A Registered Report of Error-Related Negativity and Reward Positivity as Biomarkers of
Depression: P-Curving the Evidence

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Abstract

Performance-monitoring event-related brain potentials (ERPs), such as the error-related negativity (ERN) and reward positivity (RewP), are advocated as biomarkers of depression symptoms and risk. However, a recent meta-analysis indicated effect size heterogeneity in the ERN and RewP literatures. Hence, advocating these ERPs as biomarkers of depression might be premature or possibly misguided due to the selective reporting of significant analyses on the part of researchers (e.g., *p*-hacking or omission of non-significant findings). The present study quantified the degree of selective reporting and the evidential value for a true relationship between depression and ERN and RewP using a *p*-curve analysis. We predicted that the ERN and RewP literatures would fail to show evidential value for a relationship between each ERP and depression. Contrary to expectations, both literatures showed evidential value, albeit weak. The statistical power of the included ERN studies was between 20% and 25%, and the statistical power of the RewP was around 27%. Taken together, these findings provide support for a relationship between these ERPs and depression, which strengthens claims that these ERPs represent candidate biomarkers of depression symptoms and risk. In light of the evidence for these relationships being weak, some recommendations moving forward include conducting *a priori* power analyses, increasing sample sizes to improve statistical power, assessing the internal consistency of ERP scores, and carefully planning statistical approaches to maximize power.

Keywords: performance monitoring, depression, *p* curve, error-related negativity (ERN), reward positivity (RewP)

Performance-monitoring event-related brain potentials (ERPs), such as the error-related negativity (ERN) and reward positivity (RewP), are advocated as biomarkers or endophenotypes of depression symptoms and risk (e.g., Meyer, Bress, Hajcak, & Gibb, 2018; Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). However, recent meta-analytic work indicates that both ERN and RewP literatures are replete with heterogeneous effect sizes (Moran, Schroder, Kneip, & Moser, 2017). Although heterogeneous effect sizes might be due to known or unknown moderating factors, it is possible that heterogeneity is due to the selective reporting of significant analyses or undisclosed use of other questionable research practices (QRPs), and this possibility was not examined in the meta-analysis. If effect size heterogeneity is due to selective reporting/omission of non-significant findings or other QRPs, then advocating performance-monitoring ERPs as biomarkers of depression would be misguided or at least premature. The aim of the present registered report was to quantify the degree of selective reporting and the evidential value for a true relationship between depression and ERN and RewP using *a priori* registered hypotheses and methods.

There is ambiguity surrounding the precise operational definition of a biomarker (see Miller & Rockstroh, 2013). For example, “biomarkers are sometimes viewed as merely correlates, sometimes as necessary (though not sufficient) signs, and sometimes are mere epiphenomena” (Miller & Rockstroh, 2013, p. 185). Although ERN and RewP have been proposed as biomarkers, ERN was originally proposed as an endophenotype for internalizing disorders, including depression (Olivet & Hajcak, 2008), and for a measure to be considered an endophenotype, it must satisfy additional constraints. For ERN to be an endophenotype for depression, it must be associated with depression in the population, be heritable, be present regardless of changes in state characteristics or whether depression symptoms are experienced,

co-segregate with illness within families, occur at higher rates in unaffected family relatives than in the general population, be measured reliably, and potentially demonstrate specificity for depression (see Chan & Gottesman, 2008; Gottesman & Gould, 2003; Hasler, Drevets, Gould, Gottesman, & Manji, 2006). Regardless of whether ERN or RewP is classified as a biomarker or endophenotype, these ERPs must at least demonstrate consistent and robust relationships with depression symptoms.

The ERN represents activity of an internal monitoring process that is sensitive to the detection of performance errors (Larson, Clayson, & Clawson, 2014). The ERN is a negative deflection in the scalp-recorded ERP that occurs within 100ms of error commission (Falkenstein, Hohnsbein, Hoormann, & Banke, 1991; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) and represents a manifestation of activity in the anterior cingulate cortex (Carter & van Veen, 2007; van Veen & Carter, 2002). There are multiple prominent theoretical models for explaining ERN, such as the mismatch theory (Falkenstein et al., 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), the conflict monitoring theory (Botvinick, Carter, Braver, Barch, & Cohen, 2001; Larson et al., 2014; Yeung, Botvinick, & Cohen, 2004), and the reinforcement learning theory (Holroyd & Coles, 2002). The consensus among these theories is that ERN represents a manifestation of a performance-monitoring system that serves to adaptively adjust behavior (Weinberg, Dieterich, & Riesel, 2015), which is consistent with relationships between ERN and various cognitive and motivational processes (Larson, Clayson, & Baldwin, 2012; Larson et al., 2014; Olvet & Hajcak, 2008; Proudfit, Inzlicht, & Mennin, 2013; Weinberg et al., 2015). Thus, ERN reflects the evaluation that performance is not in-line with goals and that there is need for additional cognitive control (Holroyd & Yeung, 2012; Larson et al., 2014; Weinberg et al., 2015).

As previously mentioned, increased ERN amplitude (i.e., more negative) is proposed as an endophenotype of internalizing disorders, including depression (Olvet & Hajcak, 2008). Enhanced ERN might reflect core features of depression including hypersensitivity to negative environmental cues (Ingram, Miranda, & Segal, 2006), impaired inhibition of negative information (Goeleven, De Raedt, Baert, & Koster, 2006), heightened sensitivity to aversive events (Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003), and a negative cognitive bias (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Some studies have shown enhanced ERN in depression (e.g., Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010), whereas others have shown similar or smaller ERN amplitudes in people with depression than in healthy controls (Georgiadi, Liotti, Nixon, & Liddle, 2011; Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2009). Differences in experimental tasks, depression severity, and the presence of comorbid anxiety symptoms might account for these inconsistent findings (for review, see Weinberg et al., 2015). Publication bias and effect size heterogeneity notwithstanding, a meta-analysis of 1,757 people across 28 studies indicated that ERN is larger in people with depression than in healthy controls (Hedge's $g = 0.21$, Moran et al., 2017). The meta-analysis did not observe any significant moderators of the relationship between ERN and depression but noted high variability in effect sizes for moderators such as population, experimental task, and the use of affective vs. non-affective stimuli.

Whereas ERN is associated with the internal monitoring of errors, the monitoring of external feedback and reward is indexed by RewP (Proudfit, 2015), which has also been referred to as the feedback negativity (FN) or the medial frontal negativity (MFN; for review, see Glazer, Kelley, Pornpattananangkul, Mittal, & Nusslock, 2018). The RewP is characterized by a positive deflection in the scalp-recorded ERP approximately 200 to 300ms following presentation of

feedback (Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Kujawa, Smith, Luhmann, & Hajcak, 2013; Proudfit, 2015), is more positive for reward trials than for neutral or loss trials, and represents a manifestation of activity in the ventral striatum and medial prefrontal cortex (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti, Weinberg, Dien, & Hajcak, 2011). The prominent account of RewP is the reinforcement learning theory, which suggests that RewP represents a reward prediction error indicating a discrepancy between the prior expected outcome (i.e., reward or loss) and the actual outcome (Holroyd & Coles, 2002). Meta-analytic findings support predictions of the reinforcement learning theory and demonstrate that RewP is sensitive not only to the expected likelihood of an outcome but also to the magnitude of the received outcome (Sambrook & Goslin, 2015).

Blunted RewP is proposed as a biomarker for increased depression symptoms and risk for developing depression (Proudfit, 2015). In studies of individuals with depression, alterations in RewP are believed to represent deficits in reward processing due to consummatory anhedonia (Keren et al., 2018; Nelson et al., 2016). Although the pattern of findings for a reduced RewP in depression appears more consistent than the pattern observed for ERN, RewP findings remain fairly mixed. For example, some studies of people with depression indicate attenuated RewP in depression (e.g., Liu et al., 2014; Whitton et al., 2016) and others show enhanced RewP in depression (e.g., Mies et al., 2011; Mueller, Pechtel, Cohen, Douglas, & Pizzagalli, 2015). Despite these mixed findings, attenuated RewP is related to a family history of depression (Foti, Kotov, Klein, & Hajcak, 2011; Kujawa, Proudfit, & Klein, 2014) and prospectively predicts increases in depression symptoms (Bress, Foti, Kotov, Klein, & Hajcak, 2013; Bress, Meyer, & Proudfit, 2015; Nelson et al., 2016). These promising findings notwithstanding, a recent meta-analysis of 878 people across 18 studies observed no significant alterations in RewP in people

with depression, although this might have been due to the heterogeneity of effect sizes (Moran et al., 2017). The meta-analysis observed that studies using gambling/guessing tasks observed smaller RewP in depression than studies using reinforcement learning tasks. However, the meta-analysis did not determine whether effect size heterogeneity might be due to the selective reporting of analyses, omission of non-significant effects, or other QRPs. Such a possibility would undermine any attempts to identify moderators across the RewP literature.

As mentioned previously, recent meta-analytic work observed possible publication bias among ERN findings (Moran et al., 2017). It has been known for decades that research reporting statistically significant findings is more likely to be published than research reporting nonsignificant results (Greenwald, 1975; Rosenthal, 1979; Sterling, 1959). A consequence of this prejudice against nonsignificant findings is the overrepresentation of significant studies in the literature (i.e., publication bias) and the inflation of effect sizes. Another consequence of this filter for significant studies is that researcher behavior might change to accommodate the significant-study filter observed among journals. For example, researchers might selectively report only analyses in their studies that are significant (i.e., the garden of forking paths¹; Gelman & Loken, 2014) or exploit researcher degrees of freedom or ambiguity to obtain significant results (i.e., *p*-hacking; Simonsohn, Nelson, & Simmons, 2014b). These QRPs and others are quite common, with over 90% of psychology researchers admitting to using at least one QRP (John, Loewenstein, & Prelec, 2012).

Selective reporting in the garden of forking paths is particularly problematic when researchers generate hypotheses from their collected data and then present those data-generated hypotheses as *a priori* predictions (Nosek, Ebersole, DeHaven, & Mellor, 2018). This selective reporting of significant results consequently biases the published literature and misrepresents the

“true” effect in the population of interest (typically by inflating the effect size due to the significance filter for findings). Thus, although it was observed that the ERN and RewP literatures contain significant effect size heterogeneity, it is unclear whether evidential value for a true relationship between ERN/RewP and depression remains when accounting for the selective reporting of significant analyses on the part of the researcher. Without evidential value, any meta-analytic interpretation of moderators between ERN/RewP and depression across studies would be unsupported and possibly misleading.

To quantify the level of selective reporting and determine whether the ERN and RewP depression literatures contain evidential value, the present study conducted *p*-curve analyses (Simmons & Simonsohn, 2017; Simonsohn, Nelson, & Simmons, 2014a; Simonsohn et al., 2014b; Simonsohn, Simmons, & Nelson, 2015). A *p*-curve analysis operates under the assumption that there is publication bias in the literature. Unlike other meta-analytic techniques (Simonsohn et al., 2014a; Simonsohn et al., 2015), the *p*-curve analysis determines whether selective reporting of significant analyses (i.e., garden of forking paths, *p*-hacking, or other QRPs), rather than just publication bias, accounts for an observed effect across published studies. A set of studies contains evidential value when it is statistically ruled out that selective reporting of analyses accounts for observed findings. A *p*-curve analysis examines the frequency distribution of *p* values and only considers statistical results that show significant effects. The shape of the *p* curve can be used to determine whether a set of significant findings contains evidential value. In the absence of both a “true” effect and selective reporting, *p* values are uniformly distributed between 0 and .05 (see also Ulrich & Miller, 2018). That is, the *p* curve between 0 and .05 is flat when the null hypothesis is true. When there is a “true” effect in a set of studies and when no selective reporting has occurred, the distribution of the *p* curve shows a

right skew, because low p values (e.g., .01) are more likely than high p values (e.g., .049) in the presence of true differences. Hence, a p -curve distribution with a right skew is considered to contain evidential value.

The discussion of p -curve analyses thus far considered how the distribution of significant p values looks in the absence of selective reporting. In the presence of selective reporting, the p curve shows a left skew. In the garden of forking paths, when an analysis route taken by a researcher is not prespecified but rather is dependent on the data, there can be a large number of comparisons conducted that appear consistent with the researcher's hypothesis (Gelman & Loken, 2014). In these instances, it is likely that a researcher stops analyzing the data once a significant finding is obtained. Such significant p values are more likely to be close to .05 than to 0, and they are more likely due to chance than due to the observation of a true effect (see Simonsohn et al., 2014b). As a result, the p -curve distribution between 0 and .05 is left skewed, which is evidence of selective reporting and suggests that a set of studies lacks evidential value.

The p -curve's shape can also be used to estimate the average effect size and the average statistical power after correcting for selective reporting. The shape of the p curve is a function of the sample size for a set of studies and the average effect size (Simonsohn et al., 2014a). As a result, the shape of the p curve and the sample size can be used to calculate the average effect size and average statistical power while correcting for selective reporting. To do so, a p curve for the observed sample size and for a given estimate of an effect size is modeled. Then this modeled p curve is compared to the observed p curve to determine how closely they match. A range of effect sizes is iteratively evaluated to model p curves until the modeled p curve matches the observed p curve. The effect size for the model with the best overall fit is considered to yield the average effect size for a given set of studies. Notably, in the presence of selective reporting and

other QRPs, the p -curve approach to estimating the effect size of a set of studies outperforms other conventional methods, such as the trim-and-fill procedure, which was used in the ERN/RewP meta-analysis, and meta-regression techniques (Carter, Schönbrodt, Gervais, & Hilgard, 2018; Simonsohn et al., 2014a; Simonsohn et al., 2015). For example, a recent simulation study indicated that in the presence of selective reporting and QRPs the trim-and-fill procedure is unable to identify the true effect size across a set of studies, which results in inflated false positives (Carter et al., 2018). The iterative procedure that is used to estimate the average effect size is also used to estimate the average statistical power in a set of studies included in the p -curve analysis.

The p -curve approach to effect-size estimation, but not statistical power estimation, requires that all statistical effects are in the same direction (i.e., homogeneous; Simonsohn et al., 2014a). For example, all included ERN studies in the estimation of effect size need to show larger amplitudes in depression than healthy controls for accurate inclusion in a p -curve analysis. In order to determine the average effect size of ERN and RewP depression studies, only those studies with effects in the same direction were considered. For ERN, those studies showing larger ERN and smaller ERN in depression than in healthy controls were examined separately. Both predictions of larger and smaller ERN in depression than in healthy controls have some support in the literature (see Olvet & Hajcak, 2008; Weinberg et al., 2016b). Only studies showing smaller RewP in depression than in healthy controls were examined based on recommendations that small RewP represents a biomarker for depression risk and severity (Proudfit, 2015).

Although there is enthusiasm for using ERN and RewP values as biomarkers of depression severity and risk (e.g., Meyer et al., 2018; Nelson et al., 2016), there is also

substantial effect size heterogeneity in both literatures (Moran et al., 2017). The current registered report built upon the recent ERN/RewP meta-analysis by quantifying the degree of selective reporting of significant analyses in these literatures, providing a better estimate of the true effect size after accounting for selective reporting, and determining whether there is evidential value for a relationship between depression and ERN and between depression and RewP in the extant published literature. Based on the observed heterogeneity in the literatures related to depression, we predicted that the ERN and RewP literatures would fail to show evidential value. Without evidential value for such relationships, ERN and RewP would represent poor candidate biomarkers for depression, and any attempt using meta-analytic approaches to identify moderators of the relationship between these ERPs and depression would be uninformative. We also sought to determine the average effect size and average statistical power in the literature for ERN and RewP. For the calculation of the average effect size of ERN and RewP depression studies, only those studies reporting larger ERN, smaller ERN, or smaller RewP in people with depression than in healthy controls were examined.

Method

The present manuscript represents a registered report. Hence, study hypotheses and methods were registered prior to collecting, analyzing, or interpreting the data. Raw data for this registered report are posted on Open Science Framework at <https://osf.io/ckab7/>.

Manuscript Selection Criteria

The first rule for article selection was all articles included in the meta-analysis performed by Moran et al. (2017). Then, similar to the meta-analysis, we searched EMBASE, ProQuest, PubMed, PsycInfo, Scopus, Web of Science, Google Scholar, and Psych Filedrawer databases using the following search terms: depression, major depressive disorder, depressive episode,

depress*, error-related negativity, ERN, feedback negativity, FN, action-monitoring and response-monitoring. Unlike the meta-analysis, we also included the search terms reward positivity and RewP. Although it is unclear the precise dates from which articles were included in the meta-analysis, we contained our search to articles published from January 1, 2016 (which was expected to overlap with articles published in the meta-analysis) to April 23, 2019 (the date when the present manuscript received an in-principle acceptance as a registered report).

A study was included in the *p*-curve analysis if it satisfied each of the following inclusion criteria. 1) The study was published in English. 2) The study included either participants diagnosed with depression using the DSM or ICD classification systems or participants with scores on a depression measure. Thus, consistent with Moran et al. (2017), people with both clinical and subclinical depression were examined. 3) The study examined response-locked ERN or feedback-locked RewP. 4) The study reported statistical tests and degrees of freedom (this inclusion criterion was different than the meta-analysis, which required the reporting of enough information for the calculation of effect sizes).

Selecting Statistical Tests

As outlined by Simonsohn et al. (2014b), the following steps were used to identify statistical tests to include in analyses. The information for all steps for each study were posted on Open Science Framework in the form of the standardized “*p*-curve disclosure table”. This information provides transparency for how each step was implemented for each study, and the table is considered the raw data for the present study. Consistent with recommendations (Simonsohn et al., 2014a, 2014b; Simonsohn et al., 2015), only one *p* value per independent experiment and sample were extracted to satisfy the independence criterion for the *p*-curve analysis (see steps below).

First, the researcher's stated hypothesis or hypotheses about the relationship between ERN/RewP and depression was extracted and quoted in full in the *p*-curve disclosure table. This information was used to characterize what study design and analysis technique were used to test the hypothesis, which is reported in the *p*-curve disclosure table. Second, the statistical result from the analysis designed to test the specified hypothesis was identified according to guidelines provided by Simonsohn et al. (2014b) and their subsequent clarification (Simonsohn et al., 2015). Third, the identified statistical result was quoted directly in the *p*-curve disclosure table, and the direction of the effect was specified. Fourth, precise *p* values were recomputed based on a two-tailed test of significance and reported. In the event that the authors did not report the statistical result or the *p* value could not be recomputed based on the information provided, "Not Reported" was specified in the *p*-curve disclosure table with justification for exclusion in the caption. Fifth, in the event that multiple statistical analyses were reported on the same measure for the hypothesis or hypotheses of interest, each statistical analysis was included in the *p*-curve disclosure table. The primary *p*-curve analysis of interest was the analysis conducted on the *first* reported significant result of the hypothesis related to the relationship between ERN/RewP and depression, and a *p*-curve analysis performed on the alternative *p*-value selections was conducted as a test of robustness of the first *p* curve. Sixth, the *p*-curve analysis requires that independent samples are used for each *p* value. In the event that the same sample of participants appeared in multiple ERN or RewP papers, only the study with the largest sample size was included in the *p*-curve analyses. This was determined based on authors reporting whether a study represented a re-analysis of a sample that was already included in the *p*-curve analysis. In the event that the authors reported a re-analysis of previously published data, the previous studies were denoted as "Previous Study" in the *p*-curve disclosure table with justification for exclusion in the caption.

Lastly, in the event that the depression sample included comorbid anxiety, the study was coded as a 1; otherwise, the study was coded as a 0. This information was used to determine whether there is a consistent pattern of effects among studies examining depression and comorbid anxiety (see exploratory analyses below).

***p*-curve Analysis**

p-curve analyses were performed using the online *p*-curve web application (v.4.06, available at <http://www.p-curve.com/app4>; Simonsohn et al., 2015). Extracted statistical tests and degrees of freedom were separately examined for ERN studies and RewP studies. The *p*-curve analysis first determined whether there was evidential value in the literature. To this end, the individual probability of observing a *p* value as extreme as the one reported in each study given no true effect was calculated and then combined using Stouffer's Method (Simonsohn et al., 2015). The *p* curve was first examined using all *p*-value probabilities (i.e., the "full" *p* curve) and then using only *p* values less than .025 (i.e., the "half" *p* curve). The analysis of the half *p* curve was used to circumvent ambitious selective reporting (i.e., when the goal was to report the lowest *p*-value, as opposed to one just below .05; Simonsohn et al., 2015). As recommended by Simonsohn et al. (2015), we concluded a set of studies contained evidential value if the half *p* curve had a $p < .05$ right-skew test, or if both the full and half *p* curves had a $p < .10$ right-skew test. To determine whether the results of this *p*-curve analysis were due to outliers, the *p*-curve analysis was rerun after excluding any *p* values that are 1,000 times smaller than the largest *p* values. This procedure was only used once as a robustness check of the RewP *p* curve; there were no outlier *p* values based on this criterion for the set of ERN studies.

In the event that a set of studies was determined to have evidential value, we also assessed whether the evidential value was inadequate or absent based on the test of 33%

statistical power. For the 33% statistical power test, the probability of observing an extracted p value as extreme as the one reported had the study been powered at 33% was calculated. The probabilities were again combined using Stouffer's Method (Simonsohn et al., 2015). A binomial test was then conducted comparing the percent of observed p values less than .025 to the percent of p values less than .025 that would have been expected if studies were powered at 33%. This test effectively determined whether the p curve is flatter than would be expected if the studies had only 33% statistical power. Consistent with Simonsohn et al. (2015), we concluded that a set of studies had insufficient evidential value for a true effect if the extracted full p curve is significantly flatter ($p < .05$) than the p curve powered at 33% or if the half p -curve analysis and binomial test were significant ($ps < .10$). To determine whether the results of this p -curve analysis were due to outliers, the p -curve analysis was rerun after excluding any p values that are 1,000 times smaller than the largest p values. Although there is no left-skewed test of the p curve, a left-skewed p curve suggests that a set of studies does not contain evidential value or that evidential value reflects selective reporting (Simonsohn et al., 2014b).

To compute the average statistical power and effect sizes for a set of studies, iterative procedures were used. First, the observed p curve was compared to modeled p curves for each possible value of statistical power between 5% and 99%. The statistical power used for the modeled p curve that most closely matches the observed p curve was considered the average statistical power of a set of studies. The modeled p curve leading to $Z = 0$ and $p = .5$ using the Stouffer's Method was considered the model with the best fit (Simonsohn et al., 2015). All procedures thus far relied on the online p -curve web application, but the analyses for determining average effect sizes used code provided by Simonsohn et al. (2014a). An iterative procedure was used to determine the average effect size of a set of studies. For those studies with homogeneous

effects (larger ERN, smaller ERN, and smaller RewP in depression than in controls), an expected p curve was modeled using the observed sample sizes and an estimated effect size. This expected p curve was compared to the observed p curve, and the effect size of the expected p curve that most closely matches the observed p curve is considered the average effect size of a set of studies. The Stouffer's Method was again used to determine the best model fit.

Primary Tests of Hypotheses

We hypothesized that the ERN and RewP literatures would not show evidential value. *A priori*, we determined that these hypotheses would be supported in the following instances. First, the half p -curve right-skew test was nonsignificant at $p < .05$ and both the full and half p curves right-skew tests were nonsignificant at $p < .10$. If those conditions were met, we would then determine that the ERN or RewP literature does not contain evidential value. If those conditions were not met, we then determined whether the evidential value is inadequate. We concluded that evidential value was inadequate if the full p curve was significantly flatter than the p curve powered at 33% at $p < .05$ or the same half p -curve analysis and binomial test were significant ($ps < .10$).

Exploratory Analyses

It is possible that larger ERN in depression samples is due to the presence of comorbid anxiety symptoms (Weinberg et al., 2015; Weinberg et al., 2016b). We tested whether studies that include participants with depression and comorbid anxiety contain evidential value. If this set of studies demonstrates adequate statistical power (.90) using the procedures outlined below, these exploratory analyses used the same hypothesis testing procedure just described above to determine whether these studies contain evidential value.

Power Analysis

The power of the p -curve analysis is contingent on the number of studies included, the power of the original studies, and how many of those studies find non-significant results (Simmons & Simonsohn, 2017). Because all of these factors are outside the present authors' control, the power of the p -curve analysis was necessarily limited by the available literature. To compute an *a priori* preliminary estimate of the power of the p -curve analysis, the code provided by Simmons and Simonsohn (2017) was used to determine the statistical power to detect evidential value and the statistical power to conclude a p curve is flat. The estimates for the number of studies that would be included in the p -curve analysis was based on the recent ERN and RewP depression meta-analysis, but it was expected that additional ERN and RewP studies would be included after coding recent papers. Thus, the power analyses below were considered conservative. In the meta-analysis, 28 ERN studies (average sample size was approximately 63) and 18 RewP studies (average sample size was approximately 49) were included. Analyses used an *a priori* statistical power of .90 and an alpha level of .05.

The present study would obtain a statistical power of .90 to detect evidential value in the 28 ERN studies if those studies are powered to .33. If nine of the ERN studies have a statistical power of .90 and the remaining studies have null results, the p -curve analysis would be powered at .90 to detect evidential value. For an average sample size of 63 participants across 28 studies, the ERN p -curve analysis is powered at .93 to conclude that the p curve is flatter than would be expected if the studies had statistical power of .33.

For RewP, the p -curve analysis would obtain a statistical power of .90 to detect evidential value in the 18 RewP studies if those studies are powered to .43. If seven of the RewP studies have a statistical power of .90 and the remaining studies are null results, the p -curve analysis would be powered at .90 to detect evidential value. For an average sample size of 49 participants

across 18 studies, the RewP p -curve analysis is powered at .79 to conclude that the p curve is flatter than would be expected if the studies had only .33 statistical power.

The power of the RewP p -curve analysis to conclude that the p curve is flatter than would be expected if the RewP studies had statistical power of .33 falls short of .90 statistical power. However, the actual power of the p -curve analysis should be higher because additional studies have been published since the publication of the meta-analysis. For example, a large study of RewP with 444 participants was relatively recently published (Nelson et al., 2016). Furthermore, if the studies demonstrate an average statistical power greater than .33, it is possible the final analyses were sufficiently powered to conclude that the p curve is flatter than would be expected if the studies had statistical power of .33 (regardless of whether there are additional studies to included). Considering that one purpose of the present study was to determine the average statistical power in ERN and RewP depression studies, we recomputed these power analyses after coding all papers to determine whether insufficient power might account for study findings.

Quality Check

All papers were separately coded by authors KAC and PEC, and disagreements were resolved by unanimous consent of KAC, PEC, and MJL. When all papers were coded and compiled, power analyses were rerun to determine obtained statistical power for the p -curve analyses to detect evidential value and the statistical power to conclude a p curve is flat (Simmons & Simonsohn, 2017). As an additional quality check, power analyses on those sets of studies used for effect size estimation were conducted. That is, an additional power analysis was conducted on those studies that showed larger ERN, smaller ERN, and smaller RewP in people with depression than in healthy controls.

Deviations from Preregistered Protocol

In order to be as comprehensive as possible in our inclusion of relevant literature and to maximize achieved power, we deviated from inclusion criterion 4: the study reported statistical tests and degrees of freedom. Many articles failed to report the necessary statistical tests and/or degrees of freedom. When all of the information was provided in the manuscript to compute these values, the article was included in the *p*-curve analyses. Computing these values for papers resulted in the inclusion of nine additional ERN papers and fifteen additional RewP papers.

Results

A flow chart of article and *p*-value selection for ERN and RewP studies is shown in Figure 1.

ERN

A total of 50 ERN papers were coded (see Table 1 and supplementary material on OSF for more information). Twenty-three of these papers satisfied inclusion criteria and reported significant effects for the primary or alternative result and were included in the *p*-curve.

Primary *p* curve. The primary *p*-curve analysis used the results of the statistical test of the main hypothesis related to depression and examined 20 studies with a total of 1,568 participants. The *p*-curve analysis obtained a statistical power of .92 if the underlying studies were powered to .41. If at least eight of the ERN studies have a statistical power of .90, the *p*-curve analysis is considered powered at .90 to detect evidential value. For the average sample size of 78 participants across the 20 studies, the ERN *p*-curve analysis was powered at .84 to conclude the *p* curve is flatter than would be expected if the studies had a statistical power of .33.

Results of the primary *p*-curve analysis for ERN indicated the half *p*-curve right skew test was not significant, $Z = -1.58$, $p = .06$, and the full *p*-curve test was significant, $Z = -1.76$, $p = .04$ (see Figure 2). Because both tests have *p* values below .10, these findings suggest that this set of

studies contains evidential value. Furthermore, the 33% power test for both the half and full p -curve were not significant, $Z_s > -.82$, $p_s > .21$, suggesting that evidential value was not inadequate. The estimated power of this set of studies was low at 20% (90% CI: 5%, 48%). For the ten studies that showed a small ERN relates to high depressive symptoms, the effect size was estimated as a Cohen's d of .59. For the ten studies that showed a large ERN relates to high depressive symptoms, the statistical model failed to converge, and the effect size could not be estimated.

Robustness of results. First, it was concluded that there were no outlier p values, because no p values were 1,000 times smaller than the largest included p value. Hence, secondary analyses were not conducted on data with outliers removed.

Second, a p -curve analysis was performed on alternative significant results of tests of the relationship between depression and ERN as a test of robustness, when studies reported such tests. Twenty studies were initially included in the secondary p -curve analysis, but one was removed for having a recomputed p value over .05. Results indicated that the half p -curve right skew test was not significant, $Z = -.98$, $p = .16$, and the full p -curve test was significant, $Z = -2.13$, $p = .02$ (see Figure 3), which suggests that this set of studies does not contain evidential value. However, the 33% power test for both the half and full p -curve were not significant, $Z_s > -.47$, $p_s > .32$, and the binomial test was not significant, $p = .52$. These findings suggest that the p curve is not flatter than would be expected if the studies had 33% statistical power. Taken together, the secondary p -curve analysis was inconclusive and appeared to be underpowered due to the inclusion of too few studies and/or too few participants in those studies. Statistical power of the tests included in the secondary p curve was estimated at only 25% (90% CI: 7%, 54%).

Exploratory analyses. We planned in the initial registration to explore whether those

studies that assess the relationship between ERN and depression in those with comorbid anxiety disorders demonstrated evidential value. There were ten studies of participants with depression and comorbid anxiety that reported significant results. The p -curve analysis obtained a statistical power of .92 if the underlying studies were powered to .60. If at least six of the ERN studies have a statistical power of .90, the p -curve analysis is considered powered at .90 to detect evidential value. For the average sample size of 54 participants across the 10 studies, the ERN p -curve analysis was powered at .54 to conclude the p curve is flatter than would be expected if the studies had a statistical power of .33. The achieved statistical power of this set of studies was estimated at 17% (90% CI: 5%, 58%). Because the underlying studies were not powered to at least .60, we failed to achieve the 90% statistical power criterion for conducting the p -curve analysis. Hence, we did not examine this set of studies to determine whether they contain evidential value.

Exploratory *post-hoc* sensitivity analyses were conducted on the primary ERN p -curve analyses (see Figure 4). In a p -curve sensitivity analysis, the impact of systematically dropping the lowest and highest p values on the p curve results is examined, and this approach can be used to determine how sensitive observed evidential value is to the exclusion of a few studies (Carbine & Larson, 2019; Simonsohn et al., 2015; Taylor & Munafò, 2016). The impact of dropping up to half of all the included p values is shown in Figure 4. Excluding the lowest p value would result in a nonsignificant half p -curve right skew test and a nonsignificant full p -curve test ($ps > .10$). Hence, removing only one study would result in an inconclusive p curve analysis, but it would require removing six of the lowest p values for the p curve to be flatter than would be expected if the studies had 33% statistical power.

Interim Summary. Evidential value was observed for the relationship between ERN and depression, but the sensitivity analysis indicated that the removal of one study led to inconclusive support for evidential value. The estimated power for the primary p curve was low at 20% (90% CI: 5%, 48%), and the estimated power for the secondary p curve was similarly low at 25% (90% CI: 7%, 54%).

RewP

A total of 45 RewP papers were coded (see Table 2 and supplementary material on OSF for more information). Twenty-nine of these papers satisfied inclusion criteria and reported significant effects for the primary or alternative result.

Primary p curve. The primary p -curve analysis used the results of the statistical test of the primary hypothesis and examined 26 studies of a total of 2,160 participants. (Two studies were excluded for having recomputed p values over .05.) The p -curve analysis obtained a statistical power of .92 if the underlying studies were powered to .36. If at least nine of the RewP studies have a statistical power of .90, the p -curve analysis is considered powered at .90 to detect evidential value. For the average sample size of 83 participants across the 26 studies, the RewP p -curve analysis was powered at .92 to conclude the p curve is flatter than would be expected if the studies had a statistical power of .33.

Results of the primary p -curve analysis for RewP indicated the half p -curve right skew test was significant, $Z = -2.10$, $p = .02$, and the full p -curve test was significant, $Z = -2.74$, $p = .003$ (see Figure 5). These findings suggest that this set of studies contains evidential value. Furthermore, the 33% power test for both the half and full p -curve were not significant, $Z_s > -.47$, $p_s > .32$, suggesting that evidential value is not inadequate. The estimated power of this set

of studies was low at 27% (90% CI: 10%, 51%). For the 22 studies that showed a small RewP relates to high depressive symptoms, the effect size was estimated as a Cohen's d of .48.

Robustness of results. One study showed an outlier p value that was more than 1,000 times smaller than largest significant p value. After excluding this study from the p curve analysis, the half p -curve right skew test was no longer significant, $Z = -.69$, $p = .24$ (see Figure 6). The full p -curve test was also not significant, $Z = -1.57$, $p = .06$. These findings suggest that this set of studies failed to show evidential value for a relationship between RewP and depression when the outlier was removed. We next determined the p curve was flatter than would be expected if the studies had 33% statistical power. The full p curve was not significantly flatter than the p curve powered at 33%, $Z = -1.37$, $p = .09$. The half p -curve analysis, $Z = 3.5$, $p = .9998$, and binomial test, $p = .17$, were also not significant. Taken together, the robustness test of this set of studies was inconclusive.

A p -curve analysis was also performed on alternative significant results of tests of the relationship between RewP and depression, when studies reported such tests. Twenty studies were initially included in the secondary p -curve analysis, but two were removed for having recomputed p values over .05. The half p -curve right skew test was significant, $Z = -2.67$, $p = .004$, and the full p -curve test was significant, $Z = -2.15$, $p = .02$ (see Figure 7). The 33% power test for both the half and full p -curve were not significant, $Zs > -.40$, $ps > .34$. These findings suggest that this set of studies contained evidential value. The estimated statistical power was 27% (90% CI: 7%, 56%).

Exploratory analyses. Exploratory sensitivity analyses were conducted on the primary RewP p -curve analyses (see Figure 8). Excluding the lowest two p values, the two p values that were removed in the robustness analysis above and are from a single study, would result in a

nonsignificant half p -curve right skew test and a nonsignificant full p -curve test ($ps > .10$).

However, removing half of the total p values included in the analysis would not result in a flatter p curve than would be expected if the studies had 33% statistical power.

Interim Summary. Evidential value was observed for the relationship between RewP and depression, but the robustness check indicated that the removal of one study led to inconclusive support for evidential value. The estimated power for the primary p curve was low at 27% (90% CI: 10%, 51%), and the estimated power of the secondary p curve was similarly low at 27% (90% CI: 7%, 56%).

Discussion

Contrary to expectations, evidential value was observed for a relationship between ERN and depression and for a relationship between RewP and depression in the primary p -curve analyses. However, an exploratory ERN sensitivity analysis and a planned RewP robustness check indicated that dropping the p values from a single study from each set of ERP studies would render each p -curve analysis inconclusive. Hence, these findings provide weak support for the ERN and RewP candidate ERPs to be considered biomarkers of depression. The statistical power for the included ERN and RewP studies was quite low (point estimates: 20% - 27%). The effect sizes of the relationship between high depressive symptoms and small ERN/RewP were estimated in the medium range (Cohen's $d \sim .50$). The model for estimating the effect size of the relationship between large ERN and high depressive symptoms failed to converge, possibly due to heterogeneity of effects or the inclusion of too few participants, and was consequently inconclusive. Taken together, these findings suggest that the relationships between ERN/RewP and depression might not be primarily due to the selective reporting of significant analyses or undisclosed use of other QRPs, but the evidence in support of this conclusion is weak.

The present *p*-curve analyses yielded larger effect sizes for a relationship between ERN/RewP and depression than the previous meta-analysis (Moran et al., 2017). For the subset of ERN studies that showed a small ERN relates to high depressive symptoms, a medium effect size was observed (Cohen's $d = .59$), but the analysis of studies that showed a large ERN relates to high depressive symptoms was inconclusive. This inconclusive finding suggests that the included studies were underpowered, not that there is no evidence for a large ERN relating to depression. For those RewP studies that showed a small RewP was related to high depressive symptoms, the effect size was estimated as medium (Cohen's $d = .48$). The previous meta-analysis estimated a fairly small, statistically significant effect between large ERN and high depression (Hedge's $g = .21$, 95% CI: 0.02, 0.40) and a nonsignificant relationship between RewP and depression (Hedge's $g = -.14$, 95% CI: -.47, 0.18; Moran et al., 2017). The discrepancy between effect size estimates in the *p*-curve analysis and previous meta-analysis may be due to an overestimate of the true effect sizes in the *p*-curve analysis due to effect size heterogeneity (Simonsohn et al., 2014a; van Aert, Wicherts, & van Assen, 2016), which is a limitation of the *p*-curve approach. Specifically, we only included studies in the *p*-curve that were in the same direction. Although this limitation of the *p*-curve approach can be circumvented by stratifying results on each level of a moderator, the included studies were underpowered to do so (e.g., *p*-curve of depression and comorbid anxiety). In light of the findings that there is evidential value for relationships between ERN/RewP and depression, the present analyses at least support the interpretation of moderators of these relationships from the previous meta-analysis.

However, the evidence for evidential value in the current ERN/RewP and depression literature is weak based on the sensitivity and robustness checks of the primary *p*-curve analyses.

The removal of the lowest p values from a single ERN study (see exploratory sensitivity analysis) and a single RewP study (see robustness check) rendered the right-skew tests of the half and full p curves nonsignificant. The alternative ERN p curve for ERN was also inconclusive, but the alternative RewP did show evidential value. Notably, there was clear statistical evidence that the distribution of p values was not flatter than would be expected with an average statistical power of 33%. Hence, after excluding a single study from each primary p curve, the p curve analyses failed to conclude that there is evidential value but were also unable to conclude that there is no evidential value. In order to make a stronger claim in either direction more studies that are adequately powered to detect a relationship between these ERPs and depression are needed.

The estimated statistical power of both literatures was low. The primary ERN studies had an estimated statistical power of 20%, and the primary RewP studies had an estimated statistical power of 27%. Such low statistical power is common in other meta-analyses of ERN studies. For example, the average statistical power of studies relating ERN to anxiety symptoms (Moser, Moran, Kneip, Schroder, & Larson, 2016) was conservatively estimated at around 10% to detect the estimated effect size of a Cohen's $d = -.36$ (Baldwin, 2017). Notably, the present estimates of statistical power are lower than those from the broader field. For example, the statistical power to detect a medium size effect is estimated to be 44% in the fields of cognitive neuroscience and psychology (Szucs & Ioannidis, 2017). A preregistered analysis of psychophysiology studies using the p -curve approach showed relatively low average statistical power of recent studies (45%; Carbine, Lindsey, Rodeback, & Larson, 2019). Another preregistered review of 150 ERP studies estimated an achieved power of .35-.73 for medium effect sizes (Clayson, Carbine, Baldwin, & Larson, 2019). Although low statistical power seems endemic to cognitive

neuroscience, with ERP studies and other psychological research being no exception, the statistical power of studies of the relationship between ERN/RewP and depression appears lower than what is common for the broader field.

Three important determinants of statistical power that are relevant for the examination of ERN/RewP and depression include the internal consistency of ERP scores, sample size, and statistical design. ERP scores with low internal consistency can lead to low statistical power (Boudewyn, Luck, Farrens, & Kappenman, 2017; Clayson & Miller, 2017b; Fischer, Klein, & Ullsperger, 2017; Kolossa & Kopp, 2018; Luck & Gaspelin, 2017). For example, low internal consistency due to noisy measurement can reduce effect sizes in ERP studies, and this relationship has been observed in both between-group (Hajcak, Meyer, & Kotov, 2017) and within-person (Clayson & Miller, 2017a) ERP studies. To our knowledge, none of the studies reported in the meta-analysis examined the internal consistency of ERN or RewP scores to ensure adequate reliability or comparable reliability between groups, when relevant. For example, one study to date has examined the internal consistency of ERN scores in participants with major depressive disorder (Baldwin, Larson, & Clayson, 2015). In order to achieve adequate internal consistency (reliability estimates $> .70$), controls needed at least 13 trials, but participants with major depressive disorder needed 23 (Baldwin et al., 2015). A recent meta-analysis showed that ERN score reliability is moderated by the clinical status of participants and the type of paradigm used in a study (Clayson, 2019), which is also notable considering that many different types of paradigms have been used to record ERN in people with depression (see Moran et al., 2017). Generalizability of such findings notwithstanding, it is possible that lower reliability scores in people with depression could be biasing observed effect sizes.

With regard to sample size, the average sample size across the 20 ERN studies included in the primary p -curve analysis was 78, and the average sample size across the 26 RewP studies was 83. These sample sizes are considerably larger than the typical ERP study, which includes an average of 29 participants (Clayson et al., 2019). However, these sample sizes are too low for the effect sizes of interest, which were estimated in the medium range. Such small sample sizes limit the ability to identify replicable biomarkers, particularly when the effect size is small (Miller, Clayson, & Yee, 2014). In order to achieve 80% statistical power to detect a Cohen's d of .50 between a healthy control and depression group, a total of 128 participants (64 participants per group) would be required (Clayson et al., 2019). When considering the small effect sizes from the recent meta-analysis (Moran et al., 2017), a total of 788 participants (394 participants per group) would be required (Clayson et al., 2019) to achieve 80% statistical power to detect a Cohen's d of .20 between two groups. Studies of ERN/RewP and depression would benefit from conducting and reporting *a priori* power analyses to justify sample sizes. Although *a priori* power analyses are uncommon in ERP studies (Larson & Carbine, 2017), a relatively recent ERP guidelines paper states statistical power should be explicitly justified and reported (Keil et al., 2014).

In light of the weak evidence for evidential value of ERN and RewP studies of depression, there are some other solutions for moving forward. First, some papers could not be included in the p -curve analysis due to not statistically comparing control and depression groups. For example, instead of examining the interaction of a 2-Group (controls, depression) x 2-Event (correct, incorrect) repeated measures analysis of variance (ANOVA), within-group comparisons were conducted, and group differences were subsequently inferred. However, absence of within-group differences in one group but not another group is not evidence for differences between two

groups. Unfortunately, failing to directly compare within-group differences across groups is quite common in cognitive neuroscience, but to support the claim of group differences, groups must be statistically compared (Nieuwenhuis, Forstmann, & Wagenmakers, 2011).

Second, there were also several instances when the data processing pipeline used to reduce EEG recordings to ERP amplitudes was unclear or incomplete. We recently reviewed 150 ERP studies and found that studies only reported an average of 63% of key methodological parameters EEG data reduction (Clayson et al., 2019). We encourage the use the recent ERP reporting guidelines paper (Keil et al., 2014) to facilitate communication among ERP researchers. Such clear communication of ERP data processing is essential for the reproducibility and replicability of research (Clayson et al., 2019).

Third, some of the reviewed papers included many factors in the primary ANOVAs of interest, and this approach is quite common in ERP studies (Clayson et al., 2019; Luck & Gaspelin, 2017) and psychology more broadly (Cramer et al., 2015). Including many factors in an ANOVA increases the family wise error rate due to the need to control for multiple comparisons (Cramer et al., 2015; Luck & Gaspelin, 2017). Although there are procedures available for controlling the family-wise error rate (e.g., Bonferroni correction), these procedures typically reduce statistical power, which was already quite low in the ERN/RewP studies of depression. Hence, it is recommended that researchers home in on the specific analyses of interest to avoid unnecessary comparisons. This sentiment was well phrased by Luck and Gaspelin (2017, p. 152), “a researcher who wants to avoid significant but bogus effects—and promote real scientific progress—would be advised to include as few factors as possible”.

Fourth, the approach to clustering participants into groups was sometimes unclear or was based on depression symptom measures, which can be problematic. For example, some studies

failed to include information about how diagnostic status or a depression diagnosis was made. Other studies forced participants into depression groups based on depression symptom scores that were measured continuously, but this practice is inadvisable and can lead to misleading associations (Altman, 1994; Altman & Royston, 2006; Dawson & Weiss, 2012; Naggara et al., 2011; Royston, Altman, & Sauerbrei, 2005; Thoresen, 2019). The dichotomization or categorization of a continuous variable should typically be avoided, because it can lead to reduced statistical power, biased estimates, an increased in type I error rates, concealment of non-linearity (in the case of dichotomization), propagation of arbitrary cut points, and spurious interactions. Furthermore, these problems are exacerbated in studies of small samples (Altman & Royston, 2006; Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002), such as those observed in the included ERP studies of depression.

In order for ERN and RewP to be classified as biomarkers or endophenotypes, these ERPs must demonstrate consistent and robust relationships with depression symptoms (Miller & Rockstroh, 2013). Present findings only provide some support for this claim, as evidential value was weak for each ERP. It is possible that ERN and RewP might be biomarkers for risk for the development of depression psychopathology categorically, rather than depression symptoms dimensionally. ERN has been proposed as a biomarker of risk for different forms of psychopathology, including depression and anxiety (see Hajcak, Klawohn, & Meyer, 2019). More robust relationships between ERN/RewP and depression might be shown in those studies that only examine participants with clinical depression. More specifically, there are many different types of relationships that were examined in the previous meta-analysis (Moran et al., 2017) and the present *p*-curve analysis that might contribute to the heterogeneity of effect sizes. Types of analyses include examining relationships between ERN/RewP and depression

symptoms in healthy undergraduates (e.g., Chang, Davies, & Gavin, 2010) or participants with diagnosed with clinical major depressive disorder (e.g., Ruchow et al., 2006), relationships between remitted and non-remitted in participants with depressive disorders (e.g., Weinberg, Liu, & Shankman, 2016a), and correlations between ERN and depression symptoms and response to treatment (Schrijvers et al., 2009). It is possible that the relationship between ERN/RewP and depression is only evident in those participants with a risk for developing depression or those participants with depressive disorders.

There are some additional limitations to note. First, as with any meta-analytic review, the inclusion/exclusion criteria constrained the studies included in our analyses. However, to limit potential researcher bias, we chose to closely adhere to the inclusion/exclusion criteria of the previous meta-analysis by Moran et al. (2017) and to pre-register our approach using the registered report format. Second, our *a priori* power analysis was based on estimates from the previous meta-analysis, but we failed to consider that not all of the included studies would have significant effects. Hence, our achieved statistical power for our primary *p*-curve analyses was lower than we had anticipated, and we failed to achieve adequate power for examining the evidential value for a relationship between ERN and depression in samples of people with depression and comorbid anxiety disorders.

Contrary to our expectations, the present analyses indicated that there is evidential value, albeit weak, for relationships between ERN/RewP and depression. The presence of evidential value supports the interpretation of moderators of the relationships between these ERPs and depression by suggesting that these relationships are not primarily due to QRPs. Furthermore, present findings provide some support for a robust relationship between ERN/RewP and depression, which strengthens the claim that these ERPs might represent biomarkers or

endophenotypes of depression. In light of the evidence for evidential value being weak, we provided some suggestions for moving this work forward. These suggestions included increasing sample sizes and statistical power, conducting *a priori* power analyses, assessing internal consistency of ERP scores, and carefully planning statistical approaches to obviate the need for extraneous comparisons due to unnecessary ANOVA factors and for the dichotomization or categorization of depression symptoms.

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Footnote

1. We choose to use the term garden of forking paths over p hacking, because the former term is less pejorative than the latter. The garden of forking paths emphasizes how researchers might make seemingly reasonable, *post hoc* decisions when analyzing their data that inflates the chance of finding a significant effect. Because the data determine the decision tree, the probability of finding an erroneous or inflated effect is increased. In contrast, p hacking implies a seemingly malicious intent to exhaust data flexibility (or researcher degrees of freedom) to obtain significant p values, such as trying multiple analytic techniques, excluding participants or subsets of data, or analyzing data with or without covariates. Regardless of the intent, p -curve analyses were used to determine whether a set of studies contains evidential value.
2. We chose not to cite papers as examples of some of the shortcomings we observed in the reviewed research. These issues are mentioned because we observed them across multiple studies. Thus, we view these issues as limitations of this field, rather than the shortcoming of any particular study.

Table 1

ERN p-Curve Disclosure Table

Original Paper	Quoted Text Indicating Prediction of Interest	Study Design	Key Statistical Result	Quoted text with Statistical Results	Results	Robustness Results	Alternative results
Aarts, Vanderhasselt, Otte, Baeken, and Pourtois (2013)	We surmised that MDD patients might show a larger ERN/Ne than HCs (Olvet & Hajcak, 2008).	A 2 (accuracy: error vs. hit) by 2 (group) repeated measures ANOVA, with the alpha cutoff set to $p < .05$.	2 x 2 Attenuated Interaction	This interaction became significant when including speed as a covariate given that on average MDD patients were slower than HCs, $F(1, 37) = 4.64, p < .05$.	$F(1, 37) = 4.64; p < .05$	N/A	A 2 (accuracy: error vs. hit) x 2 (ERP component: ERN vs. Pe) x 2 (group: HCs vs. MDDs) ANOVA was carried out... a significant main effect of group was evidenced, $F(1, 38) = 5.30, p < .05$
Alexopoulos et al. (2007)	ERN and Pe during an emotional go/nogo task distinguish depressed elders who remain symptomatic from those who achieve remission after treatment with escitalopram.	Comparisons were conducted using Student's t-test.	2-Cell	Nonremitters had larger amplitude of ERN and smaller amplitude of Pe in these leads... $t(10) = -2.95, p = .04$	$t(10) = -2.95; p = .04$	N/A	Nonremitters had larger amplitude of ERN and smaller amplitude of Pe in these leads... $t(10) = -2.24, p = .05$
Chang et al. (2010)	We hypothesized... depression... will collectively account for significant interindividual variability of both ERN and Pe amplitudes in healthy adults. In addition, we hypothesized that these characteristics will be significantly related to both ERN and Pe amplitudes.	Then, we performed two-step hierarchical multiple regression analyses.	Linear Trend	Examination of the beta weights revealed that both depression and antisocial personality measures were significant predictors for the ERN peak-to-peak amplitude... $\beta = 0.701; t = 2.405; p = 0.025$	$t(22) = 2.405; p = 0.025$	N/A	The beta weight showed that both depression and antisocial personality were significant predictors... ERN Peak: $\beta = -0.705; t(21) = -2.348; p = 0.028$
Chiu and Deldin (2007)	Event-related potentials associated with sequential aspects of error processing were measured as participants with and without major depressive disorder performed a common speeded response-competition task that reliably elicits errors and their accompanying physiological processes.	An initial 5x5x2 multivariate analysis of variance (MANOVA) with Wilks's lambda correction was performed for the peak amplitudes for error-related negativity and error positivity in the neutral condition.	2-Cell	Moreover, the depressed group showed higher amplitudes of error-related negativity than the comparison subjects (group effect: $F=4.6, df=1, 33, p<0.05$).	$F(1,33)=4.6; p<0.05$	N/A	In addition, the depressed group exhibited greater error-related negativity than the comparison group (group effect: $F=6.3, df=1, 33, p<0.05$).

Endrass et al. (2014)	Finally, we examined in an explorative fashion group differences with regard to the association between ERN and symptom expression .	Pearson correlations were calculated between ERN amplitude and clinical symptoms (BDI-II, OCI-R, Y-BOCS, and LSAS-SR).	Linear Trend	Significant correlations of ERN and CRN with depressive symptoms (BDI-II) and of ERN with obsessive-compulsive symptoms (OCI-R) were found... $r = -.40, p < .01$	$r(70) = -.40; p < .01$	N/A	BDI-II and ERN correlation for just participants with SAD: $r(22) = -.54, p < .01$
Fissler et al. (2017)	We hypothesized that chronically depressed patients would show a reduced ERN relative to healthy controls , and that mindfulness training would significantly increase the ERN in patients, whereas this would not be the case in the resting control condition.	A series of multivariate analyses of variance (ANOVAs)	2-Cell	A series of multivariate analyses of variance... indicated that, as compared to the depressed group, healthy controls had significantly higher ERN and Δ ERN magnitudes at both FCz and Fz.	ERN FCz: $F(2,74) = 6.25; p = .015$	N/A	ERN Fz: $F(2,74) = 6.88; p = .011$
Gajewski et al. (2017)	Given a possible overlap of burnout and depression we assume that in subjects scoring high in burnout the Ne is enhanced , while the FRN is reduced, as it is in patients with depression.	Repeated measures ANOVA with the factors Trial Type (correct vs. error) and OLBI Group (EE+ vs. EE-) or PHQ-9 Group (DE+ vs. DE-).	2 x 2 Attenuated Interaction	The effect of the PHQ-9 Group ($F(1,49) = 2.4, p = 0.125, \eta^2 = 0.047$) or the interaction Trial Type x Group ($F(1,49) = 2.0, p = 0.160, \eta^2 = 0.040$) did not reach significance.	N.S.	N/A	A negative relationship between the amplitude of the Ne and the PHQ-9 scores was found... $\beta = -.29; F(1,47) = 4.63; p = .036$
Georgiadi et al. (2011)	Consistent with previous neuroimaging findings in remitted depression (e.g., Liotti et al., 2002), it was predicted that depressed individuals currently in remission would exhibit larger ERN amplitudes relative to healthy participants and patients with acute MDD , possibly reflecting increased sensitivity, whether conscious or not, to erroneous performance during the remission phase.	The primary ERN analysis employed a three-group analysis of covariance (ANCOVA)	3-Cell (Treatment 1 vs. Control)	A three-group ANCOVA revealed a significant main effect of group... due to a greater ERN amplitude in the remitted group relative to both the control group and the acute group... peak amplitude: $F(1,34) = 7.99, p = .008$	$F(1,34) = 7.99; p = .008$	N/A	The main effect of group was qualified by a significant Age x Group interaction (peak amplitude: $F(2,66) = 4.04, p = .022$).

Hanna et al. (2016)	The aims of the study were to examine the relation of the ERN to the OCD symptom dimensions noted earlier and Child Behavior Checklist (CBCL) Syndrome Scales.	Multiple linear regression analyses were used to examine the relation of the ERN and Δ ERN to OCD symptom dimensions.	Linear Trend	The ERN had significant associations only with the Withdrawn/Depressed scale scores ($p = .014$)	$r(158) = -.30$; $p = .014$	N/A	The delta ERN had significant associations only with the Withdrawn/Depressed scale scores... $r(158) = -.25$; $p = .03$
Hanna et al. (2018)	The first aim was to compare accuracy and ERN amplitudes in OCD patients, MDD patients , and HC, followed by similar comparisons in OCD patients with MDD (OCD + MDD), OCD patients without MDD (OCD – MDD), MDD patients, and HC.	A repeated-measure ANCOVA with group (OCD patients, MDD patients, and HC) as a between-subject factor and response type (correct and error) as a within-subject factor and with age and accuracy as covariates.	3-Cell (Control vs Group II)	There was a significant group effect, $F(2, 173) = 3.69$, $p = .027$... ERN amplitudes were not significantly different either between OCD and MDD patients ($p = .17$) or MDD patients and HC ($p = .49$).	N.S.	N/A	The ERN was significantly enlarged in OCD + MDD patients compared to HC, $F(1, 99) = 6.71$, $p = .011$
Holmes and Pizzagalli (2008)	Therefore, on the basis of prior findings, we hypothesized that unmedicated patients with MDD would show... increased scalp ERN and rostral ACC activation after committing errors.	For the ERN/CRN data, mixed 2x2x4 ANOVAs with group, condition (ERN and CRN), and site (Fz, FCz, Cz, and Pz) as factors were run separately on amplitude and latency scores	2-Cell	Main effect of group ($F[1,34]=5.53$; $P=.03$; partial eta squared =0.14)... Relative to comparison subjects, patients with MDD had significantly larger ERN ($p = .006$; Cohen $d=0.75$) but comparable CRN ($p=.19$).	$F(1,34)=5.53$; $p=.03$	N/A	The main effect of group was confirmed ($F[1,34]=7.12$; $p=.01$).
Holmes and Pizzagalli (2010)	We hypothesized that, relative to controls, MDD participants would show larger ERN to errors , particularly under no-incentive conditions.	A mixed 2 X 2 X 2 X 3 ANOVA with group (MDD participants, controls), condition (reward, no incentive), response (correct, incorrect) and site (Fz, FCz, Cz) as factors was run.	2 x 2 Attenuated Interaction	A significant group X response interaction emerged [$F(1,28) = 4.22$, $p = .049$]... driven by increased ERN amplitudes for the MDD relative to the control ($p < .053$) participants.	$F(1,28) = 4.22$; $p = .049$	N/A	N/A

Kalayam and Alexopoulos (2003)	This study tested the hypothesis that frontal error negative wave amplitude during Stroop activation distinguishes depressed elders who remain symptomatic from those who achieve remission.	Multivariate analysis of variance (MANOVA) to test the overall group effect, lead effect, and lead-by-group interaction effect.	2 x 6 Attenuated Interaction	MANOVA showed a significant overall group effect ($F=4.89$, $df=6$, 15 , $p<0.006$)... and a significant group-by-lead interaction effect ($F=6.24$, $df=5$, 16 , $p<0.01$), indicating that the significance of group effect depends on the location of the lead.	$F(5, 16)=6.24$; $p<0.01$	Didn't report necessary analyses	Amplitude of left frontal F3 error negative waves was correlated with percent change of depressive symptoms (according to the Hamilton depression scale) from baseline ($r=0.56$, $df=21$, $p<0.01$).
Ladouceur et al. (2012)	On the basis of previous findings of abnormally reduced ACC activity associated with a clinical state of depression (George et al., 1997; Liotti and Mayberg, 2001), we hypothesized that, relative to low-risk healthy controls, depressed youth would show reduced ERN amplitude .	ERP measures were analyzed using a mixed MANCOVA model with group (MDD, HC) as between-subject variable and response type (correct, error) as within-subject variables. Age was included as a covariate variable in both models.	2 x 2 Attenuated Interaction	There was a significant diagnostic group by response type interaction, $F(1, 34) = 10.76$, $p = .002$, partial eta squared = .24 ... ERN amplitude was significantly less negative on error trials in the MDD compared to the HC group.	$F(1, 34) = 10.76$; $p = .002$	N/A	After removing participants with other comorbid conditions (mostly disruptive disorders), the group by response type, $F(1, 26) = 6.4$, $p = .018$, partial eta squared = .20 ... remained significant.
Olvet et al. (2010)	Based on the literature, we hypothesized that the ERN would be larger in individuals with MDD compared to healthy controls.	Mixed ANOVA with Trial Type (Error vs. Correct) as the within-subjects factor and Group (HC vs. MDD) as the between-subjects factor.	2x2 Attenuated Interaction	Thus, both the ERN and the CRN were similar in the MDD and HC groups, and the Δ ERN was also comparable between the MDD and HC groups.	N.S.	N/A	In the MDD group, there were significant correlations between the Δ ERN and the Anhedonic Depression subscale.. ($r(20)=0.53$, $p=0.01$)
Peters et al. (2018)	We also hypothesized that by contrast youth with greater depressive symptoms would exhibit a blunted ERN . We expected that depression would moderate the effects of anxiety and pubertal timing, such that high levels of depression would be associated with a reduced ERN only among early developing, anxious youth.	Generalized multiple linear regression.... model included the main effects of diagnosis (coded [HC = 0], [AD = 1]), pubertal timing, and CDI depression t scores, as well as all two-way interactions, and a diagnosis $\times$ pubertal timing $\times$ CDI depression three-way interaction.	2 x 2 Attenuated Interaction	Two-way interaction between diagnostic group and depressive symptoms (b-weight = -.80 , $SE = .33$, $p = 0.016$). Follow-up analyses indicated that among AD participants, higher depression was associated with an attenuated Δ ERN, but not among HC participants.	$t(51) = 2.49$; $p = 0.016$	N/A	There was also a two-way interaction between depressive symptoms and pubertal timing (b-weight = .22 , $SE = .07$, $p = 0.003$).

Schoenberg (2014)	Based on heightened sensitivity to negative cues, it was predicted enhancements in both components of the waveform would be observed in patients.	Condition (Go, NoGo) × Group (CD, RD, HC) repeated measures ANOVA (r-ANOVA), with Greenhouse Geisser correction where appropriate... Sex was also examined as an IV via a Condition × Group × Sex r-ANOVA.	2 x 2 Attenuated Interaction	Significant Group × Sex ($F(1, 77) = 6.156, p = 0.02$)... interactions revealed male patients had significantly lower ERN amplitudes compared to male HCs.	$F(1, 77) = 6.156; p = 0.02$	N/A	Two-level Group × Sex IV analyses indicated main effects of... Group ($F(1, 68) = 7.132, p = 0.009$), highlighting a general pattern of lower ERN amplitudes in patients compared to HCs.
Schrijvers et al. (2008) ¹	We hypothesized that, compared to healthy controls, patients with MDD would show reduced action monitoring as reflected in reduced Ne/ERN amplitudes	Repeated measures general linear model (GLM) with group (2 levels: control vs MDD) as between-subjects factor	2-Cell	The Ne/ERN amplitudes of the controls did not significantly differ from those of the patients [$F(1,49) = 1.18, p = .283$].	N.S.	N/A	The benzo subgroup did demonstrate a significant difference in Ne/ERN amplitude relative to the controls [$F(1,36) = 4.11, p = .05$].
Schrijvers et al. (2009)	We expected the dysregulation of the action-monitoring process during the early stages of a severe depressive episode to improve when depression severity substantially decreases.	Finally, in the patient group correlations were used to assess relationships between the changes in ERP amplitudes and behavioural data on the one and the changes in symptom severity on the other hand.	Linear Trend	Between-session changes in Ne/ERN amplitudes at Cz correlated... with the percentage of change in HDRS scores [$r = 0.74, p = 0.01$] and BDI-II scores [$r = 0.6, p = 0.05$].	$r(8) = 0.74; p = 0.01$	N/A	Between-session changes in Ne/ERN amplitudes at Cz correlated (nearly significantly with the percentage of change in HDRS scores [$r = 0.74, p = 0.01$] and BDI-II scores [$r(28) = 0.6, p = 0.05$]
Tabachnick et al. (2018)	We also hypothesized that more depressive symptoms would be associated with a blunted ERN among all children.	Correlational analyses	Linear Trend	The ERN was positively correlated with depressive symptoms, such that more symptoms were associated with smaller ERNs ($r = .22, p < .05$).	$r(78) = .22; p < .05$	N/A	The final model was significant... as was the interaction term (b -weight = 3.94, $SE = 1.72, p = .03; t(73) = 2.21$)

Tang et al. (2013)	We hypothesized that: (1) individuals with depression show a heightened sensitivity to error responses reflected by an enhanced ERN amplitude	A repeated measures analysis of variance (ANOVA) was applied... The between-group factor was Group (depression and healthy controls) and the within-group factor was Recording Site (Fz, FC1, FCz, and FC2).	2-Cell	There was a significant group effect on ERN amplitude ($F(1,43) = 5.68, p = 0.05$). ERN amplitudes in individuals with depression were significantly enhanced in comparison to those in healthy controls.	$F(1,43) = 5.68; p = 0.05$	N/A	N/A
Weinberg et al. (2012)	However, it was further anticipated that the ERN would be reduced in individuals who meet criteria for both GAD and MDD compared with those with GAD alone.	We conducted three 2 (response type: correct or error) $\times$ 3 (group: GAD, comorbid, and HC) mixed-model ANOVAs.	3 x 2 Attenuated Trends	However, the difference between the ERN and CRN varied according to group, reflected in a significant group by response interaction, $F(2, 82) = 3.72, p < .05$, partial eta squared = .08 .	$F(2, 82) = 3.72; p < .05$	2 x 2 interaction not reported	N/A
Weinberg et al. (2016a)	Following this, we examined potential opposing effects of depression symptoms, which we expected would be associated with a decreased ERN	Multiple linear regression analyses	Linear Trend	Symptoms of depression and checking showing opposing associations with delta ERN magnitude, such that symptoms of depression related to reduced delta ERN... $\beta = .15, t = 2.93, p < .01$	$t(510) = 2.93; p < .01$	N/A	Correlation with depression and ERN: $r(513) = .10; p < .05$
Weinberg et al. (2016b)	We predicted that the magnitude of the ERN would be blunted in remitted melancholic depression relative to both controls and remitted nonmelancholic depression.	2 (response type: correct or error) $\times$ 3 (group: HC, rNMD, and rMD) mixed-model ANOVAs were conducted	3 x 2 Attenuated Trends	However, the difference between the ERN and CRN (delta ERN) varied according to group, reflected in a significant group by response interaction $F(2, 102) = 3.68, p = .03$, partial eta squared = .07 .	$F(2, 102) = 3.68; p = .03$	2 x 2 interaction not reported	The interaction of group and response type remained significant when including accuracy as a covariate in the model: $F(2, 101) = 3.76, p = .03$.

Note. For ease of references studies included in the ERN *p*-curve disclosure table are denoted using a * in the Reference section. Information on all coded articles is shown in the supplementary material posted on Open Science Framework. † Indicates a study with a test statistic that was entered into the secondary *p*-curve analyses but was excluded by the software for having a *p* value > .05.

Table 2

RewP p-Curve Disclosure Table

Original Paper	Quoted Text Indicating Prediction of Interest	Study Design	Key Statistical Result	Quoted text with Statistical Results	Results	Robustness Results	Alternative results
Bakic et al. (2016)	We assessed if MDD could impair internal (ERN) and/or external (FRN) RPE signals , and whether it would be associated with decreased RL (at the behavioral level) compared to HCs in this task.	A mixed-model ANOVA was performed on the average mean amplitudes with group as between subjects and condition and response accuracy as within subject factors.	2 x 2 x 3 Attenuated Interaction	By comparison, for the FRN, the analysis revealed a significant group $\times$ accuracy $\times$ condition interaction ($F(2,138) = 3.84, p = .025, \eta^2 = .05$)	$F(2,138) = 3.84; p = .025$	N/A	N/A
Barch et al. (2019) ¹	We predicted that compared with the waitlist (WL) control, children undergoing PCIT-ED treatment would show an increase in the RewP to wins... and that the magnitude of the increase would correlate with the degree of depression and anhedonia reduction.	Whether changes in Winresid from pre- to posttreatment in the PCIT-ED group were correlated with depressive or anhedonia symptom change, using partial correlations of difference scores (postbaseline), controlling for age	Linear Trend	Partial correlations controlling for age demonstrated that in the treatment group a greater increase in Winresid from baseline to posttreatment was marginally associated with a greater decrease in MDD symptoms ($r = -.24; p = .05$)	$r(58) = -.24; p = .05$	N/A	N/A
Belden et al. (2016)	Based upon the findings from older children and adolescents discussed above, we hypothesized the following: that treatment-naïve children with PO-MDD would show significantly smaller responses to reward (i.e., RewP) than typically developing same-age peers; and that among children with PO-MDD, children who were more severely depressed would show significantly smaller responses to reward (i.e., RewP).	Our primary analysis was a repeated-measures analysis of variance comparing the two groups across the two conditions (reward and loss). We also conducted a univariate analysis of covariance to examine whether the hypothesized effect of PO-MDD on RewP was significant when covarying for children's concurrent diagnosis of ODD, ADHD, and GAD.	2 x 2 Attenuated Interaction	The results indicated a significant condition-by-diagnostic group interaction effect... children in the PO-MDD group had a smaller response to reward trials compared to healthy children ($t[76] = 2.72, p = .008$), but the PO-MDD and healthy children did not differ in response to loss trials ($t[76] = 1.05, p = .30$).	$F(1,76) = 7.39; p = .008$	N/A	The children with MDD showed a significant reduction compared to the healthy controls in the residualized reward scores ($t[76] = 3.16, p = .002$).

Berry et al. (2019)	In light of findings in MDD (Brush et al., 2018), it was hypothesized that RewP would show more negative within-task amplitude changes relative to FN for participants with relatively high levels of depression symptoms.	Model 1 examined whether depression score moderated the effects of experimental condition on within-task RewP/FN amplitude changes and included cross-level interactions between depression, experimental condition, and trial number.	2 x 2 x 2 Reversal of Attenuated Interaction	A significant Trial × Condition × Depression interaction was observed... RewP amplitudes showed more positive slopes in relation to higher depression scores, as evidenced by a significant Trial × Depression interaction... $t(12070) = 3.92, p < 0.001$. The same interaction term... revealed the opposite pattern, with FN... showing more negative slopes in relation to higher depression symptoms... $t(12070) = -4.37, p < 0.001$	$t(12070) = 3.92, p < 0.001$ $t(12070) = -4.37, p < 0.001$	N/A	ΔRewP was significantly positively correlated with total and affective depression scores on the PAI-DEP... $r(43) = 0.36$
Bowyer et al. (2019)	The difference in brain response to gain versus loss that defines the RewP will relate negatively to persistent depressive symptoms to a significant degree , and this association will account for any corresponding relationship with current depressive symptomatology.	Each ANOVA included a within-subject feedback factor representing gain and loss, along with either persistent depression or current depression score as a continuous between-subjects factor	2 x 2 Attenuated Interaction	Also consistent with prediction, a significant Persistent Depression × Gain/Loss interaction was observed, $F(1, 199) = 8.758, p = 0.003$, reflecting reduced gain-loss differentiation (RewP) for individuals scoring higher on the persistent depression symptom variable.	$F(1, 199) = 8.758; p = 0.003$	N/A	Number of lifetime MDEs correlated with RewP: $r(199) = -0.14$
Bress et al. (2012)	We predicted that, consistent with findings from behavioral and neuroimaging studies, the FN would differentiate monetary losses from monetary gains and that, as in adults, the difference between loss and gain trials would be attenuated among children with higher depressive symptom scores.	A series of bivariate correlations was performed in order to relate the FN (i.e., losses minus gains) to psychological and developmental measures.	Linear Trend	However, the FN was significantly correlated with CDI:T ($r = .38, p < .01$)	$r(62) = .38; p < .01$	N/A	When CDI:T was broken down into its component measures, FN was significantly correlated with CDI:SR ($r(62) = .33, p < .01$)

Bress et al. (2013)	We interviewed the same participants nearly 2 years after their initial visit in order to determine whether blunted FN amplitude at baseline would predict first MDE and severity of depressive symptoms.	Independent-samples t tests were used to compare the FN in the groups with and without MDEs over the follow-up period.	Linear Trend	Depression severity was significantly predicted by the ERP response to gain ($r = -.26, p < .05$), but not to loss ($r = -.12, p = .33$).	$r(66) = -.26; p < .05$	N/A	The group with MDEs had a smaller FN at initial evaluation than the group without... $t(66) = -2.04$.
Brush et al. (2018)	We hypothesized an attenuated delta RewP for individuals with MDD relative to healthy control individuals.	An independent-samples t test was conducted to test the primary hypothesis of differences in Δ RewP (and Δ RewPresid) by depression status.	2-Cell	Individuals with MDD had a significantly smaller Δ RewP ($2.01 \pm 2.52 \mu V$) than control participants ($4.13 \pm 3.33 \mu V$) ($t(98) = 3.61, p < .001, d = 0.72$)	$t(98) = 3.61; p < .001$	N/A	A trend toward a smaller RewP for individuals with MDD relative to control... ($t(98) = 2.10, p_{corrected} = .04$).
Burkhouse et al. (2018) ²	We first sought to replicate previous findings demonstrating a negative relationship between RewP and depressive symptomatology among patients and healthy controls at baseline	We also conducted a series of bivariate correlations to examine relations between depressive and anxiety symptoms and RewP at baseline.	Linear Trend	Bivariate correlations revealed that participants with higher depressive symptoms exhibited a more attenuated RewP at baseline ($r = -0.27, p = .01$).	$r(61) = -0.27, p = .01$	N/A	Within patients who received SSRIs, a more attenuated T1 RewP was associated with greater reduction in depressive symptoms (Beta = $-0.39, t(28) = -2.04, p < .05$)
Burkhouse et al. (2016)	Second, given the well-established link between depression and reward responsiveness,[8, 9, 15, 21, 22] we examined whether the RewP would correlate with depressive symptom change among individuals with CAD.	Hierarchal regression	Attenuated Interaction	There was a significant RewP $\times$ diagnostic status interaction, $t(47) = 2.51, p = .02, \beta = 5.35$. The RewP predicted BDI symptom change among the CAD group, $t(19) = 2.73, p = .01, \beta = 1.13$, but not the pure anxious group, $t(27) = -.18, p = .86, \beta = -.04$.	$t(47) = 2.51; p = .02$	N/A	The RewP predicted BDI symptom change among the CAD group, $t(19) = 2.73, p = .01, \beta = 1.13$

Burkhouse et al. (2017)	We expected that there would be a negative relation between RewP and distress/misery symptoms , whereas there would be no relation between the RewP and fear-based symptoms of anxiety.	Next, we tested the effect of RewP on each factor using linear regression analyses.	Linear Trend	Individuals with greater distress/misery symptoms (i.e., factor 1) exhibited a greater reduction in the RewP, $\beta = -.24$, $t = -2.17$, $p = .03$, $r = -.24$	$t(76) = -2.17$; $p = .03$	N/A	This relation remained significant after adjusting for current GAF scores... $p < .05$, $r(75) = -.23$
Distefano et al. (2018)	We hypothesized that greater depression symptoms would be associated with a smaller RewP , but we had no specific hypotheses regarding differences between monetary and social tasks.	Outcome [favorable (like) vs unfavorable (dislike)] x Location (Fz, FCz and Cz) x Peer Sex (male vs. female) x Dysphoria mixed-measures ANCOVA, with dysphoria symptoms included as the continuous covariate	3-Way Interaction (Attenuation of attenuation)	A Peer Sex x Outcome x Dysphoria interaction, $F(1, 112) = 4.01$, $p = .048$... Results indicated that greater dysphoria symptoms were associated with a smaller social RewP to female feedback... but not male feedback.	$F(1, 112) = 4.01$; $p = 0.048$	N/A	N/A
Foti and Hajcak (2009)	Additionally, based on the notion that depression might be related to reduced sensitivity to rewards versus non-rewards, we predicted that the magnitude of the FN would be inversely related to levels of depression .	To assess for the specificity of this relationship, three additional bivariate correlations were performed between the FN and depression, anxiety, and stress subscale scores of the DASS-21.	Linear Trend	Similar to general distress, the FN was inversely related to depression ($r = .23$, $p < .05$)	$r(83) = .23$; $p < .05$	N/A	Associations with depression and stress scores were as robust when examining individuals with a total DASS-21 score greater than zero ($r = .24$, $p < .05$).
Foti et al. (2014)	Based on these definitions, we expected individuals with melancholic MDD to exhibit profound reward dysfunction, as evidenced by diminished FN amplitude and VS activation.	We analyzed FN amplitude using a mixed-model ANOVA including Feedback as the within subjects factor (Win vs. Loss) and Group as the between-subjects factor (MDD vs. Control).	2 x 2 Attenuated Interaction	The ANOVA yielded... an interaction with Group ($F(1,74) = 6.08$, $p < .05$, $\eta^2 = .08$).	$F(1,74) = 6.08$; $p < .05$	N/A	The FN was substantially blunted among those depressed individuals reporting impaired mood reactivity... $t(51) = 4.96$, $p < .001$

Glazer et al. (2019)	First, following immediate outcomes, we predicted that proneness to hypomania and depressive tendencies would be related to an elevated and blunted RewP , respectively, consistent with prior studies.	Next, we examined the Pearson correlations between self-report measures (i.e., HPS and 7 Down) and each ERP difference wave (i.e., the RewP, P3, and LPP) at each delay.	Linear Trend	The RewP following immediate outcomes showed a... significant but opposite correlation with 7 Down ($r = -0.313$, $p = 0.032$).	$r(45) = -0.313$; $p = 0.032$	N/A	N/A
Kujawa et al. (2017)	Our final goal was to test the relevance of these measures to individual differences by examining correlations with symptoms of depression and social anxiety.	Bivariate correlations were computed to examine associations between ERP, self-report, behavioral measures, and symptoms of depression and social anxiety.	Linear Trend	Greater depressive symptoms were associated with... a blunted RewP component to social acceptance... $r(410) = -0.10$	$r(410) = -0.10$	N/A	N/A
Liu et al. (2014)	We hypothesized that, consistent with findings from non-clinical studies, the magnitude of FN would be attenuated in patients with depression compared to healthy individuals.	Repeated-measures analysis of variance (ANOVA) with group (depressed group, healthy controls) and feedback (gain, loss) as factors for FN on gain and loss trials and independent-sample t-test for the difference waves were performed.	2 x 2 Attenuated Interaction	ANOVA with group (depressed group, healthy controls) and feedback (gain, loss) as factors showed a significant... interaction effect of feedback and group ($F(1,52) = 6.263$, $p = 0.016$, $\eta^2 = 0.107$).	$F(1,52) = 6.263$; $p = 0.016$	N/A	For the depressed group, high levels of depressive symptoms measured by the HRSD ($r(25) = -0.430$, $p = 0.025$) were associated with reduction in response to gain feedback.
Mueller et al. (2015)	We hypothesized that depressed individuals would be characterized by increased FRN amplitudes and aMCC activity, although reduced sensitivity for negative feedback may be observed in the context of elevated anhedonic symptoms within the MDD group.	For FRN and FMT, the between-subjects factor was Group (Control vs. MDD) and the within-subjects factors were Electrode and Feedback Valence (Training phase: positive vs. negative feedback) and the critical P-value was set to .05.	2 x 2 Attenuated Interaction	There was a significant Group×Feedback interaction ($F(1,27) = 5.99$, $p < .025$), which was due to increased differentiation between positive and negative feedback in the MDD versus control group.	$F(1,27) = 5.99$ $p < .025$	N/A	There was a Group × Feedback × Electrode interaction indicating that the Group × Feedback interaction was most pronounced at centromedial electrode ($F(1,27) = 10.33$, $p < .003$)

Mulligan et al. (2018a)	In the present study, we hypothesized that the RewP would explain variance in current depressive symptoms that is independent from variance explained by other socioeconomic, psychological, and psychosocial risk factors.	A linear regression predicting PHQ-9 depressive symptoms with RewP, PRQ scores, and their interaction term entered as predictors was conducted to examine whether the RewP and PRQ interact to predict antenatal depressive symptoms.	Linear Trend	The RewP ($\beta = .20$, $t(85) = 2.59$, $p = .01$) ... [was an] independent significant predictors of depressive symptoms.	$t(85) = 2.59$; $p = .01$	N/A	The RewP ($\beta = -.10$, $t(85) = -2.33$, $p = .02$) [was a] significant independent predictor of antenatal depressive symptoms.
Mulligan et al. (2018b)	It was also hypothesized that increased depression symptoms would be associated with a reduced neural response to reward , greater levels of progesterone, and reduced levels of estradiol.	A repeated measures analysis of covariance (ANCOVA) was conducted on the traditional and PCA-derived ERP responses with menstrual cycle phase (follicular vs. luteal) and trial outcome (gain vs. loss) entered as within-subject factors, and depression score entered as a between-subjects covariate.	3-Way Interaction (Attenuation of Attenuation)	There was a significant interaction between menstrual phase, trial outcome, and depression score, $F(1, 41) = 5.41$, $p = 0.03$, $\eta^2 = 0.12$... [showing] a significant interaction between menstrual phase and depression scores for gain trials but not for loss trials.	$F(1, 41) = 5.41$; $p = 0.03$	N/A	In the traditional ERP data... [there was a] significant three-way interaction between menstrual phase, feedback..., and depression score, $F(1, 41) = 4.56$, $p = 0.04$
Parvaz et al. (2016) ¹	We hypothesize that the RewP amplitude would be specifically associated with the severity of anhedonia, and not that of depression , in individuals with cocaine use disorders (iCUD).	The association between the amplitude of the isolated PCA factor (i.e., TF3SF1) denoting RewP and the severity of anhedonia and depression (assessed via CSSA) were ascertained using the Spearman Rank correlation.	Linear Trend	The RewP amplitude was negatively correlated with anhedonia ($r_s = -0.409$, $p = 0.005$) and depression ($r = -0.287$, $p = 0.05$) scores	$r(44) = -0.287$; $p = 0.05$	N/A	N/A
Peng et al. (2015)	Therefore, we hypothesized that neutral feedback would elicit comparable FRN amplitudes to negative feedback in the depressive group .	A two-way ANOVA analysis was carried out on the FRN amplitude at the electrode FCz with feedback valence (positive, negative & neutral) as an independent variable and with depression (high BDI & low BDI) as a between-subject variable.	3 x 2 Attenuation Trends	Moreover, the interaction between valence and depression also reached significance, $F(2, 72) = 4.02$, $p < .03$, $g^2 = 0.1$.	$F(2, 72) = 4.02$, $p < .03$	High vs Low comparisons not reported	N/A

Ruchsnow et al. (2004)	With regard to the feedback ERN/Ne as a function of the previous trial, we reasoned that depressed patients should have smaller amplitudes possibly reflecting underactivity in a central reward pathway.	The ANOVA for lateral electrodes included the factors group, hemisphere, electrode position, and condition (error/error, error/correct, correct/error, correct/correct); the ANOVA for midline electrodes included the factors group, electrode position, and condition (error/error, error/correct, correct/error, correct/correct).	2 x 4 Attenuated Interaction	In the time window of the feedback ERN/Ne component (200–350ms) we found at both lateral and midline frontal electrodes... a significant Condition by Group interaction (lateral: $F(3,90)=4.89, p < .01$; midline: $F(3,90)=5.70, p < .01$).	$F(3,90)=4.89$; $p < .01$	N/A	A significant Condition by Group interaction (midline: $F(3,90)=5.70, p < .01$).
Ruchsnow et al. (2006)	We reasoned that patients with MDD should have smaller amplitudes in condition EE possibly reflecting underactivity in a central reward pathway (Elliott et al., 1998).	The ANOVA for lateral electrodes included the factors group, hemisphere, electrode position and condition (EE/EC/CE/CC), the ANOVA for midline electrodes included the factors group, electrode position and condition (EE/EC/CE/CC).	2 x 4 Attenuated Interaction	For lateral and midline centro-parietal electrodes we found... a significant condition by group interaction (lateral: $[F(3,54) = 4.14; p < 0.05]$; midline: $[F(3,54) = 3.31; p < 0.05]$).	$F(3,54) = 4.14$; $p < 0.05$	N/A	A significant condition by group interaction (midline: $[F(3,54) = 3.31; p < 0.05]$).
Santesso et al. (2008) ²	Using a probabilistic punishment learning task, we hypothesized that, relative to controls, RD subjects would show increased sensitivity to negative feedback as indexed by (1) stronger response bias against the more frequently punished stimulus; and (2) larger FRNs .	Mixed ANOVAs were used to analyze the FRN with Group and Site (Fz, FCz, Cz) as factors. Follow-up independent t-tests were performed to decompose significant effects.	2-Cell	A main effect of Group was also significant ($F(1,25)=7.50, p<0.015$), due to an overall larger FRN for RD than control subjects (0.54 ± 1.89 vs. $2.69\pm2.14 \mu V$)	$F(1,25)=7.50$; $p<0.015$	N/A	Pearson's correlations indicated that higher BDI-II total scores were related to larger FRN, $r(24) = -0.38, p < 0.05$.
Tucker et al. (2003)	We hypothesized that even when demands for response correction are minimized by the delayed feedback procedure, depressed subjects would show exaggerated MFN in evaluating negative feedback.	We conducted a mixed-design analysis of variance (ANOVA) to examine the statistical reliability of hypothesized feedback response differences in the medial frontal cortex.	3x2 Attenuated Trends	This effect was clarified by a Diagnosis $\times$ Feedback interaction, $F(2, 84) = 3.68, p < .03$, reflecting the differential feedback effect for the two groups	$F(2, 84) = 3.68$; $p < .03$	High vs Low comparisons not reported	There was a significant main effect of diagnosis, $F(1, 42)=8.74, p < .01$, reflecting the greater MFN for subjects with depression for all feedback conditions.

Umemoto and Holroyd (2017)	We predicted that participants who self-report high reward sensitivity would exhibit a relatively large feedback related RewP (Bress and Hajcak, 2013; Cooper et al., 2014; Liu et al., 2014; Parvaz et al., 2016), whereas those high in depression-related personality traits would exhibit a small feedback-related RewP (Proudfit, 2015).	In addition, a multiple linear regression analysis was conducted separately on the amplitude of each ERP component with the personality traits as predictors, using the backward method in which all of the predictors were entered into the model first and then noncontributing predictors were step-wise eliminated (with the SPSS default minimum p-value of 0.1 for retaining each predictor in the model	Linear Trend	The multiple linear regression analyses on feedback-RewP conducted separately for each probability condition indicates that participants... low in depression scores produced a large feedback-RewP, particularly when rewards were highly expected; $\beta = .26, t = 2.1; p = .04$	$t(65) = 2.1; p = .04$	N/A	N/A
Weinberg and Shankman (2017)	We predicted that the magnitude of the RewP would be blunted in remitted melancholic depression relative to both controls and remitted nonmelancholic depression.	History of melancholic depression (0 = controls, 1 = nonmelancholic depression, 2 = melancholic depression), psychiatric medication usage (yes/no), and feedback condition (rewards vs. nonrewards) were fixed factors in a full factorial linear mixed model MANOVA design.	3x2 Attenuated Trends	However, the effect of feedback was significantly qualified by an interaction with melancholic group, $F(2, 137.00) = 5.71, p < .01$.	$F(2, 137.00) = 5.71; p < .01$	High vs Low comparisons not reported	However, as earlier, the effect of feedback was qualified by an interaction with melancholic group, $F(2, 142.00) = 6.68, p < .01$.

Note. For ease of references studies included in the ERN *p*-curve disclosure table are denoted using a ^ in the Reference section. Information on all coded articles is shown in the supplementary material posted on Open Science Framework. ¹Indicates a study with a test statistic that was entered into the primary *p*-curve analyses but was excluded by the software for having a *p* value > .05. ²Indicates a study with a test statistic that was entered into the secondary *p*-curve analyses but was excluded by the software for having a *p* value > .05.

Figure Captions

Figure 1. Flow chart of article and p -value selection for ERN and RewP studies. Note that some studies were included in only the primary or secondary p -curve analyses.

Figure 2. Primary p -curve analysis of the significant results from tests of the primary error-related negativity (ERN) and depression-related hypotheses from 20 studies. The blue line shows that distribution of the p values in this set of studies. The dotted red line shows the expected distribution of p values if there no effect. The dashed green line shows the expected distribution of p values if the effect existed but the set of studies was powered at 33%. This figure was generated using the p curve app v4.06.

Figure 3. Secondary p -curve analysis on alternative significant results of 20 error-related negativity (ERN) studies. One study was removed for having a recomputed p value over .05. The blue line shows that distribution of the p values in this set of studies. The dotted red line shows the expected distribution of p values if there no effect. The dashed green line shows the expected distribution of p values if the effect existed but the set of studies was powered at 33%. This figure was generated using the p curve app v4.06.

Figure 4. The first column shows the impact of dropping the lowest p values on the right skew test of the full p curve (first column, top), the right skew test of the half p curve (first column, middle), and the test of the expected distribution of p values if the effect existed but the set of studies was powered at 33% (first column, bottom). The right column shows the impact of dropping the highest p values on the right skew test of the full p curve (second column, top) and

the test of the expected distribution of p values if the effect existed but the set of studies was powered at 33% (second column, bottom).

Figure 5. Primary p -curve analysis of the significant results from tests of the primary reward positivity (RewP) and depression-related hypotheses from 28 studies. Two studies were excluded for having recomputed p values over .05. Note that for one study in the sample, two p values were entered rather than one, because tests of each simple effect were included. The blue line shows that distribution of the p values in this set of studies. The dotted red line shows the expected distribution of p values if there no effect. The dashed green line shows the expected distribution of p values if the effect existed but the set of studies was powered at 33%. This figure was generated using the p curve app v4.06.

Figure 6. P -curve analysis of the robustness of significant results from tests of the primary reward positivity (RewP) and depression-related hypotheses of 27 studies. Two studies were removed for having recomputed p values over .05. The blue line shows that distribution of the p values in this set of studies. The dotted red line shows the expected distribution of p values if there no effect. The dashed green line shows the expected distribution of p values if the effect existed but the set of studies was powered at 33%. This figure was generated using the p curve app v4.06.

Figure 7. Secondary p -curve analysis on alternative significant results of 20 reward positivity (RewP) studies. Two studies were removed for having recomputed p values over .05. The blue line shows that distribution of the p values in this set of studies. The dotted red line shows the

expected distribution of p values if there no effect. The dashed green line shows the expected distribution of p values if the effect existed but the set of studies was powered at 33%. This figure was generated using the p curve app v4.06.

Figure 8. The first column shows the impact of dropping the lowest p values on the right skew test of the full p curve (first column, top), the right skew test of the half p curve (first column, middle), and the test of the expected distribution of p values if the effect existed but the set of studies was powered at 33% (first column, bottom). The right column shows the impact of dropping the highest p values on the right skew test of the full p curve (second column, top) and the test of the expected distribution of p values if the effect existed but the set of studies was powered at 33% (second column, bottom).

Figure 1

Figure 2

Note: The observed p -curve includes 20 statistically significant ($p < .05$) results, of which 13 are $p < .025$. There were no non-significant results entered.

Figure 3

Note: The observed p -curve includes 19 statistically significant ($p < .05$) results, of which 13 are $p < .025$. There was one additional result entered but excluded from p -curve because it was $p > .05$.

Figure 4

Figure 5

Note: The observed p -curve includes 27 statistically significant ($p < .05$) results, of which 17 are $p < .025$. There were 2 additional results entered but excluded from p -curve because they were $p > .05$.

Figure 6

Note: The observed p -curve includes 25 statistically significant ($p < .05$) results, of which 15 are $p < .025$. There were 2 additional results entered but excluded from p -curve because they were $p > .05$.

Figure 7

Note: The observed p -curve includes 18 statistically significant ($p < .05$) results, of which 11 are $p < .025$. There were 2 additional results entered but excluded from p -curve because they were $p > .05$.

Figure 8