



# An investigation of the relationship between positive affect regulation and depression

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## ABSTRACT

There is preliminary evidence that dysphoric symptoms are associated with maladaptive regulation of positive emotion. We investigated to what extent this pattern is unique to depression symptoms, persists in recovery, and extends to apprehension of intense emotion experience. In Study 1, in a sample of undergraduates ( $N = 112$ ), dysphoria was associated with apprehension about experiencing intense emotion and dampening of positive emotion. Reductions in the amplification of positive emotion experience were uniquely associated with anhedonic depressive symptoms. Study 2 compared a recovered depressed and never-depressed student sample ( $N = 123$ ), and found that recovered individuals reported using more maladaptive responses to positive affect. In Study 3 we examined community-recruited depressed, recovered and never-depressed groups ( $N = 50$ ), and found that depressed individuals reported a greater tendency to dampen positive emotion than their never-depressed counterparts, but did not significantly differ from recovered depressed individuals. Greater dampening and reduced amplification of positive experience were again uniquely associated with anhedonic depressive symptoms. Our findings converge on the proposal that current depressive symptoms, rather than a history of depression, are more strongly linked to difficulties with emotion regulation, and suggest that targeting positive emotion could reduce anhedonia and improve treatment outcomes.

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Emotion regulation refers to the processes used by individuals to influence how their emotions are experienced and expressed (Gross, 1998). There is now a growing recognition that emotion regulation involves considerable effort and control, and that some emotion regulation strategies are more helpful than others (Gross, 1999). Depression has been increasingly characterised as a disorder of dysregulated emotion, and there is evidence that clinically depressed groups use less adaptive, more dysfunctional and less flexible strategies than their non-depressed counterparts (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Garnefski & Kraaij, 2006; Kashdan & Rottenberg, 2010). Interestingly, disturbances in emotion regulation do not appear to be limited to the acute phase of depression (i.e., when individuals are symptomatic) as there is now preliminary evidence that individuals who have experienced depression in the past employ more dysfunctional strategies (e.g., catastrophising and rumination) and less functional strategies (e.g., putting things into

perspective) than never-depressed controls (Ehring, Fischer, Schnulle, Bosterling, & Tuschen-Caffier, 2008; Ehring, Tuschen-Caffier, Schnulle, Fischer, & Gross, 2010). This suggests that emotion regulation difficulties may play a key role not only in current depression, but also in the euthymic phase when the individual no longer meets criteria for a major depressive episode, with possible implications for both acute depression and vulnerability to recurrence.

Research on the role of emotion regulation in depression has predominantly focused on negative emotional experience, which is understandable from a conceptual viewpoint because depression is classified as a disorder of elevated negative affectivity. However, it is surprising that the other cardinal symptom of depression – anhedonia (i.e., a diminished ability to respond to positive stimuli) – has been relatively less researched. In the context of depression, understanding the regulation of positive emotion is of clear clinical importance. Specifically, current gold-standard pharmacological and psychological therapies for depression struggle to successfully treat anhedonia (Dunn, 2012; Treadway & Zald, 2011). For example, while encouraging individuals to reengage with potentially rewarding activities is a central component of cognitive-behavioural therapy and behavioural activation (Martell, Addis, & Jacobson,

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2001), there is very little understanding of how to help depressed clients gain enjoyment from these events. While studies are beginning to explore the impact of mindfulness based cognitive therapy and positive psychology approaches on positive affect (e.g. Geschwind, Peeters, Drukker, van Os, & Wichers, 2011; Sin & Lyubomirsky, 2009), insight into the underlying mechanism of change is largely absent. Improving understanding of what mechanisms underpin anhedonia in depression is likely to lead to novel, targeted interventions that will in turn improve treatment outcome. In particular, deficits in positive affect predict a poor depression prognosis (Morris, Blysm, & Rottenberg, 2009; Shankman, Nelson, Harrow, & Faull, 2011; Wood & Joseph, 2010), perhaps because positive affect makes individuals more resilient to negative life events (Fredrickson & Levenson, 1998; Tugade & Fredrickson, 2004). One possibility that follows is that by building positive emotion experience vulnerable individuals may be protected from an initial episode of depression or from subsequent relapse.

A critical issue that needs to be explored is whether depressed individuals employ self-regulatory cognitive strategies when they encounter positive material which might underpin their anhedonic experience. That is, it would be helpful to know whether depressed individuals engage in any conscious mental strategies with the intention of reducing the intensity of positive emotional experience. There is some preliminary evidence that depressive symptoms are associated with effortful attempts to dampen and reduce positive affect (PA; Feldman, Joormann, & Johnson, 2008; Raes, Daems, Feldman, Johnson, & Van Gucht, 2009). In these studies, undergraduate students were administered the Responses to Positive Affect Questionnaire (RPA; Feldman et al., 2008) which assesses cognitive responding to positive experience. The RPA has three subscales – two which measure attempts to amplify positive emotional experience (emotion-focus and self-focus) and the third of which measures attempts to down-regulate positive emotion (dampening). The self-reported tendency to engage in mental strategies that dampen positive emotional experience have been found to be associated with dysphoric symptoms (Feldman et al., 2008; Raes et al., 2009). However, no consistent pattern of findings has emerged between the subscales measuring attempts to amplify positive emotion and dysphoric symptoms. For example, in the Feldman et al. (2008) study, there was a small negative correlation between depressive symptoms and the self-focus subscale, but no correlation with the emotion-focus subscale. Conversely, Raes et al. (2009) found a small negative correlation between depressive symptoms and the emotion-focus subscale, but no link with the self-focus subscale. Further, a more recent study found scores on the dampening subscale (but not the emotion-focus or self-focus subscales) were predictive of dysphoric symptoms at three month and five month follow-up, even when baseline dysphoria was taken into account (Raes, Smets, Nelis, & Schoofs, 2012). From this research it is clear that dampening of PA is cross-sectionally and prospectively linked to dysphoric symptoms. However, there has been no consistent pattern of findings for the two amplification subscales on this measure, which is not surprising given the recent development of the instrument. Further investigation is needed to better understand dysphoric individuals' cognitive responses to PA.

The aim of this cross-sectional series of studies was to further investigate positive emotion regulation in depression, extending previous work in a number of ways. First, drawing from research establishing that apprehension towards the experience of negative emotion is associated with depression (Liverant, Brown, Barlow, & Roemer, 2008), we wanted to examine whether the same would hold for positive emotion. That is, we wanted to test whether apprehension towards experiencing intense emotion in general, and more specifically, positive emotion, would be linked to

dysphoria. A fear of positive emotion could be one important underlying factor driving anhedonia and therefore become a target for intervention in therapy (for example, using cognitive restructuring techniques).

Second, existing studies (e.g., Raes et al., 2012) have relied on measures of dysphoric symptoms (e.g. Beck Depression Inventory-II (BDI-II); Beck, Steer, & Brown, 1996), which are not specific measures of depression severity. In particular, individuals with anxiety presentations also score highly on the BDI-II. It is important to establish to what extent positive emotion regulation deficits are driven by anxiety symptoms, depression symptoms, or a combination of the two, both to aid differential diagnosis and to help develop effective and targeted clinical interventions. One way to establish specificity within continuous designs is to examine how candidate processes relate to symptoms common across diagnoses versus those relatively specific to a diagnosis. For example, the tripartite framework (Clark & Watson, 1991; Clark, Watson, & Mineka, 1994) suggests that common to both anxiety and depression is 'general distress', consisting of non-specific 'negative affect' symptoms (e.g., depressed mood, anxious mood, insomnia). Unique to anxiety are physiological hyper-arousal and somatic tension symptoms (e.g., dizziness, shortness of breath), whereas unique to depression are 'positive affect' symptoms of anhedonia and apathy (e.g., loss of interest, feeling nothing is enjoyable). The Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991; Watson et al., 1995) has been developed as a self-report measure of these three symptom clusters. Previous work suggests that evidence of depression versus anxiety specificity can be gleaned using this measure. For example, reductions in positive judgement biases are unique to anhedonia symptoms (Dunn, Stefanovitch, Buchan, Lawrence, & Dalgleish, 2009), whereas hyper-awareness of the body is largely driven by anxious arousal symptoms (Dunn et al., 2010).

Such an approach has yet to be taken to examine positive emotion regulation in depression. It may be the case that these different symptom clusters drive positive emotion regulation in different ways. Consequently, global measures of dysphoria severity may not be sensitive enough to detect subtle relationships in the data. For example, mixed findings regarding the relationship between the amplifying scales of the RPA across different studies (Feldman et al., 2008; Raes et al., 2009) could reflect variation in the specific symptom clusters that the sample predominantly presented with. In particular, it is likely that reduced positive amplification would be most strongly linked to the anhedonic symptoms of depression, and previous studies may have differed in how anhedonic their samples were. It also seems likely that dampening of positive emotion would be most strongly associated with anhedonic symptoms, but this possibility has yet to be tested.

A third critical issue to clarify is whether positive emotion regulation disturbances are limited to the acute phase of depression or also persist in recovery. Existing research suggests that deficits in the regulation of negative emotion persist in the euthymic phase of the disorder (Ehring et al., 2008; Werner-Seidler & Moulds, 2012). This might also be the case for the regulation of positive emotional states, although this has not yet been tested. If problems in regulating positive emotion during recovery from depression emerge, longitudinal studies would need to establish whether these difficulties pose a risk for recurrence (and therefore might be a sensible target for relapse prevention approaches). To explore this issue it is first necessary to take a diagnostic approach and compare participants who are in remission to never-depressed individuals to ascertain whether deficits exist in their regulation of positive emotion.

To address these goals we conducted three cross-sectional studies. The first was a correlational study in which we recruited

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