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**Poor Emotion Regulation Ability Mediates the Link between Depressive Symptoms and
Affective Bipolarity**

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Abstract

People's relationship between positive and negative affect varies on a continuum from relatively independent to bipolar opposites, with stronger bipolar opposition being termed *affective bipolarity*. Experiencing more depressive symptoms is associated with increased bipolarity, but the processes underlying this relation are not yet understood. Here, we sought to replicate this link, and to examine the role of two potential mediating mechanisms: emotion regulation ability, and trait brooding. Drawing from the *Dynamic Model of Affect*, we hypothesized that (a) a poor ability to regulate negative emotion, and (b) the tendency to brood over one's depressed feelings would predict stronger affective bipolarity, and mediate the relationship between depressive symptoms and affective bipolarity. To measure affective bipolarity, we calculated within-person affect correlations using two weeks of experience sampling data from a community sample ($n = 100$). Mediation analyses indicated that baseline assessments of an inability to regulate negative emotions in general, but not brooding specifically, mediated the relation between depressive symptoms and affective bipolarity. These findings highlight an initial mechanism through which depressive symptoms are associated with lower emotional complexity and flexibility.

Keywords: affective bipolarity, depressive symptoms, emotion regulation ability, brooding

Poor Emotion Regulation Ability Mediates the Link between Depressive Symptoms and Affective Bipolarity

People who suffer from depressive symptoms typically experience intense emotional distress. Not only are there alterations in *average levels* of positive (PA) and negative affect (NA; reflected by depression's two core symptoms, anhedonia and depressed mood; Clark & Watson, 1991), the *mutual relation* between these affective states is also transformed (Dejonckheere et al., 2018). While PA and NA are, on average, only moderately negatively correlated in a healthy population, the emotional life of people with depressive complaints is characterized by affective bipolarity. That is, in depressed individuals, PA and NA tend to be more polarized, representing mutually exclusive ends of a single affective dimension (Watson & Kendal, 1989).

Depressive Symptoms and Affective Bipolarity

The *Dynamic Model of Affect* (DMA; Zautra, Potter, & Reich, 1997) posits that the within-person relation between PA and NA is not invariant, but varies as function of stressful life conditions. Under normal circumstances, the DMA theorizes that people process PA and NA largely in parallel. The relatively independent nature of PA and NA promotes in-depth information processing of one's environment, effectively signaling threats and opportunities for optimal survival (Zautra et al., 1997). In times of distress, however, one's attentional focus becomes narrowed to ensure efficient coping with the stressor (Linville, 1987), shifting the balance in favor of negative over positive information processing. As negative reactivity to stressful conditions challenges one's cognitive resources, the DMA argues that heuristic emotional judgment is desired over rigorous consideration (Zautra et al., 1997). Consequently, the DMA posits that the affective space shrinks, fusing PA and NA onto a single bipolar continuum.

Although the DMA was originally concerned with within-person changes in the affect relation, this theory has also proven helpful in understanding between-person differences in the affective space as function of chronic distress (Davis, Zautra, & Smith, 2004). More specifically, drawing from the *Tripartite Model of Anxiety and Depression* (Clark & Watson, 1991), we recently demonstrated that affective bipolarity is associated with depressive symptoms, but not anxiety-related distress (Dejonckheere et al., 2018). Furthermore, results from a longitudinal experience sampling (ESM) study show that affective bipolarity temporally follows from depressive symptomatology, but the opposite direction does not hold true (Dejonckheere et al., 2018), providing initial insight in the direction of this relation.

As research only recently established a link between depressive symptoms and affective bipolarity, the processes underlying this relation have yet to be investigated. As affective bipolarity leaves people who experience depressive symptoms with less complex and inflexible emotional lives (Feldman, 1995), gaining insight in the associated mechanisms may provide direction in building interventions which address affective bipolarity. Here, we investigate one potential process: emotion regulation (ER). The DMA suggests that constraints in emotional processing as function of experienced distress play a role in pushing one's affective system towards bipolarity (Zautra et al., 1997). Emotion regulation capacity is important in dealing with distressing experiences, but depressed individuals typically show severe ER deficits (Gotlib & Joormann, 2010). Thus, it could be that people with more depressive symptoms cannot manage distressing information well, and this helps to understand why they experience more affective bipolarity.

Depressive Symptoms and Emotion Regulation Deficits

The link between depressive symptoms and impaired ER is well-established (for a review, see Liu & Thompson, 2017), with especially the inability to down-regulate negative emotions typifying depression (Gross & Muñoz, 1995). ER difficulties in depressed

individuals have been investigated with respect to both (a) a general *inability* to effectively use ER strategies, as well as (b) the *selection* of specific maladaptive ER strategies (Liu & Thompson, 2017). Accordingly, in the present paper, we will investigate both the ability to regulate negative emotions in general, and the tendency to brood over one's depressed feelings specifically.

General emotion regulation ability. Depression is related to higher levels of self-reported ER difficulties (Catanzaro & Mearns, 1990). Although depressed individuals can generally successfully implement many ER strategies when instructed to do so (Liu & Thompson, 2017), neuro-imaging studies suggest impaired regulatory influence on amygdala activation in response to emotional challenges (Beauregard, Paquette, & Lévesque, 2006). Consequently, ineffective ER skills are thought to play an important role in both the instigation and maintenance of this disorder (Garnefski & Kraaij, 2006).

Specific emotion regulation strategy selection. Of the many ER strategies, trait rumination has received most research attention in the depression literature. Rumination refers to a dispositional tendency to repetitively focus on the nature and repercussions of one's negative feelings (Nolen-Hoeksema, 1991), with especially its *brooding* component, which involves passively thinking about one's depressed mood, relating to higher levels of depressive symptoms (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). In contrast, the *reflective* or *pondering* component captures active problem solving, which is generally associated with less depressive symptoms over time.

Emotion Regulation Deficits and Affective Bipolarity

We hypothesize that ER deficits in depression may explain the relation between depressive symptoms and affective bipolarity. There is evidence for this pathway for both (a) general emotion regulation ability broadly, and (b) ruminative brooding specifically.

Emotion regulation ability. First, the general inability to regulate negative emotions associated with depressive symptoms may leave people with these symptoms easily overwhelmed when confronted with distressing information (Myin-Germeys et al., 2003). As a result, the DMA would predict that people with depressive symptoms rely more on emotional heuristics, potentially introducing a stronger bipolar affective experience (Zautra et al., 1997).

Brooding. Similarly, research shows that frequently brooding about one's symptoms and negative mood, as is common in depression, constrains a person's cognitive resources (Gotlib & Joormann, 2010). This cognitive burden may hamper deliberate information processing, presumably pushing one's affective system towards bipolarity.

The Present Study

To investigate the role of these two mediating mechanisms, we used ESM data from a large community sample ($n = 100$). Participants reported their momentary PA and NA multiple times a day, which allowed us to compute a within-person PA-NA relation for each individual. First, we hypothesized that more depressive symptoms assessed at baseline would be associated with a stronger bipolar PA-NA relation, replicating the main finding of our previous study (Dejonckheere et al., 2018). Second, we hypothesized that both (a) people's inability to regulate negative emotions, and (b) their chronic tendency to brood about their depressed feelings would statistically mediate the relationship between depressive symptoms and affective bipolarity.

Methods

Participants. We aimed to recruit a community sample of 100 participants, allowing us to detect a small to medium effect size at the between-person level ($r = .30$, $\alpha = .05$). In a first step, we advertised online and via flyers and invited people to complete an online survey, collecting their basic demographics and contact information. A pool of 306 individuals

completed this initial questionnaire (78% female, $M_{age} = 24.99$, $SD_{age} = 12.52$). Of this group, we randomly contacted potential participants to take part in the actual study until we accomplished the desired sample size. In the end, we invited 104 participants to the lab to complete the baseline session and start the ESM protocol (78% female, $M_{age} = 24.04$, $SD_{age} = 6.78$).

Four participants showed poor compliance with the ESM protocol (i.e., < 50% response rate). Their mean compliance was only 25.51% ($SD = 25.39\%$), which implied we could not compute a reliable PA-NA correlation. Therefore, they were excluded from analyses, leaving us with a final sample of 100 participants (77% female, $M_{age} = 24.12$, $SD_{age} = 6.87$). The final sample completed an average of 88.90% measurement occasions ($SD = 9.50\%$). No other data was excluded.

Procedure and materials. We invited participants to the lab in small groups, and collectively explained the purpose and procedure of the study¹. Participants completed several baseline questionnaires, including two surveys assessing depressive symptomatology, and two questionnaires assessing ER. Next, they took part in a two-week ESM study, in which they reported their momentary experience of PA and NA multiple times a day. Table 1 presents descriptive statistics for all measures.

Depressive symptomatology. To check for robustness, we assessed participants' depressive symptoms with two questionnaires that differed considerably in their item content (Fried, 2017). First, they completed the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). This 20-item survey evaluates the frequency of a range of depressive symptoms participants experienced during the last week (e.g., *I could not 'get going'*), ranging from 0 (*rarely or none of the time*) to 3 (*most or all of the time*). Second, participants completed the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). This

¹ For a full protocol, the data, and a reproducible R-code, see: <http://osf.io/mqwyyv>.

questionnaire assesses the intensity of 20 depression symptoms² participants experienced in the past two weeks (e.g., *I get very little pleasure from the things I used to enjoy.*), ranging from 0 (*absent*) to 3 (*very severe*). Internal consistencies for both scales were high ($\alpha_{\text{CES-D}} = .87$; $\alpha_{\text{BDI-II}} = .88$), and they were highly correlated ($r = .76$).

Emotion regulation ability. We assessed participants' self-reported general capacity to regulate negative emotions with the *regulating of emotions in the self* subscale of the emotional intelligence (EI) questionnaire developed by Tett, Fox, & Wang (2005). Their general EI questionnaire assesses emotional ability across a number of facets, with the emotion regulation ability (ERA) subscale that we use in the current study focusing on the degree of control one has over negative emotions when confronted with distressing situations (e.g., *I can keep myself calm, even in highly stressful situations.*; *When I get upset, it takes me a long time to settle down.* [reversed]). The ERA subscale consists of 12 (six positive and six negative) items, and participants evaluate each statement on a 7-point scale ranging from *strongly disagree* to *strongly agree*. Internal consistency was high ($\alpha_{\text{ERA}} = .89$). Higher scores indicate better emotion regulation ability.

We used this scale because it provided information about participants general ability to manage their emotions, rather than focusing on strategy specific effects. Previous work by Tett and colleagues (2005) indicated that this subscale has excellent test-retest reliability (.88), and shows moderate to strong associations with related constructs (e.g., negative relations with neuroticism and trait negative affect, and positive relations with emotional resilience).

Rumination (brooding). We evaluated participants' tendency to ruminate using the Ruminative Response Scale (RRS; Treynor et al., 2003). More specifically, we focused on the *brooding* subscale (RRS-br), as this rumination style shows strong relations with depression.

² Due to ethical constraints, suicidal ideation was not assessed.

This 5-item subscale captures the extent to which people passively think about the consequences of their depressed mood (e.g., *I think about a recent situation, wishing it had gone better.*). Participants rated each item on a scale from 1 (*almost never*) to 4 (*almost always*). Internal consistency was good ($\alpha_{\text{RRS-br}} = .75$). Higher scores indicate brooding more frequently.

ESM protocol. Using experience sampling, we assessed the within-person relation between momentary positive and negative affect for each participant. After providing them with a Motorola smartphone, we asked participants to rate their momentary experience of two positive (*happy, relaxed*) and three negative (*sad, angry, stressed*) emotions seven times a day (*how ___ are you right now?*) for 14 consecutive days. Adopting a stratified random interval scheme, smartphones were programmed to beep randomly between 10 AM and 10 PM, with an average duration of 1 hour and 43 minutes between two consecutive beeps ($SD = 42$ minutes). Participants provided answers on a continuous slider, ranging from 0 (*not at all*) to 100 (*very much*). We instructed participants to take the phone everywhere they went, and they had to respond to each beep within 90 seconds or the survey would be missed. All items had to be answered for the survey to be recorded, so there was no partial missing data.

To create a measure for PA and NA, we averaged ratings of same-valenced emotions for each beep. Internal consistencies showed excellent reliability at the person-level ($\alpha_{\text{PA}} = .97$; $\alpha_{\text{NA}} = .97$), and acceptable to low reliability at the item-level ($\alpha_{\text{PA}} = .70$; $\alpha_{\text{NA}} = .42$)³. Next, we calculated the correlation between our PA and NA composites for each individual across measurement occasions⁴.

³ Item-level internal consistency reflects to what extent the individual items that make up the global composite co-vary in a similar way within the average person across time. The lower internal consistency for NA may be a consequence of people's ability to differentiate between various negative emotional states (e.g., *At this moment, I feel stressed, but not sad.*; Erbas, Ceulemans, Koval, & Kuppens, 2015).

⁴ Although the nested data structure of our ESM protocol suggests the use of multilevel regression, we use a correlational approach to model the relation between PA and NA within individuals. Our rationale, as well as

Statistical Analysis

In our mediation analyses, we modeled depressive symptoms as the predictor (either assessed with the CES-D or the BDI-II), and included both ER ability and brooding as potential mediators (separately). We always modeled affective bipolarity as the outcome variable. Thus, in total we tested four different mediation models (see Figure 1). Although direct causal claims about this sequence cannot be made with the current observational data, and we thus are limited to *statistical* mediation (Disabato, 2016), various arguments exist to hypothesize this particular order. First, we previously demonstrated in a multi-wave ESM study that depressive symptomatology temporally precedes affective bipolarity (and not vice versa; Dejonckheere et al., 2018), supporting the assignment of predictor and outcome. Second, this particular sequence closely matches the theoretical framework of the DMA (Davis et al., 2004), which considers affective bipolarity to be the product of limited cognitive resources due to the experience of chronic stress. Similarly, although the link between depressive symptoms and ER difficulties may be circular (Gotlib & Joorman, 2010), we are interested in how affective bipolarity is maintained in people with depressive symptoms, with our hypothesis that it is the inability to manage emotional situations that perpetuates this relationship. Nevertheless, we recognize that ER difficulties likely play a role both before and after depressive symptoms occur.

the equivalent multilevel models (which fully replicate our correlation results) can be found in Supplemental Materials 1.

Table 1. Means, standard deviations and correlations among all measures.

Measures	Mean (SD)	Correlations			
		1	2	3	4
1. CES-D	0.62 (0.39)				
2. BDI-II	0.43 (0.37)	.76***			
3. ERA	4.20 (1.11)	-.53***	-.58***		
4. RRS-br	2.19 (0.63)	.42***	.47***	-.62***	
5. PA-NA correlation	-.61 (0.16)	-.22*	-.28**	.31**	-.21*

Note. CES-D = Center for Epidemiologic Studies Depression Scale; BDI-II = Beck Depression Inventory-II; ERA = Emotion Regulation Ability Questionnaire; RRS-br = Ruminative Response Scale – brooding; PA = Positive Affect; NA = Negative Affect.

* $p < .050$. ** $p < .010$. *** $p < .001$.

Results

Descriptive statistics and correlations. People displayed considerable differences in the strength of their relation between momentary PA and NA (range = $-.86 - .02$). While some individuals showed a PA-NA relation approaching -1.00 , indicating affective bipolarity, other people's affective states were unrelated (i.e., PA-NA relation around $.0$), suggesting affective independence. Differences in this affective relation were meaningfully related to participants' depressive symptom scores, as measured both with the CES-D and BDI-II (see Table 1), which replicates our findings reported in Dejonckheere et al. (2018). People with higher depressive symptom scores displayed a stronger inverse relation between PA and NA⁵.

⁵ This relation holds after controlling for mean levels of, and variability in, PA and NA, and is robust across various PA and NA item operationalizations (see Supplemental Materials 2 and 3).

In addition, participants' PA-NA relation was linked to both ER indices. A tendency to brood (RRS-br), as well as poor regulation control over negative emotions (ERA) were associated with stronger affective bipolarity.

Mediation analysis for poor negative emotion regulation ability and brooding. To test whether participants' inability to regulate negative emotions, or their tendency to brood over depressed feelings would mediate the relation between depressive symptoms and affective bipolarity, we conducted mediation analyses (see Figure 1). In the prediction of participants' PA-NA relation, we evaluated the direct and indirect pathways of our depression constructs, and bootstrapped these effects to test their significance ($n = 10,000$ resamples).

Figure 1. Mediation results for the relation between participants' depressive symptoms levels, either assessed with the CES-D (Panel A and C) or BDI-II (Panel B and D) and affective bipolarity. The mediator either represents their ERA scores (Panel A and B) or their RRS-br scores (Panel C and D). * $p < .050$. ** $p < .010$. *** $p < .001$.

Emotion regulation ability. As Panel A (CES-D scores) and B (BDI-II scores) of Figure 1 illustrate, the link between depressive symptoms and emotion regulation ability was significant (CES-D: $\beta = -.535$, $SE = .085$, $p < .001$; BDI-II: $\beta = -.581$, $SE = .082$, $p < .001$). In turn, emotion regulation ability was a significant predictor of participants' PA-NA relation

(ERA: $\beta = .31$, $SE = .100$, $p < .010$). Focal to our research question, controlling for the predictive effect of emotion regulation ability rendered the relation between depressive symptoms and participants' PA-NA relation non-significant (CES-D: $\beta = -.076$, $SE = .113$, $p = .505$; BDI-II: $\beta = -.154$, $SE = .120$, $p = .190$). This was confirmed by our bootstrap procedure yielding a significant indirect pathway (CES-D: $a.b = -.170$, 95% CI[-.280, -.035], $p < .01$; BDI-II: $a.b = -.180$, 95% CI[-.252, -.005], $p < .050$), and non-significant direct pathway (CES-D: $c' = -.076$, 95% CI[-.275, .161], $p = .480$; BDI-II: $c' = -.154$, 95% CI[-.366, .031], $p = .100$). In sum, these results provide evidence that emotion regulation ability mediated the relationship between depressive symptoms and affective bipolarity.

Rumination (brooding). Panel C and D present the results with brooding as a potential mediator between participants' depressive symptom scores and PA-NA relation. Again, depressive symptomatology and brooding were significantly related (CES-D: $\beta = .420$, $SE = .091$, $p < .001$; BDI-II: $\beta = .47$, $SE = .089$, $p < .001$), as were brooding and participants' PA-NA relation (RRS-br: $\beta = -.213$, $SE = .098$, $p < .05$). When controlling for the predictive capacity of brooding, participants' BDI-II scores still meaningfully predicted affective bipolarity ($\beta = -.234$, $SE = .110$, $p < .050$), while their CES-D scores became non-significant ($\beta = -.158$, $SE = .108$, $p = .145$). Contrary to our hypothesis, bootstrapping produced a non-significant indirect pathway for both depressive symptom measures, indicating an absence of mediation via people's tendency to brood (CES-D: $a.b = -.090$, 95% CI[-.184, .018], $p = .160$; BDI-II: $a.b = -.101$, 95% CI[-.172, -.0042], $p = .330$). The direct pathway remained significant for participants' BDI-II scores ($c' = -.234$, 95% CI[-.403, -.070], $p < .001$), but not for their CES-D scores ($c' = -.158$, 95% CI[-.331, .055], $p = .130$). In sum, contrary to our

expectations, there was no evidence that brooding mediated the relation between depressive symptoms and affective bipolarity⁶.

Discussion

Our primary aim was to begin to understand the processes underlying the relation between depressive symptoms and affective bipolarity. Specifically, we investigated the potential role of two ER processes: the ability to regulate negative emotions, and a tendency to brood over one's depressed feelings. Using ESM data from a two-week study conducted with a large community sample, we replicated the link between depressive symptoms and affective bipolarity (Dejonckheere et al., 2018). Building on this work, mediation analyses indicated that an inability to regulate negative emotions in general, but not brooding specifically, mediated this link. Providing evidence for the robustness of this effect, this finding replicated across two depressive symptom scales, each composed of different items (Fried, 2017), and assessing different symptom dimensions (i.e., frequency versus severity)⁷.

The present results support our theoretical propositions drawn from the DMA (Zautra et al., 1997), and reveal an initial mechanism through which depressive symptoms are associated with a more simplified and inflexible emotional life. As people with depressive symptoms are easily distressed (Myin-Germeys et al., 2003), yet lack the capacity to effectively down-regulate negative emotions (Gross & Muñoz, 1995), processing PA and NA in parallel may be challenging. Instead, the cognitive burden associated with depressive symptoms is likely to narrow a person's affective system, fusing PA and NA to become the mutually exclusive ends of a single affective continuum (Davis et al., 2004). In this way,

⁶ A model in which we entered both mediators simultaneously replicates the results of the separate mediation models that we describe here (see Supplemental Materials 4 for the full results of this model).

⁷ Interestingly, the mediating effect of emotion regulation ability is slightly stronger for the CES-D than the BDI-II. The CES-D's greater emphasis on emotional disturbances (versus the BDI-II's primary focus on cognitive distortions; e.g., Fried, 2017) may explain this finding.

one's emotional repertoire is not only less complex, solely ranging from feeling bad to good, it may also be less flexible, as affective bipolarity hinders subtle emotional responding to ambiguous situations.

Contrary to our expectations, brooding did not mediate the relation between depressive symptoms and affective bipolarity. Although this ineffective ER strategy is commonly adopted by people with depressive symptoms (Nolen-Hoeksema, 1991), it seems that affective bipolarity may be related to broader difficulties in emotion regulation. Indeed, ER research shows that depression is associated with deficits across a number of strategies (Liu & Thompson, 2017). Similarly, our results suggest that affective bipolarity may show stronger relations with people's general inability to regulate negative emotions, rather than the habitual selection of specific ER strategies. Finally, it may also be that the problems experienced in ER are not at the strategy level, but indicate broader difficulties, for example with the flexible or contextually sensitive implementation of ER strategies (Bonanno & Burton, 2013). Thus, before developing interventions based on our findings, we believe more targeted research needs to be done to pinpoint how exactly low ER ability is associated with a more bipolar emotional life. One potential mechanism is cognitive control, which may be associated with reduced affective bipolarity (e.g., Davis et al., 2004), and is linked to effective emotion regulation (Ochsner & Gross, 2005).

Finally, these findings may also have implications for work on emotion regulation more generally. A large body of research has established how ER strategies influence mean levels of positive and negative affect, but considerably less research has investigated how emotion regulation is implicated in the more complex ways in which emotion varies. This work suggests that ER processes may play a role in affective bipolarity, highlighting the potential utility that moving beyond mean levels of PA and NA may have in understanding effective emotion regulation. Of course, this research only examined broad trait scales

assessing ER, and we hope that moving forward, researchers will delve more deeply into the specifics of this relationship.

Limitations and Future Directions

There are limitations with this research that should be addressed in future studies. First, we used a community sample, and all participants were relatively healthy. A direct application of our findings to clinical depression may be unwarranted.

Second, the individual affect items that made up the PA-NA relation did not fully cover the affective space, making it difficult to assess a possible influence of the arousal dimension. Although multiverse analyses in Dejonckheere et al. (2018) do not suggest a confounding effect of arousal on the relation between depressive symptoms and affective bipolarity, it remains to be explored whether this dimension impacts the relation between affective bipolarity and both ER deficits. Future research could particularly benefit from including high arousal PA items (e.g., *excited*), and low arousal NA items (e.g., *depressed*).

Third, all measures are based on self-report, raising the question whether people can adequately reflect on their ER ability. Lab experiments in which researchers, (a) assess participants' emotion regulation implicitly via experimental tasks, and (b) effectively differentiate between adequate ER selection and successful implementation, could provide additional insight in the mediating role of emotion regulation. Relatedly, an experimental setting could address concerns regarding conducting mediation analyses with cross-sectional measures (however, see Disabato [2016] for a counter-concern). Although depressive symptomatology temporarily precedes affective bipolarity (Dejonckheere et al., 2018), and the current mediation model closely follows the DMA rationale, direct causal claims about this relation cannot be made with these observational data. Indeed, it is likely that ER deficits and depressive symptoms form a feedback loop, which could be disentangled using a larger longitudinal design.

Conclusion

We aimed to gain insight into the processes involved in the relation between depressive symptoms and a stronger bipolar experience of PA and NA. Results suggest that an inability to regulate negative emotions in general, but not brooding specifically, statistically mediates the relation between depressive complaints and affective bipolarity. This finding reveals an initial mechanism through which depressive symptoms are associated with reductions in emotional complexity and flexibility.

Declaration of Conflicting Interests

The authors declare no conflict of interest.

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Supplemental Materials – Introduction

To check the robustness of the relation between affective bipolarity and depressive symptoms we performed various checks, described in Supplemental Materials. First, we illustrate that standardized multilevel models fully replicate the correlational results described in our main text, showing robustness across approaches (see Supplemental Materials 1). Second, hierarchical multiple regression indicates that the relation between affective bipolarity and depressive symptoms is not driven by differences in mean levels of, or variability in, positive (PA) and negative affect (NA). In fact, a stronger bipolar relation between PA and NA is predicted by participants' depressive symptoms, independent of these covariates. Furthermore, mediation analyses in which we additionally control for average levels of PA and NA largely yield identical conclusions (see Supplemental Materials 2). Third, a multiverse analysis shows that the relation between affect bipolarity and depressive symptoms is largely robust across different PA and NA construct operationalizations (see Supplemental Materials 3). Finally, we show that a single model, in which we enter both emotion regulation (ER) variables as potential mediators simultaneously, fully replicates the results of our separate mediation models described in the main text (see Supplemental Materials 4).

Supplemental Materials 1: Standardized Multilevel Models

Although the nested data structure of our ESM protocol (i.e., repeated emotional assessments per individual) suggests the potential to use multilevel regression, in our main analyses, we use a correlational approach to model the relation between PA and NA within individuals. We do this because we aim to investigate how PA and NA form a unidimensional construct as a function of depressive symptoms, meaning we are essentially evaluating the reciprocal relation between these affective states (i.e., the *symmetrical* dependency). In contrast, multilevel regression is inherently *asymmetrical*, meaning we have to specify PA, for instance, as an outcome, and NA as a predictor (or vice versa). Not only is this assignment quite arbitrary (Ong, Zautra, & Finan, 2017), changing outcome and predictor may produce different results. To overcome this asymmetry, we computed a Pearson correlation between PA and NA per participant (i.e., to see how these affective states *mutually* relate), and investigated how this correlation was related to people's level of depressive symptoms. A similar approach is adopted in other studies investigating between-person differences in the PA-NA relation (e.g., Bagozzi et al., 1999; Carstensen et al., 2011; Davis et al., 2004; Dejonckheere et al., 2018).

However, one way to obtain symmetry in a multilevel context is to use within-person standardized PA and NA variables as outcome and predictor (Schoorman, Ferrer, de Boer-Sonnenschein, & Hamaker, 2016). In this way, predicting PA on NA or NA on PA yields identical results. In this section, we show that a multilevel approach with person-mean standardized variables generates similar results and supports identical conclusions, fully replicating our correlational findings.

Table 1S presents all multilevel models with participants' within-person standardized PA and NA scores. In both models, the momentary-level slope represents the average within-person affect relation observed in our sample ($r = -.61$). Adding participants' (grand-mean

standardized) CES-D or BDI-II scores at the person-level indicates how this PA-NA relation varies as a function of between person-differences in depressive symptoms. This yields the slope to become significantly more negative, indicating affective bipolarity.

Table 1S. Multilevel analyses modelling the relation between momentary PA and NA as a function of depressive symptom severity.

	β	SE	t	p
Model 1 – predicting standardized NA				
Level 1: Standardized PA	-0.615	0.016	-38.917	<.001
Level 2: CES-D	-0.091	0.037	-2.483	.015
Model 2 – predicting standardized PA				
Level 1: Standardized NA	-0.615	0.016	-38.917	<.001
Level 2: CES-D	-0.091	0.037	-2.483	.015
Model 3 – predicting standardized NA				
Level 1: Standardized PA	-0.615	0.016	-38.917	<.001
Level 2: BDI-II	-0.124	0.037	-3.387	.001
Model 4 – predicting standardized PA				
Level 1: Standardized NA	-0.615	0.016	-38.917	<.001
Level 2: BDI-II	-0.124	0.037	-3.387	.001

Supplemental Materials 2: Controlling for mean and variability in PA and NA

Because participants' PA-NA relation may be function of how positive or negative they feel on average (e.g., low NA mean levels may attenuate the PA-NA correlation), or the amount of variability they display in these affective states (e.g., no variance in PA or NA yields zero PA-NA correlations), we examined the unique effect of our variables of interest on people's PA-NA relation, controlling for mean levels of, and variability in, these affective states using hierarchical multiple regression (see Table 2S). Participants' PA-NA relation was our key outcome in all models. Model 1 only included our focal predictor, being either participants' CES-D, BDI-II, ERA or RRS-br scores. In Model 2, we entered participants' average levels of PA and NA as additional predictors. Finally, in Model 3, we additionally introduced participants' standard deviations of PA and NA as an indicator of variability.

The results in Table 2S indicate that both participants' depressive symptom scores (CES-D and BDI-II), as well as their inability to regulate negative emotion (ERA) predict stronger affective bipolarity, above and beyond these covariates. That is, mean levels of, and variability in PA and NA did not fully explain the current findings. For brooding (RRS-br), the association with affective bipolarity became marginally significant when controlling for mean levels ($p = .059$), and non-significant when additionally taking into account variability in PA and NA ($p = .160$).

Table 2S. Hierarchical multiple regression on the PA-NA correlation for CES-D, BDI-II, ERA and RRS-br scores.

Models	CES-D as predictor		BDI-II as predictor		ERA as predictor		RRS-br as predictor	
	R^2 (adj)	β	R^2 (adj)	β	R^2 (adj)	β	R^2 (adj)	β
Model 1	.04*		.07**		.09**		.04*	
Intercept		-.56***		-.56***		-.81***		-.49***
Predictor		-.09*		-.13**		.05**		-.06*
Model 2	.08*		.09**		.11**		.06*	
Intercept		-.23		-.27 [†]		-.52***		-.23
Predictor		-.10*		-.13**		.044**		-.05 [†]
Mean momentary PA		-.004*		-.004*		-.004*		-.003 [†]
Mean momentary NA		-.004 [†]		-.004		-.004 [†]		-.004*
Model 3	.09*		.10**		.12**		.07*	
Intercept		-.22		-.26 [†]		-.46**		-.23
Predictor		-.09*		-.11*		.04**		-.04
Mean momentary PA		-.003 [†]		-.003 [†]		-.003		-.003
Mean momentary NA		-.002		-.002		-.002		-.002
SD momentary PA		-.001		-.001		-.004		-.002
SD momentary NA		-.007		-.006		-.004		-.006

Note. The within-person PA-NA relation was our key dependent variable in all models. The predictor either refers to participants' CES-D, BDI-II, ERA or RRS brooding scores.

[†] $p < .100$ * $p < .050$. ** $p < .010$. *** $p < .001$

Finally, because emotion regulation is known to influence average levels of positive and negative emotionality, we reran the four mediation models described in our main text, with mean levels of PA and NA as additional covariates. The results of these analyses can be found in Table 3S.

Table 3S. Significance for the mediating pathways of our four mediation models, with mean levels of PA and NA as additional covariates.

Predictor	Mediator					
	ERA scores			RRS-br scores		
	<i>a.b</i>	95% CI	<i>p</i>	<i>a.b</i>	95% CI	<i>p</i>
CES-D scores	-.127	[-.249, -.020]	.019	-.051	[-.168, .030]	.227
BDI-II scores	-.119	[-.242, .010]	.060	-.042	[-.153, .040]	.390

Note. *a.b* refers to the indirect pathway between depressive symptoms and affective bipolarity.

In sum, these models largely replicate our main findings. Trait rumination is still not a significant mediator of the relation between depressive symptoms (both with the CES-D and BDI-II) and affective bipolarity, when the predictive power of mean levels of positive and negative affect is taken into account. In contrast, people's self-reported ability to regulate negative emotions remains a significant mediator of this relation, when depressive symptoms are assessed with the CES-D, and a marginally significant mediator when the BDI-II was used (see Table 3S).

Supplemental Materials 3 – Multiverse Analysis for PA and NA constructs

Because there is no theoretical consensus on the definition of PA and NA, and various PA and NA composites may produce differences in the strength of their mutual relation (e.g., in terms of truly being semantic antonyms; Barrett & Russell, 1998), this could also influence the observed relation between affective bipolarity and depressive symptoms, emotion regulation ability, or brooding. To examine how our results changed as a function of the particular PA and NA item combination we adopted, we performed a multiverse analysis (Steegen, Tuerlinckx, Gelman, & Vanpaemel, 2016). In this analysis, we calculated every possible PA-NA relation from the two positive and three negative affect items ($n = 21$). Next, we related each of these different PA-NA relations to both participants' depressive symptom levels (i.e., an average of their standardized CES-D and BDI-II scores) and ER measures (i.e., both their ERA and RRS-br scores).

As Figure 1S illustrates, the vast majority of different PA-NA relations (each based on a different combination of affect items) showed a meaningful link with participants' depressive symptom levels ($n = 17$; Panel A), suggesting that the relation between affective bipolarity and depressive symptoms is largely robust across various compositions of our PA and NA constructs. For the association with participants' ERA and RRS-br scores, the amount of significant correlations was slightly lower ($n = 11$ and 7 , respectively; Panel B and C).

A. Depressive Symptoms

HA	.362	.003	.036	.054	.041	.010	.025
RE	.318	.004	.001	.048	.006	.001	.006
HA RE	.321	.002	.001	.040	.009	.001	.007
	SA	AN	ST	SA AN	SA ST	AN ST	SA AN ST

B. Emotion Regulation Ability

HA	.074	.074	.288	.019	.076	.109	.039
RE	.309	.249	.000	.148	.005	.000	.004
HA RE	.103	.084	.001	.025	.003	.001	.002
	SA	AN	ST	SA AN	SA ST	AN ST	SA AN ST

C. Rumination (brooding)

HA	.163	.073	.529	.045	.190	.275	.122
RE	.381	.385	.017	.292	.058	.025	.058
HA RE	.227	.140	.029	.093	.045	.025	.033
	SA	AN	ST	SA AN	SA ST	AN ST	SA AN ST

Figure 1S. Multiverse results for the relation between participants' PA-NA correlation and depressive symptoms (Panel A), emotion regulation capacity (Panel B) and rumination (brooding; Panel C). Values indicate the p -value for all possible PA-NA relations ($n = 21$). Gray cells are significant, white cells are non-significant. HA = Happy; RE = Relaxed; SA = Sad; AN = Angry; ST = Stressed.

Next, to get a better understanding of the factors that influenced the strength of the correlation between the PA-NA relation and each variable of interest, we performed an exploratory trend analysis on our multiverse results. Specifically, we investigated whether (a) the number of items that make up our PA and NA constructs, (b) and the degree to which emotion words truly represent semantic antonyms, impact the observed association between

participants' PA-NA relation and their depressive symptoms, emotion regulation ability, and trait brooding. Table 4S presents the results of these exploratory trend analyses.

Table 4S. Exploratory trend analysis for multiverse results.

Trend	Depressive Symptoms		Emotion Regulation Ability		Rumination (brooding)	
	β	p	β	p	β	p
1. Number of items	-0.019	.343	0.046	.032	-0.027	.040
2. Semantic opposites	0.016	.737	0.063	.242	-0.036	.270

First, the number of items that make up the PA-NA relation (operationalized as the sum of all PA and NA items) significantly impacted the relation between affective bipolarity and emotion regulation ability and trait brooding, but not depressive symptoms. That is, PA-NA relations based on more emotion items show stronger associations with emotion regulation ability and trait brooding. A possible explanation for this effect may be that single-item constructs typically exhibit more measurement error, and therefore are less reliable. Because single-item PA-NA relations may be more unreliable, this may attenuate their association with external criteria (e.g., Green, Goldman, & Salovey, 1993).

Second, the degree to which emotion words truly represent semantic antonyms (operationalized as a binary variable with e.g., *relaxed-sad* = 0 and *relaxed-stressed* = 1) did not significantly impact the association between affective bipolarity and participants' depressive symptoms, emotion regulation ability, and trait rumination (e.g., Barrett & Russell, 1998).

Finally, we did not assess the potential impact of different arousal levels, as the current affect items do not fully cover the affective space (i.e., we did not assess any high arousal PA items [e.g., *excited*], or low arousal NA items [e.g., *depressed*]).

Supplemental Materials 4 – Mediation Models with the two Mediators simultaneously

In a final step, we aimed to investigate whether a single mediation model, in which we simultaneously entered both ER variables as potential mediators, yielded similar conclusions. That is, participants' poor ability to regulate negative emotions in general, but not brooding specifically, would mediate the link between their depressive symptoms and affective bipolarity.

In a first step, we removed the overlap between the two mediators, as both ER variables were considerably correlated ($-.62$). In this way, we could establish an independent pathway for each mediating variable, allowing us to evaluate the unique mediating effect for each ER variable. This was achieved via two separate regression models, in which both mediators were regressed on each other (i.e., trait brooding predicting emotion regulation ability, and vice versa). From these models, we extracted the residuals, which reflect the unique variance for each ER variable, and entered these as mediators in a single mediation model, in which affective bipolarity was predicted by participants' depressive symptom scores (either assessed with the CES-D or the BDI-II).

Figure 2S. Multiple mediation models for the link between participants' CES-D (Panel A) or BDI-II scores (Panel B) and affective bipolarity. Note that the unique residuals (res) of participants' RRS-br and ERA scores were used to achieve independence between both mediating pathways.

* $p < .050$. ** $p < .010$. *** $p < .001$.

Figure 2S visualizes the results for both depressive symptom questionnaires. First, the link between depressive symptoms and (the unique variance of) emotion regulation ability was significant (CES-D: $\beta = -.274$, $SE = .074$, $p < .001$; BDI-II: $\beta = -.286$, $SE = .073$, $p < .001$). In turn, (the unique variance of) emotion regulation ability was a significant predictor of participants' PA-NA relation (ERA_{res}: $\beta = .290$, $SE = .130$, $p = .023$). In contrast, (the unique variance of) trait rumination was not significantly predicted by participants' depressive symptoms (CES-D: $\beta = .087$, $SE = .078$, $p = .266$; BDI-II: $\beta = .112$, $SE = .078$, $p = .153$), nor did (the unique variance of) trait rumination meaningfully predict participants' PA-NA relation (RRS-br_{res}: $\beta = -.034$, $SE = .130$, $p = .795$). Focal to our research question, controlling for the predictive effect of (the unique variance of) emotion regulation ability and trait brooding rendered the relation between depressive symptoms and participants' PA-NA relation non-significant (CES-D: $\beta = -.073$, $SE = .115$, $p = .527$; BDI-II: $\beta = -.154$, $SE = .119$, $p = .202$). This was confirmed by our bootstrap procedure ($n = 10,000$ resamples), which produced a significant pathway for (the unique variance of) emotion regulation ability (CES-D: $a.b = -.146$, 95% CI $[-.281, -.040]$, $p = .007$; BDI-II: $a.b = -.131$, 95% CI $[-.260, -.010]$, $p = .037$), a non-significant indirect pathway for (the unique variance of) trait rumination (CES-D: $a.b = -.087$, 95% CI $[-.219, .006]$, $p = .052$; BDI-II: $a.b = -.077$, 95% CI $[-.200, .020]$, $p = .150$), and a non-significant direct pathway for depressive symptoms (CES-D: $c' = -.073$, 95% CI $[-.261, .160]$, $p = .515$; BDI-II: $c' = -.154$, 95% CI $[-.352, .030]$, $p = .089$).

In sum, these results fully replicate the findings of our separate mediation models: a poor ability to regulate negative emotions in general, but not brooding specifically, mediates the link between depressive symptoms and affective bipolarity.

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