Intracerebral mechanisms explaining the impact of incidental feedback on mood state and risky choice

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Abstract

Identifying factors whose fluctuations are associated with choice inconsistency is a major issue for rational decision theory. Here, we investigated the neuro-computational mechanisms through which mood fluctuations may bias human choice behavior. Intracerebral EEG data were collected in a large group of participants ($n = 30$), while they were performing interleaved quiz and choice tasks. Neural baseline activity preceding choice onset was confronted first to mood level, estimated by a computational model integrating the feedbacks received in the quiz task, and then to the weighting of option attributes, in a computational model predicting risk attitude in the choice task. Results showed that 1) elevated broadband gamma activity (BGA) in the ventromedial prefrontal cortex (vmPFC) and dorsal anterior insula (daIns) was respectively signaling periods of high and low mood, 2) increased vmPFC and daIns BGA respectively promoted and tempered risk taking by overweighting gain versus loss prospects. Thus, incidental feedbacks induce brain states that correspond to different moods and bias the comparison of safe and risky options. More generally, these findings might explain why people experiencing positive (or negative) outcome in some part of their life tend to expect success (or failure) in any other.

Keywords

Mood, decision, reward, risk, electrophysiology, oscillatory activity, broadband gamma, computational model, ventromedial prefrontal cortex, anterior insula

Introduction

Humans often makes inconsistent decisions, even when facing seemingly identical choices. This surprising variability is a major difficulty for rational decision theory and a reason for introducing stochastic functions in choice models. However, even if stochastic functions help mimicking behavior on average, they cannot help predicting individual choice. For this, one need to identify factors whose fluctuations are systematically associated with changes in preference, in order to replace randomness with bias. Influent factors can be related to the internal state of the decision maker and/or the external context of the choice situation. In neuroscience, many studies have shown that brain activity preceding the presentation of choice options might provide a bias on the eventual choice, particularly when alternatives are safe versus risky options (Chew et al., 2019; Huang et al., 2014; Kuhnen and Knutson, 2005; Lopez-Persem et al., 2016; Padoa-Schioppa, 2013; Vinckier et al., 2018).

In some cases, baseline brain activity could be related to mental constructs such as pleasantness, satiety or mood, which were themselves under the influence of external events (Abitbol et al., 2015; Vinckier et al., 2018). These findings therefore provide a putative mechanism explaining why mood is predictive of risky choice: positive/negative events that increase/decrease mood at the mental level also modulate baseline activity at the neural level, changing the way dedicated brain regions valuate risky options. More precisely, good mood would lead to overweighting gain prospects, and bad mood to overweighting loss prospects, making the overall expected value of a risky option positive or negative, depending on circumstances. Such a mechanism could account for the well-documented impact of mood on buying lottery tickets or investing in financial markets (Bassi et al., 2013; Otto et al., 2016; Saunders, 1993), an effect that has been reproduced in the lab (Arkes et al., 1988; Chou et al., 2007; Eldar and Niv, 2015). It could also account for why depressed and manic patients have opposite attitudes toward risk, respectively focusing on negative and positive outcomes of potential actions (Leahy, 1999; Yuen and Lee, 2003).

Despite the importance of this phenomenon, producing disastrous decisions at both the individual scale in psychiatric conditions and the societal scale in real-life economics, the underlying mechanism at the neural level is still poorly understood. This is due to the limitations of functional magnetic resonance imaging (fMRI), which has been mostly used because mood fluctuations are difficult to track in animals, while invasive techniques are forbidden in humans, for obvious ethical reasons. There is nonetheless a particular clinical situation that offers the opportunity to record intra-electroencephalographic (iEEG) activity from deep electrodes, when patients with refractory epilepsy are prior to surgery. These iEEG recordings have already been used to successfully decode mood from resting-state activity, a technical achievement that may open the route to closed-loop stimulation procedures (Bijanazadeh et al., 2019; Sani et al., 2018). They have also been used to specify the impact of lateral orbitofrontal cortex stimulation aiming at improving mood in moderately depressed patients (Rao et al., 2018). There is

however a discrepancy between these pioneering iEEG studies, which reported that changes in mood states might correspond to changes in the frequency of oscillatory activity (Bijanzadeh et al., 2019; Kirkby et al., 2018; Rao et al., 2018; Sani et al., 2018), and fMRI studies that have related mood fluctuations to the relative activity of opponent brain systems associated to either reward or punishment processing.

To examine whether good and bad mood are associated to activity in different brain regions or different frequency bands, we recorded iEEG activity while patients were performing a task similar to that used in a previous fMRI study to induce mood fluctuations and test their impact on risky choices (Vinckier et al., 2018). Mood fluctuations were induced by feedbacks provided to participants on their responses to general knowledge questions. Choices were about whether to accept a challenge rewarded with monetary gains in case of success but punished with monetary losses in case of failure. To avoid repeating self-report too frequently, a computational model was developed, building on previous suggestion (Eldar and Niv, 2015), to generate mood level as an integration of past feedbacks, the perception of which was itself modulated by mood level. This theoretical mood level was positively correlated with activity in brain regions classically associated with reward, such as the ventromedial prefrontal cortex (vmPFC), and negatively correlated with regions associated with punishment, such as the anterior insula (aIns). In turn, baseline activity in these regions (prior to the presentation of choice options) was predicting a bias on choice, relative to an expected utility model. Indeed, high vmPFC activity favored the risky option (accepting the challenge) by increasing the weight on potential gain, whereas high aIns activity favored the safe option (declining the challenge) by increasing the weight on potential loss. Yet, due to the poor temporal resolution of fMRI, there was a remaining gap in the explanation, regarding the contribution of activity in mood-related regions during decision making. Here, leveraging the excellent temporal resolution of iEEG, we intended to separate brain activity related to feedback events, to overall mood level and to the choice process.

Thus, in an attempt to clarify the neuro-computational mechanism through which mood fluctuations might arise and bias decisions under risk, we collected iEEG data in a large group of participants ($n = 30$), while they were performing interleaved quiz and choice tasks. The objectives of our analytical approach were 1) to identify brain regions in which activity was related to both mood state (good or bad) and choice (safe or risky), and 2) to examine whether mood fluctuations and related decisions were associated to a shift in frequency bands or in anatomical location of oscillatory iEEG activity.

Results

The aim of this study was to elucidate how intracerebral activity may underpin the impact of mood fluctuations on risky choices. Intracerebral electroencephalographic (iEEG) data were collected in thirty patients suffering from refractory epilepsy (39.5 ± 1.9 years old, 14 females, see demographical details in Table S1), while they performed three unrelated but interleaved tasks. The first was a quiz task designed as a mood induction procedure; the second was a rating task used to quantify mood level; the third was a choice task used to unravel the effects of mood fluctuations on economic choices (Figure 1a). In the quiz task, subjects were asked to answer general knowledge questions and received feedback on their response (correct, incorrect or too late). In order to predictably modulate mood, the difficulty of questions and the validity of feedbacks were manipulated, unbeknownst to the subjects, so as to create episodes of high and low correct feedback rate (see Methods). In 25% of the trials, the effect of feedback history was assessed by asking subjects to rate their mood on a visual analog scale. Otherwise (in 75 % of trials), the quiz and choice task were separated by a four-seconds rest period (black screen). In the choice task, subjects had to decide whether to accept or reject a given offer before performing a challenge consisting in stopping a moving ball inside a target. The offer comprised a gain prospect (i.e., the amount of money they would win in case of success), a loss prospect (i.e., the amount of loss in case of failure) and the difficulty of the upcoming challenge (target size). When accepting the offer, subjects played for the proposed amounts of money, otherwise, when declining, they played for minimal stakes (winning or losing 10 cents). All three choice dimensions (gain, loss and difficulty) were varied on a trial-by-trial basis. In order to avoid learning, and additional effects on mood, no feedback was provided regarding performance in the challenge (actual success rate was around 30% on average but significantly varied with difficulty). Subjects were explicitly informed that the three interleaved tasks were independent, such that responses to the quiz or mood ratings had no influence on choice options and hence on their monetary earnings.

Choice behavior

We first tested whether subjects performed the choice task correctly by checking that the three dimensions of the offer (gain prospect, loss prospect and challenge difficulty) were properly integrated into their choices (Figure 1b). Choice acceptance was fitted at the individual level using a logistic regression model that included the three dimensions and significance of regression estimates was assessed at the group level (across $n = 30$ subjects) using two-sided, one-sample, Student's t-tests. Results show that acceptance rate significantly increased with gain ($\beta_{\text{gain}} = 0.12 \pm 0.02$, $t(29) = 6.85$, $p = 2.10^{-7}$), but significantly decreased with loss ($\beta_{\text{loss}} = -0.10 \pm 0.02$, $t(29) = -5.51$, $p = 6.10^{-6}$) and difficulty ($\beta_{\text{diff}} = -0.03 \pm 0.01$, $t(29) = -2.97$, $p = 6.10^{-3}$). These results were expected given previous behavioral evidence with a similar choice task that subjects with epilepsy can integrate these three dimensions when making a decision (Vinckier et al., 2018).

We next assessed whether mood fluctuations impacted choices above and beyond the influence of these three dimensions. We thus tested the link between mood ratings and choice residuals, once the three dimensions had been regressed out (Figure 1c). To account for interactions and non-linearities in a principled way, we fitted a model based on expected utility theory, which was previously shown to best capture choices in this task (see Vinckier et al., 2018). In brief (see Methods for details), acceptance likelihood was estimated as a softmax function of expected utility, calculated as potential gain multiplied by probability of success (p_s , inferred from target size) minus potential loss multiplied by probability of failure ($1 - p_s$), with gain and loss terms weighted by distinct parameters (k_g and k_l respectively). Residuals of this choice model were then regressed against mood ratings at the individual level, and significance of regression estimates was assessed at the group level, using two-sided, one-sample, Student's t-test. Results indicate a significantly positive association between mood ratings and choice residuals ($\beta = 0.05 \pm 0.02$, $t(29) = 2.32$, $p = 0.027$), meaning that variability in choices, beyond that induced by the three dimensions of the offer, was indeed partially explained by mood fluctuations.

Modeling mood fluctuations

Because frequent assessment of subjective emotional states can lead to inconsistent ratings (Kahneman et al., 2004), mood ratings were collected in a minority of the trials (25%). To retrieve mood levels in trials where no rating was provided, we used computational modeling. To ensure the model was based on solid grounds, we checked beforehand that mood ratings were influenced by the expected factors related to the quiz task.

We first verified that our manipulation of feedbacks was effective (Figure 1d): indeed the proportion of correct feedbacks was higher in episodes with intended high versus low correct feedback rate (0.60 ± 0.01 vs. 0.21 ± 0.01 ; $t(29) = 22.99$, $p = 4.10^{-20}$; two-sided, paired Student's t-test) and so was mood rating (0.22 ± 0.04 vs. -0.12 ± 0.06 ; $t(29) = 3.76$, $p = 8.10^{-4}$, two-sided, paired, Student's t-test).

Figure 1: Behavioral task and results. **a** Trial structure. Each trial included a quiz task, a rest or mood assessment period and a choice task followed by a challenge. In the quiz task, subjects answered a general knowledge question and received a feedback. The quiz task was followed by a rest period (75% of trials) or a mood rating task (25% of trials) on a visual analog scale. In the choice task, subjects had to decide whether to accept or reject a given challenge by taking into account gain prospects (represented by a bunch of regular 10-cent coins), loss prospects (crossed out 10-cent coins) and difficulty (inversely proportional to the size of the blue bar in the middle of the screen). The challenge consisted in stopping a moving ball, invisible when inside the blue target. **b** Choice behavior. Acceptance probability is plotted as a function of the three objective dimensions (gain, loss, difficulty) and modeled subjective utility of the proposed challenge. Circles are binned data averaged across subjects. Yellow dotted lines represent acceptance probability as computed by the choice model. Error bars represent inter-subject S.E.M. **c** Impact of mood on the choice model residual error (actual choice – modeled acceptance probability). Left panel: residual error is plotted as a function of mood rating. Right panel: the weight of mood on residual error is shown as individual regression estimates. Circles represent individual data and horizontal line represents mean across subjects (as in d, left panel). **d** Mood fluctuations. Left panel: Effect of correct feedback rate on mood rating and theoretical mood level (TML). Right panel: difference in model evidence between TML and a null model in which feedback had no impact on mood. Bars show subjects ranked in ascending order. Stars indicate significance ($p < 0.05$).

We then fitted individual mood ratings with a previously established computational model (Vinckier et al., 2018) that formalizes a reciprocal influence between mood and feedback (see Methods). This means that mood level increases with positive feedback (reciprocally, decreases with negative feedback) and in turn that feedbacks are perceived as more positive when mood is higher (reciprocally, more negative when mood is lower). Posterior estimates of free parameters indicated that indeed, the weight of feedback on mood (parameter ω_f), was significantly positive across subjects ($t(29) = 4.72$, $p = 6.10^{-5}$; two-sided, one-sample, Student's t-test). Conversely, the weight of mood on feedback (parameter δ) was also significantly positive ($t(29) = 3.30$, $p = 0.0026$). The forgetting factor was 0.69 ± 0.04 , meaning that feedback received five trials earlier still had an impact corresponding to 23% of the most recent feedback. In addition, the model included a linear effect of time, weighted by parameter ω_t , which was not significantly different from zero, meaning that task duration had no significant influence on mood. Using fitted parameters for every patient, a theoretical mood level (TML) was then estimated on a trial-by-trial basis. As seen with mood rating (Figure 1d), TML was significantly higher during episodes with high vs. low correct feedback rate ($t(29) = 4.19$, $p = 2.10^{-4}$; two-sided, paired, Student's t-test). At the group level, Bayesian comparison indicated that the mood model was far more plausible (exceedance probability $X_p = 1$) than the null model assuming no influence of feedback but only the effect of time. However, the results of model comparison were more variable at the individual level (see Figure 1d), with the null model doing better than the mood model in 6 subjects (out of 30).

Intracerebral activity underpinning mood fluctuations

The next step was to establish a link between mood level and intracerebral electroencephalographic signals (iEEG). The iEEG dataset included a total of 3494 recording sites (bipolar montage, see Methods) acquired from 30 patients (Figure S1). For each patient, recording sites were localized within the native anatomical brain scan and labeled according to either MarsAtlas (Auzias et al., 2016), Destrieux (Destrieux et al., 2010) or Fischl (Fischl et al., 2002) parcellation schemes, with slight modifications (see Methods). Out of the 3494 recording sites, 3188 were free from artefacts and located within the grey matter of the 50 covered areas. Among these areas, 42 regions ($n = 3154$ sites) with at least 10 recording sites across at least four patients were retained in the electrophysiological analyses (see Methods). We initially focused on broadband gamma activity (50-150 Hz, BGA), as converging lines of evidence showed that BGA correlates positively with both fMRI and spiking activity (Lachaux et al., 2007; Manning et al., 2009; Mukamel, 2005; Niessing et al., 2005; Nir et al., 2007; Winawer et al., 2013), but subsequently took all frequency bands into consideration.

We extracted BGA time series from all retained recording sites and performed multiple linear regressions (one per time point) across trials between BGA and mood rating or TML. We time-locked this analysis to the [-4 to 0 s] time window before prospects onset, i.e. during the time period corresponding to the rest or mood assessment period. Significance was tested across all recording sites

within each ROI, using two-sided, one-sample Student's t-tests on regression estimates, with correction for multiple comparisons across time points using a non-parametric cluster-level statistic (see Methods). To identify brain regions that were reliably associated with mood fluctuations (Figure 2a), we focused our analyses on ROIs within which BGA was significantly associated with both mood rating and TML, with the additional constraint that the sign of the correlation had to be consistent.

The vmPFC was the only ROI for which we found a positive correlation (Figure 2b) between BGA and both mood rating (best cluster: -1.37 to -1.04 s, $\text{sum}(t_{(90)}) = 122.3$, $p_{\text{corr}} = 0.010$) and TML (best cluster: -0.57 to -0.13 s, $\text{sum}(t_{(90)}) = 132.4$, $p_{\text{corr}} = 8.10^{-3}$). Conversely, we found a negative correlation in a larger brain network encompassing the daIns (Figure 2b), in which BGA was negatively associated with both mood rating (best cluster: -3.36 to -2.51 s, $\text{sum}(t_{(85)}) = -325.8$, $p_{\text{corr}} < 1.7.10^{-5}$) and TML (best cluster: -3.13 to -2.72 s, $\text{sum}(t_{(85)}) = -136.4$, $p_{\text{corr}} = 9.10^{-3}$). The additional brain regions in which BGA was negatively associated with mood level involved the dorsolateral prefrontal cortex, the visual cortex, the motor cortex, the dorsomedial premotor cortex, the ventral somatosensory cortex and the ventral inferior parietal lobule. Note that in the ventral anterior insula, BGA was negatively associated with mood rating, but not with TML.

Thus, our two a priori ROIs signaled mood level with opposite signs, whether the correlation with BGA was tested during mood rating only or extended to all trials using TML to extrapolate mood ratings. We also verified that the association between TML and BGA (averaged within the best temporal cluster for each ROI) remained significant when only considering trials with no rating (vmPFC: $\beta = 0.031 \pm 9.10^{-3}$, $t(90) = 3.59$, $p = 5.10^{-4}$; daIns: $\beta = -0.02 \pm 8.10^{-3}$, $t(85) = -2.67$, $p = 9.10^{-3}$). To assess whether vmPFC and daIns regions would represent separate components of mood, we entered them into a single regression model meant to explain TML. In order to obtain the time course of mood expression in the two ROIs (Figure 2c), we performed multiple regression between TML and BGA from all possible pairs of vmPFC and daIns recording sites recorded in a same subject ($n = 247$ pairs of recording sites, see Methods) and tested the regression estimates across pairs within each ROI for each time point. The regression estimates were significant predictors of TML in both regions, but with opposite signs (best cluster for the vmPFC: -0.71 to 0 s, $\text{sum}(t_{(246)}) = 414.1$, $p_{\text{corr}} < 1.7.10^{-5}$; best cluster for daIns: -3.05 to -2.76 s, $\text{sum}(t_{(246)}) = -113.8$, $p_{\text{corr}} = 0.018$).

Thus, BGA in the two main ROIs carried non-redundant information about mood level. To further investigate which component of mood was signaled by each region (Figure 2d), we regressed BGA against TML separately for high- and low-mood trials (35% with highest vs. lowest TML). In the vmPFC, regression estimates were significantly positive for high-mood trials only ($\beta_{\text{high TML}} = 0.17 \pm 0.08$, $t(90) = 2.10$, $p = 0.039$; two-sided, one-sample, Student's t-test), not for low-mood trials. Conversely, in the daIns, regression estimates only reached significance for low-mood trials ($\beta_{\text{low TML}} = -0.14 \pm 0.06$, $t(85) = -2.39$, $p = 0.019$), not high-mood trials. This double dissociation suggests that the

vmPFC signals mood level when it gets better than average, and the daIns when it gets worse than average.

In order to check that TML was expressed above and beyond the component related to the last feedback (Figure 2e), we splitted TML into two regressors, one representing the last feedback only (+1 or -1) and one representing the integration of all preceding feedbacks, which is equivalent to TML estimated at the previous trial (see Methods). BGA in the best time clusters identified above were still significant predictors of TML (after removing the influence of the last feedback) in both the vmPFC ($\beta_{[-0.57 -0.13]} = 0.024 \pm 8.10^{-3}$, $t(90) = 2.99$, $p = 4.10^{-3}$) and daIns ($\beta_{[-3.13 -2.72]} = -0.021 \pm 8.10^{-3}$, $t(85) = -2.64$, $p = 0.010$; two-sided, one-sample, Student's t-test).

Finally, to ensure that our a priori focus on BGA was justified, we explored activity in other frequency bands (Figure 2f). For each frequency band and recording site, power time series were averaged over the pre-choice time window (-4 to 0 s) and regressed against TML. In the vmPFC, regression estimates were only significant in the high-gamma band ($\beta_{H\gamma} = 0.091 \pm 0.04$, $t(90) = 2.41$, $p = 0.018$; two-sided, one-sample, Student's t-test). In the daIns however, regression estimates were not only significantly negative in the gamma and high-gamma bands ($\beta_{H\gamma} = -0.067 \pm 0.03$, $t(85) = -2.18$, $p = 0.032$; $\beta_{\gamma} = -0.067 \pm 0.03$, $t(85) = -2.0$, $p = 0.048$), but also significantly positive in the theta and beta bands ($\beta_{\theta} = 0.082 \pm 0.04$, $t(85) = 2.31$, $p = 0.023$; $\beta_{\beta} = 0.062 \pm 0.02$, $t(85) = 2.85$, $p = 6.10^{-3}$). To check whether low-frequency bands could provide additional information about mood level, we fitted TML with all possible GLMs containing BGA together with any combination of other frequency bands (see Methods). In both vmPFC and daIns, Bayesian model selection designated the BGA-only GLM as providing the best account of TML (vmPFC: expected frequency $E_f = 0.99$, exceedance probability $X_p = 1$; daIns: $E_f = 0.99$, $X_p = 1$). Thus, even if other frequency band activity was significantly related to TML in daIns, it carried redundant information relative to that extracted from BGA. Hence, BGA was the best neural proxy for mood level, at least in our two main ROIs.

Figure 2: Intracerebral activity underpinning mood fluctuations. **a** Anatomical location of the vmPFC (blue) and daIns (red) in the standard Montreal Neurological Institute template brain, along with all recording sites located in these areas (dots) and the entire insula (in yellow). Anterior (A), posterior (P), dorsal (D), ventral (V), left (L) and right (R) directions are indicated. **b** Time course of estimates obtained from the regression of BGA against mood rating (left) or TML (right). **c** Time course of estimates obtained from the regression of vmPFC and daIns BGA included in a same GLM fitted to TML. In panels b-c, lines indicate means and shaded areas $\pm$ SEM across recording sites. Bold lines indicate significant clusters ($p_{\text{corr}} < 0.05$). Grey hatched areas indicate the time window within which the quiz feedback was provided to subjects. **d** Average estimates (within the best temporal cluster for each ROI) obtained from the regression of BGA against the 35% lower or higher TML. Bars are means and error bars are SEM across recording sites. **e** Average estimates (over the baseline window: -4 to 0 s before choice onset) obtained from the regression of BGA against TML after excluding the last feedback. Dots represent individual regression estimates for each recording site, horizontal lines and error bars respectively represent mean and SEM across sites within each ROI. **f** Association between TML and activity in frequency bands. For each frequency band, power time series were averaged over the baseline window and entered in a regression model meant to explain TML. θ : 4-8 Hz; α : 8-13 Hz; β : 13-33 Hz; γ : 33-49 Hz; $H\gamma$: 50-150 Hz. In panels d-f, stars indicate significance ($p < 0.05$) of regression estimates (two-sided, one-sample, Student's t-test).

Impact of baseline intracerebral activity on decision making

To identify which mood-related regions impacted choices, we regressed across trials the residuals of choice model fit against BGA (time-locked to choice onset), for every time point and recording site. Among regions that encoded mood levels in their baseline, regression estimates were significant in five ROIs (Figure 3a and Table S2), including positive correlation in vmPFC (best cluster: -1.64 to -1.31 s, $\text{sum}(t_{(90)}) = 91.2$, $p_{\text{corr}} = 0.020$) and negative correlation in daIns (best cluster: -0.95 to -0.67 s, $\text{sum}(t_{(85)}) = -85.2$, $p_{\text{corr}} = 0.029$). Taken together, these results mean that vmPFC and daIns baseline BGA not only express mood in opposite fashion, but also had opposite influence on upcoming choice.

Next, we tested whether baseline activity in our two ROIs was carried over choice-related activity so as to bias decision making (Figure 3b). For each contact of a given ROI, we regressed choices against BGA separately for trials with high and low baseline BGA (in the time window identified in the preceding analysis for each ROI). For the vmPFC, we found that when baseline BGA was high, choice-related BGA positively predicted choices (best cluster: -1.41 to -1.16 s, $\text{sum}(t_{(90)}) = 70.4$, $p_{\text{corr}} = 0.035$). In contrast, for the daIns, when baseline BGA was high, choice-related BGA negatively predicts choices (best cluster: -0.74 to -0.48 s, $\text{sum}(t_{(85)}) = -78.2$, $p_{\text{corr}} = 0.022$).

Finally, we investigated the computational mechanism through which baseline BGA (in the time window identified above for each ROI) could modulate decision making. For each contact of a given ROI, we fitted choices with a model in which the weights on the different terms of expected utility (k_0 , k_g , k_l , and σ ; see Methods) were modulated by baseline BGA (Figure 3c). Comparing posterior parameters obtained with high versus low BGA trials (obtained by median split), we found that k_g was significantly enhanced by vmPFC baseline ($t(90) = 3.16$, $p = 2.10^{-3}$; two-sided, paired, Student's t-test) whereas k_l was significantly enhanced by daIns baseline ($t(85) = 2.90$, $p = 5.10^{-3}$). This suggests that iEEG baseline fluctuations in these two mood-related regions had opposite effects: increased vmPFC BGA would lead to overestimating gain prospects, while increased daIns BGA would lead to overestimating loss prospects.

Figure 3: Impact of intracerebral activity on decision making. **a** iEEG activity predicting choice. Plots show the time course of estimates obtained from the regression of choice residuals against BGA in vmPFC (top, blue) and daIns (bottom, red), averaged across recording sites $\pm$ SEM (shaded areas). Bold lines indicate significant clusters ($p_{\text{corr}} < 0.05$). Grey hatched areas highlight the time window within which quiz feedback was provided to subjects. **b** Time course of estimates from the regression of choices against BGA performed separately for high vs. low baseline BGA trials, in vmPFC (top, blue) and daIns (bottom, red). Vertical line indicates the minimum response time of trials used in the regression (1.6 sec). **c** Impact of baseline BGA on choice model parameters. Plots show the difference in model weights (posterior parameters) between fits to high- vs. low-BGA trials. Significant modulation was only found for k_g (weight on potential gain) with vmPFC BGA and for k_l (weight on potential loss) with daIns BGA. Dots represent individual data, horizontal lines and error bars respectively represent mean and SEM across recording sites. Stars indicate significance ($p < 0.05$).

Discussion

In the present study, we used a large dataset of iEEG signals recorded in 30 subjects to provide a neuro-computational account of how mood fluctuations arise and impact risky choice. We found that 1) baseline BGA in vmPFC and daIns was respectively signaling periods of high and low mood induced by incidental feedbacks, 2) beyond BGA, oscillatory activity in other frequency bands did not provide any additional information about mood level, 3) high vmPFC baseline BGA promoted risk taking by selectively increasing the weight of potential gains, whereas high daIns baseline BGA tempered risk taking by increasing the weight of potential losses, 4) baseline BGA in both regions was carried over BGA during decision making, which was predictive of the eventual choice. In the following, we successively discuss how BGA in these two regions relate to mood and choice.

BGA and mood

To avoid asking subjects to rate their mood on every trial, we used a computational model to interpolate mood level between mood ratings. The model was inspired from previous suggestions (Eldar and Niv, 2015; Rutledge et al., 2014) and already validated in a previous publication (Vinckier et al., 2018). The basic assumption is that mood is nothing but a weighted sum of positive and negative events (here, feedbacks received in the quiz task), the more recent having more weight than the more distant in the past. In addition, the model postulates that the way feedbacks are perceived are also affected by mood, in the sense that a same feedback is perceived as more positive when we are in a better mood. This makes reciprocal the influence between internal states (mood) and external events (feedback). The model was inverted on the basis of mood ratings and compared to a null model, where mood is just drifting with time, without any influence of feedback. Even if the mood model was largely winning the Bayesian comparison at the group level, its evidence was surpassing the null model in about two thirds of participants, a proportion close to that reported in the previous publication (Vinckier et al., 2018). We nevertheless kept all patients in subsequent analyses to reach more robust conclusions that might be generalized to the entire population. Posterior estimates of free parameters confirmed the reciprocal influence between feedback and mood, the forgetting factor suggesting that mood was still impacted by the feedback received five trials in the past (about two minutes before), with a weight about four times lesser than the last feedback. Thus, the time scale of mood fluctuations was longer than acute emotional reactions to single stimuli, but much shorter than those observed in mood disorders.

When looking for neural correlates of mood fluctuations, we used both raw ratings and modeled mood levels as probes. The results were qualitatively similar but statistically more significant with theoretical mood level because it allowed including all trials in the analysis. To identify brain regions that could mediate the impact of mood on choice, we selected all regions that were significantly associated to both mood level (good or bad) and choice (safe or risky). We found the two main bilateral regions that were already identified with fMRI (Vinckier et al., 2018), plus unilateral visual or motor regions that were

likely related to the side of the behavioral response. We note however that the anterior insula region defined anatomically corresponds to a dorsal part of the cluster identified with fMRI (hence the appellation of daIns) and that the vmPFC region included many recorded sites located in more ventral areas than the fMRI cluster. As with fMRI, we observed that correlation with mood level was positive in the vmPFC and negative in the daIns. This is consistent with many studies implicating the vmPFC in reward learning and the daIns in punishment learning (Fouragnan et al., 2018; Garrison et al., 2013; Liu et al., 2011; Palminteri and Pessiglione, 2017), including studies using iEEG (Gueguen et al., in press). What we additionally show here is that the vmPFC and daIns are not just sensitive to the last reward or punishment outcome as in learning paradigms, but integrate feedbacks over a longer time scale. Indeed, the time course of regression estimates showed significant association with mood in time windows much later than the response to the last feedback, and the association remained significant when removing the impact of the last feedback from the computation of theoretical mood level used as regressor. We note that some expected regions are missing in our list, notably the ventral striatum, which has been shown to correlate positively with mood level (Eldar and Niv, 2015; Rutledge et al., 2014; Young and Nusslock, 2016). This region could not be investigated here because it was simply not sampled by the electrodes implanted for clinical purposes (i.e., for localization of epileptic foci). More generally, an inherent limitation to iEEG is that parts of the brain are less sampled than others, so null results should be considered with caution, because of huge differences in statistical power across regions. Nevertheless, whole-brain analysis using fMRI showed that with this induction procedure, activity in other regions such as the ventral striatum or dorsal anterior cingulate cortex adds nothing to decoding of mood level (Vinckier et al., 2018).

Having identified the two main regions reflecting mood fluctuations, we tested whether mood fluctuations were associated to similar shifts in the frequency of oscillatory activity as was previously suggested (Bijanzadeh et al., 2019; Kirkby et al., 2018; Rao et al., 2018). On the contrary, we observed that higher mood had opposite effects on high-frequency oscillations in the two regions, with increased BGA in the vmPFC but decreased BGA in the daIns. Thus, mood fluctuations were better accounted for by relative high-frequency activity (BGA) in the two opponent regions. Indeed, when included in the same model, vmPFC and daIns BGA were both significant predictors of mood level. Conversely, including low-frequency activity in the model did not help predicting mood level. The information carried in low-frequency activity was therefore at best redundant with that of high-frequency activity, whereas the two regions carried at least partly independent information. When analyzing separately bad, neutral and good mood levels, we found that higher BGA signaled the two extreme tertiles: good mood in the vmPFC and bad mood in the daIns. This result suggests that the two regions were specifically concerned with good and bad mood, explaining why they were not just anti-correlated. Although we used a unidimensional rating going from bad to good mood, it could be that mood is in fact bidimensional, meaning that good and bad mood would be better conceived as independent components

(Watson et al., 1988), relying on distinct brain systems. This is in keeping with clinical practice: absence of positive affect or excess of negative affect are considered as two independent criteria for depression.

The observation that mood is signaled by distinct brain systems and not different frequency bands all over the brain as in previous iEEG studies (Bijanzadeh et al., 2019; Kirkby et al., 2018; Rao et al., 2018) might be related to the use of positive and negative events to induce mood fluctuations and not direct stimulation of the cortical surface, which might put constraints on oscillatory activity. Besides, the good correspondence with previous fMRI results validates the shared view that BGA represents a time-resolved neural index of local neuronal activity, which typically correlates with both hemodynamic response and local spiking activity (Lachaux et al., 2007; Manning et al., 2009; Mukamel, 2005; Niessing et al., 2005; Nir et al., 2007; Winawer et al., 2013). We have confirmed this correspondence every time we compared iEEG and fMRI activity during the same behavioral paradigm (Gueguen et al., in press; Lopez-Persem et al., 2020).

BGA and risky choice

Choice to accept or reject the challenge in our task was significantly modulated by the three attributes displayed on screen: gain prospect (in case of success), loss prospect (in case of failure) and difficulty of the challenge. We combined the three attributes using a standard expected utility model and examined the residuals after removing the variance explained by the model. Those residuals were significantly impacted by mood level, meaning that on top of the other factors, good / bad mood inclined subjects to accept / reject the challenge. The same was true for neural correlates of mood: higher baseline BGA in the vmPFC / daIns was associated to higher accept / reject rates, relative to predictions of the choice model. Thus, different mood levels might translate into different brain states that predispose subjects to make risky or safe decisions.

The remaining question is how baseline BGA in these regions can be mechanistically related to risky choice. To address this question, we examined BGA during decision making, which could be separated from baseline BGA thanks to the temporal resolution of iEEG recordings. We found some carry-over: higher BGA in the vmPFC / daIns again predicted the likelihood of accept / reject choices. This does not mean that the link between BGA and choice was maintained all along, it was actually lost at the offer onset and retrieved before the behavioral response. This pattern could not be observed with fMRI but was already reported regarding the subjective value signals (Lopez-Persem et al., 2020). This result suggests that the impact of baseline activity in mood-related regions was mediated by the contribution of the same regions (vmPFC and daIns) to the decision-making process. These regions have already been implicated in computing the subjective values that are compared to make a choice, with the vmPFC and daIns respectively providing positive and negative value signals (Bartra et al., 2013; Pessiglione and Delgado, 2015). Thus, the relative activity of two regions may provide an impulse to accept or reject an offer, depending on whether the differential is positive or negative.

An additional explanation would be that the two regions not only provide subjective values with opposite signs, but also that they provide values that differ in how the different attributes are weighted. To test this idea, we compared parameters of the choice model fitted separately onto trials with high versus low baseline BGA in the vmPFC and daIns. We found that high BGA in the vmPFC / daIns was respectively associated to overweighting of gain and loss prospects, and not just the constant that captures the global tendency to accept or reject. This result makes the link with the idea that we may see a glass half-full or half-empty when we are in a good or bad mood. It is also consistent with reports that pre-stimulus spiking or hemodynamic activity in the vmPFC predicts how much rewards are liked (Abitbol et al., 2015; Lopez-Persem et al., 2020), whereas pre-stimulus activity in the daIns predicts how aversive punishments are perceived (Caria et al., 2010; Wiech et al., 2010). Overall, our findings suggest that mood-related neural fluctuations impact the valuation process in a valence-specific way, implemented by functionally opponent brain systems.

An obvious limitation of these findings is that although the dataset was collected in patients with epilepsy, we make interpretations as if it was collected in healthy subjects, assuming that pathological activity did not influence the neural or cognitive processes of interest. What was reassuring is that the behavior was remarkably comparable to that of young participants performing a similar task, while the iEEG results were fully compatible with fMRI results (Vinckier et al., 2018). Also, it does not seem very plausible that artifacts would by chance correlate, across task trials in patients with different forms of epilepsy, with the computational variables that were related to both neural and behavioral results. Indeed, in previous studies where we compared results with and without removing artifactual trials, results were unchanged. In fact, those trials can only degrade the correlation with computational variables, such that keeping them ensures that results would be more robust to replication.

In summary, we used intracerebral recordings in humans to specify the neuro-computational mechanisms through which mood fluctuations might arise from external events and impact risky choice. At longer time scales, these mechanisms could explain why people take more gambles (or less) after incidental events such as the victory (or defeat) of their favorite sport team, an effect that might be exacerbated in pathological conditions such as during manic (or depressive) episodes. The impact of mood on choice is a form of generalization, across different sources of reward and punishment, that may be catastrophic in pathological cases, leading people to believe that because they experienced positive (or negative) outcome in some part of their life, they are likely to succeed (or fail) in any other.

Material & Methods

Patients and electrode implantation

Intracerebral recordings were obtained from 30 patients suffering from drug-resistant focal epilepsy (39.5 ± 1.9 years old, 14 females, see demographical details in Table S1) in 7 different epilepsy centers (Rennes University Hospital: $n = 10$; Grenoble University Hospital: $n = 9$; Lyon Neurological Hospital: $n = 3$; Prague Motol University Hospital: $n = 3$; Marseille La Timone Hospital: $n = 2$; Toulouse University Hospital: $n = 2$; Nancy University Hospital: $n = 1$). These patients underwent intracerebral recordings by means of stereotactically implanted multilead depth electrodes (sEEG) in order to locate epileptic foci that could not be identified by non-invasive methods. Electrode implantation was performed according to routine clinical procedures and all target structures for the pre-surgical evaluation were selected strictly according to clinical considerations with no reference to the current study. Nine to twenty semi-rigid electrodes were implanted per patient. Each electrode had a diameter of 0.8 mm and, depending on the target structure, contained 6-18 contact leads of 2 mm wide and 1.5-4.1 mm apart (Dixi Medical, Besançon, France). All patients gave written, informed consent before their inclusion in the present study, which received approval from the local ethics committees (CPP 09-CHUG-12, study 0907; CPP18-001b / 2017-A03248-45; IRB00003888; CER No. 47-0913).

Intracerebral EEG recordings

Neuronal recordings were performed using video-EEG monitoring systems that allowed for simultaneous recording of 128 to 256 depth-EEG channels sampled at 512, 1024 or 2048 Hz (depending on the epilepsy center). Acquisitions were made with Micromed (Treviso, Italy) system and online band-pass filtering from 0.1 to 200 Hz in all centers, except for Prague (Two Natus systems were used: either a NicoletteOne with a 0.16-134 Hz band-pass filtering or a Quantum NeuroWorks with a 0.01-682 Hz band-pass filtering) and Marseille (Deltamed system, 0.16 Hz high-pass filtering). Data were acquired using a referential montage with reference electrode chosen in the white matter. Before analysis, all signals were re-referenced to their nearest neighbor on the same electrode, yielding a bipolar montage.

Behavioral tasks

Presentation of visual stimuli and acquisition of behavioral data were performed on a PC using custom Matlab scripts implementing the PsychToolBox libraries (Brainard, 1997). All patient responses were done with a gamepad (Logitech F310S) using both hands. Subjects performed a choice task combined with a mood induction procedure adapted from a previous study (Vinckier et al., 2018). Subjects completed two ($n = 6$) or three ($n = 24$) sessions of the experiment, consisting of 64 trials each, for a total of 128 or 192 trials. Each trial included 3 sub-parts: a quiz task, a rest or mood assessment period and a choice task.

Quiz task. In this task, a question and four possible answers were displayed on the screen. The question was randomly selected from a set of 256 possible questions that were adapted from the French version of the “trivial pursuit” game (e.g. Where is Park Güell located?) which were slightly adapted for Prague’s epilepsy center. Subjects had to select the correct answer using the up and down keys and confirm their answer using the confirmation key within a maximum of 8 seconds. A feedback of 1 second was finally given (either a smiling face with a bell sound or a grimacing face with a buzzer sound) immediately after the answer or at the end of the available time if no answer had been made.

In order to predictably manipulate subjects’ mood, episodes of high and low correct response rates were created unbeknownst to them by handling questions difficulty and feedbacks. Thus, questions were sorted by difficulty (assessed by mean accuracy estimated previously in a sample of healthy subjects (Vinckier et al., 2018)) and grouped so as to create easy and hard episodes. Within a given session of 64 trials, we created one episode of 20 trials with easy questions followed by one episode of 20 trials with hard questions and three episodes of 8 trials with questions of medium difficulty. The episodes were organized so that easy and hard episodes were always preceded and followed by episodes of medium difficulty (e.g., medium – easy – medium – hard – medium). The order of easy and difficult episodes was counterbalanced across sessions and subjects. Furthermore, feedback was biased such that a wrong answer could lead to a positive feedback (whereas a correct answer always led to a positive feedback). The proportion of biased feedback depended on the difficulty of the episode: 50% in easy, 25% in medium and 0% in hard episode. Post-hoc debriefing showed that no patient was aware of this manipulation.

Mood rating/rest period. Mood assessment or rest period began with a 500 ± 100 ms black screen used to ensure that a reasonable delay occurred after the last quiz feedback (which lasted 1 s). In 25% of the trials (i.e., in 16 out of 64 trials), the quiz task was followed by a mood rating task in which subjects were explicitly asked to rate their mood by answering the following question “How do you feel right now?”. Subjects had to answer by moving a cursor from left (very bad) to right (very good) along a continuous visual analog scale (100 steps) with left and right hand response buttons. Subjects were given at least 4 seconds to confirm their ratings with the confirmation button. Their response was then maintained on the screen until the end of the 4 seconds, so that they had no reason to speed-up their estimation. However, subjects were also discouraged from being too long to respond, as when they had not confirmed their rating within 4 seconds, a red message was displayed on the screen saying “Please validate your answer”. The initial position of the cursor on the scale was randomized to avoid confounding mood level with movements’ quantity. The position of mood ratings across trials within a session was evenly distributed and pseudo-randomized so that mood ratings were not predictable for the subjects, with the additional constraints that two mood ratings were spaced by a minimum of 2 trials and a maximum of 6 trials. In the remaining 75% of trials, the quiz task was followed by a rest period

consisting of a 4-second black screen. Therefore, the delay between the end of the quiz feedback and the beginning of the choice task was kept to a minimum of 4.5 ± 0.1 seconds.

Choice task: The choice task began with the presentation of an offer consisting of three dimensions: a gain prospect (represented by a bunch of 10-cent coins, range: 1-5€), a loss prospect (represented by crossed out 10-cent coins, range: 1-5€) and the upcoming challenge difficulty (represented by the size of a target window located at screen center, range: 1-5 corresponding to 75-35% theoretical success; see training section for further details about how difficulty was adjusted to each participant). Subjects were asked to accept or reject this offer, by pressing a left or right button depending on where the choice option (“yes” or “no”) was displayed. Subjects’ choice determined the amount of money at stake: accepting meant that they would eventually win the gain prospect or lose the loss prospect based on their performance in the upcoming challenge, whereas declining the offer meant playing the challenge for minimal stakes (winning 10 cents or losing 10 cents).

The sequence of trials was pseudo-randomized such that all possible combinations of the three dimensions (gains, losses and challenge difficulty), continuously sampled along four intervals ([1-2], [2-3], [3-4], [4-5]), were displayed for one patient across sessions, with greater sampling of medium difficulty combinations ([2-3] and [3-4]) to maximize the occurrence of undetermined choices during which small ongoing fluctuations were previously shown to bias subsequent choices (Padoa-Schioppa, 2013). The positions of gain and loss prospects were randomly determined to be either displayed on top or bottom of the screen and similarly, the choice options (“yes” or “no”) were randomly displayed on the left or right.

Subjects had a free time delay to accept or decline the offer. If they declined the offer, a 500 ms screen displayed the new offer (only gains and loss prospects changed so that subjects performed the challenge for a minimal stake of 10 cents). Thus, an important feature of the choice task was that the challenge was performed regardless of the choice answer to prevent subjects to eventually reject more offers to decrease experiment duration.

The challenge started 200ms after choice confirmation: a ball appeared on the left of the screen and moved, horizontally and at constant speed, towards screen center. Subjects were asked to press the confirmation button when they thought the ball was inside the basket displayed at screen center (i.e., the target window which size index the difficulty of the challenge). To facilitate the challenge, which was performed without any feedback, the ball always reached the center of the target after 1s. Thus, the size of the target window (i.e. the difficulty of the trial) represented the margin of error tolerated in patient response time (target: 1s after the movement onset of the ball). The larger the basket, the greater the tolerated spatiotemporal error was to consider a trial as successful, and therefore the easier was the trial. Importantly, the moving ball could only be seen during the first 500ms (half of the trajectory), and subjects had to extrapolate the last 500ms portion of the ball’s trajectory to assess whether the ball was

inside the target. No feedback was given to subjects about their performance or payoff after the challenge to prevent learning effects and also choice feedback effects on mood. However, to improve subjects' motivation to perform the task as accurately as possible, the total amount of money earned by the subjects during a session (calculated by adding gains and losses across all trials) was displayed at the end of a session.

Training: Before the main experiment, a training - divided into three steps - familiarized subjects with all sub-parts of the task. In the first step, subjects were familiarized with the challenge by performing 28 trials with a tolerated margin of error from ± 130 to ± 80 ms (56 trials if the accuracy of the first 28 trials was below 50%). Each training trial was followed by feedback informing whether the challenge was successful ("ok" in green) or missed ("too slow" or "too fast" in red). In the second step, subjects performed 64 trials of the full choice (i.e., the challenge was always preceded by an offer), and a feedback on the money won/lost in the trial was displayed at the end of each trial. The goal was to train subjects to properly integrate the three dimensions of the offer (gains, losses and difficulty) when making their choice. To help subjects learning the correspondence between the target size and challenge difficulty, trials were displayed by increasing difficulty level. Finally, the third and last part of the training (10 trials including 2 mood ratings) was totally similar to the main task to allow subjects to familiarize with the quiz task and the mood ratings.

Another purpose of the training was also to tailor the difficulty of the challenge to each patient's ability. To do so, a tolerated margin of error was computed for each difficulty level, ranging from 75% (level 1) to 35% (level 5) of theoretical success which we estimated from each individual subjects by assuming that errors were normally distributed. Note that during training, the difficulty levels were updated after each trial (average and standard deviation of challenge performance were updated), while in the main task, the mean and standard deviation of patient performance (and therefore difficulty levels) were set based on every challenge performed during the training. The range of tolerated margins of error between subjects ranged from $[\pm 72 \text{ ms (level 1) to } \pm 22 \text{ ms (level 5)}]$ in the most precise patient to $[\pm 198 \text{ ms to } \pm 123 \text{ ms}]$ in less precise one.

Computational models

Mood ratings and choices were fitted using published computational framework (Vinckier et al., 2018).

Mood model: As mood was sampled in 25% of the trials, we linearly interpolated ratings in order to get one data point per trial. In all analyses, mood ratings were z-scored. A theoretical mood level (TML) was computed for each trial through the integration of quiz feedback as follows:

$$TML(t) = \omega_0 + \omega_f \sum_{j=1}^t \gamma^{t-j} F(j) + \omega_t t$$

where t is the trial index, γ and all ω are free parameters (ω_0 is a constant and all other ω are weights on the different components; γ , with $0 \leq \gamma \leq 1$, is a forgetting factor that adjusts the influence of recent events relative to older ones) and F is the subjective perception of feedback. This feedback was subjective as its perception was in return biased by TML:

$$F(t) = \text{Feedback}(t) + \delta \times TML(t - 1)$$

where δ is a free parameter and $TML(t - 1)$ is the TML carried from previous trial, before updating based on the feedback received in the current trial. The model assumes that mood effect is additive so that good mood leads events to be seen as more positive than they objectively are (a multiplicative effect would imply that a negative feedback is perceived as even worst when one is in a good mood) and allowed an asymmetrical influence of positive and negative events on mood, using a free parameter R (with $R > 0$) for positive feedback instead of 1:

$$\text{Feedback}(t) = R \text{ or } -1$$

This model was compared to a control model in which only time was taken into account (linear function of trial index).

Choice model: Acceptance probability was calculated as a sigmoid function (softmax) of expected utility:

$$p(\text{accept}, t) = \frac{1}{1 + e^{-(\text{utility} + k_t \times t)}}$$

where k_t is a free parameter that accounts for a linear drift with time (trial index t) in order to capture fatigue effects. The utility function is based on expected utility theory where potential gains and losses are multiplied by probability of success (p_s) vs. failure ($1 - p_s$):

$$\text{utility} = k_0 + p_s \times k_g \times \text{gain} - (1 - p_s) \times k_l \times \text{loss}$$

However, distinct weights were used for the gain and loss components (k_g and k_l respectively), and a constant k_0 was added in order to capture a possible bias. The subjective probability of success (p_s) was inferred from the target size. The model assumes that subjects have a representation of their precision following a Gaussian assumption, meaning that the subjective distribution of their performance could be defined by its mean (the required 1 second to reach target center) and its width (i.e., standard deviation) captured by a free parameter σ . Thus, the probability of success was the integral of this Gaussian bounded by the target window:

$$p_s = \frac{1}{\sigma\sqrt{2\pi}} \int_{1-\text{Size}/2}^{1+\text{Size}/2} e^{\frac{-(x-1)^2}{2\sigma^2}} dx$$

Both models (mood and choice models) were inverted, for each patient separately with behavioral data, using the Matlab VBA toolbox (available at <https://mbb-team.github.io/VBA-toolbox/>), which implements Variational Bayesian analysis under the Laplace approximation (Daunizeau et al., 2014). This algorithm not only inverts nonlinear models, but also estimates the model evidence, which represents a trade-off between accuracy (goodness of fit) and complexity (degrees of freedom) (Robert, 2007).

Behavioral analysis

Statistical analyses were performed with Matlab Statistical Toolbox (Matlab R2018a, The MathWorks, Inc., USA). All dependent variables (raw or z-scored behavioral measures, regression estimates and model outputs) were analyzed at the subject level and tested for significance at the group level using two-sided, one or paired-sample, Student's t-tests. All means are reported along with the standard error of the mean.

sEEG pre-processing

Before analysis, bad channels were removed based on a supervised machine-learning model trained on a learning database with channels already classified by experts and using a set of features quantifying the signal variance, spatiotemporal correlation and non-linear properties (Tuyisenge et al., 2018). All signals were then re-referenced with a local bipolar montage between adjacent contacts of the same electrode to increase the spatial specificity of the effects by canceling out effects of distant sources that spread equally to both adjacent contacts through volume conduction. The average number of recording sites (one site corresponding to a bipolar contact-pair) recorded per patient was 116 ± 7 . Finally, all signals were down-sampled to 512 Hz.

Neuroanatomy

The electrode contacts were localized and anatomically labeled using the IntrAnat Electrodes software (Deman et al., 2018), developed as a BrainVisa (Rivière et al., 2009) toolbox. Briefly, the pre-operative anatomical MRI (3D T1 contrast) and the post-operative image with the sEEG electrodes (3D T1 MRI or CT scan), obtained for each patient, were co-registered using a rigid-body transformation computed by the Statistical Parametric Mapping 12 (SPM12) (Ashburner, 2009) software. The gray and white matter volumes were segmented from the pre-implantation MRI using Morphologist as included in BrainVisa. The electrode contact positions were computed in the native and MNI referential using the spatial normalization of SPM12 software. Coordinates of recording sites were then computed as the mean of the MNI coordinates of the two contacts composing the bipole. For each patient, cortical parcels were obtained for the MarsAtlas (Auzias et al., 2016) and Destrieux (Destrieux et al., 2010) anatomical atlases, while subcortical structures were generated from Fischl et al. (Fischl et al., 2002, as included in

Freesurfer). Each electrode contact was assigned to the gray or white matter and to specific anatomical parcels by taking the most frequent voxel label in a sphere of 3 mm radius around each contact center.

The MarsAtlas parcellation scheme was mainly used to label each recording site. This atlas relies on a surface-based method using the identification of sulci and a set of 41 regions of interest (ROIs) per hemisphere. These regions were completed with 7 subcortical regions, obtained from the procedure described by Fischl et al. (as included in Freesurfer (Deman et al., 2018)). However, based on the literature, we applied slight modifications concerning our regions of interest. First, boundaries based on MNI coordinates were set to the ventromedial prefrontal cortex (vmPFC) region so that contacts more lateral than $x = \pm 12$ and more dorsal than $z = 10$ were excluded from the parcel (Lopez-Persem et al., 2019). Secondly, MarsAtlas parcellation scheme involved the insular cortex as a single region, making it impossible to distinguish sub-insular areas that appear to have distinct functional properties in decision-making (Droutman et al., 2015). We therefore used the Destrieux atlas (performed by Freesurfer (Deman et al., 2018)) and MNI coordinates to segment the region corresponding to the insula into 3 sub-regions: (i) the ventral anterior insula (vaIns) corresponds to the anterior part ($y < 5$ in MNI space) of parcels 18 (G_insular_short), 47 (S_circular_insula_ant) and 48 (S_circular_insula_inf) of the Destrieux atlas, (ii) the dorsal anterior insula (daIns) corresponds to the anterior part ($y < 5$ in MNI space) of parcel 49 (S_circular_insula_sup) of the Destrieux atlas, and finally (iii) the posterior insula (pINS) corresponds to the posterior part ($y > 5$ in MNI space) of parcels 17 (G_Ins_lg_and_S_cent_ins), 48 and 49 of the Destrieux atlas, leading to a total of 50 ROIs.

For statistical analyses, only the 42 ROIs with at least 10 recording sites recorded across at least four subjects were retained. Among the 3494 initial recorded sites, 3154 recording sites were located within one of these 42 regions and were therefore kept for analysis. Note that data from both hemispheres were collapsed to improve statistical power.

Extraction of frequency envelopes

The time course of broadband gamma activity (BGA) was obtained by band-pass filtering of continuous sEEG signals in multiple successive 10 Hz-wide frequency bands (i.e., 10 bands, beginning from 50-60 Hz up to 140-150 Hz) using a zero-phase shift non-causal finite impulse filter with 0.5 Hz roll-off. The envelope of each band-pass filtered signal was next computed using the standard Hilbert transform. For each frequency band, this envelope signal (i.e., time varying amplitude) was divided by its mean across the entire recording session and multiplied by 100. This yields instantaneous envelope values expressed in percentage (%) of the mean. Finally, the envelope signals computed for each consecutive frequency band were averaged together to provide a single time series (the broadband gamma envelope) across the entire session. By construction, the mean value of that time series across the recording session is equal to 100. Note that computing the Hilbert envelopes in 10Hz sub-bands and normalizing them individually before averaging over the broadband interval allows to counteract a bias toward the lower frequencies

of the interval induced by the 1/f drop-off in amplitude. Finally, the obtained time series were smoothed using a sliding window of 250 ms, to get rid of potential artifacts, and down-sampled at 100 Hz (i.e., one-time sample every 10 ms).

The envelopes of theta, alpha, beta and gamma bands were extracted in a similar manner as the BGA except that steps were 1 Hz for theta and alpha, 5 Hz for beta and 4 Hz for gamma. BGA frequency range was defined as 50-150 Hz, gamma as 33-49 Hz, beta as 13-33 Hz, alpha as 8-13 Hz and theta as 4-8 Hz.

Electrophysiological analyses

The frequency envelopes of each recording site were epoched at each trial from 5600 ms prior choice onset to 500 ms after choice onset, encompassing the quiz feedback, the rest period (or mood assessment) between quiz and choice tasks and the choice onset (display of the offer). Electrophysiological data were analyzed using General Linear Models (GLM), providing a regression estimate for each time point and contact.

In a first GLM aimed at identifying areas underpinning mood fluctuations, power P (normalized envelope) was regressed across trials against mood M (real mood ratings or TML, normalized within subjects) at every time point:

$$P = \alpha + \beta M + \epsilon$$

With α corresponding to the intercept, β corresponding to the regression estimate on which statistical tests are conducted and ϵ corresponding to the error term.

To investigate whether vmPFC and daIns were sensitive to a specific mood state, the same GLM was performed except that BGA of each ROI was averaged over the best cluster identified in this first GLM (from -0.57 to -0.13 s before choice onset for the vmPFC and from -3.13 to -2.72 s for the daIns) and regressed across the 35% trials with the best or worst TML.

Next, to assess whether vmPFC and daIns regions would represent separate components of mood, TML was regressed across trials against BGA of these two regions at every time point as follows:

$$TML = \alpha + \beta_1 BGA_{vmPFC} + \beta_2 BGA_{daINS} + \epsilon$$

With α corresponding to the intercept, β_1 and β_2 corresponding to the regression estimates on which statistical tests are conducted and ϵ corresponding to the error term. For each of the 18 subjects with electrodes in both ROIs, regression was done for all possible pairs of vmPFC and daIns recording sites recorded within a given patient, leading to a total of $n = 247$ pairs of recording sites.

To check that TML was expressed above and beyond the variance induced by the last feedback, BGA was regressed across trials t against last feedback along with TML from the previous trial at every time point:

$$BGA(t) = \alpha + \beta_1 Feedback(t) + \beta_2 TML(t - 1) + \epsilon$$

Where α corresponds to the intercept, β_1 and β_2 correspond to the regression estimates on which statistical tests are conducted and ϵ corresponds to the error term.

Next, to assess the effect of pre-choice brain activity on acceptance rate, the residual error of choice model fit C was regressed across trials against power P (normalized envelope) at every time point as follows:

$$C = \alpha + \beta P + \epsilon$$

With α corresponding to the intercept, β corresponding to the regression estimate on which statistical tests are conducted and ϵ corresponding to the error term.

Finally, to assess how baseline activity affects choice, the frequency envelopes of each recording site were epoched at each trial from 2000 ms prior choice response to 500 ms after choice response, and trials were split into “high” or “low” baseline activity based on the average pre-choice BGA in the best cluster identified in the GLM used to investigate effect of pre-choice brain activity on acceptance rate (from -1.64 to -1.31 s before choice onset for the vmPFC and from -0.95 to -0.67 s for the daIns). Choices were then regressed across the 40% highest or lowest baseline activity trials against BGA at every time point, as in the previous GLM. To ensure that baseline activity does not interfere with the reported effect, trials with a choice response time shorter than 1.5 up to 2 secs were eliminated from this analysis. Note that the RT threshold (from 1.5 to 2s) did not affect the result. We reported results from the analysis removing trials with a response time shorter than 1.6 seconds (longest response time removing less than 25% of trials).

For all GLMs, the statistical significance of each recording site was assessed through permutation tests. The pairing between responses and predictors across trials was shuffled randomly 300 times for each site. The maximal cluster-level statistics (the maximal sum of t-values across contiguous time points passing a significance threshold of 0.05) were extracted for each shuffle to compute a ‘null’ distribution of effect size across a time window of -4 to 0 s before choice onset (the baseline corresponding to the rest or mood assessment period). The p-value of each cluster in the original (non-shuffled) data was finally obtained by computing the proportion of clusters with higher statistics in the null distribution, and reported as the ‘cluster-level corrected’ p-value (p_{corr}).

For each ROI, a t-value was computed across all recording sites of the given ROI for each time point of the baseline (-4 to 0 s before choice onset), independently of patient identity, using two-sided, one-

sample, Student's t-tests. The statistical significance of effects within each ROI was then assessed with permutation tests, as described above, except that the null distribution was computed from 60,000 random combinations of all contacts of a ROI, drawn from the 300 shuffles previously calculated for each site.

Contribution of frequency bands

To assess the contribution of the different frequency bands to mood representation, TML was regressed across trials against power P (normalized envelope) of each frequency band, averaged over time between -4 to 0 s before choice onset:

$$TML = \alpha + \beta P + \epsilon$$

With α corresponding to the intercept and ϵ to the error term. The significance of the regression estimates β was assessed across recording sites using two-sided, one-sample, Student's t-tests.

In order to determine whether other frequency bands provided additional information relative to the BGA, the following sixteen GLMs were compared:

$$\begin{aligned} TML &= \beta_{BGA} \times BGA \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) \\ TML &= \beta_{BGA} \times BGA + \beta_{\beta} \times P(\beta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\alpha} \times P(\alpha) \\ TML &= \beta_{BGA} \times BGA + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\beta} \times P(\beta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\alpha} \times P(\alpha) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\beta} \times P(\beta) + \beta_{\alpha} \times P(\alpha) \\ TML &= \beta_{BGA} \times BGA + \beta_{\beta} \times P(\beta) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\alpha} \times P(\alpha) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\beta} \times P(\beta) + \beta_{\alpha} \times P(\alpha) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\beta} \times P(\beta) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\alpha} \times P(\alpha) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\beta} \times P(\beta) + \beta_{\alpha} \times P(\alpha) + \beta_{\theta} \times P(\theta) \\ TML &= \beta_{BGA} \times BGA + \beta_{\gamma} \times P(\gamma) + \beta_{\beta} \times P(\beta) + \beta_{\alpha} \times P(\alpha) + \beta_{\theta} \times P(\theta) \end{aligned}$$

With β corresponding to the regression estimates, and P each power time series averaged between -4 to 0 s before choice onset in the high-gamma (BGA), gamma (γ), beta (β), alpha (α) and theta (θ) frequency bands.

The model comparison was conducted using the VBA toolbox (Daunizeau et al., 2014). Log-model evidence obtained in each recording site was taken to a group-level, random-effect, Bayesian model selection (RFX-BMS) procedure (Rigoux et al., 2014). RFX-BMS provides an exceedance probability that measures how likely it is that a given model is more frequently implemented, relative to all the others considered in the model space, in the population from which samples were drawn.

Computational analysis of baseline activity effect on choices

For each electrode of a given ROI, BGA was averaged in each trial over the window corresponding to the best significant cluster obtained with our second GLM (regression of residual error of choice model fit against power). More specifically, activity was averaged from -1.64 to -1.31 s before choice onset for the vmPFC and from -0.95 to -0.67 s for the daIns. The choice model was then run separately with data from trials whose averaged activity was above or below the median baseline activity across trials. As we expected small effects, data were restricted to trials that were not overly determined by choice parameters (i.e., trials for which acceptance probability, as computed by the choice model with all trials, was between 2/7 and 5/7 of the patient's acceptance range). We also ensured that the mean and variance of choice dimensions (gain, loss and difficulty) were identical between our two trial subsets (high vs. low baseline BGA). All free parameters of the expected utility were free to fluctuate (the constant k_0 , gain weight k_g , loss weight k_l and the weight associated with difficulty σ), while k_i was set with values obtained from the previously computed model (with all trials). Finally, significance of the difference between posterior parameters obtained with the two trial subsets (high versus low activity) was tested across all contacts of a given region using two-sided, paired, Student's t-tests.

Data availability

Raw data cannot be shared due to ethics committee restrictions. Intermediate as well as final processed data that support the findings of this study are available from the corresponding author (JB) upon reasonable request.

Code availability

The custom codes used to generate the figures and statistics are available from the lead contact (JB) upon request.

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Author Contributions

RC, FV, MP and JB designed the experiment. RC collected the data. FV provided preprocessing scripts. RC performed the data analysis. JH, PM, AT, NA, PK, EB, MD, LM, LM and SR did the intracerebral investigation and allowed the collection of iEEG data. RC, MP and JB wrote the manuscript. All the authors discussed the results and commented on the manuscript.

Declaration of Interests

The authors declare no competing interests.

References

- Abitbol, R., Lebreton, M., Hollard, G., Richmond, B.J., Bouret, S., and Pessiglione, M. (2015). Neural Mechanisms Underlying Contextual Dependency of Subjective Values: Converging Evidence from Monkeys and Humans. *Journal of Neuroscience* 35, 2308–2320.
- Arkes, H.R., Herren, L.T., and Isen, A.M. (1988). The role of potential loss in the influence of affect on risk-taking behavior. *Organizational Behavior and Human Decision Processes* 42, 181–193.
- Ashburner, J. (2009). Computational anatomy with the SPM software. *Magnetic Resonance Imaging* 27, 1163–1174.
- Auzias, G., Coulon, O., and Brovelli, A. (2016). MarsAtlas : A cortical parcellation atlas for functional mapping: MarsAtlas. *Human Brain Mapping* 37, 1573–1592.
- Bartra, O., McGuire, J.T., and Kable, J.W. (2013). The valuation system: A coordinate-based meta-analysis of BOLD fMRI experiments examining neural correlates of subjective value. *NeuroImage* 76, 412–427.
- Bassi, A., Colacito, R., and Fulghieri, P. (2013). 'O Sole Mio: An Experimental Analysis of Weather and Risk Attitudes in Financial Decisions. *Review of Financial Studies* 26, 1824–1852.
- Bijanzadeh, M., Desai, M., Wallace, D.L., Mummaneni, N., Kunwar, N., Dawes, H.E., and Chang, E.F. (2019). Decoding Natural Positive Emotional Behaviors from Human Fronto-Temporal Mesolimbic Structures. In 2019 9th International IEEE/EMBS Conference on Neural Engineering (NER), (San Francisco, CA, USA: IEEE), pp. 1088–1092.
- Brainard, D.H. (1997). The Psychophysics Toolbox. *Spatial Vision* 10, 433–436.
- Caria, A., Sitaram, R., Veit, R., Begliomini, C., and Birbaumer, N. (2010). Volitional Control of Anterior Insula Activity Modulates the Response to Aversive Stimuli. A Real-Time Functional Magnetic Resonance Imaging Study. *Biological Psychiatry* 68, 425–432.
- Chew, B., Hauser, T.U., Papoutsis, M., Magerkurth, J., Dolan, R.J., and Rutledge, R.B. (2019). Endogenous fluctuations in the dopaminergic midbrain drive behavioral choice variability. *Proceedings of the National Academy of Sciences* 116, 18732–18737.
- Chou, K.-L., Lee, T.M.C., and Ho, A.H.Y. (2007). Does mood state change risk taking tendency in older adults? *Psychology and Aging* 22, 310–318.
- Daunizeau, J., Adam, V., and Rigoux, L. (2014). VBA: A Probabilistic Treatment of Nonlinear Models for Neurobiological and Behavioural Data. *PLoS Computational Biology* 10, e1003441.
- Demian, P., Bhattacharjee, M., Tadel, F., Job, A.-S., Rivière, D., Cointepas, Y., Kahane, P., and David, O. (2018). IntrAnat Electrodes: A Free Database and Visualization Software for Intracranial Electroencephalographic Data Processed for Case and Group Studies. *Frontiers in Neuroinformatics* 12.
- Destrieux, C., Fischl, B., Dale, A., and Halgren, E. (2010). Automatic parcellation of human cortical gyri and sulci using standard anatomical nomenclature. *NeuroImage* 53, 1–15.
- Droutman, V., Bechara, A., and Read, S.J. (2015). Roles of the Different Sub-Regions of the Insular Cortex in Various Phases of the Decision-Making Process. *Frontiers in Behavioral Neuroscience* 9.
- Eldar, E., and Niv, Y. (2015). Interaction between emotional state and learning underlies mood instability. *Nature Communications* 6, 6149.

Fischl, B., Salat, D.H., Busa, E., Albert, M., Dieterich, M., Haselgrove, C., van der Kouwe, A., Killiany, R., Kennedy, D., Klaveness, S., et al. (2002). Whole Brain Segmentation. *Neuron* 33, 341–355.

Fouragnan, E., Retzler, C., and Philiastides, M.G. (2018). Separate neural representations of prediction error valence and surprise: Evidence from an fMRI meta-analysis. *Human Brain Mapping* 39, 2887–2906.

Garrison, J., Erdeniz, B., and Done, J. (2013). Prediction error in reinforcement learning: A meta-analysis of neuroimaging studies. *Neuroscience & Biobehavioral Reviews* 37, 1297–1310.

Gueguen, M.C., Billeke, P., Lachaux, J.-P., Rheims, S., Kahane, P., Minotti, L., David, O., Pessiglione, M., and Bastin, J. (2020). Opponent intracerebral signals for reward and punishment prediction errors in humans. *BioRxiv* 2020.06.11.145433.

Huang, Y.-F., Soon, C.S., Mullette-Gillman, O.A., and Hsieh, P.-J. (2014). Pre-existing brain states predict risky choices. *NeuroImage* 101, 466–472.

Kahneman, D., Krueger, A.B., Schkade, D.A., Schwarz, N., and Stone, A.A. (2004). A Survey Method for Characterizing Daily Life Experience: The Day Reconstruction Method. *Science* 306, 1776–1780.

Kirkby, L.A., Luongo, F.J., Lee, M.B., Nahum, M., Van Vleet, T.M., Rao, V.R., Dawes, H.E., Chang, E.F., and Sohal, V.S. (2018). An Amygdala-Hippocampus Subnetwork that Encodes Variation in Human Mood. *Cell* 175, 1688-1700.e14.

Kuhnen, C.M., and Knutson, B. (2005). The Neural Basis of Financial Risk Taking. *Neuron* 47, 763–770.

Lachaux, J.-P., Fonlupt, P., Kahane, P., Minotti, L., Hoffmann, D., Bertrand, O., and Baciou, M. (2007). Relationship between task-related gamma oscillations and BOLD signal: New insights from combined fMRI and intracranial EEG. *Human Brain Mapping* 28, 1368–1375.

Leahy, R.L. (1999). Decision making and mania. *Journal of Cognitive Psychotherapy* 13, 83–105.

Liu, X., Hairston, J., Schrier, M., and Fan, J. (2011). Common and distinct networks underlying reward valence and processing stages: A meta-analysis of functional neuroimaging studies. *Neuroscience & Biobehavioral Reviews* 35, 1219–1236.

Lopez-Persem, A., Domenech, P., and Pessiglione, M. (2016). How prior preferences determine decision-making frames and biases in the human brain. *Elife* 5, e20317.

Lopez-Persem, A., Verhagen, L., Amiez, C., Petrides, M., and Sallet, J. (2019). The Human Ventromedial Prefrontal Cortex: Sulcal Morphology and Its Influence on Functional Organization. *J. Neurosci.* 39, 3627–3639.

Lopez-Persem, A., Bastin, J., Petton, M., Abitbol, R., Lehongre, K., Adam, C., Navarro, V., Rheims, S., Kahane, P., Domenech, P., et al. (2020). Four core properties of the human brain valuation system demonstrated in intracranial signals. *Nature Neuroscience* 23, 664–675.

Manning, J.R., Jacobs, J., Fried, I., and Kahana, M.J. (2009). Broadband Shifts in Local Field Potential Power Spectra Are Correlated with Single-Neuron Spiking in Humans. *Journal of Neuroscience* 29, 13613–13620.

Mukamel, R. (2005). Coupling Between Neuronal Firing, Field Potentials, and fMRI in Human Auditory Cortex. *Science* 309, 951–954.

Niessing, J., Ebisch, B., Schmidt, K.E., Niessing, M., Singer, W., and Galuske, R.A.W. (2005). Hemodynamic signals correlate tightly with synchronized gamma oscillations. *Science* 309, 948–951.

Nir, Y., Fisch, L., Mukamel, R., Gelbard-Sagiv, H., Arieli, A., Fried, I., and Malach, R. (2007). Coupling between Neuronal Firing Rate, Gamma LFP, and BOLD fMRI Is Related to Interneuronal Correlations. *Current Biology* 17, 1275–1285.

Otto, A.R., Fleming, S.M., and Glimcher, P.W. (2016). Unexpected but Incidental Positive Outcomes Predict Real-World Gambling. *Psychol Sci* 27, 299–311.

Padoa-Schioppa, C. (2013). Neuronal Origins of Choice Variability in Economic Decisions. *Neuron* 80, 1322–1336.

Palminteri, S., and Pessiglione, M. (2017). Opponent Brain Systems for Reward and Punishment Learning. In *Decision Neuroscience*, (Elsevier), pp. 291–303.

Pessiglione, M., and Delgado, M.R. (2015). The good, the bad and the brain: neural correlates of appetitive and aversive values underlying decision making. *Current Opinion in Behavioral Sciences* 5, 78–84.

Rao, V.R., Sellers, K.K., Wallace, D.L., Lee, M.B., Bijanzadeh, M., Sani, O.G., Yang, Y., Shanechi, M.M., Dawes, H.E., and Chang, E.F. (2018). Direct Electrical Stimulation of Lateral Orbitofrontal Cortex Acutely Improves Mood in Individuals with Symptoms of Depression. *Current Biology* 28, 3893-3902.e4.

Rigoux, L., Stephan, K.E., Friston, K.J., and Daunizeau, J. (2014). Bayesian model selection for group studies — Revisited. *NeuroImage* 84, 971–985.

Rivière, D., Geffroy, D., Denghien, I., Souedet, N., and Cointepas, Y. (2009). BrainVISA: an extensible software environment for sharing multimodal neuroimaging data and processing tools. *NeuroImage Supplement 1*, S163.

Robert, C.P. (2007). *The Bayesian choice: from decision-theoretic foundations to computational implementation* (New York: Springer).

Rutledge, R.B., Skandali, N., Dayan, P., and Dolan, R.J. (2014). A computational and neural model of momentary subjective well-being. *Proceedings of the National Academy of Sciences* 111, 12252–12257.

Sani, O.G., Yang, Y., Lee, M.B., Dawes, H.E., Chang, E.F., and Shanechi, M.M. (2018). Mood variations decoded from multi-site intracranial human brain activity. *Nature Biotechnology* 36, 954–961.

Saunders, E.M. (1993). Stock Prices and Wall Street Weather. *The American Economic Review* 83, 1337–1345.

Tuyisenge, V., Trebaul, L., Bhattacharjee, M., Chanteloup-Forêt, B., Saubat-Guigui, C., Mîndruță, I., Rheims, S., Maillard, L., Kahane, P., Taussig, D., et al. (2018). Automatic bad channel detection in intracranial electroencephalographic recordings using ensemble machine learning. *Clinical Neurophysiology* 129, 548–554.

Vinckier, F., Rigoux, L., Oudiette, D., and Pessiglione, M. (2018). Neuro-computational account of how mood fluctuations arise and affect decision making. *Nature Communications* 9, 1708.

Watson, D., Clark, L.A., and Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *J Pers Soc Psychol* 54, 1063–1070.

Wiech, K., Lin, C. -s., Brodersen, K.H., Bingel, U., Ploner, M., and Tracey, I. (2010). Anterior Insula Integrates Information about Salience into Perceptual Decisions about Pain. *Journal of Neuroscience* 30, 16324–16331.

Winawer, J., Kay, K.N., Foster, B.L., Rauschecker, A.M., Parvizi, J., and Wandell, B.A. (2013). Asynchronous Broadband Signals Are the Principal Source of the BOLD Response in Human Visual Cortex. *Current Biology* 23, 1145–1153.

Young, C.B., and Nusslock, R. (2016). Positive mood enhances reward-related neural activity. *Social Cognitive and Affective Neuroscience* 11, 934–944.

Yuen, K.S.L., and Lee, T.M.C. (2003). Could mood state affect risk-taking decisions? *Journal of Affective Disorders* 75, 11–18.