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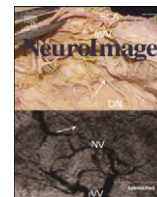
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Electrophysiological correlates of anticipating improbable but desired events

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ABSTRACT

Psychological studies have emphasized that motivation is regulated by the anticipation of the emotional impact from the possible occurrence of unexpected rewarding events. Here, we scrutinized the existence of a corresponding neural signal by means of event-related potentials (ERPs) and computational modeling. In the first experiment, we designed a task that manipulated the probability of gaining a monetary reward and measured ERPs during anticipation and at reward delivery. A sustained frontocentral neural activity (i.e., the stimulus preceding negativity, SPN) was evidenced during the anticipation period. Critically, the SPN was found to increase in amplitude as the reward became more unexpected. Changes in the SPN were found to be predictive of individual differences in risk seeking, suggesting that a greater risk attitude involved a greater motivational state for receiving an improbable reward. In the second experiment, SPN results associated with unexpected monetary gains were replicated in a condition in which participants avoided monetary losses and the occurrence of unexpected rewards was also associated with an increase in the amount of self-reported pleasure. These findings support the existence of a neural ERP signature that encodes the process of tuning our motivation to the possibility of receiving a desirable but improbable rewarding outcome.

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Introduction

Neuroeconomists and psychologists have largely emphasized that motivation is strongly influenced by the expected hedonic consequences of the outcome of one's choice (Mellers and McGraw, 2001). This is so because humans are endowed with the ability to mentally simulate the emotional consequences that events are likely to engender (Gilbert and Wilson, 2007). For instance, a person often exhibits a high level of excitement while awaiting the result of a lottery ticket, even though our midbrain dopaminergic neural system has anticipated with precision that the probability of gaining is remarkably remote (Knutson et al., 2001; Schultz, 2011; Tobler et al., 2005). One possible explanation for such elevated motivational expectations can be derived from the processing of highly unexpected but desired rewarding events (Mellers et al., 1997, 1999). Indeed, psychologists have highlighted the emotional amplification that occurs when receiving an unforeseen

rewarding event and the subsequent elevation of motivational expectation for similar future rewards (Mellers and McGraw, 2001). However, the brain mechanisms responsible for the changes in attentional or motivational state during the anticipation of improbable rewarding events remain unexplored and this constitutes the aim of the present research.

To determine to what extent brain anticipatory responses expressed differences in motivational states, event-related potentials (ERPs) were measured in two separate ERP experiments. We used ERPs here because, unlike other neuroimaging approaches, they allow for the assessment of how neural activity evolves during the time leading up to an event. Additionally, the fine-grained time resolution of ERPs permits the separation of neural state signals in response to different events that occur very close in time, such as the anticipation and impact of a reward.

In the present investigation, we used a sustained frontocentral negative component known as the stimulus preceding negativity (SPN) as an index of the degree of a participant's reward expectation. The SPN is easily observed in the 'waiting period' expressing the motivational/attentional engagement due to possible informative or emotionally relevant feedback (Brunia et al., 2011). The impact of reward delivery was evaluated through the modulation of the amplitude of the feedback-related negativity (FRN) and the P3. The FRN is thought to represent

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the manifestation of a rapidly evaluating motivational system provided by the feedback stimulus that is especially sensitive in expressing the degree by which an outcome is better or worse than expected (Gehring and Willoughby, 2002; Holroyd and Coles, 2002). The P3 has been shown to provide a suitable neural coding response for the degree of unexpectedness or surprise of an event (Duncan-Johnson and Donchin, 1977; Sutton et al., 1965) (see Polich, 2007 for a review).

We tested the prediction that unexpected rewards would induce motivational/attentional states by examining how experimental manipulations and individual differences can modulate neural signals elicited during anticipation and at reward delivery. In the first ERP and computational modeling experiment, we aimed to evaluate the relative contributions of neural signals to anticipation under 5 blocked conditions in which reward was increasingly less likely, with probabilities ranging from 0.9 down to 0.1 in each of the blocks. Because reward magnitude was equated between probability conditions, we hypothesized that (i) differences in motivational/attentional brain states during reward anticipation (measured through the SPN) should vary as a function of reward unexpectedness, (ii) the magnitude of neural responses (measured through the FRN and P3) at reward delivery activity should increase according to the degree of participants' surprise, and (iii) individual differences in SPN magnitude should covary with differences in participants' risk seeking because anticipated pleasure has been related to risk attitude (Mellers and McGraw, 2001). In the second ERP experiment, we aimed to replicate the previous findings regarding SPN and the probability of receiving unexpected monetary gains and we further evaluated the degree that these findings could be extended to an experimental situation in which participants instead of exclusively gaining money were requested to avoid a possible punishment or negative outcome (monetary losses). Finally, we sought to additionally test in the second ERP experiment whether the amount of surprise after a reward was delivered was related to increases in self-reported feelings of pleasure.

Material and methods

Experiment 1

Participants

Sixteen participants were recruited for this study (twelve females; mean age, 23.5; SD, 1.6 years). All participants were healthy, right-handed and reported neither vision problems nor neurological disorders. Informed consent was obtained from participants in accordance with procedures approved by the Ethics Committee of the University of Barcelona.

Task design

The paradigm consisted of a task that required the participants to learn the correct association between a picture and a button press (see Fig. 1A). Participants were instructed that each cue (i.e., pictures) was associated with a single correct response. They were explicitly told that pressing the correct button was sometimes, but not always, followed by a 'gain' outcome (+0.04€). In addition, they were told that trials with an erroneous button press would always be followed by a 'no-gain' outcome (0€). The experiment consisted of 5 identical trial-structure blocks in which the probability to have a rewarded trial, in the case of a correct picture–button association, was parametrically manipulated. The 5 blocks involved a particular probability to obtain a reward: 0.1, 0.3, 0.5, 0.7 and 0.9, which covered the entire probability density. The order of the blocks was pseudorandomized. However, blocks of 0.1 and 0.3 were never presented at the beginning of the experiment. Based on previous pilot studies, this criterion was set to avoid the possibility that participants would not learn any picture–response association during these blocks. Each block contained a set of 6 different familiar black and white pictures (Snodgrass and Vanderwart, 1980) that were randomly assigned to 1 out of 3 possible buttons on a standard PC keyboard ('V'; 'B'; 'N'). Each of the pictures

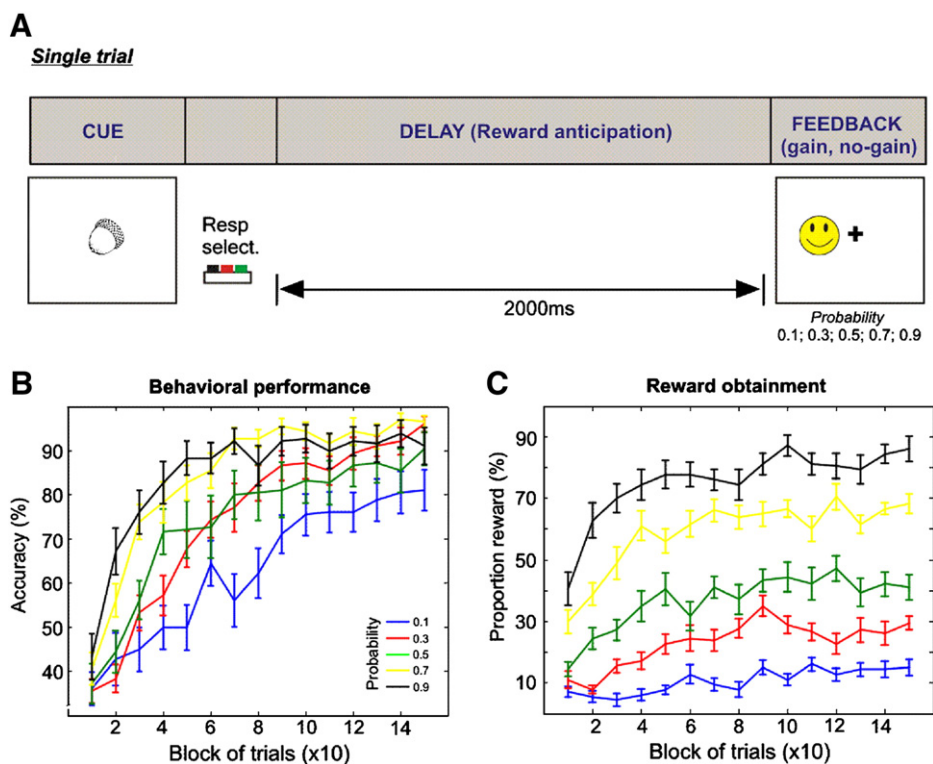


Fig. 1. (A) Schematic representation of a single trial. A cue-picture was presented and participants were instructed to learn which correct button to press so that after a fixed delay of 2 s the correct button press would give them a reward (5 different independent blocks with $p = 0.1, 0.3, 0.5, 0.7$ and 0.9 to obtain a reward). (B) Percentage of trials in which a correct response was given. Error bars denote the standard error of the mean. (C) Percentage of rewards (gain trial) obtained across participants throughout each experimental block.

was randomly presented 150 times during a block, and participants had to learn the correct picture–response association by trial and error. Each button was associated with only 2 pictures. For each participant, before the experiment started, the entire picture set (6×5) was randomly assigned to each block. Half of the participants were requested to respond with the right hand and the other half with the left hand. Participants responded with their right-hand index ('V'), middle ('B') and ring ('N') fingers and vice versa for the left-hand responders. Each trial started with a fixation cross that appeared in the middle of the screen for 400 ms. This was followed by a black and white picture that appeared for 800 ms. A blank screen, lasting 800 ms, followed the picture's appearance. A fixation cross followed and participants were explicitly instructed that this was the moment to respond with one of the three possible buttons. If the participants did not respond within this time period, the trial stopped and a message informing the participant to 'respond faster' was displayed on the screen; then, the next picture trial started. In the case where a participant responded within the 400 ms response time, a delay period of 2000 ms preceded the outcome. The feedback indicated whether the trial was compensated ('gain' trial) or not ('no-gain' trial). Gain feedback was indicated by the appearance of a smiling face, while a non-smiling face (neutral expression) indicated that subjects did not gain in that trial ('no-gain' trial) (Camara et al., 2010a; Marco-Pallarés et al., 2008, 2009). The feedback stimuli remained for 1000 ms. Feedback signaling 'gain' always appeared to the left side of the fixation cross, and the 'no-gain' sign was always shown on the right side. To ensure that participants were attending to feedback appearance, they were instructed to indicate the corresponding position of the outcome by pressing with the index (right position) and middle (left position) fingers (vice versa for left-hand responders) when the stimuli disappeared. The cumulative gaining amount was displayed at the end of each block and participants could rest for a brief period of time. When the task was finished, participants were screened in their risk-seeking attitude by means of the 17 Impulsiveness Questionnaire (Eysenck et al., 1985), which measures impulsivity, risk-taking behavior (venturesomeness) and empathy.

EEG recording

EEG was recorded (band-pass filter: 0.01–250 Hz, notch filter at 50 Hz and 500 Hz sampling rate) from the scalp using a BrainAmp amplifier and tin electrodes mounted in an electrocap (Electro-Cap International) located at 29 standard positions (Fp1/2, Fz, F7/8, F3/4, Fc1/2, Fc5/6, Cz, C3/4, T3/4, Cp1/2, Cp5/6, Pz, P3/4, T5/6, PO1/2, O1/2) and at the left and right mastoids. An electrode placed at the lateral outer canthus of the right eye served as an online reference. EEG was re-referenced offline to the linked mastoids. Vertical eye movements were monitored with an electrode at the infraorbital ridge of the right eye. Electrode impedances were kept below 3 k Ω . EEG was low-pass filtered offline at < 16 Hz for ERP analysis.

ERP analysis

For each participant, EEG data were studied from trials that consisted of a correctly learned cue–response association. A cue–response association was considered to be learned when 3 consecutive correct answers were given. ERPs to the anticipation of reward delivery were studied by extracting response-locked EEG epochs of 2100 ms starting at 100 ms before the button press (baseline). This time period corresponds to the interval preceding the arrival of the outcome, the time window at which we expect to observe modulations of the SPN. SPN amplitude values were calculated by averaging the signal from a 200–1800 ms time window. ERP components that were associated with the impact of outcome delivery (gain, no-gain) were investigated starting at 100 ms before the outcome (baseline) to 600 ms after the outcome onset. Following previous studies (Gehring and Willoughby, 2002; Marco-Pallarés et al., 2008; Nieuwenhuis et al., 2004), the FRN amplitude was calculated as the difference between gain and no-gain trials within a window of 200–300 ms at Fz. Given that P3 effects have been reported over frontal, central and

parietal scalp areas (Duncan-Johnson and Donchin, 1977; Mars et al., 2008; Yeung and Sanfey, 2004), we centered our analysis to changes in the P3 amplitude at central line electrodes (i.e., Fz, Cz and Pz) by averaging amplitudes within a window of 300–500 ms. ERP trials during the anticipation period and at the outcome appearance with a base-to-peak electro-oculogram (EOG) amplitude of more than 100 μ V, trials with amplifier saturation, or trials with a baseline shift exceeding 200 μ V/s were automatically rejected offline. ERPs were then averaged separately for each individual and probability condition. Statistically significant effects were determined using ANOVAs. A statistical threshold of $P < 0.05$ was used to determine significant effects.

Surprise model

We modeled trial-by-trial surprise at feedback appearance throughout learning following similar parameters as previously described (Mars et al., 2008). The model assumed that participants started each block conferring an equal probability that each event might happen and assumed that they updated their estimate of the probability of each event type on each trial, based on the events they previously observed. The same procedure was repeated for each block; i.e., the maximum number of observations was the number of trials in a block. During the course of learning, participants had to learn whether their button-press response was correct, but they also had to estimate the probability that the correct cue–response association could be rewarded (gain trial). In terms of formal treatment, at the start of each block, a single cue–picture could be associated with 3 possible buttons with 2 types of outcomes each (gain, no-gain). For each cue, we entered into the model the possibility that it was followed by an incorrect button response followed always by a no-gain feedback, a correct response followed by a reward ('correct + gain') and a correct response followed with no-reward ('correct + no-gain'). Formally then, we considered that a cue ($N = 6$) could be followed by four possibilities: correct button + gain, correct button + no-gain, first out of two possible incorrect button + no-gain and second incorrect button + no-gain. This distribution can be parameterized by the random vector $P(x) = [p_{1,1}, p_{1,2}, p_{1,3}, p_{1,4}, p_{2,1}, p_{2,1}, \dots, p_{6,4}]$ (which we abbreviate using $P(x) = p$), with c being the cue ($1 \leq c \leq 6$) and f one of the four above-mentioned response–feedback combinations ($1 \leq f \leq 4$).

As proposed by Mars et al. (2008), the probability of an event occurring in the trial j can be inferred from previous trials following the expression

$$p_{cf}^j = \frac{n_{cf}^{j-1} + 1}{N^{j-1} + K}$$

with n_{cf}^{j-1} being the number of occurrences of this type of cue–response–feedback combination in previous trials, K the number of total conditions ($K = 6 \times 4 = 24$ in current study) and N^{j-1} the number of observations up to the trial preceding j , that is

$$N^{j-1} = \sum_{c=1}^6 \sum_{f=1}^4 n_{cf}^{j-1}.$$

We quantified the surprise I on each trial as follows (Mars et al., 2008; Shannon, 1948):

$$I(x^j) = -\log_2 p_{cf}^j.$$

This states that the surprise of observing a certain event type at the j th trial is equal to the negative log of its predicted probability given all preceding trials. Accordingly, the amount of surprise conveyed by the occurrence of an event is high when an infrequent stimulus occurs in a stimulus sequence with high predictability. For example, a gain trial with a condition of $p = 0.1$ of obtaining a reward is as surprising as a no-gain trial under the condition of obtaining a reward of $p = 0.9$.

Following previous results (Barcelo and Knight, 2007; Donchin, 1981; Duncan-Johnson and Donchin, 1977; Mars et al., 2008), we statistically tested this hypothesis at the single-trial level by correlating (Pearson coefficient) individual participants' trial-by-trial changes in the P300 at Pz (mean: 300–500 ms) to single-trial values of surprise (I). Additionally, if C is the number of participants showing a significant correlation, then $P(C)$ follows a binomial distribution with a correct probability of p and $n = 16$ (the number of participants). We tested against the null hypotheses that the computational model fit our sample at the chance level ($p = 0.5$) by using the normal approximation to the binomial density, which allowed us to compute P values. A P value of 0.005, for example, corresponds to ~13/16 participants showing a significant correlation between ERP amplitude changes and I estimates.

Experiment 2

Task design

We ran a 2-condition variant of the experiment 1 with a separate sample of sixteen healthy right-handed participants (eight females, mean age, 20.2; SD, 1.6 years). The paradigm design was similar to those in Experiment 1. Briefly, participants were required to learn the correct association between cue-pictures and a button-press. However, they could either gain/not-gain money (i.e., similar to the design used in Experiment 1; condition A or 'monetary gains') or lose/avoid-losing money (condition B or 'avoid-losing money'). In the condition 'avoid-losing money' participants knew that a correct button press would avoid losing money on such trial. Incorrect trials resulted in a monetary loss ('-0.04€'). As in Experiment 1, both the 'monetary gains' and 'avoid losing money' conditions contained 5 separate probability blocks (0.1, 0.3, 0.5, 0.7 and 0.9). For instance, in the probability block of 0.1, only 1 out of 10 correct responses would lead to 'monetary gain' in one condition or 'avoid losing money' in the other. Feedback stimuli were presented in the center of the screen and, contrary to what was instructed of them in Experiment 1, participants were not required to indicate the appearance of the feedback stimulus on the screen. The order of the conditions to be performed in the first session was counterbalanced across participants. Participants read detailed instructions of the experiment and were explicitly informed about the goal (gain or avoid losing money) of the task for each session day. Participants received 50€ before they began the experiment. They were instructed to conceal this amount in their bag or jacket pocket (which was stored securely outside the EEG room) and told that this amount was theirs to keep and, at the end of the experiment, would be topped up by any amount they gain, but equally they would have to pay the experimenter back any amount they lose. The cumulative winnings or losses were displayed at the end of each of the 5 runs. As in the main EEG experiment, each run involved 6 different pictures that were randomly presented 30 times during each block and each of them was randomly assigned to one correct button response. The cue stimulus time duration, anticipation period, feedback time duration and ISI characteristics were as described in Experiment 1. Participants were also requested to use the three button press used in the main experiment and right and left hands were counterbalanced across participants and experimental sessions. This time however, participants were allowed to press the button whenever they wanted without the need to wait for the disappearance of the cue stimulus from the screen. Cue stimuli remained in the screen until the participants responded.

Besides, participants were instructed that during the task they were required to answer, without a time limit, the question 'How do you feel?' that would suddenly appear in the center of the screen just after some feedback outcomes. They were told to indicate, in a scale from 1 ('very unpleasant') to 7 ('very pleasant'), their subjective feeling of their current pleasure state. Participants were asked about their subjective pleasure state 22 times in each probability block. Measures were randomly collected throughout the run so that we

could screen changes of subjective pleasure within each block with the inclusion of two requirements. First, participants were always firstly asked just after the end of the first trial of each run. And second, the following measure was always taken just after they obtained the first reward. The remaining 20 questions were then distributed equidistantly until the last trial of the block, at which the last measure of pleasure was taken. Analysis was conducted separately for ratings of pleasure that were reported after feedbacks indicating gain or no-gain. Thus, for each probability condition we obtained a measure of subject pleasure that corresponded to two theoretically distinct instant utility values. Gain and no-gain subjective pleasure measures were then averaged separately for each participant and probability block condition.

EEG recording and ERP analysis

EEG recording parameters were the same as those used in Experiment 1. EEG was recorded (band-pass filter: 0.01–250 Hz, notch filter at 50 Hz and 500 Hz sampling rate) from the scalp using a BrainAmp amplifier from the scalp using tin electrodes mounted in an electrocap (Electro-Cap International) and located at 29 standard positions (Fp1/2, Fz, F7/8, F3/4, FCz, Fc1/2, Fc5/6, Cz, C3/4, T3/4, Cp1/2, Cp5/6, Pz, P3/4, T5/6, PO1/2, Oz) and at left and right mastoids. An electrode placed at the lateral outer canthus of the right eye served as an online reference. EEG was re-referenced off-line to the linked mastoids. Vertical eye movements were monitored with an electrode at the infraorbital ridge of the right eye. Electrode impedances were kept below 3 k Ω . EEG was offline low-pass filtered at <16 Hz for ERP analysis. ERP analysis followed the same parameters as detailed in Experiment 1.

Results

Experiment 1: unexpected monetary gains

Behavioral performance

Participants successfully learned the correct cue-response button association (Fig. 1B). The mean response accuracy was 81.25% (STD = 18.6%) for $p = 0.1$, 95.62% (STD = 7.88%) for $p = 0.3$, 91.87% (STD = 15.49%) for $p = 0.5$, 98.12% (STD = 3.90%) for $p = 0.7$ and 90.62% (STD = 18.19%) for $p = 0.9$. Participants' learning was confirmed by analyzing the differences in response accuracy over time. An ANOVA, including reward probability (5 levels) and the amount of training (3 levels: low amount – mean of the first 20 trials of each cue, middle amount – trials 70 to 90, and high amount – trials 130 to 150) as the within-subject factors, confirmed that accuracy increased over time (main effect of the amount of training, $F(2,30) = 314.62$, $P < 0.001$). In addition, the higher the probability of reward obtainment, the faster the participant learned (reward probability \times amount of training, $F(8,120) = 668.96$, $P < 0.001$).

EEG during the anticipation and reward delivery

As expected, the study of ERPs in response to the anticipation of reward delivery revealed an SPN component. The SPN amplitude changes were parametrically modulated by reward probability (Figs. 2A and B) (main effect: $F(4,60) = 4.34$, $P < 0.01$; linear trend: $F(1,15) = 13.27$, $P < 0.01$), and this effect was maximal at frontal regions (Fz) (main electrode effect: $F(2,30) = 61.44$, $P < 0.01$) (number of trials included in the ERP analysis can be found in Inline Supplementary Table S1). We also explored whether these effects were sustained throughout the entire 2 s anticipation period. We centered this analysis on the Fz electrode given that it was the electrode with maximal SPN expression in our analysis. We further tested whether the SPN linear amplitude modulation observed across probabilities could be influenced by the baseline period. An ANOVA at Fz confirmed that the linear SPN effects were consistent even at different baseline periods such as -200 to 0 ($F(4,60) = 5.39$, $P < 0.01$, linear trend $F(1,15) = 16.1$, $P < 0.01$) and

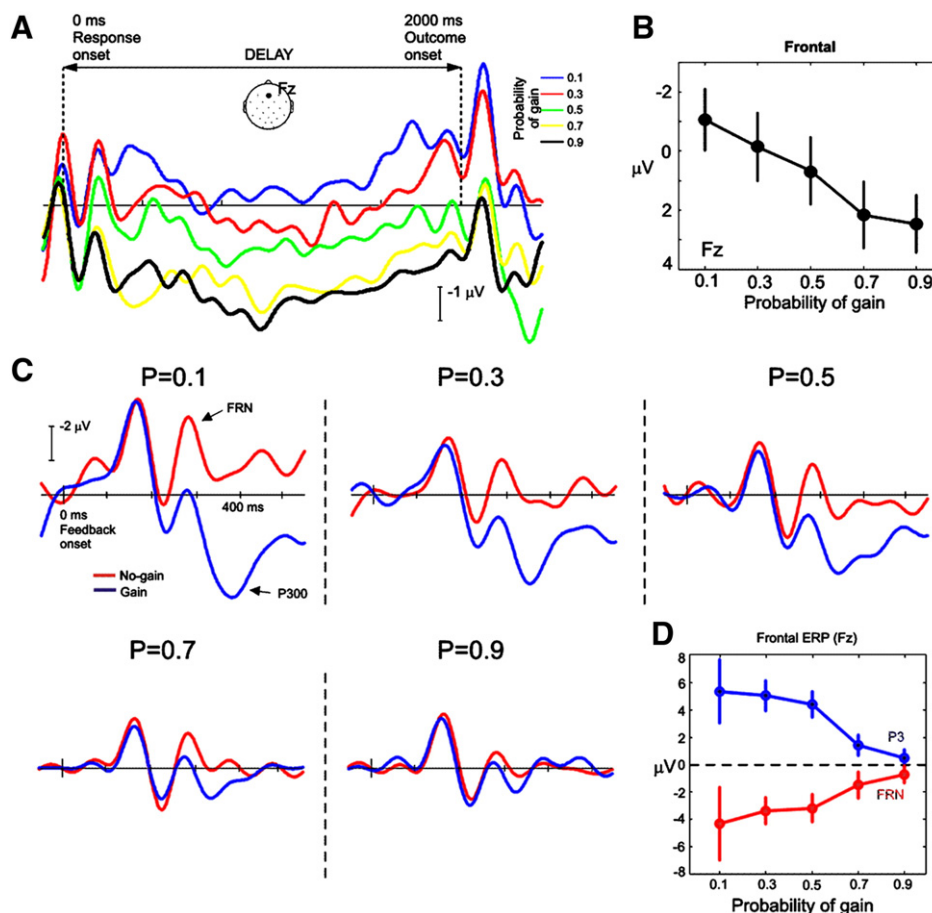


Fig. 2. (A) ERPs at the Fz electrode during anticipation of reward for each probability condition. A 6 Hz low-pass filter was only used to display the SPN. (B) Linear amplitude modulation of the SPN for the anticipation of reward (mean amplitude over the 2 s delay) for each probability condition. (C) Mean ERPs at the Fz electrode in response to gain and no-gain trials for each probability condition across participants. Each plot displays the ERPs associated with the gaining and no-gaining probability (e.g., when the gaining and no-gaining probability was set to $p = 0.1$). The plot shows that a clear modulation of the FRN is associated with no-gain trials and the P3 is associated with gain trials. (D) FRN (differences in waveform of no-gain–gain trials) and P3 mean amplitudes as a function of probability to obtain a reward at Fz. Error bars in B and D denote the standard error of the mean.

–300 to 0 ($F(4,60) = 4.39$, $P < 0.01$, linear trend $F(1,15) = 12.16$, $P < 0.01$). We further confirmed that SPN modulation as a function of probability was not affected by differences in ERP amplitude because a similar linear trend was found when ERPs were equated to all at '0 μV ' at time point 0 ms (linear trend $F(1,15) = 4.58$, $P < 0.05$).

Inline Supplementary Table S1 can be found online at <http://dx.doi.org/10.1016/j.neuroimage.2013.03.062>.

There were clear FRN and P3 ERP components after the presentation of feedback. An ANOVA, including reward probability and electrode (frontal – Fz, central – Cz, parietal – Pz) as within-subject factors, showed that the FRN (calculated as the difference between gain and no-gain outcomes) was maximal at Fz (electrode effect: $F(2,28) = 4.49$, $P < 0.05$), and that the FRN changed as a function of the probability of the predicted reward (reward probability effect: $F(4,56) = 3.21$, $P < 0.05$), reaching a maximum when the reward was highly improbable (i.e., $p = 0.1$) (Fig. 2C). A trend analysis of the FRN amplitude at Fz confirmed the monotonic decrease as a function of reward probability ($F(1,14) = 11.42$, $P < 0.01$) (Fig. 2D).¹

A repeated-measures ANOVA, including reward probability, electrode and feedback type (gain, no-gain) as within-subject factors, confirmed a larger P3 in gain than no-gain trials (feedback type effect: $F(1,15) = 11.27$, $P < 0.05$), and the magnitude of the P3 increased as

a function of the unexpectedness of the reward obtained (reward probability effect: $F(4,60) = 3.85$, $P < 0.01$) (Figs. 2C and D). The P3 magnitude changes were, on average, higher at parietal regions (electrode effect: $F(2,30) = 41.51$, $P < 0.01$) although feedback type and reward probability effects were not altered by the electrode location (feedback type \times reward probability \times electrode interaction: $F(8,120) = 0.28$, $P > 0.05$).

A correlation analysis (Pearson coefficient) of the resulting signal from averaging ERPs across probability conditions revealed that when the SPN amplitude during the anticipation phase increased (larger negative values), the amplitude of the P3 at outcome delivery also increased (measured at Pz) ($r = -0.58$, $P < 0.05$ for gain and $r = -0.65$, $P < 0.01$ for no-gain trials). No significant correlation was observed between the SPN and the FRN amplitudes (calculated as the difference between the no-gain and gain conditions, $r = -0.02$, $P > 0.05$). Similarly, no statistically significant results were found when the SPN was correlated separately from the FRN amplitude for the gain ($r = -0.15$, $P > 0.05$) and no-gain conditions ($r = -0.19$, $P > 0.47$).

Individual differences in risk-taking

We next evaluated whether such ERP variations during anticipation and at outcome events covaried with individual differences in risk-taking. We found a significant negative correlation between individual risk attitudes and the SPN magnitude ($r = -0.61$, $P < 0.02$) (Fig. 3A). Additionally, variations in P3 amplitude (averaged over gain and no-gain conditions) were correlated positively with individual differences

¹ One outlier was excluded from the FRN statistical analysis because the FRN amplitude at Fz exceeded more than 2 standard deviations from the sample mean at the $p = 0.1$ probability condition.

in risk attitude ($r = 0.54, P < 0.05$) (Fig. 3B). This correlation remained significant for the P3 responses elicited for gain ($r = 0.55, P < 0.05$) and no-gain conditions ($r = 0.54, P < 0.05$) separately. No significant correlation was found between risk attitude and FRN amplitude (gain minus no-gain) ($r = 0.21, P = 0.43$).

Modeling the behavioral single-trial degree of surprise and EEG

Behaviorally, the computational model showed that highly unexpected events provided a higher degree of surprise (I). In addition, high I values appeared only when participants had already learned the correct association between the cue and response button (Fig. 4A). At the neuronal level, participants showed a trial-by-trial correlation between the amplitude of the P3 at Pz and I . 15 out of 16 participants showed a trial-by-trial correlation between amplitude of the P3 at Pz and the degree of surprise (I) (Pearson coefficient threshold at $P < 0.05$) (Figs. 4B and C) (binomial probability distribution test, $P = 0.0002$). I values were correlated to neither SPN amplitude changes nor to other ERP component amplitude variations at outcomes which are to be sensitive to the income of salient events (i.e., P2 extracted from an averaged 200–250 ms and N2, from 260 to 300 ms, both measured at Fz) (Luck and Hillyard, 1994) (Fig. 4D). A trial-by-trial correlation between the amplitude of the P3 at Pz and I values was obtained when only correct gain trials ($P < 0.05$ in 15 out of 16 participants; $P = 0.0002$ binomial test at group level) but not when only correct no-gain trials were included in the analysis ($P < 0.05$ in 6 out of 16 participants; $P = 0.84$ binomial test at group level). These findings fit well with the idea that P3 amplitude changes vary according to the degree of surprise reward occurrence.

Previous animal research has shown that the obtainment of an unexpected reward boosts the desire to obtain it in the future (Berridge, 2003; Berridge and Aldridge, 2008). To address this possibility in our participants, we tested whether the degree of behavioral surprise at an outcome could relate to the anticipation of a reward in the subsequent trial. In the current experiment, this effect should be reflected as an increase in the SPN during reward anticipation that correlates with the I in the previous trial. To test this prediction, we selected 20 trials with the highest I values ('high surprise') and 20 trials with the lowest I values ('low surprise') at feedback for each participant and probability condition. We then averaged the frontal EEG activity during the anticipation of reward on the following trial. This analysis showed a significant increase in negativity after 'high surprise' events (repeated measures t -test; $t(14) = 3.11; P < 0.01$) (Fig. 4E). This difference was not significant when the trials used for this analysis (i.e., 40 trials for each probability condition) were binned according to whether they indicated gain or no-gain at outcome display ($t(14) < 1$) or when this analysis was performed on anticipatory EEG activity at posterior scalp regions (i.e., Pz) that were binned according to the amount of surprise or outcome valence (both, $t(14) < 1$).

Experiment 2: unexpected monetary losses

Behavioral performance

The mean response accuracy in condition A was 95% (STD = 9.21%) for $p = 0.1$, 97.31% (STD = 5.41%) for $p = 0.3$, 95% (STD = 11.77%) for $p = 0.5$, 94.24% (STD = 15.91%) for $p = 0.7$ and 98.84% (STD = 2.88%) for $p = 0.9$. Similar accuracy rates were found for condition B, 93.85% (STD = 11.12%) for $p = 0.1$, 88.08% (STD = 11.69%) for $p = 0.3$, 93.46% (STD = 7.93%) for $p = 0.5$, 95.38% (STD = 6.34%) for $p = 0.7$ and 96.92% (STD = 6.94%) for $p = 0.9$. An ANOVA including probability (5 levels), amount of training (3 levels) and condition (2 levels) indicated that participants' response accuracy increased over time (main effect of amount of training $F(2,24) = 178.58; P < 0.01$). Furthermore, a significant triple interaction effect of probability \times amount of training \times condition ($F(8,96) = 6.45; P < 0.01$) indicated that the probability of the outcome affected differentially the learning response accuracy progression between conditions. Thus, although separate ANOVAs for each condition confirmed that in the two conditions (A and B), participants' response accuracy increase was faster the higher the probability of the associated outcome ($F(8,96) = 3.12; P < 0.01$), and participants learned faster in condition A than in B (condition \times amount of training $F(2,24) = 333.81; P < 0.01$).

EEG during the anticipation and at outcome delivery

As in the EEG analysis procedure in Experiment 1, here we only studied EEG data from those trials for which the cue-response association was correctly learned (number of trials included in the ERP analysis can be found in Inline Supplementary Table S2). Similarly, a criterion of 3 consecutive correct answers was required to consider that the cue-response association was learned. 3 participants were excluded from further analysis because less than 5 trials were available for the ERP analysis to feedback stimuli in the probability block of $p = 0.1$.

Inline Supplementary Table S2 can be found online at <http://dx.doi.org/10.1016/j.neuroimage.2013.03.062>.

SPN was studied by extracting response-locked EEG epochs of 2100 ms starting 100 ms before the button press (baseline). A repeated measures ANOVA was calculated including condition (A or B) \times reward probability (5 levels) \times electrode (Fz, Cz, Pz) as within subject factors. The results showed that the SPN amplitude changes were again parametrically modulated by the likelihood of occurrence of condition A (gaining money) or condition B (avoid losing money) (reward probability effect: $F(4,48) = 2.34, P = 0.06$; linear trend: $F(1,12) = 16.12, P < 0.05$; main effect of condition and all effects interacting with condition were $F < 1$), and this effect was maximal at frontal regions (Fz) (main electrode effect: $F(2,24) = 85.32, P < 0.01$) (Figs. 5A and B).

FRN amplitude was calculated as the difference between gain and no-gain trials in condition A and no-loss and loss trials in condition B within a window of 200–300 ms. P300 analysis was performed by

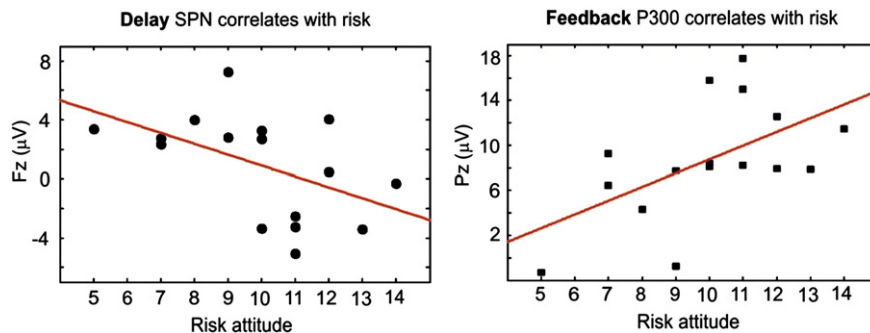


Fig. 3. Individual differences in risk attitude predicted the amplitude of the SPN during anticipation of reward (left panel) and the P3 elicited at outcome delivery (right panel). Note that because the EEG component associated with the anticipation to reward is negative, the higher its amplitude, the riskier is the attitude, which is expressed as a correlation with negative sign.

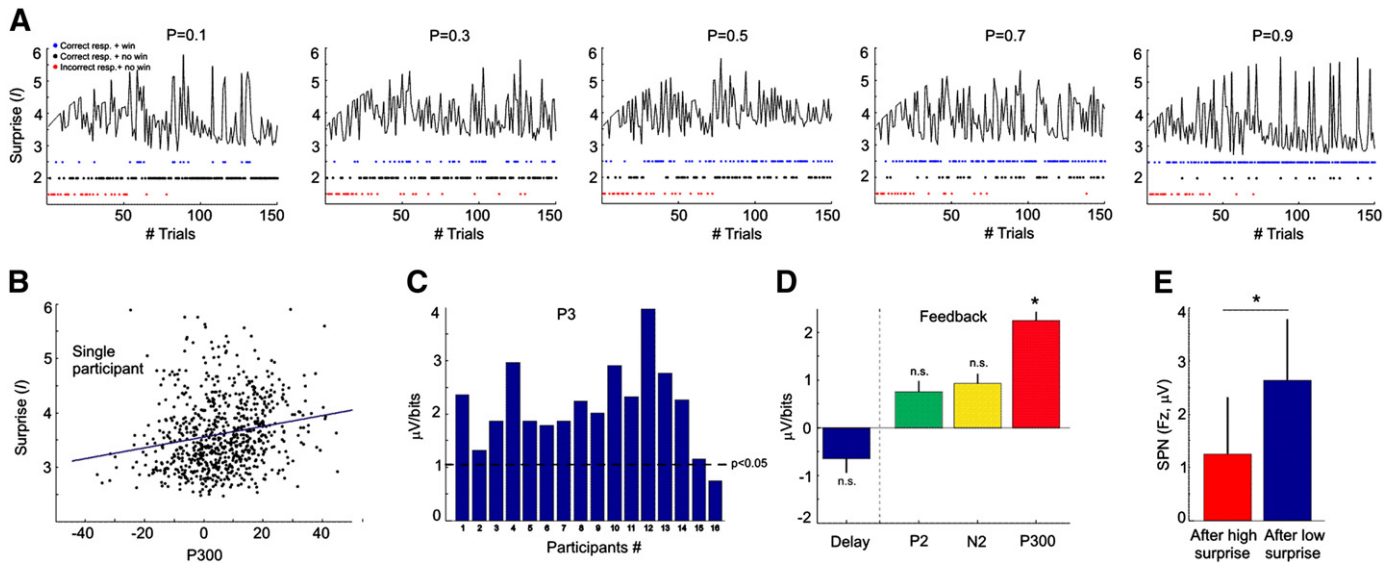


Fig. 4. (A) Example of the behavioral model for one subject. (B) P3 and surprise value (I) for a representative subject. (C) Model-based analysis of the EEG data. (D) Single subject model-based analysis of the P3 ERP component. (E) EEG anticipation of reward activity is dependent on previous feedback impact. Error bars denote the standard error of the mean. * denotes $P < 0.05$.

averaging amplitudes within a window of 300–500 ms. The appearance of the feedback in this experiment elicited clear FRN and P300 ERPs in both conditions A and B (Fig. 5C). In fact, an ANOVA, including reward probability (5 levels), electrode (Fz, Cz and Pz) and condition (A or B)

as within-subject factors, replicated our reported FRN findings in Experiment 1 that it was maximal at Fz (electrode effect: $F(2,24) = 10.27$, $P < 0.05$) and that it changed parametrically as a function of the probability of the predicted reward (reward probability effect:

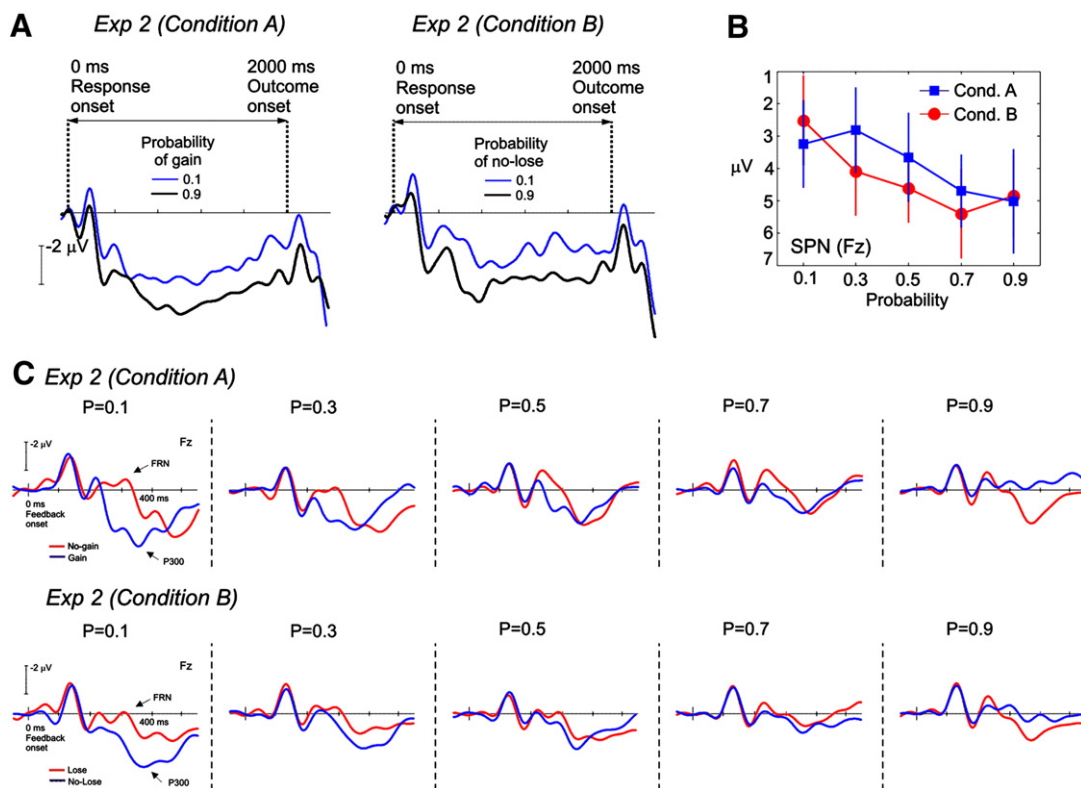


Fig. 5. (A) SPN ERP during anticipation of feedback for each probability and condition in Experiment 2. A 6 Hz low-pass filter was only used to display the SPN. (B) Linear amplitude modulation of the SPN to anticipation of feedback for conditions A and B in Experiment 2 (mean amplitude over the 2 s delay) for each probability run. (C) Grand average ERPs to feedback stimuli for each probability block and condition at Fz. Clear FRN and P3 were elicited in both conditions A and B. Each plot in condition A displays the ERPs associated with the probability of gaining but also the similar probability of not gaining (i.e., from left to right, in the first case, the ERPs are depicted when gaining probability was set at $p = 0.1$ and no-gaining probability was set also at $p = 0.1$). Similarly, in condition B, the plot to the left depicts ERPs when the probability of avoiding loss was set at $p = 0.1$ and the probability of losing set at $p = 0.1$.

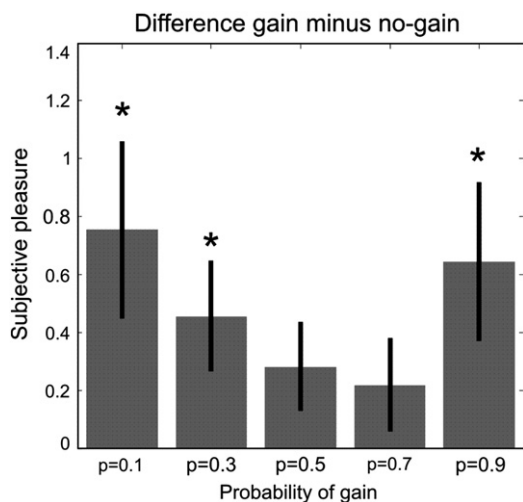


Fig. 6. The difference between subjective ratings to gain and no-gain trials for each condition in Experiment 2. Bars represent the mean across participants. Error bars denote the standard error of the mean. * denotes $P < 0.05$.

$F(4,48) = 11.45, P < 0.05$), being maximal not only when the reward obtained was very improbable but also when FRN amplitudes did not differ between conditions A and B (main effect of condition and all effects interacting with condition were all $P > 0.1$). As in the main experiment, FRN amplitudes decreased linearly across probabilities ($F(1,12) = 25.21, P < 0.01$). In terms of P300, an ANOVA, including reward probability, electrode, feedback type (gain/no-gain or no-loss/loss) and condition (A or B) as within-subject factors, revealed larger P300 amplitudes to unexpected feedbacks (reward probability effect: $F(4,48) = 4.16, P < 0.01$; feedback \times reward probability interaction: $F(4,48) = 10.03, P < 0.01$) and that this effect was maximally shown at Pz (electrode effect: $F(2,24) = 34.99, P < 0.01$).

Subjective measures of pleasure

In Experiment 2, we further tested if our EEG results were also associated with differences in subjective measures of pleasure after gain and no-gain outcomes. To test the prediction that unexpectedness of reward obtainment boosted feelings of pleasure, we compared the individual ratings of pleasure to gain and no-gain trials separately for each probability block. In accordance with our hypothesis, ratings of subjective pleasure after having won were larger than those after not gaining when gaining was highly unexpected ($p = 0.1$ and $p = 0.3$ were both $t(12) > 2.3; P < 0.05$). The pleasure ratings were equivalent when the expectancy of obtaining a reward increased ($p = 0.5$ and $p = 0.7$, both $t(12) < 1.9; P > 0.05$). At $p = 0.9$, higher ratings of pleasure were recorded after rewarding trials ($t(12) = 2.4; P < 0.05$), probably mixed with possible deceiving feelings elicited by not gaining in such contextual circumstances (Fig. 6).

Discussion

In the present study we used electrophysiological recordings to elucidate the existence of anticipatory neural signals that could be related to increased motivational/attentional states when expecting desired rewards that are unlikely to appear. To characterize these states during the anticipation period we measured the SPN amplitude during the interval following a specific cue that predicted a positive monetary reward with a fixed probability. The SPN is believed to be primarily sensitive to the expectation of motivationally relevant stimuli or stimuli with emotional valence, presumably reflecting the engagement of appetitive and defensive motivational systems of the brain (see van Boxtel and Böcker, 2004 for a review). For example, the SPN does not reflect general attentional or cognitive demands for tasks that do not have motivational value (Hillman et al., 2000; Kotani and Aihara, 1999) or

general responsiveness to feedback with no task-significance (Damen and Brunia, 1994; Kotani and Aihara, 1999; Kotani et al., 2003).

In the first ERP experiment, after the participants learned a certain association (cue–response mapping), they were able to predict the number of times that a fixed reward would appear. We used blocks of different probabilities (from 0.1 to 0.9) to investigate the motivational impact of highly expected (0.9), uncertain (0.5) or unexpected monetary gains (0.1). The most striking result was that as the reward became more unexpected (when the correct response was followed by a reward less often on average), the SPN component at frontal regions increased in amplitude more than that under other reward probability conditions (see Fig. 2). This result was further replicated in the second ERP experiment in which we added a condition in which participants avoided losing money (see Fig. 5). This indicates that the SPN results can be generalized to motivational states related to the improbable avoidance of negative events or possible punishments and are not exclusively associated with unexpected monetary gains.

An increased SPN amplitude after choices that were followed by very low probability of reward may seem counterintuitive from an ecological point of view and does not apparently converge with the data from normative reward prediction error models. According to these models, the probability of reward obtainment is expressed as a parametric increase during anticipation in phasic activity in mid-brain dopaminergic neurons (Schultz et al., 1997; Tobler et al., 2005) and the striatum (Knutson et al., 2001 but see Guitart-Masip et al., 2011). Furthermore, our data on the electrophysiological responses recorded during the anticipatory delay did not conform to either animal (Fiorillo et al., 2003) or human (Tobler et al., 2009) findings that show uncertainty-induced neural activity for anticipation of reward delivery. These studies revealed the existence of a signal that increased as the probability of receiving a reward rose from 0.0 to 0.5 and then decreased toward zero as the probability grew further to 1.0. If the SPN was a signal of this sort, we would have expected the SPN magnitude to be dependent on the amount of information conveyed by the feedback, as the association would have been completely predicted. Indeed, the condition that had the least predictive information corresponded to the condition in which the probability of receiving or not receiving a reward was more uncertain ($p = 0.5$). Therefore, this probability condition would be expected to have the largest increase of the SPN component; however, this was not found to be the case in our study.

Instead, we argue that the SPN amplitude modulation observed here reflects the process of tuning our motivation or attention to the possibility of receiving a desirable but highly unlikely rewarding outcome. Our findings suggest that our motivational system in the brain is regulated, or at least partially influenced, by the emotional value that an imminent reward could evoke. In fact, the process of anticipating the emotional consequences of our decisions represents a pivotal aspect of psychological (Mellers and McGraw, 2001) and social cognition (Gilbert and Wilson, 2007) theories of decision making. For instance, the decision affect theory (DAT) (Mellers et al., 1997) emphasizes the major role that unexpectedness plays in amplifying the emotional experience to an outcome. Mellers et al. (1999) showed that in a gambling scenario participants anticipated more intense emotional reactions to gaining outcomes the less likely they were to occur.

In the present study, we found two important findings that lend support to the psychological effects derived from receiving unexpected rewards proposed in the DAT (Mellers et al., 1997). First, the impact of very unlikely rewards was reflected as stronger neural responses in participants than those observed following rewards that were expected. Thus, the degree of anticipation of positive rewards modulated the impact of feedback processing, and this was reflected in the amplitude changes in the FRN and P3. Second, the P3 amplitude changes at outcome delivery fit well with a recent single-trial behavioral computational model of surprise (Mars et al.,

2008). From this study, the P3 arose as a formal quantification of the degree of surprise associated with reward occurrence.

We predicted that if the SPN was indexing the degree of anticipation for the possibility of receiving a desired outcome, individual differences in the way this emotional reaction took place would also be accompanied by concomitant variations in attention or motivation during the anticipation period. Consistent with this notion, our findings showed that the magnitude of the P3, which indexed the degree of surprise at reward delivery, was correlated with the amplitude of the SPN during anticipation; the greater the surprise at reward delivery, the greater was the SPN amplitude during the expectation of reward delivery. We further showed that such individual variations in anticipating and processing rewarding outcomes were also related to individual differences in risk attitude. In fact, DAT envisages that greater pleasure at anticipation of unforeseen rewarding events is associated with risk seeking behavior (Mellers and McGraw, 2001). In the present study, the magnitude of the SPN and P3 predicted individual differences in risk-taking tendencies; so that the greater the risk seeking attitude, the greater is the response amplitude of the P300 and of the SPN. An important question derived from the current findings is whether such individual differences in reward-related personality traits (i.e., risk-taking), which have been shown to modulate activity in brain reward circuitry (Camara et al., 2010b; Schultz, 2011) can be altered in pathological behavior in psychiatric disorders (Clark et al., 2009).

Albeit speculative, the SPN results from the current study could partially reflect the concept of 'wanting' in some animal models (Berridge and Kringelbach, 2008). These studies have helped to delineate important predictions regarding the nature of affective reactions when unexpected rewards are presented. The concept of 'incentive salience' is used to describe the process by which pleasurable events are more likely to become motivationally 'wanted' the less likely they are to appear, similarly to what we argue in the current study. When incentive salience is attributed to the reward-related stimulus, it transforms the brain's representation from a mere perception or memory into a motivationally potent incentive (Berridge, 2007; Berridge and Kringelbach, 2008; Berridge and Robinson, 1998; Berridge et al., 2009). Incentive salience drives 'wanting' behaviors and has been shown, theoretically (Berridge and Robinson, 1998) and empirically (Smith et al., 2011), to be operationally separated from 'liking', which refers to the hedonic impact of the rewarding event. In the present experiment, the anticipatory phase of the present study may trigger expectations of hedonic states upon its arrival which could create a sense of 'wanting'. One could even speculate that the increase in this signal kept our participants engaged in those blocks with low probability of reward. Unfortunately, empirical findings fully establishing the role of 'wanting' and 'liking' in humans are still lacking. While we await further studies, the current results could indicate a possible way to investigate this aspect in healthy populations as well as in psychiatric patients in which the motivation/wanting dimension of decision making might be altered (Barch and Dowd, 2010).

In summary, the present findings highlight the critical role of unexpected and highly desired rewards in tuning motivational and attentional processes during anticipation or 'wanting' states. Our results indicate that such a motivational state is reflected as a sustained frontocentral activity during the anticipation of an upcoming reward. Larger anticipatory-related EEG activity corresponded to conditions in which the expectance of a reward was very unlikely and it was accompanied by an enhancement of the subjective pleasure when the unforeseen reward was delivered. These data help bridge the gap between animal (Berridge and Kringelbach, 2008) and psychological (Mellers et al., 1999) findings arguing that motivation is directly modulated by the affective reactions to unexpected positive outcomes. Such an integrative map of motivational states during anticipation, the effects of when the outcome is displayed and the effects at the subjective level, will offer valuable insight for understanding

human desirability aspects of decision making and may also provide an important step for investigating and treating pathological behaviors derived from wanting/motivation problems that are prevalent across several psychiatric disorders.

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Conflict of interest

There is no conflict of interest.

References

- Barcelo, F., Knight, R.T., 2007. An information-theoretical approach to contextual processing in the human brain: evidence from prefrontal lesions. *Cereb. Cortex* 17 (Suppl. 1), i51–i60.
- Barch, D.M., Dowd, E.C., 2010. Goal representations and motivational drive in schizophrenia: the role of prefrontal–striatal interactions. *Schizophr. Bull.* 36, 919–934.
- Berridge, K.C., 2003. Pleasures of the brain. *Brain Cogn.* 52, 106–128.
- Berridge, K.C., 2007. The debate over dopamine's role in reward: the case for incentive salience. *Psychopharmacology (Berl)* 191, 391–431.
- Berridge, K.C., Aldridge, J.W., 2008. Decision utility, the brain, and pursuit of hedonic goals. *Soc. Cogn.* 26, 621–646.
- Berridge, K.C., Kringelbach, M.L., 2008. Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology (Berl)* 199, 457–480.
- Berridge, K.C., Robinson, T.E., 1998. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Res. Brain Res. Rev.* 28, 309–369.
- Berridge, K.C., Robinson, T.E., Aldridge, J.W., 2009. Dissecting components of reward: 'liking', 'wanting', and learning. *Curr. Opin. Pharmacol.* 9, 65–73.
- Brunia, C.H., Hackley, S.A., van Boxtel, G.J., Kotani, Y., Ohgami, Y., 2011. Waiting to perceive: reward or punishment? *Clin. Neurophysiol.* 122, 858–868.
- Camara, E., Kramer, U.M., Cunillera, T., Marco-Pallares, J., Cucurell, D., Nager, W., Mestres-Misse, A., Bauer, P., Schule, R., Schols, L., Tempelmann, C., Rodriguez-Fornells, A., Munte, T.F., 2010a. The effects of COMT (Val108/158Met) and DRD4 (SNP –521) dopamine genotypes on brain activations related to valence and magnitude of rewards. *Cereb. Cortex* 20, 1985–1996.
- Camara, E., Rodriguez-Fornells, A., Munte, T.F., 2010b. Microstructural brain differences predict functional hemodynamic responses in a reward processing task. *J. Neurosci.* 30, 11398–11402.
- Clark, L., Lawrence, A.J., Stley-Jones, F., Gray, N., 2009. Gambling near-misses enhance motivation to gamble and recruit win-related brain circuitry. *Neuron* 61, 481–490.
- Damen, E.J., Brunia, C.H., 1994. Is a stimulus conveying task-relevant information a sufficient condition to elicit a stimulus-preceding negativity? *Psychophysiology* 31, 129–139.
- Donchin, E., 1981. Presidential address, 1980. Surprise!..surprise? *Psychophysiology* 18, 493–513.
- Duncan-Johnson, C.C., Donchin, E., 1977. On quantifying surprise: the variation of event-related potentials with subjective probability. *Psychophysiology* 14, 456–467.
- Eysenck, S.B.G., Pearson, P.R., Easting, G., Allsopp, J.F., 1985. Age norms for impulsiveness, venturesomeness and empathy in adults. *Pers. Individ. Differ.* 6, 613–619.
- Fiorillo, C.D., Tobler, P.N., Schultz, W., 2003. Discrete coding of reward probability and uncertainty by dopamine neurons. *Science* 299, 1898–1902.
- Gehring, W.J., Willoughby, A.R., 2002. The medial frontal cortex and the rapid processing of monetary gains and losses. *Science* 295, 2279–2282.
- Gilbert, D.T., Wilson, T.D., 2007. Prospection: experiencing the future. *Science* 317, 1351–1354.
- Guitart-Masip, M., Fuentemilla, L., Bach, D.R., Huys, Q.J., Dayan, P., Dolan, R.J., Duzel, E., 2011. Action dominates valence in anticipatory representations in the human striatum and dopaminergic midbrain. *J. Neurosci.* 31, 7867–7875.
- Hillman, C.H., Apparies, R.J., Hatfield, B.D., 2000. Motor and nonmotor event-related potentials during a complex processing task. *Psychophysiology* 37, 731–736.
- Holroyd, C.B., Coles, M.G., 2002. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. *Psychol. Rev.* 109, 679–709.
- Knutson, B., Adams, C.M., Fong, G.W., Hommer, D., 2001. Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *J. Neurosci.* 21, RC159.
- Kotani, Y., Aihara, Y., 1999. The effect of stimulus discriminability on stimulus-preceding negativities prior to instructive and feedback stimuli. *Biol. Psychol.* 50, 1–18.
- Kotani, Y., Kishida, S., Hiraku, S., Suda, K., Ishii, M., Aihara, Y., 2003. Effects of information and reward on stimulus-preceding negativity prior to feedback stimuli. *Psychophysiology* 40, 818–826.
- Luck, S.J., Hillyard, S.A., 1994. Electrophysiological correlates of feature analysis during visual search. *Psychophysiology* 31, 291–308.

- Marco-Pallarés, J., Cucurell, D., Cunillera, T., Garcia, R., Andres-Pueyo, A., Munte, T.F., Rodríguez-Fornells, A., 2008. Human oscillatory activity associated to reward processing in a gambling task. *Neuropsychologia* 46, 241–248.
- Marco-Pallarés, J., Cucurell, D., Cunillera, T., Krämer, U.M., Càmarà, E., Nager, W., Bauer, P., Schüle, R., Schöls, L., Münte, T.F., Rodríguez-Fornells, A., 2009. Genetic variability in the dopamine system (DRD4, COMT) modulates neurophysiological responses to gains and losses. *Biol. Psychiatry* 66 (2), 154–161.
- Mars, R.B., Debener, S., Gladwin, T.E., Harrison, L.M., Haggard, P., Rothwell, J.C., Bestmann, S., 2008. Trial-by-trial fluctuations in the event-related electroencephalogram reflect dynamic changes in the degree of surprise. *J. Neurosci.* 28, 12539–12545.
- Mellers, B.A., McGraw, P., 2001. Anticipated emotions as guides to choice. *Curr. Dir. Psychol. Sci.* 10, 210–214.
- Mellers, B.A., Schwartz, A., Ho, K., Ritov, I., 1997. Decision affect theory: emotional reactions to the outcomes of risky options. *Psychol. Sci.* 8, 423–429.
- Mellers, B.A., Schwartz, A., Ritov, I., 1999. Emotion-based choice. *J. Exp. Psychol. Gen.* 128, 332–345.
- Nieuwenhuis, S., Yeung, N., Holroyd, C.B., Schurger, A., Cohen, J.D., 2004. Sensitivity of electrophysiological activity from medial frontal cortex to utilitarian and performance feedback. *Cereb. Cortex* 14, 741–747.
- Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. *Clin. Neurophysiol.* 118, 2128–2148.
- Schultz, W., 2011. Potential vulnerabilities of neuronal reward, risk, and decision mechanisms to addictive drugs. *Neuron* 69, 603–617.
- Schultz, W., Dayan, P., Montague, P.R., 1997. A neural substrate of prediction and reward. *Science* 275, 1593–1599.
- Shannon, C.E., 1948. A mathematical theory of communication. *Bell Syst. Tech. J.* 27, 379–423.
- Smith, K.S., Berridge, K.C., Aldridge, J.W., 2011. Disentangling pleasure from incentive salience and learning signals in brain reward circuitry. *Proc. Natl. Acad. Sci. U. S. A.*
- Snodgrass, J.G., Vanderwart, M., 1980. A standardized set of 260 pictures: norms for name agreement, image agreement, familiarity, and visual complexity. *J. Exp. Psychol. Hum. Learn.* 6, 174–215.
- Sutton, S., Braren, M., Zubin, J., John, E.R., 1965. Evoked-potential correlates of stimulus uncertainty. *Science* 150, 1187–1188.
- Tobler, P.N., Fiorillo, C.D., Schultz, W., 2005. Adaptive coding of reward value by dopamine neurons. *Science* 307, 1642–1645.
- Tobler, P.N., Christopoulos, G.I., O'Doherty, J.P., Dolan, R.J., Schultz, W., 2009. Risk-dependent reward value signal in human prefrontal cortex. *Proc. Natl. Acad. Sci. U. S. A.* 106, 7185–7190.
- van Boxtel, G.J.M., Böcker, K.B.E., 2004. Cortical measures of anticipation. *J. Psychophysiol.* 18, 61–76.
- Yeung, N., Sanfey, A.G., 2004. Independent coding of reward magnitude and valence in the human brain. *J. Neurosci.* 24, 6258–6264.