Concurrent Relations among Cigarette Smoking Status, Resting Heart Rate Variability, and Erectile Response

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ABSTRACT

Introduction. Heart rate variability (HRV) is a marker of sympathovagal balance; it has been implicated in erectile function and is also altered by tobacco use. Furthermore, smoking and erectile health are strongly related, given that smokers are at increased risk for erectile dysfunction. Few studies have explored the interrelationships between smoking, HRV, and erectile function concurrently.

Aim. The aim of this study was to examine potential mechanisms underlying tobacco's effects on penile hemodynamics by exploring the mediating role of HRV.

Methods. The sample comprised 119 men (smokers = 64; nonsmokers = 55) (mean age 28.90 years; standard deviation (SD) 11.68; range 18–58) selected from the control conditions of three previously published experiments. Participants were free from a history of cardiovascular disease, myocardial infarct, and/or cardiac/cardiovascular medication use. During a laboratory visit, self-report, anthropometric, cardiovascular, and electrocardiographic data were assessed, as well as sexual arousal responses elicited from viewing an erotic film.

Main Outcome Measures. Objective sexual arousal indices (circumferential change via penile plethysmography), self-reported erectile function (per the erectile function domain score of the International Index of Erectile Function [IIEF-EF]), and time- (SD of beat-to-beat intervals) and frequency-domain parameters of HRV (ratio of low-frequency [LF] power to high-frequency [HF] power [LF/HF ratio]) were assessed.

Results. Being a current long-term cigarette smoker was associated with dysregulated sympathovagal balance (higher LF/HF ratios, indicative of sympathetic nervous system dominance), which in turn showed inverse relations with magnitude of erectile tumescence. HRV did not mediate relations between tobacco use and either IIEF-EF scores or resting penile circumference.

Conclusions. Findings suggest that dysfunctional cardiac autonomic tone may be an underlying mechanism by which tobacco exerts its deleterious effects on erectile health. Further research is necessary to determine whether this relationship is mechanistic in nature, or whether it is better explained by other health factors. **Harte CB. Concurrent relations among cigarette smoking status, resting heart rate variability, and erectile response. J Sex Med 2014;11:1230–1239.**

Key Words. Smoking; Nicotine; Heart Rate Variability; Cardiac Autonomic Function; Sexual Arousal; Erectile Tumescence; Erectile Physiology

Introduction

Tobacco use is the most preventable cause of morbidity and mortality in the world today [1] and is responsible for introducing many adverse health conditions, including erectile dysfunction (ED). Large cross-sectional studies have indicated that smokers have approximately a twofold risk of reporting ED compared with nonsmokers [2–4]. Moreover, laboratory studies have shown that cigarette consumption [5] and isolated nicotine ingestion [6,7] both acutely reduce genital

arousal responses in men, whereas discontinuing cigarette use promotes improvement in erectile tumescence [8–10].

Despite a large body of literature delineating associations between smoking and male sexual health, a paucity of research has focused on mechanisms by which tobacco use may deleteriously affect erectile function (EF). Smoking may exert its adverse effects on penile hemodynamics via central [11] or biochemical [12] mechanisms, as well as peripherally [13], via changes in heart rate variability (HRV). HRV is a marker of physical and mental health [14,15] and is capable of noninvasively assessing vagal cardiac tone. Elevations in HRV (parasympathetic nervous system [PNS] dominance, evidenced by high variability in beat-to-beat intervals) represent healthy cardiac function, whereas reductions in HRV (sympathetic nervous system [SNS] dominance, characterized by low inter-beat variability), leave the heart vulnerable to arrhythmia and sudden death. Given that erectile hemodynamics and cardiac function are both largely under the influence of the autonomic nervous system (ANS), it is reasonable to believe that disruptions in cardiac autonomic balance (typically reflected as increased SNS and decreased PNS activity [16,17]) may serve as an underlying mechanism responsible for the pathophysiology of penile erection.

A growing body of literature has begun to explore the role of cardiac autonomic function as it pertains to tobacco use and erectile health. Regarding the former relationship, it has been shown that long-term cigarette smokers free from cardiac and cardiovascular disease (CVD), compared with nonsmokers, evidence increased SNS parameters and dysregulated (i.e., reduced) indices of HRV [18]. In addition, studies investigating temporal effects of cigarette smoking on cardiac function have provided additional evidence supporting the covariation between tobacco use and HRV. Specifically, among chronic smokers, cardiac dysregulation is acutely instigated by cigarette smoking [19,20], whereas smoking cessation improves a number of ANS indices, including HRV [21–23]. Regarding the potential link between HRV and ED, studies have shown that individuals with ED (compared with healthy controls) display dysregulated HRV, as evidenced by lower levels of both time-domain (i.e., standard deviation [SD] of the normal inter-beat intervals) and frequency-domain indices (power spectrum data capturing high-frequency (HF) and low-frequency (LF) ranges) [24–27].

Of particular interest is that smoking, HRV, and erectile health may be interrelated. In a recent study by Harte and Meston [28], who investigated smoking intensity, HRV, and erectile response among long-term male smokers, reductions in cigarette use over time was associated with temporal increases in HRV, which in turn was positively related to longitudinal changes in erectile tumescence. Although in need of replication, these findings underscore the potential underlying role of cardiac autonomic function in the physiology of erectile hemodynamics and point toward a possible mechanism that explains the link between tobacco use and erectile impairment. Given the public health consequences associated with both smoking and ED, it is an important goal to better understand mechanisms by which smoking may exert deleterious effects on EF.

Aim

This study attempted to build upon the scant literature examining the potential mechanisms underlying tobacco's effects on penile hemodynamics by exploring the mediating role of cardiac autonomic function (assessed via HRV). Specifically, this study attempted to determine whether resting HRV mediated concurrent associations between cigarette smoking status (smoker, nonsmoker) and several measures of erectile health (resting penile circumference, erectile tumescence, self-reported EF) among a sample of men from the United States without a history of CVD or myocardial infarct (MI). This study adds to the extant literature by exploring associations between smoking status, electrocardiographic (ECG) parameters, and erectile response in a concurrent fashion, among a sample of both smokers and nonsmokers. HRV was chosen as a potential mediator, given that this variable is a marker of sympathovagal balance, which is a chief underlying mechanism of erectile response [29].

Method

Participants

Participants were male volunteers recruited from an undergraduate psychology subject pool and via community and online advertisements between 2006 and 2010. Participants were selected from three experiments previously completed and published elsewhere [6,10,30]. Inclusion criteria for all studies comprised being at least 18 years of age,