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Brief mindfulness training de-couples the anxiogenic effects of distress intolerance on reactivity to and recovery from stress among deprived smokers



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ABSTRACT

Objective: We tested whether mindfulness de-couples the expected anxiogenic effects of distress intolerance on psychological and physiological reactivity to and recovery from an anxiogenic stressor among participants experimentally sensitized to experience distress.

Method: N = 104 daily smokers underwent 18-hours of biochemically-verified smoking deprivation. Participants were then randomized to a 7-min analogue mindfulness intervention (present moment attention and awareness training; PMAA) or a cope-as-usual control condition; and subsequently exposed to a 2.5-min paced over breathing (hyperventilation) stressor designed to elicit acute anxious arousal. Psychological and physiological indices of anxious arousal (Skin Conductance Levels; SCL) as well as emotion (dys)regulation (Respiratory Sinus Arrhythmia; RSA) were measured before, during and following the stressor.

Results: We found that PMAA reduced psycho-physiological dysregulation in response to an anxiogenic stressor, as well as moderated the anxiogenic effect of distress intolerance on psychological but not physiological responding to the stressor among smokers pre-disposed to experience distress via deprivation.

Conclusions: The present study findings have a number of theoretical and clinical implications for work on mindfulness mechanisms, distress tolerance, emotion regulation, and smoking cessation interventions.

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Distress tolerance is the capacity to withstand distress related to aversive affective, cognitive, and/or physical states (e.g., negative affect, physical discomfort; see Leyro, Zvolensky, & Bernstein, 2010 for a review). Distress intolerance facilitates avoidance, control or otherwise potentially maladaptive down-regulation of negative emotions and/or related aversive states; this may result in a paradoxical amplification of these aversive states, as well as reinforced learning that distress indeed cannot be tolerated (Simons & Gaher, 2005; Zvolensky, Vujanovic, Bernstein, & Leyro, 2010). In contrast, distress tolerance facilitates willingness to experience and approach negative emotions and related aversive states, and thereby the capacity to flexibly choose adaptive behavioral responding to such states, as well as enable learning that these

aversive states may be successfully tolerated. Hence, distress intolerance may affect a variety of key psycho-behavioral processes important to maladaptation, such as deployment of attention to potential emotional threat cues, appraisals of distress, and modulation of responses to distress (Leyro et al., 2010; Simons & Gaher, 2005). Accordingly, distress intolerance may contribute to the development or persistence of several forms of psychopathology (e.g., substance use, anxiety, mood, and personality disorders; Buckner, Keough, & Schmidt, 2007; Dennhardt & Murphy, 2011; Gorka, Ali, & Daughters, 2012; Gross & Muñoz, 1995; Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010; Leyro et al., 2010; Lynch & Bronner, 2006, pp. 217–236; Marshall-Berenz, Vujanovic, Bonn-Miller, Bernstein, & Zvolensky, 2010; Mennin, Heimberg, Turk, & Fresco, 2002; Norr et al., 2013; Zvolensky & Otto, 2007).

Accordingly, knowledge of specific interventions capable of promoting distress tolerance or buffering the psychopathogenic effects of distress intolerance has important clinical implications (e.g., Barlow, Allen, & Choate, 2004; Brown et al., 2013; Hayes,

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Strosahl, & Wilson, 1999; Linehan, 1993; Orsillo & Batten, 2005; Orsillo, Roemer, & Holowka, 2005, pp. 3-35). Indeed, the potential clinical importance of targeting distress intolerance is reflected by numerous psychotherapeutic approaches that directly or indirectly target distress intolerance and related processes (e.g., Hayes et al., 1999; Kohlenberg & Tsai, 1991; Linehan, 1993; Segal, Williams, & Teasdale, 2002). Yet, only a limited body of controlled experimental research – analogue laboratory, experimental psychopathology, or randomized controlled intervention research has directly tested specific intervention methods or specific intervention targets that may be promising candidates to buffer or decouple the effects of distress intolerance on psycho-bio-behavioral reactivity and recovery to distressing aversive internal states (e.g., anxious arousal; e.g., Berg, Snyder, & Hamilton, 2008; Brown et al., 2008). This is an important yet understudied area of research, with particularly important clinical implications for the delivery of brief specialized interventions (Zvolensky, Schmidt, Bernstein, & Keough, 2006).

In this vein, we focus on mindfulness, a promising intervention target and methodology that may be particularly well-suited to buffer the maladaptive or psychopathogenic effects of distress intolerance. Mindfulness has been conceptualized as present moment attention and awareness (PMAA), characterized by clarity of attention, flexibility of attention, and nondiscriminatory awareness (Anālayo, 2003; Bishop et al., 2004; Segal, Williams, & Teasdale, 2012; Thanissaro, 1996). Mindfulness also refers to practices or techniques, such as various meditative training exercises, grounded in the contemplative Buddhist tradition (e.g., Dahl, Lutz, & Davidson, 2015).

We theorize that mindfulness is well-suited to break the connection between distress intolerance and maladaptive reactivity or slowed recovery to an emotional stressor. First, an attitude of acceptance or willingness - that may also be conceptualized as a core component of equanimity (Desbordes et al., 2014; Hadash, Segev, Goldstein, Tanay, & Bernstein, 2016) – may allow a person to respond to a stressor in a less reactive manner regardless of her/ his "predisposition" to avoid or control unwanted experiences (i.e. distress intolerance). Indeed, mindfulness has been linked to: reduced experiential avoidance (Roemer & Orsillo, 2007), greater emotional acceptance (Hayes et al., 1999; Linehan, 1993; Segal et al., 2002), willingness to tolerate uncomfortable emotions and sensations (Eifert & Heffner, 2003; Erisman & Roemer, 2010; Levitt, Brown, Orsillo, & Barlow, 2004), and greater distress tolerance (Lotan, Tanay, & Bernstein, 2013). Second, meta-awareness induced by PMAA facilitates reduced identification with internal experiences (Bernstein et al., 2015). In response to acute subjective distress, such decentering or disidentification may lead to greater tolerance of distress and negative emotions (Kross & Ayduk, 2008; Kross, Ayduk, & Mischel, 2005), reduced self-referential thought (Farb et al., 2007; Ives-Deliperi, Solms, & Meintjes, 2011; Northoff & Bermpohl, 2004), as well as reduced reactivity to thought content (Bernstein et al., 2015; Hayes, Strosahl, & Wilson, 2012; Segal et al., 2012) which, in turn, may lead to reduced habitual processing (e.g., avoidance, catastrophizing, etc.) and greater behavioral choice in response to distress, as well as improved emotional and physiological (i.e., increased heart rate variability, reduced skin conductance) recovery following acute distress (Britton, Shahar, Szepsenwol, & Jacobs, 2012; Garland, Gaylord, Boettiger, & Howard, 2010; Goldin & Gross, 2010; Goleman & Schwartz, 1976; Vago & Silbersweig, 2012). We thus theorized that mindfulness may be particularly well-suited to de-couple the psychopathogenic effects broadly, and anxiogenic effects specifically, of distress intolerance.

We thus tested whether a laboratory analogue of a mindfulness intervention, a 7-min PMAA intervention, would moderate or buffer the effect of distress intolerance on reactivity to and recovery from an anxiogenic stressor (2.5-min paced over-breathing or hyperventilation) among deprived smokers. Smoking deprivation permits a strong experimental context for manipulating internal distress and sensitizing participants to experience distress in response to the anxiogenic stressor (Bernstein, Trafton, Ilgen, & Zvolensky, 2008; Farris, Zvolensky, Otto, & Leyro, 2015; Zvolensky & Bernstein, 2005); in turn, the anxiogenic stressor provides a strong experimental means to elicit acute and controlled anxious arousal (Asmundson, Norton, Wilson, & Sandler, 1994; Rapee, Brown, Antony, & Barlow, 1992; Zvolensky & Eifert, 2001). Moreover, smoking is a critical marker of psychiatric vulnerability, and thus, study of daily smokers permits over-sampling of persons atrisk for prevalent forms of psychopathology (Ziedonis et al., 2008).

First, we tested the effects of PMAA on reactivity and recovery. Specifically, we tested whether PMAA would lead to reduced psychological and physiological anxious arousal in response to and in recovery following an acute stressor - as reflected in subjective anxious arousal and skin conductance (e.g., Britton et al., 2012; Serpa, Taylor, & Tillisch, 2014). We also tested whether PMAA would also or alternatively enable greater physiological regulation during and then following the stressor – as reflected in a component of heart rate variability (respiratory sinus arrhythmia) under vagal control and indicative of parasympathetic nervous system activity (e.g., Campbell-Sills, Barlow, Brown, & Hofmann, 2006). We thus aimed to test whether PMAA reduces degree of emotional reactivity as reflected by anxious responding to the stressor or whether the effects of PMAA are limited to physiological regulation of that arousal. Second, we tested whether PMAA changes the anxiogenic effects of distress intolerance. Specifically, we predicted that whereas distress intolerance would lead to greater psychological and physiological reactivity and poorer recovery to the acute anxiogenic stressor, PMAA would buffer this maladaptive mechanism.

1. Method

1.1. Participants

One-hundred-four adult ($M(SD)_{age} = 26.02(5.36)$) daily smokers (>10 cigarettes per day) were recruited from the general city of Haifa community (Please see Table 1 for additional demographic information). Participants reported being a daily smoker for a M(SD) = 8.98(5.41) years and initiating smoking at M(SD) = 17.18(2.23) years of age. Potential participants met study inclusion criteria if they: (a) were between 18 and 65 years of age; (b) smoked regularly for at least one year; (c) currently smoke an average of at least 10 cigarettes per day; (d) exhaled carbon monoxide > 10 ppm; (e) have not reduced number of cigarettes smoked per day by more than half in the past six months; (f) reported Hebrew-language fluency; and (g) reported normal or fullycorrected (glasses) vision. Potential participants met study exclusion criteria in the event that they reported: (a) possible pregnancy; (b) seizure disorder; (c) current or past cardiopulmonary illness (e.g., heart disease) or other major chronic illness (e.g. cancer); (d) current acute respiratory illness (e.g., bronchitis); (e) current psychotic symptoms; (f) current nicotine replacement therapy or Zyban; (g) current use of other tobacco products more than once a week; (h) current homicidal or suicidal ideation; (i) current psychotropic medication also used in treatment of smoking cessation due to the possible confounding effects of medication on cessation and related study measures; (j) inability to give informed, written consent. Participants received financial compensation (\$50) for their participation. To participate in session 2, participants needed to demonstrate biochemically-verified abstinence from smoking

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