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BRIEF ARTICLE

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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 hypothesised 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.

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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 characterised by affective bipolarity. That is, in depressed individuals, PA and NA tend to be more polarised, representing mutually exclusive ends of a single affective dimension (Watson & Kendall, 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 theorises 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 signalling 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 favour 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.

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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.

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 hypothesise 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 hypothesised 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 hypothesised 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 study.[1] 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 questionnaire assesses the intensity of 20 depression symptoms[2] 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 ($\alpha_{CES\text{-}D} = .87$; $\alpha_{BDI\text{-}II} = .88$), 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, and Wang (2005). Their general EI questionnaire assesses emotional ability across a number of facets, with the emotion regulation ability (ERA) subscale

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

		Correlations			
Measures	Mean (SD)	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.
*$p < .050$, **$p < .010$, ***$p < .001$.

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 ($\alpha_{ERA} = .89$). 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 et al. (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. 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 ($\alpha_{RRS\text{-}br} = .75$). 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 h and 43 min between two consecutive beeps ($SD = 42$ min). 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 s 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 ($\alpha_{PA} = .97$; $\alpha_{NA} = .97$), and acceptable to low reliability at the item-level ($\alpha_{PA} = .70$; $\alpha_{NA} = .42$).[3] Next, we calculated the correlation between our PA and NA composites for each individual across measurement occasions.[4]

Statistical analysis

In our mediation analyses, we modelled 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 modelled 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 hypothesise this particular order. First, we previously demonstrated in a multi-wave ESM study that depressive

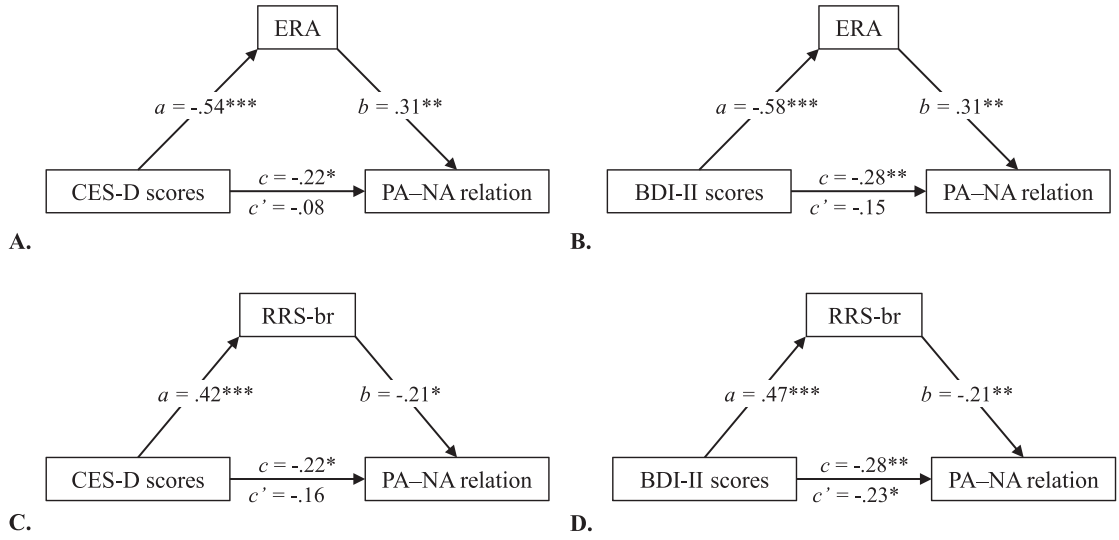

Figure 1. Mediation results for the relation between participants' depressive symptoms levels, either assessed with the CES-D (Panel A and C) or BDI-II (Panel B and D) and affective bipolarity. The mediator either represents their ERA scores (Panel A and B) or their RRS-br scores (Panel C and D). *$p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

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 & Joormann, 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 recognise that ER difficulties likely play a role both before and after depressive symptoms occur.

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 NA.[5]

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).

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, −. 0.042], $p = .330$). 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 bipolarity.[6]

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, 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.

Notes

1. For a full protocol, the data, and a reproducible R-code, see: http://osf.io/mqwyv.
2. Due to ethical constraints, suicidal ideation was not assessed.
3. Item-level internal consistency reflects to what extent the individual items that make up the global composite covary 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 the equivalent multilevel models (which fully replicate our correlation results) can be found in Supplemental Materials 1.
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).
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.

Disclosure statement

No potential conflict of interest was reported by the authors.

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References

Beauregard, M., Paquette, V., & Lévesque, J. (2006). Dysfunction in the neural circuitry of emotional self-regulation in major depressive disorder. *Neuroreport*, *17*, 843–846.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the beck depression inventory–II*. San Antonio, TX: Psychological Corporation.

Bonanno, G. A., & Burton, C. L. (2013). Regulatory flexibility: An individual differences perspective on coping and emotion regulation. *Perspectives on Psychological Science*, *8*, 591–612.

Catanzaro, S. J., & Mearns, J. (1990). Measuring generalized expectancies for negative mood regulation: Initial scale development and implications. *Journal of Personality Assessment*, *54*, 546–563.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*, 316–336.

Davis, M. C., Zautra, A. J., & Smith, B. (2004). Chronic pain, stress, and the dynamics of affective differentiation. *Journal of Personality*, *72*, 1133–1160.

Dejonckheere, E., Mestdagh, M., Houben, M., Erbas, Y., Pe, M., Koval, P., … Kuppens, P. (2018). The bipolarity of affect and depressive symptoms. *Journal of Personality and Social Psychology*, *114*, 323–341.

Disabato, D. (2016, May 22). *The double standard against cross-sectional mediation*. Retrieved from http://www.daviddisabato.com/blog/2016/5/22/the-double-standard-against-cross-sectional-mediation

Erbas, Y., Ceulemans, E., Koval, P., & Kuppens, P. (2015). The role of valence focus and appraisal overlap in emotion differentiation. *Emotion*, *15*, 373–382.

Feldman, L. A. (1995). Valence focus and arousal focus: Individual differences in the structure of affective experience. *Journal of Personality and Social Psychology*, *69*, 153–166.

Fried, E. I. (2017). The 52 symptoms of major depression: Lack of content overlap among seven common depression scales. *Journal of Affective Disorders*, *208*, 191–197.

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, *6*, 285–312.

Gross, J. J., & Muñoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice*, *2*, 151–164.

Linville, P. W. (1987). Self-complexity as a cognitive buffer against stress-related illness and depression. *Journal of Personality and Social Psychology*, *52*, 663–676.

Liu, D. Y., & Thompson, R. J. (2017). Selection and implementation of emotion regulation strategies in major depressive disorder: An integrative review. *Clinical Psychology Review*, *57*, 183–194.

Myin-Germeys, I., Peeters, F., Havermans, R., Nicolson, N. A., DeVries, M. W., Delespaul, P., & Van Os, J. (2003). Emotional reactivity to daily life stress in psychosis and affective disorder: An experience sampling study. *Acta Psychiatrica Scandinavica*, *107*, 124–131.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, *100*, 569–582.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *TRENDS in Cognitive Sciences*, *9*, 242–249.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*, 385–401.

Tett, R. P., Fox, K. E., & Wang, A. (2005). Development and validation of a self-report measure of emotional intelligence as a multidimensional trait domain. *Personality and Social Psychology Bulletin*, *31*, 859–888.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, *27*, 247–259.

Watson, D., & Kendall, P. C. (1989). Understanding anxiety and depression: Their relation to negative and positive affective states. In P. C. Kendall & D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features* (pp. 3–26). New York, NY: Academic Press.

Zautra, A. J., Potter, P. T., & Reich, J. W. (1997). The independence of affects is context-dependent: An integrative model of the relationship between positive and negative affect. In M. P. Lawton & S. K.W. (Eds.), *Annual review of gerontology and geriatrics* (pp. 75–102). New York, NY: Springer Publishing Company.