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Addressing Neuroticism in Psychological Treatment

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Neuroticism has long been associated with psychopathology and there is increasing evidence that this trait represents a shared vulnerability responsible for the development and maintenance of a range of common mental disorders. Given that neuroticism may be more malleable than previously thought, targeting this trait in treatment, rather than its specific manifestations (e.g., anxiety, mood, and personality disorders), may represent a more efficient and cost-effective approach to psychological treatment. The goals of the current manuscript are to (a) review the role of neuroticism in the development of common mental disorders, (b) describe the evidence of its malleability, and (c) review interventions that have been explicitly developed to target this trait in treatment. Implications for shifting the focus of psychological treatment to underlying vulnerabilities, such as neuroticism, rather than on the manifest symptoms of mental health conditions, are also discussed.

Keywords: neuroticism, temperament, treatment

Neuroticism is typically defined as the tendency to experience frequent and intense negative emotions in response to various sources of stress. While the emotions considered within the purview of this trait include the range of negative affect (e.g., fear, irritability, anger, and sadness), the greatest focus has been on the experience of anxious and depressive mood. The perception that the world is a dangerous and threatening place also accompanies this exaggerated negative emotionality, along with beliefs about one's lack of agency to handle challenging events. Manifestations of this trait may include heightened focus on criticism, either self-generated or from others, as confirming a general sense of inadequacy and perceptions of lack of control over salient events (Barlow, 2002; Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014; Clark & Watson, 2008; Eysenck, 1947; Goldberg, 1993).

Barlow and colleagues (Barlow et al., 2014) have forwarded a model to account for the development of neuroticism informed by an interaction of biological and psychological vulnerabilities. To summarize, the general biological risk is evidenced by high heritability estimates (40 to 60%) for this trait (e.g., Bouchard & Loehlin, 2001; Clark, Watson, & Mineka, 1994; Kendler, Prescott, Myers, & Neale, 2003), with genetically mediated neuroticism linked to the neurobiological tendency for heightened reactivity in emotion-generating structures, including amygdala hyperexcitability, and reduced inhibitory control by prefrontal systems (Keightley et al., 2003; Stein, Simmons, Feinstein, & Paulus, 2007; Westlye, Bjørnebekk, Grydeland, Fjell, & Walhovd, 2011). These neural circuits are also influenced by a general psychological vulnerability that includes stressful, traumatic, or related developmental experiences during childhood, cultivating the perception that life events are unpredictable and uncontrollable (Gunnar & Quevedo, 2007; Lanius, Frewen, Vermetten, & Yehuda, 2010; Rosen & Schulkin, 1998). Taken together, these general biological and psychological vulnerabilities build upon each other to produce the neurotic phenotype. More important, these interacting vulnerabilities were originally described as part of an etiological model for trait anxiety and emotional disorders more generally (Barlow, 2000, 2002), suggesting that a neurotic temperament may be a necessary component for the development of a range of psychological conditions. In fact, all the temporal covariance among the *Diagnostic and Statistical Manual for Mental Disorders* (*DSM*; American Psychiatric Association, 2013) constructs of depression and several anxiety disorders can be accounted for by neuroticism (Brown, 2007).

Neuroticism has been associated with a wide range of public health problems (see: Lahey, 2009). For example, this trait strongly predicts a variety of mental disorders as well as comorbidity among them (Clark et al., 1994; Khan, Jacobson, Gardner, Prescott, & Kendler, 2005; Krueger & Markon, 2006; Weinstock & Whisman, 2006), including personality disorders (Henriques-Calado, Duarte-Silva, Junqueira, Sacoto, & Keong, 2014; Sauer-Zavala & Barlow, 2014; Widiger, Verheul, & van den Brink, 1999). Neuroticism has also been associated with a range of physical problems including cardiovascular disease, eczema, asthma, and irritable bowel syndrome (Brickman, Yount, Blaney, Rothberg, & De-Nour, 1996; Smith & MacKenzie, 2006; Suls & Bunde, 2005). Additionally, this trait predicts treatment seeking and response to treatment for both mental disorders and general health concerns (Shipley, Weiss, Der, Taylor, & Deary, 2007). Considering these public health consequences, it is not surprising that the economic cost of this trait exceeds the cost of common mental disorders (Cuijpers et al., 2010).

A discussion of the public health costs associated with neuroticism prompts consideration of how this trait may be addressed. As noted above, the transactional relationship between genetic and environmental inputs for neuroticism suggests that the inherited contributions of this trait may serve as a predisposition, but not a

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mandate. This opens the question of whether neuroticism can be treated directly, rather than separately targeting each manifestation of this trait in the form of separate mental health and physical disorders. This approach is consistent with National Institute of Mental Health's Research Domain Criteria (RDoC) initiative that challenges researchers to look beyond diagnoses to identify core processes implicated in the development and maintenance of symptoms across a range of disorders (Insel et al., 2010). Targeting neuroticism itself may represent a more efficient and cost-effective means of addressing the wide swath of public health problems associated with it.

Malleability of Neuroticism

Although personality traits have long been considered stable and inflexible across time (American Psychiatric Association, 2013), there is increasing evidence that neuroticism may be more malleable than originally believed. For example, in a review of the literature regarding the stability of personality disorders, Clark (2009) notes that the traits associated with these diagnoses do change slowly over time, with the greatest shifts occurring in the behavioral manifestations of these traits. Further, longitudinal studies of the general population also show gradual age-related decreases in neuroticism and related constructs (i.e., internalizing) that continue into old age (Eaton, Krueger, & Oltmanns, 2011; Roberts & Mroczek, 2008; Roberts, Walton, & Viechtbauer, 2006). Overall, this pattern of results suggests that, on average, neuroticism decreases across time; however, when change in neuroticism has been examined at the individual level using growth modeling (Mroczek & Spiro, 2003), there appears to be great variability in the extent of change, with some people maintaining a stable level of this trait and others changing considerably (Helson, Jones, & Kwan, 2002; Small, Hertzog, Hultsch, & Dixon, 2003). Indeed, there is evidence to suggest that individuals with higher initial levels of neuroticism tend to show less change in this dimension over time, and conversely, individuals with lower initial levels of neuroticism tend to evidence greater change (Brown, 2007).

Change in neuroticism has also been explored in the context of individuals seeking treatment for *DSM* disorders. For example, in a study that tracked 41 individuals with major depressive disorder (MDD) across 8 months (most of whom received some kind of treatment during this time), neuroticism remained stable, despite changes in clinical state (Eaton et al., 2011). Specifically, neuroticism displayed the same high level of temporal stability in individuals who no longer met criteria for MDD as it did in the subgroup of participants who were depressed at both assessment points. In contrast, others have indeed found changes in neuroticism as a function of time and treatment. For example, Brown and colleagues (2007; Brown & Barlow, 2009) examined the temporal course of neuroticism and its relationship to the *DSM–IV* disorder constructs of depression, social anxiety disorder, and generalized anxiety disorder in a large sample ($N = 606$) of outpatients with these disorders. Participants were assessed at intake and then reassessed at 1-year and 2-year follow-ups with the majority (76%) of patients receiving some kind of treatment (of varying quality and duration) during the follow-up period. As expected, *DSM–IV* disorder constructs improved significantly over time and other temperamental variables (e.g., extraversion) remained stable; neuroticism, however, evidenced the greatest amount of temporal change and was the dimension associated with the largest treatment effect.

In addition to studies exploring the malleability of neuroticism in treatment-seeking samples where the treatment received was unspecified, changes in neuroticism have also been examined in the context of specific interventions. First, Kring, Persons, and Thomas (2007) assessed the degree to which neuroticism (described as negative affectivity in this study) and symptoms of depression and anxiety changed across a course of cognitive–behavioral therapy. Results suggest that neuroticism, as well as symptoms of depression and anxiety, decreased following the 12-week course of treatment; unfortunately, it is difficult to determine whether change in temperament is driving change in symptoms or vice versa as the study did not include multiple repeated measures of these variables. Additionally, in a large randomized-controlled trial of cognitive therapy (CT) compared with placebo for adults with major depressive disorder (Tang et al., 2009), CT produced greater changes in both neuroticism and extraversion than placebo. However, contrary to Brown's (2007) results, after controlling for changes in depressive symptoms, the effect of CT on these temperamental variables remained significant only for extraversion. Finally, levels of neuroticism did not appear to change significantly after a course of treatment with dialectical behavior therapy for individuals with borderline personality disorder (Davenport, Bore, & Campbell, 2010).

The inconsistent results described above may be because of the fact that the measurement of neuroticism consists of some combination of stable temperamental variance and variability attributable to generalized distress that is apt to covary with temporal fluctuations in the severity of disorders. While this measurement issue, known as mood state distortion, is one possible explanation of the diverse findings with regard to temperamental malleability, there is also a considerable body of compelling research to suggest that these fluctuations represent actual changes in the level of neuroticism secondary to situational stressors. Given that neuroticism is chiefly defined as the tendency to experience negative emotions, it is reasonable to consider mood state fluctuations that affect self-reported levels of neuroticism as direct expressions of this trait (Costa, Bagby, Herbst, & McCrae, 2005; Widiger & Smith, 2008). Indeed, results from a recent study indicate that measures of neuroticism primarily capture true temperamental variance even in individuals with emotional disorders (Naragon-Gainey, Gallagher, & Brown, 2013).

Similarly, to the extent that high neuroticism is necessary for the development of a range of common mental disorders, one would expect that this trait would vary in accordance with the onset or remission of symptoms. In fact, a number of longitudinal studies have controlled for the periodic occurrence of anxious or depressive symptoms and still found neuroticism to act independently in predicting anxiety and mood (Lahey, 2009; Spijker, de Graaf, Oldehinkel, Nolen, & Ormel, 2007). Further, while neuroticism predicts the course *DSM* disorders, with higher levels of this trait reflecting less change in symptoms across time, the converse does not appear to occur; that is, initial levels of *DSM* disorders do not predict changes in temperament over time (Brown & Rosellini, 2011; Gershuny & Sher, 1998; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Meyer, Johnson, & Winters, 2001). This pattern of

results, personality traits predicting symptoms but not vice versa, has also been found for personality disorders (Warner et al., 2004).

In summary, there appears to be evidence that neuroticism is subject to change slowly over across the course of the life span (e.g., Clark, 2009; Roberts & Mroczek, 2008); however, the degree to which this and other temperamental variables are malleable in response to treatment remains unclear. The studies described above examined changes in neuroticism in the context of naturalistic treatments or treatments targeting disorder-specific symptoms and have yielded mixed results. Indeed, the research reviewed raises questions about the mechanisms through which neuroticism changes and whether directly targeting this trait in treatment would lead to more definitive results.

Treatment of Neuroticism

Most studies that have examined changes in temperament in response to psychological treatment, including the studies described in the previous section, utilize interventions that were not specifically designed to target neuroticism itself, but rather to address *DSM* disorder symptoms. It is possible that the lack of a priori specification regarding how and why these treatments impact temperament may have led to the mixed findings on the responsiveness of neuroticism to treatment; in other words, some interventions may be more suited to address neuroticism than others. Emerging research, however, suggests that when neuroticism is addressed directly with interventions explicitly designed to target it, change on this construct is more robust. This literature, including specific interventions for neuroticism and the putative pathways through which they target it, are reviewed below.

Most of the studies that examine interventions specifically designed to target temperament have come from the psychopharmacology literature (for review, see: Ilieva, 2015; Soskin, Carl, Alpert, & Fava, 2012). Serotonergic drug agents (i.e., selective serotonin reuptake inhibitors) appear to produce dampening effects on neuroticism (Fu et al., 2004; Harmer, Mackay, Reid, Cowen, & Goodwin, 2006; Harmer et al., 2009; Murphy, Yiend, Lester, Cowen, & Harmer, 2009; Quilty, Meusel, & Bagby, 2008), while catecholaminergic (i.e., noradrenergic/dopaminergic) drugs enhance extraversion (McCabe, Mishor, Cowen, & Harmer, 2010; Tomarken, Dichter, Freid, Addington, & Shelton, 2004). Clear hypotheses relating these agents to neurobiological changes have been forwarded that underscore their effect on temperament. For example, serotonergic agents likely produce dampening of neuroticism because of their ability to decrease hyperreactivity of the amygdala in response to fear-inducing stimuli and to inhibit dopaminergic neurotransmission in areas of the prefrontal cortex. In contrast, catecholaminergic agents may be associated with increased extraversion because of their ability upregulate noradrenergic and dopaminergic neurotransmission, particularly within the mesolimbic reward circuitry (Soskin et al., 2012). The fact that specific pharmacological agents display preferential effects on dimensions of temperament suggests that there may be utility in designing behavioral treatments to selectively address temperamental variables.

Currently, there are few behavioral interventions that are explicitly designed to target temperament. One of the first examples of a treatment developed to address temperamental vulnerabilities, rather than psychological symptoms, is Rapee, Kennedy, Ingram, Edwards, and Sweeney (2005) intervention for behavioral-inhibited children; the goal of addressing behavioral inhibition was to prevent the onset of future anxiety disorders. The program focuses on parenting training to minimize augmentation of the child's biological nature through environmental interactions and includes psychoeducation about the nature of anxiety, traditional cognitive–behavioral strategies (i.e., exposure and cognitive restructuring) directed toward personal concerns, and training in behavior management techniques that prevent an overprotective parenting style. Results from randomized controlled trials (Rapee et al., 2005; Rapee, Kennedy, Ingram, Edwards, & Sweeney, 2010) suggest that this program is indeed successful at preventing anxiety disorders. Although a brief version of this program did not appear to produce significant changes in behavioral inhibition measured via parent report or laboratory observation (Rapee et al., 2010), a more intensive version with higher risk children indeed led to significantly greater reductions in this trait compared with those who did not receive the treatment (Kennedy, Rapee, & Edwards, 2009). Differences among groups increased over time, leading Rapee et al. (2010) to speculate that more intensive interventions directed at temperament might produce an increasing trajectory of change in temperament compared with addressing more surface-level disorder symptoms.

The Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (UP; Barlow et al., 2011) represents another example of an intervention explicitly developed to address temperamental vulnerabilities, in this case neuroticism, associated with common mental health conditions. As noted above, neuroticism is implicated in the development of the range of anxiety, depressive, and related disorders (e.g., somatic symptom disorders, trauma-related disorders), also known as "emotional disorders" (Barlow, 2002) given the role of strong emotions in their development. In addition to frequently occurring negative emotions, emotional disorders are also characterized by aversive reactions to emotional experiences, leading to problematic efforts to escape or avoid them (see: Barlow et al., 2014; Sauer-Zavala & Barlow, 2014). To address neuroticism, the UP consists of six core treatment modules aimed at extinguishing distress in response to the experience of strong emotions. The rationale for this aim is that with fewer aversive reactions to negative emotions when they occur, reliance on avoidant emotion regulation strategies that exacerbate symptoms is reduced, which in turn leads to less frequent and intense negative emotions over time. This approach has shown efficacy for the range of anxiety and unipolar depressive disorders (Barlow et al., 2016; Boswell, Anderson, & Barlow, 2014; Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010; Farchione et al., 2012) and there is preliminary support for the use of the UP with bipolar disorder (Ellard, Deckersbach, Sylvia, Nierenberg, & Barlow, 2012) and posttraumatic stress disorder (PTSD; Gallagher, 2015).

In addition, the emotional disorders framework has been extended BPD and the related phenomena of suicidal and nonsuicidal self-injurious behavior (Bentley, Nock, & Barlow, 2014; Bentley et al., 2016; Sauer-Zavala & Barlow, 2014). The presentation of BPD is largely accounted for by neuroticism, with 4 out of 7 traits (emotional lability, anxiousness, separation insecurity, and depressivity) included in the in *DSM–5*'s alternative model of BPD stemming from the parent factor of neuroticism from the Five Factor Model (FFM) of personality (American Psychiatric Asso-

ciation, 2013). Additionally, the impulsive behaviors that characterize BPD, including suicidal and nonsuicidal self-injury, are often conceptualized as maladaptive attempts to reduce or escape from intense negative affect (Chapman, Gratz, & Brown, 2006). Although potentially more life-threatening, these BPD-related behaviors serve a similar function (relief from negative emotions) as leaving a social situation might for an anxiety patient or taking a nap for a depressed patient. Consequently, skills from the UP targeting the aversive, avoidant reactions to emotions that maintain both neuroticism and symptoms of emotional disorders are also relevant for BPD. In fact, the UP or its skill modules have been used to successfully treat BPD (Sauer-Zavala, Bentley, & Wilner, 2016), nonsuicidal self-injury (Bentley, Nock, Sauer-Zavala, Gorman, & Barlow, 2016), and suicidal thoughts and behaviors (Bentley et al., 2016).

The modules of the UP have been described in detail elsewhere (e.g., Payne, Ellard, Farchione, Fairholme, & Barlow, 2014); however, a summary of how the six core modules cultivate a more accepting stance toward emotional experiences is described here. First, by providing information about the adaptive, functional nature of emotions in the psychoeducation module (core module 1), patients begin to cultivate the stance that emotions provide useful information and should not be avoided. Next, patients receive instruction on *how* to engage willingly, versus with avoidance, with their emotional experiences via mindfulness training (core module 2); specifically, patients are taught the benefits of a present-focused, nonjudgmental attitude toward their emotions through three complimentary experiential exercises.

Following the presentation of the UP orientation (e.g., cultivating a more accepting stance toward emotions), patients are taught specific skills that map onto three interacting components of an emotional experience (i.e., thoughts, behaviors, and physical sensations). First, patients are encouraged to be more flexible in the way they appraise emotion-eliciting situations (core module 3); patients are instructed to question the automaticity of interpretations, rather than to change maladaptive cognitions—in line with the UPs emphasis on acceptance. The following module, countering emotional behaviors (core module 4), involves the identification of patient-specific avoidance behaviors that hinder full exposure to strong emotions. Patients are then instructed to act counter to their emotion-driven behavioral urges by engaging in activities that may put them in contact with strong emotions in the short-term. Next, patients cultivate a greater tolerance of physical sensations through interoceptive exercises (e.g., hyperventilation, breathing through a thin straw) that deliberately provoke the physiological feelings associated with strong emotions (core module 5). Finally, treatment culminates with emotion exposures (core module 6) in which patients engage in a series a series of activities that elicit strong or uncomfortable emotions. Through this practice, patients' aversive reactions to emotions are gradually extinguished via new learning that emotions are temporary and can be tolerated.

There is evidence to suggest that the approach to targeting temperament utilized in the UP indeed leads decreases in aversive reactions to emotions that in turn leads to changes in negative affectivity (Sauer-Zavala et al., 2012). Additionally, in the context of a small randomized-controlled trial (i.e., Farchione et al., 2012), the UP produced small to moderate effects on measures of neuroticism from pre- to posttreatment compared with a waitlist group, and these changes were maintained at the 6-month follow-up assessment point (Carl, Gallagher, Sauer-Zavala, Bentley, & Barlow, 2014). In addition, these changes in neuroticism predicted decreased anxiety and depressive symptoms and reductions in functional impairment. Overall, the results of this investigation suggest in a preliminary manner that neuroticism can be successfully targeted in treatment and that reductions in this temperamental vulnerability also affect treatment outcomes.

More recently, Armstrong and Rimes (2016) conducted a pilot study examining the effect of mindfulness-based cognitive therapy (MBCT; Segal, Teasdale, & Williams, 2002) that had been explicitly modified to directly target levels of neuroticism. The version of MBCT used in this study included references to neuroticism-related constructs, rather than depression-related themes. For example, Session 1 covered stress-reactivity by introducing the fight-or-flight response, the role of the hypothalamic-pituitary-adrenal axis, and unhelpful ways of responding to stress (i.e., overthinking and avoiding). Session 2 involved discussion about the relationship between thoughts and feelings as well as common interpretation biases, and in Session 3, genetic and environmental contributions that make an individual susceptible to stress are discussed. In Session 4 patients learn about the long- and short-term consequences of avoiding stress-eliciting situations and Session 5 extends this discussion by presenting the notion that avoiding such situations leads to increased suffering. Sessions 6 and 7 cover additional maladaptive, neurotic responses to stress including overthinking and self-criticism. Finally, Session 8 ties together the skills for "stress management" that had been learned previously in the treatment. After 8-weeks of treatment, participants in the MBCT condition demonstrated significantly greater reductions in neuroticism than participants in the Internet-based self-help control condition.

Conclusions

Contrary to theoretical conceptions of personality, research suggests that neuroticism may be malleable over time or in response to treatment; however, such findings have been mixed, indicating that additional work is necessary to identify the specific conditions that impact temperament. Interventions that were explicitly developed to target neuroticism appear to have the most consistent effects on this trait. Rapee and colleagues' (Rapee et al., 2005, 2010) work with behaviorally inhibited children addresses the neuroticism by targeting the environmental factors (e.g., parenting styles) that contribute to the general psychological vulnerability (e.g., the belief that the world is a dangerous, uncontrollable place; Barlow et al., 2014) necessary for this trait's development. In adults, both the UP and neuroticism-focused MCBT address neuroticism by targeting the aversive, avoidant reactions to emotions that, paradoxically, increase the frequency and intensity of future negative emotions (Armstrong & Rimes, 2016; Carl et al., 2014). In sum, these efforts provide preliminary support for the notion that psychological interventions can indeed address temperamental vulnerabilities, and that such improvements are associated with a range of beneficial treatment outcomes (Carl et al., 2014; Farchione et al., 2012; Kennedy et al., 2009).

There are several advantages to shifting the focus of treatment to core temperamental vulnerabilities, rather than focusing disorder-specific symptoms. First, the rates of comorbidity among common mental disorders, including emotional disorders and personality disorders, are quite high (Grant et al., 2008; Zanarini et al., 1998). Rather than prioritizing treatment of one condition over another, an

intervention focused on a shared vulnerability (e.g., neuroticism) could lead to simultaneous improvement across symptoms of all co-occurring disorders. Additionally, separate treatments for each Diagnostic and Statistical Manual diagnosis places a burden on practitioners to receive training to competently provide these interventions; targeting common temperamental mechanisms may reduce the number of treatments, increasingly the likelihood that empirically supported treatments will be more widely disseminated.

Although there are there are theoretical advantages to addressing neuroticism directly in treatment, there are practical implications associated with moving away from the field's disorder-specific focus. For example, it is customary, and often necessary, to provide a *DSM* diagnosis (rather than levels of neuroticism) for psychotherapy to be reimbursed by insurance companies. This issue is addressable as targeting disorder symptoms and the core temperamental factors that maintain them are not mutually exclusive; the goal of addressing neuroticism in treatment *is* remission of *DSM* disorders. There may be benefit, however, for insurance companies to shift focus from presence or absence of a particular disorder to levels dimensional variables, like personality traits, as the latter may be more informative with regard to an individual's functioning (Widiger & Trull, 2007). Additional research is necessary to establish clinically significant thresholds for neuroticism that would point to whether treatment should be applied or if meaningful enough gains have been to warrant ending treatment.

Additionally, it is important to note that all three behavioral treatments developed to target neuroticism consist of multiple components. Given that they have each been tested only in their entirety, it is unclear whether each component contributes equally to the observed reductions in neuroticism. Future research should explore the unique contributions of each skill included in these treatment packages to identify the components that lead to the most robust and efficient changes in temperament. In addition, future research must also explore the possibility of intervention before a neurotic temperament fully develops. In fact, there is evidence to suggest that behavioral inhibition can be detected as early as 4 months old from crying and salivary cortisol in response to novel stimuli (Moehler et al., 2008). The animal literature provides insight into strategies that may be useful in preventing neuroticism; for example, exposing newborn rats to novel environments for 3 min a day resulted in less behavioral inhibition (defined as time spent in exploration after weaning) compared with newborn rats that stayed in their home cage (Tang, Reeb-Sutherland, Romeo, & McEwen, 2012). As such, it is important to identify early biological and behavioral markers that may indicate a vulnerability for the development of a neurotic temperament and that could serve as triggers for intervention.

In summary, treatments developed to directly target neuroticism indeed appear to impact this trait. However, to our knowledge, only three behavioral trials have been published with the aim of impacting temperamental factors, despite the advantages conferred by targeting shared vulnerabilities instead of disorder symptoms. Future research should not only continue to develop treatments for temperament but also explore which components included in these treatments for neuroticism are the most successful at reducing this trait, as well as how early interventions can occur.

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