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Smoking's effects on respiratory sinus arrhythmia in adolescent smokers ☆

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ABSTRACT

Respiratory sinus arrhythmia (RSA) has emerged as an indicator of how well the body maintains homeostasis and flexibly responds to environmental demands. Previous research has shown that smoking has both acute and chronic effects on RSA in adults. More recent work has focused on adolescent smokers because the natural decrease in RSA over the lifespan might be hastened by smoking at an early age. The goal of the current study, then, was to examine the acute effects of smoking on RSA and mean heart rate (HR) in a group of adolescent smokers. Participants completed two experimental sessions separated by 6–10 weeks, during which resting electrocardiogram (EKG) data were collected before and after smoking or not smoking a single cigarette ad libitum. Results indicate that smoking significantly decreased resting RSA and increased mean HR. In addition, those who smoked their first cigarette earlier in life (i.e., before age 8 or 10) evidenced a greater decrease in RSA during their smoking session relative to those who tried smoking after age 10. Importantly, these findings are largely consistent with the adult literature and suggest that smoking has acute effects on both RSA and HR in adolescence.

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1. Introduction

The negative health consequences of smoking are well-documented, as cigarette smokers experience greater rates of stroke, cancer, and emphysema than non-smokers (Department of Health and Human Services, DHHS, 2004). Smoking also has serious and wide-ranging effects on cardiac health, including an increased risk for heart attack (Price et al., 1999) and greater dysfunction in the dilation of blood vessels in the heart, which is one of the first indicators of atherosclerosis (Ambrose and Barua, 2004). Examining the processes that may contribute to these health risks is crucial to prevention efforts. To that end, greater empirical investigation of non-invasive indicators of cardiovascular health, such as respiratory sinus arrhythmia (RSA), may be particularly valuable.

RSA is the rhythmic fluctuation of heart rate (HR) during the respiratory cycle and has emerged as a state and trait indicator of the extent to which the vagus nerve mediates parasympathetic influences on the heart so the body can flexibly respond to environmental demands (Porges, 1995, 1997, 2007; Thayer and Lane, 2000). More specifically, the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) work together in a negative feedback loop to regulate numerous body systems, including cardiac functioning. The vagus nerve is specifically responsible for the PNS influences on HR, and a large body of evidence has indicated that these fast-acting vagal influences mediate more complex behaviors as well (Berntson et al., 1998; Porges, 2007). Thus, RSA is often conceptualized as a more general marker of self-regulatory capabilities (e.g., ability to modulate experience of emotion and arousal). Due to its close association with overall health, individual differences in RSA are impacted by various health-related behaviors, particularly smoking (e.g., Levenson and Ditto, 1981; Levin et al., 1992).

Previous research has shown that smoking has both acute and chronic effects on RSA in adults (Acharya et al., 2006). In laboratory studies, adult smokers typically manifest a decrease in RSA after smoking a single cigarette ad libitum (Hayano et al., 1990; Karakaya et al., 2013). Indeed, a small dose of nicotine (4 mg) is enough to affect autonomic nervous system (ANS) function (Sjoberg and Saint, 2011), while chronic use results in more long-lasting cardiac effects that persist beyond acute nicotine ingestion. For instance, smokers show an increased or abnormal resting HR, as well as decreased RSA, compared with non-smokers (Levin et al., 1992). Further, when trained in biofeedback, smokers are less able to decrease their HR than non-smokers, in large part due to differences in their physical condition and respiratory health (Levenson and Ditto, 1981). The negative effects of smoking are

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not limited to current smokers. Non-smokers exposed to secondhand (i.e., environmental) or sidestream (i.e., given off by the cigarette itself) smoke show similarly increased HR and decreased RSA compared with those in a smoke-free setting (Felber Dietrich et al., 2007; Pope et al., 2001; Valenti et al., 2013). Thus, the robust negative effects of smoking on cardiac health, as demonstrated by both acute and chronic decreases in RSA, are evident in a variety of settings.

It is important to note that the majority of existing studies on the acute (and chronic) effects of smoking on RSA have been conducted in adults. However, adolescence is considered a particularly sensitive time for smoking research and intervention as cigarettes have remained the substance most often used on a daily basis by high school students (Johnston et al., 2009). To our knowledge, only one previous study has focused on a younger sample, particularly young adults (below the age of 40). Results indicated that smokers and non-smokers had comparable levels of heart-rate variability (Erdem et al., 2015). Notably, this finding is discrepant with studies reporting group differences in RSA in older adult smokers relative to non-smokers (Levin et al., 1992), suggesting that age may indeed impact the association between smoking and cardiac functioning. Erdem et al. (2015) did not assess acute changes in RSA as a result of smoking, however, so this question remains unexplored.

This question is critical given that smoking's effects on RSA have important health implications for adolescents. Data indicate that low RSA (i.e., increased sympathetic and decreased parasympathetic influences) leads to chronic energy expenditure that precipitates premature aging, immune dysfunction, inflammation, and disease (Brook and Julius, 2000). Deficits in vagal functioning also appear to increase risk for cardiac disease, hypertension (Schroeder et al., 2003), abnormal fasting blood glucose levels (Singh et al., 2000), increased cholesterol (Kupari et al., 1993), and inflammatory markers (Janszky et al., 2004). Therefore, although adolescents may not be at immediate risk for cardiac disease, even acute reductions in RSA during nicotine exposure may weaken immune functioning and increase vulnerability to long-lasting and pervasive negative health outcomes.

The goal of the current study, then, was to examine the acute effects of smoking on RSA and mean HR in a group of adolescent smokers using a two-session (smoke vs. no-smoke) within-subjects design. The two experimental sessions were separated by 6–10 weeks and resting electrocardiogram (EKG) data were collected before and after smoking or not smoking a single cigarette ad libitum. We examined the effects of various individual difference variables (e.g., smoking behavior, nicotine dependence) on RSA and mean HR because it is unclear whether they modulate smoking's effects on cardiac functioning. With regard to the acute effects of smoking, we anticipated that responses would parallel those of adult smokers: RSA would decrease after smoking, as compared with not smoking, whereas mean HR would increase.

2. Material and methods

2.1. Participants

The current study draws from a cohort of adolescents who participated in a larger program project that examined the social and emotional contexts of smoking using various methodologies. Participants from 16 Chicago-area high schools were recruited via survey, which gathered information about smoking behavior, intentions for continued smoking, demographics, and parental smoking status. Based on their responses to this screening questionnaire, they were oversampled for current and past smoking behavior. Participants were excluded only if they were former regular users (i.e., smoked 100 cigarettes in their lifetime but none in the past 30 days) or former occasional smokers (i.e., smoked less than 100 cigarettes but none in the past 12 months); current high rate regular users (smoked 5 or more cigarettes daily in the past 30 days); or provided inconsistent smoking information during screening. Over 1400 eligible students and their parents were invited to participate in the longitudinal study and subsequent follow-up assessments. Of those, 1263 9th and 10th graders enrolled in the overall program project and have been followed longitudinally through a seven year follow-up.

Between 2006 and 2008, a subgroup of 217 adolescents participated in the lab-based study from which the current sample originates, "Smoking's Effect on Emotion in Adolescent Smokers." Though gender was not of particular interest in the current study, it was a focus of the larger project, and recruitment was targeted to obtain a roughly equal number of male and female participants. The goals of this lab-based study were to determine whether 1) adolescent smokers derive affective benefit from smoking a cigarette, 2) smoking deprivation results in nicotine withdrawal symptoms, and 3) individual differences in smoking's effect on emotional response and withdrawal reduction are predictive of subsequent developmental smoking behavior and patterns (see main results in Conrad, 2015 and Kassel et al., 2015). All participants completed two experimental sessions separated by 6–10 weeks, and participants who reported smoking at least one cigarette in the last two weeks without a desire to quit qualified as "smokers." They were offered the chance to smoke ad libitum at only one of their visits, which was chosen randomly. Of those who chose to smoke, 49 participants smoked during Visit 1 and 45 smoked during Visit 2. Of interest in the current study is how acute smoking might affect RSA. Therefore, we initially narrowed the sample to include only those participants who chose to smoke at one of these two visits; that is, they had both a smoking and non-smoking session ($n = 94$). The analytic approach used for the current study also required complete data for all participants, which meant we had to exclude those with missing or incomplete data from either session due to experimenter or equipment error ($n = 21$). Thus, the analyses reported here include 73 participants. Of note, those with incomplete data did not differ significantly from those with complete data on any demographic or smoking variables.

2.2. Procedure

Upon arrival in the lab for each study session (Time 1), all participants provided an expired breath carbon monoxide (CO) reading (Vitalograph EC 50 CO monitor, Vitalograph, Lenexa, KS). Prior to each CO reading, participants were instructed to take a deep breath, hold it for 15 s, and then blow forcefully into a mouthpiece so that all the air left their lungs. This procedure generally ensures the most accurate CO reading possible. They also completed various self-report questionnaires before being fitted with psychophysiological electrodes. Participants then sat quietly for 3–5 min while the research assistant set up recording software. EKG data were collected for a total of 2 min while participants were seated in an upright position and instructed to view a blank computer screen. This time period is consistent with American Psychological Association (APA) Record Keeping Guidelines when studying stress as measured via psychophysiology (American Psychological Association, 1993). Of note, respiration was not controlled or monitored in the present study. During the session in which they chose to smoke ("smoking session"), participants then lit a cigarette before placing it in a CReSS device, which examines many different aspects of smoking topography, including both volume and duration of each puff (results reported elsewhere; see Veilleux et al., 2011). They were told to smoke normally ad libitum (i.e., as much or as little of the cigarette as they desired) and remove the cigarette butt when finished. During the session in which they did not smoke ("non-smoking session"), participants were offered a magazine and asked to sit and relax for 9 min. This amount of time was chosen to approximately match the average amount of time it took participants to light a cigarette, insert it into the CReSS machine, smoke ad libitum, remove the cigarette from the CReSS machine, and extinguish the cigarette during the smoking session. Immediately after smoking (Time 2), participants provided a second CO reading; no CO reading was taken after relaxing during the non-smoking session. EKG data were collected for an additional 2 min,