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Review

Reinforcement sensitivity, depression and anxiety: A meta-analysis and meta-analytic structural equation model

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HIGHLIGHTS

- Sensitivity to punishment positively predicts both depression and anxiety.
- Sensitivity to reward discriminates between them, negatively predicting depression.
- This pattern was observed even when directly controlling for comorbidity.
- Depression's effect sizes are uniquely sensitive to clinical state.
- Depression's effect sizes are also moderated by method of clinical assessment.

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ABSTRACT

Reinforcement Sensitivity Theory (RST) posits that individual differences in reward and punishment processing predict differences in cognition, behavior, and psychopathology. We performed a quantitative review of the relationships between reinforcement sensitivity, depression and anxiety, in two separate sets of analyses. First, we reviewed 204 studies that reported either correlations between reinforcement sensitivity and self-reported symptom severity or differences in reinforcement sensitivity between diagnosed and healthy participants, yielding 483 effect sizes. Both depression (Hedges' g = .99) and anxiety (g = 1.21) were found to be high on punishment sensitivity. Reward sensitivity negatively predicted only depressive disorders (g = −.21). More severe clinical states (e.g., acute vs remission) predicted larger effect sizes for depression but not anxiety. Next, we reviewed an additional 39 studies that reported correlations between reinforcement sensitivity and both depression and anxiety, yielding 156 effect sizes. We then performed meta-analytic structural equation modeling to simultaneously estimate all covariances and control for comorbidity. Again we found punishment sensitivity to predict depression (β = .37) and anxiety (β = .35), with reward sensitivity only predicting depression (β = −.07). The transdiagnostic role of punishment sensitivity and the discriminatory role of reward sensitivity support a hierarchical approach to RST and psychopathology.

1. Introduction

Gray's Reinforcement Sensitivity Theory (RST; Gray, 1970, 1987; Gray & McNaughton, 2000) posits that sensitivity to appetitive and aversive stimuli serves as a biological basis of human personality. According to the original theory, the Behavioral Approach System (BAS) governs processes related to appetitive stimuli, whereas the separate, independent Behavioral Inhibition System (BIS) governs responses to aversive stimuli (Corr, 2008). Individuals' varied sensitivities in BAS and BIS functioning then lead to individual differences in reward processing, punishment processing, and personality. Those with a more sensitive BAS tend to show more cognitive styles and behaviors associated with reward promotion and the broad personality dimension of extraversion (Corr & McNaughton, 2008; Depue & Collins, 1999; Gray, 1987). A more sensitive BIS, on the other hand, impacts behaviors and psychological processes related to punishment and neuroticism (Gray, 1970; Smits & Boeck, 2006).

In 2000, RST was revised (Gray & McNaughton, 2000) to include three systems (Corr, 2008). The BAS system remained governing all appetitive processing, while aversive processing was ascribed to the newly named Fight/Flight/Freeze System (FFFS) instead of the original BIS. The BIS in the revised version was theorized to be activated when the goals of the BAS and FFFS came into approach-avoidance conflicts. New measures (e.g., Corr & Cooper, 2016; Jackson, 2009) have been

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developed with the purpose of differentiating the revised BIS from the FFFS (i.e., original BIS). In practice, they both correlate with measures of BIS developed under the original RST and much of the literature on RST, motivation and psychopathology continues to use the original terminology of BAS for appetitive sensitivity and BIS for aversive sensitivity (e.g., Bijttebier, Beck, Claes, & Vandereycken, 2009; Trew, 2011). The current meta-analysis, in reviewing this literature, uses the terminology of the original RST when referring to basic appetitive and aversive processes as well.

When appetitive and aversive sensitivities are dysregulated, they are theorized to be diatheses for subsequent psychopathology. BAS hyposensitivity is indicated by deficits in anticipatory pleasure, consummatory pleasure, and appetitive motivation (Alloy, Olino, Freed, & Nusslock, 2016; Olino, McMakin, & Forbes, 2018; Zald & Treadway, 2017). This anhedonic experience is a hallmark of dysthymia and major depressive disorder (American Psychiatric Association, 2013; Watson et al., 1995). Accordingly, numerous studies have found reduced BAS sensitivity to be associated with depression among clinical (e.g., DelDonno et al., 2015; Greenwood et al., 2013; Zaninotto et al., 2015) and nonclinical samples (e.g., Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007; Jarmolowicz et al., 2014; Peirson & Heuchert, 2001) – though not always (e.g., Carver & Johnson, 2009). Among depressed patients, BAS hyposensitivity was found to predict greater concurrent symptom severity and a worse outcome following treatment (Kasch, Rottenberg, Arnow, & Gotlib, 2002). Anxiety, on the other hand, is theorized to be weakly related to BAS sensitivity, if at all (Gray, 1987; Gray & McNaughton, 2000; McNaughton & Corr, 2004). Rather, anxiety is theorized to primarily occur as a function of aversive sensitivities, conceptualized as BIS hypersensitivity in the original RST (Gray, 1982, 1987). BIS hypersensitivity is associated with increased apprehension in the face of impending negative outcomes and more intense reactivity to punishment, which are experiences salient to anxiety disorders (Carver & White, 1994; Corr & McNaughton, 2008; Hollon, 2019; Leen-Feldner, Zvolensky, Feldner, & Lejuez, 2004). Later investigations, however, have found BIS levels to predict depression to a large degree as well (Eshel & Roiser, 2010; Hundt et al., 2007; Johnson, Turner, & Iwata, 2003; Pekka Jylhä & Isometsä, 2006).

Thus, it appears that the BIS predicts both depression and anxiety while the BAS uniquely predicts depression (Bijttebier et al., 2009; Khan, Jacobson, Gardner, Prescott, & Kendler, 2005). This pattern has been linked to similar hierarchical models of psychopathology such as the tripartite model, where general distress is present across depression and anxiety disorders, but deficits in positive affect are unique to depression (Clark & Watson, 1991; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017; Zinbarg & Yoon, 2008).

The multidisciplinary literature on the relationship between reinforcement sensitivity dysregulation and mood disorders has been summarized in a number of influential theoretical frameworks and models. The *BAS dysregulation model* highlights the unique role that reward sensitivity plays in the development and maintenance of depression and bipolar disorder (Alloy et al., 2016; Stange et al., 2013). The *joint system hypothesis* suggests that internalizing pathology is most closely predicted by a confluence of hyposensitive BAS and hypersensitive BIS (Corr, 2001; Eddington, Majestic, & Silvia, 2012; Hundt et al., 2007). Furthermore, RST has provided a theoretical basis for disorder classification models related to depression and anxiety in particular (Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007; Zinbarg & Yoon, 2008), and mental illness in general (Bijttebier et al., 2009). RST has even served key roles in explaining how individual differences in general may impact the etiology (Kimbrel, 2008; Trew, 2011), severity (e.g., Brown, Chorpita, & Barlow, 1998; Cloninger, Bayon, & Svrakic, 1998) and classification (e.g., Caspi et al., 2014; Clark & Watson, 1991; Eaton et al., 2013; Krueger & Markon, 2006; Watson, 2009) of mental disorders. The National Institute of Mental Health has since included reward and punishment processing among its Research Domain Criteria (RDoC; Insel et al., 2010), further emphasizing the central role that RST plays in basic clinical science.

The current theoretical work, however, is limited by its reliance on narrative reviews, which often under-represent nonsignificant effects, unpublished effects, or effects that were secondary to the study at hand (Easterbrook, Gopalan, Berlin, & Matthews, 1991; Rosenthal & DiMatteo, 2001; Sterne, Gavaghan, & Egger, 2000). This biased source selection may have theoretical implications. Constructs such as positive emotionality (Watson & Naragon-Gainey, 2010) and extraversion (Krueger, Caspi, Moffitt, Silva, & McGee, 1996) have been hypothesized to differentiate depression from anxiety. However, when subjected to the scrutiny of meta-analyses, neither construct conclusively discriminated between these disorders (Khazanov & Ruscio, 2016; Kotov, Gamez, Schmidt, & Watson, 2010). Similarly, the relationship between BAS hyposensitivity and anhedonic depression has been highlighted in narrative reviews of findings (e.g., Alloy et al., 2016; Zinbarg & Yoon, 2008) derived from biological (e.g., DelDonno et al., 2015), behavioral (e.g., Treadway, Bossaller, Shelton, & Zald, 2012), and self-report data (e.g., Kircanski, Mazur, & Gotlib, 2013), with some studies based upon quite sizeable samples (e.g., Johnson et al., 2003). However, it is also possible that the unique relationship between BAS sensitivity and anhedonia may not withstand the scrutiny of meta-analysis, as was the case of positive emotionality and extraversion.

Attempts to discriminate between depression and anxiety are further complicated by their high comorbidity (Watson, 2009). Depression and anxiety are noted risk factors for each other (Jacobson & Newman, 2017), with overlapping symptom criteria (Borsboom & Cramer, 2013), some shared basic mechanisms (Nolen-Hoeksema & Watkins, 2011; Ruscio, Seitchik, Gentes, Jones, & Hallion, 2011), and are especially comorbid among more severe clinical samples (Kessler, Chiu, Demler, & Walters, 2005). To understand each disorder's unique relationship with reinforcement sensitivity, it is necessary to control for their shared variance. Only meta-analytic tools enable that, either by indirectly projecting shared covariance (e.g., Khazanov & Ruscio, 2016) or by using structural equation modeling to directly control for it (M. W.-L. Cheung & Chan, 2005; Jak, 2015). Thus, a systematic quantitative review is needed to adequately estimate the unique relationships between BAS, BIS, depression and anxiety, after controlling for the disorders' high rates of comorbidity.

Furthermore, a meta-analysis of the relationship between reinforcement sensitivity, depression and anxiety may answer the calls for identifying potential moderators to the relationship (see Bijttebier et al., 2009). One moderator of note is clinical state. Indeed, differences have been found between acute depressed patients and those in remission in measures of both reinforcement sensitivity (Pinto-Meza et al., 2006) and temperament (Nery et al., 2009; Takahashi et al., 2013). This was particularly true for punishment sensitivity (Hansenne & Bianchi, 2009). To the best of our knowledge, however, the moderating role of clinical state on otherwise stable traits is largely neglected in the meta-analytic literature (Kotov et al., 2010; cf. Zaninotto et al., 2016), including the literature on reinforcement sensitivity.

In sum, numerous multimodal lines of research have found that dysregulated reinforcement sensitivity plays a critical role in the development and maintenance of depression and anxiety. Summaries of these findings, however, have been limited to narrative reviews. Without a quantitative summary of the literature, the range of observed effect sizes remains unknown, central hypotheses laid out in RST remain untested, and potential moderators remain unexamined. These questions impact the nature of reinforcement sensitivity's relationship with depression and anxiety, and warrant the performance of a meta-analysis.

2. The current studies and hypotheses

The current meta-analyses aimed to quantify the relationships between reinforcement sensitivity, depression and anxiety, and to examine the factors that may moderate these relationships. First, we

examined the broad relationships between reward and punishment sensitivity, and a single pathology factor consisting of both depression and anxiety (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Caspi et al., 2014). In doing so, we aimed to provide, for the first time, an estimation of the true effect size of the relationships between the components of reinforcement sensitivity, depression and anxiety.

We then examined potential moderators for these relationships. The moderator of primary interest was disorder cluster (i.e., anxiety vs depression). By including disorder cluster as a moderator, we could examine whether punishment sensitivity or reward sensitivity could reliably discriminate between anxiety and depression. We examined these relationships in two ways. First, using a large sample of studies, we used subgroup analysis to examine the bivariate relationships between the two components of reinforcement sensitivity and disorder cluster. A significant difference in BAS or BIS levels between disorder clusters would indicate that the reinforcement sensitivity system generally discriminates between depression and anxiety in the current literature. To more directly control comorbidity, we also performed a meta-analytic structural equation model (MASEM; Hunter & Schmidt, 2004) analysis on a subgroup of eligible studies that estimated the unique covariance between all four elements (i.e., BIS, BAS, depression and anxiety). This allowed us, for example, to estimate the relationship between BAS and anxiety, independent of possible confounding relationships between BAS and depression. To maintain the independence of both forms of analysis, we partitioned the studies used in the MASEM away from the other studies. This ultimately led to the two separate meta-analytic studies presented below.

We also examined the role of diagnostic method plays moderating the observed effects. Indeed, as has been seen elsewhere (e.g., Aldao et al., 2010), effects that are taken from self-report measures of psychopathology (e.g., Beck Depression Inventory; Beck, Wartenberg, Mendelson, Mock, & Erbaugh, 1961) may systematically differ from those taken from clinical diagnosis. Such differences are confounded by the fact that effects taken from self-report measures are usually derived from correlational data, while those taken from clinically diagnosed groups are usually derived from standardized mean comparisons with healthy control groups. This difference in analytic method may inflate the effect sizes of comparison groups due to the selection of statistically extreme participants (e.g., high in depression vs healthy; Kircanski et al., 2013). Thus, differences observed between self-report effects and diagnosed-healthy comparison effects could potentially be confounded by differences in clinical severity. To address this concern, we also examined whether clinical state (e.g., acute episode vs remission) would moderate effects among diagnosed-healthy comparisons. In doing so, we were able to quantify the moderating role of current clinical severity, within a diagnosed population. Significant moderators in this case would indicate meaningful differences in effect sizes, based on the clinical status of the participants (i.e., self-report vs diagnosed; acute vs remission). We examined the role of clinical status as a moderator for reinforcement sensitivity's relationships with psychopathology both across disorder clusters as well as within depression and anxiety.

2.1. Operationalization of reinforcement sensitivity and pathology

Reward and punishment sensitivities were operationalized in the form of self-report measures derived directly from RST (e.g., BIS/BAS; Carver & White, 1994) or from measures with subscales generated to measure particular RST systems (e.g., Tridimensional Personality Questionnaire – Novelty Seeking and Harm Avoidance; Cloninger, 1987; Klein, Kotov, & Bufferd, 2011; for a fuller review, see Torrubia, Avila, & Caseras, 2008). Psychopathology was operationalized either in the form of self-report measures of depression or anxiety (e.g., Beck Depression Inventory; Beck et al., 1961), or in the form of a diagnosed anxiety, depressive, or mixed disorder.

2.2. Hypotheses

In Study 1, we first expected to find reinforcement sensitivity to predict psychopathology (Hypothesis 1). Specifically, based on the close relationship between BAS hyposensitivity and affective symptoms such as anhedonia (Zald & Treadway, 2017), we expected a negative relationship between BAS sensitivity and psychopathology (Hypothesis 1a). On the other hand, due to the literature on punishment hypersensitivity (e.g., Smits & Boeck, 2006), we expected to find a positive relationship between BIS sensitivity and psychopathology (Hypothesis 1b).

Second, we expected the role of reinforcement sensitivity to change as a function of disorder cluster (Hypothesis 2). BAS was predicted to play a discriminatory role (Alloy et al., 2016), in the form of a negative relationship with depressive disorders (Hypothesis 2a) which would be significantly larger than anxiety (Hypothesis 2b). We did not expect a significant relationship between BAS and anxiety. On the other hand, we expected the BIS would impact both depression and anxiety (Smits & Boeck, 2006; Zinbarg & Yoon, 2008). This would take the form of a positive relationship between the BIS and depression (Hypothesis 2c), as well as anxiety (Hypothesis 2d). We did not expect a difference between the two disorder clusters. Finally, we expected to see this pattern both when comparing between bivariate relationships using subgroup analysis (see Study 1), as well as when simultaneously estimating all relationships (e.g., between depression and anxiety) using MASEM (see Study 2).

Third, we expected clinical status to impact the observed relationships (Hypothesis 3). This would be reflected in the form of larger effect sizes for populations with more severe clinical statuses, both with regards to BAS (Hypothesis 3a) as well as BIS (Hypothesis 3b). Specifically, we expected that effects derived from self-report correlations would be smaller than those derived from clinical-healthy comparisons, which select for participants high and low in symptom severity. In keeping with this pattern, we also expected that clinical participants who currently suffer from more severe pathology would differ more from healthy controls in reinforcement sensitivity than those who are currently in remission. We examined this moderator across disorders, as well as within the depression and anxiety clusters.

3. Method

3.1. Literature search

Studies were identified through a set of 30 searches in PsycInfo and PubMed between 1991 and October 2017. Searches entailed permutations of (a) keywords related to reinforcement sensitivity theory and its scales - *RST*, "*Reinforcement Sensitivity*", "*Reward Sensitivity*", "*Punishment Sensitivity*", "*Reward Dependence*", *BIS, BAS,* "*Behavioral Activation System*", "*Behavioral Approach*", "*Behavioral Inhibition*", *SPSRQ,* "*Sensitivity to Reward Questionnaire*", "*Brief Sensitivity to Punishment*", "*Appetitive Motivation Scale*", *RSQ, RST-PQ,* "*Gray-Wilson Personality Questionnaire*", "*General Reward and Punishment Expectancy Scale*", "*Tridimensional Personality Questionnaire*", *TPQ,* "*Temperament and Character Inventory*", *TCI* and (b) depression, anxiety and their measures - *depress*, MDD, dysth*, "mood disorder", "affective disorder", anxiety, GAD, fear, panic, STAI, "post*traumatic stress disorder", PTSD, "social phobia", "social anxiety", agoraphobia, phob*, "obsessive-compulsive disorder", OCD*. The asterisk in search terms allowed for multiple word endings (e.g., *phob** allowed for both *phobia* and *phobic*). An invitation for published and unpublished manuscripts was also publicized on ResearchGate. The reference sections of narrative literature reviews on the topic were reviewed for additional potential articles (Alloy & Abramson, 2010; Bienvenu et al., 2001; Bijttebier et al., 2009; Khazanov & Ruscio, 2016; Kimbrel, 2008; Klein et al., 2011; Kotov et al., 2010; Nusslock & Alloy, 2017; Trew, 2011; Urosević, Abramson, Harmon-Jones, & Alloy, 2008; Zald & Treadway, 2017). All searches

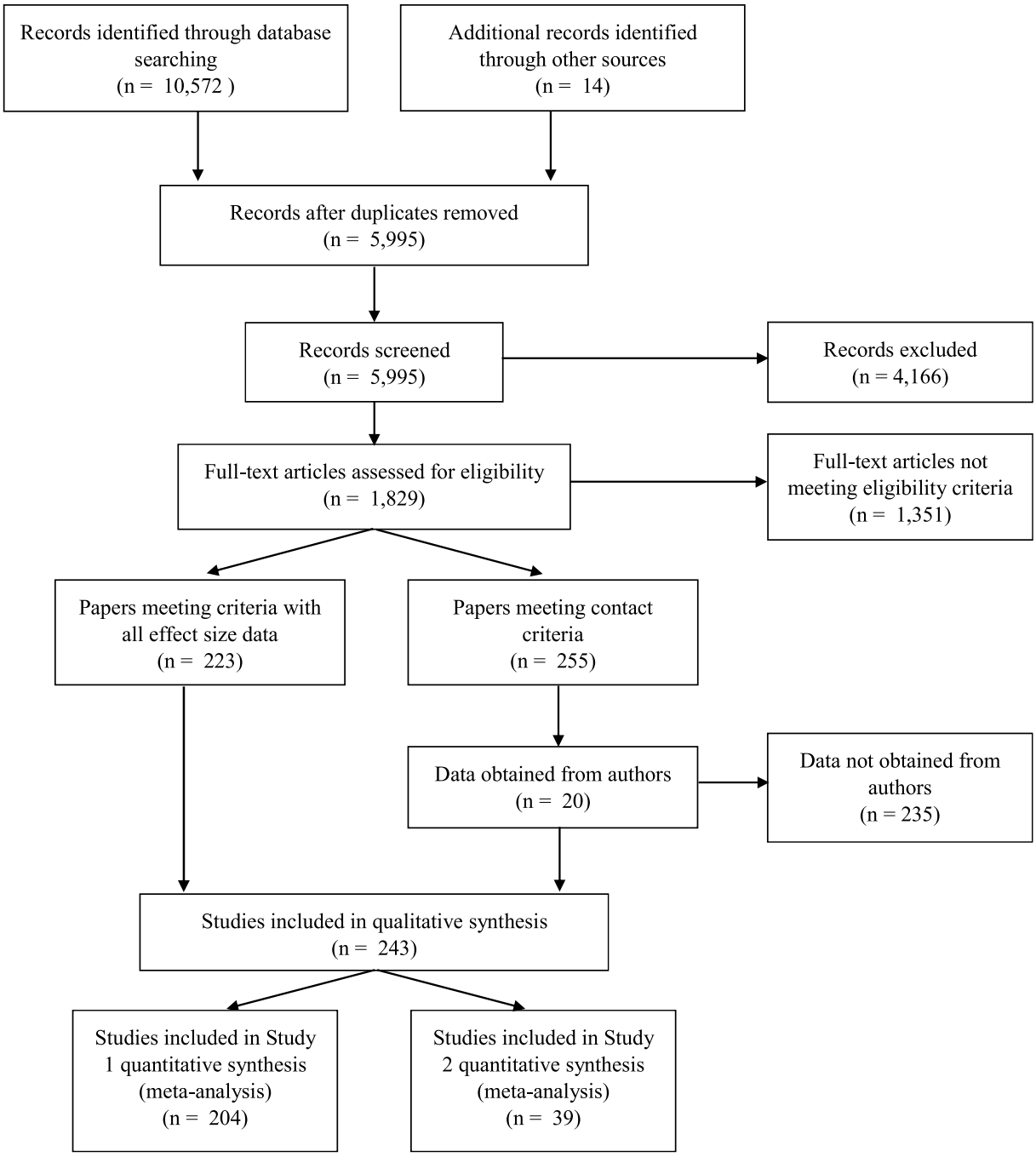

Fig. 1. Derivation of analysis samples.

were performed between May and June, 2017. A search protocol may be found in the Supplemental Materials at https://osf.io/n6gv4/. This process yielded 10,572 references. We then uploaded the references to Endnote X8.2 and scanned for duplicates. Following the elimination of duplicates, 5995 references were approved for abstract screening (see Fig. 1 for summary).

3.2. Inclusion/exclusion criteria

A study was evaluated according to its ability to furnish a unique effect size on the relationship between depressive/anxious symptomatology and sensitivity to reward and punishment. Specifically, a manuscript had to report one of three effects. The first possible effect was a correlation between a validated self-report symptom measure (e.g., the Beck Depression Inventory; Beck et al., 1961) and a validated measure of reinforcement sensitivity (e.g., the BIS/BAS scale; Carver & White, 1994). The second effect consisted of standard mean differences in reinforcement sensitivity between two groups derived from a symmetrical division (e.g., median split) of participants based on a self-report symptom measure (e.g., Aarts & Pourtois, 2010). Third, effects were included if they compared reinforcement sensitivity levels between participants diagnosed with a depressive or anxiety disorder, and healthy controls. Only manuscripts written in English were included, though the research itself may have been performed in any language.

A number of exclusion criteria were applied as well. First, in order to aggregate comparable effect sizes, only trait self-report data was considered eligible for inclusion. Thus, clinical reinforcement sensitivity studies that did not include self-report data (e.g., only behavioral

data; Kunisato et al., 2012) or only state data (e.g., State-Trait Anxiety Inventory-State; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) were rejected. Second, standard mean differences among non-clinical participants were only included if they were symmetrical. Studies with non-symmetrical division of participants (e.g., high BAS vs moderate BAS; Stange et al., 2013) were not included. Third, in order to minimize the probability of confounding effects and over-sampling of certain disorders, studies were rejected if the diagnosed group was selected on the basis of comorbidity with a disorder beyond the purview of the meta-analysis (e.g., eating disorders; Reba-Harrelson et al., 2008) or on the basis of any other clinically relevant criterion foreign to the meta-analysis (e.g., alcohol use; Sellman & Joyce, 1996). Studies were included, however, if a correlation between RST and self-report symptom measures could be obtained from within the non-clinical controls (e.g., Senín-Calderón, Perona-Garcelán, Fuentes-Márquez, & Rodríguez-Testal, 2017). Fourth, in the event that participants diagnosed with a depressive or anxiety disorder were included in the sample, a study would only be included if it included a healthy control group as well, in order to calculate a standard mean difference (cf. Kotov et al., 2010). Fifth, experiments and treatment studies were included only if data was collected before any intervention took place. The first author (BAK) sorted all studies in both the abstract review and full text review stages. The second author (KM) independently sorted a subset consisting of ten percent of the studies, randomly selected. Interrater reliability was high ($rs > .86$) for all stages of the sorting process. Two hundred and fifty-five manuscripts indicated the collection of data appropriate for the meta-analysis, but did not report all of what was necessary for inclusion. Authors of these manuscripts were contacted for further information between February and April 2018, with 20 agreeing to send the unpublished data. Altogether, 244 manuscripts were included in the meta-analysis.

3.3. Coding of studies

In the case of a single group, we recorded correlations between BAS/BIS and depression/anxiety measures. In the case of a dichotomous comparison between two groups (e.g., diagnosed depression and healthy control group), we recorded the data necessary for the calculation of standard mean differences (e.g., *M* and *SD* of BIS). A number of moderators were coded as well. We recorded demographic variables known to correlate with reinforcement sensitivity: average age and proportion of female participants (Gray, Hanna, Gillen, & Rushe, 2016; Torrubia et al., 2008). We also recorded sample size and publication status as meta-data for each article (i.e., published = 1, unpublished = 0). We also recorded the measures used to assess reinforcement sensitivity, clinical severity and the diagnosis of participants in a clinical group.

The clinical status of the sample was recorded as well. This was done in two steps. First, the sample was coded based on whether effect sizes were derived from comparisons between clinical and healthy groups or from correlations with self-report measures (i.e., diagnostic = 1, self-report = 0). All correlational data was classified as *self-report*, along with two (i.e., Aarts & Pourtois, 2010; Duley, Hillman, Coombes, & Janelle, 2007) out of 118 standard mean differences. These two studies utilized self-report data to perform a between-group analysis based on participants' reinforcement sensitivities. Studies with diagnosed participants were then coded based on their clinical status (Zaninotto et al., 2016). Participants who were currently experiencing a depressive episode or were diagnosed with a current, clinical-level, untreated anxiety disorder were classified as *Episode/Acute*. Participants who reported a lifetime incidence of a depressive or anxiety disorder, but were below the threshold for a current depressive episode or anxiety diagnosis were classified as *Euthymic/Remission*. In addition to these two groups, there was a high incidence of studies where participants were recruited as outpatients in ambulatory clinics, but their current clinical state was not specified. Because such populations often represent participants at both clinical and sub-clinical levels of symptom severity, these participants were classified in a separate group, *Outpatient*.

The first author (BAK) coded all 244 studies. The second author (KM) independently coded a subset consisting of 102 studies (41.8%) randomly selected from the pool of coded studies. Interrater reliability was high ($r = .96$ or above) for all variables. Disagreements in ratings were discussed until a consensus was reached.

3.3.1. Coding decisions

A number of decisions needed to be made during the coding process when studies contained multiple measures. First, when multiple clinical or reinforcement sensitivity measures were included in the same sample, steps were taken to include all collected data while nevertheless maintaining the assumption of independence of all samples' effect sizes. Specifically, in studies with correlational data, the correlations were averaged together (Aldao et al., 2010). In studies consisting of group differences, the distributions of multiple measures were merged in order to generate two groups for comparison (i.e., an aggregated clinical group and an aggregated healthy control) using the standard analysis-of-variance approach to partitioning variance (Borenstein, Hedges, Higgins, & Rothstein, 2009; Kirk, 1995). Multiple means were summed together and multiple standard deviations were merged by taking the square root of the pooled variances (Kotov et al., 2010). In the case of multiple clinical groups with a single healthy control group ($k = 23$), separate standard mean differences were calculated between each clinical group and the control group. The sample size of the control group was then evenly divided by the number of comparisons for which it was used (Borenstein et al., 2009; cf. Kotov et al., 2010). Finally, owing to the small portion of studies with longitudinal data ($k = 11$), only the initial cross-sectional effect was recorded.

3.4. Division of studies

The two present meta-analytic studies utilized separate methodologies with different inclusion requirements. In Study 1, we aimed to quantify the bivariate relationships between reward/punishment sensitivity, depression and anxiety, and to identify moderators for these effects. To do so, we performed a series of meta-analyses and moderator analyses, which required studies that reported relationships between at least one self-report symptom measure (i.e., depression and/or anxiety) and at least one reinforcement sensitivity measure (i.e., BAS and/or BIS). This intentionally broad criterion allowed for a comprehensive summary of the current literature and a well-powered moderator analysis. Study 2 examined the extent to which relationships observed in Study 1 may have been confounded by the high covariance between depression and anxiety. To address this question, we utilized the meta-analytic structural equation model (MASEM; Hunter & Schmidt, 2004) method.

Estimating a MASEM requires a series of decisions that impact which studies are to be included. One approach to study selection is pairwise inclusion, wherein every correlation available is included in a meta-analysis (e.g., BAS-depression), even in the absence of other correlations (e.g., BAS-anxiety). This method includes a wider range of data but is also accompanied by complications related to questions of ideal sample size (e.g., average sample size vs sum of all samples, etc.; Cheung & Chan, 2005) as well as by the risk of generating a non-positive definite matrix that is ineligible for the maximum likelihood estimator used in structural equation modeling (Kline, 2015; Naragon-Gainey, McMahon, & Chacko, 2017; Wothke, 1993). For these reasons, we used a more conservative listwise inclusion of studies (M. W.-L. Cheung & Chan, 2005; Hom, Caranikas-Walker, Prussia, & Griffeth, 1992), wherein studies were only included in this MASEM if they reported correlations between all four elements in the model (i.e., BAS, BIS, depression and anxiety).

To ensure the independence of Study 1 and Study 2, effects included in Study 2 were not included in Study 1. Thus, following coding, 299 samples were partitioned into two groups. The majority of articles were included in Study 1 (k = 204). Those that reported correlations between all four elements were included in Study 2 (k = 39). Databases and analyses for Studies 1 and 2 may be found in the Supplemental Materials (https://osf.io/n6gv4/). No differences were found with regards to the meta-data of the self-report effects in Study 1 and the effects in Study 2. Furthermore, sensitivity analyses confirmed that no findings in Study 1 were impacted by the removal of studies for Study 2 (see Supplemental Materials; S1–S2).

4. Study 1

4.1. Data analytic plan

All effect sizes were transformed to standard mean differences using standard formulae (H. Cooper, Hedges, & Valentine, 2009). To correct for small sample sizes, Hedges' g was used instead of Cohen's d (Hedges & Olkin, 1984), with non-clinical participants coded as *0* and clinical participants coded as *1*. Thus, positive effect sizes indicated elevated reinforcement sensitivity among those with higher clinical measures. We considered an absolute value of Hedges' g under |.10| to be trivial, between |.10| and |0.49| to be a small effect size, between |0.50| and |0.79| to be medium, and greater than |.80| to be large (Cohen, 1988). In keeping with other meta-analyses that assess unified constructs via self-report measures (e.g., Kotov et al., 2010; Naragon-Gainey et al., 2017), multiple measures of the same construct were included under the same analysis. Separate meta-analyses for each measure are reported in the supplemental materials (S3-S4).

First, to estimate the relationships between psychopathology and reinforcement sensitivity, we performed two separate meta-analyses – one for reward sensitivity (Hypothesis 1a) and one for punishment sensitivity (Hypothesis 1b). These meta-analyses followed procedures laid out by Borenstein et al. (2009). Due to the assumption of true differences between studies and an interest in generalizing beyond the current dataset, we used a random-effects model (Schmidt, Oh, & Hayes, 2009). The summary effect size was calculated as a weighted average, using the inverse of sample size as weights. Standard errors were calculated as a function of sampling error and between-study variance. Analyses were performed using R version 3.6.2 (R Core Team, 2017). Effect sizes were calculated using the package 'compute.es' version 0.2-4 (Del Re, 2013); the meta analysis was performed using 'meta' version 4.10-0 (Schwarzer, 2007) and 'robumeta' version 2.0 (Fisher, Tipton, & Zhipeng, 2017; for a full review of available packages, see Polanin, Hennessy, & Tanner-Smith, 2017).

Next, to examine the role of disorder cluster (i.e., depression vs anxiety) as a moderator, we performed a mixed-effects subgroup analysis for the BAS (Hypotheses 2a and 2b) and BIS (Hypothesis 2c and 2d) meta-analyses using disorder as a grouping variable (Borenstein et al., 2009). Four studies involved data taken from patients with mixed depression/anxiety (Battaglia, Przybeck, Bellodi, & Cloninger, 1996; Brown, Svrakic, Przybeck, & Cloninger, 1992; Minaya & Fresán, 2009; Minelli, Pedrini, Magni, & Rotondo, 2009) and were ineligible for this stage of analysis. A series of mixed-effect subgroup analyses were then performed to examine the moderating roles of participant clinical status (Hypothesis 3) and publication status. Then, to examine the role of continuous variables as moderators, and to control for possible covariance between moderators, a two-step metaregression was undertaken (Gonzalez-Mulé & Aguinis, 2018; Piotrowska, Stride, Croft, & Rowe, 2015). First, we performed a series of univariate regressions on all of the above categorical moderators, as well as on the continuous moderators of sample size, age and percent of women included in the sample. Categorical variables were dummy-coded into two levels. Then, all significant and non-trivial moderators from the univariate regressions (i.e., $beta > |.10|$, $p < .05$) were combined into a multivariate regression. Owing to the intercorrelation between moderators, a robust variance estimator was used (Hedges, Tipton, & Johnson, 2010).

Two sets of analysis were not included in the main analysis due to complications arising from the dataset. First, we intended to examine whether individual disorders (e.g., social phobia vs generalized anxiety disorder) may have explained differences between depression and anxiety. However, the small sample sizes of each disorder (range k = 0–14) other than major depressive disorder (k = 63) did not supply adequate statistical power to perform subgroup analysis (Borenstein et al., 2009). Second, we intended to analyze whether there would be significant differences based on reinforcement sensitivity measure. However, many studies (e.g., Gomez & Gomez, 2005; Harnett, Loxton, & Jackson, 2013) used more than one measure to assess reinforcement sensitivity. Owing to the dependency between measures' participant samples, subgroup analysis was inappropriate (Borenstein et al., 2009; Cooper et al., 2009). Thus, we report effect sizes grouped based on measure and disorder in the supplemental materials (S5), but do not test for differences.

Finally, small study effects (i.e., publication bias; Bakker, van Dijk, & Wicherts, 2012) were considered. Most effect sizes were extracted from large tables with numerous findings, and were often tangential to the studies from which they came. Their statistical significance tended to be less required for publication, and we therefore expected bias to be minimal (e.g., Aldao et al., 2010). Nevertheless, bias was examined in two ways. First, the Egger's test was performed (Egger, Smith, Schneider, & Minder, 1997; Sterne et al., 2000). The Egger's test is a regression of the effect size on the standard error, with weights provided by inverse variance, and provides a statistical test parallel to a "funnel plot" (i.e., a figure used to visualize bias based on sample size). To quantify the possible impact of effect size asymmetry, we then performed Duval and Tweedie's (2000) "trim-and-fill" procedure. First, studies that were missing because of publication bias were estimated and imputed into the database. Then a new meta-analysis was performed with an estimate of a possible alternative effect size. Both the Egger's test and the "trim-and-fill" procedure were performed on the main effects of BAS and BIS.

4.2. Results

4.2.1. Description of studies

Four hundred and eighty-three effect sizes were calculated, based on 260 distinct samples drawn from 204 articles that were published between 1991 and 2017 (see Table 1). The majority of samples (k = 223) provided effect size data for both BAS as well as BIS. Five samples only provided effect size data for BAS while 32 samples provided for only BIS. The total sample size (N) for this study was 79,657, with samples ranging from 18 to 4778 participants (M = 306.37, SD = 562.00). Participants comprised a wide mean age range (M = 30.45, SD = 11.69, range = 11.43 - 80.40). Two hundred and thirty-seven samples included in this study provided all the information necessary for calculating effect sizes while 23 samples required access to unpublished data.

4.2.2. Strength of link between reinforcement sensitivity and pathology

We predicted in Hypothesis 1 that BAS would have a negative relationship across disorders (Hypothesis 1a), and BIS would have a positive one (Hypothesis 1b). Analyses are summarized for BAS at the top of Table 2a, and for BIS at the top of Table 2b. Indeed, a small, negative relationship was observed between BAS and psychopathology across disorders, g = −.16, 95% CI [−.21; −.11]. A homogeneity of variance test found a large portion of real variance in the literature, Q (227) = 1908.70, $p < .0001$; tau^2 = 0.11; I^2 = 88.1% [86.8%; 89.3%]. This large heterogeneity was also reflected in a wide prediction interval of observed effect sizes, *95% PI* [−.82; .51]. BIS, on the other hand, had a large, positive relationship with pathology, g = 1.10, 95% CI [1.02; 1.19]. There was much heterogeneity in this sample as well, Q

Table 1
Studies on the bivariate relationships between reinforcement sensitivity, depression and anxiety (Study 1).

ID_Text	N	PercWom	Age	Disorder cluster	Clinical status	RST scales	Pub status	Data type	*g* BAS	*vg* BAS	*g* BIS	*vg* BIS
Aarts and Pourtois (2010)	32	.88	18.81	Anxiety	Healthy	BIS/BAS	Y	*d*	.13	.12	.84	.13
Abbate-Daga, Buzzichelli, Marzola, Amianto, and Fassino (2014)	59	1.00	25.08	Depression	Healthy	TCI	N	*r*	.23	.07	1.13	.08
Alfimova, Korovaitseva, Lezheiko, and Golimbet (2014)	266	.59	30.80	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	1.00	.02
Alonso et al. (2008)	76	.37	32.30	Anxiety	Outpatients	TCI	Y	*d*	−.85	.06	1.71	.07
	44	.41	30.70	Anxiety	Episode/Acute	TCI	Y	*d*	−.70	.09	2.52	.16
Ammerman, Kleiman, Jenkins, Berman, and McCloskey (2017)	1912	.63	20.89	Depression	Healthy	SPSRQ	Y	*r*	.39	.00	.75	.00
Applegate, El-Deredy, and Bentall (2009)	516	.66	21.75	Depression	Healthy	BIS/BAS	Y	*r*	−.45	.01	.41	.01
Avila and Parcet (2001)	45	1.00	19.02	Anxiety	Healthy	SPSRQ	Y	*r*	−.06	.09	1.08	.10
Baggio et al. (2015)	4778	.00	20.00	Depression	Healthy	BIS/BAS	N	*r*	.05	.00	−.43	.00
Bajraktarov, Gudeva-Nikovska, Manuseva, and Arsova (2017)	40	NA	NA	Depression	Episode/Acute	TCI	Y	*d*	−1.01	.11	1.96	.14
Balsamo (2013)	230	.90	20.90	Depression	Healthy	TCI-R	Y	*r*	−.19	.02	−.32	.02
Battaglia et al. (1996)	68	.63	36.10	Other	Remission	TPQ	Y	*d*	.17	.07	1.08	.08
	71	.63	36.10	Anxiety	Remission	TPQ	Y	*d*	.44	.07	1.21	.08
Battaglia, Bertella, Bajo, Politi, and Bellodi (1998)	63	1.00	34.60	Anxiety	Remission	TPQ	Y	*d*	.08	.07	1.08	.08
Bensaeed, Ghanbari Jolfaei, Jomehri, and Moradi (2014)	167	.54	33.80	Depression	Remission	TCI	Y	*d*	−.57	.03	.24	.03
Bergdahl and Bergdahl (2003)	74	.84	NA	Anxiety	Episode/Acute	TCI	Y	*d*	.87	.06	−.19	.05
Berger and Anaki (2014)	314	.54	33.70	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	−.77	.01
Bey et al. (2017)	326	.60	33.60	Anxiety	Episode/Acute	TCI-HA	Y	*d*	NA	NA	4.28	.04
Bodas, Siman-Tov, Kreitler, and Peleg (2017)	385	.52	40.60	Anxiety	Healthy	BIS/BAS	Y	*r*	.04	.01	.89	.01
Booth and Hasking (2009)	454	.80	21.40	Anxiety	Healthy	BIS/BAS	Y	*r*	−.35	.01	.64	.01
Borgomaneri, Vitale, and Avenanti (2017)	26	.42	23.20	Anxiety	Healthy	BIS/BAS	N	*r*	.28	.15	.39	.15
Brailean, Koster, Hoorelbeke, and De Raedt (2014)	85	NA	NA	Depression	Episode/Acute	BIS/BAS	Y	*d*	−.50	.05	.76	.05
Brown et al. (1992)	1060	.51	44.90	Other	Outpatients	TPQ	Y	*d*	.48	.03	1.80	.03
Buchman et al. (2014)	40	NA	80.40	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	.81	.10
Carver and Johnson (2009); Study 1	235	.57	19.50	Depression	Healthy	BIS/BAS	Y	*r*	.06	.02	.41	.02
Carver and Johnson (2009); Study 2	394	.67	19.50	Depression	Healthy	BIS/BAS	Y	*r*	.11	.01	.45	.01
Caseras, Ávila, and Torrubia (2003)	538	.78	19.94	Anxiety	Healthy	TPQ; SPSRQ; BIS/BAS; GRAPES; MS-BIS	Y	*r*	−.27	.01	1.34	.01
Celikel et al. (2009)	132	.77	31.39	Depression	Outpatients	TCI	Y	*d*	−.67	.03	1.11	.04
Chang et al. (2013)	932	.49	40.26	Depression	Remission	TPQ	Y	*d*	−.10	.00	.05	.00
Chang et al. (2013a)	250	.58	37.66	Anxiety	Outpatients	TPQ	Y	*d*	−.17	.03	.78	.03
Chang et al. (2013b)	224	.41	NA	Anxiety	Episode/Acute	TPQ	Y	*d*	.29	.04	.56	.04
Chatterjee, Sunitha, Velayudhan, and Khanna (1997)	40	.00	27.10	Anxiety	Episode/Acute	TCI	Y	*d*	−1.65	.13	4.15	.31
Chen, Lu, and Kitamura (2011)	469	.50	41.70	Depression	Healthy	TCI	Y	*r*	.05	.01	−.61	.01
Cheung and Todd-Oldehaver (2006)	74	.65	74.80	Depression	Episode/Acute	TCI-HA	Y	*d*	NA	NA	.67	.06
Claes, Bijttebier, Mitchell, de Zwaan, and Mueller (2011)	211	1.00	22.56	Depression	Healthy	BIS/BAS	N	*r*	.37	.02	.48	.02
Cloninger et al. (1998)	804	.57	46.00	Depression	Healthy	TCI	Y	*r*	−.12	.00	1.12	.01
Contractor, Elhai, Ractliffe, and Forbes (2013)	308	.64	42.51	Anxiety	Healthy	SPSRQ	Y	*r*	.26	.01	.69	.01
Cooper and Gomez (2008)	327	.66	21.86	Anxiety	Healthy	SPSRQ-S	Y	*r*	−.02	.01	2.07	.02
Cooper, Duke, Pickering, and Smillie (2014)	38	.47	24.39	Depression	Healthy	BIS/BAS	Y	*r*	.05	.10	.04	.10
Corr and Cooper (2016)	362	.76	23.34	Anxiety	Healthy	RST-PQ	Y	*r*	.04	.01	.91	.01
Cowley, Roy-Byrne, Greenblatt, and Hommer (1993)	29	NA	30.42	Anxiety	Episode/Acute	TPQ	Y	*d*	−.62	.15	1.34	.17
	22	NA	32.53	Anxiety	Episode/Acute	TPQ	Y	*d*	−.33	.17	1.52	.22
Cremers et al. (2015)	40	.45	28.40	Anxiety	Episode/Acute	BIS/BAS-BIS, RR	Y	*d*	−.38	.10	1.59	.13
Cruz-Fuentes, Blas, Gonzalez, Camarena, and Nicolini (2004)	108	.57	34.00	Anxiety	Episode/Acute	TCI	Y	*d*	−.61	.04	1.36	.05
Dalbudak et al. (2013)	319	.73	21.29	Anxiety	Episode/Acute	TCI	Y	*d*	−.14	.02	.52	.02
Davenport, Houston, and Griffiths (2012)	134	1.00	22.00	Anxiety	Healthy	SPSRQ-SR	Y	*r*	.65	.03	NA	NA
de la Torre-Luque, Fiol-Veny, Balle, and Bornas (2016)	50	.62	13.00	Anxiety	Episode/Acute	SPSRQ-J	Y	*d*	NA	NA	2.40	.14
DelDonno et al. (2015); Study 1	54	.76	28.98	Depression	Episode/Acute	BIS/BAS-BIS,RR,D	Y	*d*	−1.34	.09	1.91	.11
DelDonno et al. (2015); Study 2	60	.63	21.25	Depression	Remission	BIS/BAS-BIS,RR,D	Y	*d*	−.10	.07	.13	.07
De Pascalis, Cozzuto, and Russo (2013)	51	1.00	24.60	Anxiety	Healthy	BIS/BAS	Y	*r*	.28	.08	1.16	.09
Dennis (2007)	36	.72	21.42	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	1.81	.15
Derntl et al. (2011)	30	.60	33.50	Depression	Episode/Acute	BIS/BAS- BAS	Y	*d*	−2.05	.20	3.68	.35
Díaz and Pickering (1993)	89	.00	27.60	Anxiety	Healthy	SPSRQ-SP	Y	*r*	NA	NA	1.61	.06
	82	1.00	27.60	Anxiety	Healthy	SPSRQ-SP	Y	*r*	NA	NA	1.00	.05
Dinovo and Vasey (2011)	477	.50	19.20	Depression	Healthy	BIS/BAS	Y	*r*	.56	.01	.49	.01
Dodd, Mansell, Bentall, and Tai (2011)	175	.88	19.75	Depression	Healthy	BIS/BAS	Y	*r*	.11	.02	.63	.02

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Table 1 (*continued*)

ID_Text	N	PercWom	Age	Disorder cluster	Clinical status	RST scales	Pub status	Data type	*g* BAS	*vg* BAS	*g* BIS	*vg* BIS
Dufey, Fernández, and Mourgues (2011)	233	1.00	20.08	Anxiety	Healthy	SPSRQ	Y	*r*	.16	.02	1.51	.02
	201	.00	20.08	Anxiety	Healthy	SPSRQ	Y	*r*	.51	.02	1.67	.03
Duley et al. (2007)	38	.34	21.35	Anxiety	Healthy	BIS/BAS	Y	*d*	−.30	.11	2.47	.19
Ekinci, Albayrak, and Ekinci (2012)	160	.56	34.20	Depression	Remission	TCI	Y	*d*	−.12	.02	1.50	.03
Elovainio et al. (2004)	2149	.59	31.40	Depression	Healthy	TCI	Y	*r*	−.02	.00	1.12	.00
Ettelt et al. (2008)	150	.67	37.70	Anxiety	Episode/Acute	TPQ-HA	Y	*d*	NA	NA	1.24	.03
Farmer and Seeley (2009)	591	.57	51.00	Depression	Healthy	TCI	Y	*r*	−.05	.01	.80	.01
Gazioglu et al. (2014)	73	.56	35.10	Depression	Healthy	TCI	Y	*r*	NA	NA	1.05	.06
Gil (2005)	180	.58	23.40	Anxiety	Episode/Acute	TPQ	Y	*d*	−.32	.04	.31	.04
Gillath, Giesbrecht, and Shaver (2009); Study 1	83	.77	19.00	Anxiety	Healthy	BIS/BAS	N	*r*	−.12	.05	.59	.05
Gillath et al. (2009); Study 2	101	.66	20.00	Anxiety	Healthy	BIS/BAS	N	*r*	−.25	.04	1.38	.05
Goekoop et al. (2011)	164	NA	68.00	Depression	Episode/Acute	TCI	Y	*d*	−.24	.02	1.50	.03
	127	NA	68.00	Depression	Remission	TCI	Y	*d*	.11	.04	.86	.04
Gökdağ and Arkar (2016)	255	NA	NA	Depression	Episode/Acute	TCI	Y	*d*	.01	.02	1.37	.02
Gomez and Gomez (2005)	358	.55	24.06	Anxiety	Healthy	BIS/BAS; GRAPES	Y	*r*	−.63	.01	1.62	.01
Greenwood et al. (2013)	135	.62	45.00	Depression	Remission	TCI	N	*d*	NA	NA	1.02	.07
Gudiño (2013)	148	.56	11.43	Anxiety	Healthy	BIS/BAS	Y	*r*	.55	.03	.71	.03
Hagenaars (2016)	1154	.63	50.48	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	.82	.00
Halvorsen et al. (2009)	105	.45	47.40	Depression	Episode/Acute	TCI	Y	*d*	−.56	.04	2.08	.06
	138	.48	46.91	Depression	Remission	TCI	Y	*d*	−.06	.03	.97	.04
Han et al. (2006)	277	.00	17.36	Anxiety	Healthy	TCI	Y	*r*	−.10	.01	2.49	.03
Hannan and Orcutt (2013)	282	.63	19.68	Anxiety	Healthy	J5	Y	*r*	−.22	.01	.25	.01
Hansenne and Ansseau (2001)	128	.50	37.79	Depression	Episode/Acute	TCI	Y	*d*	−.40	.03	2.08	.05
Hansenne and Bianchi (2009)	108	.59	44.50	Depression	Episode/Acute	TCI-R	Y	*d*	−.91	.04	1.94	.05
	40	.75	47.50	Depression	Remission	TCI-R	Y	*d*	−.39	.10	.97	.11
Harmon-Jones and Allen (1997)	37	1.00	18.50	Anxiety	Healthy	BIS/BAS	Y	*r*	−1.01	.12	.66	.11
Hasler, Allen, Sbarra, Bootzin, and Bernert (2010)	208	.67	19.23	Depression	Episode/Acute	BIS/BAS	Y	*r*	−1.18	.02	1.12	.02
Hellerstein, Kocsis, Chapman, Stewart, and Harrison (2000)	178	.00	42.00	Depression	Episode/Acute	TPQ	Y	*d*	.17	.02	1.75	.03
	532	.50	42.00	Depression	Episode/Acute	TPQ	Y	*d*	.31	.01	1.54	.01
Highsmith, Wuensch, Tran, Stephenson, and Everhart (2017)	18	.56	20.40	Anxiety	Healthy	BIS/BAS-FS	Y	*r*	−1.15	.24	1.62	.27
Hirano et al. (2002)	163	.58	42.59	Depression	Episode/Acute	TCI	Y	*d*	−.34	.03	1.69	.04
Holmes et al. (2012)	1050	NA	21.37	Anxiety	Healthy	TCI-HA; BIS/BAS-BIS	Y	*r*	NA	NA	1.40	.00
Hoyer, Braeuer, Crawcour, Klumbies, and Kirschbaum (2013)	88	.45	26.95	Anxiety	Episode/Acute	TPQ	Y	*d*	.34	.05	.46	.05
Hur and Kim (2009)	112	.66	38.70	Depression	Episode/Acute	TCI	Y	*d*	−.30	.04	.87	.04
Izci et al. (2014)	122	.67	35.70	Anxiety	Outpatients	TCI	Y	*d*	−.14	.03	.41	.03
Iidaka et al. (2006)	56	.46	22.30	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	.81	.08
Jackson (2009); Study 1	972	.72	22.00	Anxiety	Healthy	J-5	Y	*r*	.00	.00	1.04	.00
Jackson (2009); Study 2	190	.61	18.89	Anxiety	Healthy	J5; BIS/BAS	Y	*r*	−.36	.00	.84	.00
Jarmolowicz et al. (2014)	100	.49	30.70	Depression	Healthy	BIS/BAS	Y	*r*	−.14	.04	.41	.04
Jiang et al. (2003)	162	.74	19.40	Anxiety	Healthy	TCI	Y	*r*	−.23	.02	1.66	.03
	89	.49	23.20	Anxiety	Healthy	TCI	Y	*r*	−.32	.04	1.78	.06
Jiang and Tiliopoulos (2014)	225	.68	19.52	Anxiety	Healthy	BIS/BAS; RST-PQ	Y	*r*	−.55	.02	1.25	.02
Johnson et al. (2003)	1296	.47	19.00	Anxiety	Other	BIS/BAS-FS and RR	Y	*d*	.09	.01	NA	NA
Johnson and Carver (2006); Study 1	138	.68	18.00	Depression	Healthy	BIS/BAS	Y	*r*	−.21	.03	.41	.03
Johnson and Carver (2006); Study 2	285	.68	18.00	Depression	Healthy	BIS/BAS	Y	*r*	−.07	.01	.28	.01
Jokela and Keltikangas-Järvinen (2011)	1591	.57	37.80	Depression	Healthy	TCI	Y	*r*	−.05	.00	1.58	.00
Jones and Day (2008)	231	.79	28.52	Depression	Healthy	BIS/BAS	Y	*r*	−.15	.02	.48	.02
Josefsson, Larsson, Sydsjö, and Nylander (2007)	136	1.00	35.63	Depression	Outpatients	TCI	Y	*d*	−.37	.03	1.49	.04
Joyce, Light, Rowe, Cloninger, and Kennedy (2010)	412	.67	48.20	Depression	Not specified	TCI	Y	*d*	−.08	.01	.94	.01
Jung et al. (2011)	40	.35	25.23	Anxiety	Episode/Acute	BIS/BAS	Y	*d*	−.40	.10	.76	.10
Jylhä et al. (2011)	398	.69	NA	Depression	Episode/Acute	TCI-R	Y	*d*	−.54	.01	1.08	.01
Kambouropoulos, Egan, O'Connor, and Staiger (2014)	218	.73	32.84	Anxiety	Healthy	J5 - FFFS	Y	*r*	NA	NA	.63	.02
Kaneda, Yasui-Furukori, Nakagami, Sato, and Kaneko (2011)	166	.66	44.38	Depression	Episode/Acute	TCI	Y	*d*	−.69	.03	1.45	.03
Kang, Han, Hannon, Hall, and Choi (2015)	120	.00	20.48	Anxiety	Healthy	TCI	Y	*r*	NA	NA	.36	.03
Kasch et al. (2002)	89	.69	34.60	Depression	Episode/Acute	BIS/BAS	Y	*d*	−1.55	.07	1.15	.06
Kashdan and Roberts (2006); Study 1	104	.55	NA	Anxiety	Healthy	BIS/BAS	Y	*r*	−.36	.04	1.65	.05
Kashdan and Roberts (2006); Study 2	90	.50	19.38	Anxiety	Healthy	BIS/BAS	Y	*r*	−.36	.04	1.00	.05
Kerremans, Claes, and Bijttebier (2010)	239	.47	16.60	Depression	Healthy	BIS/BAS	N	*r*	−.47	.02	.54	.02
Keune, Bostanov, Kotchoubey, and Hautzinger (2012)	35	.69	24.14	Depression	Healthy	BIS/BAS	Y	*r*	−.10	.11	.06	.11
Kim and Grant (2001)	81	.27	41.45	Anxiety	Outpatients	TPQ	Y	*d*	−.15	.05	1.60	.06
Kim, Kang, and Kim (2009)	315	.38	34.59	Anxiety	Episode/Acute	TCI	Y	*d*	−.05	.01	1.28	.02

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Table 1 (*continued*)

ID_Text	N	PercWom	Age	Disorder cluster	Clinical status	RST scales	Pub status	Data type	*g* BAS	*vg* BAS	*g* BIS	*vg* BIS
Kimbrel, Cobb, Mitchell, Hundt, and Nelson-Gray (2008)	128	1.00	18.84	Anxiety	Healthy	SPSRQ	Y	*r*	−.14	.03	1.42	.04
Kimbrel, Nelson-Gray, and Mitchell (2012)	207	.67	19.10	Anxiety	Healthy	BIS/BAS	Y	*r*	−.25	.02	1.51	.02
Kimura et al. (2000)	117	.00	43.40	Depression	Remission	TCI	Y	*d*	−.31	.03	1.57	.04
	168	1.00	43.40	Depression	Remission	TCI	Y	*d*	−.20	.02	1.12	.03
Kircanski et al. (2013)	152	1.00	44.00	Depression	Remission	BIS/BAS	Y	*d*	−.50	.03	.71	.03
Knyazev and Slobodskaya (2003)	47	.87	23.69	Anxiety	Healthy	GWPQ	Y	*r*	.30	.08	1.44	.10
Knyazev et al. (2004)	345	.73	20.00	Anxiety	Healthy	GWPQ; BIS/BAS	Y	*r*	.11	.01	1.00	.01
Knyazev, Bocharov, Slobodskaya, and Ryabichenko (2008)	292	.41	15.50	Anxiety	Healthy	GWPQ	Y	*r*	.16	.01	1.18	.02
Knyazev, Levin, and Savostyanov (2008)	51	.69	20.00	Anxiety	Healthy	GWPQ; BIS/BAS-BIS	Y	*r*	NA	NA	1.23	.09
Kramer, Rodriguez, and Kertz (2015)	120	.53	44.09	Anxiety	Episode/Acute	J5	Y	*d*	−.58	.04	−.31	.04
Kushner, Abrams, Thuras, and Hanson (2000)	49	1.00	47.00	Anxiety	Healthy	TPQ	Y	*r*	.51	.08	1.60	.11
	24	.00	23.50	Anxiety	Healthy	TPQ	Y	*r*	.25	.16	2.49	.28
Kusunoki et al. (2000)	26	.00	35.20	Anxiety	Remission	TCI	Y	*d*	−.48	.16	2.31	.26
	39	1.00	35.20	Anxiety	Remission	TCI	Y	*d*	−.39	.11	2.28	.18
	25	.00	36.00	Depression	Remission	TCI	Y	*d*	−.13	.17	1.74	.23
	39	1.00	36.00	Depression	Remission	TCI	Y	*d*	.15	.11	1.20	.13
Landman, Nieuwenhuys, and Oudejans (2016)	59	.00	37.90	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	1.03	.07
Lee et al. (2012)	344	.62	42.75	Depression	Not specified	TCI	Y	*d*	−.15	.04	.98	.04
Leikas, Lindeman, Roininen, and Lähteenmäki (2007)	1270	.51	45.76	Anxiety	Healthy	BIS/BAS	Y	*r*	.26	.00	.80	.00
Lemaire, El-Hage, and Frangou (2014)	101	.49	43.90	Anxiety	Healthy	TPQ	N	*r*	−.10	.04	1.78	.05
Lemogne et al. (2009)	60	.50	23.45	Depression	Healthy	TCI	N	*r*	−.15	.07	.47	.07
Levinson, Rodebaugh, and Frye (2011); Study 1	723	.68	19.14	Anxiety	Healthy	BIS/BAS	Y	*r*	−.44	.01	.80	.01
Li, Xu, and Chen (2015)	330	.58	16.95	Depression	Healthy	BIS/BAS	Y	*r*	.08	.01	.63	.01
Lochner et al. (2007)	109	.70	35.80	Anxiety	Episode/Acute	TCI-NS and HA	Y	*d*	−.44	.04	1.06	.05
Lorian and Grisham (2010)	55	.73	20.24	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	1.40	.09
Lovallo et al. (2014)	314	.57	23.50	Depression	Healthy	TPQ	N	*r*	−.33	.01	.93	.01
Lövdahl, Bøen, Falkum, Hynnekleiv, and Malt (2010)	26	.85	33.60	Depression	Not specified	TCI	Y	*d*	.27	.18	.78	.20
Lyoo, Yoon, Kang, and Kwon (2003)	70	.37	28.70	Anxiety	Episode/Acute	TCI	Y	*d*	−.43	.06	1.20	.07
Lyvers, Duric, and Thorberg (2014)	106	.63	21.21	Anxiety	Healthy	SPSRQ	Y	*r*	.58	.04	.62	.04
Lyvers, Lysychka, and Thorberg (2014)	113	.69	22.11	Anxiety	Healthy	SPSRQ	Y	*r*	.65	.04	.67	.04
Maack, Tull, and Gratz (2012)	91	.63	24.78	Anxiety	Episode/Acute	BIS/BAS	Y	*d*	−.15	.08	1.17	.09
Maack, Buchanan, and Young (2015)	291	.75	20.38	Anxiety	Healthy	BIS/BAS	Y	*r*	−.13	.01	.47	.01
Mansell, Rigby, Tai, and Lowe (2008)	191	.84	20.00	Depression	Healthy	BIS/BAS	Y	*r*	.09	.02	.70	.02
Marchesi, Cantoni, Fontò, Giannelli, and Maggini (2006)	142	.69	36.00	Anxiety	Episode/Acute	TCI	Y	*d*	−.15	.03	.83	.03
Mardaga and Hansenne (2009)	40	.55	42.80	Depression	Episode/Acute	TCI-R; BIS/BAS	Y	*d*	−.03	.10	1.27	.12
Mardaga and Iakimova (2014)	38	.71	42.80	Depression	Episode/Acute	TCI-R	Y	*d*	−.67	.11	.75	.11
Marteinsdottir, Tillfors, Furmark, Anderberg, and Ekselius (2003)	431	NA	33.50	Anxiety	Episode/Acute	TCI-R	Y	*d*	−.32	.03	1.19	.04
Meerkerk, van den Eijnden, Franken, and Garretsen (2010)	1369	.63	42.00	Depression	Episode/Acute	TCI	Y	*d*	−.44	.00	1.12	.00
Mertol and Alkın (2012)	79	.57	41.50	Anxiety	Outpatients	TCI	Y	*d*	2.31	.08	5.50	.24
Minaya and Fresán (2009)	82	.67	34.20	Depression	Outpatients	TCI	Y	*d*	−.31	.05	.55	.05
	87	.67	34.20	Other	Outpatients	TCI	Y	*d*	.27	.05	.45	.05
Minelli et al. (2009)	327	.59	50.30	Other	Episode/Acute	TCI	Y	*d*	−.41	.02	1.06	.02
Mitchell and Nelson-Gray (2006)	184	.70	18.92	Anxiety	Healthy	BIS/BAS; SPSRQ	Y	*r*	.16	.02	.86	.02
Mitsui et al. (2013)	479	.00	18.70	Depression	Episode/Acute	TCI	Y	*d*	.18	.04	.79	.04
	203	1.00	18.55	Depression	Episode/Acute	TCI	Y	*d*	.12	.07	.91	.07
	489	.00	18.70	Depression	Episode/Acute	TCI	Y	*d*	.00	.03	.63	.03
	250	1.00	18.40	Depression	Episode/Acute	TCI	Y	*d*	.25	.02	.24	.02
Miyoshi et al. (2016)	85	.45	26.24	Depression	Episode/Acute	TCI	Y	*d*	−2.68	.13	5.89	.30
Mommersteeg et al. (2011)	92	.07	28.50	Anxiety	Episode/Acute	TCI	N	*d*	.63	.26	.81	.26
Mörtberg, Bejerot, and Wistedt (2007)	459	.51	34.90	Anxiety	Episode/Acute	TCI	Y	*d*	−.32	.02	1.89	.02
Movius and Allen (2005)	98	.52	19.00	Anxiety	Healthy	BIS/BAS	Y	*r*	−.17	.04	.97	.05
Mueller et al. (2011)	286	1.00	22.90	Depression	Healthy	BIS/BAS	Y	*r*	.39	.01	.56	.01
	124	.00	22.90	Depression	Healthy	BIS/BAS	Y	*r*	.47	.03	.51	.03
Mulder, Joyce, and Cloninger (1994)	148	.51	41.50	Depression	Episode/Acute	TPQ	Y	*d*	−.43	.03	3.47	.07
Müller and Wytykowska (2005)	202	NA	NA	Anxiety	Healthy	BIS/BAS	Y	*r*	−.18	.02	.92	.02
Naito, Kijima, and Kitamura (2000)	220	.50	20.90	Depression	Healthy	TCI	Y	*r*	.00	.02	1.21	.02
Nery et al. (2009)	75	.33	38.10	Depression	Episode/Acute	TCI	Y	*d*	.46	.06	2.05	.08
	45	.33	39.10	Depression	Remission	TCI	Y	*d*	.35	.10	.48	.10
Nicholls, Staiger, Williams, Richardson, and Kambouropoulos (2014)	350	.71	34.21	Anxiety	Healthy	SPSRQ	N	*r*	.05	.01	2.55	.02
Norris, Larsen, Crawford, and Cacioppo (2011); Study 1	65	1.00	NA	Depression	Healthy	BIS/BAS	Y	*r*	.08	.06	1.30	.07

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Table 1 (*continued*)

ID_Text	N	PercWom	Age	Disorder cluster	Clinical status	RST scales	Pub status	Data type	*g* BAS	*vg* BAS	*g* BIS	*vg* BIS
North and Cloninger (2012)	151	.48	43.00	Anxiety	Episode/Acute	TCI-NS and HA	N	*d*	.26	.03	.85	.03
Nowakowska, Strong, Santosa, Wang, and Ketter (2005)	71	.45	33.60	Depression	Remission	TCI	Y	*d*	.49	.08	1.26	.10
Nyman et al. (2011)	3902	.50	16.00	Depression	Remission	TCI	Y	*d*	.10	.00	.67	.00
O'Connor, Staiger, Kambouropoulos, and Smillie (2014)	402	.78	32.49	Anxiety	Healthy	BIS/BAS	Y	*r*	−.58	.01	1.42	.01
Panayiotou, Karekla, and Panayiotou (2014)	127	.82	21.22	Anxiety	Healthy	SPSRQ	Y	*r*	.45	.03	1.49	.04
Peirson and Heuchert (2001)	471	.76	19.19	Depression	Healthy	TCI	Y	*r*	−.05	.01	.98	.01
Perich, Manicavasagar, Mitchell, and Ball (2011)	69	.75	41.18	Depression	Remission	BIS/BAS	Y	*d*	−.40	.06	.21	.06
Perkins and Corr (2006)	83	1.00	29.03	Anxiety	Healthy	BIS/BAS	Y	*r*	.01	.05	.99	.05
	58	.00	29.03	Anxiety	Healthy	BIS/BAS	Y	*r*	.25	.07	.80	.07
Perkins et al. (2007)	101	.41	20.00	Anxiety	Healthy	BIS/BAS	Y	*r*	−1.06	.04	1.14	.05
Perkins, Cooper, Abdelall, Smillie, and Corr (2010); Study 1	173	.54	23.90	Anxiety	Healthy	BIS/BAS	Y	*r*	−.05	.02	1.29	.03
Perkins et al. (2010); Study 2	97	.77	23.05	Anxiety	Healthy	BIS/BAS	Y	*r*	−.15	.04	.90	.04
Pfohl, Black, Noyes, Kelley, and Blum (1990)	60	.63	38.00	Anxiety	Episode/Acute	TPQ	Y	*d*	−.41	.07	2.12	.10
Pickett, Bardeen, and Orcutt (2011)	851	1.00	19.48	Anxiety	Healthy	BIS/BAS	Y	*r*	.03	.00	.49	.00
Pinciotti, Seligowski, and Orcutt (2017)	322	.72	21.30	Anxiety	Healthy	J5-FFFS	Y	*r*	NA	NA	−.46	.01
Pinto-Meza et al. (2006)	30	NA	22.00	Depression	Episode/Acute	SPSRQ	Y	*d*	−1.55	.17	1.45	.16
	50	NA	35.00	Depression	Remission	SPSRQ	Y	*d*	−.92	.10	.00	.09
Prochwicz and Gawęda (2016)	492	.89	21.58	Depression	Healthy	TCI	Y	*r*	.02	.01	1.06	.01
Putman, van Peer, Maimari, and van der Werff (2010)	28	1.00	22.70	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	2.22	.22
Reimold et al. (2008)	29	.45	46.30	Depression	Episode/Acute	TCI	Y	*d*	−.21	.14	2.35	.24
Richman and Frueh (1997)	515	.00	45.10	Anxiety	Episode/Acute	TPQ	Y	*d*	.62	.02	1.97	.02
Richter, Summerfeldt, Joffe, and Swinson (1996)	64	.41	35.50	Anxiety	Episode/Acute	TPQ	Y	*d*	−.31	.06	1.29	.07
Richter, Eisemann, and Richter (2000)	192	.62	44.80	Depression	Episode/Acute	TCI	Y	*d*	−.92	.03	1.46	.03
Richter, Polak, and Eisenmann (2003)	466	.00	36.84	Depression	Episode/Acute	TCI	Y	*d*	−.44	.01	1.21	.01
	837	1.00	38.28	Depression	Episode/Acute	TCI	Y	*d*	−.37	.01	.99	.01
Rönnlund, Vestergren, Mäntylä, and Nilsson (2011)	255	.48	75.00	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	.77	.02
Rybakowski, Slopien, Zakrzewska, Hornowska, and Rajewski (2004)	60	1.00	15.90	Depression	Healthy	TCI	Y	*r*	−.13	.07	1.41	.08
Salter (2013)	36	.14	43.10	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	−.74	.11
Sasayama et al. (2011)	122	.00	35.19	Depression	Outpatients	TCI	Y	*d*	−.67	.04	1.84	.05
	122	1.00	37.93	Depression	Outpatients	TCI	Y	*d*	−.53	.04	1.56	.04
Sato et al. (1999)	141	.25	47.43	Depression	Outpatients	TCI	Y	*d*	−.22	.03	1.92	.04
Saviotti et al. (1991)	66	.82	30.70	Anxiety	Remission	TPQ	Y	*d*	−.07	.06	2.05	.09
Segarra, Poy, López, and Moltó (2014)	329	.55	20.23	Anxiety	Healthy	BIS/BAS	Y	*r*	.14	.01	1.21	.01
Shachar, Aderka, and Gilboa-Schechtman (2014)	1362	.53	14.44	Anxiety	Healthy	JTCI-HA	Y	*r*	NA	NA	.65	.00
Sharma et al. (2017)	58	.52	36.72	Depression	Not specified	BIS/BAS-RR	Y	*d*	−.67	.07	NA	NA
Slessareva and Muraven (2004)	146	NA	NA	Depression	Healthy	BIS	Y	*r*	NA	NA	.43	.03
Smillie, Dalgleish, and Jackson (2007); Study 1	60	.62	20.60	Anxiety	Healthy	BIS/BAS	Y	*r*	−.99	.07	1.41	.08
Smillie et al. (2007); Study 2	81	.65	20.70	Anxiety	Healthy	BIS/BAS	Y	*r*	.32	.05	1.34	.06
Smith, Duffy, Stewart, Muir, and Blackwood (2005)	39	.00	21.20	Depression	Remission	TCI	Y	*d*	.55	.11	1.63	.14
	102	1.00	21.20	Depression	Remission	TCI	Y	*d*	−.04	.04	1.54	.05
Soler et al. (2014)	145	.93	34.49	Depression	Not specified	SPSRQ	Y	*d*	.14	.03	.93	.03
Stein, Chartier, Lizak, and Jang (2001)	55	.62	41.71	Anxiety	Episode/Acute	TPQ	Y	*d*	.01	.07	.58	.07
Stewart, Donaghey, Deary, and Ebmeier (2008)	872	.61	20.89	Anxiety	Healthy	TPQ	N	*r*	−.16	.00	2.29	.01
Takahashi et al. (2013)	122	.25	37.33	Depression	Episode/Acute	TCI	Y	*d*	−.59	.04	1.85	.05
	118	.27	37.76	Depression	Remission	TCI	Y	*d*	−.25	.04	.71	.05
Takahashi, Ozaki, Roberts, and Ando (2012); Study 1	489	NA	19.68	Depression	Healthy	BIS/BAS-BAS	Y	*r*	−.34	.01	NA	NA
Takahashi et al. (2012); Study 2	109	NA	20.31	Depression	Healthy	BIS/BAS-BAS	Y	*r*	−.72	.04	NA	NA
Takahashi, Roberts, Yamagata, and Kijima (2015)	319	.69	18.77	Depression	Healthy	BIS/BAS	Y	*r*	−.14	.01	.75	.01
Thierry et al. (2004)	132	1.00	32.80	Anxiety	Outpatients	TCI	Y	*d*	−.50	.03	1.56	.04
Torrubia et al. (2001); Study 3a	96	.00	20.14	Anxiety	Healthy	SPSRQ	Y	*r*	−.06	.04	1.84	.06
Torrubia et al. (2001); Study 3b	276	1.00	20.14	Anxiety	Healthy	SPSRQ	Y	*r*	.20	.01	1.46	.02
Tse, Rochelle, and Cheung (2011)	902	.47	24.55	Depression	Healthy	TCI-HA	Y	*r*	NA	NA	.72	.00
Ubl et al. (2015)	46	.65	41.96	Depression	Remission	TPQ	Y	*d*	−.25	.08	1.41	.11
Uzieblo, Verschuere, and Crombez (2007)	431	.58	18.81	Anxiety	Healthy	BIS/BAS	Y	*r*	.22	.01	1.06	.01
Van der Gucht, Morriss, Lancaster, Kinderman, and Bentall (2009)	44	.66	46.72	Depression	Episode/Acute	BIS/BAS	Y	*d*	−.26	.10	.94	.11
Van Meter and Youngstrom (2015)	313	.62	19.85	Depression	Not specified	BIS/BAS	Y	*d*	−.55	.05	.08	.05
Vangberg (2012)	1239	.51	16.80	Depression	Healthy	JTCI	Y	*r*	−.25	.00	1.35	.00

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Table 1 (*continued*)

ID_Text	N	PercWom	Age	Disorder cluster	Clinical status	RST scales	Pub status	Data type	*g* BAS	*vg* BAS	*g* BIS	*vg* BIS
Vergara and Roberts (2011)	83	.57	19.35	Depression	Healthy	SPSRQ	Y	*r*	.18	.05	1.00	.05
Vervoort et al. (2010)	70	.00	13.11	Anxiety	Outpatients	BIS/BAS	Y	*d*	.06	.07	1.01	.08
	105	1.00	13.11	Anxiety	Outpatients	BIS/BAS	Y	*d*	−.75	.04	.48	.04
Voth et al. (2014)	31	.81	39.07	Depression	Healthy	BIS/BAS	N	*r*	.14	.12	.51	.13
Wachleski et al. (2008)	270	.74	38.10	Anxiety	Outpatients	TCI	Y	*d*	.07	.01	1.54	.02
Wagener, Baeyens, and Blairy (2016)	504	.74	36.10	Depression	Healthy	BIS/BAS	Y	*r*	−.09	.01	1.03	.01
Wang, Mason, Charney, and Yehuda (1997)	363	.00	42.40	Anxiety	Episode/Acute	TPQ	Y	*d*	−4.08	.05	1.61	.03
Wang et al. (2014)	323	.48	23.75	Depression	Healthy	TPQ-HA	Y	*r*	NA	NA	.79	.01
Westlye, Bjørnebekk, Grydeland, Fjell, and Walhovd (2011)	263	.57	50.00	Depression	Healthy	TPQ-HA	Y	*r*	NA	NA	.95	.02
Wiborg, Falkum, Dahl, and Gullberg (2005)	182	.72	35.42	Anxiety	Outpatients	TPQ	Y	*d*	.19	.03	1.26	.03
Windle (1994)	4462	NA	37.83	Depression	Healthy	MS-BIS; MMPI-MAC	Y	*r*	−.24	.00	1.62	.00
Wright, Scerpella, and Lisdahl (2016)	42	.52	21.10	Depression	Healthy	BIS/BAS	N	*r*	.22	.09	−.17	.09
Wu et al. (2012)	152	.70	44.51	Depression	Outpatients	TPQ	Y	*d*	−.01	.03	1.87	.04
Wytykowska, Fajkowska, Domaradzka, and Jankowski (2017); Study 1	1512	.54	39.41	Anxiety	Healthy	RST-PQ	Y	*r*	−.42	.00	1.06	.00
Wytykowska et al. (2017); Study 2	124	.59	38.23	Depression	Episode/Acute	RST-PQ	Y	*d*	−2.08	.05	1.96	.05
Yen et al. (2012)	2282	.52	20.96	Anxiety	Not specified	BIS/BAS	Y	*d*	.57	.01	1.32	.01
Young et al. (1995)	47	.00	38.90	Depression	Outpatients	TPQ	Y	*d*	−.45	.08	1.04	.09
	65	1.00	38.80	Depression	Outpatients	TPQ	Y	*d*	.14	.06	.38	.06
Zaninotto et al. (2015)	165	.65	44.86	Depression	Episode/Acute	TCI	N	*d*	−.32	.03	.42	.03
Zhao, Cheng, Harris, and Vigo (2015)	108	.61	19.28	Anxiety	Healthy	BIS/BAS-BIS	Y	*r*	NA	NA	.86	.04
Zhao, Harris, and Vigo (2016)	98	1.00	NA	Anxiety	Healthy	BIS/BAS-BIS	N	*r*	NA	NA	.85	.04
	62	.00	NA	Anxiety	Healthy	BIS/BAS-BIS	N	*r*	NA	NA	.48	.06
Zvolensky, Feldner, Eifert, and Stewart (2001)	60	.57	NA	Anxiety	Healthy	BIS/BAS	Y	*r*	.16	.07	1.44	.08

Note. BIS/BAS: Behavioral Inhibition System/Behavioral Activation scale, BIS/BAS- Dr FS and RR: BIS/BAS subscales Drive Fun Seeking and Reward Responsiveness, GRAPES: Generalized Reward and Punishment Expectancy Scales, GWPQ: Gray-Wilson Personality Questionnaire, J5: Jackson 5, J5-FFFS: J5 Fight Flight Freeze Scale, JTCI: Junior Temperament and Character Inventory, JTCI-HA: JTCI Harm Avoidance Subscale, MMPI-MAC: Minnesota Multiphasic Personality Inventory's MAC scale, MS-BIS: MacAndrew and Steele's (1991) Behavior Inhibition Scale, RST-PQ: Reinforcement Sensitivity Theory Personality Questionnaire, SPSRQ: Sensitivity to Punishment, Sensitivity to Reward Questionnaire, TPQ: Tridimensional Personality Questionnaire, TCI: Temperament and Character Inventory, TCI-HA and NS: TCI Harm Avoidance and Novelty Seeking subscales, TCI-R: Revised TCI.
Data type indicates the type of effect size selected. d = standard mean difference; r = correlation.

Table 2a
Analysis summary of BAS and categorical moderators.

Moderator	*k*	Hedges' g	95% CI	Test of subgroup differences
Main effect	228	−.16	[−.21; −.11]	
Disorder				$Q(1) = 3.83, p = .05$
Depression	116	−.21	[−.28; −.15]	
Anxiety	108	−.11	[−.19; −.02]	
Diagnostic method				$Q(1) = 15.60, p < .0001$
Clinical diagnosis	111	−.28	[−.38; −.19]	
Self-report	117	−.06	[−.12 −.00]	
Source				$Q(1) = 2.76, p = .10$
Published	208	−.17	[−.23; −.12]	
Unpublished	20	−.06	[−.18; .05]	

Table 2b
Analysis summary of BIS and categorical moderators.

Moderator	*k*	Hedges' g	95% CI	Test of subgroup differences
Main effect	255	1.11	[1.02; 1.19]	
Disorder				$Q(1) = 6.57, p = .01$
Depression	125	.99	[.87; 1.12]	
Anxiety	126	1.21	[1.10; 1.32]	
Diagnostic method				$Q(1) = 15.50, p < .0001$
Clinical diagnosis	114	1.29	[1.17; 1.42]	
Self-report	141	.96	[.85; 1.07]	
Source				$Q(1) = 1.18, p = .28$
Published	232	1.12	[1.04; 1.19]	
Unpublished	23	.85	[.38; 1.32]	

Table 3a
Random-effects models of significant moderators of BAS.

Moderator	Univariate metaregression				Multivariate metaregression		
	Beta	SE	95% CI	R^2	*Beta*	SE	95% CI
Disorder	−.12*	.05	[−.22; −.02]	.000	−.07	.08	[−.22; .08]
Publication status	−.13	.09	[−.31; .05]	.000			
Assessment method	−.22***	.05	[−.32; −.11]	.037	−.24**	.07	[−.38; −.09]
N	.00	.00	[−.00; .00]	.000			
Age	−.01*	.00	[−.01; −.00]	.020	.00	.00	[−.01; .01]
Percent women	.16	.10	[−.05; .36]	.000			

Note. Disorder: 0 = anxiety, 1 = depression; Publication status: 0 = unpublished, 1 = published; Clinical status: 0 = healthy, 1 = clinical; N = sample size of study; Age = mean age of participants; Percent women: Percent of sample that was female.
*p < .05; **p < .01; ***p < .001

(254) = 6489.16, p < .0001; tau^2 = 0.43; I^2 = 96.1% [95.8%; 96.3%]. This large heterogeneity was reflected in a wide prediction interval for BIS as well, though the majority of effect sizes were in the same direction, 95% PI [−.18; 2.40]. These findings were consistent with Hypothesis 1, indicating a small, negative effect for BAS across disorders, and a large, positive, effect for BIS.

Next, in order to test Hypothesis 2, we examined disorder cluster as a possible moderator for the above, relationships (see Tables 2a and 2b). BAS was found to have a small relationship with depressive disorders, g = −.21; 95% CI [−.28; −.15], and a trivial relationship with anxiety disorders, g = −.11; 95% CI [−.19; −.02]. This was consistent with Hypothesis 2a. The difference between the two types of disorders was found to be significant, $Q(1)$ = 3.83, p = .05, which was consistent with Hypothesis 2b. BIS had a large, positive relationship with depression, g = .99; 95% CI [.87; 1.12], as well as anxiety g = 1.21; 95% CI [1.10; 1.32], which was consistent with Hypotheses 2c and 2d. However, contrary to expectations, the effect sizes of the two groups significantly differed from each other, $Q(1)$ = 6.57, p = .01. Taken together, BIS was found to have a large, positive effect size across disorders, which was significantly larger for anxiety. BAS, on the other hand, was found to have a small, negative effect size for depression, and a trivial one for anxiety. This was generally consistent with the patterns predicted in Hypothesis 2.

4.2.3. Moderator analysis

The large portions of variance due to heterogeneity of effect sizes called for a further analysis of moderators. First, a series of subgroup analyses were performed to measure the impact of categorical moderators, in addition to disorder (Tables 2a and 2b). Consistent with our predictions in Hypothesis 3, greater effect sizes were observed when clinical assessment was derived from diagnostic means than from self-report measures with regards to BAS (g = −.28 vs −.06) as well as BIS (g = 1.29 vs .96). These differences were significant (ps < .0001). No other moderators were found to be significant for both the BAS and BIS (ps > .09).

Table 4
Intercorrelation of the Study 1 moderator variables.

	g_BAS	g_BIS	N	Dep	Clin	Src	Age	Women
g_BAS	–							
g_BIS	−.23**	–						
N	.09	−.11	–					
Disorder	−.12	−.14*	.09	–				
Assessment	−.19**	.22**	−.16*	.21**	–			
Source	−.10	.11	−.04	−.02	.14*	–		
Age	−.09	.10	−.07	.27**	.49**	.07	–	
Percent Women	.09	−.15*	−.09	.01	−.09	.02	−.09	–

Note. g_BAS = BAS effect size; g_BIS = BIS effect sizes measured in Hedges' g; N = total N; Disorder is the pathology measure used with depression = 1, anxiety = 0; Assessment is the assessment strategy with self-report correlation = 0, diagnosed-healthy comparison = 1; Source is the source of the data with "published data" = 1, "unpublished data" = 0; Age is mean age; Percent Women is calculated as n_wom/n.
* < .05; ** < .01

Next, to examine the role of continuous variables as moderators, a series of univariate regressions were performed (See Tables 3a and 3b). For BAS, the average age of participants was a significant moderator, but to a negligibly small degree (b = −.01, R^2 < 0.02). The percent of women in the sample was found to significantly impact BIS, b = −.38, p = .02, 95% CI [−.69; −.07], but not BAS b = .16, p = .13, 95% CI [−.05; .36]. Interpretation of the univariate analyses, however, was limited by the intercorrelation of moderator variables (see Table 4). Thus, a series of multivariate analyses were performed only for the significant moderators of BAS and BIS, including disorder. Clinical assessment method was found to be the only significant moderator in the multivariate analysis of BAS, with clinical diagnosis predicting a more negative effect size than self-report, b = −.24, p = .001, 95% CI [−.38; −.09]. BIS was significantly moderated by both clinical assessment method, b = .40, p < .0001, 95% CI [.20; .60], disorder, b = −.31, p = .005, 95% CI [−.50; −.12], and percent women,

Table 3b
Random-effects models of significant moderators of BIS.

Moderator	Univariate metaregression				Multivariate metaregression		
	Beta	SE	95% CI	R^2	*Beta*	SE	95% CI
Disorder	−.22**	.09	[−.39; −.06]	.022	−.31***	.10	[−.50; −.12]
Publication status	.26	.14	[−.01; .54]	.136			
Assessment method	.34***	.09	[.17; .51]	.025	.40***	.10	[.20; .60]
N	−.00	.00	[−.00; .00]	.027			
Age	.01	.00	[−.00; .00]	.051			
Percent women	−.38*	.16	[−.69; −.07]	.061	−.32**	.16	[−.60; −.01]

Note. Disorder: 0 = anxiety, 1 = depression; Publication status: 0 = unpublished, 1 = published; Clinical status: 0 = healthy, 1 = clinical; N = sample size of study; Age = mean age of participants; Percent women: Percent of sample that was female.
*p < .05; **p < .01; ***p < .001

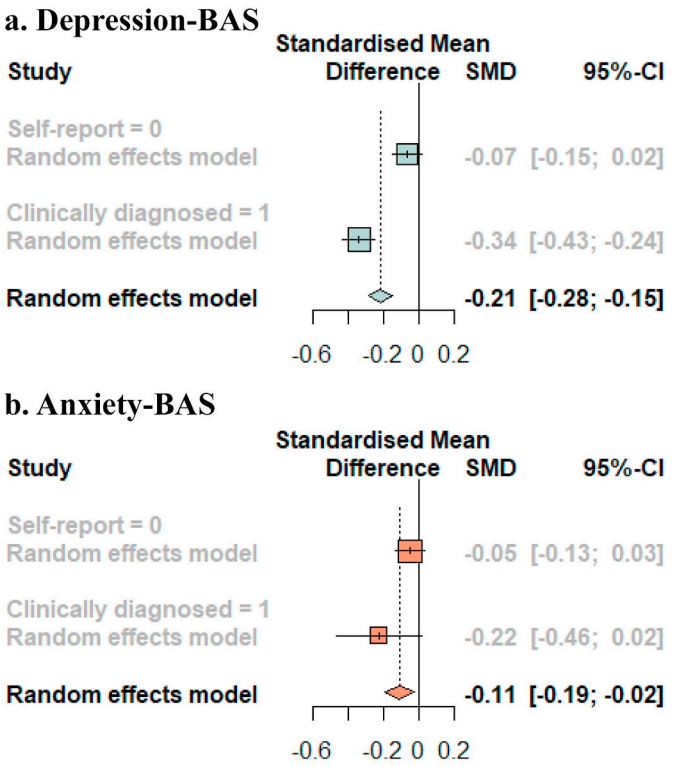

Fig. 2. (a–b) Forest plot of BAS interaction between disorder and assessment method.

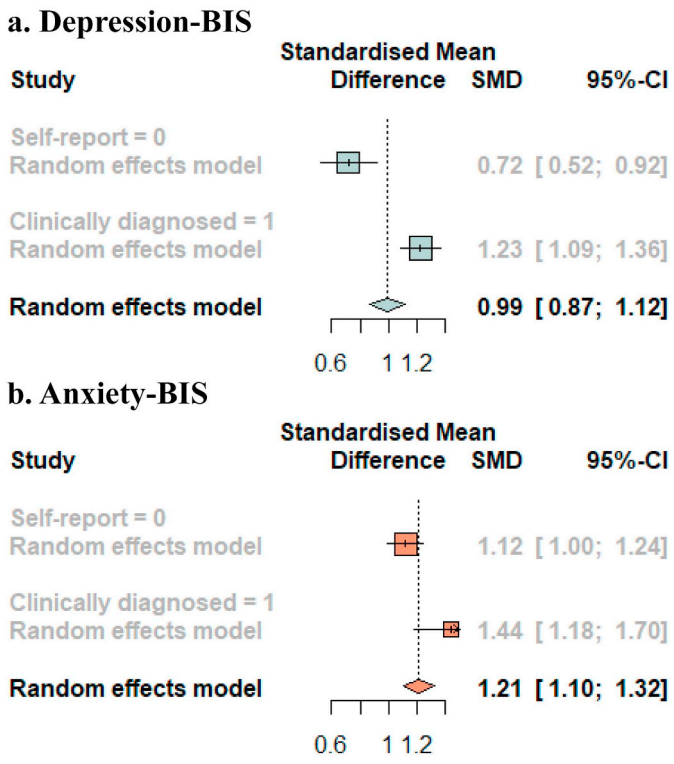

Fig. 3. (a–b) Forest plot of BIS interaction between disorder and assessment method.

$b = -.32$, $p = .04$, 95% CI [−.62; −.01]. Thus, when controlling for disorder cluster and gender, clinical assessment method increased effect sizes for both BAS and BIS. When controlling for clinical assessment method and gender, larger effect sizes were found for anxiety (compared to depression) with regards to BIS, but not BAS.

4.2.4. Interaction between disorder cluster and assessment method of sample

In order to better understand the interaction between clinical assessment method and disorder, a series of subgroup analyses were performed to compare patterns of moderation in depression and anxiety samples. First, analyses were performed separately for studies that collected depression and anxiety data, with clinical assessment method as the grouping variable (see Figs. 2a,b and 3a,b). Anxiety and depression followed two distinct patterns. For depression a small, significant relationship was found between BAS and depression when clinical diagnosis was used ($g = -.34$, 95% CI[−.43; −.24]). No such relationship was found for self-report clinical assessment ($g = -.07$; 95% CI [−.15; .02]). This led to a significant difference between the subgroups, $Q(1) = 17.88$, $p < .0001$. There was also a difference between the large positive effects found between depression and BIS among diagnosed ($g = 1.23$, 95% CI [1.09; 1.36]) and self-reporting ($g = .72$, 95% CI [.52; .92]) samples that was statistically significant, $Q(1) = 17.10$, $p < .0001$. Anxiety showed different patterns depending upon reinforcement system. Anxiety and BAS did not show a significant relationship among self-report correlations ($g = -.05$, 95% CI [−.13; .03]) or comparisons between diagnosed and healthy participants ($g = -.22$, 95% CI [−.46; .02]), with no significant difference between these groups, $Q(1) = 1.83$, $p = .18$. On the other hand, a large positive effect size was found between anxiety and BIS both among diagnosed-healthy comparisons ($g = 1.44$, 95% CI [1.18; 1.70] as well as self-report correlations ($g = 1.12$, 95% CI [1.00; 1.24]), with a significant difference between, $Q(1) = 4.83$, $p = .03$. However, the difference in BAS effect sizes between the two groups was quite smaller for anxiety ($Q(1) = 4.83$) than it was for depression ($Q(1) = 17.10$).

Thus, an interaction was revealed between diagnostic method and disorder cluster. The relationship between BAS and depression was only significant for diagnosed-healthy comparisons, and not for self-report correlations. These groups significantly differed from each other. The relationship between BIS and depression also revealed a significant difference between groups, though both still maintained large effect sizes. For anxiety, neither group's BAS effect sizes were significantly different from zero, nor were the groups different from each other. Anxiety's groups did significantly differed from each other for BIS effect sizes, though to a much smaller degree than depression.

These between-group differences, however, may have been confounded by the fact that the majority of effect sizes drawn self-report assessment were based off of correlational data, whereas all the effect sizes drawn from clinical diagnosis were based off of standard mean differences. Thus, it remains unclear from these findings whether differences in effect sizes resulted from a meaningful differences between the groups, or from a statistical artifact (see Aldao et al., 2010). Indeed, it is possible that the differences observed between self-report effects and clinical diagnosis effects were caused by greater levels of symptom severity observed in diagnosed samples. In order to better understand the role symptom severity in predicting reinforcement sensitivity, it would be necessary to compare participants recruited through similar diagnostic methods, but who differ in current symptom severity.

To circumvent the above statistical issue, a series of subgroup analyses were performed only on data derived from diagnosed-healthy comparison studies ($k = 112$ for BAS and 116 for BIS), comparing the effect sizes based on the clinical status of the participants (Tables 5a and 5b). Importantly, all effect sizes in this analysis were based on group differences between diagnosed and healthy participants. They only differed based on the severity of participants' current clinical state at the time of data collection. Depression's effect sizes did show significant differences both with regards to BAS, $Q(2) = 13.02$, $p = .002$, as well as BIS, $Q(2) = 16.33$, $p = .0003$. An examination of overlapping confidence intervals revealed that depression's BAS effect sizes tended to cluster around two groups, with the weighted average effect size for

Table 5a
Clinical status-by-disorder for BAS.

Moderator	Total			Depression			Anxiety		
	k	Hedges' *g*	*95% CI*	*k*	Hedges' *g*	95% CI	*k*	Hedges' *g*	*95% CI*
Main effect	112	**−.29**	**[−.39; −.20]**	65	**−.36**	**[−.47; −.25]**	43	**−.22**	**[−.43; −.01]**
Clinical status									
Episode/Acute	64	**−.43**	**[−.58; −.29]**	35	**−.51**	**[−.67; −.34]**	28	**−.33**	**[−.61; −.04]**
Outpatients	21	−.12	[−.34; .10]	9	**−.35**	**[−.53; −.17]**	10	−.02	[−.32; .28]
Euthymic/Remission	27	**−.11**	**[−.22; −.00]**	21	**−.14**	**[−.26; −.01]**	5	−.02	[−.40; .37]

Note. Bolded effect sizes are significantly different from zero.

Euthymic/Remission participants (g = −.14, 95% CI [−.26; −.01]) distinct from those of the Episode/Acute group (g = −.51, 95% CI [−.67; −.34]). The Outpatient group overlapped with the other groups (g = −.35, 95% CI [−.53; −.17]). A similar pattern emerged with regards to depression and BIS. There, the Episode/Acute group (g = 1.48, 95% CI [1.29; 1.67]) was distinct from the Euthymic/Remission group (g = .87, 95% CI [.65; 1.10]). Again, the Outpatient group (g = 1.32, 95% CI [.96; 1.68]) overlapped with the two others. The effects of anxiety disorders were not moderated by clinical status with regards to BAS, $Q(2)$ = 2.68, p = .26, or BIS, $Q(2)$ = 1.53, p = .47.

Thus, two patterns emerged based on disorder. Depression's effect sizes were significantly larger when participants were selected for the sample based on clinical diagnosis. Furthermore, within diagnosed-healthy comparisons, effects were larger when clinical groups were in a current depressive episode than among those in remission. Anxiety, on the other hand, did not have these differences. No relationship was found between anxiety and BAS, regardless of diagnostic method or clinical state. Similarly, BIS and anxiety had a consistently large effect size. Differences were observed when comparing effect sizes derived from diagnosed-healthy comparisons versus self-report correlations. However, these differences were not maintained when comparing between clinical statuses within diagnosed-healthy comparisons.

4.2.5. Publication bias analysis

First, Egger's tests were conducted, to examine the possibility of a systematic bias. No such bias was found with regards to BAS, $t(226)$ = −1.06, p = .29. BIS, on the other hand, was found to contain significant bias, $t(253)$ = 3.11, p = .002. Despite Egger's test only being significant with regards to BIS, a trim-and-fill procedure was applied to adjust for possible publication bias in both BAS and BIS (see Fig. 4a and b). While the procedure reduced the average effect sizes, they nevertheless remained significant. Twenty-two studies were imputed into the BAS meta-analysis, reducing the average effect size from a small effect size, g = −.16, to a significant, albeit very small effect, g = −.06, 95% CI [−.12; −.01], p = .03. Ninety-two studies were imputed into the BIS meta-analysis, reducing the effect size from a large effect, g = 1.11, to a medium one, g = .67, 95% CI [.58; .76]. Thus, a robust significant effect remained with regards to BAS and BIS, though there is a possibility of some underreported findings, particularly with

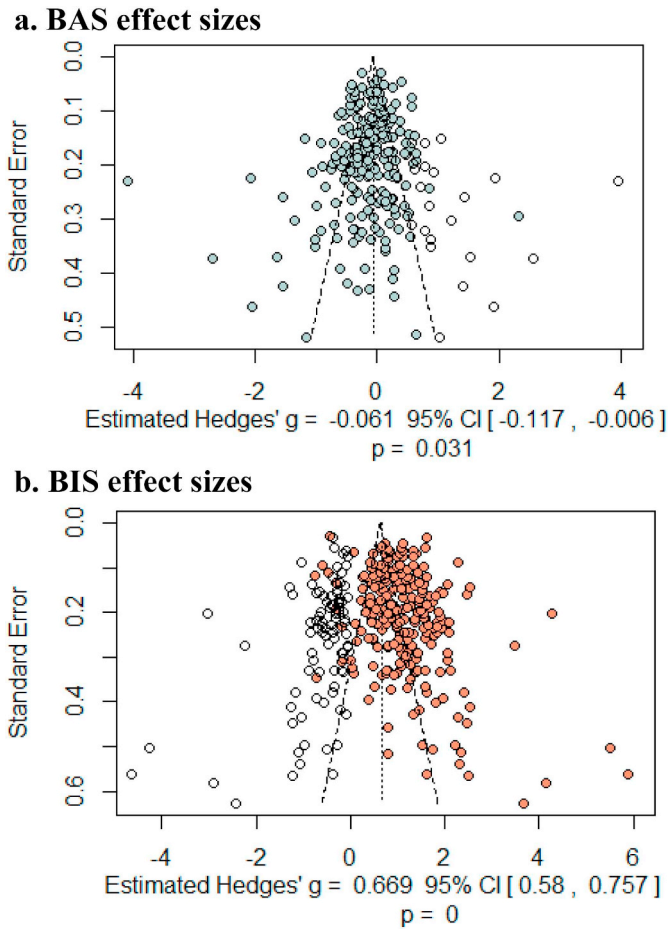

Fig. 4. (a–b) Funnel plots of BAS and BIS effect sizes following the trim-and-fill procedure.

Table 5b
Clinical status-by-disorder for BIS.

Moderator	Total			Depression			Anxiety		
	k	Hedges' *g*	*95% CI*	*k*	Hedges' *g*	95% CI	*k*	Hedges' *g*	*95% CI*
Main Effect	116	1.31	[1.18; 1.44]	68	1.35	[1.18; 1.52]	45	1.40	[1.14; 1.65]
Clinical status									
Episode/Acute	67	1.40	[1.24; 1.57]	37	1.64	[1.41; 1.87]	30	1.31	[.98; 1.65]
Outpatients	21	1.36	[1.07; 1.65]	9	1.32	[.96; 1.68]	10	1.48	[.99; 1.97]
Euthymic/Remission	28	1.01	[.80; 1.23]	22	.87	[.65; 1.10]	5	1.70	[1.18; 2.23]

Note. All effect sizes are significantly different from zero.

regard to BIS.

4.3. Discussion

The findings of the analyses performed in Study 1 tell a story that becomes richer at higher resolutions. First, BAS was found to have a small, negative relationship with pathology across depression and anxiety, whereas BIS has a large, positive one. Then, an interaction with disorder type was observed. On one hand, BAS was found to have a small, negative relationship with depression, whereas its relationship with anxiety was found to be trivial, albeit significant. On the other hand, BIS showed large, positive effects across disorder clusters, that were larger for anxiety than for depression. Finally, a series of moderator analyses found clinical status to interact with disorder type in predicting effect sizes. For depression, effect sizes grew larger as participants' clinical state became more severe. For anxiety, no such moderating effect was observed.

Taken together, these findings suggest a set of interrelated trends. In terms of reinforcement sensitivity, BAS was a small, negative predictor of pathology that was more closely related to depression. BIS, on the other hand, was found to be a large, shared predictor of depression and anxiety that was more strongly related with anxiety. Thus, BAS was confirmed to be a discriminating factor as theorized (Alloy et al., 2016; Stange et al., 2013). Similarly, BIS effects were consistent with both the current RST framework of affective pathology that frames BIS as a shared, neuroticism-like factor (Bijttebier et al., 2009; Caspi et al., 2014; Corr & Cooper, 2016; Jackson, 2009; Knyazev, Slobodskaya, & Wilson, 2004; Slobodskaya, 2007; Smillie, Jackson, & Dalgleish, 2006; Smits & Boeck, 2006) and the older framework that more closely related it to anxiety (Bijttebier et al., 2009). Furthermore, depression was consistent with the joint system hypothesis wherein severest pathology entailed a combination of low BAS functioning and high BIS functioning (Corr, 2001; Eddington et al., 2012).

Finally, differences were found between depression and anxiety regarding their interactions with clinical state. Depression showed significantly larger relationships with both BAS and BIS when participants in the clinical group were undergoing an episode than when participants were euthymic. Anxiety, on the other hand, was found to have similar effect sizes across clinical states. Thus, when compared to healthy controls, participants with current depression showed larger differences in reinforcement sensitivity than did their peers in remission. Participants with anxiety, on the other hand, showed levels of reinforcement sensitivity that had similar sizes of difference from healthy controls, regardless of symptom severity.

Study 1 was limited by only quantifying bivariate relationships between reinforcement sensitivity, depression and anxiety. Importantly, despite the high comorbidity rates between them (Watson, 2009), depression and anxiety were estimated separately. Thus, their shared variance was only indirectly estimated via robust variance estimation. A more exact estimate of the reinforcement sensitivity's relationship with depression and anxiety would directly control for covariance between the disorder clusters as well.

5. Study 2

In order to account for high levels of covariance between depression and anxiety, Study 2 aimed to simultaneously estimate each relationship between appetitive sensitivity (i.e., BAS), aversive sensitivity (i.e., BIS), depression and anxiety. This was done using a meta-analytic structural equation modeling approach (MASEM; Hunter & Schmidt, 2004).

5.1. Data analysis plan

The MASEM was performed in three steps (M. W.-L. Cheung & Chan, 2005; Hagger, Chan, Protogerou, & Chatzisarantis, 2016; Riketta, 2008; Viswesvaran & Ones, 1995). First, we assembled a matrix consisting of the weighted average of every bivariate correlation. Because all articles included in this study reported correlational data, no effect transformation was necessary. As in Study 1, each weighted average was calculated using a random effects meta-analysis, weighted as a function of sample size (Borenstein et al., 2009). Second, we performed a path analysis using the assembled average correlation matrix as input using a maximum likelihood estimator. The sum of all studies' sample sizes was used as the sample size in the model. This practice is preferred over alternatives (e.g., average sample size) in order to increase the sensitivity of significance tests (M. W.-L. Cheung & Chan, 2005). Because all objects in the matrix were derived from standardized correlations, variances in the path model were constrained to one. In so doing, all possible loadings were able to be included in the initial model, while having it remain eligible for goodness of fit statistics (see Nohe, Meier, Sonntag, & Michel, 2014). Finally, we considered alternate models in order to distill the most parsimonious one. This was done by systematically removing relationships between elements and comparing the new model's fit statistics to those of the original. In the event of model equivalence, the new model with more degrees of freedom was retained as the more parsimonious of the two. In the event of model non-equivalence, the one with the better fit was retained.

The χ^2 test was calculated as an initial measure of goodness of fit, and was used as the measure of relative fit when comparing alternate models. However, due to the χ^2 statistic's overestimation of lack of model fit, particularly among studies with a large sample size (Bollen, 1989; Browne & Cudeck, 1993), we reviewed additional indices of fit as well. Recommended cutoff scores (Hu & Bentler, 1999; Kline, 2015) were: comparative fit index (CFI) above .95, root mean square error of approximation index (RMSEA) below .06 and standardized root means residual (SRMR) below .08. Analyses were performed using R version 3.6.2 (R Core Team, 2017). The meta-analysis was performed using 'meta' version 4.10-0 (Schwarzer, 2007) and path analysis using 'lavaan' version 0.5-23.1097 (Rosseel, 2012).

5.2. Results

5.2.1. Description of studies

One hundred and fifty-six effect sizes were calculated, based on 39 distinct samples (k) drawn from 39 articles that were published between 1997 and 2018 (see Table 6). The total sample size (N) for this study was 13,572, with samples ranging from 20 to 2725 participants (M = 348, SD = 455.38). The mean age centered around young adults (M = 25.90, SD = 9.10, range = 16.0 – 47.6). Twenty-eight samples offered data already available in the published article, whereas 11 samples entailed unpublished data.

Importantly, constraints placed on study selection required correlations to be reported between depression and anxiety symptom severity, as well as punishment and reward sensitivity. No clinical-healthy comparison studies offered every correlation between these four elements. Thus, this analysis includes only studies with healthy participants.

5.2.2. Path analysis

First, we performed a series of meta-analyses on the correlations between each element in the path analysis (Table 7). The sizes and directions of the correlations between reinforcement sensitivity, depression and anxiety were consistent with those found among healthy participants in Study 1. The large correlation between depression and anxiety (r = .65, p < .001) was consistent with other findings (Jacobson & Newman, 2017), and the trivial, nonsignificant correlation between BAS and BIS (r = .01, p = .90) was consistent with their theorized orthogonal relationship (Corr, 2008).

We then performed a path analysis using the above correlations as an input matrix. The initial model estimated covariance between BAS and BIS, and each loaded on both depression and anxiety. Covariance

Table 6
Studies on the inter-relationships between reinforcement sensitivity, depression and anxiety (Study 2).

ID_Text	N	Published data?	RST Scales	BAS-depression	BAS-anxiety	BIS-depression	BIS-Anxiety	BAS-BIS	Depression-Anxiety
Albrecht, Staiger, Hall, Kambouropoulos, and Best (2016)	204	Y	BIS/BAS	−.02	.10	.27	.30	−.14	.58
Ak et al. (2012)	55	N	TCI	−.14	−.10	.27	.10	.80	.65
Atkinson, Sharp, Schmitz, and Yaroslavsky (2012)	448	N	BIS/BAS	−.04	−.10	.28	.22	.10	.71
Beevers and Meyer (2002)	171	Y	BIS/BAS	−.21	.07	.20	.24	.11	.27
Brook and Willoughby (2016)	1132	N	BIS/BAS	−.27	.07	.41	.42	−.04	.37
Brunborg et al. (2010)	61	Y	BIS/BAS	−.02	.10	.02	.54	.03	.41
Brunborg et al. (2010)	204	Y	BIS/BAS	−.06	−.12	.27	.30	−.14	.58
Ceschi, Hearn, Billieux, and Van der Linden (2011)	122	Y	SPSRQ	−.21	.07	.44	.35	.14	.29
Chen et al. (2011)	556	Y	TCI; BIS/BAS	−.09	.17	.46	.37	−.16	.63
Choi et al. (2014)	21	N	BIS/BAS; TCI	.28	.33	.58	.59	−.10	.31
Dennis and Chen (2007)	67	Y	BIS/BAS-BAS	.14	.36	.02	.52	−.07	.55
Fayazi and Hasani (2017)	453	Y	J-5	−.09	−.03	.09	.17	.17	.43
Feil and Hasking (2008)	161	Y	BIS/BAS	−.09	.01	.21	.23	.05	.57
Gawęda and Kokoszka (2014)	161	Y	TCI	−.11	−.13	.54	.47	.17	.57
Goncalves and Cloninger (2010)	595	Y	TCI	−.11	.09	.43	.43	−.34	.65
Hamill, Pickett, Amsbaugh, and Aho (2015)	467	Y	BIS/BAS	−.01	−.01	.35	.35	.06	.67
Harnett, Reid, Loxton, and Lee (2016)	452	Y	J-5	−.15	−.08	.16	.17	−.04	.58
Hundt et al. (2007)	285	Y	BIS/BAS; SPSRQ	.01	.09	.32	.21	.08	.33
Hundt et al. (2013)	293	Y	BIS/BAS	−.14	−.06	.28	.31	.02	.49
Izadpanah, Schumacher, and Barnow (2017)	274	Y	ARES	−.16	.02	.43	.31	−.23	.55
Jiménez-Murcia et al. (2015)	50	N	TCI	.12	−.02	.41	.35	−.41	.59
Jorm et al. (1998)	2725	N	BIS/BAS	.03	−.29	.29	.23	.20	.77
Jylhä and Isometsä (2006)	184	Y	TCI-R	−.06	.10	.58	.57	−.44	.74
	163	Y	TCI-R	−.20	−.03	.49	.52	−.38	.71
Katz and Yovel (2018)	512	N	BIS/BAS	−.25	−.10	.34	.32	.02	.60
Liao et al. (2017)	87	N	TPQ	−.09	−.14	.40	.41	.04	.48
Lu et al. (2012)	184	Y	TCI	−.17	−.13	.35	.24	−.12	.53
Lyvers, Karantonis, Edwards, and Thorberg (2016)	86	Y	SPSRQ	−.13	−.09	.37	.34	.18	.73
Manfredi et al. (2011)	307	Y	TPQ	.03	−.13	.39	.31	−.28	.53
Markarian, Pickett, Deveson, and Kanona (2013)	459	Y	BIS/BAS	.08	.34	.21	.23	.01	.67
Matsudaira and Kitamura (2006)	541	Y	TCI	.01	.05	.26	.44	−.27	.49
Park et al. (2013)	201	Y	BIS/BAS	−.09	.02	.38	.19	.09	.55
Rubinart, Fornieles, and Deus (2017)	20	N	TCI	.08	−.16	.17	.13	−.54	.73
Sanders and Abaied (2015)	170	Y	BIS/BAS	−.09	.07	.40	.28	−.14	−.33
Scott-Parker, Watson, King, and Hyde (2012)	761	Y	SPSRQ	.05	.12	.43	.39	.08	.70
Rodríguez-Testal et al. (2016)	287	Y	SRQ	−.34	.09	.43	.42	.14	.66
Tanaka, Sakamoto, Kijima, and Kitamura (1998)	223	Y	TCI	.15	.16	.48	.36	−.23	.64
Taubitz et al. (2015)	497	N	BIS/BAS	−.12	−.19	.36	.56	.07	.73
Wichelns, Renna, and Mennin (2016)	115	Y	BIS/BAS	.11	.14	.30	.02	.93	.78

Note. ARES: Action Regulating Emotion Systems scale, BIS/BAS: Behavioral Inhibition System/Behavioral Activation scale, J-5: Jackson 5, SPSRQ: Sensitivity to Punishment, Sensitivity to Reward Questionnaire, TCI: Temperament and Character Inventory, TCI-R: Revised TCI.

for depression and anxiety was estimated as well (see Fig. 5a). Results showed that reward and punishment sensitivity did not covary ($\beta = .01$, $p = .54$, 95% CI [−.01; .02]), whereas depression and anxiety shared a large covariance ($\beta = .64$, $p < .001$, 95% CI [.63; .65]). Reward sensitivity only loaded significantly on depression ($\beta = -.07$, $p < .001$, 95% CI [−.08; −.05]) but not on anxiety ($\beta = .01$, $p = .21$, 95% CI [−.01; .03]). Punishment sensitivity, on the other hand, loaded similarly on both anxiety ($\beta = .35$, $p < .001$, 95% CI [.33; .37]) and depression ($\beta = .37$, $p < .001$, 95% CI [.35; .38]).

Next, we considered alternative models (see Table 8). First, we removed the covariance between BAS and BIS from the model (path *a* in Fig. 5a), as per their theorized orthogonality (Corr, 2008; Gray & McNaughton, 2000). A comparison of goodness of fit found the two models to be equivalent, $\Delta\chi^2(1) = .37$, $p = .54$. Thus, the second model was retained as the more parsimonious of the two. Next, we considered removing paths between BAS and the psychopathology elements, due to their small effect sizes. In a third model, BAS did not load on depression (path *b* in Fig. 5a). That model proved to be a significantly worse fit than the second, $\Delta\chi^2(1) = 60.27$, $p < .001$, and was therefore rejected. Thus, the loading of BAS on depression was maintained. Finally, we considered a fourth model with the loading of BAS on anxiety removed (path *c* in Fig. 5a). When compared to the second model, the fourth model was an equally good fit and thus

Table 7
Meta analysis of correlations between reinforcement sensitivity, depression and anxiety.

Variables	*r*	95% CI	*p* value
BAS-BIS	.01	−.08; .09	.90
BAS-Depression	−.07	−.10; −.03	.001
BAS-Anxiety	.01	−.03; .05	.55
BIS-Depression	.37	.32;.41	< .0001
BIS-Anxiety	.35	.30; .39	< .0001
Depression-Anxiety	.65	.57; .73	< .0001

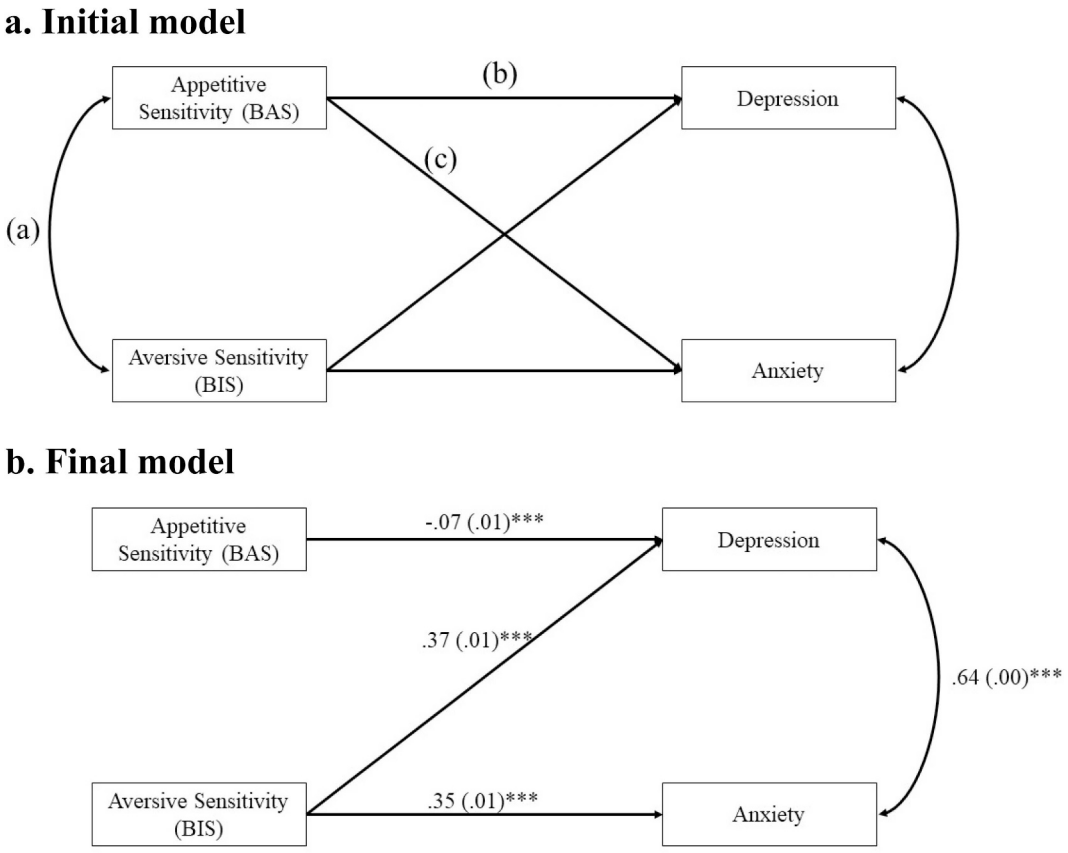

Fig. 5. (a–b) Path models of reinforcement sensitivity predicting depression and anxiety.

maintained as the most parsimonious, $\Delta\chi^2(1) = 1.55$, $p = .21$. All other paths led to significantly worse-fitting models when removed ($ps < .001$).

In the final model (see Fig. 5b), BAS loaded on depression with a very small effect size ($\beta = -.07$, $p < .001$, 95% CI [−.09; −.06]). BIS loaded with a small effect on both anxiety ($\beta = .35$, $p < .001$, 95% CI [.33; .36]) and depression ($\beta = .37$, $p < .001$, 95% CI [.35; .38]). Depression and anxiety shared a moderate portion of their variance ($\beta = .64$, $p < .001$, 95% CI [.63; .65]). Altogether, this model was consistent with the predictions laid out in Hypothesis 2 that predicted a negative relationship between BAS and depression (Hypothesis 2a) which would be larger than that of BAS and anxiety (Hypothesis 2b). Furthermore, we expected a positive relationship between BIS and depression (Hypothesis 2c) as well as BIS and anxiety (Hypothesis 2d).

Results of the χ^2 test indicated a bad fit for the final model, $\chi^2(6) = 182.84$, $p < .0001$. Other indices, however, indicated a good fit: CFI = .982, RMSEA = .047, 95% CI [.041,.053], SRMR = .070. In light of the large sample of participants in this analysis, we concluded that the fit indices indicate a good fit to the final model.

Table 8
Summary of model fit statistics for the alternative path models.

Model	χ^2	*df*	CFI	RMSEA	RMSEA 90% CI	SRMR
First model	180.92	4	.982	.057	.050 – .064	.070
Second model	181.29	5	.982	.051	.045 – .057	.070
Third model	241.56	6	.976	.054	.048 – .060	.073
Fourth model	182.84	6	.982	.047	.041 – .053	.070

Note. CFI = comparative fit index; RMSEA = root-mean-square error of approximation; CI = confidence interval; SRMR = Standardized root mean square residual.

5.3. Discussion

Study 2 aimed to simultaneously estimate the effects of BAS and BIS on both depression and anxiety while controlling for covariance between depression and anxiety. Doing so affirmed the general relationships between reinforcement sensitivity, depression and anxiety that were observed in Study 1. As in Study 1, punishment sensitivity was a shared predictor of both depression and anxiety (Bijttebier et al., 2009; Smillie et al., 2006). Similarly, reward sensitivity impacted only depression and not anxiety (Alloy & Abramson, 2010; Bijttebier et al., 2009; Trew, 2011), with the joint effects of hyposensitive BAS and hypersensitive BIS predicting depression together (Corr, 2001; Eddington et al., 2012). As opposed to Study 1, BIS did not have a stronger relationship with anxiety than it did with depression. Rather, the relationships of the BIS with anxiety and depression were virtually the same. Similarly, the theorized independence of the BAS and BIS (Gray & McNaughton, 2000) and the high covariance between depression and anxiety (Jacobson & Newman, 2017) were confirmed in the final model.

Importantly, Studies 1 and 2 differ in that Study 1 includes clinical-healthy comparisons, whereas Study 2 includes only non-clinical participants. Thus, the role of clinical state in this model remains unclear. Furthermore, as seen in Study 1, effects derived from correlations taken among non-clinical samples are smaller than standardized mean differences taken from clinical samples (see Figs. 2a,b and 3a,b). This may explain why the effect sizes in Study 2 were smaller overall. On one hand, these smaller effect sizes may impact the robustness of the final structural equation model among smaller samples. On the other hand, it is notable that the relationships between reinforcement sensitivity depression and anxiety were confirmed despite smaller effect sizes.

6. General discussion

The relationships between reinforcement sensitivity (Corr & McNaughton, 2008; Gray, 1970, 1987; Gray & McNaughton, 2000) depression and anxiety have been widely studied and brought together through influential theoretical narratives (e.g., Alloy et al., 2016; Kimbrel, 2008; McNaughton & Corr, 2008; Trew, 2011; Zinbarg & Yoon, 2008). However, until now, there has been no attempt to summarize the literature quantitatively or to systematically consider factors that may moderate these relationships across studies. To address this need, two complementary sets of meta-analyses were performed to summarize the cross-sectional relationships between reinforcement sensitivity, depression and anxiety, and to trace the extent to which these relationships differ between depression and anxiety, or between different levels of clinical severity. In the first study, a broad range of bivariate relationships between reward sensitivity, punishment sensitivity, depression, and anxiety were summarized and examined for moderators. In the second study, stricter inclusion criteria were set to allow for a meta-analytic structural equation model (MASEM) that directly controlled for the comorbidity between depression and anxiety.

Across studies and meta-analytic methodologies, certain findings consistently emerged. Sensitivity to aversive stimuli, or BIS sensitivity, was found to play a common role in the form of large, positive relationships with both depression and anxiety. Sensitivity to appetitive stimuli, or BAS sensitivity, discriminated between depression and anxiety. A small, negative relationship was observed between BAS and depression, and a significantly smaller, trivial-to-nonsignificant relationship was observed with anxiety. Clinical characteristics of the sample interacted with disorder cluster to moderate effect sizes as well. Clinical diagnosis and greater clinical severity (i.e., Acute/Episode vs Euthymic/Remission) predicted larger effect sizes, but only for depression. However, they did not moderate effect sizes for anxiety.

When brought together, these findings are generally consistent with clinical models of RST, particularly with the joint subsystems hypothesis (Carver, Johnson, & Joormann, 2008; Corr, 2002; Eddington et al., 2012; Kimbrel, Mitchell, Hundt, Robertson, & Nelson-Gray, 2012). According to this theoretical approach, under certain conditions, the two subsystems may antagonize or facilitate each other. Thus, clinically relevant phenomena are best understood in the context of both reinforcement sensitivities' effects on affect and behavior. In depression, for example, BAS hyposensitivity and BIS hypersensitivity may coalesce to inhibit goal pursuit (Trew, 2011). In the current meta-analysis, the results are consistent with models wherein a hypersensitive BIS predicts pathology in general, but a hyposensitive BAS makes the general pathology depressogenic (Knyazev & Wilson, 2004; Watson, 2009; Watson, Clark, et al., 1995). Indeed, hyposensitive BAS may only be pathological insofar as it is in the presence of a hypersensitive BIS (Hundt et al., 2007). Anxiety, on the other hand, would be most likely in a case of hypersensitive BIS, with a trivial relationship with BAS sensitivity, if at all (Bijttebier et al., 2009; Hollon, 2019; cf. Corr, 2002). However, it is worth noting that most studies of the joint subsystem hypothesis were performed within normative levels of reinforcement sensitivity (Bijttebier et al., 2009). For example, in externalizing symptoms such as substance use and ADHD, the joint subsystem hypothesis only has partial support (Coplan, Wilson, Frohlick, & Zelenski, 2006; Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Knyazev & Wilson, 2004). Further research is required to directly assess whether of one form of reinforcement sensitivity will impact the extent to which the other one predicts psychopathology (e.g., Harnett et al., 2013).

The distinctive roles of a common factor BIS and discriminatory BAS parallel other nosological models of pathology, most closely the tripartite model of depression and anxiety (Anderson & Hope, 2008; Watson et al., 1995; Watson, Clark, et al., 1995). The tripartite model first posits a general distress dimension shared among these disorders. Then, it suggests that there are disorder-specific factors as well, with anhedonia uniquely predicting depression and somatic arousal uniquely predicting anxiety. It has been suggested elsewhere that punishment sensitivity may parallel the shared general distress dimension and that reward hyposensitivity may parallel anhedonia (Shankman & Klein, 2003; Zinbarg & Yoon, 2008). The current findings are consistent with such a claim and highlight the role of the BIS as a possible common factor behind the substantial comorbidity between depression and anxiety (e.g., Kessler et al., 2005). Furthermore, the current findings are consistent with other hierarchical models such as the Hierarchical Taxonomy of Psychopathology (HiTOP; Conway et al., 2019; Kotov et al., 2017; Widiger et al., 2018). In the HiTOP, disorders of distress (e.g., major depressive disorder, general anxiety disorder) and disorders of fear (e.g., specific phobia, obsessive compulsive disorder) are predicted by factors common to all internalizing disorders, as well as disorder-specific factors. The high rates of BIS across disorder clusters make it a candidate for being a higher-ordered, common factor, and the discriminatory role of low BAS may identify it as a depression-specific factor.

In summary, the current findings represent a call for greater theoretical work on the interplay between reward sensitivity and punishment sensitivity. Clinical theories that focus on positively valenced constructs such as positive emotionality, reward sensitivity, reward motivation, and extraversion may be complemented with closer study of their negatively valenced counterparts (e.g., Carl, Soskin, Kerns, & Barlow, 2013; Khazanov & Ruscio, 2016; Millgram, Joormann, Huppert, Lampert, & Tamir, 2019; Naragon-Gainey & Watson, 2014; Naragon-Gainey, Watson, & Markon, 2009; Watson, Stasik, Ellickson-Larew, & Stanton, 2015). The BAS dysregulation model, for example, has accurately identified the central role that the BAS plays in longitudinally predicting fluctuations in mania and depression (Alloy, Bender, Wagner, Abramson, & Urosevic, 2009; Urosević et al., 2008). However, it has been found that some aspects of bipolar disorder's course, such as progression to bipolar I disorder, are only predicted by the BAS in the presence of a high BIS (Alloy et al., 2012). Based on the present findings, similar interactions are likely to be present for depression as well, and accounting for them has been found to improve the fit of predictive models based on reinforcement sensitivity (Gershuny & Sher, 1998). Indeed, cross-sectional and temporal models of depression, general anxiety disorder and social phobia account for the majority of variance when they include reinforcement sensitivity, positive affect and negative affect (Brown, 2007; Brown et al., 1998). However, that is not always the case (Jorm et al., 2000).

The current findings also indicate ways through which reinforcement sensitivity and symptom severity may interrelate. Ample evidence supports the emergence of dysregulated levels of reinforcement sensitivity prior to the development of psychopathology (Alloy et al., 2008; Carver & White, 1994; Meyer, Johnson, & Carver, 1999; Meyer & Hofmann, 2005; Urosević et al., 2008). However, different models predict different levels of reciprocity to this relationship. Some models interpret reinforcement sensitivity as a stable trait temperament that serves as a diathesis to the future development and maintenance of affective psychopathology (Klein et al., 2011; Kotov et al., 2010; Naragon-Gainey, Gallagher, & Brown, 2013). Others posit more dynamic, reciprocal models wherein temperaments such as reinforcement sensitivity function as a combination of diathetic personality traits alongside pathology-induced "personality states" incurred during acute episodes (Brown, 2007; Clark, Vittengl, Kraft, & Jarrett, 2003; Cole, Martin, & Steiger, 2005; Kendall et al., 2015).

In Study 1, effect sizes were compared between diagnosed-healthy comparison studies, based on current clinical severity. For depression, participants in a current depressive episode (i.e., *Acute/Episode*) showed larger differences from healthy controls than did participants with lifetime depression who were currently euthymic (i.e., *Euthymic/Remission*). For anxiety, no such differences were observed based on whether participants currently suffered from a clinical anxiety disorder (i.e., *Acute/Episode*) or from a previous one that is now at subclinical levels (i.e., *Euthymic/Remission*). This interaction between disorder

cluster and clinical state may indicate that reinforcement sensitivity has varied relationships with symptom severity, depending on the disorder. For depression, it seems that reinforcement sensitivity is best construed according to the dynamic models (Brown, 2007; Clark et al., 2003). This may reflect the episodic nature of depression, which is designated by discrete periods of clinical severity (American Psychiatric Association, 2013). The relationship between reinforcement sensitivity and anxiety, on the other hand, was not moderated by clinical state. As opposed to depression, anxiety was most consistent with models that emphasize the stability of trait reinforcement sensitivity across clinical states (Klein et al., 2011). The observed pattern of relationships between RST and anxiety was also consistent with current conceptualizations of anxiety that approach it as a continuum ranging from calmness to high arousal, as opposed to a set of discrete states (Clark, Cuthbert, Lewis-Fernández, Narrow, & Reed, 2017; Siddaway, Taylor, & Wood, 2018; Vautier & Pohl, 2009). As opposed to depression, where different clinical states led to categorically different effect sizes, levels of reinforcement sensitivity for anxiety were revealed to have a much larger overlap across clinical states. Thus, the present findings strongly indicate that the models used to understand the nature of reinforcement sensitivity's relationship with psychopathology may best be developed in a disorder-specific fashion.

Finally, the current findings also support contemporary therapeutic trends in developing interventions for both negatively and positively valenced basic processes that underlie affective disorders. The Unified Protocol for Treatment (Barlow et al., 2010; Carl, Gallagher, Sauer-Zavala, Bentley, & Barlow, 2014; Griffith et al., 2010), for example, was originally developed to reduce the deleterious effects of neuroticism across disorders (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014; Griffith et al., 2010). Recent augmentations to the Unified Protocol have aimed to impact positive emotionality as well (Carl, Gallagher, & Barlow, 2018). Indeed, recent calls have been made for a new wave of cognitive behavioral interventions that target transdiagnostic basic processes in addition to those specific to particular disorders (Hofmann & Hayes, 2019). The current meta-analyses emphasize the need for transdiagnostic interventions for punishment sensitivity, such as distress tolerance and acceptance-based exercises (Katz, Breznitz, & Yovel, 2019; Zvolensky, Vujanovic, Bernstein, & Leyro, 2010), alongside disorder-specific modules that promote rewarding experiences, such as behavioral activation (Cuijpers, van Straten, & Warmerdam, 2007; Hofmann & Hayes, 2019).

Despite the overall similarity in the conclusions derived from both studies, certain inconsistencies are worth noting. First, the effects in Study 2 were smaller in size than those observed in Study 1. This was perhaps a result of the fact that Study 2 only drew effects from healthy populations. Indeed, the ranges of effect sizes observed in Study 2 were similar to those observed among healthy participants in Study 1. It is also worth noting that the role of BIS differed somewhat between the two studies. In Study 1, BIS was found to have a larger relationship with anxiety than with depression. This difference was not observed in Study 2. It is unlikely that this is due to a difference in statistical power, as the weighted average correlations between BIS-anxiety and BIS-depression in this study were nearly equal (r = .35 vs .34, respectively; see Fig. 5b). It is also unlikely that this incongruity is a result of differences in the studies' populations. As seen in Study 1, depression and anxiety actually had weighted means that were further apart among healthy groups (g = .72 vs 1.12, respectively) than among clinical groups (g = 1.23 vs 1.39, respectively). Thus, the large positive role of BIS across anxiety and depression may be considered robust across meta-analytic studies, but it remains a question for future study whether it has a larger relationship with anxiety, and if so, under what conditions.

6.1. Limitations and future directions

The present findings should be evaluated in light of certain qualifications. First, the effects included in the present studies were cross-sectional. This limits our ability to extract etiological conclusions, which are inherently longitudinal (Brown, 2007). Clinical state's role as a moderator, for example, provides an indirect, between-subject indication that reinforcement sensitivity functions at different levels depending on depression severity. Longitudinal studies (e.g., Alloy et al., 2012), however, may more directly estimate these questions by accounting for within-subject variance as well. Currently, such studies (e.g., Goekoop, De Winter, & Goekoop, 2011) are too limited in number to provide the statistical power necessary for a meaningful meta-analysis. Future studies may utilize repeated measures of reinforcement sensitivity and symptom severity to better understand the role of reinforcement sensitivity as a predictor of etiology and course of depressive and anxiety disorders (Alloy et al., 2016).

Second, it is worth noting that reinforcement sensitivity was assessed in all studies included in the present meta-analyses using self-report measures, as was symptom severity among non-diagnosed populations. This raises both theoretical and methodological issues. On a theoretical level, RST was originally based in the link between biology and behavior (Corr, 2008; Gray, 1987), and self-report is thus an indirect proxy of the intended constructs. Methodologically, this also raises a concern for shared method effects (Eid, Lischetzke, Nussbeck, & Trierweiler, 2003). Indeed, some RST measures themselves even contain items that reference clinically relevant phenomena. The BIS subscale of the BIS/BAS (Carver & White, 1994), for example, references worry (e.g., *I worry about making mistakes*), a basic process closely linked with anxiety (Watkins, 2008). It is worth noting that group comparisons between clinical and non-clinical populations used different methods – psychiatric diagnosis and self-reported reinforcement sensitivity. Nevertheless, they showed larger effect sizes than correlations in non-diagnosed populations that perhaps benefited from this type of shared method variance. Furthermore, the use of self-report reinforcement sensitivity ensured that only measures with prior validation would be included in the systematic review. However, assessment of reinforcement sensitivity was only done through self-report methods. As such, some findings may have been partially impacted by other factors associated with clinical severity, such as limited insight, current mood and response style (Chmielewski & Watson, 2009; Klein et al., 2011; Watson, 2004). Future meta-analyses may account for these concerns by including behavioral (e.g., Millgram et al., 2019; Treadway et al., 2012) and biological measures (e.g., DelDonno et al., 2015) in their assessments of reinforcement sensitivity. These measures are more theoretically aligned with the biological-behavioral foundation of RST (Gray, 1970, 1987) and are also less susceptible to spurious shared-method covariance and biased responses.

Third, the majority of reinforcement sensitivity measures included in the present meta-analyses (Ball & Zuckerman, 1990; Carver & White, 1994; Cloninger, 1991, 1994; MacAndrew & Steele, 1991; Torrubia, Ávila, Moltó, & Caseras, 2001) were developed under the original version of RST (Gray, 1970, 1987). In 2000, a major revision was undertaken that significantly realigned the theoretical structure of punishment processing (Bijttebier et al., 2009; Corr, 2008; Gray & McNaughton, 2000). The novel Fight/Flight/Freeze system (FFFS) became responsible for the processing of aversive stimuli, while the revised BIS (r-BIS) became responsible for detection of threat and reduction of goal conflicts (Bijttebier et al., 2009; Corr, 2008). While self-report measures have been developed directly for the revised RST (Corr & Cooper, 2016; Jackson, 2009), they were released too recently to have supplanted more traditional measures of RST such as the BIS/BAS (Carver & White, 1994). Comparison studies have found that BAS measures from the original RST are closely related to measures of BAS in the revised RST, and measures of the original BIS correlate with measures of both the revised BIS and the revised FFFS – particularly the Flight and Freeze subscales of FFFS measures (Jackson, 2009; Krupić, Corr, Ručević, Križanić, & Gračanin, 2016; Torrubia et al., 2008). Thus, the current meta-analyses are best understood as summaries of the relationship between reward sensitivity, punishment sensitivity,

depression and anxiety. These basic processes are already the subjects of numerous lines of clinical research (Insel et al., 2010). However, future studies that utilize measures based on revised RST (e.g., J-5; Jackson, 2009) will be better able to explore which of the revised subsystems are more closely linked with which disorder.

Similarly, because the current meta-analyses consider the broad constructs of reward sensitivity and punishment sensitivity, they do not consider potential differences among each sensitivity's subtypes (Corr, 2013; Zald & Treadway, 2017). Indeed, reward sensitivity and punishment sensitivity are multifaceted, evolving constructs with multiple subtypes (Insel et al., 2010; Krupić, Gračanin, & Corr, 2016). These subtypes may have unique clinical ramifications both within reward sensitivity (e.g., reward responsiveness vs. fun seeking; Taubitz, Pedersen, & Larson, 2015) and punishment sensitivity (e.g., loss aversion vs. punishment responsiveness; Sokol-Hessner & Rutledge, 2019). Future reviews may examine the extent to which the varied subtypes of reward sensitivity and punishment sensitivity carry meaningful difference, whether assessed through the subscales of self-report measures, or through varied physiological and behavioral paradigms (e.g., physiological response to anticipatory vs consummatory pleasure; Corr, 2013; Insel et al., 2010; Nusslock & Alloy, 2017).

Additionally, future studies may benefit from a closer resolution of clinical phenomena on the level of disorder or even symptom. Indeed, it is possible that the relatively larger variances observed in the anxiety cluster reflect a real heterogeneity between the different anxiety disorders. Low positive affect, for example, has been found to be uniquely associated with social phobia (Naragon-Gainey et al., 2009; Watson, Clark, & Tellegen, 1988) and post-traumatic stress disorder (Nawijn et al., 2015), as compared to other anxiety disorders. This may reflect disorder-specific deficiencies of reward processing (Anderson & Hope, 2008). The current meta-analysis, however, did not have an adequate number of studies that represented each disorder to assess such a possibility. Similarly, low-arousal anxiety disorders and high-arousal fear disorders are theorized to have different relationships with the subsystems of revised RST (Perkins, Kemp, & Corr, 2007) and have been found to load on separate factors within internalizing disorders (Eaton et al., 2013). Additionally, different symptoms within the same disorder may carry different relationships with reinforcement sensitivity as well (Watson, 2009). For example, it is possible that BAS hyposensitivity relates specifically to symptoms unique to depression, such as anhedonia, whereas BIS hypersensitivity is more closely connected to symptoms that appear in multiple disorders, such as disturbed sleep (Borsboom & Cramer, 2013; Hundt et al., 2007). Future studies may quantify such differences by examining whether reinforcement sensitivity's relationships vary among different disorders within a single cluster, or among different symptoms within a single disorder.

Finally, the effects included in the meta-analysis are also impacted by decisions made within their component studies. For example, participants' data were only included in the meta-analysis if the relevant effect sizes were made available. While 244 manuscripts were identified as potentially having data which could have been included, data for only 20 were furnished upon request. While the impact of publication bias was estimated to be small, the current meta-analyses should be evaluated in light of the significant number of effect sizes which were not included. Similarly, participants were only included in the meta-analysis if they were approved by their component study's inclusion and exclusion criteria. Thus, exclusion criteria common to clinical studies (e.g., psychosis) were likely *de facto* applied to the current meta-analyses as well.

7. Conclusion

The present meta-analyses summarize the current state of RST, depression and anxiety, and signal new avenues of future inquiry. Ultimately, we found robust support for a hierarchical approach to reinforcement sensitivity (e.g., Zinbarg & Yoon, 2008) that may be consistent with the joint subsystem hypothesis (e.g., Corr, 2002). Punishment hypersensitivity was found to be a higher-order, shared factor for both depression and anxiety, whereas reward hyposensitivity was specific to depression. Furthermore, we found that clinical diagnosis and more severe levels of depression predicted larger effect sizes for both reward sensitivity and punishment sensitivity, which was not found with anxiety. Attention to the ways such differences interact, covary and change may improve how the nature of affective pathology is understood, and ultimately, how more effective, personalized treatments may be developed.

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Appendix A. Supplementary data

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