Biological Psychology 88 (2011) 1–12

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Biological mechanisms underlying the relationship between stress and smoking: State of the science and directions for future work

Jessica M. Richards[a,*], Brooke A. Stipelman[b], Marina A. Bornovalova[c], Stacey B. Daughters[d], Rajita Sinha[e,f], C.W. Lejuez[a,f]

[a] Department of Psychology, Center for Addictions, Personality, and Emotion Research (CAPER), University of Maryland, 2103 Cole Activities Building, College Park, MD 20742, USA
[b] The National Cancer Institute, Rockville, MD, USA
[c] Department of Psychology & Department of Mental Health Law and Policy, University of South Florida, Tampa, FL 33620, USA
[d] Department of Behavioral and Community Health, School of Public Health, University of Maryland, College Park, MD, USA
[e] Department of Psychiatry, Yale University School of Medicine, New Haven, CT, USA
[f] Yale Child Study Center, New Haven, CT 06520-7900, USA

ARTICLE INFO

Article history:
Received 6 October 2010
Accepted 22 June 2011
Available online 7 July 2011

Keywords:
HPA axis
Stress
Cortisol
Autonomic nervous system
Smoking
Nicotine dependence

ABSTRACT

Theories of addiction implicate stress as a crucial mechanism underlying initiation, maintenance, and relapse to cigarette smoking. Examinations of the biological stress systems, including functioning of the hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system (ANS), have provided additional insights into the relationship between stress and smoking. To date, convergent data suggests that chronic cigarette smoking is associated with alterations in HPA and ANS functioning; however, less is known about the role of HPA and ANS functioning in smoking initiation and relapse following cessation. In order to organize existing findings and stimulate future research, the current paper summarizes the available literature on the roles of HPA axis and ANS functioning in the relationship between stress and cigarette smoking, highlights limitations within the existing literature, and suggests directions for future research to address unanswered questions in the extant literature on the biological mechanisms underlying the relationship between stress and smoking.

Contents

* Corresponding author. Tel.: +1 301 405 8441; fax: +1 301 405 3223.
E-mail addresses: jrichards@psyc.umd.edu, jess0526@gmail.com (J.M. Richards).

0301-0511/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.biopsycho.2011.06.009

1. Overview

The CDC estimates that approximately 19.8% of U.S. adults currently smoke (CDC, 2008a). Moreover, cigarette smoking continues to be the leading preventable cause of death and disability in the United States accounting for approximately 1 of every 5 deaths (443,000 people) each year (CDC, 2008b). Because of its enormous public health impact, it is imperative to gain a comprehensive understanding of the mechanisms involved in the initiation, maintenance, and relapse of cigarette smoking.

In considering mechanisms within addiction, theories have emphasized the role of stress (Conger, 1956; Khantzian, 1985; Koob and Le Moal, 1997, 2001; Leventhal and Cleary, 1980; Marlatt and Gordon, 1985; Russell and Mehrabian, 1975; Sher and Levenson, 1982; Shiffman, 1982; Sinha, 2001; Solomon, 1977; Tomkins, 1966; Wikler, 1948; Wills and Shiffman, 1985). A number of studies have provided evidence that acute and chronic stress are related to smoking across multiple stages of the addiction process including initiation (Wills et al., 1996, 2002), maintenance (McEwen et al., 2008; Payne et al., 1991), and relapse to cigarette smoking (Cohen and Lichtenstein, 1990; Falba et al., 2005; Heishman, 1999; Hymowitz et al., 1991; Matheny and Weatherman, 1998; Siahpush and Carlin, 2006; Wewers, 1988).

Although there is a large body of research on the relationship between smoking and stress, this literature historically has been limited to self-report measurement (Cummings et al., 1985; O'Connell and Martin, 1987; Shiffman, 1982; Swan et al., 1988; Wills et al., 1996, 2002) which often relies on retrospective memories of stressful events and internal experiences, and may be subject to reporter biases (e.g., Turner et al., 1998). Despite the advancements made by self-report studies of the relationship between stress and smoking, many questions remain about the complex relationship between stress and smoking. A multimodal approach may provide one strategy for gaining clarity in this complex relationship. As a compliment to self-report, biological measures of stress may provide information about potential mediators and moderators of the relationship between stress and smoking. Two biological stress systems of interest are the hypothalamic–pituitary–adrenal (HPA) axis, which can be assessed via serum/plasma and salivary cortisol concentrations, and the autonomic nervous system (ANS) which is routinely examined by measuring catecholamine levels and cardiovascular responses. Available evidence suggests that chronic cigarette smoking is associated with altered functioning in these systems, and in some cases, these alterations have been shown to increase the reinforcing effects of acute smoking, particularly in the context of stress; however, many questions remain regarding the processes through which biological stress reactivity affects the relationship between stress and smoking across the stages of addiction. Thus, the aim of the current paper is to summarize the existing literature regarding the role of HPA axis and ANS functioning in the relationship between stress and smoking at each stage of nicotine addiction, to identify limitations and gaps within the extant literature, and to suggest clear directions for future research in order to specifically address the remaining questions in the literature.

2. Acute effects of nicotine and psychological stress

Striking parallels exist between the effects of acute nicotine exposure and psychological stress on neurobiological mechanisms involved in both stress regulation and reward. These neurobiological parallels point to important processes that can function to increase the reinforcing effects of nicotine, particularly in the face of acute stress. As such, a brief overview of the neurobiological effects of both acute nicotine and psychological stress exposure is in order.

2.1. Acute effects of nicotine

Acute nicotine administration is associated with increased HPA axis activation (Rohleder and Kirschbaum, 2006). Specifically, nicotine induces the release of corticotrophin releasing hormone (CRH) by binding to cholinergic receptors in the locus coeruleus and hypothalamus (e.g., Fuxe et al., 1989; Matta et al., 1998; Rosencrans and Karin, 1998). Subsequently, ACTH is released from the pituitary, followed by cortisol secretion by the adrenal glands, resulting in an overall increase in ACTH and cortisol. Indeed, research among human smokers has shown that cortisol reliably increases after smoking a minimum of two cigarettes (Caggiula et al., 1998; Kirschbaum et al., 1992, 1994) and the magnitude of acute nicotine-induced HPA activation (as well as changes in heart rate and positive subjective effects of smoking) appear to be dose-dependent (Gilbert et al., 1992, 2000; Kirschbaum et al., 1992; Meliska and Gilbert, 1991; Mendelson et al., 2005; Newhouse et al., 1990; Seyler et al., 1984; Winternitz and Quillen, 1977).

Cigarette smoking also reliably activates the autonomic nervous system (ANS), which is most frequently studied by assessing changes in plasma catecholamine levels (i.e., epinephrine and norepinephrine), as well as cardiovascular responses including systolic blood pressure (SBP), diastolic blood pressure (DSB), heart rate (HR) and heart rate variability (HRV). Specifically, acute cigarette smoking is associated with increased cardiovascular activation, which is mediated in part by catecholamine release (Cryer et al., 1976; Grassi et al., 1994), muscle sympathetic nerve excitation (Narkiewicz et al., 1998), and nicotinic receptor stimulation (Haass and Kubler, 1997). Research on ANS functioning among smokers has contributed greatly to our understanding of the physiological effects of both acute and chronic smoking (Adamopoulos et al., 2008; Cryer et al., 1976; Grassi et al., 1994; Haass and Kubler, 1997; Najem et al., 2006; Narkiewicz et al., 1998), as well as the mechanisms underlying the relationship between chronic cigarette smoking and cardiovascular disease (Adamopoulos et al., 2008).

In addition to activation in stress circuits, nicotine also activates reward circuits of the brain by binding to the nicotinic acetylcholin-