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Emotion in Aging and Bipolar Disorder: Similarities, Differences, and Lessons for Further Research

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Abstract

In this article, we consider similarities and differences in emotion research on older adults and individuals with bipolar disorder (BD). Recent research and theory within both areas has focused on the importance of positive emotion, but the case of older adults is generally considered a case of “adaptive” positivity whereas BD is usually considered maladaptive positivity. We explore the paradox of the same phenomenon being labeled as adaptive in one group and yet maladaptive in another, with attention to commonalities and distinctions between these two groups. We identify only limited areas of overlap, and suggest a refinement of models of positive emotionality in the two populations.

Keywords

aging; bipolar disorder; emotion; emotion reactivity; emotion regulation; late life; mania

Older adults report relatively more positive affective experience than their younger counterparts (Carstensen et al., 2011). This phenomenon has been the focus of considerable research (see Charles, 2010), but without consideration of other populations characterized by elevations in positive affective experience. Elevated positive affective experience is a well-documented aspect of bipolar disorder (BD) and is a cardinal feature of manic episodes. In this article, we take a comparative approach to considering emotion in aging and bipolar disorder. What makes this comparison provocative is that overly positive emotion is considered healthy in the former and disordered in the latter. This suggests from the outset a need to determine mechanisms that could lead to both healthy and unhealthy variants of elevated positive emotion. Hence there are two goals of this article: to consider (a) the extent

of overlap in the positive emotionality observed in late life as compared to BD and (b) similarities and differences in the emotion-relevant processes of these two groups.

Charting the Territory: Older Adults versus BD

How do we define older adults? Aging is a continuous process, though the studies we draw on for this comparison tend to group older adults as those aged 60 and above in comparison to some other age group(s). In a cross-sectional sample of adults ranging from age 18 to 74, older individuals reported both higher self-reported positive affect and lower self-reported negative affect than did their younger counterparts (Mroczek & Kolarz, 1998). A similarly positive profile of age effects has emerged in longitudinal studies (Charles, Reynolds, & Gatz, 2001). However, other studies indicate that the affective picture of the oldest-old (80s and above) might not be as positive as for the younger-old (e.g., Isaacowitz & Smith, 2003). Nonetheless, the bulk of self-report evidence suggests a more positive affective profile associated with age, and these findings dovetail with research suggesting low rates of clinical depression among older adults (Gatz, Kasl-Godley, & Karel, 1996).

Whereas the emotional changes with age tend to be viewed positively, BD is a disabling psychiatric disorder defined by episodes of mania or hypomania. The core diagnostic criterion for mania is a distinct period of abnormally and persistently elevated or irritable mood, accompanied by a number of associated symptoms (e.g., grandiosity, increased goal-directed activity). Depressive episodes are not necessary for the diagnosis of BD but are commonly present during the life course of people with mania. Given the extreme highs and lows, disruptions in emotion responses and regulation are often suggested to be central in explaining BD.

Both remitted BD and subsyndromal BD are associated with greater self-reported dispositional positive affectivity (e.g., Lovejoy & Steuerwald, 1995) and greater positive emotions in response to rewards as measured by self-report, psychophysiology, and behavior (for a review see Johnson, Edge, Holmes, & Carver, 2012). Most importantly, positive affectivity (e.g., Akiskal et al., 1995) and responsivity to rewards predict increases in manic symptoms over time, as well as the onset of BD or conversion from milder to more severe forms (Johnson et al., 2012). Hence elevations of positive affectivity are clearly maladaptive in BD.

Although BD and older age are both posited to involve elevations of positive affective experience, the prevalence of BD is substantially lower (1%) in older adults compared to the prevalence among younger adults (4%; Byers, Yaffe, Covinsky, Friedman, & Bruce, 2010; Merikangas et al., 2007). This diminished prevalence further suggests that the positive affectivity displayed during late life differs from that observed in BD.

We place emphasis in our review on laboratory studies of two processes: *emotion reactivity* and *emotion regulation*. Within each category, we will consider available behavioral, physiological, and neural evidence. While not universally accepted (e.g., Gross & Barrett, 2011), some researchers consider reactivity the initial unregulated response to an emotional stimulus, whereas regulation involves active efforts to manage the response to the stimulus.

We are limited in some cases by a lack of research in making direct comparisons of the aging and BD literatures, and we will note those gaps as they occur.

When possible, we draw a distinction between stimuli that are passively viewed (e.g., standardized facial expressions of emotion) and those that are personally engaging (e.g., recalling an autobiographical memory, or receiving positive or negative feedback on a performance task) or are shown to elicit an emotional response. Personally engaging stimuli are more likely to actually induce an emotional state and thus lead to reactivity and potential regulation. Ideal studies of emotion regulation require two steps: providing a controlled stimulus that would evoke emotion and then examining the slope of recovery from an initial reaction, so we highlight studies with these features.

Emotion Reactivity

Behavioral and Physiological Studies of Emotion Reactivity

Aging—Several studies have investigated whether there are age differences in physiological responsiveness to negative emotion-inducing situations. While early findings indicated an age-related decline in magnitude of autonomic response (e.g., Levenson, Carstensen, Friesen, & Ekman, 1991), one study found equivalent responsivity when elicitors were age-relevant (Kunzmann & Grühn, 2005), and no studies of this type have focused on positive reactivity. Other research has looked at cognitive processing of emotional stimuli in aging. While these studies tend not to examine reactivity per se, they do speak to how older adults respond behaviorally when presented with emotional stimuli. Guided by socioemotional selectivity theory (Carstensen, 2006), a motivational account of changes in emotional goal priority with age, many studies have observed preferences in attention and memory toward positive or away from negative information; this has been referred to as an age-related positivity effect (Carstensen & Mikels, 2005; cf. Grühn, Smith, & Baltes, 2005). However, older adults are just as quick as younger adults at detecting negative stimuli in an array of non-emotional distracters (Mather & Knight, 2006), suggesting age maintenance of automatic responses to negative stimuli.

Bipolar disorder—Behavioral studies provide support for greater emotional reactivity to positive stimuli, and particularly to personally engaging approach-relevant stimuli, among adult bipolar samples (e.g., Hayden et al., 2008), as well as among individuals at risk for BD (e.g., Johnson, Ruggero, & Carver, 2005; cf. Pizzagalli, Goetz, Ostacher, Iosifescu, & Perlis, 2008). Some studies find greater emotional reactivity to negative stimuli among those with BD (e.g., Cuellar, Johnson, & Ruggero, 2009; Depue, Kleinman, Davis, Hutchinson, & Krauss, 1985), but those effects are identified only in samples with depressive symptoms present.

Neuroimaging Studies of Emotion Reactivity

Aging—Some studies find diminished responsivity of the amygdala to negatively valenced stimuli among older compared to young adults (e.g., Mather et al., 2004). However, when negative but novel stimuli (in contrast to familiar, nonemotional stimuli) are presented, fast detection and robust amygdala activity have been observed in older adult samples (Wright,

Wedig, Williams, Rauch, & Albert, 2006). Older adults' maintained neural sensitivity to negative emotional inputs when not instructed to regulate (Wright et al., 2006) suggests that the diminished emotional responsivity to negative stimuli may be specific to regulatory contexts. The pattern of age differences in amygdala activation is sometimes reversed for positive stimuli, with older adults showing greater amygdala activity toward positive relative to negative pictures (Mather et al., 2004). Older adults have also been shown to be more responsive to positive relative to negative stimuli in prefrontal regions (such as the ventromedial prefrontal cortex [VMPFC]) than are younger adults (Leclerc & Kensinger, 2008)

It therefore does not appear that older adults show decreased neural reactivity for all negative stimuli. Rather, older adults show blunted responsivity of the amygdala for familiar stimuli, but also show greater activation of the amygdala to positive stimuli, and normative activation of the amygdala for novel negative stimuli. Diminished responsivity to certain types of negative stimuli may reflect effective emotion regulation processes in old age. However, initial responsiveness to novel negative stimuli may be an adaptive component of regulation as well, as individuals need to be able to detect unexpected threatening stimuli in the environment.

Bipolar disorder—Compared to the findings from old age, findings are perhaps more mixed regarding neural responses to negatively valenced stimuli among bipolar participants relative to controls (e.g., increased, decreased, or no differences in activity within a suite of brain regions). For example, some studies find reduced amygdala activation to negative stimuli among currently depressed (e.g., Chang, Wagner, Garrett, Howe, & Reiss, 2008) or remitted participants (Lagopoulos & Malhi, 2007), whereas other studies have not found group differences in amygdala activation among depressed (e.g., Altshuler et al., 2008) or euthymic (e.g., Hassel et al., 2008) BD participants. Similarly, some studies observe reduced cingulate cortex response to negative stimuli among currently manic participants relative to controls (e.g., Lennox, Jacob, Calder, Lupson, & Bullmore, 2004), whereas others find increased activation in the dorsal/rostral anterior cingulate among euthymic participants in response to a negative stimulus (Krüger, Seminowicz, Goldapple, Kennedy, & Mayberg, 2003). Finally, some studies find reduced dorsolateral response to negative stimuli among depressed (Altshuler et al., 2008) or euthymic participants (e.g., Hassel et al., 2008; Pavuluri, O'Connor, Harral, & Sweeney, 2008). Other studies, however, fail to obtain a frontal response to negative stimuli among euthymic participants (e.g., Malhi et al., 2007), or show increased frontal activation among euthymic and depressed bipolar patients (Lawrence et al., 2004). Thus, findings on neural responsivity to negatively valenced stimuli in BD have been mixed.

Considerably less research is available regarding neural activity in response to positively valenced stimuli. Some studies have shown increased amygdala activation to positively valenced stimuli across phases of illness (e.g., Bermppohl et al., 2009; Lawrence et al., 2004; Malhi et al., 2004) relative to controls. Other studies, however, find no significant differences among manic (Lennox et al., 2004), euthymic (Hassel et al., 2008; Pavuluri et al., 2008), or mixed-state BD participants (Chen et al., 2006) relative to controls. Findings regarding a frontal response to positive stimuli have also been mixed. During a gambling

task, currently manic participants demonstrated reduced activation in the anterior ventromedial prefrontal cortex (Brodmann Area [BA] 10), but increased activation in the dorsal anterior cingulate (BA 32) as compared to control participants (Rubinsztein et al., 2001). Other studies, however, showed an increased prefrontal activation (ventrolateral; BA 10, 11) among a mixed-state sample in response to positive stimuli (Lawrence et al., 2004). Thus, no clear pattern of neural response to either negatively or positively valenced stimuli has emerged.

Emotion Regulation

Experimental Studies of Emotion Regulation

We next consider studies that have directly investigated regulation of an elicited emotional response. We first describe studies of negative emotion regulation followed by studies examining positive emotion regulation. While research on bipolar disorder has investigated regulation of positive and negative states, most regulation studies in the aging literature have focused only on negative states.

Aging—Older adults *report* having better emotion regulation ability as compared to younger adults (Gross et al., 1997), endorsing a more adaptive profile of strategies, including more reappraisal and less suppression, than younger adults. In laboratory studies on down-regulation of negative emotional responses after a sad-mood induction, older adults reported larger initial increases in negative affect but also more effective mood regulation as compared to younger adults (Kliegel, Jäger, & Phillips, 2007). Thus, even though older adults appeared to be more emotionally reactive, they were also more successful at regulating their negative affective state.

Gaze preferences in response to a negative mood induction have also been studied to understand emotion regulation. In one study (Isaacowitz, Toner, Goren, & Wilson, 2008), young adults reporting a negative mood demonstrated mood-congruent gaze preferences: they fixated significantly toward negative facial expressions. On the other hand, older adults in a negative mood demonstrated regulatory gaze preferences: they fixated more toward positive and away from negative faces. Among older adults, such positive gaze preferences may predict less mood decline (Isaacowitz, Toner, & Neupert, 2009).

Other studies have investigated age differences in the preferential use, or effectiveness, of particular emotion regulation strategies. A common finding is the lack of age differences in expressive suppression (inhibiting behavioral expressions of emotion; e.g., Phillips, Henry, Hosie, & Milne, 2008; Shiota & Levenson, 2009). Findings have been mixed for reappraisal. In one study, older adults who were instructed to use a reappraisal strategy in response to a film clip were more successful than younger adults at decreasing self-reported negative affect (Phillips et al., 2008). However, the effectiveness of reappraisal may depend on the type of reappraisal strategy used. Shiota and Levenson (2009) instructed participants to decrease their emotional response to negative film clips by using one of three emotion regulation strategies (detached reappraisal in which they were instructed to adopt an unemotional tone, positive reappraisal in which they were asked to think of positive aspects of the situation, and expressive suppression). There was age-related decline in the

effectiveness of detached reappraisal, but improvement in the effectiveness of positive reappraisal. These age effects may reflect the underlying resources necessary for the particular strategies: detached reappraisal requires participants to inhibit processing of salient, negative stimuli and thus may rely heavily on cognitive control and executive functioning resources (Opitz, Rauch, Terry, & Urry, 2012), whereas positive reappraisal may not.

Scheibe and Blanchard-Fields (2009) found that down-regulating a disgust response had a negative impact on young adults' performance of an n-back task, but did not impair older adults' performance on the n-back. These findings suggest that older adults may benefit from their lifetime of experience in regulating their feeling states, such that they may have become more efficient in doing so (Blanchard-Fields, 2007), perhaps due to repeated rehearsal of successful regulatory strategies.

In sum, research suggests that older adults describe themselves as more skilled at emotion regulation than younger adults, and they endorse a more adaptive range of strategies. They appear to be able to implement certain emotion regulation strategies more effectively in laboratory settings than do younger adults, and to experience less interference from cognitive demand in doing so. On the other hand, the choice of strategy may be particularly important; the greater regulatory ability of older adults may only be apparent when they are using strategies that match their resources (Urry & Gross, 2010).

Bipolar disorder—Little research has examined regulation after positive-mood inductions. Findings of one study demonstrated that after a mood induction involving reward, the control group's positive affect scores returned to baseline levels, whereas the bipolar group's scores remained elevated (Farmer et al., 2006), highlighting the importance of examining the slope of recovery from an emotional stimulus.

Two experimental mood manipulation studies have examined the ability of bipolar participants to regulate negative emotion. In one study, participants were asked to describe a difficult life stressor during a dyadic interaction with a confederate. The confederate delivered a standardized, critical statement that took a blaming tone. People with remitted BD did not differ significantly from control participants in their initial affect or their affective recovery during the 9 minutes after the criticism (Cuellar et al., 2009). A different study, though, suggested that it might be important to consider a longer time period and mood state at the time of the challenge: patients diagnosed with cyclothymia (a mild form of BD) showed poor cortisol regulation 3 hours after a time pressured math test (Depue et al., 1985).

Consistent with the idea of a very slow recovery from mood states, one study identified effects of mood inductions on the ability to fall asleep. People with BD took longer to fall asleep than did controls after positive-mood induction but not after a negative-mood induction (Talbot, Hairston, Eidelman, Gruber, & Harvey, 2009). As sleep disturbance can be a trigger of mania, these results highlight the potentially deleterious outcomes of difficulties in regulating positive mood.

What cognitive and regulatory responses might help explain the slower recovery from mood inductions within BD? People with BD report engaging in many different strategies to help regulate their emotions, as well as early signs of symptoms. For example, people at risk for BD endorse tendencies to dampen and control positive emotions (Johnson & Jones, 2009). Similarly, in naturalistic studies, people with BD reported that they reduced goal-directed activity (e.g., reduced social and occupational activities, increased rest) in the context of early manic symptoms (Lam & Wong, 1997); such strategies have been linked to lower risk of relapse over time (Lam, Wong, & Sham, 2001). Tendencies to dampen positive affect may reflect a perceived need to down-regulate more intense and frequent positive moods. In laboratory settings, emotion regulation strategies do seem to help people with remitted BD to reduce high moods (Gruber, Harvey, & Johnson, 2009). One possibility is that people with BD are able to employ these strategies effectively until moods escalate. Hence there is a need to examine the engagement of regulatory responses in daily life, outside of laboratory studies that provide direct instruction to regulate in a given moment.

In parallel, people with BD reported increased goal engagement in response to early signs of depressive symptoms (e.g., kept busy, became more social; Lam & Wong, 1997). At the same time, people with BD, even during remission, endorsed frequent use of responses to negative moods that may be maladaptive, such as ruminative (e.g., thinking about one's shortcomings, failures, and faults) and sensation-seeking responses (e.g., pleasant distraction; Thomas, Knowles, Tai, & Bental, 2007).

Neuroimaging Studies of Emotion Regulation

Ageing—Recent research has compared neural mechanisms involved in emotional responding and emotion regulation in younger and older adults. A number of studies have observed coupling of PFC (namely medial PFC) with the amygdala when processing emotionally evocative negative stimuli (Leclerc & Kensinger, 2011; St. Jacques, Dolcos, & Cabeza, 2010). Diminished amygdala activity in response to negative stimuli (or increased activity in response to positive stimuli; Mather et al., 2004), as well as activation within regions associated with cognitive control (e.g., VMPFC), may be reflective of emotion regulation processes. However, this evidence cannot speak directly to whether such response patterns reflect emotion regulation, as participants were not instructed to regulate. Thus, more pertinent evidence comes from studies examining neural activation patterns in response to explicit emotion regulation tasks.

First, age is not always related to activation of several of these regions: older and younger adults have similar regulation-related activity within dorsolateral (BA 45) and dorsomedial (BA 6, 9) PFC, as well as similar connectivity between lateral PFC and amygdala (Winecoff, LaBar, Madden, Cabeza, & Huettel, 2011), during voluntary emotion regulation tasks. Regions within medial PFC (namely dorsomedial prefrontal cortex, BA 6; dorsal cingulate gyrus, BA 32) have been associated with changes in amygdala activity predictive of physiological arousal in response to instructions to reappraise negative stimuli in old age (Urry, van Reekum, Johnstone, & Davidson, 2009). Given that researchers have found preservation of the structure and function of the amygdala in older adult samples (Grieve, Clark, Williams, Peduto, & Gordon, 2005), findings of diminished amygdala activity in

response to emotion regulation prompts in old age are more likely to result from controlled emotional reactivity via the PFC rather than neural degradation of the amygdala (as suggested by the aging brain model; Caccioppo, Berntson, Bechara, Tranel, & Hawkley, 2011).

Additional evidence of amygdala–PFC (namely medial PFC) interactions during voluntary emotion regulation comes from studies assessing only older adult samples. In one study, decreased amygdala activity corresponded with increased activity in VMPFC (BA 11, 32) when older adults were attempting to decrease as opposed to amplify their reactions to negative stimuli (Urry et al., 2006). Activation within dorsomedial and ventrolateral PFC along with decreased amygdala activation has also been observed when older adults dampen their reaction to negative stimuli (van Reekum et al., 2007). Thus, while older adults may show declines in cognitive control abilities, the relative structural preservation within certain prefrontal regions (e.g., medial prefrontal cortex [mPFC]; Tisserand et al., 2002), as well as adequate connectivity with the amygdala, suggests that older adults are able to recruit neural resources necessary for successful emotion regulation.

Bipolar disorder—In the only research using positron emission tomography (PET) to study emotion regulation in BD, persons with BD demonstrated diminished blood flow in the dorsal and ventral medial frontal cortex (BA 9/10) and orbitofrontal cortex (BA 11) after a negative mood induction. Remitted bipolar patients exhibited more robust decreases in activation of the medial frontal cortex than the depressed BD participants, and also showed unique increases in activation of the dorsal anterior cingulate cortex (ACC; BA 24a/32) compared to healthy controls (Krüger et al., 2003). Since the medial frontal cortex is implicated in reappraisal of personally relevant emotional stimuli (e.g., Fossati et al., 2003), reduced activation in this region may reflect a potentially stable vulnerability among BD participants in regulating negative information (Krüger et al., 2003). The dorsal ACC is thought to be involved in attention to salience of, and monitoring of emotional response to, stimuli (e.g., Whalen et al., 1998). Thus, the pattern for remitted patients of more robust decreases in activation of the medial frontal cortex, along with the unique activation of the dorsal ACC, suggests that there are regional interactions that may underlie difficulties in emotion regulation (Krüger et al., 2003).

Three studies have used functional connectivity methods to examine brain activity while participants with BD label negative (either sad or fear) or happy facial expressions. These studies have examined how tightly coupled activity in the amygdala is with activity in regions involved in emotion regulation, such as the perigenual anterior cingulate cortex (Wang et al., 2009), the orbitomedial prefrontal cortex (BA 11, 47, and 25; Almeida et al., 2009), and the ventrolateral prefrontal cortex (BA 11; Versace et al., 2010). The findings of these studies have been mixed regarding responses to negative stimuli. In contrast to the mixed findings regarding negative stimuli, findings consistently indicate diminished functional connectivity of prefrontal regions with the amygdala while participants with BD label happy faces. That is, bipolar participants have been found to exhibit diminished connectivity of the perigenual ACC (Wang et al., 2009), the OMPFC (BA 11, 47, and 25; Almeida et al., 2009), and the ventrolateral prefrontal cortex (VLPFC) (BA 11; Versace et al., 2010) with the amygdala as compared to controls. Almeida's analyses were specific in

indicating that the diminished connectivity was due to decrements in bottom–up connectivity, and noting that this pattern was distinct from that observed among persons with major depressive disorder (MDD).

In summary, three studies indicate that BD is characterized by diminished connectivity of prefrontal cortex regions with the amygdala while evaluating positive stimuli, a profile that could help explain the findings we have noted of sustained positive mood elevations in behavioral studies of this population (cf. Farmer et al., 2006).

Conclusions: Similarities and Differences in the Emotionality of Late Life and BD

Evidence for increases in positive trait affectivity up until very old age, maintained or increased emotion regulation competency with age, and prioritization of positive relative to negative stimuli in information processing among older adults suggests a positive age-related affective trajectory. While older adults show maintained detection of negative stimuli (Mather & Knight, 2006), top–down control of amygdala responses to negative stimuli in ventral and medial regions of PFC during voluntary emotion regulation tasks suggests good emotion regulation ability in aging (Urry et al., 2006).

For those with BD, behavioral and neural evidence provides evidence for greater reactivity to positively valenced stimuli, and more specifically to cues of reward among those with BD, but not for greater reactivity to negative stimuli. Some of the most clear-cut behavioral evidence suggests a pattern of prolonged response to positive feedback, and functional connectivity studies indicate that people with BD may display differences in regulatory (but not reactivity) regions (e.g., perigenual ACC, orbitofrontal and adjacent medial prefrontal cortex [OMPFC]) in response to positive stimuli as well as diminished activation of the prefrontal cortex during exposure to negatively valenced stimuli. Taken together, these findings indicate that increased reactivity to positive stimuli, coupled with deficits in the ability to regulate responses to emotionally relevant stimuli of either valence, may be core to the disorder, and that these deficits may be biologically based. Despite this evidence that people with BD have difficulty regulating their moods, they endorse more frequent use of strategies to try to regulate their moods than do healthy controls, and in laboratory studies, they can use these strategies to good effect. This would suggest that at least some of these strategies are failing in more demanding contexts or not being implemented at the right time. Aging may bring better top–down control and selection of emotion regulatory strategies, whereas the poor neural connectivity of individuals with BD may be a biological constraint on their ability to select and deploy emotion regulatory strategies in situations where they are needed.

This leads to a few key domains that seem to share a good deal of commonality, and others that diverge. BD and aging both appear to involve elevated positive affective traits and may perhaps involve positive emotional reactivity. Some caution is warranted in that different measures and methodologies have been used in the two literatures, and the lack of research on reactivity to positive stimuli in aging constrains this particular parallel. In contrast, compared to those with BD, elderly individuals appear to be better at down-regulating

negative emotion, though this is true only for some regulatory strategies (e.g., positive reappraisal). This conclusion has some support on the neural level as well, given the relatively strong evidence suggesting connectivity between the amygdala and prefrontal control regions (namely medial PFC) during explicit emotion regulation of negative stimuli (Urry et al., 2009; Urry et al., 2006). Furthermore, older adults' enhanced activation of both the amygdala and PFC in response to positive relative to negative stimuli (Leclerc & Kensinger, 2010, 2011) could also reflect age-related improvements in the regulation of negative affect that balance the positive.

Studies with bipolar patients provide mixed support for connectivity between the amygdala and prefrontal regions in response to negative stimuli, but consistent support for reduced connectivity in response to positive stimuli (Almeida et al., 2009; Versace et al., 2010; Wang et al., 2009). To the extent that functional connectivity is an index of regulatory ability, these findings are suggestive of the idea that persons with BD may have difficulties in their ability to recover from a positive emotional state.

While our ability to draw conclusions from these patterns is constrained by the lack of fully symmetric data (i.e., no studies of regulation of positive emotional states in aging), these patterns nonetheless suggest that the adaptiveness of displaying high levels of trait positive affectivity and positive emotion reactivity depends on regulatory capacity. Older adults seem able to use their regulatory abilities to minimize negative responses and, presumably, to maintain positive ones as well. These regulatory abilities are expressed behaviorally, and appear to have parallels in neural functioning. In contrast, the positive affectivity in the context of deficits in regulatory ability observed in BD appears to be maladaptive. Both the behavioral and neural data suggest an inability to dampen positive states. Of course, testing this model will depend on more careful research on how older people respond to positive emotions, whether they endorse different regulatory responses, and whether those responses differentially influence the duration of a positive mood. Little is known about the neural correlates of positive mood regulation in late life either.

Taking a step back from the observed similarities and differences in the phenomenology of emotion in aging and BD, some conclusions can be drawn from the conceptual models used to explain why the regulatory capacity diverges for these two target populations. Socioemotional selectivity theory (Carstensen, 2006) posits that age-related changes in regulatory capacity result from motivational processes that lead older adults to prioritize well-being and emotion regulation over other possible goals. In other words, aging may bring with it positive affective experience and regulation because older adults select feeling good rather than, for example, learning new information or having new experiences. From this perspective, possible age differences in neural responding could result from changing goals (Samanez-Larkin & Carstensen, 2011). Age-related differences could also come from a lifetime of experience regulating emotions (Blanchard-Fields, 2007). While an alternative viewpoint has been posited by Cacioppo and colleagues' (2011) aging brain model, in which changes in the brain are thought to be key to lifespan changes in emotional experience and responding, the findings we reviewed suggest that adaptive regulation of negative inputs in old age may be more influenced by top-down factors than simply a beneficial side effect of neural decrement.

In contrast, BD is believed to be strongly heritable (McGuffin et al., 2003), and there is little support for a motivational account. So it would make some sense to differentiate positive affectivity that is based on a top-down effortful regulatory process, as compared to positive affectivity that might be biologically based, and to involve a deficit in the ability to engage brain regions involved in regulation.

That is, in a population without regulatory deficits, trait-like positive affectivity and positive reactivity would seem to be largely beneficial. In the context of poor regulatory constraints, a different picture emerges, characterized by difficulties activating key prefrontal cortical regions, cognitive deficits, and more sustained affective states. A take-home point for the bipolar literature would be that it may be difficult to take a trait or emotion approach to this disorder without considering how the underlying biology comes into play. At a behavioral level, regulatory capacity may be the key feature of positive emotionality to consider. Without such models, the field can easily end up describing psychological traits that provide only unsatisfying answers to the question of what factors define vulnerability to BD. In this case, the very traits that are seen as related to BD and its manic symptoms appear to be related to beneficial aspects of aging.

At the same time, these contrasting conceptual frameworks are not independent of which outcomes are conceptualized as adaptive versus maladaptive. In other words, positive emotionality in aging is thought to be adaptive largely because it is often considered as the outcome of motivational processes, whereas positive emotionality in BD is considered maladaptive because it is thought to be rooted in genetics and biological processes and believed to be related to a worse course of symptoms. But, to the extent that age differences correspond to neural differences as well, there is no logical reason for this strict dichotomy. Furthermore, to some extent these theories have constrained the questions that have been asked and the methods used to investigate those questions. While there may be good empirical reasons to favor one set of explanations for aging and another for BD, one message of the current analysis is that both fields may be served by keeping an open mind to diverse frameworks and methods. Why not, for example, investigate the neural correlates of up-regulation of positive emotion in older adults, or the implementation of positive reappraisal in those with BD?

In sum, evidence does support the idea that both aging and BD relate to elevations of positive emotionality. Contrasting the two populations, though, suggests the need to be more specific in conducting research on the neural and regulatory mechanisms guiding this positive affectivity. It is our hope that the comparison of these two conditions leads researchers to be more interested in defining the adaptive and maladaptive aspects of positive emotionality within both populations.

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