<.001* BF = 12 ++	<.001* BF = 10.42 ++	<.001* BF = 12.26 ++
5	*Expectancy for Reward*	Unexpected reward Fz- expected reward Fz	<.001* BF = 4.64 ++[a]	<.001* BF = 11.83 ++	.442 BF = −2.87 –	<.001* BF = 6.79 ++
6	*Expectancy for NoReward*	Unexpected NoReward Fz - expected NoReward Fz	.369 BF = −2.92 -	.023* BF = −.42	<.001* BF = 3.45 ++[b]	.696 BF = −3.8 - -
7	*Valence for Unexpected*	Unexpected reward Fz- unexpected NoReward Fz	<.001* BF = 38.94 +++	<.001* BF = 41.94 +++	<.001* BF = 24.87 +++	<.001* BF = 26.36 +++
8	*Valence for Expected*	Expected reward Fz- Expected NoReward Fz	<.001* BF = 23.99 +++	<.001* BF = 33.6 +++	<.001* BF = 26.37 +++	<.001* BF = 25.21 +++

Note. First value refers to the p-Map value, an asterisk indicating a significant effect, BF = logarithmic Bayes Factor (*BF*) of H1. +++/– - - indicates strong evidence in favor of/against H1. ++/– - positive evidence. ± weak evidence. Diff = Difference NoReward – Reward outcome. When using the original pre-registered quantification time window following the original study, similar results were obtained unless specified otherwise in the footnotes. RobTest = Robustness Test.

[a] The pre-registered quantification showed an opposite effect: .092; BF = −.94.

[b] The pre-registered quantification showed an opposite effect: .239; BF = −2.34.

Table 4 – Bayes factor analysis for the different robustness tests of P300 component at Pz.

			Original		Current Standards	
			Peak	Mean	Peak	Mean
1	*Expectancy for Reward*	Unexpected reward - expected reward	<.001* BF = 28.76 +++	<.001* BF = 26.83 +++	<.001* BF = 27.88 +++	<.001* BF = 23.07 +++
2	*Expectancy for NoReward*	Unexpected NoReward - expected NoReward	<.001* BF = 16.41 ++	<.001* BF = 17.94 ++	<.001* BF = 21.49 +++	<.001* BF = 14.84 ++
3	*Valence for Unexpected*	Unexpected reward - unexpected NoReward	<.001* BF = 8.85 ++	<.001* BF = 7.84 ++	<.001* BF = 7.84 ++	.001* BF = 5.24 ++
4	*Valence for Expected*	Expected reward - expected NoReward	<.001* BF = 8.07 ++	<.001* BF = 4.71 ++	<.001* BF = 7.31 ++	<.001* BF = 4.72 ++

Note. First value refers to the p-Map value, an asterisk indicating a significant effect, BF = BF_{10} = logarithmic Bayes Factor (*BF*) of H1. +++ indicates strong evidence in favor of H1. ++ positive evidence.

For the P300 component, we found positive to strong evidence for the main effect of Expectancy (BF_{10} = 14.84–28.76, $p < .001$) across all valence types, but also positive evidence for the main effect of Valence (BF_{10} = 4.72–8.85, $p < .001$) across all expectancy types. This pattern was robustly found across all preprocessing and quantification methods.

3.4. *Meta-analysis (robustness test 5)*

For the meta-analysis, forest and funnel plots were computed. We report and plot median and distribution of the weighted effect sizes, 95% confidence intervals, and the number of labs successfully replicating the original effect.

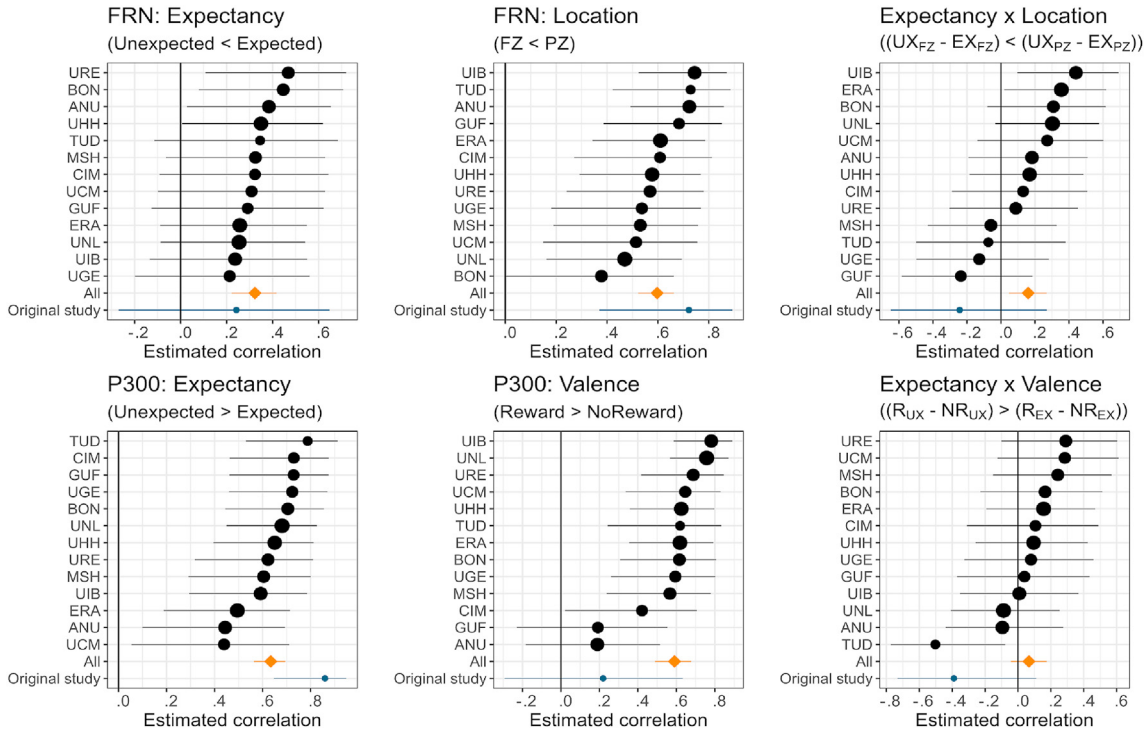

Fig. 5 – Forest Plots. Correlation coefficients (converted from partial eta squared) for various laboratories. Circle size corresponds to sample size, indicating the robustness of findings in each lab. The orange square shows the meta-analytically aggregated score. The blue circle shows the effect size from Hajcak et al. (2005) (derived from the reported F statistic). Correlation coefficients are coded in such a way that positive values are evidence in favor of the expected effect under consideration (noted in the caption). UX = Unexpected. EX = Expected. R = Reward. NR = NoReward. Please note that the FRN is a negative potential, hence a smaller (more negative) amplitude shows a stronger effect. ANU = Australian National University, Australia. BON = Bond University, Australia. CIM = Central Institute of Mental Health Mannheim, Germany. ERA = Erasmus University Rotterdam, The Netherlands. GUF = Goethe University Frankfurt am Main, Germany. MSH = Medical School Hamburg, Germany. TUD = Technical University Dresden, Germany. UCM = CINPSI Neurocog UCMaule, Chile. UGE = Ghent University, Belgium. UHH = University Hamburg, Germany. UIB = University of Bergen, Norway. UNL = Leiden University, The Netherlands. URE = University of Regensburg, Germany

The meta-analysis on the FRN/RewP showed significant main effects of Expectancy [$r = .32$, $p < .001$, 95% CI (.22, .42), $Q(12) = 2.3$, $p = .999$, $I^2 = .0\%$] and Location [$r = .60$, $p < .001$, 95% CI (.52, .66), $Q(12) = 8.6$, $p = .733$, $I^2 = .0\%$], as well as interaction between these two factors [$r = .16$, $p = .005$, 95% CI (.05, .27), $Q(12) = 13.1$, $p = .359$, $I^2 = 6.7\%$]. The large main effect of Location was robustly detected across all labs except one (i.e., 12 out of 13 labs showed a significant effect in the expected direction, with the FRN/RewP the largest at Fz > Cz > Pz). While all labs showed that the FRN/RewP was numerically larger for unexpected compared to expected outcomes, this relatively small effect was only significant in a few of them (i.e., 4 out of 13 labs showed it). Moreover, the interaction between Location and Expectancy was only significant in 2 out of 13 labs, and some of them showed even opposite effects (see Fig. 5 and Supplementary Fig. 4).

The meta-analysis on the P300 showed significant main effects of Expectancy [$r = .63$, $p < .001$, 95% CI (.56, .69), $Q(12) = 9.5$, $p = .661$, $I^2 = .0\%$] and Valence [$r = .59$, $p < .001$, 95% CI (.49, .68), $Q(12) = 20.3$, $p = .062$, $I^2 = 41.3\%$], while the interaction between them was not significant [$r = .07$, $p = .23$, 95% CI (−.04, .17), $Q(12) = 11.9$, $p = .457$, $I^2 = .0\%$]. The large main effect of Expectancy was robustly detected across all labs (i.e., all 13 labs showed a significant effect in the expected direction, with the P300 being larger for unexpected compared to expected outcomes). Similarly, the large main effect of Valence was robustly detected in a majority of labs (i.e., 11 out of 13 labs showed a significant effect in the expected direction, with the P300 being larger for reward compared to no-reward outcomes). In comparison, the interaction between Valence and Expectancy was only significant in one lab, where the effect was reversed compared to most other labs (see Fig. 5 and Supplementary Fig. 4).

For all effects, the aggregated effect sizes across all labs fell within the estimated confidence interval of the original sample, which were quite wide. However, for the previously reported significant effects (i.e., main effect of Location for the FRN, main effect of Expectancy for the P300 component), the aggregated effect sizes were smaller than the ones reported in

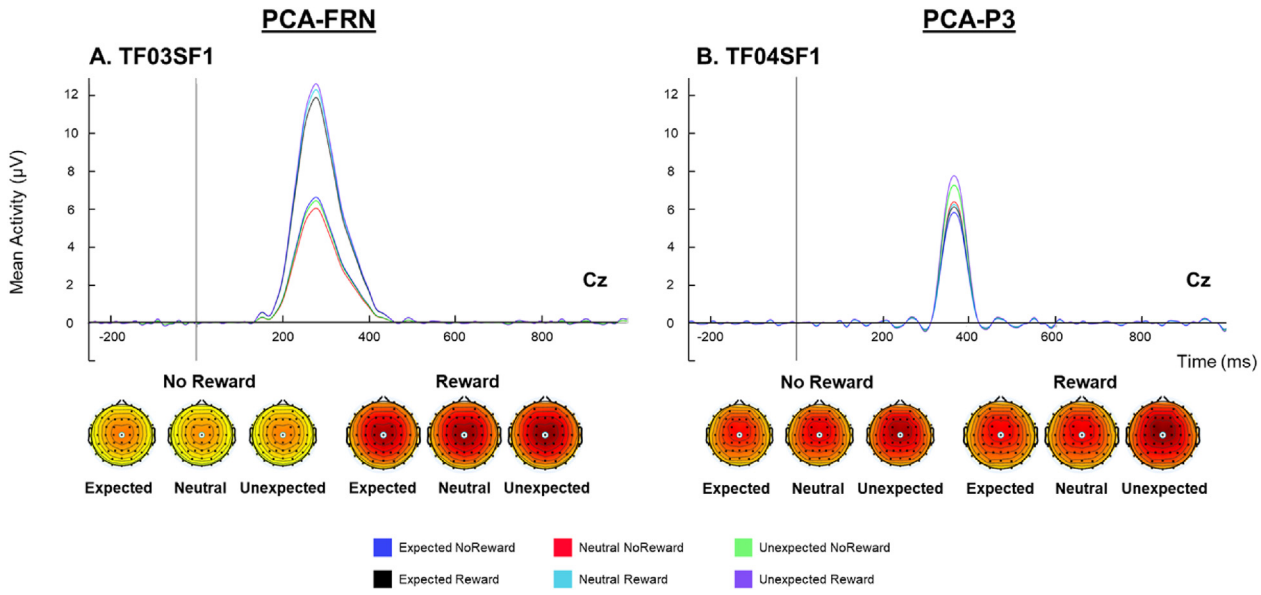

Fig. 6 – Activation over time and topographical plots for the PCA factors corresponding to the RewP and P300 components: (A) The factor TF03SF1 (corresponding to the RewP) peaks at 276 msec at the central area. (B) The factor TF04SF1 (corresponding to the P3) peaks at 366 msec at the central area.

the original study. In comparison, for the previously reported non-significant effects (i.e., main effect of Expectancy for the FRN, main effect of Valence for the P300 component), the aggregated effect sizes were larger than the ones reported in that study.

3.5. *Temporospatial principal component analysis (robustness test 6)*

For the PCA analysis, the data of 230 participants coming from 8 labs could be used (the other ones did not include the relevant channels). Based on the time course and scalp distribution, four temporospatial factors were identified that closely corresponded to the FRN/RewP and P300 components. One of these factors captured the spatiotemporal variations of the FRN/RewP component, while the remaining ones captured that of the P300 component (see Fig. 6).

The PCA factor TF03SF1, corresponding to the FRN/RewP component, exhibited a peak latency at 276 msec over the central area (maximal at Cz). The robust ANOVA revealed a significant main effect of Valence ($T_{WJt}/c_{1.0,198.0} = 271.02$, $p < .001$, MSe = 38.75), exhibiting a larger positivity for reward than no-reward outcomes ($M_{\text{Reward}} = 12.08$ μV, $sd = .04$ *vs* $M_{\text{NoReward}} = 6.15$ μV, $SD = .03$). In contrast, the main effect of Expectancy was not significant ($T_{WJt}/c_{2.0,176.0} = 1.67$, $p = .189$, MSe = 13.01). Moreover, the interaction between them was significant ($T_{WJt}/c_{2.0,176.0} = 6.16$, $p < .002$, MSe = 5.28), with this PCA factor differentiating better reward from no-reward outcomes for unexpected compared to expected outcomes. The positivity was larger for unexpected reward compared to expected rewards, while the opposite pattern was true for no-reward outcomes ($M_{\text{Unexpected Reward}} = 12.38$ μV, $sd = .04$ *vs* $M_{\text{Expected Reward}} = 11.66$ μV, $sd = .03$ *vs* $M_{\text{Unexpected NoReward}} = 6.29$ μV, $sd = .03$ *vs* $M_{\text{Expected NoReward}} = 6.35$ μV, $sd = .03$).

The PCA factor TF04SF1, which corresponded to the P300 component, exhibited a peak latency at 366 msec over the central area (maximal at Cz). Although the robust ANOVA revealed no significant main effect of Valence ($T_{WJt}/c_{1.0,198.0} = 1.03$, $p = .311$, $MSe = 15.76$), the effect of Expectancy reached significance ($T_{WJt}/c_{2.0,176.0} = 37.78$, $p < .001$, $MSe = 7.91$), exhibiting the largest positivity for the unexpected outcomes ($M_{\text{Unexpected}} = 7.50$ μV, $sd = .02$ *vs* $M_{\text{Expected}} = 5.85$ μV, $sd = .02$ *vs* $M_{\text{Neutral}} = 6.22$ μV, $sd = .02$). The interaction between Valence and Expectancy was not significant ($T_{WJt}/c_{2.0,176.0} = 3.29$, $p = .0389$, $MSe = 4.98$).

Two additional PCA factors could be related to the P300 component and are described in the supplementary material since their latency was later than the average peak of this ERP component after 400 msec, although still falling within the time interval of the P300 component according to some models; see (Polich, 2007). These two additional factors were both significantly modulated by Valence, while only one of them additionally showed a significant main effect of Expectancy. None of them showed a significant interaction effect, see Supplementary section 4.

4. Discussion

In this study, we directly replicated Hajcak et al. (2005) as part of the #EEGManyLabs project (Pavlov et al., 2021). We examined the sensitivity of the FRN and P300 components to outcome valence and expectancy using a simple guessing task. Hajcak et al. (2005) found that the FRN distinguished reward from no-reward outcomes regardless of expectancy, while the P300 differentiated unexpected from expected outcomes, independent of valence. This led to a two-stage model of feedback processing: valence is processed at the FRN level,

while expectancy mostly influences the P300. Our replication, with an unprecedented sample size of up to 360 participants across 13 laboratories worldwide, partly corroborates these findings but contradicts this simple two-stage model. Unlike Hajcak et al. (2005), we found that both the FRN/RewP and the P300 components were significantly modulated by both outcome expectancy and valence. In addition to the exact replication using the same EEG pre-processing and scoring methods, we conducted several robustness tests, a meta-analysis including laboratory as a variable, and a PCA. These methods consistently confirmed our findings for the FRN/RewP and P300 components.

The original study reported significant effects of expectancy only for the P300 (valence was not tested for the FRN) while, for the FRN/RewP and P300 components, it reported null-effects of expectancy and valence, respectively. Therefore, we aimed for a large sample size in our study to detect small but relevant effects (Paul et al., 2020). In comparison, the original study had a modest sample size ($n = 17$), common in neurophysiology at that time (Picton et al., 2000). However, with a well-powered sample, we observed that expectancy had a small to moderate effect on the FRN/RewP component. With only 17 participants, detecting a similar significant effect would have been rather unlikely given that the statistical power to detect an effect of $\eta p^2 = .08$ with 17 participants is only around 40%. Consequently, the previously reported "insensitivity" of the FRN/RewP to expectancy was most likely a false negative finding, emphasizing that absence of evidence does not equate to evidence of absence.

Our study shows instead that the FRN/RewP is robustly modulated by expectancy, albeit to a lesser extent than by valence, and to a lesser extent than the P300 component. This result challenges the view that the FRN/RewP solely represents binary outcome valence processing (Hajcak, Moser, Holroyd, & Simons, 2006; Kujawa, Smith, Luhmann, & Hajcak, 2013). To explain it, the reinforcement learning framework provides a more plausible model, according to which the FRN/RewP captures activity in a dopaminergic fronto-striatal network where both valence and expectancy are processed concurrently (Holroyd & Coles, 2002; Ullsperger, Fischer, et al., 2014). At the same time, it remains to be determined which role the P300 component could play in this ERN-RL framework. Moreover, using a guessing task rather than a learning task, our replication indicates that reinforcement learning (see Sutton & Barto, 1998) is not required to produce these ERP effects. This implies that the cue information about reward probability was sufficient to influence feedback processing. These findings support the idea that subjective expectancy, rather than reward probability maximization, could actually drive these FRN/RewP amplitude changes (Walentowska, Severo, Moors, & Pourtois, 2019). Notably, other ERP findings suggest that even when reward probabilities were held constant, the FRN/RewP amplitudes could vary depending if reward probabilities were perceived as better or worse than previously experienced (Mushtaq, Stoet, Bland, & Schaefer, 2013). In this vein, later results from Hajcak et al. (2007) are also informative: using the same guessing task as used here, the authors asked participants about their reward expectations either before or after the information cue. They found that the FRN/RewP component was sensitive to the expectancy manipulation only when participants rated their expectations after the presentation of the information cue, suggesting that this effect depends on the close coupling of (subjective) predictions and outcomes. In the current study, our results show that participants reported paying attention to both the reward probability cue and the feedback, possibly indicating they sought to maximize reward, even though outcomes were unrelated to any behavioral strategy. Thus, it is possible that the effect of (objective) expectancy on the FRN/RewP component becomes larger the more explicitly subjective expectations align with manipulated variables. We suggest that a potentially fruitful line of future study could be to directly compare the impact of subjective and objective reward probabilities.

The second discrepancy worth-mentioning between our results and the original study is that also the P300 component is robustly modulated by both valence and expectancy, and not only expectancy as postulated by the two-stage model outlined above. While expectancy's influence on the P300 is well-documented across various domains (Polich, 2007), valence effects on this ERP component during performance monitoring are less consistent (Ullsperger, Fischer, et al., 2014). Some have even argued that it is blind to outcome valence (Hajcak et al., 2006; Kujawa et al., 2013). Our replication clearly demonstrates that the P300 amplitude is significantly modulated by outcome valence, being larger for reward than no-reward outcomes. This suggests that its amplitude variations likely reflect a motivational effect (Nieuwenhuis, Aston-Jones, & Cohen, 2005; San Martín, 2012). Although the specific processes underlying the P300 remain unclear (Verleger, 2020), our findings indicate that this component is enhanced for favorable outcomes, possibly reflecting approach motivation (Harmon-Jones, Harmon-Jones, & Price, 2013). This aligns with a study on social feedback processing, which also found enhanced P300 activity for favorable, expected outcomes (Van der Veen, van der Molen, Sahibdin, & Franken, 2014). Nevertheless, since we did not include a loss condition (only no-reward versus reward), it remains an open question how these effects compare to unfavorable outcomes. Additional EEG research is needed to address this question and directly assess the extent to which motivationally relevant or meaningful outcomes could influence the P300 component (Glazer et al., 2018; San Martín, 2012; Stewardson & Sambrook, 2020). In this context, it appears important to clarify whether relevance, memory updating, or perhaps another cognitive or emotional process drives this neurophysiological effect.

Given that the FRN/RewP and P300 components rapidly follow each other, overlapping effects of expectancy and valence may be artificially inflated. This makes our additional PCA analysis particularly important, as both components clearly distinguish between reward and no-reward outcomes. The PCA allowed us to disentangle successive and overlapping ERP components (Dien, 2012). While carefully controlling for the influence of other spatiotemporal components, the PCA revealed that the valence effect at the FRN/RewP level was distinct and independent from that of the P300. Our findings therefore suggest that valence processing is multifaceted and influences both the FRN/RewP and P300, which likely capture distinct facets of it. Speculatively, the FRN/RewP may reflect

early hedonic feedback processing ("liking"), while the subsequent P300 may represent its motivational value ("wanting"), consistent with theoretical frameworks that decompose brain pleasure mechanisms into liking and wanting components (see Berridge, Schmeichel, & España, 2012). A related effect could be shown when considering saturation to (e.g., food-related) rewards, which affected only the P300 component (Huverman et al., 2021). Even more speculatively, a similar division might be applied to the processing of outcome expectancy because the PCA analysis confirmed distinct and independent effects of it on the FRN/RewP and the P300. This implies that, similar to valence, expectancy processing during performance monitoring could involve multiple components. While the PCA effectively disentangles these components, the functional significance of these successive expectancy (as well as valence) effects remains challenging to grasp. Because it could not be addressed directly with the current ERP analyses, future studies are needed to shed light on it and eventually improve or amend current theoretical models of performance monitoring. Moreover, at the methodological level, since we used visual inspection to select the main PCA factors corresponding to the FRN/RewP and P300, we believe that replicability could be enhanced in the future if automated procedures or algorithms would be used to carry out this selection.

Based on our results, one could hypothesize that the FRN/RewP reflects a "crude" reward prediction error in a midbrain-dependent fronto-striatal loop (Schultz, 2016). Consistent with this hypothesis, single-trial ERP studies have shown that both FRN/RewP and P300 are influenced by prediction errors but this influence varies depending on the context (Hoy et al., 2021; Weber & Bellebaum, 2024). Interestingly, even cerebellar output is crucial for learning from action outcomes, as disruptions in cerebellar function impair the FRN/RewP component (Huvermann et al., 2024). The fronto-striatal reward prediction error signal is then being relayed to areas such as the hippocampus or entorhinal cortex involved in memory or reinforcement learning, potentially giving rise to the P300 component (Soltani & Knight, 2000).

Besides the theoretical implications and better functional delineation of the FRN/RewP and P300 components during performance monitoring, our replication highlights their sensitivity to different EEG data processing methods. Embedded in the #EEGManyLabs project, our replication aimed to address methodological limitations of previous EEG research, such as small sample sizes and lack of preregistration (Pavlov et al., 2021). We performed an almost exact replication with sufficient statistical power and supplemented it with robustness tests, including a PCA and a meta-analysis. Overall, these analyses largely concurred on a robust amplitude modulation of the FRN/RewP by expectancy. Nonetheless, there are some differences between them worth mentioning, as they might explain some of the discrepant results reported earlier in the literature. First, the peak-scoring method showed the expectancy effect of the FRN/RewP most robustly (see also Paul et al., 2020). In contrast, the mean-scoring method yielded more topographical precision as it was confined to Fz, where this component is expected to reach its maximum amplitude given its intracranial generators are presumably located in the dorsal medial prefrontal cortex (Hauser et al., 2014). At the same time, the liability of the peak-scoring to noise (see Luck, 2005) led to a larger SME as a measure of within-subject variability across trials (see Supplementary Table 1). Fortunately, the pre-processing strategies did not have a large influence on the pattern of results. However, they were aimed to be as similar as possible, with the largest difference concerning the correction of ocular artifacts. Other important methodological choices, e.g., the choice of reference or the time-window used to define the ERP components, were not investigated, but are probably worth exploring further in future EEG studies. Multiverse analyses, which systematically explore the influence of methodological choices across multiple analytical pipelines, could also provide valuable insights into these questions (see Clayson, 2024).

Aligned with the ERN-RL framework, the FRN/RewP difference was found to be larger for unexpected events, with a stronger response observed for unexpected versus expected outcomes. Alongside the traditional difference-wave approach (reward versus no-reward) used to assess the influence of expectancy effects on the FRN/RewP component, we further assessed the components separately for reward and no-reward outcomes to investigate whether the expectancy effect was driven by one type of outcome, providing additional insights into its mechanism. Prior research suggests that the RewP (in response to reward) can serve as the counterpart to the FRN (in response no-reward or loss) with opposite polarity (Kappenman, Farrens, Zhang, Stewart, & Luck, 2021; Proudfit, 2015). Although difference-waves are used to analyze these effects in this framework, the RewP and FRN's different spatio-temporal properties may obscure distinct modulatory effects of expectancy and valence for reward and no-reward outcome, respectively (Gheza et al., 2018). Our findings strongly support this distinction, showing that expectancy effects were stronger and more robust for rewards than no-rewards, indicating that the RewP/FRN component was boosted in particular in response to unexpected reward outcomes, while the RewP/FRN component was not influenced by the expectancy of no-reward. Bayes factors provided evidence for the absence of an expectancy effect for no-rewards (when using mean-scoring, which is more suitable to define the RewP in the absence of a clear peak). This finding is not surprising, as rewards were more relevant to participants in the current task, while no-rewards were presumably less informative. Therefore, the relevance or informativeness of feedback, which may be closely linked to the participants' curiosity and motivation for information-seeking, should be considered in future study designs (Kidd & Hayden, 2015). It is possible that expectancy impacts feedback processing at the level of the FRN/RewP component only when the feedback is meaningful to the participant (Walentowska, Paul, Severo, Moors, & Pourtois, 2018). Additionally, prior research using the absence of aversive outcomes as positive outcomes (i.e., "rewarding") has often failed to find that the RewP/FRN component (defined as a difference score) is larger for unexpected than expected feedback, highlighting the importance of outcome type in determining expectancy effects (e.g., Talmi, Atkinson, & El-Deredy, 2013; Bauer et al., 2024). More broadly, these findings suggest that using difference waves may not be ideal for examining the modulatory effects of expectancy on early performance monitoring ERP components, as this method can

obscure potentially asymmetrical effects on the overlapping RewP and FRN components.

At the methodological level, our series of robustness tests allowed us to compare various analytical approaches for data collected across multiple labs. These approaches included an ANOVA on the entire dataset without accounting for potential differences between labs, a Bayesian multilevel model (MLM) with random intercepts and slopes for each lab, and a random-effects meta-analysis across all 13 labs, which accounted for the lab effect and estimated heterogeneity. Importantly, accounting for the differences between labs did not significantly alter the effect sizes (e.g., $\eta_p^2 = .08$ in ANOVA *vs* $\eta_p^2 = .10$ in the meta-analysis for the FRN expectancy effect, and $\eta_p^2 = .32$ in ANOVA *vs* $\eta_p^2 = .35$ in the meta-analysis for the P300 valence effect). The use of Bayesian MLM, compared to the meta-analysis, did not noticeably affect the results either, at least for the peak scoring approach.

We collected data from 13 laboratories across seven countries on three continents. Despite noticeable differences in hardware and potential variations in local populations, the overall effect, exemplified by the expectancy effect on FRN, remained consistent. Strikingly, conventional heterogeneity estimates indicated no variability. This result is important because it indirectly suggests that the effects in this task are quite robust. Notably, the effect size for the expectancy effect on FRN in the ANOVA in our replication turned out to be exactly the same as our estimate of the effect size in the original study. Moreover, our effect sizes for all effects of interest fell within the confidence interval of the original study. The diverse nature of our sample, along with the absence of variability in the results, further supports the robustness of the observed effects.

In addition to these methodological insights, our study provides some recommendations for future EEG studies on the FRN/RewP and P300 components regarding sample size estimation, should the same task be used (see Supplementary Table 8). In short, for each ERP component and effect under consideration (i.e., Location, Valence, Expectancy, or their interaction), we have used the effect size reported in this study and computed a sample size estimation. We believe this information could be valuable to researchers working on performance monitoring. Moreover, because the data and scripts of this replication are publicly available, they could easily be used in future studies to perform additional analyses (e.g., time-frequency decompositions). Similarly, our data could be pooled together with other EEG data sets available in the literature and contribute to mega-studies or mega-analyses (Costafreda, 2009). These efforts would have the potential to provide a more precise estimate of the effect size under scrutiny or to identify possible moderators (e.g., learning, different feedback types or stimuli used). Additionally, since we collected some personality questionnaires, pooling with existing data could allow the investigation of interindividual differences in feedback processing. Furthermore, this study highlights the broader significance of replication studies in advancing psychological theories. Replication not only validates previous findings but also refines and challenges existing theories in cognitive neuroscience, ensuring that they are robust and generalizable. Thus, we can uncover nuances and inconsistencies that lead to a deeper understanding of psychological processes.

In conclusion, our replication underscores the complexity of feedback processing in the brain and reveals several advantages of a large and collaborative EEG data collection to gain novel insights. Crucially, we found no support of the two-stage model of feedback processing. Instead, our new results suggest that the premises of the ERN-RL model might also include the P300 component, besides the FRN/RewP. In light of them, we suggest an integrated model of evaluative feedback processing where both valence and expectancy are concurrently processed across multiple stages. Furthermore, we advocate for more stringent methods, including the use of preregistration and the consideration of effect sizes to determine appropriate sample sizes, and hope the present replication and associated resources could be used to guide future research on the electrophysiological correlates of feedback processing.

Scientific transparency statement

The responses provided regarding Pre-registration of Study Procedures and Pre-registration of Analysis Plans may not yet be compliant with the transparency guidelines. This is likely to result in delays and additional inquiries unless changes are made to the manuscript and/or the Scientific Transparency Report.
DATA: All raw and processed data supporting this research are publicly available: https://gin.g-node.org/EEGManyLabs/EEGManyLabs_Replication_HajcakHolroyd2005, https://osf.io/t9g2e/.
CODE: All analysis code supporting this research is publicly available: https://osf.io/ftgm4/, https://osf.io/xt4c6/.
MATERIALS: All study materials supporting this research are publicly available: https://osf.io/vuy2q/.
DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.
PRE-REGISTRATION: At least part of the study procedures was pre-registered in a time-stamped, institutional registry prior to the research being conducted: https://osf.io/db4rs At least part of the analysis plans was pre-registered in a time-stamped, institutional registry prior to the research being conducted: https://osf.io/db4rs The analyses that were undertaken deviated from the preregistered analysis plans. All such deviations are fully disclosed in the manuscript.
For full details, see the *Scientific Transparency Report* in the supplementary data to the online version of this article.

CRediT authorship contribution statement

Katharina Paul: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Douglas J. Angus:** Writing – review & editing, Supervision. **Florian Bublatzky:** Writing – review & editing, Supervision, Funding acquisition. **Raoul Dietrich:** Writing –

review & editing, Methodology, Investigation. **Tanja Endrass:** Writing – review & editing, Supervision. **Lisa-Marie Greenwood:** Writing – review & editing, Supervision, Investigation. **Greg Hajcak:** Writing – review & editing. **Bradley N. Jack:** Writing – review & editing, Supervision, Investigation, Funding acquisition. **Sebastian P. Korinth:** Investigation. **Leon O.H. Kroczek:** Investigation, Supervision, Writing – review & editing. **Boris Lucero:** Investigation, Funding acquisition. **Annakarina Mundorf:** Writing – review & editing, Investigation. **Sophie Nolden:** Writing – review & editing, Supervision. **Jutta Peterburs:** Writing – review & editing, Supervision, Methodology, Funding acquisition. **Daniela M. Pfabigan:** Writing – review & editing, Supervision, Software, Methodology, Funding acquisition. **Antonio Schettino:** Writing – review & editing, Validation, Software, Methodology, Formal analysis, Data curation. **Mario C. Severo:** Writing – review & editing, Visualization, Validation, Formal analysis. **Yee Lee Shing:** Writing – review & editing, Supervision, Funding acquisition. **Gözem Turan:** Writing – review & editing, Investigation. **Melle J.W. van der Molen:** Writing – review & editing, Supervision. **Matthias J. Wieser:** Supervision, Investigation. **Niclas Willscheid:** Writing – review & editing, Investigation. **Faisal Mushtaq:** Writing – review & editing, Project administration, Conceptualization. **Yuri G. Pavlov:** Writing – review & editing, Project administration, Funding acquisition, Conceptualization. **Gilles Pourtois:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Funding acquisition, Conceptualization.

Conflict of interest

The authors declare that there is no conflict of interest. Funders and employers had no role in study design or the decision to submit the work for publication.

Open practices

The study in this article has earned Open Data, Open Materials and Preregistered badges for transparent practices. The data, materials and preregistered studies are available at: https://osf.io/2w9gy.

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Supplementary data

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