A Novel Stress-Based Intervention to Reduce Cigarette Use in Non-Treatment Seeking Smokers

Alexandra Barnabe

Department of Psychology

McGill University, Montreal, Quebec

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This thesis is dedicated to the late Dr. Harold Frank
who inspired me to study memory.

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ABSTRACT

Tobacco use is the leading cause of preventable mortality worldwide. Since current smoking cessation aids show only modest efficacy, new interventions are needed. Given the evidence that stress is a potent trigger for smoking, the present randomized clinical trial tested whether stress could augment the effects of an intervention (known as "memory updating") that entails reactivating smoking-related memories and then weakening them following principles of memory reconsolidation blockade. A total of 1365 non-treatment seeking cigarette smokers were screened, 118 were deemed eligible, 76 agreed to participate further and 62 completed all study sessions. Participants were assigned to one of four reactivation conditions composed of either a stressful or non-stressful psychosocial challenge followed by either smoking or neutral cues. Ten minutes after this manipulation, all underwent a 60-minute extinction procedure during which they viewed smoking-related videos and images and manipulated smoking paraphernalia. Physiological measures (skin conductance, blood pressure, and heart rate) and craving questionnaires (Questionnaire on Smoking Urges – Brief and Tobacco Craving Questionnaire – Short Form) were administered before and after reactivation and again after extinction. Twenty-four hours, two weeks and six weeks after the intervention, participants completed cue reactivity test sessions evaluating their psychophysiological responses to new smoking-related videos. Compared to participants who were not exposed to the laboratory stressor, the stressor-exposed groups exhibited greater psychophysiological responses during their intervention and greater decreases in cigarette use at two- and six-weeks follow-up independent of smoking cue exposure. All groups showed increases in motivation to quit and decreases in cigarette dependence and cue reactivity at follow-up. The role of personality factors and childhood adverse events were examined and, while neither affected the efficacy of the intervention, physical neglect emerged

as an independent predictor of decreased cigarette use. Together, these findings suggest that the ability of stress to activate cigarette seeking processes can be exploited to decrease cigarette use in people with low motivation to quit.

RÉSUMÉ

Mondialement, le tabagisme est la cause principale de décès évitables. Comme les outils actuels pour arrêter de fumer ne montrent qu'une efficacité modeste, de nouvelles interventions sont nécessaires. Compte tenu que le stress est un puissant déclencheur de la consommation de cigarettes, le présent essai clinique randomisé a testé si le stress pouvait augmenter les effets d'une intervention d'actualisation de la mémoire (« memory updating ») qui consiste à réactiver les souvenirs liés à la consommation de cigarettes, puis les affaiblir suivant les principes de reconsolidation de la mémoire. Au total, 1365 fumeurs non-motivés à cesser de fumer ont participé au processus de sélection initial. D'entre eux, 118 ont rempli les critères d'éligibilité, 76 ont accepté de participer à l'étude et 62 ont complété toutes les séances. Les participants ont été assignés à l'une des quatre conditions de réactivation composées soit d'un défi psychosocial stressant ou non stressant, suivi de vidéos reliés au tabagisme ou présentant des scènes neutres. Dix minutes après cette manipulation, les participants ont pris part à une procédure d'extinction de 60 minutes au cours de laquelle ils ont visionné des vidéos et des images liées au tabagisme et manipulé des articles pour fumeurs. Des mesures physiologiques (conductance cutanée, tension artérielle et fréquence cardiaque) et des questionnaires sur les envies de fumer (le « Questionnaire on Smoking Urges – Brief » et le « Tobacco Craving Questionnaire – Short Form ») ont été administrés avant et après la réactivation et à nouveau après l'extinction. Vingt-quatre heures, deux semaines et six semaines après l'intervention, les participants ont effectué des tests de réactivité évaluant leurs réponses psychophysiologiques à de nouvelles vidéos liées au tabagisme. Comparativement aux participants qui n'ont pas été exposés au stress en laboratoire, les groupes exposés au stress ont démontré des réponses psychophysiologiques plus importantes au cours de leur intervention et des diminutions plus

marquées de la consommation de cigarettes à deux et à six semaines de suivi, indépendamment de l'exposition aux stimuli liés au tabagisme. Tous les groupes ont démontré une augmentation de la motivation à arrêter de fumer et une diminution de la dépendance à la cigarette, et de la réactivité aux stimuli lors des suivis. Le rôle des facteurs de personnalité et du stress pendant l'enfance a été examiné et, bien qu'aucun facteur n'ait affecté l'efficacité de l'intervention, la négligence physique est apparue comme un élément indépendant prédisant la diminution de la consommation de cigarettes. Ensemble, ces résultats suggèrent que la capacité du stress à activer les processus comportementaux liés au tabagisme pourrait être exploitée pour réduire la consommation de cigarettes chez les personnes peu motivées à arrêter de fumer.

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Completing this project would have been far less enjoyable without the enthusiasm and dedication of the army of undergraduate students who assisted with recruitment, screening interviews, video coding, data entry, analyses, and literature reviews for months on end. It would have been insufferable without support and comic relief from my brilliant friends, family, and friends who have become family. Alex #1, C, Courtney, Etienne, Francis, Fred, Jon, Julien, Karmalak, Katharina (and Mischa), Kathryn, Pam, Richard, Ryan and Sven: I can't imagine the last few years without your memes, late night conversations, cat photos, and existential debates. Mom, thank you for passing on your resilience. Dad, thank you for sharing your intellectual curiosity and love of learning. These acknowledgements wouldn't be complete without thanking my fur kids, Pepperoni and Chewy, for their companionship and regular Zoom appearances.

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CONTRIBUTIONS TO ORIGINAL KNOWLEDGE

The dogma of memories as fixed and unchangeable entities was challenged a few decades ago with the rediscovery of memory reconsolidation. Reconsolidation work suggests that memories are susceptible to modification after storage. Studies have harnessed this potential for the treatment of anxiety-related disorders, such as phobias and post-traumatic stress disorder, and substance use disorders. Much of this research has focused on the use of pharmacological interventions, but more recent work has tested the potential of less invasive behavioral approaches.

The present thesis adds to the small body of literature on reconsolidation in substance use disorders and to the even smaller body of literature on reconsolidation-based behavioral interventions to treat addiction. Herein, I describe a novel intervention for cigarette smoking, using stress for the first time (to my knowledge) to reactivate smoking-related memories, potentially enhancing the ability to disrupt their reconsolidation. Our findings not only provide preliminary insight into the influence of stress on a behavioral reconsolidation paradigm, but also indicate that this could be a promising treatment to reduce cigarette use in smokers with low motivation to quit. Further, our results suggest that this stress-based intervention can be successfully applied regardless of individual factors, such as early life stress or personality traits.

CONTRIBUTION OF AUTHORS

The present thesis contains a novel experiment to study a memory updating manipulation in non-treatment seeking cigarette smokers. The experiment was designed by Dr. Karim Nader, Dr. Marco Leyton, Karine Gamache and me. Recruitment and screening interviews were performed by me and a large team of undergraduate students (Nora Boudghène, Alexane Doucet, Sara Eldabaa, Amanda Gallant, Victoria Gilmore, Joshua Iverson, Adelaide Jensen, Sofia Osorno, João Vitor Paes de Camargo, Regan Palmerio, Mathilde Rioux, Stefanie Todesco and Alexander Toundjian). Following the screening interviews, I conducted diagnostic interviews of potential participants. Dr. Leyton and I then determined study eligibility.

The study comprised five in-person sessions and subsequent telephone follow-ups with each participant. Materials used in these sessions included video and image cues, smoking-related paraphernalia, and a psychosocial stress task. I acquired the images and videos from online sources and other research groups. Video and image cues were rated and counterbalanced by two undergraduate students (Alexane Doucet and Adelaide Jensen) and myself, and I then assembled and edited the material with video editor Elena Pérez Gómez. The psychosocial stressor, its control task, and associated scripts were provided by Dr. Jens Pruessner.

I performed all sessions and collected physiological and self-report data. The data was then entered, coded and verified by many undergraduate students (Erin Allen-Flanagan, Mathew Apostolatos, Sara Eldabaa, Amanda Gallant, Joshua Iverson, Adelaide Jensen, Paige Johnston, Bronwen Lathrop, Nilufar Mokhtarian, João Vitor Paes de Camargo, Regan Palmerio, Mathilde Rioux, Anne-Marie Saucier, Justin Simo, Victoria Laurin, Stefanie Todesco and Alexander Toundjian) and I. Data analysis and interpretation was carried out by me and assisted by Dr.

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All the suffering, stress and addiction comes from not realizing you already are who you are looking for.

- Jon Kabat-Zinn

INTRODUCTION

Overview

Tobacco use is the primary cause of preventable death worldwide (Centers for Disease Control and Prevention, 2020). The effect is large, with direct use and second-hand smoke leading to over eight million deaths per year (World Health Organization, 2020). While most tobacco users are familiar with the consequences of smoking, abstinence remains challenging for many. Among the most commonly reported barriers are the perceived difficulties of quitting and coping with stress (Hughes, 2009; Twyman et al., 2014; Villanti et al., 2016), potentially related to the ability of stressful events to augment responses to cigarette cues (Dagher et al., 2009).

Cessation aids and therapies are available, but are infrequently used (Cokkinides et al., 2005) owing partly to their modest efficacy (Jiloha, 2014; Piasecki, 2006; Teneggi et al., 2002; Waters et al., 2004) and high cost (Gross et al., 2008; Rosenthal et al., 2013). Together, these observations highlight the need for new treatments that can increase motivation to quit and improve remission rates.

In the following sections, I provide (1) an overview of the epidemiology and psychopharmacology of cigarette use and addiction; (2) details about two primary triggers for smoking: exposure to stress and cigarette-related cues; (3) the influence of childhood adversity; (4) the role of personality factors; and (5) the effectiveness of current treatments, including pharmacotherapies, nonpharmacological interventions, and cue exposure therapy. Given the shortcomings of these treatments, a new behavioral intervention called stress-based memory updating is proposed. A feasibility study tested whether this intervention could be effective in non-treatment seeking smokers. The methods, results and implications are discussed below.

Cigarette Use: Epidemiology

Tobacco use remains rampant despite widespread efforts at tobacco prevention and control over several decades (World Health Organization, 2014; Xiao & Wang, 2019). While use has decreased across the globe, more than 1 billion people – over 20% of adults – smoke cigarettes (Perez-Warnisher et al., 2018; Reitsma et al., 2021; World Health Organization, 2021). In Canada, approximately 4.2 million people above the age of 12 smoked cigarettes in 2020[1] (Statistics Canada, 2021b). Although this represents a decrease from previous years, rates in the 18 to 34 (14.9%), 35 to 49 (15.1%) and 50 to 64 (16.1%) age groups continue to be higher than other age groups (Statistics Canada, 2021b). Men remain more likely to smoke than women, with smoking rates of 15.8% compared to 10.1%, as are individuals with lower socioeconomic status and less education (Chaiton & Callard, 2019; Statistics Canada, 2021b; Wellman et al., 2018).

Quitting smoking is challenging. Despite this, over 40% of Canadian smokers reported attempting cessation at least once in the previous year and 30% reported trying more than once (Statistics Canada, 2021a). The majority (65%) made their quit attempts without cessation aids, 32% used nicotine replacement and 29% tried another method, including online support or prescription medication (Statistics Canada, 2021a). Their efforts were largely unsuccessful. Only an estimated 12% of smokers who attempt to quit remain abstinent the following year (Reid et al., 2015; Reid et al., 2019). This low success rate contributes to why the prevalence of former smokers has remained unchanged in recent years (Carpenter et al., 2010; Physicians for a Smoke-Free Canada, 2022; Statistics Canada, 2021a). Indeed, there is evidence that the decreases in smoking rates are more closely related to population turnover than improvements in cessation (Physicians for a Smoke-Free Canada, 2022; Statistics Canada, 2021a).

[1] Estimates only include Canadian provinces (not territories).

Acute Triggers of Cigarette Use

Cigarette-Paired Cues

Cigarette use disorder can be conceptualized as a disorder of learning and memory (Bevins & Palmatier, 2004; Everitt & Robbins, 2005). Within 30 seconds of a cigarette puff inhalation, nicotine reaches the brain where it binds to nicotinic receptors and triggers a cascade to signal reward (Benowitz, 2008; Liakoni et al., 2019). Over time, the brain forms associations between these rewarding effects and co-occurring cues (people, places, and objects) (Perkins et al., 2017; Rupprecht et al., 2015), such that the cues alone can come to elicit psychophysiological responses[2] including those that trigger drug-seeking behaviors (Carter & Tiffany, 1999; Waters et al., 2004). These conditioned effects are thought to include the ability to elicit approach (reward), the ability to sustain effort to obtain the cues (reinforcement), and the ability to augment efforts to obtain the drug itself (Cardinal et al., 2002; Everitt et al., 2001; Milton & Everitt, 2010).

Stress

Stressful events can also potently trigger cigarette cravings (Buchmann et al., 2008; Childs & de Wit, 2010) and use (Nakajima et al., 2020). Smokers under acute stress smoke more intensely[3] and are less likely to resist smoking (McKee et al., 2011). They report increased satisfaction from use (McKee et al., 2011) and commonly believe it will reduce stress (Kassel et al., 2003; Scales et al., 2009). However, there is little evidence to support this belief (Childs & de

[2] Psychophysiological responses to cues can include, but are not limited to, decreased heart rate and increased craving, blood pressure and skin conductance. Heart rate deceleration may index increased attention to cues (Graham & Clifton, 1966) while cardiac responsivity (sympathetic arousal) may index the motivational state triggered by the cues (Boucsein, 2012; Naqvi & Bechara, 2006; Niaura et al., 1988).

[3] In addition to increasing cravings (a psychological response) and smoking (a behavioural response), acute stress typically increases physiological responses, including heart rate, blood pressure and skin conductance (Feldman et al., 1999; Weber et al., 2022).

Wit, 2010; Heishman, 1999; Kassel et al., 2003). Rather, stress may dampen the effects of nicotine, inducing increased cigarette use to compensate (Buchmann et al., 2008; Childs & de Wit, 2010; Winders et al., 1998). Stress may also decrease the ability to control smoking-related urges through a depletion of self-regulatory resources (Muraven & Baumeister, 2000; Slopen et al., 2013). Indeed, individuals with lower self-control are at greater risk of smoking (Bogg & Roberts, 2004; Daly et al., 2014) potentially because self-control has a stabilising influence on emotions (Bogg & Roberts, 2004; Daly et al., 2014; Tangney et al., 2018).

Additionally, stressful events can affect the stabilization (consolidation) of memories (Hyman, 2005; Paré, 2003), such as those related to cigarette use. Like nicotine, stressors can facilitate reward-associated learning and lead to habit formation (Hyman et al., 2006; Schwabe et al., 2011; Taylor et al., 2014; Wood & Rünger, 2016). The promotion of habitual over goal-directed behaviors in times of stress may hinder the reduction or cessation of cigarette use (Jager, 2003; Jesus et al., 2016; Schwabe & Wolf, 2009), especially in individuals with greater cigarette dependence (Webb et al., 2009).

Risk Factors for Cigarette Use

Adverse Childhood Events

Early life adversity can increase later stress responses and interfere with coping abilities (Chen & Baram, 2016; McLaughlin et al., 2010), potentially by altering brain development (Chen & Baram, 2016; Maniam et al., 2014). This can increase susceptibility to health-risk behaviours, such as cigarette use (Chen & Baram, 2016; Maniam et al., 2014; Topitzes et al., 2010). Indeed, childhood maltreatment predicts early onset smoking (Iakunchykova et al., 2015; Mills et al., 2014), lifetime cigarette use (Kristman-Valente et al., 2013; Power et al., 2020) and smoking persistence (smoking despite having a disease exacerbated by cigarette use) (Edwards et

al., 2007). Maltreatment alters stress-sensitive regions of the developing brain and responsivity of the hypothalamic-pituitary-adrenocortical (HPA) axis, increasing vulnerability to substance use problems (Duffy et al., 2018; Goeders, 2003; Hyman et al., 2006; Maté, 2012; Sinha, 2008; Whittle et al., 2013), such as cigarette use disorder.

Heightened stress sensitivity increases responsiveness not only to subsequent stressors but also to cigarette use (Berridge, 2012; Hellberg et al., 2019). By heightening vulnerability to cigarette use, these neuroadaptations, in combination with emotional and behavioral consequences of maltreatment, can enhance the incentive salience of reward cues and contribute to smoking persistence (Berridge, 2012; Hellberg et al., 2019; Kendall-Tackett, 2002; Pecina et al., 2006; Richards et al., 2011; Sinha, 2008; Wanat et al., 2008).

Types of Adverse Childhood Events and Cigarette Use. The different forms of childhood maltreatment have both overlapping and distinct effects on cigarette use.

Physical Abuse. Childhood physical abuse, defined as non-accidental bodily harm (Mok, 2008), increases the risk of early smoking initiation (Anda et al., 1999; Jun et al., 2008). As substantial emotion regulation abilities are developed in childhood through observational learning and modelling of parents or caregivers (Morris et al., 2007), physical abuse can lead to patterns of emotional dysregulation (Morris et al., 2007) which in turn increase the risk of cigarette use (Rogers et al., 2018; Topitzes et al., 2010; Wolff et al., 2016). Indeed, physical abuse predicts later cigarette use, with greater harm exposure leading to greater use (Anda et al., 1999).

Emotional Abuse. Adults who experienced repeated emotional and psychological maltreatment in childhood are also more likely to smoke (Alcalá et al., 2016; Chen et al., 2017) and begin smoking earlier (Anda et al., 1999). These effects may be cumulative, with more

frequent emotional abuse increasing the likelihood of smoking and leading to greater cigarette use in some (Anda et al., 1999; Taha et al., 2014) but not all (Lewis et al., 2019) studies.

Family modelling (as with physical abuse, discussed above) and peer group affiliation may play a role in the development of cigarette use (Topitzes et al., 2010; Yoon, 2020; D. Yoon et al., 2020). Indeed, emotionally abused youth disproportionately affiliate with severely antisocial peer groups whose members are more likely to smoke cigarettes (Yoon, 2020; D. Yoon et al., 2020). These youth may start smoking to conform (Hill, 1971) or maintain proximity with peers (Liu et al., 2017) and may persist in order to fulfill their needs for social acceptance and validation (Fuligni & Eccles, 1993; Liu et al., 2017).

Sexual Abuse. Any involvement of a child in sexual activity that the child is not developmentally ready for, does not fully understand, or is unable to provide informed consent to is typically considered sexual abuse (Mathews & Collin-Vézina, 2019; Wyatt & Peters, 1986). Like other forms of childhood abuse, sexual abuse increases the risk of early smoking onset (Jun et al., 2008; Roberts et al., 2008) and cigarette use in adolescence and adulthood (Anda et al., 1999; Jun et al., 2008; Kim & Williams, 2009). Familial emotional support and an increased sense of self-worth may buffer this risk (Goldstein et al., 2013; Jun et al., 2008; Kim & Williams, 2009) potentially through increased use of adaptive coping strategies (Guelzow et al., 2003).

Physical Neglect. Despite being one of the most prevalent forms of maltreatment (Afifi et al., 2011; Barbosa et al., 2014; Hussey et al., 2006; Keyes et al., 2012; Witt et al., 2017), fewer studies have examined the effect of neglect on health outcomes (Cohen et al., 2017; Stoltenborgh et al., 2013), such as smoking behaviors (Grummitt et al., 2021; Lewis et al., 2011). Physical neglect, defined as the failure to meet a child's physical needs by providing adequate nutrition, hygiene, clothing, medical care and supervision (Stoltenborgh et al., 2013), increases the risk of

future cigarette use (Cammack et al., 2019; Cohen et al., 2017). Use may increase gradually over time (S. Yoon et al., 2020) and may persist (Cammack et al., 2019) possibly because neglected youth and adults struggle to address their health-compromising behaviors without prior adaptive parental or caregiver modelling (Cammack et al., 2019; Farruggia & Sorkin, 2009; Peshevska et al., 2014).

Emotional Neglect. Childhood emotional neglect, the failure to meet a child's psychological needs by connecting emotionally and providing affection and nurturance (Crittenden, 1999; Stoltenborgh et al., 2013), also predicts later cigarette use (Cohen et al., 2017; Collado et al., 2019). Individuals who experienced emotional neglect are more likely to have limited emotion regulation abilities (Berzenski, 2019; Wolff et al., 2016) and feel lonely and isolated (Loos & Alexander, 1997). In turn, they may smoke cigarettes to foster a sense of belongingness and avoid isolation (Barahmand et al., 2016; Leigh-Hunt et al., 2017).

Overall, childhood adverse events can alter behavioral, emotional and neurobiological functioning, increasing the likelihood of later adverse health outcomes, such as early onset and lifetime cigarette use (Kim & Cicchetti, 2006; Topitzes et al., 2010). Continued smoking and difficulties with cessation are also more likely (Cammack et al., 2019; Lee et al., 2020; Smith et al., 2015; Taha et al., 2014), suggesting that it may be beneficial to incorporate emotion regulation and coping skills training in cessation interventions (Lee et al., 2020; Taha et al., 2014).

Personality Factors

Numerous studies have found associations between personality traits and important health outcomes. These associations hold across the lifespan: personality traits in childhood predict both self- and physician-rated health in adulthood (Chapman et al., 2007; Hampson et al.,

2007) as well as biomarkers of health and longevity (Hampson et al., 2013; Jokela et al., 2013; Roberts et al., 2007).

Health behaviors involve both the adoption of healthy lifestyle choices (e.g., healthy eating, exercise) and the avoidance of harmful or risky behaviors (e.g., frequent alcohol consumption, cigarette smoking) (Yañez et al., 2020). Different personality traits may underlie the implementation or avoidance of certain behaviors (Munafo et al., 2007). In terms of smoking, both its initiation and persistence have been consistently reported to be associated with approach-related traits, such as extraversion, sensation seeking, and impulsivity, as well as avoidance-related traits, such as neuroticism and anxiety sensitivity (Munafo et al., 2007). While approach-related traits may increase the predisposition to smoke because of the reinforcing aspects of cigarette use, avoidance-related traits may increase this tendency in order to soothe or regulate negative emotions (Glautier, 2004; Munafo et al., 2007).

Personality Traits and Cigarette Use. Many personality traits have been studied in association with smoking outcomes. Here, the relationship between smoking and the following personality traits will be discussed: extraversion, openness to experience, neuroticism, agreeableness, conscientiousness, anxiety sensitivity, hopelessness, sensation seeking, and impulsivity.

Extraversion. Smokers are more likely to be extraverted than non-smokers (Buczkowski et al., 2017; Munafo et al., 2007; van Loon et al., 2001), potentially because their higher levels of sociability lead to greater exposure to social smoking and smoking-related cues (De Leeuw et al., 2010; Munafo et al., 2007; Watson & Clark, 1997). They also have larger excitement-seeking tendencies which may increase their desire for the stimulating effect of nicotine (Buczkowski et al., 2017; De Leeuw et al., 2010; Eysenck & Eaves, 1980). It follows that extraverts may be less

likely to quit smoking than their more introverted counterparts (Abe et al., 2019; Helgason et al., 1995), but this has not been reliably found (Hooten et al., 2005; van Loon et al., 2005).

Openness to Experience. Being open-minded, curious, and accepting of novel experiences might also increase the risk of experimenting with new substances (Chapman et al., 2009; Turiano et al., 2012). It is therefore unsurprising that openness to experience is associated with smoking initiation (Chapman et al., 2009; McCrae & Costa Jr, 1997; Turiano et al., 2012) and lifetime cigarette use (Campbell et al., 2014; Chapman et al., 2009; Otten et al., 2008; Zvolensky et al., 2015). It is also related to a smaller likelihood of smoking cessation or abstinence following treatment (Hooten et al., 2005; Leung et al., 2013).

Neuroticism. Neuroticism, a trait characterized by emotional lability and vulnerability to stress, is linked to an increased likelihood of smoking onset in adolescents (Byrne et al., 1995; Cherry & Kiernan, 1976; Vink et al., 2003) and maintenance in adults (McCrae et al., 1978; Munafo et al., 2007). Two hypotheses may account for these associations: individuals higher in neuroticism are more likely to struggle with impulse control and engage in risk-taking behaviors (Marks & Lutgendorf, 1999; Memetovic et al., 2016; Von Ah et al., 2005), and they may smoke to soothe negative emotions or cope with stress (Eysenck & Eaves, 1980; Munafo et al., 2007; Schilling, 1991). These hypotheses may also explain why high neuroticism is associated with lower odds of smoking cessation (del Río et al., 2015; Hakulinen et al., 2015).

Agreeableness. Agreeableness is a trait characterized by trust, straightforwardness, altruism, and cooperativeness (Costa Jr et al., 1991). Lower levels of agreeableness have often been linked with smoking (Malouff et al., 2006; Schlyter et al., 2016; Terracciano & Costa Jr, 2004) and intensity of cigarette use (Paunonen, 2003). Rebelliousness, a characteristic related to low agreeableness (Gullone & Moore, 2000), may contribute to the initiation and maintenance of

cigarette use (Stewart & Livson, 1966; Terracciano & Costa Jr, 2004). Those lower in agreeableness are less likely to follow health recommendations and have less need for social approval, resulting in lower motivation to quit and poorer cessation outcomes (Kulkarni et al., 2018; Schlyter et al., 2016; Terracciano & Costa Jr, 2004).

Conscientiousness. Conscientiousness, defined as the propensity to be hardworking, responsible, and rule abiding (Roberts et al., 2009; Roberts et al., 2014), may protect against smoking initiation and maintenance (Kubička et al., 2001; Zvolensky et al., 2015). More conscientious individuals are less likely to take health risks, especially when those risks could affect others (for instance, through second-hand smoke) (Hampson et al., 2000; Kang, 2022). Their self-discipline and impulse control may help them reduce or abstain from cigarette use and could account for their reduced rates of use (Abe et al., 2019; Terracciano & Costa Jr, 2004; Zvolensky et al., 2015).

Anxiety Sensitivity. Individuals with heightened anxiety sensitivity often experience fears around bodily sensations and attribute catastrophic meanings to them (Conrod et al., 2000). They are more likely use substances, such as cigarettes, to soothe these fears (Gonzalez et al., 2008; Schlauch et al., 2015; Siu, 2011). With repeated use and soothing, the motivation to use these substances may increase, as the contrast between drug use and other non-drug options heightens (Leyton, 2021; Leyton & Vezina, 2014). This contrast, in turn, further augments drug-seeking and use (Leyton, 2021; Leyton & Vezina, 2014). Anxiety sensitivity is also linked to earlier relapse after cessation attempts (Zvolensky et al., 2006) potentially because of greater perceived intensity of withdrawal symptoms (Zvolensky et al., 2004).

Hopelessness. Hopelessness, or pervasive negative beliefs about oneself and one's future, can lead to maladaptive, risky, and unhealthy behaviors (Jalilian et al., 2014; Yip & Cheung,

2006). It is linked to difficulties with interpersonal relationships and problem solving (Fırıncık & Gürhan, 2019; Zeyrek et al., 2009), which in turn can lead to smoking by increasing social withdrawal and loneliness (Jalilian et al., 2014; Martínez-Vispo et al., 2019; Shankar et al., 2011; Wootton et al., 2021). Loneliness and decreased feelings of competence can further affect smoking cessation, resulting in reduced motivation to quit and shorter periods of abstinence (Clancy et al., 2013; Kenney et al., 2009).

Sensation Seeking. Sensation seeking, the desire for stimulating and arousing experiences, can also lead to increased risk-taking behaviors (Memetovic et al., 2016; Zuckerman, 1979). Indeed, high sensation seekers derive greater anticipatory pleasure from risky activities and tend to minimize risks compared to low sensation seekers (Zuckerman, 1979, 1991). In terms of cigarette use, however, high and low sensation seekers appear to equally appraise the risks but high sensation seekers are more likely to smoke, possibly because of their favorable attitude towards risky situations (Zuckerman, 1991; Zuckerman et al., 1990). As the novelty of smoking diminishes, high sensation seekers may be less motivated to maintain this habit, resulting in similar cessation outcomes as low sensation seekers (Carton et al., 2000; Hall et al., 2012).

Impulsivity. Higher levels of impulsivity predict smoking initiation (Kvaavik & Rise, 2012; Memetovic et al., 2016) and maintenance (Balevich et al., 2013; Flory & Manuck, 2009). Evidence suggests that this may be because impulsivity can increase positive expectancies about substance use and lead to a discounting of its consequences (Audrain-McGovern et al., 2009; Doran et al., 2007; Wittmann & Paulus, 2008). These expectancies may further account for why impulsive smokers are less likely to quit and more likely to relapse (Bloom et al., 2014; Pattij & De Vries, 2013; VanderVeen et al., 2008). They may also experience heightened cravings and

withdrawal symptoms which can delay abstinence or precipitate relapse (Bloom et al., 2014; Doran et al., 2004; Mathew et al., 2015; VanderVeen et al., 2008).

Current Smoking Cessation Treatments

Pharmacotherapies

Four smoking cessation aids are currently available in Canada: bupropion, varenicline, nicotine replacement therapy (NRT), and cytisine (Hersi et al., 2019). Bupropion and varenicline seem able to decrease withdrawal symptoms and cravings, effects that are thought to reflect actions on the catecholamines, dopamine and norepinephrine (McIvor, 2009). Prescriptions are required for these medications (Hersi et al., 2019), presenting an obstacle to their use. Over the counter options, such as NRTs, are more widely used (Hersi et al., 2019) and provide nicotine in the form of patches, chewing gum, sprays, inhalers, tablets, and lozenges (Mills et al., 2010). Although NRTs have similar clinical effectiveness as bupropion, many studies report no difference between either of these cessation aids and placebo (CADTH, 2016; Stapleton et al., 2013). Varenicline appears to improve abstinence compared to placebo (Cahill et al., 2016), yet only shows marginal success compared to NRTs (CADTH, 2016). Bupropion, varenicline and NRTs can be costly (between $200-450 per month) (Karnieg & Wang, 2018) and have mild side effect profiles that typically include dry mouth, nausea, gastrointestinal discomfort, insomnia, and vivid or abnormal dreams (Kaur et al., 2009; Richmond & Zwar, 2003).

In 2017, Health Canada approved cytisine, a naturally occurring analogue of varenicline, for sale over the counter (Karnieg & Wang, 2018). By mimicking the effects of nicotine, cytisine binds to the nicotine receptors to reduce withdrawal symptoms (Jeong et al., 2015). It has been shown to improve abstinence compared to NRTs and placebo and is more affordable (around $50 per month) than other pharmacotherapies (Karnieg & Wang, 2018). However, it is not free of

adverse events, which include dry mouth, nausea, vomiting, and sleep disturbances (Cahill et al., 2016).

Nonpharmacological Interventions

Given the modest efficacy of current pharmacotherapies (Jiloha, 2014; Piasecki, 2006; Teneggi et al., 2002; Waters et al., 2004), behavioral strategies have been tested for use alone or in combination with cessation medication. The most common ones will be reviewed here: cognitive behavior therapy (CBT), motivational interviewing (MI), mindfulness, and telephone support (quitlines).

CBT, the standard behavioral intervention for smoking cessation (Hernández-López et al., 2009), focuses on identifying smoking triggers and using adaptive coping strategies when cravings occur (Jhanjee, 2014). It is often combined with relapse prevention skills, such as relaxation training and problem solving (Jhanjee, 2014; Marlatt & Donovan, 2005). While there are few studies evaluating the effectiveness of CBT alone (without medication) (Guichenez et al., 2007), there was no difference in cigarette dependence in smokers following either three sessions of CBT or basic health education (Raja et al., 2014). However, it is possible that more sessions were required as effectiveness increases with treatment duration (Jaén et al., 2008). Six sessions of CBT combined with NRT resulted in higher rates of sustained abstinence than basic health education with NRT (Webb et al., 2010).

Unlike CBT and traditional therapies that rely on psychoeducation and skills training (Hettema & Hendricks, 2010; Jaén et al., 2008), MI uses reflective listening, highlights discrepancies between behaviors and values, and reinforces change talk to help smokers resolve their ambivalence towards quitting (Jaén et al., 2008). While it has been found effective at increasing quit attempts (Butler et al., 1999; Chan et al., 2005; Steinberg et al., 2004), it is

unclear if it improves abstinence in smokers who are motivated to quit (Butler et al., 1999; Hettema & Hendricks, 2010; Jaén et al., 2008).

Inspired by the Mindfulness-Based Stress Reduction program (Kabat-Zinn, 2013; Oikonomou et al., 2017), mindfulness training teaches smokers to identify and emotionally detach from cigarette cravings, withdrawal symptoms and negative emotions (Brewer et al., 2011). Several studies have found this eight-session program effective at reducing cigarette use and increasing abstinence (Brewer et al., 2011; de Souza et al., 2015), possibly by enhancing volitional control over smoking (Spears et al., 2017). Clinical efficacy has been found to be similar to other smoking cessation programs, such as CBT (Maglione et al., 2017; Spears et al., 2017) and quitlines (Maglione et al., 2017).

As the costs of CBT, MI and mindfulness training may present a barrier to use, free-of-charge quitlines are offered by providers in the North American Quitline Consortium or through private, employer-funded services (Asfar et al., 2012; Lichtenstein et al., 2010). While services differ across quitlines, most offer counselling and self-help materials (Cummins et al., 2007; Ossip-Klein & McIntosh, 2003). Despite moderate effectiveness in improving cessation rates (Danielsson et al., 2014; Ossip-Klein et al., 1991), they are rarely used (Cummins et al., 2007) potentially due to a lack of knowledge about quitlines, mistrust in the credibility of providers, and privacy concerns (Sheffer et al., 2011; Solomon et al., 2009).

Extinction / Cue Exposure Therapy

Most psychological treatments for smoking cessation focus on increasing motivation or emotion regulation skills (Barry, 1999; Beck et al., 1993; Newman, 2001). Cue exposure therapy (CET), on the other hand, attempts to weaken the associations between smoking-related cues and the rewarding effects of nicotine (Marlatt, 1990). Through repeated exposure in the absence of

cigarette use, the cues theoretically lose their associative strength resulting in diminished cue-elicited cravings (Marlatt, 1990; Thewissen et al., 2006). While CET appears to decrease posttreatment cigarette cravings (Choi et al., 2011; LaRowe et al., 2007; J. Lee et al., 2004) and cigarette use (Götestam & Melin, 1983; Lowe et al., 1980), long-term clinical efficacy is lacking (Conklin & Tiffany, 2002; Lowe et al., 1980; Niaura et al., 1999).

The failure of CET to prevent relapse may be due to one or more of the following mechanisms: renewal, reinstatement, spontaneous recovery, and/or reacquisition (Bouton, 2002). When smokers return to a familiar environment following CET, the extinguished responses to smoking cues can be recovered by smoking again (reinstatement) or by encountering cues in a different context (renewal) (Bouton, 2002; Conklin & Tiffany, 2002; Thewissen et al., 2006). Responses can also reoccur with the passage of time after CET (spontaneous recovery) or when use is paired anew with smoking cues (reacquisition) (Bouton, 2002; Conklin & Tiffany, 2002).

These mechanisms of relapse point to the likelihood that CET results in new learning rather than erasure or unlearning of previously learned conditioned responses (Bouton, 2002). Following from this premise, the effectiveness of CET is determined by the odds that posttreatment exposure to an extinguished smoking cue will elicit the new learning (abstinence) rather than the original learning (cigarette use) (Conklin & Tiffany, 2002). Behavioral and pharmacological interventions have been paired with CET to increase the probability of evoking new learning. Although combined CET, counselling and NRT resulted in diminished cue-induced cravings (Unrod et al., 2014), they failed to improve abstinence rates (Niaura et al., 1999).

Memory Reconsolidation Blockade

Memory reconsolidation blockade procedures might overcome the limitations of CET (Bevins & Palmatier, 2004; Bouton, 2002). Memory reconsolidation theory postulates that stable (consolidated) memories become labile when recalled and are thus susceptible to modification (Haubrich & Nader, 2016; Nader et al., 2000). While in this labile state, it is possible to interfere with their restabilization (reconsolidation) through memory reconsolidation blockade (Haubrich & Nader, 2016; Nader et al., 2000). A typical reconsolidation blockade paradigm involves three phases: (1) a memory retrieval phase in which a previously consolidated memory is recalled and becomes labile, (2) a modification phase in which a behavioral procedure or pharmacological agent is administered soon after[4] the memory's recall, and (3) a test phase in which memory retention is assessed (Nader, 2016; Schafe & LeDoux, 2000).

This paradigm first garnered interest two decades ago when it was tested in laboratory rats. In this seminal study, the rats' conditioned fear response, generated through pairing a tone with a foot-shock, was disrupted by injecting anisomycin, a protein synthesis inhibitor, into the amygdala 10 minutes following the presentation of the tone (memory retrieval) (Nader et al., 2000). The fear response was no longer observed when animals were tested up to 14 days following anisomycin administration (Debiec et al., 2002; J. L. Lee et al., 2004; Nader et al., 2000).

In humans, memory reconsolidation blockade has been replicated primarily by using the β-adrenergic receptor antagonist propranolol and has shown promise for the treatment of stress- and anxiety-related disorders (Björkstrand et al., 2017; Brunet et al., 2008; Schiller et al., 2010;

[4] While pharmacological agents are typically administered immediately after memory retrieval, some are administered before (Brunet et al., 2011; Wood et al., 2015) in order to reach their peak bioavailability soon after retrieval (Dey et al., 1986; Marino et al., 1987).

Soeter & Kindt, 2015; Telch et al., 2017). Through effects putatively mediated by the amygdala (Hurlemann et al., 2010; Simson et al., 2001), propranolol prevented the return of a conditioned fear response to a spider image (measured as fear potentiated startle) when administered prior to the retrieval of the fear memory (Kindt et al., 2009). The memory of the spider image was preserved but the fear response was attenuated at one-month follow-up (Soeter & Kindt, 2010), while fear responses were reinstated in participants who received propranolol without retrieval and placebo with retrieval (Kindt et al., 2009). These results suggest that disrupting memory reconsolidation blockade can selectively attenuate the emotional component of a memory while declarative memory remains intact (Tronson & Taylor, 2013).

More recent work has begun to apply the same principles to altering reward-related behaviors (Chen et al., 2021; Diergaarde et al., 2006; Lee et al., 2005; Milton et al., 2008; Wouda et al., 2010). In rats, pre-retrieval infusion of the transcription inhibitor zinc-finger 268 antisense oligodeoxynucleotides into the basolateral amygdala impaired a conditioned cocaine self-administration response (a nosepoke in response to light signalling cocaine availability) (Lee et al., 2005). Systemic post-retrieval propranolol infusions similarly disrupted sucrose- and cocaine-seeking (Milton et al., 2008).

Human studies have used similar paradigms to alter drug-related memories. In abstinent heroin users, propranolol administered prior to the retrieval of a previously learned word list disrupted recall for heroin-related words but not neutral words 24-hours later (Zhao et al., 2011). In cocaine users, administration of propranolol post-presentation of cocaine-related videos and paraphernalia reduced physiological and craving responses to these cues 24-hours later compared to placebo controls (Saladin et al., 2013). The propranolol group's decreased craving responses

were maintained at one-week follow-up, but were not significantly different from controls (Saladin et al., 2013).

Although the above studies are encouraging, other studies had mixed results (Jobes et al., 2015; Lonergan et al., 2016; Pachas et al., 2015). In polydrug users maintained on methadone treatment, post-retrieval propranolol (as compared to placebo) led to increased instead of decreased craving and cardiovascular responses to cocaine-related cues at one-week follow-up (Jobes et al., 2015). At five-week follow-up, there were no group differences (Jobes et al., 2015). In cigarette smokers, post-retrieval propranolol did not change craving or physiological responses to individualized smoking-related scripts (as compared to placebo) at one-week follow-up (Pachas et al., 2015).

These null findings might be explained in part by drug interactions. Propranolol has been shown to alter methadone's efficacy (Ferrari et al., 2004; Hollister & Prusmack, 1974). This in turn might increase heroin withdrawal symptoms and stimulate cocaine cravings (as heroin users often turn to cocaine to mitigate heroin withdrawal symptoms[5]) (Hunt et al., 1984; Leri et al., 2003). Cigarette smoking affects the bioavailability of propranolol (Faber et al., 2005), decreasing its efficacy (Horn & Hansten, 2007; Schaffer et al., 2009). Altering the dosage, timing or number of treatments may overcome these interactions (Faber et al., 2005). In cigarette smokers, a single dose of propranolol administered prior to retrieval decreased self-reported cigarette cravings 24 hours later (Lin et al., 2021). In a small, mixed sample of substance users, six biweekly pre-retrieval propranolol treatment sessions reduced self-reported drug cravings (Lonergan et al., 2016).

[5] Cocaine may accelerate the elimination of methadone in the body, thereby reducing methadone serum concentration (Tennant & Shannon, 1995).

Memory Updating

Behavioral paradigms, such as memory updating, circumvent the challenges of drug interactions and timing of treatment administration. Memory updating, like pharmacological reconsolidation blockade, involves modifying the strength of a memory by interfering with its reconsolidation (Lee, Nader, & Schiller, 2017). This intervention, also known as retrieval-extinction, has three distinct phases: (1) a retrieval (or reactivation) phase in which a brief reminder evokes the recall of memories, rendering them labile[6] and vulnerable to modification, (2) a short interval without memory-eliciting stimuli, and (3) an extinction phase in which a lengthy exposure to cues weakens the memories' potencies by disrupting their restabilization (reconsolidation) (Lee et al., 2017).

Memory updating was first demonstrated for fearful memories a little over a decade ago (Monfils et al., 2009; Schiller et al., 2010). In rats, a brief retrieval session (presentation of a tone) followed by extinction (repeated presentations of the tone) decreased the conditioned fear response (freezing in response to the tone) (Monfils et al., 2009). The retrieval-extinction session (compared to extinction without retrieval) also reduced renewal, reinstatement and spontaneous recovery of the fear response (Monfils et al., 2009). In humans, a single 10-minute retrieval session (exposure to geometric shapes previously paired with a mild shock) followed by extinction (repeated exposure to the geometric shapes) prevented the return of the conditioned fear response (increased skin conductance in response to the shapes) up to one year later (Schiller et al., 2010). Participants who received extinction alone or extinction outside of the reconsolidation window (6 hours after retrieval) had spontaneous recovery of the conditioned response at 24-hour and one year follow-ups (Schiller et al., 2010).

[6] The period of lability, known as the reconsolidation window, is time limited (Duvarci & Nader, 2004; Nader et al., 2000). It persists for up to six hours following memory retrieval (Nader et al., 2000).

In light of these promising results, a few studies have attempted to translate memory updating to clinical populations (Germeroth et al., 2017; Telch et al., 2017; Xue et al., 2012). In the first of these studies, recently abstinent heroin-addicted inpatients exposed to two memory updating sessions with drug-related cues reported decreased craving responses during re-exposure to the same cues (cue-induced cravings) up to six months later (Xue et al., 2012). There was no change in cravings with extinction alone or with a six-hour delay between retrieval and extinction (Xue et al., 2012). A similar approach was used to target cravings and cigarette use in treatment-seeking smokers (Germeroth et al., 2017). Two memory updating sessions, each consisting of a five-minute smoking-related video (retrieval) followed by a one-hour exposure to smoking-related cues (extinction), led to decreased cue-induced cravings and cigarette use at four-week follow-up compared to no-retrieval (extinction only) controls (Germeroth et al., 2017). There were no group differences in physiological reactivity (skin conductance, heart rate and blood pressure) to smoking cues at follow-up or in time to lapse or relapse (Germeroth et al., 2017).

Not all attempts to translate findings from rodent to human models have been as successful. Aviophobes who underwent anxiety management training did not differ in self-reported fear of flying after four sessions of memory updating using virtual reality (VR) exposure to flight stimuli or four sessions of VR extinction without retrieval (Maples-Keller et al., 2017). There were, however, differences in physiological responding to a VR flight clip at three-month follow-up assessment: the memory updating group showed increased heart rate during VR while the extinction only group showed increased skin conductance (Maples-Keller et al., 2017). In arachnophobes, memory updating using VR combined with *in vivo* exposure to spiders reduced fear up to six months later (Shiban et al., 2015). No differences in phobic

responding were found between the memory updating group and the standard (no retrieval) VR treatment group (Shiban et al., 2015). It may be challenging to draw conclusions from these studies due to the additional treatments (e.g., *in vivo* exposure or anxiety management training) combined with extinction or memory updating. Participants may have imagined the treatment process prior to the study sessions (an unplanned retrieval of fear-related memories), which could have inadvertently led to memory updating in both groups (Shiban et al., 2015). Other methodological differences may also be at play, such as the strength or length of the retrieval session (Monfils & Holmes, 2018). A one-time memory updating intervention with a longer retrieval phase increased approach behavior toward a spider image in a spider-phobic population (Björkstrand et al., 2017) and reduced phobic responses to live spiders or snakes (Telch et al., 2017).

Boundary Conditions

Several factors influence the susceptibility of a memory to undergo updating. These factors, called boundary conditions, include the age and strength of the memory as well as the duration, predictability, and novelty of the retrieval procedure (Alberini & LeDoux, 2013; Milekic & Alberini, 2002; Winters et al., 2009). Older and more strongly encoded memories, such as those formed by years of repeated cue- or stress-induced cigarette use, are more resistant to destabilization (Alberini & LeDoux, 2013; Milekic & Alberini, 2002) and require stronger or longer retrieval procedures to induce their reconsolidation (Ishikawa et al., 2016; Kida, 2019; Pedreira & Maldonado, 2003; Suzuki et al., 2004). However, longer procedures (exceeding 30 minutes) may trigger extinction rather than reconsolidation (Cassini et al., 2017; Elsey et al., 2020; Pedreira & Maldonado, 2003; Suzuki et al., 2004).

Retrieval alone is not sufficient to destabilize memories; a mismatch between expected and actual outcomes must occur (Forcato et al., 2016; Lee, 2009; Sevenster et al., 2012). This mismatch, known as a prediction error, can be triggered by either the absence or difference in magnitude of an expected outcome (Lee, 2009). For instance, exposure to drug-related cues without concomitant substance use could prompt a prediction error (Das et al., 2018; Paulus et al., 2019). However, the presence of a prediction error will not reliably lead to reconsolidation; the duration of retrieval and extent of the mismatch interact to elicit either reconsolidation or extinction (Eisenberg et al., 2003; Elsey & Kindt, 2017).

Prediction errors may also modulate attention to retrieval stimuli (Exton-McGuinness et al., 2015; Fernández et al., 2016). Unexpected stimuli and outcomes draw attention, potentially facilitating memory destabilization and reconsolidation (Exton-McGuinness et al., 2015; Lee, 2009; Sinclair & Barense, 2018). In contrast, extinction may be promoted by a gradual decrease in attention to repetitive stimuli (Exton-McGuinness et al., 2015; Robbins, 1990). While useful for memory updating, novelty alone does not suffice for memory retrieval; it must occur with a prediction error to confer its benefit (Junjiao et al., 2019).

Stress and Memory Updating

Recent work has tested whether exposure to laboratory stressors can augment retrieval (Zhao et al., 2010) and reconsolidation procedures (Drexler et al., 2015). Stress can impair or enhance memory retrieval (Buchanan & Tranel, 2008; Schönfeld et al., 2014). Its effects depend on several modulating variables, including the age and content of the memory and the timing of stress administration (Goldfarb, 2019; Schönfeld et al., 2014; Schwabe & Wolf, 2014). Retrieval of drug-related and emotionally salient memories can be triggered or enhanced by stress (Ježek et al., 2010; Stringfield et al., 2017; Zhao et al., 2010). In rats, a stressful forced swim procedure

induced the retrieval of an aversive conditioned memory (foot-shock avoidance in a left-right discrimination task) (Ježek et al., 2010). Compared to saline-treated rats, suppression of the HPA axis (stress response) with dexamethasone prior to the forced swim attenuated foot-shock avoidance, confirming that stress induced memory retrieval (Ježek et al., 2010). Antagonism of beta-adrenergic receptors with propranolol immediately after the forced swim reduced conditioned avoidance, suggesting that stress rendered the memory sufficiently labile for subsequent disruption (Ježek et al., 2010).

Similarly, in rats trained to self-administer cocaine, novelty stress (exposure to a new context) reinstated cocaine-seeking following extinction (Stringfield et al., 2017). Mifepristone, a glucocorticoid antagonist, increased corticosterone concentrations, leading to further enhancement of cocaine self-administration following stress but not home cage exposure (Stringfield et al., 2017). Mifepristone potentiated stress-induced memory retrieval, suggesting that stress can destabilize drug-related memories, increasing their susceptibility to then undergo modification (Stringfield et al., 2017).

In addition to triggering retrieval, stress can also alter memory recall (Schwabe & Wolf, 2010; Zhao et al., 2010; Zhao et al., 2009). While post-retrieval stress can impair the recall of ingrained memories (Schwabe & Wolf, 2010; Zhao et al., 2009), pre-retrieval stress may enhance it (Zhao et al., 2010). In abstinent heroin users, pre-retrieval administration of cortisol or a psychosocial stressor (compared to placebo and non-stressful conditions) enhanced recall of heroin-related words but not neutral words (Zhao et al., 2010). Pre-retrieval beta-adrenergic antagonism with propranolol blocked this enhancement, indicating that stress-enhanced memory retrieval is modulated by a glucocorticoid mechanism that also necessitates beta-adrenergic stimulation (Zhao et al., 2010).

Prior stress experiences may alter later effects of stress on memory retrieval (Yang et al., 2003; Zhao et al., 2010). Pre-retrieval stress or corticosterone administration enhanced retrieval (spatial recall in a water maze task) in stress-experienced rats but impaired it in naïve (non-stress experienced) animals (Yang et al., 2003). Greater stress before or during training led to larger enhancements of retrieval by later stress administration (Yang et al., 2003). These enhancements were prevented by mifepristone administered prior to stress-based training (Yang et al., 2003). Of note, there was no effect of stress type, such that experience with one type of stress (e.g., foot-shocks) during training altered retrieval with a different stressor (e.g., elevated platform stress) (Yang et al., 2003).

Similarly, stressful experiences and concomitant cigarette use may result in enhanced retrieval of later smoking-related memories in a stressful context (Beylin & Shors, 2003; Hunt et al., 1979; Zhao et al., 2010). Glucocorticoid activation during the initial learning of stress- and cigarette use associations may be essential for these later stress-related enhancements of retrieval (Beylin & Shors, 2003; Zhao et al., 2010). Future release of glucocorticoids may reignite these associative memories and promote cigarette use (Goeders, 2003; Zhao et al., 2010).

HYPOTHESES

Given the evidence that stress can potently trigger cigarette cravings (Buchmann et al., 2008; Childs & de Wit, 2010) and use (Hughes, 2009; Nakajima et al., 2020; Twyman et al., 2014; Villanti et al., 2016), and increase the retrieval of drug-related memories (Zhao et al., 2010), we hypothesized that a stress task could augment responsivity at retrieval, leading to a more effective memory updating procedure.

To test this, we compared four different reactivation conditions as part of a single-session intervention: a stress task followed by smoking cues (combined stress and cue-based retrieval), a stress task followed by neutral cues (stress-based retrieval), a non-stressful task followed by smoking cues (cue-based retrieval), and a non-stressful task followed by neutral cues (no retrieval). Following a 10-minute period without cues or tasks, participants in all four conditions underwent an extinction phase consisting of an extended exposure to smoking-related cues.

There is evidence that early life stress has a few long-term effects that are relevant to this thesis. First, childhood maltreatment increases susceptibility to cigarette use (Maniam et al., 2014; Topitzes et al., 2010). Second, it can sensitize later stress responses, sometimes decreasing the ability to cope with future stressors (Chen & Baram, 2016; Duffy et al., 2018; Power et al., 2020). Together, these effects might influence responses to a stress-based intervention. Based on these observations, we investigated whether the intervention triggered larger psychophysiological responses in individuals with a history of childhood maltreatment. If so, it was possible that they might also experience larger stress-induced retrieval responses thereby engendering a more potent memory updating effect.

Finally, individual differences in stress responsivity have also been linked to personality traits (Lecic-Tosevski et al., 2011). We therefore tested whether personality traits influenced the

outcome of the intervention and hypothesized that memory reactivation – and the intervention's efficacy – would occur more potently in individuals who are more prone to stress.

Based on the literature described above, we made several predictions. First, compared to all other groups, the combined stress and cue memory updating procedure would induce greater craving and physiological responses at retrieval and larger decreases in cue reactivity and cigarette use at two- and six-week follow-up. Second, stress-based memory updating alone would be at least as effective as cue-based memory updating alone. Third, the combined and stress alone procedures would induce greater cravings and physiological responses in participants with a history of childhood maltreatment or heightened stress-related personality traits, such as neuroticism and anxiety sensitivity.

METHODS

Participants

Non-treatment seeking smokers were recruited through online advertisements, flyers posted around Montreal (Quebec), and word-of-mouth. Study eligibility was determined from telephone interviews using the Fagerström Test for Cigarette Dependence (FTCD) (Fagerström, 2011; Heatherton et al., 1991) and the Mini International Neuropsychiatric Interview (Bentz et al.; Sheehan et al., 1997). Primary inclusion criteria included scoring 5 or higher on the FTCD, willingness to abstain from smoking for four hours prior to each laboratory visit and being between 18 and 65 years of age. Exclusion criteria included current use of smoking cessation products, ß-blockers, antidepressant, anxiolytic or other psychotropic medications, pregnancy, and meeting diagnostic criteria for current (untreated) psychological disorders (moderate to severe psychiatric disorders (lifetime history of psychosis or current post-traumatic stress disorder, major depressive disorder, anxiety disorder or panic disorder)).

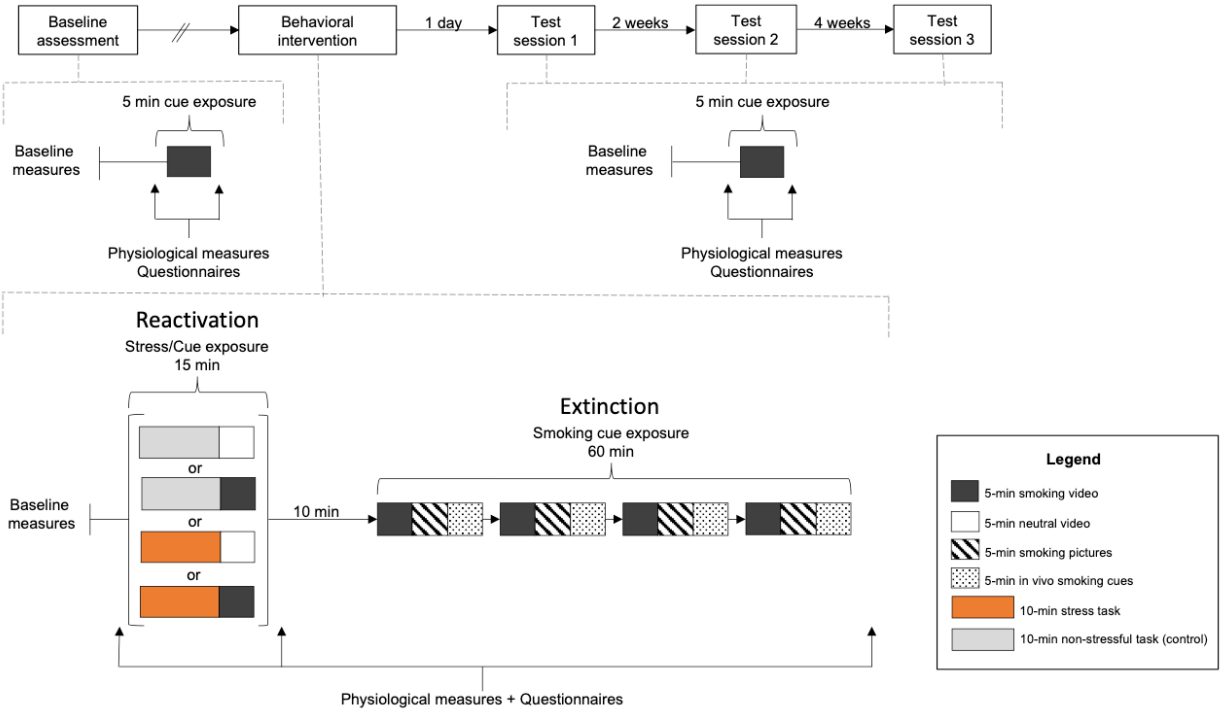
The study was carried out in accordance with the Declaration of Helsinki and approved by the McGill University Research Ethics Board. All participants were informed that study participation could affect their smoking habits and cravings prior to providing written informed consent.

Procedures

This randomized clinical trial took place between February 2019 and May 2020. The study comprised five in-person visits, including baseline, intervention, and three test sessions given 24 hours, two weeks and six weeks post-intervention (Figure 1). All visits were standardized and administered by the same experimenter.

Figure 1

Experimental Design and Timeline



Novel smoking-related videos were shown to participants during the initial assessment and test sessions 1, 2 and 3. Physiological measures (heart rate (HR) and skin conductance (SC)) were collected prior to and during the last minute of each video presentation. During these same visits, blood pressure (BP) and self-reported cigarette craving and urge to smoke scores were collected pre- and post-video. On the day of the behavioral intervention, baseline physiological measures (HR, SC, and BP) and self-report data were gathered, followed by one of four reactivation conditions: stress task and smoking cue, stress task and neutral cue, non-stressful task and smoking cue, or non-stressful task and neutral cue. Physiological and craving measures were collected and followed by a 10-minute break. All participants then went through the extinction protocol consisting of additional smoking videos, smoking images, and the manipulation of smoking-related paraphernalia. Immediately after extinction, physiological and craving measures were gathered again.

Four-hour smoking abstinence was verified at each visit by self-report and informing participants that breath carbon monoxide (CO) measures would be collected at the start of the session.

Baseline

During the first study session, participants provided demographic information, including age, sex, ethnicity, level of education, employment status, and history of cigarette use. They completed the Contemplation Ladder (Biener & Abrams, 1991) to evaluate their readiness to quit smoking and the Cigarette Withdrawal Scale (CWS-21) (Etter, 2005) to assess symptoms of withdrawal on six dimensions: craving, insomnia, depression–anxiety, appetite–weight gain, irritability–impatience, and difficulty concentrating. They then had a baseline cue reactivity assessment during which they viewed a five-minute video containing ten 30-second smoking-related clips.

Smoking-related videos were acquired from Dr. Joel Erblich's research group at Mount Sinai School of Medicine, Dr. Edythe London's research group at University of California, and YouTube. The YouTube cues were qualitatively similar to the previously validated Erblich and London lab videos (Ghahremani et al., 2018; Tong et al., 2007). Video cues were counterbalanced across the five sessions on the following criteria: number of smokers, their approximate age, sex, ethnicity, distance from camera, location, brightness, and video quality. Neutral videos were acquired from Dr. Erblich's research group and similar video clip segments from YouTube.

Smoking-related images (presented during the Intervention, see below) were acquired from the International Smoking Image Series (Gilbert & Rabinovich, 1999) and were also provided courtesy of Dr. Stephen Tiffany's research group, SUNY, University at Buffalo (Wray

et al., 2011), Dr. Charlotte Boettiger's research group at University of North Carolina at Chapel Hill (Chanon et al., 2010), Dr. Matt Field from the University of Sheffield (Mogg et al., 2005), Dr. Maartje Luitjen's research group at Radboud University (Luijten et al., 2011), and Drs. Paul Pauli and Ronald Mucha at the University of Würzburg (Geier et al., 2000; Mucha et al., 1999; Mucha et al., 2008). Pictures were categorized and counterbalanced across the four image sets on the following criteria: presence/absence of a person, their approximate age, sex, ethnicity, distance from camera, image quality and brightness.

Pre- and post-video conscious craving responses were measured using the Tobacco Craving Questionnaire – Short Form (TCQ-SF) (Heishman et al., 2008) and the Questionnaire on Smoking Urges – Brief (QSU-Brief) (Cox et al., 2001). Physiological measures, including heart rate (HR) and skin conductance (SC), were recorded for one minute pre-video and during the last minute of the five-minute video cue using the ProComp Infinity 5-channel, multi-modality encoder (Thought Technology Ltd, Montreal, Canada). Blood pressure (BP) was collected pre- and post-video cue presentation.

Intervention

Prior to the baseline session, separate blocked randomization lists were generated for males and females using an online random number generator (Sealed Envelope Ltd) to assign participants to intervention groups. Participants were randomized using a 2 x 2 factorial design to one of four retrieval conditions: stress task and smoking cue, stress task and neutral cue, control task and smoking cue, or control task and neutral cue.

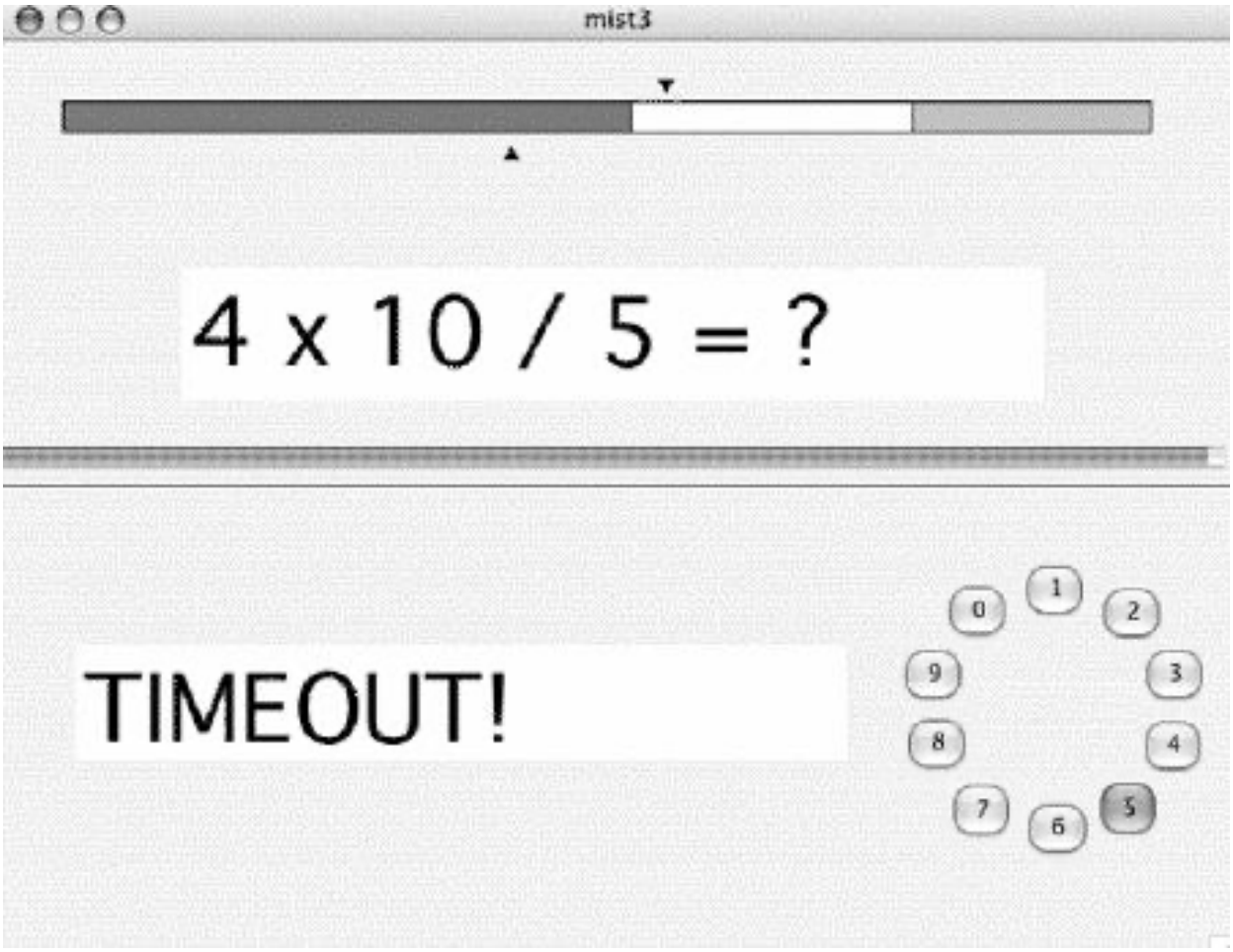
The stress task was the Montreal Imaging Stress Test (MIST) (Dedovic et al., 2005), a psychosocial challenge consisting of competitive mental arithmetic combined with negative social evaluation. During the MIST, participants were asked to solve computerized mental

arithmetic problems while receiving negative social-evaluative feedback from both the experimenter and an on-screen performance progress bar (Figure 2). The arithmetic problems consisted of timed multiplication, division, addition and subtraction tasks which, unbeknownst to participants, were adjusted to their individual performance to enforce a 55% failure rate. On the computer monitor, participants received continuous social evaluation via a performance progress bar which indicated their performance in comparison to the (fictitious) average performance of other participants. They also received negative performance commentary from the experimenter between each run of the MIST. In the non-stressful control task of the MIST, participants performed similar arithmetic problems without time constraints or social evaluation. The experimenter provided neutral feedback, unrelated to the participant's performance. All participants were exposed to three runs of the MIST (or its control version), each lasting three minutes and followed by experimenter feedback. The MIST is well-validated as a laboratory stressor, previously shown to increase blood pressure (Jones et al., 2011), heart rate (Brugnera et al., 2018; Jones et al., 2011; Voellmin et al., 2015) and cortisol responses (Jones et al., 2011; Voellmin et al., 2015). Participants were blind to group allocation and study hypotheses.

The MIST (or its control version) was immediately followed by a five-minute video cue presentation. For participants in the smoking cue condition, these video clips were similar but non-identical to those presented at baseline. For participants in the neutral cue condition, cues consisted of ten 30-second clips depicting non-smoking activities (see Baseline Methods). Neutral and smoking cues were matched on number of people in each clip, their approximate age, ethnicity, distance from the camera, and lighting.

Figure 2

Graphical User Interface of the Montreal Imaging Stress Task (MIST)



The MIST user interface includes performance indicators at the top (top arrow: (fictitious) average performance of other participants, bottom arrow: individual participant's performance), a progress bar reflecting the imposed time limit in the middle, and a text field for feedback and rotary dial for response submission at the bottom of the screen.

All four reactivation conditions were followed by a 10-minute break during which participants remained seated in front of a black screen. They then underwent a 60-minute extinction protocol. This entailed four rotations of: a five-minute video with smoking-related content (composed of similar but non-identical clips to those presented in the baseline session), a five-minute presentation of smoking images (with each image presented for three seconds, see Baseline Methods), and five minutes of manipulating smoking paraphernalia (e.g., lighter, cigarettes).

Immediately prior to and after reactivation, and immediately after extinction, HR, SC and BP were measured, and participants completed the TCQ-SF and QSU-Brief. Participants remained in the laboratory for one hour following completion of the questionnaires to minimize the chance that they would reengage the association between cigarette use and smoking-related cues. Participants who underwent the stressful MIST were debriefed. All participants were asked to see how long they could go without smoking after the session.

Test Sessions 1, 2 and 3

Participants returned to the laboratory for cue reactivity test sessions 24 hours, two weeks, and six weeks following the intervention. At the beginning of each of these sessions, SC, HR and BP were measured, and the CWS-21, Contemplation Ladder, FTCD (for tests 2 and 3), TCQ-SF and QSU-Brief were administered. Participants then viewed a new five-minute smoking cue video with SC and HR measured for one minute before video presentation and again during the last minute of the video. Immediately after the video, BP was measured and the TCQ-SF and QSU-Brief were administered again.

Between each of the test sessions, participants were asked to record their daily cigarette use in a journal provided by the experimenter. Data from the journals were collected at test sessions 2 and 3.

Telephone Follow-up

The Childhood Trauma Questionnaire (CTQ) was administered in a follow-up telephone interview. Prior studies indicate that childhood adversity increases adult cigarette use (Spratt et al., 2009; Topitzes et al., 2010) and physiological stress responses (Carpenter et al., 2011; Kuras et al., 2017). The CTQ was added to test whether early life adversity influences smoking behavior.

Statistical Analyses

Sample Size Estimation

Based on anticipated medium effect sizes between 0.4 and 0.5, sample sizes of 60 to 90 would yield power of 80%, 0.05 alpha error probability. Since power analyses tend to be conservative, we planned to test up to 80 participants. A first data check at N = 62 confirmed that our objectives had been met.

Study Variables

SPSS 26.0.0.1 (Chicago, IL) was used for all statistical analyses. Preliminary analyses indicated less than 1% missing data on all variables. Multiple imputation was used to impute missing scores. All variables were verified for normality and outliers prior to analysis. Chi square tests and analyses of variance (ANOVAs) were used to examine group differences in study characteristics at baseline for categorical and continuous variables, respectively. Daily cigarette use at test sessions 2 and 3 was calculated as the mean number of cigarettes smoked per day in the week prior to each test session. Repeated measures analyses of variance were used to

examine group differences in physiological and craving measures, daily cigarette use, Contemplation Ladder and FTCD scores. Partial eta squared were used to assess the magnitude of these effects. Greenhouse-Geisser corrections were applied when the assumption of sphericity was violated. Post-hoc analyses consisted of paired samples t-tests with Bonferroni corrections. Correlational analyses tested for potential predictors of smoking behavior changes, and the risk of false positives was decreased by using a conservative Benjamini-Hochberg procedure with a false discovery rate of 5%.

RESULTS

Study Overview

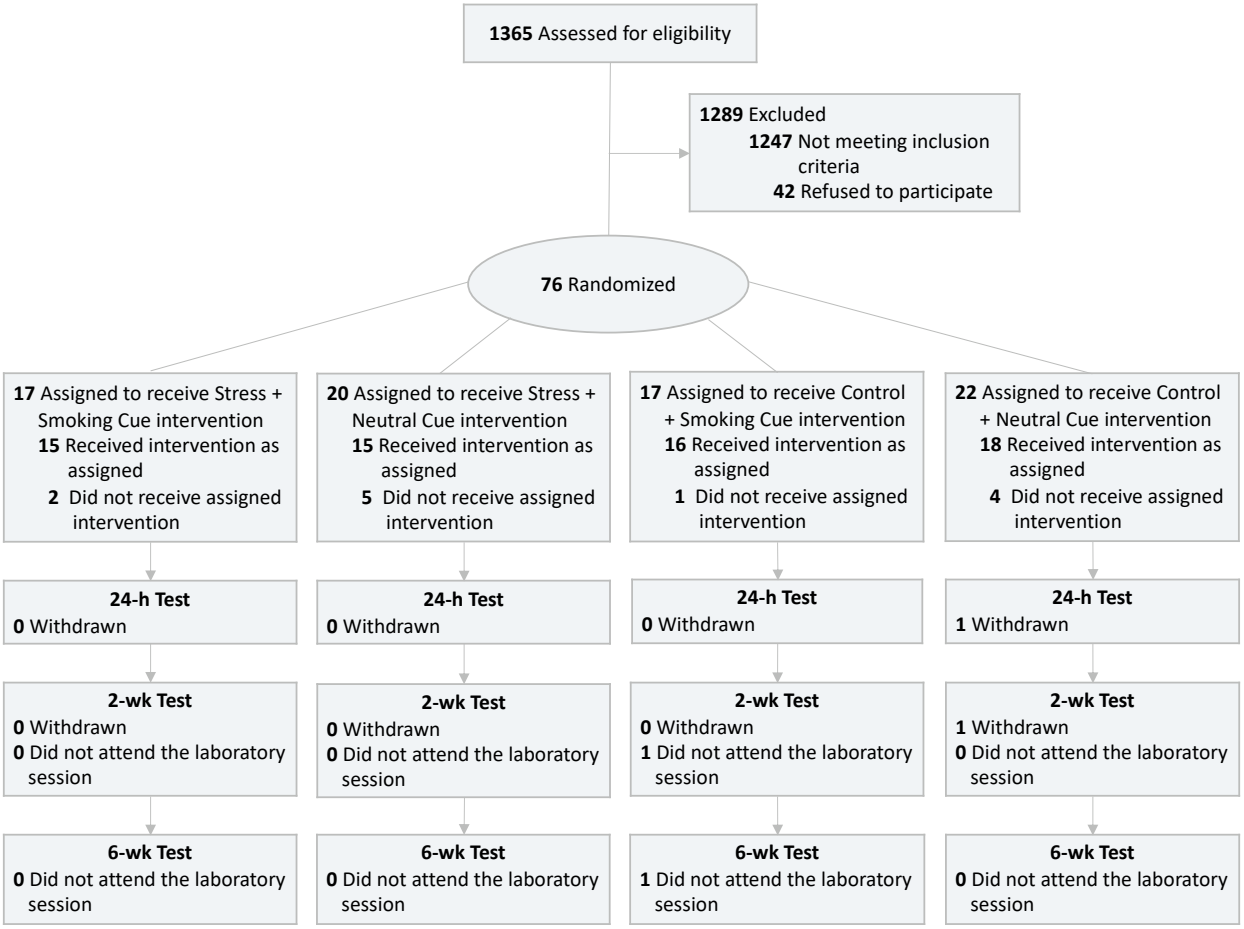
This study was a randomized controlled trial following CONSORT guidelines (Figure 3). A total of 1,365 people were screened, 926 were excluded based on not meeting preliminary entry criteria, and 321 proceeded to the psychiatric assessment. Of those who completed the assessment, 203 met criteria for a moderate to severe psychiatric disorder, 42 declined to participate in the study, and 76 were deemed eligible and agreed to participate. Overall, a total of 507 individual study sessions were performed, including 328 laboratory visits and 179 telephone follow-ups and COVID-related interviews. The laboratory sessions were exclusively retained for analysis while the telephone follow-ups (with the exception of CTQ data) were omitted due to potential confounds (e.g., onset of COVID-19 and related changes in daily habits).

Participant Characteristics

Seventy-six volunteers were deemed eligible for the study (Figure 3). Of these, 14 withdrew or were withdrawn. Reasons for withdrawal included dyscalculia, scheduling conflicts, and not meeting study requirements. Of the remaining 62 participants, one did not attend test session 2 for medical reasons and another missed test session 3 due to relocating to another province. When the participants were randomly assigned to the subgroups, there were no significant differences in tobacco use (16.9±5.9 cigarettes per day) or other demographic or clinical characteristics (Table 1).

Figure 3

CONSORT Flow Diagram



The level of cigarette use and dependence (on the FTCD) of many volunteers did not meet minimum inclusion criteria, accounting for the large number of volunteers excluded. Reasons for withdrawal after randomization included reduction or cessation in cigarette use prior to the intervention, misreporting cigarette use, and a participant having dyscalculia. Scheduling conflicts or time constraints were the primary reasons why some participants declined to participate.

Table 1

Demographic and Clinical Characteristics of Intervention Groups[a]

Characteristic	Participants, No. (%) / GROUP	CONTROL		STRESS		Statistic	*P* value
	All participants (n = 62)	Control Stressor + Neutral Cues (n = 16)	Control Stressor + Smoking Cues (n = 16)	Stressful Task + Neutral Cues (n = 15)	Stressful Task + Smoking Cues (n = 15)		
Age, mean (SD)	**35.82 (12.99)**	38.56 (13.45)	38.25 (14.85)	31.47 (1.92)	34.67 (12.18)	$F = 1.03$	.39
Sex							
Male	**34 (54.84)**	9 (56.25)	9 (56.25)	8 (53.33)	8 (53.33)	$\chi2 = .05$	1.00
Female	**28 (45.16)**	7 (43.75)	7 (43.75)	7 (46.67)	7 (46.67)		
Ethnicity							
White	**45 (72.58)**	13 (81.25)	10 (62.50)	12 (8.00)	10 (66.67)	$\chi2 = 2.10$	.55
Other[b]	**17 (27.42)**	3 (18.75)	6 (37.50)	3 (2.00)	5 (33.33)		
Employed							
Yes	**39 (62.90)**	10 (62.50)	8 (5.00)	11 (73.33)	10 (66.67)	$\chi2 = 1.93$	.59
No	**23 (37.10)**	6 (37.50)	8 (5.00)	4 (26.67)	5 (33.33)		
Education							
No HS completion	**2 (3.23)**	1 (6.25)	1 (6.25)	0 (0)	0 (0)	$\chi2 = 2.94$	.97
HS graduate	**9 (14.52)**	2 (12.50)	2 (12.50)	3 (2.00)	2 (13.33)		
College or trade school graduate	**21 (33.87)**	6 (37.50)	6 (37.50)	4 (26.67)	5 (33.33)		
University graduate	**30 (48.39)**	7 (43.75)	7 (43.75)	8 (53.33)	8 (53.33)		
Annual household income, $							
≤ 20 000	**21 (33.87)**	3 (18.75)	6 (37.50)	8 (53.33)	4 (26.67)	$\chi2 = 4.61$	.20
> 20 000	**41 (66.13)**	13 (81.25)	10 (62.50)	7 (46.67)	11 (73.33)		
No. of cigarettes smoked per day, mean (SD)	**16.87 (5.87)**	16.84 (6.17)	15.34 (5.71)	18.27 (6.64)	17.13 (5.07)	$F = .64$	.59
CO level (ppm), mean (SD)	**8.60 (5.72)**	11.25 (5.92)	6.75 (5.47)	7.67 (7.08)	8.67 (3.18)	$F = 1.92$	.14
FTCD score, mean (SD)	**6.05 (1.19)**	5.81 (1.05)	6.13 (1.02)	6.00 (1.36)	6.27 (1.39)	$F = .39$	.76
Contemplation Ladder score, mean (SD)	**4.97 (1.59)**	4.75 (1.34)	5.13 (1.78)	4.87 (1.55)	5.13 (1.77)	$F = .22$	.88
Age at first cigarette smoked, mean (SD)	**16.08 (4.14)**	15.91 (2.95)	16.56 (3.31)	14.87 (3.74)	16.97 (6.08)	$F = .73$	.54
Years of smoking, mean (SD)	**19.74 (14.07)**	22.66 (15.06)	21.69 (15.43)	16.60 (12.86)	17.70 (13.04)	$F = .67$	.57
No. of MINI diagnoses							
0	**52 (83.9)**	14 (87.50)	14 (87.50)	11 (73.33)	13 (86.67)	$\chi2 = 1.63$	.65
1	**10 (16.1)**	2 (12.50)	2 (12.50)	4 (26.67)	2 (13.33)		

CO, carbon monoxide; FTCD, Fagerström Test for Cigarette Dependence; HS, high school; MINI, Mini International Neuropsychiatric Interview. [a] All measures were collected at baseline. [b] Other self-reported ethnicities included African American, Asian, Hispanic/Latinx, Indian, Indigenous, Middle Eastern, and other.

Experimental Manipulation Check

Since this was the first time that stress and smoking cues were combined in a memory updating procedure, we began by testing whether they elicited their expected effects during the reactivation phase of the intervention session. As hypothesized, the stress task yielded the predicted effects within the smoking cue groups (*p*s < .05 for SC, systolic BP and QSU-Brief scores) and the neutral cue groups (*p*s < .05 for TCQ-SF and QSU-Brief scores). In contrast, the smoking cues, administered immediately after the stress and non-stress tasks, did not change either craving or physiological responses, not within the stress task groups (all *p*s > .30) or the non-stressful task groups (all *p*s > .10), nor were there any stress by cue interactions (all *p*s > .30). Based on these observations and an absence of cue effects on cigarette use at follow-up (all *p*s > .25), subsequent analyses combined the two stress subgroups (stress) and two non-stressful subgroups (controls), thus increasing statistical power to detect effects of stress.

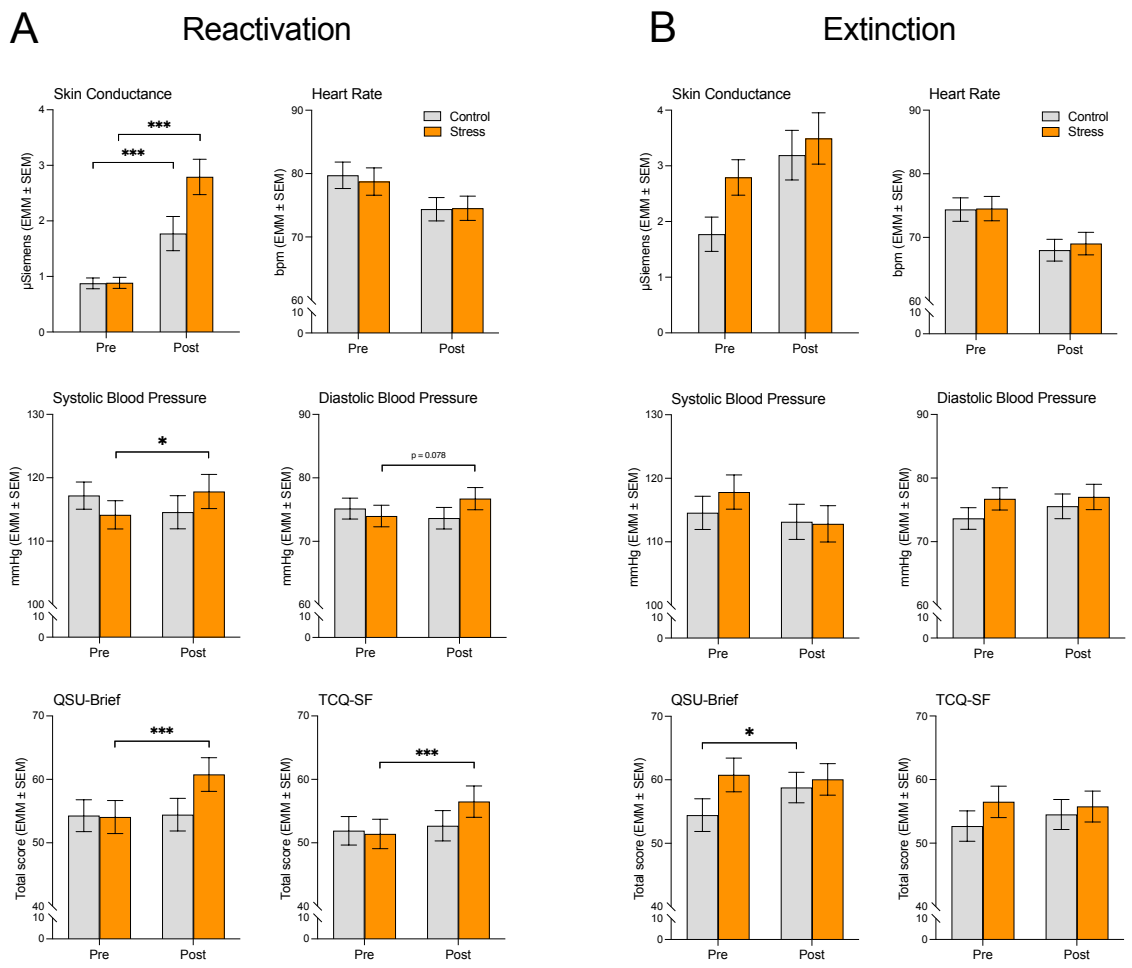
Assessment of Craving and Psychophysiological Responses

Responses to the Intervention

Reactivation Phase. When comparing scores from immediately before and after the reactivation phase, there were main effects of time reflecting anticipated decreases in HR ($F(1,60) = 61.61$, $p < .001$) and increases in SC ($F(1,60) = 63.04$, $p < .001$) and craving (QSU-Brief: $F(1,60) = 14.90$, $p < .001$; TCQ-SF: $F(1,60) = 14.32$, $p < .001$). The increases in physiological and craving scores were larger in the stress group, compared to the controls (Figure 4A), as reflected by stress condition by time interactions for SC ($F(1,60) = 8.22$, $p = .006$), BP (systolic: $F(1,60) = 8.65$, $p = .005$; diastolic: $F(1,60) = 5.52$, $p = .022$), and craving (QSU-Brief: $F(1,60) = 13.58$, $p < .001$; TCQ-SF: $F(1,60) = 7.72$, $p = .007$). There were no group differences for HR.

Figure 4

The Stress Task Induced Psychophysiological and Craving Responses During the Intervention



During the reactivation phase (**A**), participants exposed to the stress task exhibited larger increases in physiological (SC and systolic BP) and craving measures (QSU-Brief and TCQ-SF scores). During the extinction phase (**B**), physiological measures did not significantly differ between groups, but participants previously exposed to the control task exhibited greater smoking cue-induced craving responses (QSU-Brief). BP: blood pressure; SC: skin conductance; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief. EMM: estimated marginal mean; SEM: standard error of the mean. * $p < .05$, *** $p < .001$.

Extinction Phase. Across groups, the extinction procedure significantly increased SC ($F(1,60) = 29.60$, $p < .001$) and decreased both HR ($F(1,60) = 68.94$, $p < .001$) and systolic BP ($F(1,60) = 5.85$, $p = .019$). The stress and control groups were not significantly different on most measures but there was a stress condition by time interaction for QSU-Brief ($F(1,60) = 5.03$, $p = .029$) reflecting a significant increase in craving during the extinction phase only in the control group ($p = .014$) (Figure 4B).

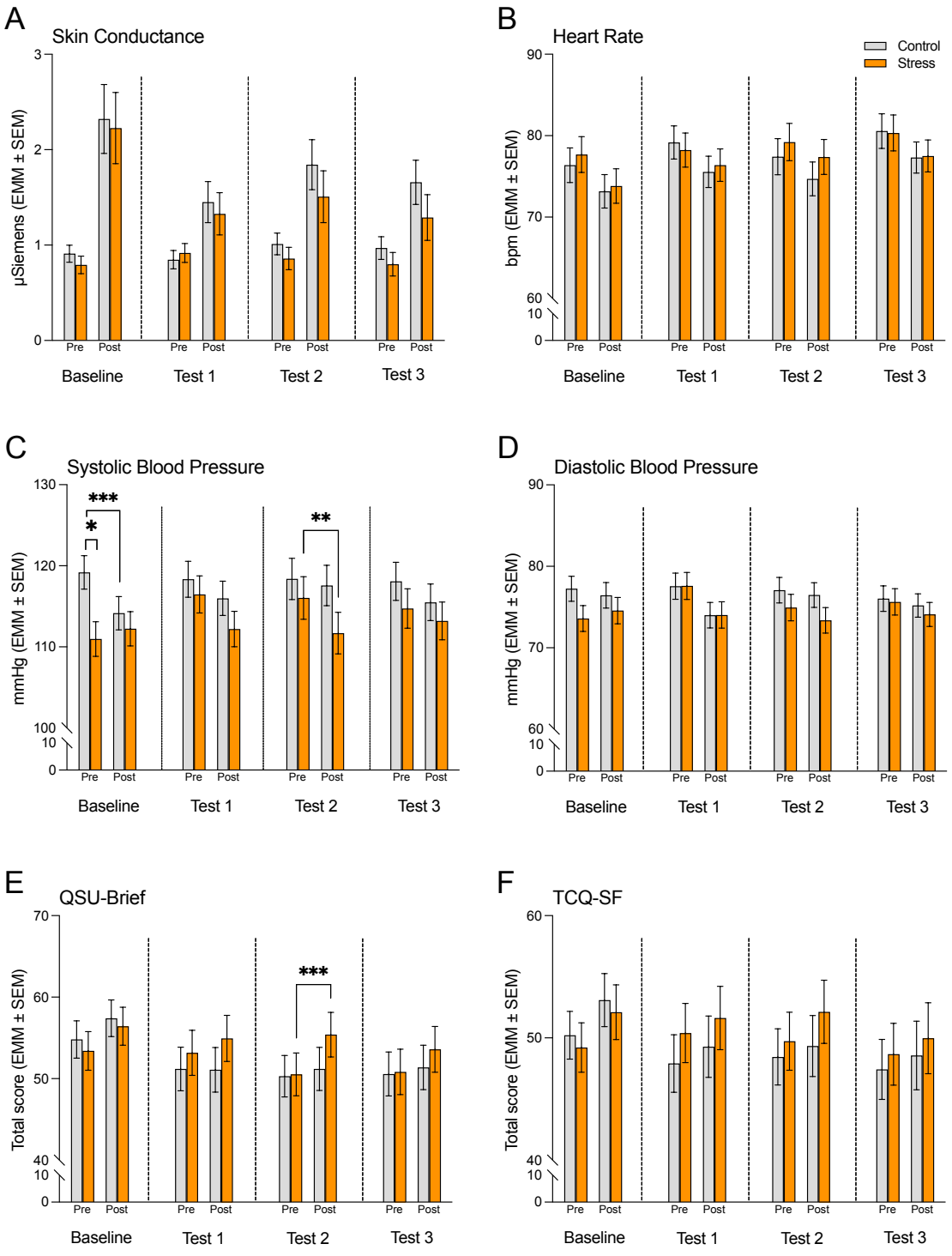
Cue Reactivity Assessments

Baseline. As expected, exposure to a smoking-related video led to a main effect of time revealing significant increases in craving (QSU-Brief ($F(1,60) = 15.01$, $p < .001$; TCQ-SF: $F(1,60) = 13.96$, $p < .001$) and SC responses ($F(1,60) = 46.67$, $p < .001$) and decreases in HR ($F(1,60) = 39.11$, $p < .001$) and systolic BP responses ($F(1,60) = 4.20$, $p = .045$). There was a single stress condition by time interaction (systolic BP: $F(1,60) = 11.76$, $p = .001$; Figure 5C), which was likely spurious given that groups were not yet treated differently. This interaction was controlled for in subsequent analyses.

Test Sessions. Participants underwent test sessions administered 24 hours, two weeks and six weeks after the intervention. At each test session, exposure to a new smoking-related video led to increased SC response (all ps $< .001$) and decreased HR (all $ps < .002$) across groups. Increases in craving were seen at test sessions 1 (TCQ-SF: $p < .05$) and 2 (QSU-Brief: $p < .001$) but not at test session 3. Details on each test session are provided below.

Figure 5

Cue Reactivity Assessments



There were no differences between groups in terms of cue reactivity (**A**, **B**, **D**, **F**) with the exception of baseline differences in systolic BP (controlled for at test) and changes in systolic BP (**C**) and QSU-Brief (**E**) responsivity at test 2 in the stress group. BP: blood pressure; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief; bpm: beats per minute; mmHg: millimetre of mercury. EMM: estimated marginal mean; SEM: standard error of the mean. ** $p < .01$, ** $p < .01$, *** $p < .001$.

Test 1 (24 hours post-intervention). Across groups, exposure to a new smoking-related video led to significantly increased craving (TCQ-SF: $F(1,60) = 5.88$, $p = .018$; but not QSU-Brief: $p > .05$) and SC responses ($F(1,60) = 2.47$, $p < .001$) and decreased HR ($F(1,60) = 24.02$, $p < .001$) and BP responses (systolic: $F(1,59) = 7.71$, $p = .007$; diastolic: $F(1,60) = 33.99$, $p < .001$). There were no group differences in craving or physiological responses to the video at this session (Figure 5).

Test 2 (2 weeks post-intervention). Across groups, exposure to a new smoking video led again to significantly increased craving (QSU-Brief: $F(1,60) = 12.25$, $p < .001$; but not TCQ-SF: $p > .05$) and SC responses ($F(1,60) = 32.58$, $p < .001$) and decreased HR ($F(1,60) = 1.67$, $p = .002$) and BP responses (systolic BP: $F(1,59) = 4.54$, $p = .037$; but not diastolic BP: $F(1,60) = 1.61$, $p > .05$). For QSU-Brief scores, there was an interaction of stress condition by time ($F(1,60) = 5.82$, $p = .019$; Figure 5E) driven by an increase in craving from pre- to post-video in stress group participants ($p < .001$) but not non-stress group participants ($p > .80$). An interaction of stress condition by time also emerged for systolic BP responses when controlling for baseline differences ($F(1,59) = 5.54$, $p = .022$; Figure 5C), revealing decreases in BP in the stress group ($p < .002$) but not in controls ($p > .90$).

Test 3 (6 weeks post-intervention). Across groups, exposure to a new smoking video led to significantly increased SC ($F(1,60) = 27.21$, $p < .001$) and decreased HR ($F(1,60) = 35.53$, $p < .001$). There were no main effects on other measures and no group differences in craving or physiological responses (Figure 5).

Across Baseline and Test Sessions. Across sessions, physiological responses to the video cues became less pronounced as shown by decreases in SC (($F(3,180) = 5.52$, $p = .001$) from baseline to test 1 ($p = .008$) and baseline to test 3 ($p = .031$)) and increases in HR

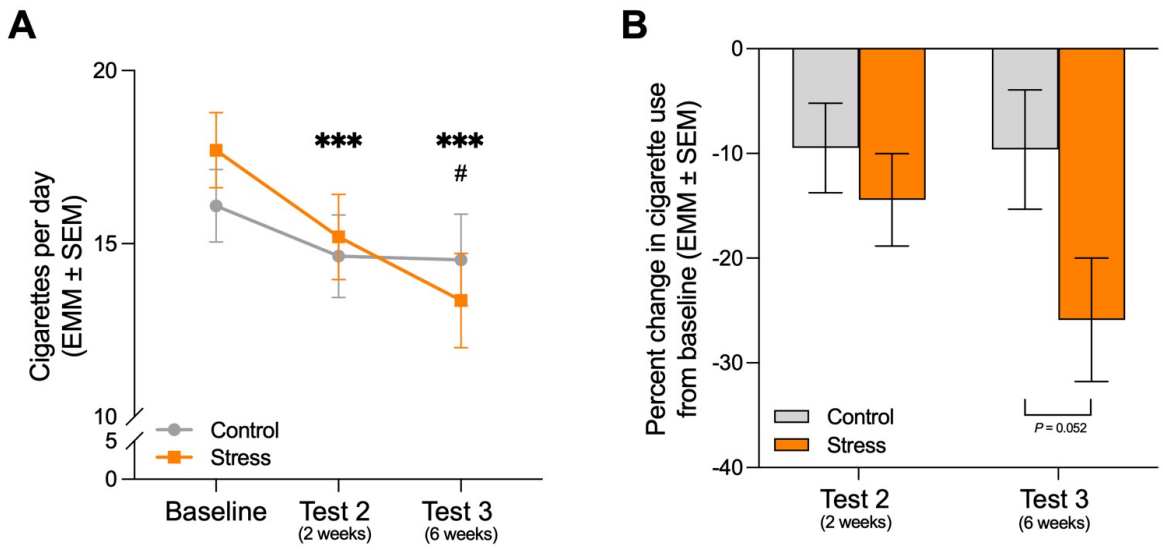
((*F*(3,180) = 2.90, *p* = .036) from baseline to test 3 (*p* = .033)). There was an interaction of session by time for SC (*F*(2.23,133.94) = 13.66, *p* < .001) reflecting decreases in post-video SC from baseline to tests 1 (*p* < .001), 2 (*p* = .028) and 3 (*p* = .005). While there was no main effect on BP across sessions, there was a stress condition by time interaction (systolic BP: *F*(1,177) = 4.77, *p* = .033, $\eta_p^2 = 0.075$) driven by within-session decreases in response to the smoking videos in the stress (*p* < .001) and control (*p* = .034) groups (Figure 5C). A time by session interaction was detected for diastolic BP (*F*(3,180) = 5.38, *p* = .001), reflecting a significant decrease in responses to the smoking videos at test 1 (*p* < .001). No across session changes or group differences in craving were observed.

Smoking Behavior

Over the period of the study, participants decreased their cigarette use as reflected by a significant main effect of session (*F*(1.66,99.35) = 16.58, *p* < .001). Stress condition by session interactions were observed for baseline *vs*. test sessions 2 and 3 (*F*(1.66,99.35) = 3.60, *p* = .039, $\eta_p^2 = 0.057$) and baseline *vs.* test session 3 alone (*F*(1,60) = 4.86, *p* = .031, $\eta_p^2 = 0.075$) (Figure 6A). These interactions reflected significant decreases in cigarette smoking in the stress group participants from baseline to test session 2 (*p* = .001) and baseline to test session 3 (*p* < .001) but not in the control group participants (all *p*s > .05). This represented decreases of 14% and 26% in the stress group compared to 9% and 10% in the controls at test sessions 2 (*F*(1,62) = .67, *p* = .42) and 3 (*F*(1,62) = 3.94, *p* = .052), respectively (Figure 6B). The magnitude of the intervention's effect increased over time such that, in the stress group, cigarettes smoked per day decreased from baseline and were lower at test session 3 compared to test 2 (*p* = .02). No significant changes in cigarette use were found in the controls.

Figure 6

The Stress-Based Intervention Led to Decreased Cigarette Use at Follow-Up



(**A**) Participants in the stress-based intervention significantly decreased their average daily cigarette use from baseline to test sessions 2 and 3 (two and six weeks after the intervention, respectively). (**B**) Cigarette use in the stress group participants decreased by 14% at two weeks (test 2) and by 26% at six weeks (test 3), while the control group showed decreases of 9% and 10%, respectively. EMM: estimated marginal mean; SEM: standard error of the mean. In the stress group: *** $p \leq .001$ compared to baseline, # $p < .05$ compared to test 2.

Across study visits, there were progressive decreases in cigarette dependence (FTCD: $F(2,120) = 1.94$, $p < .001$) and withdrawal symptoms due to craving (CWS-21 craving subscale: $F(3,180) = 8.05$, $p < .001$), and increased motivation to quit smoking scores (Contemplation Ladder: $F(2.46,147.93) = 9.86$, $p < .001$) (Table 2). There were no significant group differences in these measures and no main effects of session on other CWS-21 subscales.

Predictors of Decreased Cigarette Use

When participants from all groups were combined, higher systolic BP values following the reactivation phase predicted larger decreases in cigarette use at test session 3 ($r = -.27$, $p = .033$) (Figure 7A). Furthermore, larger decreases in systolic BP during the extinction phase predicted greater decreases in cigarette use at test session 2 ($r = .28$, $p = .03$; Figure 7B) and test session 3 ($r = .38$, $p = .002$; Figure 7C). This latter result survived the Benjamini-Hochberg correction.

Table 2

Mean Cigarette Dependence, Withdrawal Symptoms and Motivation to Quit Smoking Scores Across Sessions

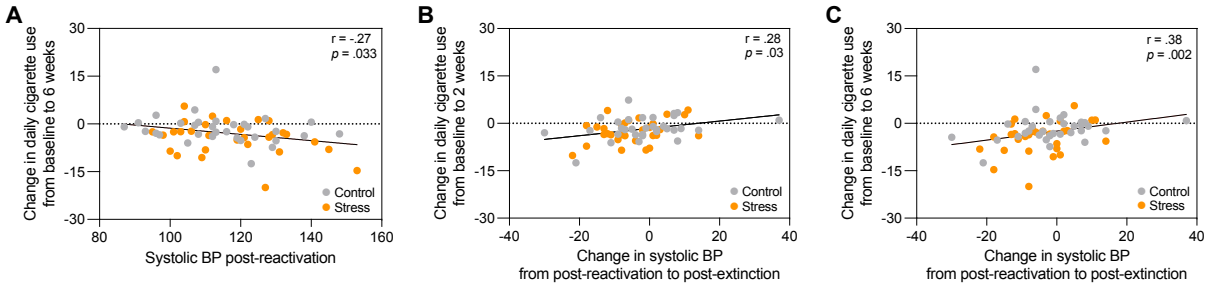
		Session				Statistics	
Measure	**Group**	**Baseline**	**Test 1**	**Test 2**	**Test 3**	**Across Sessions (*F* value)**	**Stress Condition x Session (*F* value)**
FTCD score	Control	5.97 (.21)	-	5.56 (.33)	5.18 (.37)	1.77***	.45
	Stress	6.13 (.22)	-	5.53 (.35)	4.93 (.38)		
Contemplation Ladder score	Control	4.94 (.29)	5.28 (.28)	5.75 (.29)	6.03 (.27)	9.98***	.92
	Stress	5.00 (.30)	5.30 (.29)	5.40 (.30)	5.67 (.28)		
CWS-21 total	Control	3.12 (.18)	2.65 (.17)	2.48 (.19)	2.42 (.17)	7.70***	.84
	Stress	3.02 (.18)	2.88 (.17)	2.49 (.19)	2.68 (.17)		
Depression-anxiety	Control	1.49 (.11)	1.60 (.14)	1.50 (.14)	1.56 (.13)	.17	.28
	Stress	1.83 (.11)	1.86 (.14)	1.90 (.14)	1.80 (.13)		
Irritability-impatience	Control	1.69 (.14)	1.73 (.14)	1.68 (.17)	1.78 (.16)	.44	.60
	Stress	2.23 (.14)	1.98 (.14)	2.06 (.17)	2.09 (.16)		
Difficulty concentrating	Control	1.79 (.19)	1.94 (.20)	1.91 (.21)	1.88 (.21)	.38	.44
	Stress	2.28 (.19)	2.33 (.20)	2.12 (.21)	2.18 (.21)		
Insomnia	Control	1.97 (.18)	2.18 (.21)	2.16 (.18)	2.24 (.20)	.23	1.30
	Stress	2.57 (.18)	2.56 (.21)	2.52 (.18)	2.34 (.20)		
Appetite-weight gain	Control	1.97 (.15)	2.18 (.18)	2.01 (.18)	2.23 (.23)	.86	.66
	Stress	2.06 (.15)	1.91 (.18)	1.78 (.18)	2.01 (.23)		
Craving	Control	3.11 (.18)	2.66 (.17)	2.49 (.20)	2.43 (.17)	7.52***	.76
	Stress	3.02 (.18)	2.88 (.17)	2.50 (.19)	2.68 (.16)		

CWS-21: Cigarette Withdrawal Scale – 21; FTCD: Fagerström Test for Cigarette Dependence.

*** $p < .001$.

Figure 7

Systolic BP Changes During the Intervention Correlated with Decreased Cigarette Use



Larger decreases in daily cigarette use at follow-up were associated with (**A**) higher systolic BP post-retrieval and (**B, C**) larger changes in systolic BP during the extinction phase across all groups. BP: blood pressure.

Role of Personality Characteristics

Personality traits have been linked to cigarette use and motivation to quit. The Substance Use Risk Profile Scale (SURPS) and International Personality Item Pool (IPIP) were administered at baseline to assess different personality traits.

Group Differences

The stress and control groups did not differ on any of the SURPS subscales (anxiety sensitivity, hopelessness, impulsivity and sensation seeking) (Figure 8A) or most of the IPIP subscales (extraversion, agreeableness, conscientiousness and openness) (all *p*s > .05). The only exception was the IPIP neuroticism scale in which the stress group was significantly higher than the control group ($p < .001$) (Figure 8B). These group differences did not affect the craving and physiological responses to the reactivation or extinction procedures or the changes in cigarette use over the course of the study.

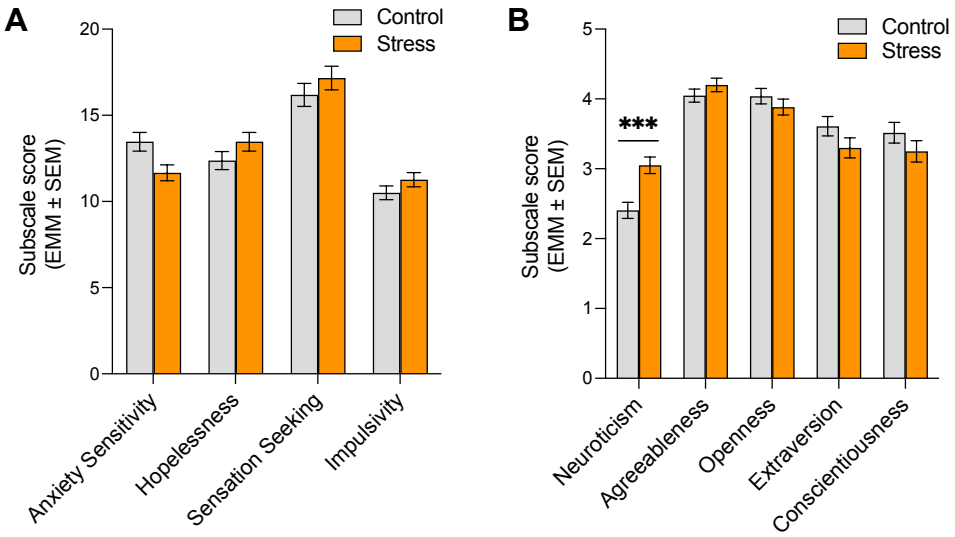
Baseline Assessments

Smoking Behavior.

SURPS. Prior to the intervention, participants with higher SURPS hopelessness scores smoked more cigarettes ($r = .30$, $p = .017$) and reported greater cigarette dependence (FTCD: $r = .28$, $p = .031$) (Table 3). They also indicated heightened withdrawal symptoms (CWS-21 total: $r = .45$, $p < .001$, survived the Benjamini-Hochberg correction), marked by depression and anxiety ($r = .48$, $p < .001$, survived the Benjamini-Hochberg correction), irritability and impatience ($r = .40$, $p = .001$), difficulty concentrating ($r = .41$, $p = .001$), insomnia ($r = .36$, $p = .004$), and increased appetite or weight gain ($r = .29$, $p = .023$).

Figure 8

Personality Traits of Participants in the Control and Stress Groups



The groups did not differ on SURPS personality traits (**A**) but were significantly different in the IPIP personality trait of Neuroticism (**B**). IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale. EMM: estimated marginal mean; SEM: standard error of the mean. *** $p < .001$.

Table 3

Correlations Between Cigarette Use Characteristics and Personality Traits

	SURPS				IPIP				
Measure	Hopelessness	Anxiety Sensitivity	Impulsivity	Sensation Seeking	Extraversion	Agreeableness	Openness	Conscientiousness	Neuroticism
No. of cigarettes smoked per day	.30*	.18	.07	-.19	-.02	-.004	-.24	-.01	.03
FTCD score	.28*	.14	-.04	-.16	.06	.02	-.09	-.08	.09
Age of first cigarette smoked	-.15	-.03	-.16	.11	.05	-.02	.03	.17	-.08
Years of smoking	.04	.03	.13	-.20	-.01	-.28	-.07	.10	-.10
Contemplation Ladder score	.52	.76	.98	.27	.14	.95	.93	.80	.75
CWS-21 total	.45**ψ	.35**	.01	-.13	-.19	.13	-.11	-.39**	.33**
Depression-anxiety	.48**ψ	.32*	-.04	.02	-.28*	.02	.19	-.33**	.33**
Irritability-impatience	.40**	.25	.07	-.18	-.10	-.002	-.11	-.35**	.46**ψ
Difficulty concentrating	.41**	.36**	.03	-.11	-.26*	.16	-.01	-.37**	.41**
Insomnia	.36**	.28*	.003	-.10	-.21	-.03	-.16	-.21	.10
Appetite-weight gain	.29*	.26*	.08	-.15	.107	-.19	-.25	-.13	-.07
Craving	.09	-.01	-.07	-.11	.129	.41**	-.10	-.15	-.03

CWS-21: Cigarette Withdrawal Scale-21; FTCD: Fagerström Test for Cigarette Dependence; IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale. * $p < .05$, ** $p < .01$. ψ Survived the Benjamini-Hochberg correction.

Similarly, anxiety sensitive participants tended to report more withdrawal symptoms (CWS-21 total: $r = .34$, $p = .007$), including depression and anxiety ($r = .32$, $p = .01$), difficulty concentrating ($r = .36$, $p = .004$) and insomnia ($r = .26$, $p = .043$). There were however no associations between anxiety sensitivity and smoking behavior nor were there other correlations of baseline smoking behavior, motivation to quit (Contemplation Ladder scores), or history of smoking behavior (i.e., age of onset, years of cigarette use) with any SURPS personality traits.

IPIP. When abstaining from cigarette use, participants higher in IPIP neuroticism reported heightened withdrawal symptoms (CWS-21 total: $r = .33$, $p = .01$), particularly depression and anxiety ($r = .33$, $p = .009$), irritability and impatience ($r = .46$, $p < .001$, survived the Benjamini-Hochberg correction), and difficulty concentrating ($r = .41$, $p = .001$) (Table 3). Conversely, during withdrawal, more extraverted participants reported fewer symptoms of anxiety and depression ($r = -.28$, $p = .025$) and less difficulty concentrating ($r = - .26$, $p = .043$) than their introverted counterparts. Participants higher in IPIP agreeableness scores had greater craving due to withdrawal (CWS-21: $r = .41$, $p = .001$) while those higher in IPIP conscientiousness had fewer withdrawal symptoms overall (CWS-21 total: $r = -.38$, $p = .002$; depression-anxiety: $r = -.33$, $p = .01$; irritability-impatience: $r = -.35$, $p = .006$; difficulty concentrating: $r = -.37$, $p = .004$).

IPIP personality traits were not associated with differences in motivation to quit smoking (Contemplation Ladder scores) or history of smoking behavior (i.e., age of onset, years of cigarette use). There were no other correlations of baseline smoking behavior with IPIP subscale scores.

Cue Reactivity.

SURPS. At baseline, greater cravings (on the QSU-Brief) in response to the smoking video were reported by participants higher in hopelessness (r = .25, p = .05) and anxiety sensitivity (r = .26, p = .045) (Table 4). There were no associations with other baseline craving and physiological responses (all ps > .08) or with the personality traits of impulsivity or sensation seeking (all ps > .07).

IPIP. More agreeable participants experienced greater increases in systolic BP in response to the video cue (r = .33, p = .008). No significant associations were observed with other craving and physiological responses at baseline (all ps > .15) or with the personality traits of conscientiousness, openness to experience, neuroticism and extraversion (all ps > .08).

Responses to the Intervention

SURPS. Hopelessness was associated with increased physiological responses at reactivation (HR: r = .27, p = .031; diastolic BP: r = .28, p = .031) and decreased responses at extinction (diastolic BP: r = -.42, p < .001) (Tables 5 and 6). Similarly, impulsivity was associated with decreased SC over the extinction phase (r = -.29, p = .024). These findings were independent of stress condition. No other craving or physiological responses were associated with these or other personality traits at reactivation (ps > .13) or extinction (ps > .06).

IPIP. For the most part, personality traits on the IPIP did not influence responses at reactivation (ps > .06) or extinction (all ps > .05) with the exception of openness to experience which was associated with SC (r = .34, p = .007) and craving responses (TCQ-SF: r = .26, p = .041) at reactivation, independent of stress condition.

Table 4

Correlations Between Baseline Cue Reactivity Measures and Personality Traits

	SURPS				IPIP				
Change in measure	Hopelessness	Anxiety Sensitivity	Impulsivity	Sensation Seeking	Extraversion	Agreeableness	Openness	Conscientiousness	Neuroticism
Heart rate	.13	-.07	-.10	-.01	.11	.09	-.14	-.06	-.13
Skin conductance	.05	< .001	-.19	.23	.09	.03	.20	.13	.04
Systolic BP	-.15	.03	.09	-.01	-.04	.33**	.14	.09	.15
Diastolic BP	-.22	-.05	.01	.13	.09	.18	-.10	.14	.22
QSU-Brief	.25*	.26*	.11	.15	-.13	-.10	-.02	-.04	.13
TCQ-SF	.03	.12	.13	.04	-.06	.09	-.12	-.01	.09

BP: blood pressure; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief; IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale.

* $p < .05$, ** $p < .01$.

Table 5

Correlations Between Changes at Reactivation and Personality Traits

	SURPS				IPIP				
Change in measure	Hopelessness	Anxiety Sensitivity	Impulsivity	Sensation Seeking	Extraversion	Agreeableness	Openness	Conscientiousness	Neuroticism
Heart rate	.27*	.11	.15	-.13	.21	-.09	-.12	-.04	.01
Skin conductance	.07	.03	-.06	.19	-.12	.04	.34**	.04	.11
Systolic BP	.06	-.02	-.08	.004	.17	-.03	-.24	.06	-.13
Diastolic BP	.28*	-.14	-.02	.12	.05	.06	-.17	.002	.13
QSU-Brief	-.09	-.12	-.08	.12	.001	.12	-.07	-.03	.22
TCQ-SF	-.09	.10	.05	.05	.14	.08	-.08	.11	.26*

BP: blood pressure; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief; IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale.

* $p < .05$, ** $p < .01$.

Table 6

Correlations Between Changes at Extinction and Personality Traits

	SURPS				IPIP				
Change in measure	Hopelessness	Anxiety Sensitivity	Impulsivity	Sensation Seeking	Extraversion	Agreeableness	Openness	Conscientiousness	Neuroticism
Heart rate	-.06	-.07	.17	.10	.08	.07	-.22	-.02	.04
Skin conductance	-.16	-.24	-.29*	.21	.17	.05	.16	-.05	-.13
Systolic BP	-.10	.03	-.07	-.07	.20	.13	-.05	.08	.01
Diastolic BP	-.42**	-.10	-.01	-.12	.19	.04	-.07	.20	-.15
QSU-Brief	-.19	.19	-.01	-.03	.18	-.25	.04	.03	.08
TCQ-SF	-.08	.04	.02	.07	.12	-.13	-.04	.07	.04

BP: blood pressure; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief; IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale.

* $p < .05$, ** $p < .01$.

Post-Intervention Assessments

SURPS. Across sessions and groups, participants higher in sensation seeking were more likely to experience increased cravings due to withdrawal (from baseline to test 3: $r = .29$, $p = .025$) (Table 7). Hopelessness, on the other hand, was associated with decreases in withdrawal symptoms (CWS-21 total: $r = -.27$, $p = .032$ from baseline to test 1 and $r = -.36$, $p = .006$ from baseline to test 3), driven primarily by decreases in insomnia ($r = -.31$, $p = .013$ from baseline to test 1 and $r = -.36$, $p = .005$ from baseline to test 3). There were no other associations between SURPS personality traits and withdrawal symptoms (ps > .05) or associations with smoking behavior (ps > .09) across sessions.

IPIP. Across sessions and groups, participants higher in agreeableness initially experienced increased depression and anxiety due to withdrawal symptoms (on the CWS-21, from baseline to test 1: $r = .26$, $p = .044$) followed by decreased cravings (on the CWS-21, from baseline to test 2: $r = -.27$, $p = .039$). From baseline to test 2, those higher in extraversion reported decreased withdrawal symptoms overall (CWS-total: $r = -.28$, $p = .03$) while those higher in openness to experience were more likely to experience insomnia due to withdrawal (on the CWS-21: $r = .26$, $p = .047$). From baseline to test session 3, neuroticism was associated with decreased cigarette dependence (on the FTCD: $r = -.25$, $p = .049$) and withdrawal symptoms (on the CWS-21: $r = -.31$, $p = .015$), particularly irritability and impatience ($r = -.31$, $p = .015$) and difficulty concentrating ($r = -.29$, $p = .023$). There were no other associations between IPIP personality traits and CWS-21 withdrawal symptoms (ps > .05) or smoking behavior (ps > .06) across sessions.

Table 7

Correlations Between Changes in Smoking Behavior and Personality Traits

	SURPS				IPIP				
Change in measure	Hopelessness	Anxiety Sensitivity	Impulsivity	Sensation Seeking	Extraversion	Agreeableness	Openness	Conscientiousness	Neuroticism
No. of cigarettes smoked per day									
Baseline to test 2	-.10	.07	-.06	.16	.16	.13	.15	-.02	-.03
Baseline to test 3	< .001	.04	.05	.18	.20	.10	.02	-.002	-.04
FTCD score									
Baseline to test 2	-.14	-.003	-.21	-.15	.11	.11	-.06	.19	-.24
Baseline to test 3	-.09	.01	-.06	-.11	.15	.11	-.08	.19	-.25*
Contemplation Ladder score									
Baseline to test 1	-.08	.09	.09	.21	.05	-.06	.02	-.06	.16
Baseline to test 2	-.03	-.02	-.11	.19	-.02	-.15	.17	-.05	-.01
Baseline to test 3	-.08	-.02	-.11	.04	-.12	-.16	.03	-.13	-.05
CWS-21 total									
Baseline to test 1	-.27*	-.06	-.03	.02	-.08	.07	.16	-.06	-.17
Baseline to test 2	-.10	.10	-.05	-.13	-.28*	.03	.23	.01	-.03
Baseline to test 3	-.35**	-.07	-.10	.05	.09	< .001	.14	.01	-.31*
Depression-anxiety									
Baseline to test 1	-.10	.01	.10	-.14	-.08	.26*	-.10	-.12	.05
Baseline to test 2	-.04	.06	-.10	-.25	-.17	.24	.04	.01	-.10
Baseline to test 3	-.11	-.12	-.04	-.09	.08	.04	-.03	.07	-.15
Irritability-impatience									
Baseline to test 1	-.15	-.15	-.13	-.03	-.09	.10	.10	-.06	-.16
Baseline to test 2	-.09	-.02	-.19	-.03	-.11	.11	.17	.14	-.15
Baseline to test 3	-.22	-.12	-.20	.11	.06	-.01	.21	.10	-.31*
Difficulty concentrating									
Baseline to test 1	-.18	-.18	-.15	.12	-.07	.02	.09	.06	-.18
Baseline to test 2	-.08	-.14	-.11	.01	-.10	.04	.17	.10	-.04
Baseline to test 3	-.09	-.19	-.18	.11	.14	< .001	.11	.06	-.29*
Insomnia									
Baseline to test 1	-.31*	-.12	.07	< .001	< .001	.06	-.05	-.03	-.17
Baseline to test 2	-.17	.02	-.18	-.11	-.09	.14	.26*	-.21	-.07
Baseline to test 3	-.36**	-.01	-.02	-.06	.23	.14	.05	-.03	-.25
Appetite-weight gain									
Baseline to test 1	-.11	.14	-.09	-.10	-.10	-.03	.22	.07	-.07
Baseline to test 2	-.11	.10	.07	-.01	-.18	< .001	.09	.04	.04
Baseline to test 3	-.18	.01	-.07	-.09	-.19	.04	.08	< .001	-.11
Craving									
Baseline to test 1	-.05	.06	-.02	.10	-.06	-.11	.01	.05	.05
Baseline to test 2	.12	.15	.08	-.07	-.17	-.27*	< .001	.06	.19
Baseline to test 3	-.01	.06	.17	.29*	.01	-.17	.04	.10	.06

CWS-21: Cigarette Withdrawal Scale-21; FTCD: Fagerström Test for Cigarette Dependence; IPIP: International Personality Item Pool; SURPS: Substance Use Risk Profile Scale. * $p < .05$, ** $p < .01$.

Role of Childhood Adversity

Childhood maltreatment can influence responses to stressors and cigarette use. The Childhood Trauma Questionnaire (CTQ) was administered to assess the influence of early life stress on smoking behavior and responses to the stress-based intervention.

Lack of Interaction with the Stress-Based Intervention

No differences were observed between the control and stress groups on any of the CTQ factors (emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect) (Figure 9). There were no interactions between these factors and stress condition at intervention (all *p*s > .10) and across sessions (all *p*s > .05) for any psychophysiological and behavioral measures, suggesting that the stress-based intervention independently affected psychophysiological responsivity at intervention and subsequent smoking behavior changes.

Cue Reactivity and Smoking Behavior

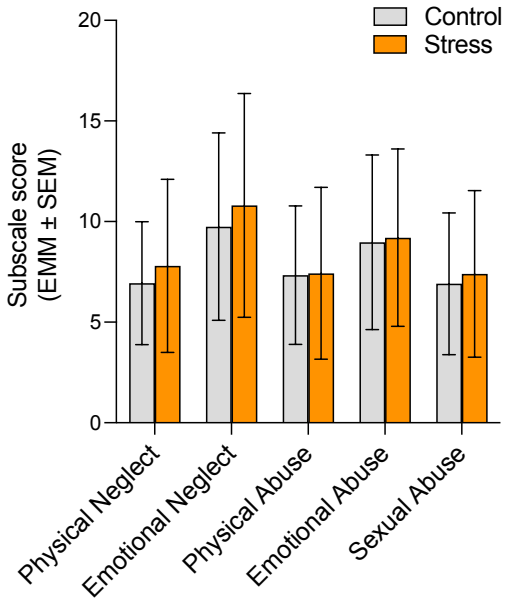
Participants who experienced emotional neglect, physical abuse, emotional abuse or sexual abuse did not show differences in cigarette use (Table 8) or craving or physiological responses to smoking cues at baseline (all *p*s ≥ .05). There was also no relationship between these subscales of the CTQ and the change in cigarette use across sessions (all *p*s > .10). However, participants who experienced physical neglect reported differences in baseline cigarette use and physiological responsivity at intervention compared to those without a history of neglect. These differences are described below.

Influence of Physical Neglect

Group Distribution. Participants were considered to have experienced childhood physical neglect (CPN) if they scored 8 or higher on this scale of the CTQ. These participants were equally distributed among the stress (n = 9) and control (n = 9) groups.

Figure 9

Mean Childhood Adverse Events of Participants in the Control and Stress Groups



The groups did not differ in CTQ scores on the subscales of physical and emotional neglect, and physical, emotional, and sexual abuse. CTQ: Childhood Trauma Questionnaire. EMM: estimated marginal mean; SEM: standard error of the mean.

Table 8

Correlations Between CTQ Scores and Cigarette Use Characteristics

	CTQ Subscales				
Measure	Physical Neglect	Emotional Neglect	Physical Abuse	Emotional Abuse	Sexual Abuse
No. of cigarettes smoked per day	.37**	.18	.10	.08	-.06
FTCD score	.06	-.06	.06	-.12	-.14
Age of first cigarette smoked	-.36**	-.33**	-.07	-.26*	-.30*
Years of smoking	.35**	.17	.32*	.23	.17
Contemplation Ladder score	< .001	-.03	.09	.12	-.03
Change in cigarette use from baseline to two weeks	-.37**	-.24	-.21	-.25	-.21
Change in cigarette use from baseline to six weeks	-.26*	-.15	-.11	-.15	-.13

CTQ: Childhood Trauma Questionnaire; FTCD: Fagerström Test for Cigarette Dependence.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Baseline Assessments.

Smoking Behavior. Prior to the intervention, participants with a history of CPN (n = 18) smoked significantly more cigarettes than participants without a history of neglect (n = 44) ($F(1,61) = 8.19$, $p = .006$; Table 9), with higher neglect scores predicting greater use ($r = .37$, $p = .003$). This was not accompanied by differences in FTCD cigarette dependence scores ($p > .95$) or motivation to quit smoking on the Contemplation Ladder ($p > .30$).

History of Smoking Behavior. Participants with greater physical neglect scores began smoking cigarettes at a younger age ($r = -.36$, $p = .004$; Figure 10A, Table 9) and smoked for a longer time ($r = .35$, $p = .005$; Figure 10B). The association between current cigarette use and CTQ physical neglect scores appears to be mediated by the duration of cigarette use ($b = .18$, $t(59) = 3.49$, $p = .001$) or age of onset ($b = -.40$, $t(59) = -2.14$, $p = .037$). While the association seems to be most closely related to the duration of use, the current mean age of participants with a history of neglect was higher ($F(1,61) = 1.73$, $p = .002$), potentially enlarging the group differences in duration of use.

Cue Reactivity. Participants with a history of CPN presented with significantly higher BP (systolic: $F(1,61) = 4.35$, $p = .041$; diastolic: $F(1,61) = 4.09$, $p = .048$) and a trend toward lower HR ($F(1,61) = 3.77$, $p = .057$) prior to the smoking video presentation compared to participants without CPN (Figure 11). Craving responses to the smoking video cues also differed, as reflected by a time by CPN status interaction (on the QSU-Brief: $F(1,60) = 71.23$, $p = .034$, but not on the TCQ-SF). Participants without a history of CPN had increased cue-induced cravings ($p < .001$) while those with a history of CPN did not ($p > .70$). There were no significant differences in pre-video SC ($p > .60$) or cravings (on the QSU-Brief, TCQ-SF or CWS-21: all ps $> .15$) or physiological responses to smoking cues (all ps $> .25$).

Table 9

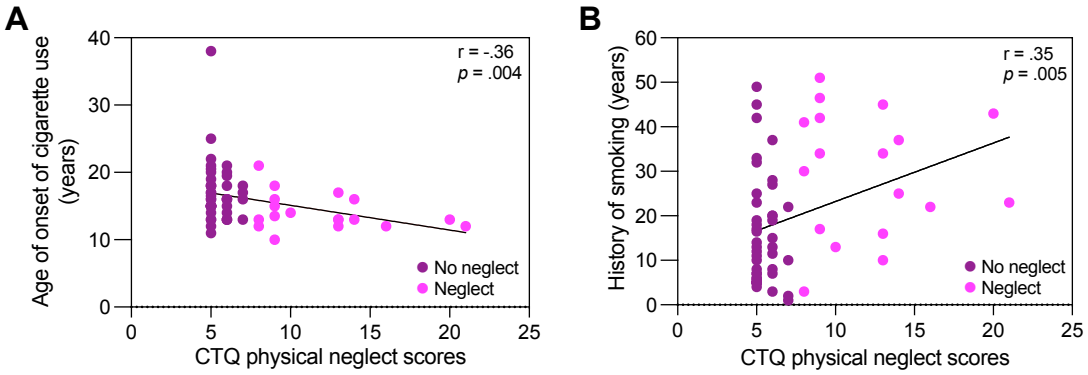
Demographic Characteristics of Childhood Physical Neglect and No Physical Neglect Groups

Characteristic	Participants, No. (%) / GROUP			Statistic	*P* Value
	All participants (n = 62)	No History of Physical Neglect (n = 44)	History of Physical Neglect (n = 18)		
Age, mean (SD)	**35.82 (12.99)**	32.61 (11.43)	43.67 (13.52)	$F = 1.73$	.002**
Sex					
Male	**34 (54.84)**	25 (56.82)	9 (5.00)	$\chi2 = .24$	.62
Female	**28 (45.16)**	19 (43.18)	9 (5.00)		
Ethnicity					
White	**45 (72.58)**	30 (68.18)	15 (83.33)	$\chi2 = 1.47$	.23
Other[a]	**17 (27.42)**	14 (31.82)	3 (16.67)		
Employed					
Yes	**39 (62.90)**	29 (65.91)	10 (55.56)	$\chi2 = .59$	.44
No	**23 (37.10)**	15 (34.09)	8 (44.44)		
Education					
No HS completion	**2 (3.23)**	2 (4.55)	0 (0)	$\chi2 = 2.21$	.53
HS graduate	**9 (14.52)**	6 (13.64)	3 (16.67)		
College or trade school graduate	**21 (33.87)**	13 (29.54)	8 (44.44)		
University graduate	**30 (48.39)**	23 (52.27)	7 (38.89)		
Annual household income, $					
≤ 20 000	**21 (33.87)**	15 (34.09)	6 (33.33)	$\chi2 = .003$	.95
> 20 000	**41 (66.13)**	29 (65.91)	12 (66.67)		
No. of cigarettes smoked per day, mean (SD)	**16.87 (5.87)**	15.58 (5.77)	2.03 (4.97)	$F = 8.19$	.006**
CO level (ppm), mean (SD)	**8.60 (5.72)**	8.77 (6.16)	8.17 (4.59)	$F = .14$	.71
FTCD score, mean (SD)	**6.05 (1.19)**	6.04 (1.22)	6.06 (1.16)	$F = .001$	.98
Contemplation Ladder score, mean (SD)	**4.97 (1.59)**	4.84 (1.46)	5.28 (1.87)	$F = .97$	.33
Age at first cigarette smoked, mean (SD)	**16.08 (4.14)**	16.90 (4.38)	14.08 (2.66)	$F = 6.43$	.014*
Years of smoking, mean (SD)	**19.74 (14.07)**	15.72 (12.06)	29.58 (14.07)	$F = 15.32$	< .001***
No. of MINI diagnoses					
0	**52 (83.9)**	37 (84.09)	15 (83.33)	$\chi2 = .005$	.94
1	**10 (16.1)**	7 (15.91)	3 (16.67)		

CO: carbon monoxide; FTCD: Fagerström Test for Cigarette Dependence; MINI: Mini International Neuropsychiatric Interview. [a] Other self-reported ethnicities included African American, Asian, Hispanic/Latinx, Indian, Indigenous, Middle Eastern, and other. ** $p < .01$, *** $p < .001$.

Figure 10

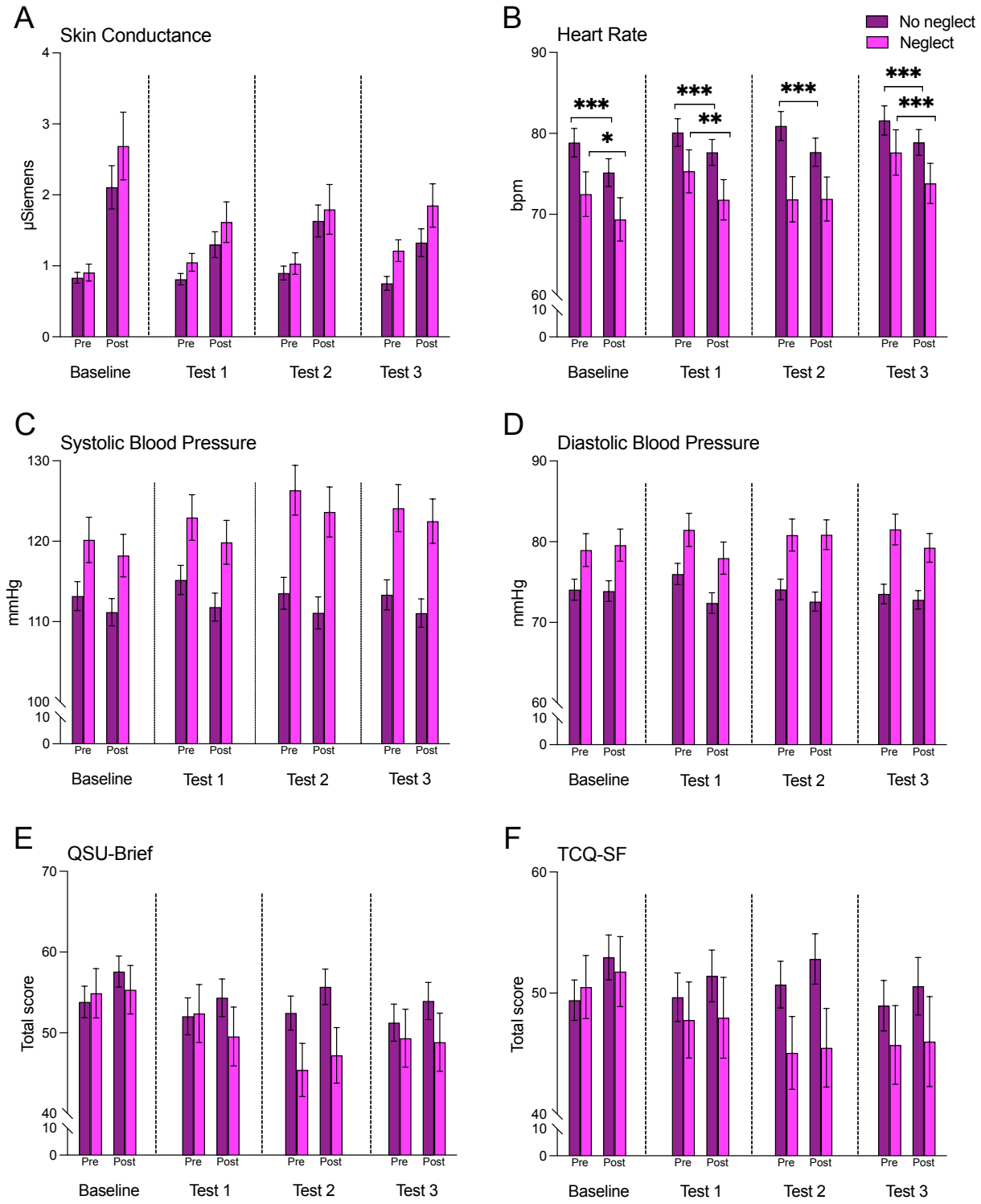
Physical Neglect Scores Correlated with the Onset and Duration of Cigarette Use



Greater physical neglect scores correlated with (**A**) earlier age of first cigarette use and (**B**) longer duration of use. Abbreviation: CTQ, Childhood Trauma Questionnaire.

Figure 11

Influence of Childhood Physical Neglect on Cue Reactivity



Across groups and across sessions, (**A**) skin conductance, (**C, D**) blood pressure and (**E, F**) cravings increased while (**B**) heart rate decreased in response to the smoking-related videos. (**B**) HR decelerations were observed in both groups, with more consistently significant effects within the no neglect group (all *p*s < .001). (**C**) When pre- and post-cue time points were combined within each session, systolic BP was typically higher in the neglect group (*p*s < .05 at test sessions 1, 2 and 3). (**E**) Across sessions, cue-induced cravings (on the QSU-Brief) were only observed in the no neglect group ($p < .01$). BP: blood pressure; TCQ-SF: Tobacco Craving Questionnaire – Short Form; QSU-Brief: Questionnaire on Smoking Urges – Brief; bpm: beats per minute; mmHg: millimetre of mercury. EMM: estimated marginal mean; SEM: standard error of the mean. ** $p < .01$, ** $p < .01$, *** $p < .001$.

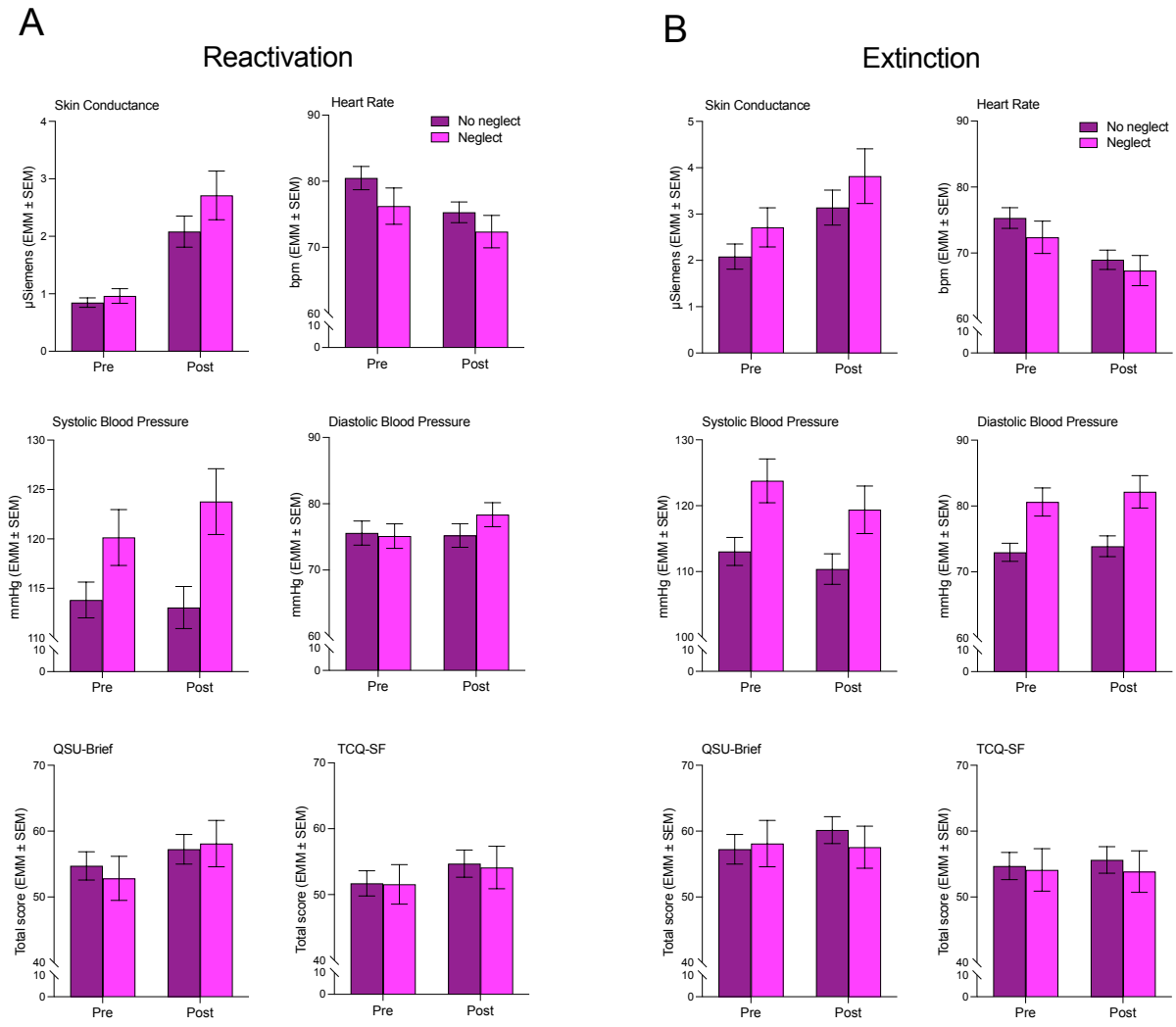
Responses to the Intervention.

Reactivation. Participants with a history of CPN exhibited greater BP overall across reactivation compared to those without a history of neglect (systolic: $F(1,60) = 6.14$, $p = .016$; diastolic: $F(1,60) = 5.99$, $p = .017$). They also showed a trend toward increased BP over the course of the reactivation phase (systolic: $F(1,60) = 3.85$, $p = .071$; diastolic: $F(1,60) = 3.83$, $p = .055$; Figure 12A). There were no significant differences in SC, HR or self-reported cravings (QSU-Brief and TCQ-SF) between participants with and without CPN as well as no significant changes in these measures over the reactivation phase (all ps > .20).

Extinction. During the extinction phase, higher BP (systolic: $F(1,60) = 6.78$, $p = .012$; diastolic: $F(1,60) = 1.46$, $p = .002$) was seen in participants with a history of CPN as compared to those without a history of CPN (Figure 12B). There were no significant differences in SC, HR or self-reported cravings (QSU-Brief and TCQ-SF) between participants with versus without CPN and no changes in these measures over the course of extinction (all ps > .15).

Figure 12

Influence of Childhood Physical Neglect on Psychophysiological Responses During the Intervention



(**A**) Participants with a history of childhood physical neglect (CPN) exhibited trends toward group by time interactions for BP (systolic: $p = .071$; diastolic: $p = .055$), reflecting greater increases in BP during the reactivation phase of the intervention. (**B**) During the extinction phase there were significant changes in systolic BP across groups (main effect of time: $p = .02$), but no differences in responses between groups. EMM: estimated marginal mean; SEM: standard error of the mean. BP: blood pressure. * $p < .05$; ** $p < .01$.

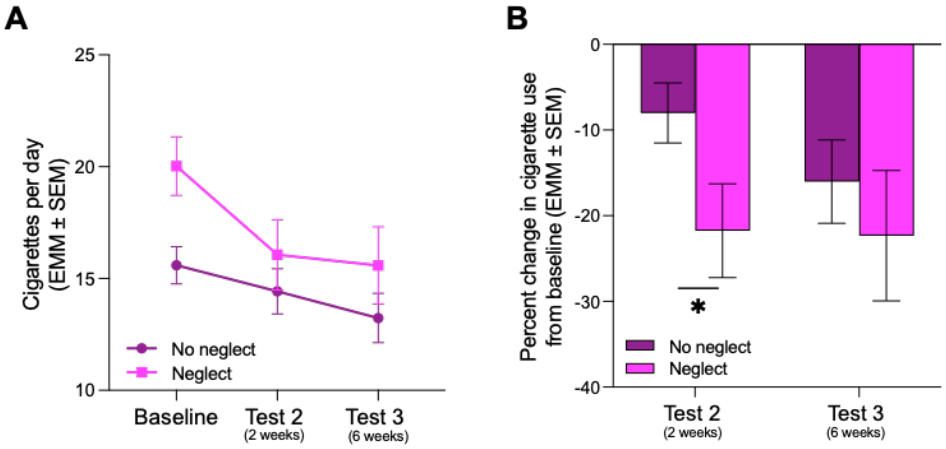
Post-Intervention Assessments.

Physiological and Craving Responses. Across all sessions, participants with a history of CPN continued to exhibit significantly higher BP (systolic: $F(1,60) = 11.15$, $p = .001$; diastolic: $F(1,60) = 1.99$, $p = .002$) as well as lower HR ($F(1,60) = 4.96$, $p = .03$) (Figure 11). They also showed an increase in BP (systolic: $F(3,180) = 2.75$, $p = .044$, but not diastolic) from baseline to test 2 ($p = .023$). There were no significant differences in overall SC, QSU-Brief or TCQ-SF scores at each session and no other changes in the pattern of responses to smoking video cues as a function of neglect (all ps > .10).

Change in Cigarette Use Across Sessions. Across all sessions, there was a trend toward a greater decrease in cigarette use in participants with a history of CPN ($F(1.59,95.25) = 3.27$, $p = .053$) (Figure 13A). An uncorrected pairwise comparison indicated that this was driven primarily by a decrease in cigarette use from baseline to test session 2 ($F(1,60) = 8.55$, $p = .005$) rather than baseline to test session 3 ($F(1,60) = 2.32$, $p = .13$) in the history of neglect versus no history of neglect groups. Regression analyses indicated that participants with greater neglect severity exhibited larger decreases in cigarette use from baseline to test 2 ($r = -.37$, $p = .003$; Figure 14A) and from baseline to test 3 ($r = -.26$, $p = .04$; Figure 14B). This represented 22% decreases in cigarette use at both two and six weeks in participants with a history of CPN compared to 7% and 15% decreases, respectively, in those without CPN histories (Figure 13B). No significant associations between CPN status and cigarette dependence or motivation to quit smoking were observed (all ps > .06).

Figure 13

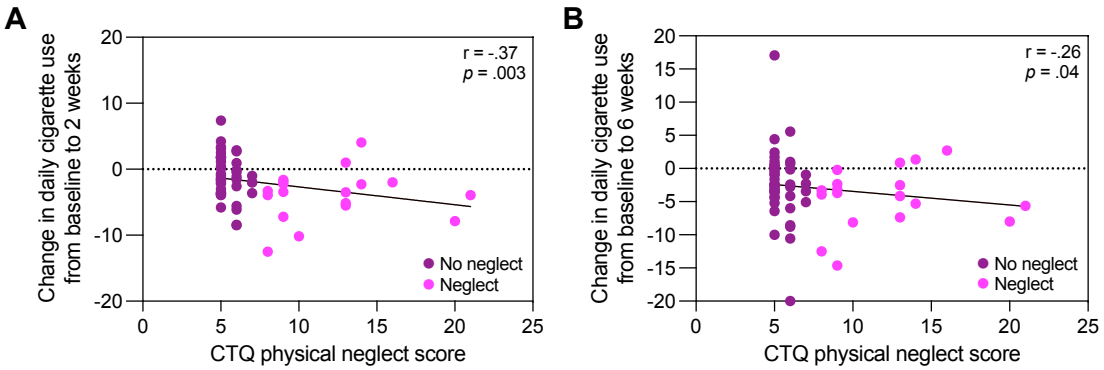
Childhood Physical Neglect Predicted Greater Decrease in Cigarette Use Two Weeks After the Intervention



(**A**) Participants with a history of childhood physical neglect (CPN) showed a trend toward a decrease in cigarette use over the duration of the study (session x group interaction: $p = .053$). (**B**) Participants with a history of CPN decreased their cigarette use by 22% at both two and six weeks, while those without a history of CPN showed decreases of 7% and 15%, respectively. EMM: estimated marginal mean; SEM: standard error of the mean. * $p < .05$.

Figure 14

Childhood Physical Neglect Scores Predicted Change in Daily Cigarette Use



Greater physical neglect scores predicted larger decreases in daily cigarette use at (**A**) two and (**B**) six-week follow-ups. Abbreviation: CTQ, Childhood Trauma Questionnaire.

DISCUSSION

Overview

The present randomized clinical trial used a memory updating paradigm to test whether (i) the ability of stressors to trigger smoking could be leveraged to decrease smoking, and (ii) individual differences in the magnitude of this response were related to pre-existing features. Together, this project permitted the exploration of many features that influence smoking behavior and responses to the stress-based intervention. The following main observations were made.

First, the results confirmed that, during the reactivation phase of the intervention, craving and physiological responses were larger following exposure to the stressor as compared to the control task. Second, and more importantly, during the weeks following the intervention, larger decreases in cigarette use were seen in the stress versus control group participants.

Third, contrary to our expectations, intervening without the stressor (i.e., only with smoking-related cues) was not effective. That is, contrary to what has been reported following a two-session memory updating protocol with smoking cues, our single-session intervention with smoking cues alone did not decrease cigarette use despite the cues inducing the anticipated psychophysiological responses at baseline and follow-up.

Finally, as certain personality traits and history of childhood maltreatment can affect stress responsivity and cigarette use, we hypothesized that these factors might influence the effectiveness of the intervention. As anticipated, physical neglect, neuroticism, anxiety sensitivity and hopelessness were associated with smoking behavior or cue reactivity at baseline, but there was little evidence that responses to the intervention were influenced by personality traits or histories of maltreatment. Although physical neglect was an independent predictor of

decreased cigarette use over the course of the study, this association was not seen for other early life stressors, and personality factors were not related to the efficacy of the intervention.

Effectiveness of the Stress-Based Intervention

As predicted, exposure to the psychosocial stress task during the reactivation phase induced physiological and craving responses. These effects have been reported previously (Al'Absi et al., 2003; Back et al., 2008; Buchmann et al., 2008; Childs & de Wit, 2010; Michalowski & Erblich, 2014). While some studies have shown that stressful events can increase the incentive salience of cues, leading to increased cue reactivity and motivation to smoke (Berridge & Robinson, 1998; Dagher et al., 2009; Niaura et al., 1992), we found the effects of the stressor to be independent of cue exposure (discussed below).

Most importantly, the stress-based intervention affected our primary outcome, cigarette use, which decreased by 26% in the stress group (as compared to 10% in controls) by six weeks. No other studies to my knowledge have harnessed the ability of stress to induce drug-seeking to reduce this same behavior. These findings could reflect a weakened association between stress and smoking. More specifically, the stress task may have reactivated smoking-related memories. In the subsequent extinction phase where smoking was not permitted, the association between stress and smoking may have weakened via memory updating mechanisms. Indeed, stress can enhance the retrieval of drug-related memories (Zhao et al., 2010), potentially increasing their susceptibility to undergo memory updating (Kuijer et al., 2020; Lee et al., 2017).

Further suggesting that the protocol's efficacy was related to the stress response comes from evidence that individual differences in decreases in cigarette use were predicted by the magnitude of cardiac responses during the intervention. That is, greater decreases in cigarette use were correlated with larger systolic BP responses post-reactivation and larger systolic BP

attenuations during extinction. Since these associations were seen across all participants, it raises the possibility that the stress task was not equally stressful to all participants and, for some, the control task was stressful. Systolic BP responses could therefore reflect both the reactivation of smoking-related processes and the efficacy of the intervention.

While this study was originally conceived as a memory updating paradigm, other processes could explain our results. One possibility is that the extinction phase alone led to decreased cigarette use. However, although extinction procedures (often in the form of exposure therapy) can produce transient reductions in cigarette use (Götestam & Melin, 1983; Lowe et al., 1980), long-term clinical efficacy is lacking (Brandon et al., 1987; Conklin & Tiffany, 2002; Götestam & Melin, 1983; Lowe et al., 1980; Niaura et al., 1999; Pericot-Valverde et al., 2019). More importantly, in the present study, all participants were exposed to a 60-minute extinction phase yet only those also exposed to the stressor exhibited significant decreases in cigarette use. It is therefore considered unlikely that extinction alone accounted for the present results.

A more plausible alternative interpretation may be that the stress procedure enhanced extinction. Few studies have investigated behavioral stress induction on extinction (as in the current study); most have used cortisol to induce a stress state. In fear-related studies, elevated stress hormones during extinction learning (endogenously or through prior cortisol administration) further reduced fear responses post-extinction and at follow-up (de Quervain et al., 2011; Meir Drexler et al., 2018; Meuret et al., 2015; Siegmund et al., 2011; Soravia et al., 2006; Yehuda et al., 2015). Pre-extinction stress induced by a cold pressor test similarly enhanced fear extinction (Antov et al., 2015) while a psychosocial stress prior to extinction blunted extinction learning and led to a greater return of fear the next day (Peyrot & Marin, 2019). In the only study (to my knowledge) examining the role of pre-extinction stress on

appetitive memories in a clinical population, administration of cortisol before exposure to alcohol cues decreased within-session cravings in those with a severe alcohol use disorder but increased cravings in those with less severe symptoms (Soravia et al., 2021). These limited results with a short follow-up (less than eight days) are challenging to interpret and compare with the current findings. Here, as well as in cue-based memory updating studies (Björkstrand et al., 2016; Germeroth et al., 2017; Zandonai et al., 2021), beneficial outcomes were evident at later time points but not during the extinction session or shortly after it. In contrast, many enhanced extinction studies report favorable effects during or soon after exposure to cues (de Quervain et al., 2011; de Quervain & Margraf, 2008; Soravia et al., 2006). These different within-exposure session effects may indicate that different mechanisms can lead to similar outcomes, though with different time-courses.

The stress-based intervention might also have improved coping. Though speculative, this could have occurred in at least three ways. First, participants' self-efficacious beliefs about their abilities to cope with stress may have increased after taking part in a stressful task without smoking. As stress is a potent trigger for smoking (Nakajima et al., 2020) and higher perceived self-efficacy predicts lower stress response and physiological arousal (Bandura et al., 1982), improved self-efficacy may have led to decreased cigarette use. Indeed, self-efficacy expectations are associated with increased motivation to quit and maintenance of smoking cessation (DiClemente et al., 1985; Gulliver et al., 1995).

Second, the stress-based intervention may have triggered the reconsolidation of ego-related memory. Psychosocial stressors can threaten ego and self-esteem, which in turn could affect ego-related memory (Smeets et al., 2007). While the psychosocial stress task may have initially increased negative self-perception, debriefing after the intervention (within the

reconsolidation window) may have induced a reappraisal of their abilities (e.g., they are more competent than they previously believed). Following reconsolidation, this updated appraisal may have boosted their self-esteem. As low self-esteem is associated with smoking (Bonaguro & Bonaguro, 1987; Croghan et al., 2006), efforts to improve self-esteem can positively affect health behaviors (Bonaguro & Bonaguro, 1987; Bonaguro et al., 1988) and lead to decreased cigarette use (Kim, 2011).

Third, the stress task alone may have induced cognitive reappraisal, in turn improving coping. An aversive situation (such as an acute stressor) can automatically trigger a negative appraisal, necessitating reappraisal to see it positively (Riepenhausen et al., 2022). The motivation to remain in the study (for example, for financial incentives, to please the experimenter, or keep a commitment) may have prompted a reappraisal of the stress task or their ability to cope with stress. As smokers tend to have difficulties using reappraisal to self-regulate emotions (Faulkner et al., 2022), the stress task may have inadvertently improved this ability, which in turn could have helped them reduce cigarette use. There is some evidence that single-session interventions can improve cognitive reappraisal (Rodriguez et al., 2019; Rodriguez et al., 2020; Rodriguez et al., 2021) and reduce substance use (Rodriguez et al., 2019), but no studies to my knowledge have used a stress-based intervention to prompt cognitive reappraisal as a treatment for substance use.

More research is needed to determine whether cognitive reappraisal, improved self-efficacy, memory updating or enhanced extinction could be the underlying mechanism of change.

Ineffectiveness of the Cue-Based Intervention

In the current study, smoking cue-based memory updating did not decrease cigarette use contrary to what was previously reported (Germeroth et al., 2017). This might be related to the inability of our smoking cues to elicit psychophysiological responses when they were combined with the arithmetic tasks (both stress and control versions). This was unexpected since our smoking cues yielded their anticipated effects (Betts et al., 2021; Saladin et al., 2012) when administered alone during the baseline and follow-up sessions. Several factors might explain this. First, the computerized arithmetic tasks may have served as distractors reducing the effectiveness of the smoking cues. Indeed, online games can distract from cravings and cigarette use (DeLaughter et al., 2016; Schlam & Baker, 2020). Second, ceiling effects may have occurred during the stressful version of the task. The smoking cues may not have been able to induce greater psychophysiological reactivity as the stress task already elicited near maximal responses. Third, habituation to the video cues from baseline to reactivation could not be ruled out, but this was considered unlikely as novel videos were presented at each session and the expected cue-induced responses re-emerged during the follow-up sessions.

There were no group differences in craving and physiological responses during the follow-up sessions, underscoring again the possibility that our paradigm did not alter responses to cues alone. This too differed from studies using cue-based memory updating interventions (Germeroth et al., 2017; Xue et al., 2012) and may reflect several procedural differences in addition to the lack of effect of cues at reactivation (noted above). As a start, we used a single intervention session whereas the previous studies provided two sessions (Germeroth et al., 2017; Xue et al., 2012). Second, the minimum duration of abstinence prior to each session was four hours as compared to at least 24 hours in previous work (Germeroth et al., 2017; Xue et al.,

2012). Third, we tested non-treatment seeking volunteers instead of those either seeking (Germeroth et al., 2017) or receiving treatment (Germeroth et al., 2017; Xue et al., 2012). Indeed, non-treatment seeking smokers exhibit larger smoking cue-reactivity responses than treatment-seekers (Sayette & Dimoff, 2016), potentially accounting for the persisting responses across test sessions. The continued ability of smoking cues to elicit craving despite changes in cigarette use could represent a dissociation between self-reported craving and substance use. Such dissociations are frequently reported and are thought to indicate that drug-seeking behaviors are driven in large part by processes outside of conscious awareness (Dagher et al., 2009; Rosenberg, 2009; Venugopalan et al., 2011). Our study's findings therefore suggest that the stress-based intervention may be more effective at targeting preconscious processes. Further studies with longer follow-up periods will be needed to determine whether the behavioral changes persist, increase further, or eventually dissipate.

Improvements in Smoking Behavior and Motivation to Quit in Controls

The control procedure also conferred some benefit during the study. As observed in the stress group, the control group reported increased motivation to quit smoking and decreased cigarette dependence and use. While this may in part be a byproduct of study participation (e.g., placebo or Hawthorne effect), it might also reflect individual differences in responsiveness to the intervention whereby the control task may have been stressful to some participants (as discussed above).

Increased attention to and awareness of smoking-related behaviors might also have played a role. Since many smoking behaviors become automatized with experience (Dar, 2018; Motschman & Tiffany, 2016), self-monitoring (such as through daily cigarette use journals used in the present study) can increase awareness of smoking and decrease automaticity (Bartlett et

al., 2014; Dar, 2018). Indeed, previous studies found that self-monitoring is associated with decreased frequency of cigarette use (Dar, 2018; Leventhal & Avis, 1976). Nonetheless, the decreases we observed in cigarette use were larger in the stress group, supporting the effectiveness of the stress-based intervention.

Influence of Individual Differences in Smoking and Response to the Intervention

As certain personality traits, such as neuroticism and anxiety sensitivity, and history of maltreatment are associated with altered stress reactivity and cigarette use (Chen & Baram, 2016; Choi et al., 2014; Guillot et al., 2016; Gunthert et al., 1999; Power et al., 2020; Zvolensky et al., 2015), we examined whether these factors influenced psychophysiological responses to the intervention and associated changes in smoking behavior. We did not find any effects of personality traits or maltreatment on the intervention's effectiveness, suggesting that our stress-based approach can be applied to a diverse population. In addition, some interesting findings emerged for the stress-related personality traits of neuroticism, anxiety sensitivity and hopelessness, as well as for one type of childhood maltreatment, physical neglect (discussed below). Aside from these findings, few conclusions could be drawn from the correlations and some of them may have been spurious, likely reflecting the large number of secondary associations evaluated.

Hopelessness

In the current study, participants with higher levels of hopelessness reported greater baseline cigarette use, dependence, and withdrawal symptoms. This was not surprising as depression and hopelessness (a predictor of depression (Rholes et al., 1985)) are well known risk factors for smoking initiation and maintenance (Anda et al., 1990; Glassman, 1993; Jalilian et al., 2014; Paperwalla et al., 2004; Winefield et al., 1989). Individuals with these personality traits

tend to smoke more cigarettes (Page, 1991; Spielberger, 1986) as smoking may be a way to self-soothe or quell feelings of emptiness created by a lack of meaningful pursuits (Clancy et al., 2013; Delfino et al., 2001; Gehricke et al., 2007).

Participants with heightened hopelessness may have also experienced more withdrawal symptoms since the severity of withdrawal increases with increasing cigarette use and dependence (McNeill et al., 1986; Ríos-Bedoya et al., 2008; Rojas et al., 1998). Greater withdrawal symptoms may additionally reflect depressive symptomatology rather than smoking abstinence since there is some overlap between the experiences of depression and withdrawal (Pomerleau et al., 2000; Pomerleau et al., 2005).

Furthermore, we found hopelessness to be associated with baseline cue-induced craving. Similarly, a previous study showed that smokers with depressive symptoms exhibit greater cravings in response to smoking-related cues (Weinberger et al., 2012), perhaps because they ascribe greater incentive salience to smoking cues (Kushnir et al., 2013). However, increased cue reactivity has also been linked to cigarette dependence (McClernon et al., 2008; Payne et al., 1996), therefore the increase in cue-induced cravings seen here may reflect greater dependence in smokers with heightened hopelessness.

Altered stress reactivity is also common in depression (Hu et al., 2016; Hughes & Stoney, 2000; Ilgen & Hutchison, 2005; Kibler & Ma, 2004) and as such it was foreseeable that hopelessness was associated with physiological changes during our intervention. Increases in physiological responses at reactivation occurred irrespective of stress condition, potentially because the control task may have been stressful for some participants (discussed above). Unpredictability and lack of control, as experienced during study participation, can certainly lead to heightened psychophysiological reactivity (Baker & Stephenson, 2000; Havranek et al., 2016),

which may be further augmented in individuals with depressive symptoms (Havranek et al., 2016).

At follow-up, hopelessness was associated with decreases in withdrawal symptoms. Considering there were no corresponding changes in cigarette use or dependence, it is unlikely that our intervention was more effective in participants with greater hopelessness. More probable is that this change reflects decreased rumination due to increased predictability and control as the study progressed. Given that we also observed improved insomnia in these participants, and that rumination is known to negatively affect sleep (Guastella & Moulds, 2007; Thomsen et al., 2003), it is possible that symptoms of insomnia may have reduced or resolved with decreased rumination, thus improving withdrawal symptoms.

Anxiety Sensitivity and Neuroticism

Studies have shown that individuals with anxiety-related traits tend to experience stronger withdrawal symptoms and cue reactivity when abstaining from smoking (Dubitzky & Schwartz, 1968; Hakulinen et al., 2015; Madden et al., 1997; Svicher et al., 2018; Watson et al., 2012; Zvolensky et al., 2014). Congruently, we found that participants higher in anxiety sensitivity and neuroticism (a predictor of anxiety (Jorm et al., 2000)) reported greater withdrawal symptoms and, in those with higher anxiety sensitivity, greater craving in response to smoking cues at baseline. Several factors may explain these findings. First, anxiety sensitive individuals may experience hypersensitivity to internal sensations during withdrawal (Johnson et al., 2012; Zvolensky et al., 2004) and exposure to smoking cues could affect those internal sensations (Zvolensky et al., 2003).

Second, anxious individuals are more likely to use ineffective coping strategies (Carmody et al., 2007; Feldner et al., 2004; Johnson et al., 2012) which may inadvertently maintain their

withdrawal symptoms (Brodbeck et al., 2013; Johnson et al., 2012) or trigger a rebound effect whereby cravings are intensified (Sayers & Sayette, 2013; Toll et al., 2001).

Third, misattribution of symptoms as withdrawal from smoking rather than situational anxiety could not be ruled out. Specifically, novel situations (such as the first study session) can be more anxiety-provoking for people with anxious personality traits (Raymond et al., 2017; Sep et al., 2019), and heightened anxiety could potentially induce greater cravings (Shiffman et al., 2003; Zvolensky et al., 2008) and affect attention and salience attributed to cues (Hellberg et al., 2019; Tanovic et al., 2018).

Neuroticism was associated with decreased withdrawal symptoms and cigarette dependence at follow-up. However, there were no changes in psychophysiological responses at reactivation as a function of anxiety-related traits, ruling out a more effective intervention in these individuals. Alternately, this could reflect improved self-esteem and self-efficacy in individuals higher in neuroticism who typically tend to struggle with their self-worth (Judge & Ilies, 2002; Strobel et al., 2011; Watson et al., 2002). Abstaining from cigarette use for long periods of time while exposed to the study's smoking cues may have enhanced these participants' confidence in maintaining abstinence, in turn reducing their cigarette dependence.

Childhood Maltreatment

Childhood maltreatment, including physical, emotional, sexual abuse and physical and emotional neglect, has been found to increase the likelihood of smoking in adulthood (Anda et al., 1999; Ford et al., 2011; Hussey et al., 2006; Mills et al., 2014; Topitzes et al., 2010). Concordantly, we showed that greater childhood physical neglect (CPN) status predicted larger baseline cigarette use. However, this was not observed for other types of neglect or abuse, potentially owing to our small sample (n = 62) and strict eligibility criteria. Whereas studies

focusing on maltreatment and smoking typically include large cohorts or population surveys without such criteria (for example, Anda et al., 1999; Kisely et al., 2020; Mills et al., 2014; Taha et al., 2014), the present study excluded volunteers with moderate to severe psychiatric disorders to optimize the sample for our primary objective (e.g., the study of memory updating). As different types of maltreatment tend to be associated with different psychopathologies (Cohen et al., 2014; Cohen et al., 2001; Ross et al., 2019), we possibly excluded some individuals who experienced certain kinds of childhood adverse events. Consequently, our results should be interpreted with caution as our sample may not be representative of the general population.

With regard to CPN, we found that participants who experienced CPN smoked more cigarettes at baseline and began smoking at earlier age. They were also older, had a longer history of smoking, and exhibited heightened blood pressure over the course of the study as blood pressure increases with age and duration of smoking (Landahl et al., 1986; Leone, 2011; Roberts & Maurer, 1977; Zhang et al., 2021).

Contrary to our expectations, participants with a history of CPN did not report increased cue-induced cravings at baseline (while cravings increased in participants without a history of CPN). This differs from cue reactivity studies in other substance users wherein cue-induced cravings were similar in maltreated and non-maltreated groups (Elton et al., 2015; Joseph et al., 2019). As there has been a general "neglect of neglect" in the literature (Dubowitz et al., 2022), few studies have specifically examined the effects of CPN and none, to my knowledge, have explored the potential relationship between CPN and cue reactivity. Therefore, we hypothesize that the lack of cue-induced cravings in our sample, but not in other maltreated groups, may reflect differences in selective attention, visual memory, and executive function, as shown in previous studies (Gould et al., 2012; Viola et al., 2013). Attention and reactivity to smoking cues

may have been reduced in our participants with a history of CPN, leading to decreased self-reported craving. In addition, CPN may have differentially affected participants with some exhibiting hyper-reactivity and others hypo-reactivity to cues, as observed previously with stress reactivity in individuals with a history of early life adversity (Agorastos et al., 2018; Boyce & Ellis, 2005; Wesarg et al., 2020). These opposing responses could have counteracted one another, obscuring differences in reactivity.

Furthermore, we observed a decrease in cigarette use in participants with a history of CPN, with greater neglect predicting greater decreases in use. Of note, these decreases in cigarette use occurred irrespective of reactivation condition and were unexpected as maltreatment is generally associated with smoking persistence and difficulty quitting (Cammack et al., 2019; Taha et al., 2014). A few reasons could account for this. First, I suspect that participants with a history of CPN may have been more susceptible to the Hawthorne effect; they may have been more likely to modify their smoking behavior out of a greater desire to please the experimenter or because they were being monitored. People with a trauma history often engage in people-pleasing behaviors and their motivation may stem from meeting the needs of others (Arai et al., 2011). Second, consistent and prolonged contact with the study team may have differentially affected participants with a history of CPN and, in turn, motivated them to reduce their cigarette use. Third, the reactivation phase may have been more effective in these participants as they may have been more prone to experiencing stress during either the control or stress task. Consistent with this hypothesis, participants with a history of CPN showed a trend towards increased blood pressure at reactivation, which may index the effectiveness of the intervention.

Limitations

While the results presented in this thesis support the use of a novel stress-based intervention for the treatment of cigarette use disorder, several limitations should be considered. First, our small sample size (n = 62) and lack of longer-term unadulterated follow-up sessions due to the COVID-19 pandemic may have precluded the observation of greater (or sustained) decreases in cigarette use over time. Although we observed a larger decrease in cigarette use in our stress group, this decrease became more pronounced over the course of the study. Even greater decreases may have been observed at longer follow-ups.

The completed sample size was within the range originally projected, but a larger sample could have allowed us to study the effects of maltreatment and personality factors in greater depth. The present study was not optimized to study these variables (as the focus was on memory updating) and applicants with certain personality traits or history of maltreatment may have been inadvertently excluded during the screening process (discussed above). Moreover, specific details about the history of the maltreatment (e.g., age and duration of exposure, protective elements) were not collected. These factors can affect later stress reactivity (Gunnar et al., 2009; Raymond et al., 2021) and therefore may have altered responses to the intervention.

We presented standardized smoking-related videos throughout the study. While these videos elicited the anticipated effects at baseline and follow-up sessions, they did not increase psychophysiological responding at reactivation. Greater responding may have occurred with personalized videos as individualized smoking cues induce greater cravings (Conklin et al., 2010). Customized smoking videos, images and paraphernalia could also have been used during the extinction phase. This may have enhanced the efficacy of the extinction procedure and, in turn, of the memory updating intervention.

As our smoking cues did not elicit the expected psychophysiological responses when combined with the arithmetic tasks, the present study may have benefited from a baseline assessment of stress and cues together. However, evidence of habituation and anticipation effects to repeated stressors (Velozo et al., 2021) made this a less viable option. Similarly, testing stress reactivity at follow-up would have been interesting as the intervention may have targeted stress responses rather than cue reactivity. While we considered presenting a variety of psychosocial stress tests at follow-up, it would not have been possible to control for differential responses to the different challenges.

Future Directions

In future studies, it would be beneficial to test the stress-based intervention in a larger sample and explore its effectiveness in treatment-seeking cigarette smokers and individuals with other substance use disorders. This would also provide the opportunity to further study the influence of early life stress and stress-related lifestyle factors on the intervention.

Future studies may also wish to investigate in greater depth the effects of stress on smoking cue reactivity. While some research suggests that stress may increase the incentive salience of cues (Dagher et al., 2009), we did not find a cue-specific effect on psychophysiological responding when cues were preceded by a stress (or control) task. Likewise, it may be of interest to study the potential distractive effects of various tasks on cue reactivity as this may have accounted for the lack of cue effect.

Considering that participants with a history of CPN decreased their cigarette use irrespective of intervention condition, it may be interesting to investigate whether study participation differentially affects individuals with a history of early life stress. It may also be beneficial to further study the effect of personality traits on cue reactivity given our evidence of

augmented responses in smokers high in anxiety sensitivity. As anxiety-related responses, such as rumination (Brozovich et al., 2015; DuPre & Spreng, 2018) and thought suppression (Erskine et al., 2007), can alter stress reactivity (LeMoult et al., 2013; Raymond et al., 2019), it could be useful to investigate their role in the stress-based intervention. This may also clarify if the intervention improved coping, which in turn could account for the decreases in cigarette use (discussed above).

Other variables, such as sleep and trauma history in adulthood, have also been shown to affect stress reactivity and memory performance (Dickie et al., 2008; Meerlo et al., 2008; Stickgold & Walker, 2005). Examining how they affect stress- and memory-related interventions could be of interest and, in the case of sleep, may potentially explain the progressively larger decreases we observed in cigarette use over time.

Finally, future studies may want to examine neurobiological underpinnings of the stress-based intervention. This could clarify differences between enhanced extinction and memory updating and elucidate the mechanism underlying the intervention's effects on smoking behavior.

Conclusion

While it is not possible to determine which mechanism (i.e., memory updating, enhanced extinction or improved coping) is involved, the present study nonetheless provides, to my knowledge, the first evidence that a single-session stress-based intervention can reduce cigarette use in non-treatment seeking smokers. Personality traits, such as hopelessness, neuroticism and anxiety sensitivity, and childhood maltreatment, such as physical neglect, may play a role in the maintenance of cigarette use, but we found the stress-based intervention to be effective irrespective of these factors, suggesting it could be used in a diverse population. With further

validation, this novel intervention could become a strategy for decreasing the use of various substances in people with little motivation to quit.

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