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Running Head: TRAIT ER AND CVR

EFFECTS OF TRAIT EMOTION REGULATION ON STRESS RESPONSIVITY

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A sizable body of literature has amassed on the topic of emotion regulation since the 1990's; with a focus on strategies that people implement to regulate their affective and physiological responses to stress. In particular, the impact of two emotion regulation strategies have received much attention; reappraisal and suppression (Gross, 1998c, 1999, 2002, 2013, 2014, 2015; Gross & Thompson, 2007; Schafer, Naumann, Holmes, Tuschen-Caffier, & Samson, 2017). Reappraisal involves reinterpreting a situation in order to alter the person's emotional response, for example looking at the positive aspects of a situation to reduce negative emotions. Whereas, suppression involves consciously inhibiting the behavioural expressions of the emotional response. Habitual use of these strategies is captured through self-report measures (Gross & John, 2003). In the laboratory, participants are instructed to employ either reappraisal or suppression in order to examine the effect of this manipulation on self-reported emotion and/or indices of sympathetic nervous system (SNS) activity.

The extended stress and coping theoretical model (Folkman, 1997) highlights the role of reappraisal in coping with stress. If stress-eliciting situations are reappraised as not "stressful" or non-threatening, it is expected that this will be reflected in the affective and physiological responses to the stressor. Reappraisal is hypothesised to buffer the negative effects of stress by changing the experience of the stressor and therefore resulting in lower negative affect (NA), greater positive affect (PA) and lower physiological arousal. In contrast, use of suppression does little to alleviate negative emotions associated with stress. Indeed, as suppression is typically initiated *after* the stress response has been elicited, it is hypothesised that the effort of inhibiting the expression of this response results in greater activation of the SNS.

Recent research has speculated that individual differences in emotion regulation may have implications for physical health; through both psychological mechanisms (the experience of

negative and positive emotions) and physiological mechanisms (via activation of stress-response systems). Prospective research provides some evidence linking emotion regulation with elevated cardiovascular disease risk. Individuals with poorer emotion regulation ability (as measured using items from the Minnesota Multiphasic Personality Inventory) were more likely to have died due to coronary heart disease (CHD) or have suffered a non-fatal myocardial infarction at a 12 year follow-up, after controlling for well-known CHD risk-factors (Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011). Furthermore, research directly assessing individual differences in suppression and reappraisal have reported a relationship between these measures and levels of C-reactive protein, which is a potential inflammatory marker of CHD (Danesh et al., 2004). Greater habitual use of reappraisal was associated with lower C-reactive protein levels, while suppression was associated with higher C-reactive protein levels (Appleton, Buka, Loucks, Gilman, & Kubzansky, 2013). The present chapter will first review research examining the relationship between emotion regulation, NA and PA and discuss how this may influence cardiovascular health. Next, we will focus on research investigating the effects of emotion regulation on SNS activity; as indexed by skin conductance and blood pressure responses. Finally, directions for future research in this area will be discussed.

Psychological outcomes of emotion regulation

A wealth of research has described the association between cardiovascular disease risk and the experience of heightened NA (for a review see; Suls & Bunde, 2005), and has highlighted the potential protective influence of positive emotions (for reviews see; Boehm & Kubzansky, 2012; Folkman, 2008; Pressman, Jenkins, & Moskowitz, 2019). Individual differences in the propensity to engage in reappraisal and suppression correlate with a range of

psychological outcomes, in particular PA, NA, depression and anxiety. Trait reappraisal has been associated with greater PA, greater satisfaction with social support, lower NA, less perceived stress, lower anxiety and lower depressive symptoms (e.g., Balzarotti, Chiarella, & Ciceri, 2017; Balzarotti, John, & Gross, 2010; Chervonsky & Hunt, 2018; Gross & John, 2003; Miklósi, Martos, Szabó, Kocsis-Bogár, & Forintos, 2014). In contrast, greater habitual use of suppression correlates with greater NA, greater depressive symptoms, lower PA and less satisfaction with social support (e.g., Appleton et al., 2013; Chervonsky & Hunt, 2018; Johnson et al., 2016; Nolen-Hoeksema & Aldao, 2011; Richardson, 2017; Rogier, Garofalo, & Velotti, 2017). A number of meta-analytic studies have also confirmed the association between reappraisal and psychological health, and the association between suppression and indices of psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Schafer et al., 2017). In cross-sectional studies, habitual use of reappraisal is associated with better indices of psychological well-being, while suppression has an inverse relationship with psychological health. Studies employing daily diary reports have found during times of stress trait reappraisers report less NA, and trait suppressors report less PA (Johnson et al., 2016; Richardson, 2017).

In experimental designs, instructed reappraisal to a range of passive stressors (e.g., film-clip stimuli, negative imagery) and active stressors (e.g., speech tasks, mental arithmetic) generally support the cross-sectional findings. Participants instructed to use reappraisal have reported less unpleasant emotions when viewing negative imagery (e.g., Dillon, Ritchey, Johnson, & LaBar, 2007) and lower distress, sadness, fear and disgust across a range of emotional film-clip stimuli compared to uninstructed conditions (Gross, 1998b; Gruber, Hay, & Gross, 2014; Richards & Gross, 2000; Shiota & Levenson, 2009; Troy, Shallcross, Brunner, Friedman, & Jones, 2018; Wolgast, Lundh, & Viborg, 2011). In contrast, instructed suppression

results in little or no change in reported affect compared to control conditions. For example, no differences have been found between suppression and control conditions in reported negative emotions in response to negative imagery and sad/distressing film-clips (Dillon et al., 2007; Dunn, Billotti, Murphy, & Dalgleish, 2009; Richards & Gross, 1999; Shiota & Levenson, 2009). Although Shiota and Levenson (2009) found instructed suppression led to reduced feelings of disgust after a disgust eliciting film-clip, others have reported no differences (Gross, 1998a; Roberts, Levenson, & Gross, 2008). Taken together, cross-sectional and experimental research support the hypothesis that reappraisal may buffer the negative affective impact of stress. However, contrary to cross-sectional findings which demonstrate greater habitual use of suppression is associated with higher levels of NA, experimental manipulations of this strategy have little or no influence on affective responding during acute stress. This may be a result of self-report bias; individuals were instructed to “feel nothing” or “hide their emotional responses” which may have influenced self-reported affect. Assessment of individual differences in strategy use overcomes this potential bias and will help elucidate how the tendency to suppress expressive behaviour during stressful situations influences positive and negative emotions.

How might psychological outcomes influence physical health?

In terms of physical health, an abundance of research has linked negative affective states with the development of cardiovascular disease (Anda et al., 1993; Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2005; Hemingway & Marmot, 1999). One mechanism believed to link emotional and stress responses with CHD is exaggerated cardiovascular responses to stressors (Kibler & Ma, 2004). The well-established cardiovascular reactivity (CVR) hypothesis

implicates exaggerated or prolonged CVR to stressors as a risk-factor for CHD; a finding supported by numerous longitudinal and cross-sectional studies (for a review see; Chida & Steptoe, 2010). In these studies, those that had heightened reactions to acute psychological stress in a laboratory setting had, at a 10-15 year follow-up, higher resting blood pressure (Carroll et al., 2001; Light, Dolan, Davis, & Sherwood, 1992), and a greater incidence of hypertension (Carroll et al., 2001; Tuomisto, Majahalme, Kahonen, Fredrikson, & Turjanmaa, 2005) and stroke (Everson et al., 2001). It is argued that exaggerated reactivity to stressors over time causes structural and functional changes in the heart, which promote the development of CHD (Treiber et al., 2003; Treiber, Turner, Davis, & Strong, 1997). Individual differences in emotion regulation may help elucidate the pathway underlying the relationship between stress responsivity and CHD.

Physiological outcomes of emotion regulation

Laboratory studies which assess physiological arousal mirror results from research examining the affective consequences of emotion regulation; in general, instructed reappraisal leads to healthful patterns of cardiovascular responding, while suppression is associated with exaggerated CVR during acute stress. Instructed reappraisal has been found to result in lower skin conductance responses to a range of emotion-eliciting film-clips (Gruber et al., 2014; Wolgast et al., 2011). Conversely, instructed use of suppression leads to heightened skin conductance and blood pressure during film-clip stimuli (e.g., Gross, 1998a; Harris, 2001; Roberts et al., 2008), as well as greater heart rate reactivity during a speech task (Hofmann, Heering, Sawyer, & Asnaani, 2009) and exaggerated blood pressure reactivity during mental arithmetic and cold pressor tasks (Quartana & Burns, 2010). This highlights a potential pathway

by which suppression can contribute to CHD risk; repeated activation of the sympathetic system over time can result in arterial damage due sheer stress, heightening the risk of hypertension and atherosclerosis (e.g., Clarkson, Manuck, & Kaplan, 1986). It is worth noting that some studies have reported no relationship between trait emotion regulation and skin conductance responding during passive stress (Dunn et al., 2009; Gross, 1998b; Kim & Hamann, 2012); however, this may be due to differences in physiological measurements, as well as task differences (in terms of the emotional nature of the stressor and the type of coping context elicited by the task) (e.g., Bolli, Amann, Hulthen, Kiowski, & Buhler, 1981; Gross & Levenson, 1993; Obrist, 1981; Obrist, Light, McCubbin, Hutcheson, & Hoffer, 1979; Saab et al., 1993; Sherwood, Allen, Obrist, & Langer, 1986). Overall, reported findings have generally corroborated the argument that reappraisal can be considered an adaptive strategy; it is associated with lower physiological arousal which may offer a protective function in terms of physical health. While use of instructed suppression demonstrates the opposite pattern.

Examining individual differences in the natural tendency to engage in these strategies can offer more insight into the longer-term psychophysiological implications of reappraisal and suppression use. However, few studies have taken the individual difference approach and only a handful include CVR to stressors as an outcome. Research that has assessed both trait emotion regulation and CVR has focussed on reappraisal and provide evidence that reappraisal moderates cardiovascular responding. For example, in response to a speech task higher trait reappraisal scores were associated with lower blood pressure reactivity; however, trait suppression had no influence on blood pressure (Memedovic, Grisham, Denson, & Moulds, 2010).

One potential avenue to explore in elucidating how emotional regulation may influence disease risk is the examination of two cardiovascular parameters that underly the blood pressure

response; total peripheral resistance (TPR) and cardiac output (CO). Cardiac output and TPR are compensatory parameters, as one increases the other should decrease (Turner, 1994). An increase in CO paired with a decrease in TPR is indicative of a myocardial response, which is classified as being approach-oriented or indicative of a challenge response to stress. An increase in TPR paired with a decrease in CO indicates a threat response, which is vascular in nature (Mendes, Reis, Seery, & Blascovich, 2003; Tomaka, Blascovich, Kibler, & Ernst, 1997). Individuals scoring high in reappraisal demonstrated this approach-orientated cardiovascular profile in response to a mental arithmetic stressor; indexed by increased CO and decreased TPR (Mauss, Cook, Cheng, & Gross, 2007). Further evidence for this stems from research experimentally manipulating the use of reappraisal; participants instructed to reappraise arousal as adaptive when completing the Trier Social Stress Task (a mental arithmetic task and speech task) demonstrated increased CO and decreased TPR, compared to individuals instructed to ignore feelings of stress, or given no instructions (Jamieson, Nock, & Mendes, 2012a, 2013). To our knowledge, no research to-date has examined the relationship between suppression and these parameters in response to active stress.

Directions for future research

Evidence from cross-sectional and experimental research advocate reappraisal as a process which buffers the negative, emotional and physiological, consequences of stress. In response to acute stress instructed use of reappraisal, as well as a greater tendency to engage in this strategy habitually, is associated with more positive emotion, less negative emotion and a challenge-orientated cardiovascular response. On the other hand, suppression does little to mitigate negative emotional responses during stress, and generally results in exaggerated SNS

activity (indexed by blood pressure or skin conductance responding). While instructed use of emotion regulation strategies typically demonstrates these distinct patterns of SNS activity, drawing on the current research it is difficult to claim that the propensity to use these strategies shows the same pattern of cardiovascular responding. Future research needs to address this gap, in particular as individual differences would reflect how individuals naturally respond to, and cope with, daily stressors; examining the psychophysiological consequences of *trait* emotion regulation would therefore offer insights into the long-term implications of reappraisal and suppression use. Furthermore, research in the area can be strengthened by addressing methodological limitations with past research (e.g., inclusion of official acclimatization and baseline periods, employment of active stress tasks) and by using more sophisticated measures of the physiological response.

Knowledge and expertise from stress reactivity research can make a substantial contribution to the emotion regulation literature. Past research examining the physiological outcomes of emotion regulation is limited by a number of methodological shortcomings. Although many of these experiments are well-controlled in terms of counterbalancing the order of stimuli, they lack key stages inherent in CVR research. For example, within the stress reactivity literature it is common to include a pre-baseline acclimatization period (e.g., Hogan et al., 2012; Howard, Hughes, & James, 2011; van Stegeren, Wolf, & Kindt, 2008) to allow participants to become adjusted to the laboratory environment and ensure participants are fully rested prior to recording physiological parameters. Emotion regulation studies rarely include such a period. Furthermore, baseline periods tend to be short, usually one minute (e.g., Gross, 1998b; Gruber et al., 2014; Richards & Gross, 1999; Shiota & Levenson, 2009), three minutes (Dunn et al., 2009) or five minutes (e.g., Jamieson et al., 2012a, 2013) with instructions to

simply “sit quietly”, “clear your mind” or “relax”. This conflicts with the recommended 10-minute Vanilla resting baseline (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992), where the inclusion of a minimally taxing task is recommended in order to avoid arousal from task anticipation, boredom or rumination. Considering that physiological baseline measurements often act as a comparison against reactivity elicited during a task, the implementation of a clear, methodological-sound baseline period is essential.

Furthermore, previous research tends to measure physiological responding in terms of skin conductance responsivity or heart rate, rather than more sophisticated indices of CVR, such as blood pressure, CO and TPR. This is a clear limitation of the emotion regulation literature considering the utility of blood pressure reactivity in predicting the development of CHD (Carroll et al., 2001; Light et al., 1992). In particular, a focus on CO and TPR will help extend research in this area. Reappraisal has already been linked to a more approach-oriented response to stress in terms of CO and TPR; but the relationship between these parameters and trait suppression needs to be examined. We would advocate the assessment of CO and TPR in future research, but also the application of the Hemodynamic Profile-Compensation Deficit (HP-CD) Model (Gregg, Matyas, & James, 2002). This computational model produces HP and CD values, calculated from CO and TPR responses. CD refers to blood pressure changes, while HP values classify the underlying hemodynamic response as myocardial (CO-dominant), vascular (TPR-dominant) or mixed (synergistic changes in CO and TPR). This more sophisticated index of hemodynamic responding offers a number of advantages over previous classifications of response type (for a review see; James, Gregg, Matyas, Hughes, & Howard, 2012).

The relationship between emotion regulation and stress reactivity is also limited by the focus of past research on stimuli that are considered “passive” tasks such as film-clips and

negative imagery; during which participants simply endure the experience and have little control over the task (e.g., Obrist, 1981; Obrist et al., 1978). Conversely, active tasks, such as speech tasks, require participants to be actively engaged during the task and participants exert some control over their performance. Within the stress reactivity literature, active tasks are argued to be more representative of everyday stressors, better predictors of future blood pressure, and are associated with a pattern of physiological responding distinct from passive tasks (e.g., Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998). In particular the use of speech tasks have been shown to successfully elicit cardiovascular responses in the laboratory, and this magnitude of response has been shown to be generalizable from laboratory to field (Johnston, Tuomisto, & Patching, 2008). Within the emotion regulation literature limitations of passive tasks have also been highlighted. Passive tasks have been argued not to elicit the same internal motivational and socio-evaluative states characteristic of active tasks (Aldao, 2013), while active tasks are posited to prompt stronger emotional response tendencies which result in changes in affect and physiological arousal (Egloff, Schmukle, Burns, & Schwerdtfeger, 2006). It may be the case that active stressors may be more reflective of coping responses elicited by daily stressors, compared to simply watching film-clips or negative imagery.

Overall, evidence from laboratory and cross-sectional research strongly suggest that reappraisal can promote adaptive stress responding, in terms of affect and physiological arousal. Conversely, use of suppression appears to exacerbate the stress response. While, in the short-term this may not negatively impact health, over time repeated activation of the SNS (for high suppressors) may confer a risk for CHD. Although the reviewed studies present evidence for a relationship between trait emotion regulation and stress responsivity, it must be acknowledged that emotion regulation research examining CVR is in its early stages. Despite the potential

importance of trait emotion regulation in elucidating the effects of stress on physical health few studies have adopted a stress reactivity research perspective. Combining strengths from these overlapping, yet separate, literatures can extend our understanding of this relationship. Future research can address the methodological issues outlined in this review; namely the inclusion of an official acclimatization period and a Vanilla resting baseline, as well as the use of active tasks and more sophisticated measures of cardiovascular responding. In doing so, future studies can help elucidate how individual differences in suppression and reappraisal influence responses to stress.

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