Journal of Affective Disorders 301 (2022) 117–129

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Reinforcement sensitivity predicts affective psychopathology via emotion regulation: Cross-sectional, longitudinal and quasi-experimental evidence

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ARTICLE INFO

Keywords:
Reinforcement Sensitivity
Reappraisal
Rumination
Internalizing Disorders
Longitudinal Study

ABSTRACT

The current article presents a model wherein reinforcement sensitivity predicts depression and anxiety via trait preferences for concomitant emotion regulation strategies. In Study 1 ($N = 593$), BAS sensitivity positively predicted reappraisal and BIS sensitivity negatively predicted it. Reappraisal then negatively predicted depression. BIS sensitivity also predicted rumination, which predicted both depression and anxiety. Study 2a confirmed the model developed in Study 1 with an independent sample ($N = 513$) and examined the relationships longitudinally. While the cross-sectional relationships were generally maintained, reinforcement sensitivity did not predict reappraisal. In Study 2b, participants ($N = 218$) were assessed a third time one year later, at the onset of the COVID-19 pandemic. During this stressful time, BAS sensitivity did longitudinally predict reappraisal. These studies highlight the role of emotion regulation in mediating the relationship between reinforcement sensitivity and affective pathology, particularly during times of high stress.

According to Reinforcement Sensitivity Theory (RST; Gray, 1970, 1987), the dual abilities to process positive and negative stimuli are governed by independent neurological systems. Positively-valenced experiences are processed by the Behavioral Approach System (BAS) while negative ones are processed by the Behavioral Inhibition System (BIS; Corr, 2008). In 2000, RST was revised to include a third system, concerned with resolving approach-avoidance conflicts (Gray and McNaughton, 2000). Since then, the BAS and BIS from the original RST have been understood best as proxies for general appetitive and aversive processing (Bijttebier et al., 2009; Depue and Collins, 1999; Insel et al., 2010; Manczak et al., 2014). Each system's sensitivity, and the interactions between them, serve as the biological basis for an individual's affect, motivation, behavior, and subjective experience (Corr, 2002).

Temperamentally abnormal levels of (original) BAS and BIS sensitivities influence the psychological disorders to which an individual is most susceptible (Bijttebier et al., 2009; Clark, 2005; Hollon, 2019; Insel et al., 2010; Kotov et al., 2017). BIS hypersensitivity is a common factor across depression and anxiety, positively predicting them both to a large degree. BAS sensitivity, on the other hand, discriminates between them, negatively predicting only depression to a small degree (Alloy et al., 2016; Griffith et al., 2010; Katz et al., 2020; Zinbarg and Yoon, 2008). Research programs often focus on how each reinforcement system directly impacts clinically relevant phenomena, such as the strong, phenomenologically overlapping relationship between BAS hypo-sensitivity and depressive anhedonia (e.g., Nusslock and Alloy, 2017). Indeed, it has been argued that reinforcement sensitivities and their related psychiatric symptoms overlap so closely that the only systematic difference between them is that symptoms take place over discrete periods while trait reinforcement sensitivity has no such temporal limit (e.g., DeYoung et al., 2020). In a given moment, however, extreme reinforcement sensitivity traits and their concomitant affective symptoms may be indistinguishable (Shankman and Klein, 2003; Van Meter and Youngstrom, 2015).

While trait reinforcement sensitivity does closely relate to affective symptoms, it influences other clinically relevant individual differences as well. Reinforcement sensitivity serves as the biological basis for traits with broad implications, such as BAS sensitivity's relationship with extraversion and BIS sensitivity's relationship with neuroticism (Corr and McNaughton, 2008; Keiser and Ross, 2011), and likely has implications for other cognitive processes as well. Indeed, multiple reviews on the role of reinforcement sensitivity in affective psychopathology have pointed out the need for further research identifying cognitive mediators of their relationship (e.g., Alloy et al., 2005; Bijttebier et al., 2009; Katz et al., 2020). This call, however, has only sporadically been answered (e.g., Dennis, 2007; Hundt et al., 2013).

Some findings indicate that reinforcement sensitivity's impact upon

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https://doi.org/10.1016/j.jad.2022.01.017
Received 19 July 2021; Received in revised form 9 November 2021; Accepted 2 January 2022
Available online 11 January 2022

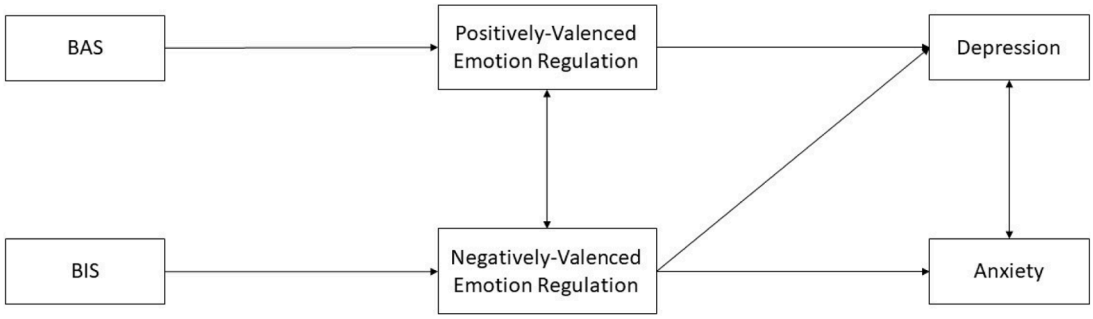


Fig. 1. A general emotion regulation model of reinforcement sensitivity, emotion regulation, and affective psychopathology.

mood dynamics may be a possible cognitive pathway towards affective psychopathology. Temperamental positive and negative affectivity impact the desirability of different emotions, along with preferences for the regulatory strategies deployed to achieve or avoid them (Hughes et al., 2020; Rottenberg, 2017). Higher levels of neuroticism, a trait closely related to BIS hypersensitivity (Corr and McNaughton, 2008; Keiser and Ross, 2011), predict more acutely felt negative emotions along with the greater use of maladaptive regulatory strategies such as rumination (Barańczuk, 2019; Ng and Diener, 2009). These patterns are also found when BIS hypersensitivity is measured directly (Gray, 1987; Leen-Feldner et al., 2004; Tull et al., 2010), especially at times of actively stressful situations (Heponiemi et al., 2003; Rackoff and Newman, 2020). On the other hand, higher levels of BAS sensitivity and extraversion predict a greater preference for most positive emotions (Khazanov et al., 2020), along with coping styles that pursue such emotions, such as positive reappraisal (Barańczuk, 2019; Carl et al., 2013; Gable et al., 2000). Thus, each reinforcement sensitivity may predict trait preferences for emotion regulation strategies, but with opposite valences (see Fig. 1). Greater BAS sensitivity predicts more positively-valenced strategies such as reappraisal (Llewellyn et al., 2013) while greater BIS sensitivity predicts more negatively-valenced strategies, such as rumination (Keune et al., 2012; Randles et al., 2010).

Some studies have taken steps to examine whether the relationship between reinforcement sensitivity and emotion regulation may serve as a mechanism through which diathetic levels of reinforcement sensitivity impact affective pathology. In one cross-sectional study (O'Connor et al., 2014), BAS sensitivity positively predicted reappraisal while BIS sensitivity negatively predicted it. Reappraisal, in turn, negatively predicted social anxiety to a small degree. Elsewhere, BIS sensitivity was found to indirectly impact depression via greater use of maladaptive strategies such as rumination, but not via lesser use of adaptive ones like positive reappraisal (Li et al., 2015). Negative emotion regulation (i.e., rumination, catastrophizing, etc.) has been found to mediate the relationship between BIS sensitivity and anxiety as well (Tortella-Feliu et al., 2010).

Taken together, these studies provide evidence for the role of emotion regulation as a mediator between reinforcement sensitivity and affective pathology. However, such studies rarely account for the high comorbidity between anxiety and depression in their models (Jacobson and Newman, 2017). Most only assess one disorder (e.g., O'Connor et al., 2014) or analyze depression and anxiety symptoms in separate models (e.g., Hundt et al., 2013; cf. Markarian et al., 2013). Doing so, however, blurs the line between specific and transdiagnostic processes (Kazdin, 2007; Rottenberg, 2017) – an issue particularly relevant to emotion regulation (Nolen-Hoeksema et al., 2008) and to reinforcement sensitivity (Zinbarg and Yoon, 2008). When bivariate relationships are examined, reappraisal negatively predicts both depression and anxiety, while rumination positively predicts them both (Aldao et al., 2010). However, when analyzed together, repetitive negative thinking is indeed found to be transdiagnostic while positive reappraisal is uniquely related to depressive symptoms (Everaert and Joormann, 2019). Similarly, BAS hyposensitivity may be found in anxiety as well as depression, but discriminates between the two after directly accounting for their high comorbidity (Jacobson and Newman, 2017; Katz et al., 2020; Khazanov and Ruscio, 2016; Kotov et al., 2010). Furthermore, most studies that examine reinforcement sensitivity, emotion regulation, and affective psychopathology use cross-sectional data. Doing so, however, produced potentially biased results and obscures the possibility of bidirectional effects (Maxwell and Cole, 2007). Thus, effective modeling of the relationships between reinforcement sensitivity, emotion regulation, and affective pathology should include measures of both depression and anxiety to parse out transdiagnostic processes from disorder-specific ones, and use repeated measures to estimate directional, less biased effects.

Furthermore, each reinforcement sensitivity may have a different relationship with different regulatory strategies. Many studies on clinical reinforcement sensitivity theory emotion regulation use broad definitions such as "emotion regulation difficulties" (e.g., Markarian et al., 2013; Tull et al., 2010; Wang et al., 2019). Others compare between formally dissimilar strategies such as cognitive reappraisal and behavioral suppression (e.g., Llewellyn et al., 2013; O'Connor et al., 2014). For this reason, many studies of emotion regulation in the context of clinical individual differences find it necessary to incorporate comparable strategies such as reappraisal and rumination, that are cognitive in nature, but with opposingly valenced interpretative biases (e.g., Denson et al., 2012; Everaert and Joormann, 2019; Millgram et al., 2019; Ray et al., 2005). This approach, however, has not been applied to the context of reinforcement sensitivity and affective pathology. No study has traced the relationship between reinforcement sensitivity and affective pathology along specific, comparable, cognitive emotion regulation strategies. For example, both positive reappraisal and rumination are formally similar emotion regulation strategies in that they recognize the content of a negative stimulus and cognitively interact with it. However, they differ in the valence of the interaction, with positive reappraisal attempting to interpret the stimulus's content more positively, while rumination entails repetitive, critical review of the stimulus (McRae et al., 2012; Sheppes et al., 2014). Furthermore, no study has attempted to identify which of these mechanisms may be transdiagnostic, and which may discriminate between depression and anxiety.

1. Current studies

The current investigation examines the role of emotion regulation as a mechanism through which BAS and BIS sensitivities impact depression and anxiety. According to both theory (Zinbarg and Yoon, 2008) and empirical findings (Katz et al., 2020), BIS sensitivity plays a large, transdiagnostic role in predicting both depression and anxiety. BAS sensitivity, on the other hand, plays a smaller, discriminatory role, only negatively predicting depression. Furthermore, each sensitivity may be mediated by a concomitant emotion regulation strategy, with BAS sensitivity primarily predicting positively-valenced coping strategies (i.

e., reappraisal) and BIS sensitivity predicting negatively-valenced coping strategies (i.e., rumination; Ng and Diener, 2009). These coping strategies may then predict psychopathology in a similar pattern to their concomitant reinforcement sensitivities. Rumination, like BIS sensitivity, transdiagnostically predicts both depression and anxiety while reappraisal, like BAS sensitivity, predicts only depression (see Everaert and Joormann, 2019).

The current model of reinforcement sensitivity, emotion regulation, and affective pathology was evaluated in three steps. First, in Study 1, we used an exploratory approach to evaluate the viability of the initial, cross-sectional model, to generate a more parsimonious version, and then to compare its fit to alternative explanatory models. The data-driven nature of the first study's model-building process, however, introduced the possibility of errors due to capitalization on chance (MacCallum et al., 1992). To address this issue, in Study 2a, we examined the model's fit in an independent sample, and, importantly, examined the effects longitudinally. Finally, in Study 2b, we examined the utility of the emotion regulation model of reinforcement sensitivity for predicting increases in depression and anxiety during a time of stress for participants – the COVID-19 pandemic. Taken together, these studies highlight the role of emotion regulation as a mediator between reinforcement sensitivity and depression and anxiety, particularly in times of stress, when such coping strategies are most necessary.

Materials, data, and analysis syntax for all studies can be found at https://osf.io/5t27r/.

2. Study 1

The first study established a model of reinforcement sensitivity, emotion regulation, and affective pathology. It did so by exploring the extent to which reinforcement sensitivity's relationship with depression and anxiety could be indirectly related via positive and negative cognitive emotion regulation. Next, using a data-based approach, the model was simplified to select for a most parsimonious representation. Finally, alternative exploratory models were considered, in terms of the extent to which they may better fit the data.

2.1. Method

2.1.1. Participants

In order to include participants representing a wide range of geographic origin, socioeconomic status, and individual differences, an Internet-based community sample was assembled. Participants were recruited through the Prolific Academic Platform, which has elsewhere been found to deliver higher rates of honest, attentive responses that reliably reproduce known effects from laboratory assessments (Palan et al., 2018, Peer et al., 2017). Only those who have previously completed at least 50 tasks on the platform, with an approval rate of at least 95% were invited to join. Additionally, only those who marked themselves as primarily English-speakers were eligible for the study.

Five hundred and ninety-three participants (male = 314, female = 275, n/a or other = 4) were included in the study. Participants' ages ranged widely ($M = 38.29$, $SD = 13.38$, range = 18 - 83). Most participants originated in the United States (82.0%) with the other participants originating in Canada (15.7%) or the United Kingdom (2.4%). Additional data on participants' race/ethnicity, education, and employment status may be found in supplementary material S1.

2.2. Measures

2.2.1. Behavioral inhibition scale/behavioral approach scale (BIS/BAS; Carver & White, 1994)

The BIS/BAS is a widespread measure of reinforcement sensitivity. The BAS subscale assesses motivation and savoring of positive experiences (e.g., "I go out of my way to get things I want."). The BIS subscale assesses avoidance of and sensitivity to negative experiences (e.g., "I feel worried when I think I have done poorly at something important."). Both the BAS (Cronbach's $\alpha = 0.84$) and the BIS ($\alpha = 0.85$) showed good reliability in the current study.

2.2.2. Emotion regulation questionnaire – reappraisal (ERQ-R; Gross and John, 2003)

The ERQ-R is a widely used six-item subscale that assesses participants' preference to change their view of a situation to either feel more positive or less negative about it (e.g., "When I want to feel *more positive* emotion, I change the way I'm thinking about the situation."). The measure was found to have excellent reliability ($\alpha = 0.91$) in the current study.

2.2.3. Rumination-reflection questionnaire (RRQ; Trapnell and Campbell, 1999)

The RRQ is a 12-item scale that assesses participants' tendency to repetitively think about past events of theirs more often and more self-critically than they would like (e.g., "I tend to "ruminate" or dwell over things that happen to me for a really long time afterward."). The measure was found to have excellent reliability ($\alpha = 0.95$) in the current study.

2.2.4. Depression, anxiety and stress scale – depression and anxiety (DASS-D & DASS-A; Lovibond & Lovibond, 1995)

In the current study, participants were assessed using the measure's depression subscale – a seven-item assessment of sadness and anhedonia (e.g., "I couldn't seem to experience any positive feeling at all.") – and the anxiety subscale – a seven-item assessment of fear-related somatic arousal (e.g., "I felt I was close to panic."). In this study, both the depression subscale ($\alpha = 0.93$) and anxiety subscale ($\alpha = 0.86$) showed very good reliability.

2.2.5. Procedure

Participants completed the self-report measures related to reinforcement sensitivity, emotion regulation, and affective symptom severity, presented in a random order to minimize systematic carryover effects from the previous measure. The study was approved by the departmental Ethics Committee. Participants provided informed consent prior to participation in the study and were compensated consistent with Prolific's standard payment practices following completion of the study.

2.2.6. Data analytic procedure

Model generation and evaluation were performed in three steps. First, an indirect effects model was assessed wherein reinforcement sensitivity predicted affective pathology via emotion regulation. The model was then improved by removing paths which would render the model more parsimonious without negatively impacting fit. Parsimony was operationally defined as increased degrees of freedom, and difference in fit was defined as a significant test of χ^2 difference (Barrett, 2007). Finally, we examined theoretically plausible alternative models for the data (see Kline, 2015). These models' goodness of fit measures were examined in order to assess their viability as alternative models.

Analysis followed Kline's (2015) recommendations for SEM path analysis, using a maximum likelihood estimator. All variables were treated as observed and all parameter values were standardized (Rosseel, 2012). Goodness-of-fit measures were considered holistically, with recommended thresholds (Hu and Bentler, 1999; Kline, 2015) referenced: (non)significance of a χ^2 test of fit, comparative fit index (CFI) above 0.95, standardized root means residual (SRMR) below 0.08, and root mean square error of approximation index (RMSEA) below 0.05 for exact fit and above 0.10 for poor fit.

Analyses were performed using R version 4.0.1 (R Core Team, 2017). Descriptive statistics were calculated using the 'psych' package, version 1.9.12.31 (Revelle, 2017) and 'qwraps2', version 0.4.2 (DeWitt, 2019). Path analyses and goodness of fit calculations were performed using 'lavaan' version 0.6–6 (Rosseel, 2012).

Table 1
Summary of goodness-of-fit statistics for models.

	χ^2 test	CFI	RMSEA [90% CI]	SRMR
Study 1				
1. Original model–Reinforcement sensitivity -> Emotion regulation-> Affective symptoms	$\chi^2(4) = 5.86, p = .21$	.998	.028 [.000; 0.073]	.017
2. Simplified model–Reinforcement sensitivity -> Emotion regulation-> Affective symptoms	$\chi^2(6) = 7.20, p = .30$	.999	.018 [.000; 0.059]	.022
3. Alternative model–Affective symptoms -> Reinforcement sensitivity -> Emotion Regulation	$\chi^2(7) = 111.79, p < .001$	.893	.159 [.134; 0.185]	.076
4. Alternative model–Emotion regulation -> Affective symptoms -> Reinforcement sensitivity	$\chi^2(6) = 330.71, p < .001$	.668	.302 [.275; 0.330]	.120
Study 2a				
5. Confirmation of Model 2	$\chi^2(6) = 42.43, p < .001$	.959	.109 [.079; 0.141]	.058
6. Longitudinal model (T1-T2)–Reinforcement sensitivity -> Emotion regulation-> Affective symptoms	$\chi^2(14) = 25.89, p = .027$	.994	.043 [.008; 0.070]	.027
Study 3				
7. Longitudinal model (T2-T3)–Reinforcement sensitivity -> Emotion regulation-> Affective symptoms	$\chi^2(14) = 31.43, p = .005$	.986	.076 [.040; 0.111]	.047

2.3. Results

All measures' means, standard deviations, and intercorrelations can be found in the supplementary materials (S2). BIS sensitivity positively correlated with both depression ($r = 0.39$; all effects are significant at $p < .001$ unless noted otherwise) and anxiety ($r = 0.33$), while BAS sensitivity correlated only with depression ($r = -0.12, p = .003$), but not anxiety ($r = -0.04$, $p = .331$). Emotion regulation strategies, on the other hand, did not directly differentiate between disorder measures. Reappraisal negatively predicted both depression ($r = -0.27$) as well as anxiety ($r = -0.12$, $p = .003$), and rumination positively predicted both disorders ($rs = 0.52$ and 0.40, respectively).

2.3.1. Model development

We next performed a series of indirect effects models to test the hypothesis that reinforcement sensitivity impacts affective pathology via concomitant emotion regulation strategies. The first model entered included all possible relationships between reinforcement sensitivity and emotion regulation, and between emotion regulation and affective pathology (Fig. 2a). In keeping with their theoretical (Corr, 2008; Gray and McNaughton, 2000) and observed independence (Katz et al., 2020), BAS and BIS were not expected to covary. In light of the large positive relationship between depression and anxiety (Jacobson and Newman, 2017), and between rumination and reappraisal (Naragon-Gainey et al., 2017), the pairs of affective symptoms and emotion regulation strategies were allowed to covary. This model was found to have an excellent fit (see Table 1; Model 1).

To make the model more parsimonious, the two non-significant paths (i.e., a and b in Fig. 2a) were removed with no negative impact on fit, $\Delta\chi^2(2) = 1.40, p = .497$. The simplified model (i.e., Model 2), can be found in Fig. 2b. This final model was found to be an excellent fit for the data (see Table 1; Model 2).

The final model was consistent with the hypothesis that emotion regulation mediates the relationship between reinforcement sensitivity and affective pathology. BIS sensitivity predicted both reappraisal negatively to a small degree ($\beta = -0.20$) and rumination positively, to a large degree ($\beta = 0.70$). BAS sensitivity, on the other hand, only predicted reappraisal ($\beta = 0.26$). In turn, emotion regulation predicted psychopathology along similar patterns as reinforcement sensitivity. Rumination, a strategy that was only predicted by BIS sensitivity, predicted both depression ($\beta = 0.40$) and anxiety ($\beta = 0.49$). However, reappraisal, which was predicted by BAS sensitivity as well as BIS sensitivity, uniquely predicted depression ($\beta = -0.16$). These effects were observed when directly controlling for the depression's large covariance with anxiety ($\beta = 0.55$) and, to a smaller degree, reappraisal's negative covariance with rumination ($\beta = -0.08$, $p = .063$).

2.3.2. Examination of alternative models

Two alternative models were examined (S3a-S3b). In Model 3, symptom severity predicted reinforcement sensitivity (see Pinto-Meza et al., 2006; Takahashi et al., 2013), which in turn predicted emotion regulation (see O'Connor et al., 2014). In Model 4, emotion regulation predicted symptom severity (see Aldao et al., 2010), which in turn predicted reinforcement sensitivity (see Pinto-Meza et al., 2006; Takahashi et al., 2013). Paths between each set of variables were borrowed from the simplified model (i.e., Model 2), with directionality defined by the theoretical orientation of the alternative explanatory model. Thus, for example, in Model 3, depression predicted both BAS sensitivity and BIS sensitivity, but anxiety only predicted BIS sensitivity. Examination of fit indices revealed that neither alternative model (see Table 1; Models 3 and 4) explained the data better than the simplified model (see Table 1; Model 2). As such, Model 2, the simplified model, was retained as the only explanatory model that fit the data.

2.4. Discussion

The first study supports the hypothesized model of reinforcement sensitivity, emotion regulation, and affective pathology as the best explanatory model for the data. Consistent with previous research, it finds a large, transdiagnostic role of BIS sensitivity alongside a small, negative relationship only between BAS sensitivity and depression (Bijttebier et al., 2009; Katz et al., 2020; Zinbarg and Yoon, 2008). These relationships' indirect relationships via emotion regulation were also found to have proportionally similar effects. Rumination, a coping strategy only predicted by BIS sensitivity, predicted depression and anxiety to a similar, larger degree. Reappraisal, a coping strategy predicted by BIS sensitivity and, to a larger extent, BAS sensitivity, uniquely predicted depression, to a small degree. These relationships were observed after controlling for the large, positive covariance between depression and anxiety, as well as the small, negative covariance between reappraisal and rumination. Importantly, the RST-emotion regulation model was also found to be the only good fit for the data, as compared to alternative explanatory models.

Due to the exploratory, data-driven nature of the first study, the final model (i.e., Model 2) was susceptible to biases such as capitalization on chance characteristics of the data (MacCallum et al., 1992). Furthermore, the cross-sectional data potentially biases mediational models and do not allow for the assessment of each relationship's longitudinal effects (Hershberger and Marcoulides, 2013; Kline, 2015; Maxwell and Cole, 2007). Thus, an independent sample was necessary to assess the model's replicability, and longitudinal data was necessary to further establish the hypothesized directional relationships.

3. Study 2a

The second study examined the RST-emotion regulation-affective pathology model in an independent sample, at two points, six months apart. It was performed with three intended goals. First, we assessed the model's replicability in an independent sample. Second, we assessed the extent to which the cross-sectional relationships were also observed longitudinally, by returning to the same sample six months later.

3.1. Method

3.1.1. Participants

For the same reasons as in Study 1, an Internet-based community sample of five hundred and thirteen participants was recruited through the Prolific Academic Platform (male = 285, female = 224, n/a or other = 4). Participants' ages ranged widely ($M = 37.27$, $SD = 12.32$, range = 18 - 74). The majority of participants originated in the United Kingdom (45.22%) or United States (48.93%), with the remaining participants (5.85%) originating in other countries (i.e., Canada, Ireland, United Arab Emirates, Azerbaijan). As in Study 1, participants were included if English was their primary language and they had participated in at least 50 previous studies with successful participation rate above 95%. No participants in the current study were included in Study 1.

Participants in Study 2a were contacted for a second assessment six months later (see Procedure below). Among the original 513 participants, 348 (male = 149, female = 196, n/a or other = 3) returned for the second assessment (T2; 67.84%). This attrition rate was within the range of other longitudinal, Internet-based studies with only online communication (e.g., 36%−79%; Bajardi et al., 2014). Only participants who completed both assessments were able to be included in the longitudinal analysis. No differences were observed in most critical T1 measures between those who returned for T2 and those who did not ($ps > 0.075$; see S4). BIS sensitivity, however, was found to be significantly higher among those who returned ($M = 3.15$, $SD = 0.60$) than those who did not ($M = 3.02$, $SD = 0.58$), to a small degree, $t(511) = 2.30$, $p = .022$, $d = 0.22$. Additional data on participants' race/ethnicity, education, and employment status may be found in supplementary material S1.

3.1.2. Materials and procedure

Measure administration followed the same procedure as in Study 1, with the addition of a reaction-time task that was part of an unrelated, multi-study project, and was completed after the self-report questionnaires. Six months later, participants were contacted again and invited to complete a second assessment, consisting of the same measures as those in the first.

The questionnaires were the same as well, with similar levels of reliability. The BIS/BAS (Carver & White, 1994) showed very good reliability for the BAS (T1 & T2 αs = 0.87) and BIS (αs = 0.86 - 0.87) subscales. The ERQ-R (Gross and John, 2003) and RRQ (Trapnell and Campbell, 1999) showed excellent reliability (αs = 0.90 - 0.91 and 0.95 - 0.96, respectively). The depression and anxiety measures of the DASS (Lovibond & Lovibond, 1995) showed very good-to-excellent reliability (αs = 0.94 - 0.95 and 0.87, respectively).

3.1.3. Data analytic procedure

Analysis and model evaluation of the cross-sectional model followed the same procedures as those followed in Study 1. However, as opposed to Study 1, the current study was analyzed with an *a priori* model to be tested – the simplified model (i.e.,. Model 2). Thus, in light of the confirmatory nature of the study, only one model was examined.

Longitudinal effects were examined using a bidirectional, cross-lagged panel path analysis (Kline, 2015; Preacher, 2015). In such an analysis, each measure would be entered as a T1 predictor variable predicting each adjacent measure's T2 criterion variable, after controlling for the criterion variable at T1, and T2 covariates specified in the model. Thus, for example, we estimated the bidirectional longitudinal relationship between BIS sensitivity and rumination by estimating BIS sensitivity (T1)'s relationship with rumination (T2) and rumination (T1)'s relationship with BIS sensitivity (T2), while controlling for rumination and BIS sensitivity at T1 and the covariance between rumination (T2) and reappraisal (T2). To control for longitudinal effects which may have occurred prior to assessment, all variables at T1 were allowed to covary (see Kline 2015 and Preacher, 2015). All variables were treated as observed. Consistent with recommendations for hypothesis-testing using missing data (Graham, 2009), the full-information maximum likelihood (FIML) method was used to impute missing data. Goodness of fit was assessed using the same criteria as in Study 1.

3.2. Results

3.2.1. Cross-sectional model confirmation

All measures' means, standard deviations, and intercorrelations can be found in supplementary materials (S5). The simplified model's (i.e., Model 2) replicability was assessed using all participants who completed the T1 assessment. Overall, the model had adequate fit across most measures (see Table 1; Model 5 & Fig. 3). The preponderance of fit statistics indicated a very good fit, CFI = 0.957, SRMR = 0.061, with the RMSEA indicating a bad fit RMSEA = 0.111, 90% [.082; 0.143]. Thus, the model developed in Study 1 was judged to be a good fit overall for the second, independent sample tested in Study 2.

Results from the path analysis in Study 2a replicated the findings from Study 1′s indirect effects model and showed similarly sized relationships as well. Again, depression and anxiety strongly, positively covaried (β = 0.49), and rumination and reappraisal negatively covaried (β = −0.21). Reappraisal was negatively predicted by BIS sensitivity to a small degree (β = −0.12, $p = .002$), and positively predicted by BAS sensitivity to a larger degree (β = 0.43). Rumination was only predicted by BIS sensitivity, to a large degree (β = 0.66). Emotion regulation also predicted psychopathology along similar patterns with similar effect sizes as those observed in Study 1. Reappraisal, the strategy characterized more by BAS sensitivity than by BIS sensitivity, negatively predicted only depression, to a small degree (β = −0.13). Rumination, a strategy predicted by only BIS sensitivity, in turn predicted both depression (β = 0.52) and anxiety (β = 0.40). Due to the goodness of fit and similarity of effect sizes, we concluded that the simplified model (i.e., Model 2) was satisfactorily replicated in a second, independent sample (i.e., Model 5).

3.2.2. Assessment of longitudinal model

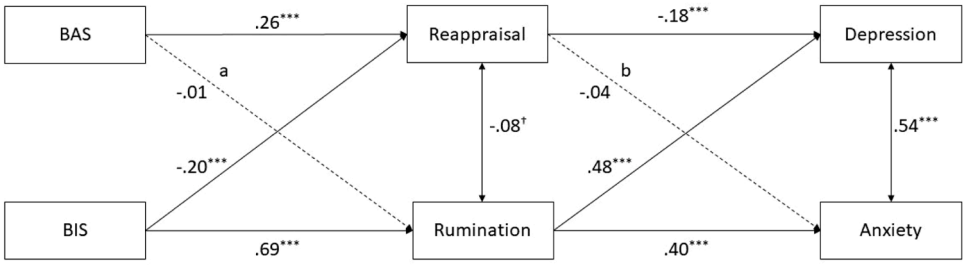
In order to assess the extent to which variables in the prior confirmed models (i.e., Models 2 and 5) longitudinally predict their criteria beyond autocorrelation, the model was assessed with a cross-lagged panel model applied to a half-longitudinal design (i.e., Model 6; Table 1 & Fig. 4). This model was found to be an excellent fit for the data, CFI = 0.998, SRMR = 0.018, RMSEA = 0.027, 90% CI [.000–0.051].

The previously observed cross-sectional relationships were generally observed longitudinally as well. BIS sensitivity (T1) loaded on rumination (T2; $\beta = 0.13$, $p = .001$) while rumination (T1) predicted BIS sensitivity (T2) as well ($\beta = 0.13$). Rumination (T1) then predicted both depression (T2; $\beta = 0.15$) and anxiety (T2; $\beta = 0.12$, $p = .007$). Contrary to expectations, reappraisal (T2) showed no significant relationship with either reinforcement sensitivity in either direction, beyond autocorrelation ($\beta = 0.63$). However, reappraisal (T1) still predicted depression (T2) to a small degree ($\beta = -0.07$, $p = .019$), but not vice versa ($\beta = -0.05$, $p = .284$). Finally, as observed in the cross-sectional model, significant covariances were observed between reappraisal (T2) and rumination (T2) ($\beta = -0.13$, $p = .018$), and between depression (T2) and anxiety (T2; $\beta = 0.54$). Taken together, most relationships observed in the cross-sectional model were observed longitudinally as well, though the expected relationship between reinforcement sensitivity and reappraisal was not observed longitudinally. Additionally, bidirectionality was indicated in the relationships between BIS sensitivity and rumination, and between rumination and depression.

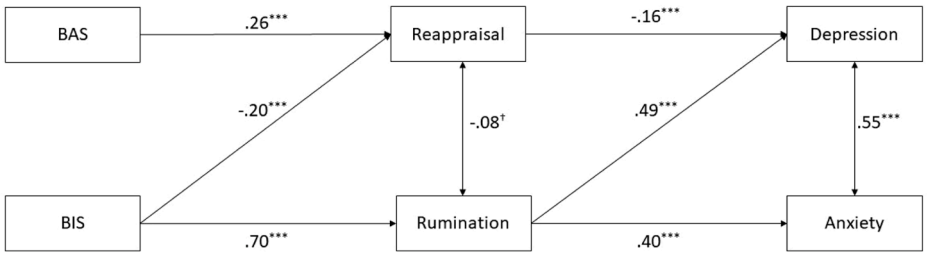
3.3. Discussion

The RST-emotion regulation-affective pathology model replicated with an independent sample using a cross-sectional design (i.e., Models 2 and 5). Furthermore, the longitudinal model (i.e., Model 6) fit the data excellently, and similar longitudinal relationships were observed as in

2a. Original model (i.e., Model 1)



2b. Simplified model (i.e., Model 2)



Figs. 2a-b. Mediation models and standardized path coefficients for relationship between reinforcement sensitivity, emotion regulation, and affective pathology in Study 1.

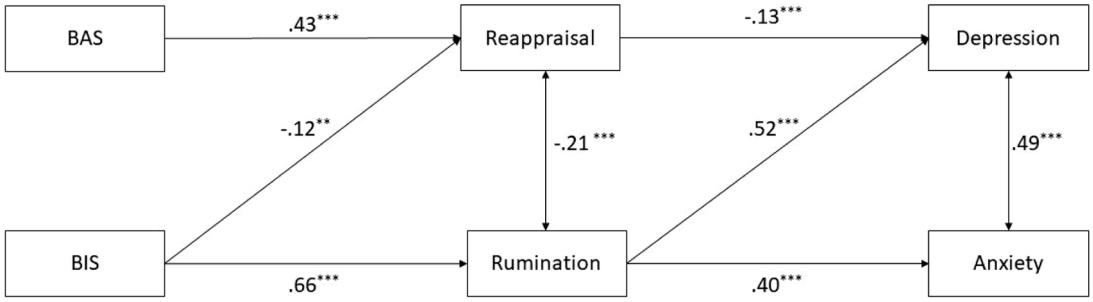


Fig. 3. Mediation model and standardized path coefficients for relationship between reinforcement sensitivity, emotion regulation, and affective pathology in Study 1 (i.e., Model 5).
Note. *** - significant at $p < .001$; ** - significant at $p < .01$

the cross-sectional model. Rumination predicted both depression and anxiety while reappraisal only predicted depression. However, neither reinforcement sensitivity predicted reappraisal beyond autocorrelation. This may be due to the fact that for most participants in the study, no major life stressors have occurred. Individual differences in emotion regulation habits are pronounced differently in times of stress, (Aldao et al., 2015; Bonanno and Burton, 2013; Heponiemi et al., 2003; Sheppes et al., 2014) and reinforcement sensitivity's clinical relevance may only be revealed following stress-inducing adverse life events (Clark, 2005; Kopala-Sibley et al., 2016; LeMoult, 2020). Thus, it remains possible that the hypothesized relationship between BAS sensitivity and reappraisal still occurs, but is dependent upon the introduction of stress for such individual differences to be expressed.

4. Study 2b

The temporal relationships in the RST-emotion regulation-affective pathology model may be dependent upon the presence of stress. To assess such a model, however, it would be necessary to assess participants before and after a group-level stressor would take place (Hammen, 2005; Ingram and Luxton, 2005; Kopala-Sibley et al., 2016). In the current study, this group-level stressor took the form of the COVID-19 (SARS-CoV-2). The pandemic was generally a time of significant, clinically relevant stress (Gruber et al., 2020; Hawes et al., 2021; Yarrington et al., 2021), and the current study's latter assessment was performed during a peak of daily deaths as a result of the pandemic (European Centre for Disease Prevention and Control, 2020; see Fig. 5). In doing so, the model's longitudinal relationships were examined under more stress-inducing conditions, while also applying a diathesis-stress framework to pandemic-related stress (Taquet et al., 2020).

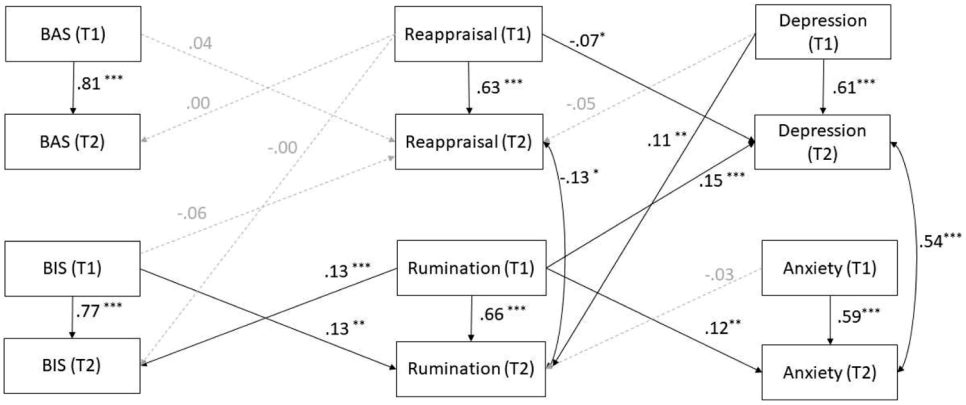


Fig. 4. Half-longitudinal cognitive coping model in Study 2a (i.e., Model 6).
Note. Covariance among T1 variables not pictured. *** - significant at $p < .001$; ** - significant at $p < .01$; * - significant at $p < .05$. Non-significant paths drawn with dotted lines in gray.

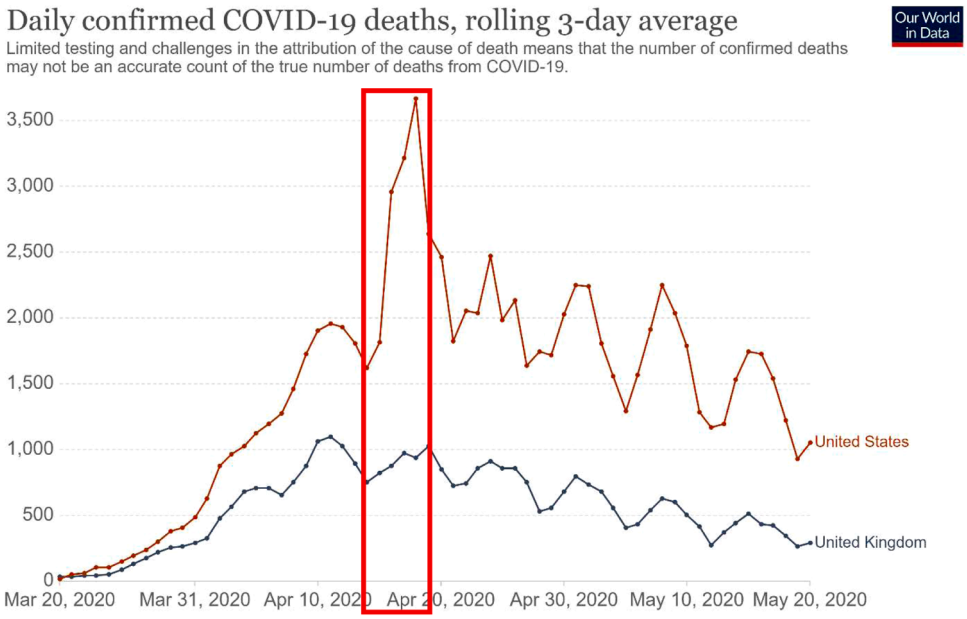


Fig. 5. Daily confirmed deaths of COVID-19 between March 20, 2020 and May 20, 2020.
Note. Figure retrieved from OurWorldInData.org/coronavirus, which visualized data reported by the European Center for Disease Control. The red rectangle indicates the dates in which the third assessment took place, between April 15 and April 20

4.1. Method

4.1.1. Participants

Participants in Study 2a were contacted exactly one year after their second assessment, to complete a third assessment during the COVID-19 pandemic (see Procedure below). Among the 348 who returned for the second assessment, 218 (62.93%) completed a third assessment (T3). As in the case of Study 2a, this attrition rate also fell within the range of Internet-based studies with similar designs (Bajardi et al., 2014).

The current study focused on the 218 participants (female = 118, male = 97, n/a or other = 3) who completed the latter assessment in Study 2a (i.e., T2) and the assessment during the COVID-19 pandemic (i.e., T3). Participants represented a wide range of ages ($M = 42.87$, $SD = 13.09$, range = 19 – 75) and resided mostly in the United States (46.57%), United Kingdom (46.12%), as well as other countries (i.e., Canada, Ireland; 7.31%). Additional data on participants' race/ethnicity, education, and employment status may be found in supplementary material S1.

Participants who returned for the third assessment were not significantly different from those who did not return in T2 levels of BIS sensitivity, reappraisal, or rumination ($ps > 0.09$). They were, however, lower in BAS sensitivity ($M = 0.2.73$, $SD = 0.50$ vs $M = 2.82$, $SD = 0.47$), $t(346) = 2.37$, $p = .02$, $d = 0.19$, depression ($M = 0.85$, $SD = 0.79$ vs $M = 1.06$, $SD = 0.62$), $t(346) = 2.99$, $p = .003$, $d = 0.30$, and anxiety ($M = 0.45$, $SD = 0.56$ vs $M = 0.64$, $SD = 0.56$), $t(346) = 2.42$, $p = .016$, $d = 0.34$, to a small degree (see S6). In light of these differences, the previous models examined in Study 2a (i.e., Models 5 and 6) were re-analyzed, only including those who returned for Study 2. No differences were observed in the models' conclusions (e.g., BAS T1 -> Reappraisal T2 changed from $\beta = 0.04$, $p = .349$ to $\beta = 0.09$, $p = .091$ and Reappraisal T1 -> Depression T2 changed from $\beta = -07$, $p = .014$ to $\beta = -0.10$, $p = 006$; see S7).

4.1.2. Materials

The measures used were the same as those included in Studies 1 and 2a and showed similar levels of reliability. The BIS/BAS (Carver & White, 1994) showed very good reliability for the BAS ($\alpha s = 0.88$) and BIS ($\alpha s = 0.88$ - 0.89) subscales. The ERQ-R (Gross and John, 2003)

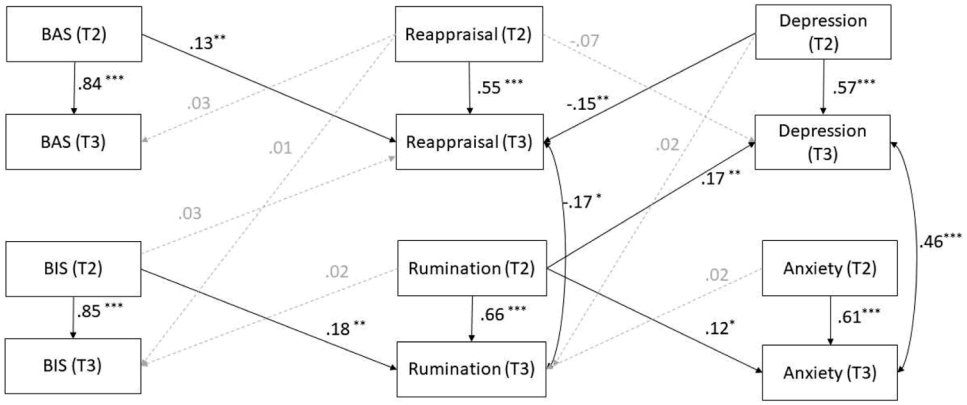


Fig. 6. Half-longitudinal mediation model and standardized path coefficients for relationship between reinforcement sensitivity, emotion regulation, and affective pathology in Study 3 (i.e., Model 7).
Note. Covariance among T2 variables not pictured. † - significant at $p < .10$; * - significant at $p < .05$; ** - significant at $p < .01$. Non-significant paths drawn with dotted lines in gray.

showed excellent reliability (αs = 0.91–0.92) as did the RRQ (Trapnell and Campbell, 1999; αs = 0.96). The depression subscale of the DASS (Lovibond & Lovibond, 1995) showed excellent reliability (αs = 0.94 - 0.95) and the anxiety subscale showed very good reliability (αs = 0.86 - 0.88).

4.1.3. COVID-19 stress items

In the assessment that took place during the COVID-19 pandemic (i.e., T3), participants also completed an ad-hoc questionnaire that assessed the extent to which the pandemic impacted their lives. This included the presence of pandemic-related environmental stressors (e.g., "became ill from possible or certain exposure to the coronavirus") in the participants' personal lives or in the life of somebody close to them. Additionally, participants were asked to rate on a scale of 0 (= none at all) to 100 (= a lot) the extent to which they experienced more loneliness and distress as a result of the pandemic.

4.1.4. Procedure

Exactly one year following the second assessment (i.e., T2; April 15–20 2019), participants were invited to complete a third assessment (i.e., T3; April 15–20, 2020) as well. This assessment took place during a peak of the COVID-19 pandemic in terms of daily deaths in their countries (European Centre for Disease Prevention and Control, 2020; see Fig. 5). Only participants who completed both initial assessments were eligible for the third assessment. All measures administered at the previous assessments were administered at the third one as well, with the addition of the ad-hoc COVID-19 stress items, which were administered after the key variables included in the RST-emotion regulation model.

4.1.5. Data analytic procedure

In order to examine the hypothesized model following the introduction of a group-level stressor, we included participants' assessments during the pandemic (i.e., T3) and their assessments exactly one year prior (i.e., T2). Analysis followed the same cross-lagged panel path analysis (Kline, 2015; Preacher, 2015) as was utilized in Study 2a (i.e., Model 6). Goodness of fit was assessed using the same criteria as in the previous studies and missing data was again imputed using the FIML method.

4.2. Results

4.2.1. Impact of pandemic

The COVID-19 pandemic introduced environmental stressors to the participants' lives. At the time of assessment (i.e., T3), most participants reported that the pandemic led to their self-isolation (63.3%), and many reported a reduction in income for either themselves or a person close to them (44.5%). Furthermore, 17.9% of respondents indicated that they or someone close to them had probably been exposed to COVID-19 and 11.0% indicated that either they or somebody close to them knew someone who had died from the disease. Participants also reported moderate levels of loneliness ($M = 42.33$, $SD = 32.74$) and stress ($M = 57.40$, $SD = 28.00$; both on a 0–100 scale) as a result of the COVID-19 pandemic.

4.2.2. Assessment of diathesis-stress model

All measures' means, standard deviations, and intercorrelations can be found in supplementary materials (S8). A half-longitudinal model was entered using the same specifications as those used in Study 2a (i.e., Model 6). This model (i.e., Model 7; Fig. 6) was found to be an excellent fit for the data on all measures of fit, CFI = 0.994, SRMR = 0.027, RMSEA = 0.043, 90% CI = [.008; 0.070].

While participants were undergoing a period of stress, BAS sensitivity (T2) longitudinally predicted reappraisal (T3; $\beta = 0.13$, $p = .010$), but not vice versa ($\beta = 0.03$, $p = .511$). No relationship was observed between BIS sensitivity and reappraisal in either direction ($\beta\langle |.03|, p\rangle .588$), nor did rumination predict BIS sensitivity ($\beta = 0.02$, $p = .730$). As in Study 2a, however, BIS sensitivity (T2) did predict rumination (T3; $\beta = 0.18$, $p = .002$). Thus, in the current model, BAS sensitivity only predicted reappraisal while BIS sensitivity only predicted rumination – and not vice versa.

Additionally, some differences were found in the relationship between emotion regulation and affective pathology as well. As in Study 2a, rumination (T2) predicted depression (T3; $\beta = 0.17$, $p = .003$) and anxiety (T3; $\beta = 0.12$, $p = .029$). However, unlike in the previous study, depression (T2) did not predict rumination (T3; $\beta = 0.02$, $p = .748$) and instead negatively predicted reappraisal (T3; $\beta = -0.15$, $p = .012$). Furthermore, reappraisal (T2) predicted depression (T3) with the same effect size as in the previous study, but due to a larger standard error, that relationship was not significant ($\beta = -0.07$, $p = .137$). Thus, in the current model, rumination longitudinally predicted depression and anxiety, and not vice versa. Additionally, depression longitudinally negatively predicted reappraisal, but reappraisal did not predict depression.

4.3. Discussion

The current study examined the hypothesized RST-emotion regulation-affective pathology model using assessments from before and during a group-level stressor, the beginning of the COVID-19 pandemic (Taquet et al., 2020). As observed in Study 2a, the model was found

to be an excellent fit for the longitudinal data. Furthermore, all relationships observed in the cross-sectional model were present: BAS sensitivity predicted reappraisal, BIS sensitivity predicted rumination, and rumination predicted depression and anxiety. These relationships were not observed in the opposite direction. On the other hand, depression longitudinally negatively predicted reappraisal, but not vice versa. Although the size of reappraisal's longitudinal prediction of depression was the same as that observed in Study 2a, it was not significant in the current study due to a larger standard error.

Taken together, this pattern was consistent with the size and specificity of each reinforcement sensitivity system. BIS sensitivity has been found to have a large, transdiagnostic impact on both depression and anxiety (Bijttebier et al., 2009; Katz et al., 2020; Zinbarg and Yoon, 2008). Here, BIS sensitivity predicted only rumination, which itself longitudinally predicted both depression and anxiety to a larger degree. BAS sensitivity, on the other hand, plays a discriminatory role in predicting psychopathology, predicting only depression but not anxiety (Alloy et al., 2016; Katz et al., 2020; Kotov et al., 2017). In the current study, BAS sensitivity predicted only reappraisal, which was also predicted by only depression. Thus, taken together, the findings of the current study show that a model that traces the links between reinforcement sensitivity, emotion regulation, and affective pathology fit the data well in the presence of a group-level stressor, in the form of the COVID-19 pandemic. The model traces the longitudinal relationships between reinforcement sensitivity, emotion regulation styles, and affective psychopathology, and shows how individual differences in BAS and BIS sensitivities may have implications for mental health at times of stress. Furthermore, it indicates a possible cyclical relationship between depression and emotion regulation (Rottenberg, 2017).

5. General discussion

The current studies found that reinforcement sensitivity predicts affective pathology via emotion regulation. Three studies tested this model and compared it against alternatives. In two independent samples, using a cross-sectional design, BAS sensitivity positively predicted reappraisal, while BIS sensitivity negatively predicted it. Reappraisal, in turn, negatively predicted only depression, to a small degree. BIS sensitivity, on the other hand, positively predicted rumination, which positively predicted both depression and anxiety to a medium degree.

These relationships were generally maintained longitudinally beyond autocorrelation as well, though not always in the same direction. Rumination's transdiagnostic positive relationship with depression and anxiety was observed in both studies, as was reappraisal's unique, negative relationship with depression. However, the relationship between emotion regulation and depression may be bidirectional, with depression positively predicting rumination in Study 2a and reappraisal in Study 2b. Similarly, while the prospective relationship between reappraisal and depression was of equal effect sizes in both studies, it was only significant in Study 2a as a result of differences in error variance. Rumination's relationship with anxiety, however, was consistently unidirectional, in both studies. The longitudinal relationships between reinforcement sensitivity and emotion regulation also differed based on the presence of stress. In Study 2a, when no group-level stressor occurred, BIS sensitivity showed a bidirectional relationship with rumination, while BAS sensitivity did not relate to reappraisal. In Study 2b, when the second assessment occurred during the COVID-19 pandemic BAS sensitivity prospectively predicted reappraisal and BIS sensitivity predicted rumination – but not vice versa.

Taken together, the current studies highlight the transdiagnostic role of BIS hypersensitivity and the discriminatory role of BAS hyposensitivity (see Bijttebier et al., 2009; Zinbarg and Yoon, 2008). These studies then build on the extant literature by pointing to emotion regulation as a mechanism that mediates these relationships, following similar patterns of shared and discriminant factors as those of reinforcement sensitivity. For example, BAS sensitivity has been shown to negatively predicts depression only to a small degree (Katz et al., 2020). Here, this discriminatory role occurs via trait reappraisal, that also only predicts depression, negatively and to a small degree. BIS sensitivity, on the other hand, has a large, positive, transdiagnostic relationship with both depression and anxiety (Katz et al., 2020; Zinbarg and Yoon, 2008). Here, this pattern of effects is observed for rumination. Thus, the complementary roles of BAS sensitivity and BIS sensitivity are themselves mediated by their concomitant emotion regulation strategies, reappraisal and rumination, respectively.

Reinforcement sensitivity, affective psychopathology, and emotion regulation may relate through the common temperamental mechanisms. Reinforcement sensitivity dysregulation may lead to core deficits in positive or negative affectivity, that in turn may underlie pathological emotion dysregulation present in depression and anxiety (Clark, 2005; Dennis, 2007; Gross and Jazaieri, 2014; Stange et al., 2013). Indeed, individuals with depression have been found to show greater use of regulatory strategies that upregulate sadness (Joormann and Stanton, 2016), and lesser use of strategies that upregulate happiness (Carl et al., 2014). High levels of negative affectivity predict depression and anxiety via general negative emotion regulation strategies, including rumination (Tortella-Feliu et al., 2010). Additionally, trait goal orientations (Higgins, 2012) also relate to reinforcement sensitivity, emotion regulation, and affective psychopathology. BAS sensitivity predicts a promotion orientation, a preference for reward-approaching goals. BIS sensitivity, on the other hand, predicts a prevention orientation, a preference for punishment avoidance (High and Solomon, 2014). These goal orientations may then be activated when choosing emotion regulation strategies (Millgram et al., 2020; Vanderlind et al., 2020). Positive reappraisal aims to maximize a positive emotional outcomes and is more consonant with a promotion orientation (McRae et al., 2012; Vishkin et al., 2020). Alternatively, rumination is a self-focused cognitive process often misused to avoid other distressing thoughts (Dickson et al., 2012; Eisma et al., 2013) in a way consistent with prevention goals. Importantly, each goal's failure also arouses emotions typical of their respective disorders. A promotion failure (i.e., non-reward) is associated with grieving and sadness (Idson et al., 2000), two emotions salient to depression (Rottenberg, 2017). Prevention failures (i.e., punishments), on the other hand, are associated with discomfort and distress (Idson et al., 2000), emotions present in both depression and anxiety (Watson, 2009).

The current studies also highlight the importance of both temperamental traits and situational context in explaining how individuals select among different emotion regulation strategies. Recent trends in emotion regulation research have emphasized individuals' autonomy in regulating towards emotions (Tamir et al., 2020) and in choosing some regulatory strategies over others (Sheppes et al., 2011). The current studies show that reinforcement sensitivity may influence regulatory preferences, and that this effect may be modified by situational context. Some individual differences that underlie emotion regulation preferences may only do so during times of stress – when emotion regulation is most needed (Carver and Connor-Smith, 2010), and differences in emotion regulation between healthy individuals and those with internalizing disorders are often more pronounced (Colombo et al., 2020; Ehring et al., 2010; Tan et al., 2012). This is true for more mundane periods of stress, such as an exam period for students (Millgram et al., 2019), or more dramatic ones, such as a natural disaster for the local population (Kopala-Sibley et al., 2016). The COVID-19 pandemic, a source of extreme stress on a global level (Ghebreyesus, 2020; Yarrington et al., 2021), served as a group-level stressor in the current study. The current results suggest that this stress played a critical role in the longitudinal relationship between BAS sensitivity and reappraisal (e.g., Sheppes et al., 2011). However, it is important to note that due to sample size limitations it was not possible to perform post-hoc analyses that could more directly compare effect sizes in the model between those who were suffering from high amounts of stress during assessment and those who were not. As such, future research should more directly

examine how reinforcement strategies may be chosen as a function of temperament and context in general (Carver and Connor-Smith, 2010), and as a function of reinforcement sensitivity and stress in particular (Hundt et al., 2007). This may be done using experimental designs in a laboratory context. For example, reinforcement sensitivity may be used to predict strategy selection during a spontaneous emotion regulation task (Katz et al., 2017). Alternatively, it is possible that emotion regulation may be more or less effective for individuals based on their underlying reinforcement sensitivities and the strategy selected (Higgins, 2005).

The current findings offer clinical ramifications as well. Reinforcement sensitivity dysregulation is often targeted as an indicator of affective pathology risk (Alloy et al., 2008; Brown and Barlow, 2009; Clark, 2005; Kotov et al., 2017; Zald and Treadway, 2017). However, temperamental reinforcement sensitivity is often quite stable, even in clinical contexts (Clark et al., 2003; Khazanov and Ruscio, 2016), and is only amenable to intervention to a small degree (Carl et al., 2014). Emotion regulation, on the other hand, is more malleable with targeted protocols (Carl et al., 2018; Renna et al., 2017) as well as nonspecific cognitive-behavioral therapies (Goldin et al., 2012; Wolitzky-Taylor et al., 2012). Clarifying the cognitive mechanisms of temperament's impact on affective pathology may be a necessary step towards better personalizing treatments to an individual's needs. Some success has been found, for example, in psychosocial interventions targeted to individuals with BAS hypersensitivity (Nusslock et al., 2009), who are at higher risk for developing bipolar disorders (Katz et al., 2021; Urošević et al., 2008). These advances may be applied to depression and anxiety as well (Cohen and DeRubeis, 2018; Eddington et al., 2015; Mennin et al., 2006). In so doing, a more integrated approach may be developed wherein dysregulated reinforcement sensitivity predicts individual risk for depression and anxiety, while intervention focuses on more malleable cognitive processes (Cohen and DeRubeis, 2018).

Furthermore, the final study highlights the need for diathesis-stress models, when identifying who is most at risk for increased affective pathology following disasters such as the COVID-19 pandemic (Gruber et al., 2020). In such disasters, populations are more likely to experience greater amounts of stress, but not necessarily a sustained increase in psychiatric symptoms (Goldmann and Galea, 2014). Rather, diathesis-stress models, which aim to identify individuals who are likely to suffer symptom increases based on temperamental risk, are preferred (Hammen, 2005; Monroe, 2008; Pizzagalli, 2014). It may thus be concluded that BAS hyposensitivity and BIS hypersensitivity are temperamental diatheses that place individuals at risk for maladaptive emotion regulation choices when faced with stress, which may itself lead to increases in psychopathology.

Finally, to compare emotion regulation strategies that were formally similar but different in valence, the current studies focused on reappraisal and rumination. However, a wide range of other strategies are available that may be impacted by reinforcement sensitivity as well. Distraction and acceptance deploy attention as a form of emotion regulation, either away from the situation or towards it wholistically (Sheppes et al., 2014). However, acceptance is putatively an adaptive strategy while distraction is considered maladaptive (Aldao et al., 2010). Some studies have linked these strategies to BIS sensitivity, which relates positively to distraction and negatively to acceptance (Llewellyn et al., 2016; Tull et al., 2010). Similarly, BAS sensitivity's close relationship with extraversion (Corr, 2008) suggests the possibility that greater BAS sensitivity could be associated with greater use of interpersonal forms of emotion regulation. A more fully realized model of reinforcement sensitivity, emotion regulation, and affective psychopathology will consider preferences for both the valence and form of strategy preferred.

5.1. Limitations and future directions

The current studies' findings should be evaluated while considering the strengths and limitations of their designs. Each study employed the same designs and measures, ensuring comparability between the original study, its replication, and longitudinal extensions. However, the current studies utilized self-report measures of reinforcement sensitivity. This is in keeping with much current work in the field that relies heavily upon self-report assessments of reinforcement sensitivity and relies upon it almost exclusively for assessments of cognitive processes (Alloy et al., 2016; Dennis, 2007; Hawes et al., 2021; Hundt et al., 2013). However, reinforcement sensitivity is a biobehavioral model (Corr, 2008) and as such, self-report assessments are an indirect proxy of the construct. Future research may incorporate behavioral measures (e.g., Effort Expenditure for Rewards Task; Treadway et al., 2009) or experimental designs (e.g., Trier Social Stress Test; Kirschbaum et al., 1993) to more directly assess the constructs of interest.

The current studies' Internet-based samples enabled a participant pool ranging more widely in demographics (Palan and Schitter, 2018) and clinical severity (Shapiro et al., 2013) than student convenience samples. However, participants were not screened for diagnosable levels of depression or anxiety (see Chandler et al., 2020). Reinforcement sensitivity and emotion regulation may function differently at such levels of symptom severity (Katz et al., 2020; Quigley et al., 2017) and future work would benefit from examining these relationships at more extreme levels of symptom severity. Ideally, future work may compare trajectories of reinforcement sensitivity, emotion regulation, and psychopathology for diagnosed and undiagnosed populations. This would allow for an unrestricted range of each variable in the model while also identifying where trajectories within diagnosed populations diverge from the general population. This design already has been successfully implemented for bipolar disorders (Alloy et al., 2008), and its implementation for depression and anxiety may follow similar methods.

Finally, the current studies identified group-level stress as a critical ingredient in activating the longitudinal relationship between BAS sensitivity and reappraisal. However, limitations of design and sample size prevent higher-resolution follow-up analyses. A larger sample, particularly at the third wave of data collection, would have allowed for more stable estimates of smaller effect sizes and sub-group comparisons between those who were high in stress at the time of assessment and those who were low in it. Future research may benefit from methodologies such as ecological momentary assessments, daily diaries, and idiographic networks in order to better capture and analyze more commonplace stressors, and the extent to which they too impact how individual differences in emotion regulation are expressed (Aldao, 2013; Fisher et al., 2017; McMahon and Naragon-Gainey, 2020; Taquet et al., 2020). These methodologies are equipped to examine the mechanisms through which reinforcement sensitivity impacts emotion regulation choice on a more granular basis. They may parse temperamental reinforcement sensitivity from "personality states" that occur during depressive episodes (Clark et al., 2003; Naragon-Gainey et al., 2013) and examine how these shifts impact changes in emotion regulation habits. They may also study how different aspects of situational context (e.g., stress intensity; Sheppes et al., 2014) moderate these shifts.

Despite the phenomenological overlap between reinforcement sensitivity dysregulation and affective symptoms (DeYoung et al., 2020), the current studies indicate that the two phenomena are indeed distinguishable and may be related vis emotion regulation. Recent models of pathology that emphasize positive and negative valence systems (e.g., the Hierarchical Taxonomy of Psychopathology; Kotov et al., 2017) may also benefit from further attention to the ways in which deficits in these systems are expressed cognitively. In doing so, steps may be taken towards a multimodal models of psychopathology, along with the possibility of more personalized clinical interventions. (Fig 2a-b)

Author statement

Both authors were involved in every stage of the current research,

including conceptualization of study, data collection, data analysis, and manuscript preparation. Both authors have approved the final manuscript.

Funding

The funding source had no role in the study design, data collection, data analysis, data interpretations, or composition of the manuscript.

Conflict of Interest Statement

The authors have no conflicts of interest to disclose.

Acknowledgments

This research was partially supported by the Israel Science Foundation grant 886/18 awarded to IY.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2022.01.017.

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