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Chronic Smoking, Trait Anxiety, and the Physiological Response to Stress

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Abstract

Background and Objectives—Both chronic smoking and trait anxiety have been associated with dysregulations in psychobiological stress response systems. However, these factors have not been studied in conjunction. We expected trait anxiety and smoking status to attenuate stress reactivity. Furthermore, we expected an allostatic load effect resulting in particularly attenuated stress reactivity in high-anxious smokers. In addition, high-anxious smokers were expected to exhibit increased urges to smoke in response to stress.

Methods—115 smokers and 37 nonsmokers, aged 18 – 64 years, completed a laboratory session including mental stressors such as evaluated public speaking and mental arithmetic. Trait anxiety was assessed using Spielberger's State-Trait Anxiety Inventory. Cardiovascular autonomic indices, salivary cortisol, and the desire to smoke were measured at baseline, during stressors, and at recovery.

Results—Regression analyses showed that smokers exhibited attenuated cardiovascular stress responses in comparison to nonsmokers. Higher trait anxiety predicted attenuated systolic blood pressure responses to stress. No interaction effect of smoking status and trait anxiety was found in stress response measures. Higher trait anxiety predicted an increased desire to smoke in response to stress among smokers.

Conclusion—Results indicate that both smoking status and trait anxiety are associated with blunted sympatho-adrenal cardiovascular stress reactivity. Elevated urges to smoke in response to

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Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

stress found among smokers with high trait anxiety suggest an important role of anxiety in smoking propensity and relapse.

Keywords

Chronic smoking; trait anxiety; mental stress; psychophysiology; cardiovascular system; sympathetic nervous system

BACKGROUND

Tobacco use causes severe health problems worldwide annually. Epidemiological studies estimated about 1.1 billion daily smokers worldwide with 81 percent male smokers (Jha, Ranson, Nguyen, & Yach, 2002; World Health Organization, 2011) and an annual death rate of approximately 5 to 6 million people globally (Mathers & Loncar, 2006; World Health Organization, 2014). Smoking plays a role in regulating negative mood and emotions (Gilbert, 1995; Leung, Gartner, Dobson, Lucke, & Hall, 2011) supporting a high prevalence of nicotine dependence among individuals with emotional dysfunctions (Capron et al., 2012; Langdon et al., 2013; Luk & Tsoh, 2010; Sonntag, Wittchen, Hofler, Kessler, & Stein, 2000). Specifically, higher rates of smoking have been found among individuals diagnosed with anxiety disorders (Goodwin et al., 2012; Grover, Goodwin, & Zvolensky, 2012; Ziedonis et al., 2008).

Smoking behavior and anxiety disorders

Individuals diagnosed with an anxiety disorder are twice as likely to smoke as the general population and smoking is a risk factor for developing an anxiety disorder (Breslau, Davis, & Schultz, 2003; Breslau & Klein, 1999). Sonntag and colleagues (2000) demonstrated in a prospective longitudinal study that social fear and social phobias increase the risk for nicotine dependence in adolescents and young adults. Similarly, individuals diagnosed with posttraumatic stress disorder (PTSD) show a greater prevalence of cigarette smoking than the general population (reviewed by Rasmusson, Picciotto, & Krishnan-Sarin, 2006). Furthermore, a population-based study revealed that panic disorder is associated with occasional and regular smoking and an increased risk for new onsets of panic attacks among prior nicotine dependent individuals (Isensee, Wittchen, Stein, Hofler, & Lieb, 2003). Individuals who quit smoking exhibit greater depressed mood, increased anxiety, and impaired brain functions, which in turn, increases the likelihood of smoking relapse (Hughes, Gust, Skoog, Keenan, & Fenwick, 1991). One study investigating anxiety in smoking relapse revealed that female smokers with current or lifetime diagnoses of anxiety disorders were less likely to be abstinent than female smokers never diagnosed with an anxiety disorder (Piper et al., 2010). In addition, Zvolensky and colleagues (2008) demonstrated that patients diagnosed with an anxiety disorder were at increased risk of relapse during the first week post-smoking cessation compared to control participants. These different observations support strong linkages between clinical anxiety and smoking behavior. However, these results only apply to a minority of smokers. To draw conclusions of the relationship between anxiety and smoking for the general population, it is necessary to investigate individual differences in nonclinical anxiety among smoking populations.

Smoking behavior and trait anxiety in the general population

Only few studies have examined the relationship of anxiety as a fundamental personality trait in the general population and smoking behavior. Trait anxiety is defined as experiencing negative feelings such as worry, discomfort, tension, and stress across specific situations on a regular basis (Spielberger, Gorusch, & Lushene, 1983). Several studies have shown higher trait anxiety levels among chronic smokers compared to nonsmokers (Houston & Schneider, 1973; Pietras, Witusik, Panek, Szemraj, & Gorski, 2011; Sheahan et al., 2006) while other studies did not find any differences (Canals, Domenech, & Blade, 1996; Farley & Lester, 1995; Henry, Jamner, & Whalen, 2012). Inconsistencies of these findings may be due to different sample populations (e.g., college students vs. non-academic samples), variation in sample sizes, and different measurements of anxiety. Some studies suggest that smoking reduces state anxiety (Pomerleau & Pomerleau, 1987; Pomerleau, Turk, & Fertig, 1984) while other research indicates smoking cessation as being an attenuating factor for state anxiety (West & Hajek, 1997). However, specifically in stressful situations anxiety-based coping strategies may contribute to a greater likelihood of nicotine intake to manage stress (Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007). These different observations produce an inconsistent picture of the relationship between trait anxiety and smoking behavior and suggest that stress may play another important role in the linkage.

Psychobiological stress responses and anxiety

The stress response involves multiple levels of physiological activation. Upon perception of stress, the corticotropin-releasing hormone (CRH) is produced in the hypothalamus. CRH stimulates the release of the adrenocorticotropic hormone (ACTH) in the anterior pituitary, which in turn initiates the production of cortisol in the adrenal cortex (i.e., the hypothalamic-pituitary-adrenocortical (HPA) axis; e.g., al'Absi & Arnett, 2000). The sympathetic nervous system (SNS), which is responsible for catecholamine release in the adrenal medulla, is also activated. This leads to an increased heart rate (HR), blood pressure (BP), and alterations in other physiological systems of the body (Hoehn-Saric & McLeod, 2000). Research examining anxiety and psychobiological stress responses has shown inconsistent results. For instance, one study found a hyperactive HPA axis with increased cortisol levels in response to stress in people suffering from social anxiety (Condren, O'Neill, Ryan, Barrett, & Thakore, 2002) compared to healthy controls while in PTSD basal cortisol levels are often found to be lower than in healthy controls (Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007). Research on trait anxiety has found a hypoactive stress response with reduced cardiovascular and cortisol responses in individuals with high trait anxiety (de Rooij, Schene, Phillips, & Roseboom, 2010; Jezova, Makatsori, Duncko, Moncek, & Jakubek, 2004). These blunted physiological stress responses may result from malfunctioning of biological stress systems. In the short-term, anxiety may heighten psychophysiological stress reactivity (e.g., increased cortisol) as shown by Boudarene and colleagues (2002). However, chronic anxiety may induce a prolonged physiological activation that may lead to systemic allostatic load, resulting in maladaptive physiological stress response patterns (McEwen, 1998, 2007).

Psychobiological stress response and smoking behavior

Interestingly, a growing body of evidence also indicates attenuated cardiovascular (Roy, Steptoe, & Kirschbaum, 1994; Straneva, Hinderliter, Wells, Lenahan, & Girdler, 2000) and neuroendocrine (al'Absi, Amunrud, & Wittmers, 2002; al'Absi, Wittmers, Erickson, Hatsukami, & Crouse, 2003; Kirschbaum, Strasburger, & Langkrar, 1993; Roy et al., 1994) stress responses in habitual smokers. The pharmacological effects of nicotine and toxic effects of other smoke constituents may have additive negative impacts in biological adaptation mechanisms, which may lead to prominent allostatic changes resulting in attenuated physiological responses to stress. Allostatic load (McEwen, 2007) may be particularly elevated in smokers with high trait anxiety, resulting in an additive effect on blunting of the psychobiological stress response. However, research investigating tobacco use, anxiety, and stress adaptation in conjunction is lacking and to the best of our knowledge, no study has directly tested this hypothesis.

OBJECTIVES

The aim of the present study was to examine associations of trait anxiety and habitual smoking with psychobiological stress responses. Based on studies reviewed above, we hypothesized that trait anxiety is associated with deterioration in stress responding, and that the effect of trait anxiety is most prominent in smokers due to additional physiological effects of smoking on stress responsivity. In addition, we expected that trait anxiety among smokers predicts an increased urge to smoke in response to stress due to partial evidence of anxiolytic effects of smoking (reviewed by Morissette et al., 2007).

METHODS

Participants

A sample of 152 participants (71women) comprising 115 smokers and 37 nonsmokers with an average age of 33.47 years (SD = 12.00) was recruited as part of a smoking relapse study (al'Absi, Nakajima, Allen, Lemieux, & Hatsukami, 2015). We note that previous studies from our laboratory have focused on stress, pain, and stress-induced analgesia in habitual smokers (al'Absi, Nakajima, & Grabowski, 2013; Nakajima and al'Absi, 2014). The majority of participants were Caucasian (62.2 % of non-smokers; 83.5% of smokers) and single (67.6 % of non-smokers; 58.3 % of smokers). Participants underwent an on-site screening session including the assessment of smoking status, alcohol, drug use, and medical history. To be eligible for the study, participants had to meet the following criteria: (1) no history of a chronic illness; (2) no current use of prescriptive medication except contraceptives; (3) no current alcohol or drug dependence, as well as no current psychiatric disorder and (4) weighted within ± 30% of Metropolitan Life Insurance norms (i.e. body mass index between 18 and 30). Smokers must have smoked at least an average of ≥10 cigarettes per day for a minimum of two years with a strong desire to quit smoking: Smokers were asked "What is your desire to quit smoking at this time?" on a 7-point rating scale of 1 (not at all) to 7 (extremely strong). A strong desire for quitting smoking required to report the score of 4 or higher to this question; M = 5.77, SD = 1.06). Smokers smoked their first cigarette at the age of M = 16.16 (SD = 3.45) years, with an average duration of M = 11.28

(*SD* = 9.52) years, a daily cigarette consumption of *M* = 18.31 (*SD* = 6.55), and a baseline carbon monoxide level of *M* = 24.01 (*SD* = 13.22) ppm. Eligible subjects read and signed a consent form that was approved by the Institutional Review Board of the University of Minnesota. After obtaining the consent, participants were asked to complete a battery of questionnaires (described below) and provided a breath sample for assessment of expired carbon monoxide (CO) and the time of their last cigarette.

Self-report measures

Demographic information and smoking history—Prior to the laboratory session, participants completed a battery of questionnaires (e.g., anxiety questionnaires, stress level assessment) and were asked for demographic information. Smokers completed additional forms on smoking history (e.g., number of cigarettes per average day).

Trait anxiety—The State-Trait Anxiety Inventory (Spielberger, Gorusch, & Lushene, 1983) including 20 statements on the trait assessment and on a 4-point rating scale from 1 (almost never) to 4 (almost always) was used to assess trait anxiety. Items such as “I worry too much over something that really doesn’t matter” were utilized (Cronbach’s alpha ranging from 0.87 – 0.93) with higher scores indicating higher anxiety levels.

Stress—Psychosocial stress levels were assessed as a trait using the Perceived Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983) including ten items on a 4 point rating scale from 1 (almost never) to 4 (almost always) such as “How often have you felt nervous and “stressed”?” (Cronbach’s alpha between 0.84 – 0.86). Higher scores indicate higher stress levels.

Urge to smoke—The brief version of the Questionnaire for Smoking Urges (QSU-B) (Cox, Tiffany, & Christen, 2001; Tiffany & Drobes, 1991) was used to assess two components of craving (factor 1: positive desire to smoke for reward including 5 items such as “I have desire for a cigarette right now”; factor 2: need to smoke for relief including 5 items such as “Nothing would be better than smoking a cigarette”; Cronbach’s alpha between 0.92 – 0.97). Higher scores indicate greater smoking urges.

Stressors

In this study we combined different stress tasks: public speaking and mental arithmetic that have been shown to modulate the physiological stress response in habitual smokers (al’Absi, Hugdahl, & Lovallo, 2002; al’Absi, Nakajima, & Grabowski, 2013).

Public speaking—Anxiety provoking stressors such as the public-speaking task has been shown to induce cardiovascular, endocrine, and emotional state changes (e.g., al’Absi et al., 1997; Dickerson & Kemeny, 2004). Participants were given one of two scenarios: In scenario one, participants were asked to imagine a situation where they were being accused of shoplifting whereas in scenario two, participants were asked to imagine a situation where they were being accused in a hit and run accident. For either scenario, participants were asked to develop a defense speech (preparation phase, 4 min) and then to give the speech in front of the experimenter audience (speech phase, 4 min). Participants were instructed that

the speech would be videotaped and evaluated by staff members at a later time. They were instructed that evaluators were going to rate their performance on persuasion, organization, articulation, and devotion. Cardiovascular measurements were taken at one minute and three minutes during preparation and delivery phases. Afterwards, participants were asked to complete a cognitive task.

Mental arithmetic—This task was chosen to elicit mental stress and it has been shown to reliably activate the HPA axis (al'Absi et al., 1997; Dickerson & Kemeny, 2004). The task required continuous addition in a ten-minute block starting with a three-digit number. Participants were instructed to add each digit of the three-digit number and adding the sum of these three digits to the three-digit number itself. This should have been continued until the experimenter told the subject that the given result was wrong. In this case, the experimenter repeated the last three-digit number and participants were instructed to start over again with the given number. They were asked to perform as quickly and accurately as possible. Cardiovascular measurements were taken at the beginning (initial measurement) and then after every two minutes during the task.

Adrenocortical and cardiovascular measures

Cortisol concentrations were assessed by saliva samples. Participants placed cotton dental rolls in their mouth until saturated and collected the rolls into a plastic tube (Salivette® tubes, Sarstedt, Rommelsdorf, Germany). Salivary cortisol samples were assayed by a timeresolved fluorescence immunoassay with a cortisol–biotin conjugate as a tracer, 43 with a sensitivity of 0.4 nmol/L and inter- and intra-assay coefficients of variation less than 10% and 12%. Systolic bloodpressure (SBP), diastolic bloodpressure (DBP), and heartrate (HR) were measured using the Dinamap oscillometric monitor system (Critikon, Tampa, FL).

Procedure

Smokers and nonsmokers were instructed to refrain from caffeine, alcoholic beverages, and physical exercise prior to the laboratory session, which started between 12 and 2 PM to control for diurnal pattern of hormonal levels. Each participant was seated in a comfortable chair and was instructed to sit upright with legs uncrossed and feet on the floor. Starting with a 45-minute baseline period, they watched documentary films (selected for emotionally neutral content) to be instrumented for cardiovascular measurements such as HR and blood pressure. Five cardiovascular measurements (including SBP, DBP, HR) were obtained during the last 20 min of this baseline. Afterward, individual stress procedures (public speaking, mental arithmetic, cold pressor) always followed a resting/recovery period of at least 20 minutes. During the public speaking and the mental arithmetic task, cardiovascular measures were collected every 2 min. After the stress procedures a 20 min recovery period and cardiovascular measurements were obtained every 5 min. Saliva samples and self-report data (QSU-B) were collected after baseline, stressors, and recovery periods. At the end of the laboratory session participants were debriefed and received monetary compensation for participation.

Data analysis

This paper presents secondary analyses obtained from a larger study conducted to examine psychobiological predictors of smoking relapse (al'Absi et al., 2015). A series of independent pooled variance estimate t-test statistics accounting for differences in sample sizes between smokers and nonsmokers was conducted to examine group differences of demographic variables, trait measures, and hormonal (salivary cortisol levels) baseline measures. We used regression analyses to determine the associations of trait anxiety and smoking group with primary outcome variables: Stress indices (difference between stress, baseline, and recovery) of hormonal and cardiovascular measures such as SBP, DBP, and HR. In a secondary analysis, the Perceived Stress Scale was included as a trait-level predictor, to explore to what degree effects were based on general stress levels of participants. Additionally, associations of the urge to smoke (stress index of the QSU-B), trait anxiety, and perceived stress were analyzed. Cardiovascular measurements were averaged for baseline, stress (speech and math), and recovery. Cortisol data were log-transformed to meet assumption for normality.

RESULTS

Participant characteristics

The smoking and nonsmoking group did not differ in age, body mass index, anxiety, and baseline salivary cortisol ($ps > 0.076$). However, smokers indicated higher daily caffeine consumption, higher levels of perceived stress, and less years of education ($ts > 2.96$, $ps < 0.004$; see Table 1).

Trait anxiety, smoking group, and perceived stress in association with stress indices

Results from regression analyses showed that trait anxiety was significantly associated with the SBP stress index in Model 1, $\beta = -0.38$, $p = 0.012$ indicating that higher trait anxiety predicted blunted SBP responses to stress (see Table 2). Similarly, the smoking group also predicted the SBP stress index, $\beta = -0.16$, $p = 0.043$ (see Table 2) demonstrating that smokers exhibited a blunted SBP response to stress. Unexpectedly, the interaction between smoking group and trait anxiety did not reach significance, $\beta = 0.24$, $p = 0.112$ [1]. Model 2 included perceived stress as an additional predictor and showed no significant influence on the SBP stress response, $\beta = 0.09$, $p = 0.744$ (see Table 2). DBP and HR stress indices were only predicted by smoking group, $\beta s < -0.22$, $ps < 0.008$ showing that smokers also exhibited attenuated DBP and HR responses to stress whereas trait anxiety and perceived stress did not have any influences on DBP and HR stress indices, $\beta s > -0.17$, $ps > 0.267$.

[1] Due to large differences in sample sizes between smokers and nonsmokers, which may bias statistical analyses (Zahn, 2009), three subsamples were randomly generated. Subsample 1(39 smokers, 37 nonsmokers) showed the same pattern: higher trait anxiety predicted blunted SBP responses to stress, $\beta = -0.36$, $p = 0.017$ and smokers exhibited blunted SBP response to stress, $\beta = -0.25$, $p = 0.023$; the interaction effect was not significant, $\beta = 0.13$, $p = 0.376$ ($R^2 = 0.16$). Subsample 2 (38 smokers, 37 nonsmokers) showed a similar pattern: higher trait anxiety predicted blunted SBP responses to stress, $\beta = -0.39$, $p = 0.014$, whereas the effect for smokers exhibiting blunted SBP response to stress was only marginally significant, $\beta = -0.21$, $p = 0.059$. Again, the interaction effect was not significant, $\beta = 0.26$, $p = 0.091$ ($R^2 = 0.13$). In subsample 3 (38 smokers, 37 nonsmokers) higher trait anxiety predicted attenuated SBP responses to stress, $\beta = -0.42$, $p = 0.010$; however, no effect was found for the smoking group factor, $\beta = -0.11$, $p = 0.341$; the interaction effect was also not significant, $\beta = 0.19$, $p = 0.233$ ($R^2 = 0.12$).

Furthermore, no associations were found for the cortisol stress index including trait anxiety, smoking group, and perceived stress as predictors in the model, $\beta s > -0.10$, $ps > 0.573$.

Trait anxiety and perceived stress in association with the urge to smoke for relief

Trait anxiety was significantly associated with the urge to smoke for relief (factor II, QSU-B) in response to stress, $\beta = 0.29$, $p = 0.002$ (see Table 3). Model 2 including perceived stress did not reach significance, $\beta = 0.08$, $p = 0.497$ and trait anxiety only showed a marginal trend, $\beta = 0.24$, $p = 0.055$ (see Table 3). However, including caffeine consumption in Model 3 showed significant effects for trait anxiety, $\beta = 0.25$, $p = 0.042$ and caffeine consumption, $\beta = 0.19$, $p = 0.040$ as predictors for the urge to smoke for relief indicating higher levels of trait anxiety and higher caffeine intake predicting greater stress-related urges to smoke for relief (see Table 3). This was not the case for perceived stress in Model 3, $\beta = 0.06$, $p = 0.607$. No effects of trait anxiety and perceived stress were found for the urge to smoke for reward (factor I, QSU-B), $\beta s < 0.15$, $ps > 0.111$.

CONCLUSIONS

This study investigated the effects of trait anxiety and chronic smoking on the physiological response to stress. Our expectation of blunted cardiovascular responses to stress among smokers was confirmed, i.e., smokers independent of their trait anxiety status exhibited blunted cardiovascular stress responses relative to nonsmokers. Furthermore, higher trait anxiety irrespective of smoking status was also associated with blunted systolic blood pressure responses to stress. However, both such effects were not found for the salivary cortisol response to stress. Contrary to our expectations and the allostatic load model, we did not find an interaction effect of trait anxiety and smoking status indicating that habitual smoking did not further accentuate the attenuating effect of trait anxiety on systolic blood pressure responses to stress. As expected, trait anxiety was associated with increased stress-related urges to smoke for relief among smokers. Overall, these findings indicate that high trait anxiety and habitual smoking were independently linked to deteriorated cardiovascular stress responses. The current study is among the first to demonstrate the role of trait anxiety and habitual smoking in response to stress and expands our prior work and work of others (al'Absi et al., 2013; al'Absi et al., 2003; Kirschbaum et al., 1993; Roy et al., 1994; Straneva et al., 2000).

Smokers, regardless of trait anxiety, showed attenuated cardiovascular stress responses, which was also shown in our recent work (e.g., al'Absi et al., 2013). Smoking has been associated with the HPA axis and autonomic nervous system (ANS) activity in response to stress. Studies examining acute effects of smoking reported an additive effect of nicotine and stress in psychobiological mechanisms (Parrott & Murphy, 2012) due to common modulating patterns in the central nervous system (CNS) where stress and nicotine play important roles in activating the cardiovascular system. In contrast, chronic smoking may lead to a prolonged ANS activation resulting in decreased sensitivity to stress as well as other potential additive effects that are not associated with nicotine (Kirschbaum, Scherer, & Strasburger, 1994) such as trait anxiety. This may explain why the expected sympatho-adrenal response was not observed among smokers compared to nonsmokers.

The finding of an association between high levels of trait anxiety with decreased cardiovascular stress reactivity is in line with Jezova and colleagues (2004) showing attenuated epinephrine and norepinephrine secretion to stress in high-anxious compared to low-anxious individuals. Results suggest that high trait anxiety is associated with an inadequate regulation of the sympatho-adrenal system during acute stress. Other studies found similar patterns of attenuated physiological responses to stress in patients with different anxiety disorders (e.g., Hoehn-Saric & McLeod, 2000; McTeague et al., 2010; Petrowski, Wintermann, Schaarschmidt, Bornstein, & Kirschbaum, 2013). It should be noted that, in the present study, DBP and HR responses to stress were not associated with trait anxiety levels. In contrast to systolic blood pressure, which is a rather pure sympathetic autonomic measure regulated almost exclusively by the sympatho-adrenal axis via beta-adrenergic receptors (e.g., Obrist, 1981; Silvestini and Gendolla, 2011), HR is dually innervated by cardiac sympathetic as well as vagal efferent pathways (Berntson et al., 1997). This may explain why it diverged from the systolic blood pressure pattern. Also, HPA axis activity did not show differential responses to stress between smoking groups and we did not find any associations between cortisol stress responses and trait anxiety. Previous research did not reveal evidence of relationships between personality traits and the cortisol stress response either (Bohnen, Nicolson, Sulon, & Jolles, 1991; Schommer, Kudielka, Hellhammer, & Kirschbaum, 1999). However, one study found associations between trait anger and the cortisol stress response (al'Absi, Carr, & Bongard, 2007). Clearly, more research is needed to address the linkage of smoking, anxiety, and stress among subclinical as well as clinical samples diagnosed with anxiety disorders. Due to the increasing literature on anxiety and smoking, adding stress to the prediction models may elucidate important mechanisms.

Furthermore, general perceived stress did not influence the physiological response to an acute stressor although higher PSS scores have been associated with psychological distress, physical symptomatology, and more life events (e.g., Cohen and Lichtenstein, 1990). In the current study the experienced level of stress did not explain more variance than trait anxiety did. Although we did not find any associations between general experienced stress and physiological stress markers, it is important to note that high anxious smokers are more likely to smoke when they experience negative affect and stress due to the expectation that smoking may alleviate their negative mood (Brandon & Baker, 1991; Langdon & Leventhal, 2014).

Results of the present study provide supportive evidence of particularly high smoking urges in response to stress among smokers with high trait anxiety. Thus, trait anxiety may play an important role in the relationship between stress and tobacco use. This is consistent with prior observations (al'Absi, Amunrud, et al., 2002; al'Absi et al., 2003): Nicotine's tranquilizing effects during anticipation of stressors are thought to provide distraction from anxiety-provoking triggers and nicotine itself may reduce approach-avoidance conflict-generated distress (see Kassel, Stroud, & Paronis, 2003 for review). It has been suggested that nicotine contributes to dopamine (DA) release in the mesolimbic system, which is an important component of the human reward system implying that rewarding properties may be enhanced by smoking during exposure to stress and anxiogenic situations (see Balfour, 1991 for review). Individuals exposed to chronic stress with an impaired ability of adapting

to stressful and anxiogenic situations may consider smoking as a reward and relief factor that may be associated with the vulnerability of developing nicotine dependence. The current finding of a positive relationship between trait anxiety and greater urges to smoke in response to stress provides experimental support to this account. Furthermore, it has been shown that negative mood and stress are associated with increased risk of smoking relapse (al'Absi, Hatsukami, & Davis, 2005; Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008). Thus, smokers with high trait anxiety may be more likely to fail smoking cessation attempts due to an increased desire to smoke for relief during stressful situations compared to smokers with low trait anxiety. We found a positive association with urges to smoke for relief and caffeine consumption which is in line with a prior work (see Swanson, Lee, and Hopp, 1994 for review; Treloar, Piasecki, McCarthy, & Baker, 2014). It has been suggested that appetitive effects of cigarettes decrease after time of smoking increases which may possibly indicate reduced dopaminergic tone (nicotine receptor desensitization; e.g., Pierce and Kumaresan, 2006). It is possible that caffeine alleviates this effect through its stimulating dopaminergic characteristics maintaining the dopaminergic tone. Caffeine intake may impact the dopaminergic system significantly when smoking urges begin to occur (for discussion see Treloar et al., 2014). The role of trait anxiety in this relationship is not clear at this point.

The results of the current study are limited by a number of factors. First, the findings cannot make any causal direction due to the cross-sectional nature of the research design. Thus, it is not clear whether chronic smoking and/or trait anxiety causes dysregulations in central stress regulatory systems, or whether such stress dysfunctions predispose individuals to smoke or have higher anxiety. Second, task difficulty was not assessed in this study. It is possible that habitual smoking and high levels of anxiety are associated with dysregulations in central motivational systems, which may lead smokers and anxious individuals to be less engaged in stress tasks, partially explaining attenuated physiological responses. Third, cardiovascular reactivity and subjective reports to laboratory stressors may not be equivalent to responses to naturalistic stressors in daily life (Gerin, Pickering, & Schwartz, 2004). Electronic diary methods integrated in smartphones could sample stress experience and nicotine use in stress situations occurring spontaneously in daily life and analyze their relationship using advanced statistical models (Mehl & Connor, 2012; Wilhelm & Grossman, 2010). Fourth, due to a different focus of examining predictors of smoking relapse in a larger study protocol (results reported elsewhere: al'Absi et al., 2015) more smokers than non-smokers were recruited. Thus, regression analysis results may have been somewhat biased due to unequal sample sizes (e.g., Zahn, 2010). Accounting for this issue, randomly generated subsamples confirmed a stable effect of trait anxiety on systolic blood pressure whereas the effect of smoking status was partially unstable. Thus, this effect should be interpreted with caution. Nonetheless, the study has several strengths such as the use of multiple biopsychological measures and the combined investigation of the role of trait anxiety in stress responses in smokers and nonsmokers.

In conclusion, the current investigation demonstrated that habitual smoking and trait anxiety were associated with attenuated cardiovascular responses to an acute stressor. The increased desire to smoke for relief associated with high trait anxiety among smokers may be an important risk factor of smoking relapse. Further research examining the relationship

between anxiety and nicotine dependence may productively focus on naturalistic observations of stress and smoking behavior in daily life in high-vs. low-anxious individuals using novel ambulatory assessment methods (Wilhelm & Grossman, 2010).

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Table 1

Summary of means (SD) and test statistics for participant characteristics and stress indices adjusting for unequal sample sizes

	Nonsmokers (*n* = 37)	**Smokers (*n* = 115)**	**Parametric test statistics**				
	M (SD)	***M (SD)***	***pooled SD***	***t* (150)**	***p***	**Cohen's *d***	**95% CI**
Age (years)	30.03 (11.10)	34.15 (11.30)	11.17	−1.79	0.076	−0.37	[−8.68, 0.44]
Education (years)	16.18 (3.12)	14.21 (2.61)	2.72	**3.50**	**0.001**	0.72	[0.85, 3.07]
BMI (kg/m^2)	24.23 (3.18)	25.12 (4.59)	4.27	−1.01	0.315	−0.21	[−2.64, 0.86]
Caffeine (drinks/day)	0.63 (0.98)	1.63 (1.80)	1.63	**−2.96**	**0.004**	−0.61	[−1.67,−0.33]
STAI - T	34.26 (9.73)	36.31 (9.00)	9.12	−1.09	0.277	−0.22	[−5.77, 1.67]
Perceived stress (PSS)	16.65 (4.59)	19.46 (4.68)	4.63	**−2.94**	**0.004**	−0.61	[−4.70,−0.92]
Salivary cortisol baseline (log nmmol/l)	2.06 (0.61)	2.06 (0.70)	0.68	0.00	1.00	0.00	[−0.25, 0.25]

Note. BMI: body mass index; STAI-T: State-Trait-Anxiety-Inventory (trait version); PSS: perceived stress scale.

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Table 2

Multiple regression analyses predicting systolic blood pressure responses to stress based on smoking status, trait anxiety, and perceived stress

	Stress reactivity in systolic blood pressure		
Predictor	**R^2**	**b**	**95% CI (b)**
Model 1	0.08		
Smoking		−3.39*	[−6.67,−0.11]
STAI - T		−0.37*	[−0.66,−0.08]
Smoking *STAI-T		2.56	[−0.60, 5.71]
Model 2	0.09		
Smoking		−3.73*	[−7.13,−0.33]
STAI-T		−0.43*	[−0.76,−0.10]
Smoking *STAI-T		2.61	[−0.56, 5.77]
PSS		0.16	[−0.26, 0.58]

Note. STAI-T: State-Trait-Anxiety-Inventory (trait version); PSS: perceived stress scale.

CI: confidence intervals (95%).

*$p < 0.05$.

Table 3

Multiple regression analyses predicting the urge to smoke for relief in response to stress among smokers based on trait anxiety, perceived stress, and caffeine intake

	Urge to smoke (for relief)		
Predictor	**R^2**	**b**	**95% CI (b)**
Model 1	0.09		
STAI - T		0.21**	[0.08, 0.34]
Model 2	0.09		
STAI - T		0.17	[−0.00, 0.34]
PSS		0.11	[−0.22, 0.44]
Model 3	0.13		
STAI - T		0.18*	[0.01, 0.35]
PSS		0.09	[−0.24, 0.41]
Caffeine intake		0.66*	[0.03, 1.29]

Note. STAI-T: State-Trait-Anxiety-Inventory (trait version); PSS: perceived stress scale.

Caffeine intake per day. CI: confidence intervals (95%). N=114.

* $p < 0.05$

** $p < 0.01$.