

Tobacco usage interacts with postdisaster psychopathology on circadian salivary cortisol

Miranda Olf^{a,*}, Marie-Louise Meewisse^a, Rolf J. Kleber^{b,c}, Peter G. van der Velden^b,
Annelieke N. Drogendijk^b, Jan G.C. van Amsterdam^d,
Antoon Opperhuizen^d, Berthold P.R. Gersons^a

^a Center for Psychological Trauma, Department of Psychiatry, Academic Medical Center/De Meren, Amsterdam, The Netherlands

^b Institute for Psychotrauma, Zaltbommel, The Netherlands

^c Department of Clinical Psychology, Utrecht University, Utrecht, The Netherlands

^d Laboratory of Toxicology, Pathology and Genetics, National Institute of Public Health and Environment, Bilthoven, The Netherlands

Received 13 October 2005; received in revised form 15 October 2005; accepted 20 October 2005

Available online 4 January 2006

Abstract

Posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) have been associated with increased rates of tobacco usage as well as with dysregulations of the hypothalamus–pituitary–adrenal (HPA) axis. At the same time tobacco also affects the HPA axis. This paper examines the relationships between PTSD, posttraumatic MDD, smoking and levels of circadian cortisol 2–3 years postdisaster. Subjects were survivors of the Enschede fireworks disaster. The sample consisted of 38 healthy survivors, 40 subjects with PTSD, and 17 subjects with posttraumatic MDD. The Composite International Diagnostic Interview was used to determine mental disorders in accordance with DSM-IV criteria. Salivary cortisol samples were collected at home immediately upon awakening, 30 min after awakening, at noon, and at 10 p.m. Quantity of smoking was measured through self-report. The results of the study show that salivary cortisol concentrations were higher in smoking subjects. Survivors with MDD following the disaster had a flatter diurnal cortisol curve than subjects with PTSD or healthy survivors. In survivors with PTSD and healthy individuals the usual dynamic pattern of increase in cortisol past awakening was present, while we did not observe this in posttraumatic MDD. These survivors with MDD tended to use more tobacco per day, and the cortisol group differences could only be revealed when we adjusted for quantity of smoking. Smoking, which may be an important palliative coping style in dealing with posttraumatic arousal symptoms, seems to mediate the relationship between traumatic stress and the HPA-axis.

© 2005 Elsevier B.V. All rights reserved.

Keywords: Smoking; Posttraumatic stress disorder; Major depression; Cortisol; Disaster

1. Introduction

The majority of people will be exposed to at least one traumatic event during their lifetime (Kessler et al., 1995). Of trauma exposed individuals, 20–30% of woman and 8–13% of men will develop posttraumatic stress disorder (PTSD) and a substantial proportion will develop major depressive disorder (MDD), substance disorder or a combination of those (Kessler et al., 1995; Olf and de Vries, 2004). The DSM-IV diagnosis

of PTSD consists of symptoms in three clusters: “reexperiencing” (intrusive recollections of the trauma that are triggered by exposure to cues symbolizing the trauma, nightmares, flashbacks); “avoidance and numbing” (diminished participation in activities and avoidance of thoughts, people, places, and memories associated with the trauma); and “hyperarousal” (difficulty sleeping, irritability, difficulty concentrating, hypervigilance, and exaggerated startle response).

Substance use, including tobacco use, has been previously described as a significant problem after traumatic events (e.g. the 9-11 attacks: Vlahov et al., 2004; sexual and physical assault: Acierno et al., 1996; exposure to childhood trauma: Walker et al., 1999; Springs and Friedrich, 1992; Felitti et al., 1998; American male veterans: Schnurr and Spiro, 1999;

* Corresponding author. Department of Psychiatry, Academic Medical Center/De Meren, Tafelbergweg 25, 1105 BC Amsterdam, The Netherlands. Tel.: +31 20 5662356; fax: +31 20 6919019.

E-mail address: m.olf@amc.uva.nl (M. Olf).

Dutch resistance veterans: Falger et al., 1992; and Israeli veterans: Shalev et al., 1990). Studies have shown that the degree of event exposure is associated with a greater degree of substance use after disasters (Beckham et al., 1997; Green et al., 1985), and subsequently that the number of cigarettes is related to higher morbidity and mortality after trauma. For instance, early death has been reported among smoking US veterans with a relative risk of 2.4 in very heavy smokers (Rogot and Murray, 1980). Relationships between psychopathology and smoking may explain part of these high mortality rates. Associations have also been found between smoking and MDD (Upadhyaya et al., 2002), and suicidal behaviour in psychiatric patients (McGee et al., 2005) and U.S. army personnel (Miller et al., 2000).

Associations between PTSD and substance use disorders have been reported in several studies (Kandel et al., 1997; Breslau et al., 1994, 2003, 1991). US data show that 60% of help-seeking PTSD patients smoke cigarettes, compared to 23% of the general population, and also that patients with PTSD are more likely to be heavy smokers (Beckham, 1999). Recently, Vlahov et al. (2004) demonstrated an increased prevalence of substance use among residents of New York City shortly after the 9-11 attacks, which persisted six to nine months later. Those persons who increased use of cigarettes were more likely to report symptoms consistent with PTSD and depression. Persons who experience major trauma may increase use of substances – either by starting to use, or by increasing in comparison to prior levels – to deal with arousal symptoms and negative affect. Smoking may thus be a coping mechanism following trauma exposure to reduce distress (Carmody, 1992), and thereby also affecting the neuroendocrine stress response (Olf et al., 2005).

Disparate results on the association between PTSD, depression and cortisol may partly be explained by the effects of smoking on the central nervous system and neuroendocrine parameters. Nicotine facilitates release of crucial neurotransmitters (e.g. acetylcholine, noradrenaline, vasopressin, dopamine, serotonin and beta-endorphin). After smoking of one cigarette both the systolic and diastolic blood pressure increase with 10 mmHg, heart frequency with 20 beats per minute and plasma adrenaline and noradrenaline with 250% and 40% respectively (Cryer et al., 1976). With heavy smoking the sympathetic nervous system is almost permanently stimulated. Smoking also activates HPA-axis. For instance, in chronic smokers compared to non-smokers increased cortisol levels have been found (e.g. Field et al., 1994), not only over the day, but as well as in response to waking (Steptoe and Ussher, in this issue). The early morning rise (EMR) of cortisol, typically with a peak half an hour after awakening, has been found to be an indicator of the stress response system and has been shown to be attenuated in e.g. PTSD (Neylan et al. 2005). Also acute effects of smoking on the HPA-axis have been found (Wilkins et al., 1982; Kirschbaum et al., 1992). On the other hand, results have not always been consistent and attenuated adrenocortical responses to stress have also been reported (e.g., al'Absi et al., 2003; Handa et al., 1994). Since nicotine facilitates neurotransmitters that modulate mood, smoking may

relieve symptoms of anxiety, irritability and depression following a traumatic event, thereby reinforcing smoking behavior. As such, apart from well-known physical health implications smoking may be an important problem in relation to mental health. One of the World Health Organization's (WHO) top priorities is the Tobacco Free Initiative (TFI), aiming at reducing tobacco use worldwide. 168 countries have signed the Framework Convention on Tobacco Control (FCTC) in April 2005. However, too little attention has been given to smoking related to psychiatric disorder.

Summarizing, although there seem to be associations between traumatic stress and smoking, traumatic stress and cortisol, and between smoking and cortisol, the relationships between traumatic stress, smoking, and cortisol have not yet received much attention. This is the first study to explore the relationships between PTSD, posttraumatic depression, smoking and levels of cortisol in a sample of trauma survivors. The aim is to examine how smoking would interact with the effects of a large disaster in the Netherlands on salivary cortisol in subjects with PTSD, posttraumatic MDD or no disorder. The cortisol response to awakening is a discrete and distinctive part of the cortisol circadian cycle (Clow et al., 2004), therefore we address both the diurnal pattern, overall production, and early morning rise of cortisol. We hypothesized that smoking would be associated with increased cortisol levels and that smoking would be an important mediator in the relationship between traumatic stress disorders and salivary cortisol.

2. Methods

2.1. Subjects

Participants of the present study were 95 Dutch speaking adults aged 20–80 years (mean 48.12, S.D. 15.38), living in the affected area at the time of the fireworks disaster in the city of Enschede, the Netherlands, on May 13, 2000. That day, the surrounding residential district was completely destroyed by the explosion of a fireworks storage depot. Twenty-two people were killed and almost one thousand were injured. Over 10,000 local residents were evacuated for one or more days, while over 1200 people lost their homes completely (Roorda et al., 2004). The participants were recruited as part of a large prospective study monitoring health after the disaster (Kamp and van der Velden, 2001). Most subjects were invited by a letter of the Dutch Ministry of Health Welfare and Sports to participate. In addition, announcements for the study were made in the media.

Two to three years after the disaster a subsample of subjects were invited by telephone to participate in an additional study concerning prevalence and predictors of postdisaster psychopathology. Of them, 263 subjects were interviewed at their homes, face to face by research employees with the Dutch version of the Composite International Diagnostic Interview (CIDI; WHO, 1997) to determine mental disorders in accordance with DSM-IV criteria (APA, 1994) and demographic data. Research employees underwent 3 days of didactic and supervised practical training in CIDI administration. The following modules of the CIDI were administered to determine

متن کامل مقاله

دریافت فوری ←

ISIArticles

مرجع مقالات تخصصی ایران

- ✓ امکان دانلود نسخه تمام متن مقالات انگلیسی
- ✓ امکان دانلود نسخه ترجمه شده مقالات
- ✓ پذیرش سفارش ترجمه تخصصی
- ✓ امکان جستجو در آرشیو جامعی از صدها موضوع و هزاران مقاله
- ✓ امکان دانلود رایگان ۲ صفحه اول هر مقاله
- ✓ امکان پرداخت اینترنتی با کلیه کارت های عضو شتاب
- ✓ دانلود فوری مقاله پس از پرداخت آنلاین
- ✓ پشتیبانی کامل خرید با بهره مندی از سیستم هوشمند رهگیری سفارشات