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Adolescent's Respiratory Sinus Arrhythmia is Associated with Smoking Rate Five Years Later

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Abstract

Introduction—Vulnerability factors like respiratory sinus arrhythmia (RSA) may help identify adolescents at risk for nicotine dependence. We examined if resting RSA and the acute effects of smoking on RSA was associated with cigarette smoking five years later among adolescents at high risk for smoking escalation and nicotine dependence.

Methods—Sixty-nine adolescents participated in a baseline laboratory session- RSA was collected before and after smoking a single cigarette ad libitum. Participants were then followed for five years.

Results—Lower pre-smoke resting RSA was related to higher past month smoking rate five years later, even after controlling for baseline smoking rate and other relevant covariates including gender, race/ethnicity, age of initiated use, and frequency of exercise at baseline ($p = .018$). Exploratory analyses suggested resting RSA is an independent predictor of increased cigarette rate beyond other baseline predictors.

Conclusions—Low resting RSA may be a vulnerability factor, helping to identify adolescents at risk for cigarette escalation.

Keywords

adolescence; cigarettes; heart rate variability (HRV); nicotine; respiratory sinus arrhythmia (RSA); tobacco

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Introduction

Cigarette smoking and other tobacco use remains the leading cause of preventable death in the United States, accounting for over 480,000 deaths annually (CDC, 2014). Adolescence represents a particularly critical developmental period for smoking initiation, as 80-90% of adults report beginning to smoke before age 18 (Santelli, Sivaramakrishnan, Edelstein, & Fried, 2013; USDHHS, 2012). In addition to the negative health outcomes associated with cigarette smoking, recent work indicates that cigarette use in adolescence and young adulthood is associated with an increased risk for using other substances, behavioral problems, and dropping out of school (Orpinas, Lacy, Nahapetyan, Dube, & Song, 2015). These factors highlight the need to identify those who are at risk for nicotine dependence, before use becomes chronic.

Vulnerability factors linked to domains associated with cigarette smoking, including reduced emotion regulation (Fucito, Juliano, & Toll, 2010; Meyers et al., 2015; Wilens, Martelon, Anderson, Shelley-Abrahamson, & Biederman, 2013) may help to identify adolescents who may be at risk for nicotine dependence. One measure that is thought to be related to individual differences in emotion regulation, amongst other possibilities, is respiratory sinus arrhythmia (RSA), a measure of the rhythmic fluctuation of heart rate during the respiratory cycle (Porges, 1995, 1997; Thayer & Lane, 2000). RSA is often interpreted as a non-invasive measure of influences of the vagus nerve (i.e., the 10th cranial nerve) on the heart (Berntson et al., 1997; Porges, 1995, 1997; Thayer & Lane, 2000; however, for interpretative caveats see (Grossman, Karemaker, & Wieling, 1991; Grossman & Taylor, 2007). The vagus nerve is connected to brain structures involved in goal-directed behavior and adaptability, and individual differences in RSA are positively associated with cognitive flexibility (Johnsen et al., 2003), modulation of emotion and affect (see Porges, 1995), and inhibitory control (Allen, Matthews, & Kenyon, 2000; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). Consistent with this literature, several studies have found that low resting RSA (i.e., RSA collected at rest as an indicator of trait-like vagal functioning) is associated with psychological disorders characterized by affective dysregulation, including substance use disorders (Brody, Krause, Veit, & Rau, 1998; Carney, Freedland, & Stein, 2000; H. Cohen et al., 1998; Friedman, 2007; Friedman & Thayer, 1998; Ingjaldsson, Laberg, & Thayer, 2003; Malpas, Whiteside, & Maling, 1991; Thayer, Friedman, & Borkovec, 1996; Thayer, Smith, Rossy, Sollers, & Friedman, 1998). Additionally, acute nicotine use has been shown to decrease RSA in adults (Ashare et al., 2012; Hayano et al., 1990; Karakaya et al., 2007). Less work has been conducted with adolescents; however, our group found a similar pattern of decreased RSA in adolescents after smoking a single cigarette in the same sample as the current study (Conrad, Gorka, & Kassel, 2015). In addition, this effect was moderated by age of initiated cigarette use, such that individuals who had initiated smoking at an earlier age demonstrated a greater decrease in RSA after smoking (Conrad et al., 2015).

The goal of the current study was to extend our previous finding that smoking has acute effects on RSA in adolescents (Conrad et al., 2015) to now examine if resting RSA and the acute effects of smoking on RSA is associated with smoking behavior five years later among adolescents at high risk for smoking escalation and nicotine dependence. Importantly, RSA was captured in this study during adolescence, when individuals had a limited history of

cigarette use. Therefore, the current study presents the unique opportunity to better understand how potential vulnerability factors, like RSA, are related to smoking escalation among adolescents at high risk for developing nicotine dependence. Consistent with prior literature and our recent findings of acute effects of smoking on RSA in this same sample, we hypothesized that lower pre-smoke RSA and a larger change in RSA after smoking would be associated with more cigarette use five years later. To understand whether RSA is a specific vulnerability factor of subsequent cigarette use, we also examined relationships between mean heart rate at baseline and cigarette use five years later.

Materials and Methods

Participants

The sample was drawn from a longitudinal program project, the Social and Emotional Contexts of Adolescent Smoking Patterns (SECASP) Study (Dierker et al., 2013), which recruited a cohort of 9th and 10th graders from Chicago-area high schools. Participants in the longitudinal study were oversampled for ever-smoking a cigarette at baseline (83% ever-smoked), and thus at high risk for smoking escalation. Participants completed questionnaires assessing substance use and psychosocial factors at baseline and five years. The Institutional Review Board at the University of Illinois at Chicago approved the study. Written parental informed consent and participant informed assent were obtained.

Ninety-four participants participated in a laboratory session in which they smoked a single cigarette at baseline as a part of the larger SECASP study. Participants were recruited as a sample of convenience/willingness. They were invited from the SECASP study to participate in the laboratory session if they met the following criteria at baseline: 1) smoked a cigarette within 2 weeks of the session; 2) smoked on average one cigarette a week over the past three months; and 3) were not currently trying to quit smoking. These criteria were assessed in two ways: 1) self-reported smoking data in a questionnaire, which was verified against interview data for anyone who appeared eligible; and 2) all subjects were re-assessed when they arrived for the laboratory visit using the same measures. Sixteen participants had incomplete data at baseline. Thirteen participants did not complete the assessment at 5 years follow-up. Four of the 13 participants missing data at year 5 also had incomplete data at baseline. Therefore 25 participants were excluded from analyses due to missing data at baseline and/or 5 years and 69 participants with complete data were included in analyses. More males ($n=19$) than females ($n=6$) were excluded from the analyses due to incomplete data, but the 25 participants did not differ significantly from those with complete data ($n=69$) on any other baseline demographic variables or on any smoking variables at baseline and 5 years.

Laboratory Visit

Participants were asked to abstain from using tobacco for four hours prior to the laboratory session. Upon arrival at the laboratory session, breath carbon monoxide (CO) reading (Vitalograph EC 50 CO monitor, Vitalograph, Lenexa, KS) was obtained. Participants were then asked to sit quietly for 3-5 minutes while the research assistant set up recording software. EKG data were collected over two minutes while participants were seated in an

upright position and instructed to view a blank computer screen, consistent with the American Psychological Association (APA) Record Keeping Guidelines for measuring stress via psychophysiology (APA, 1993). Of note, we did not monitor or control for respiration in the present study, as heart rate and RSA were not the primary variables of interest. During the study visit, participants were offered one cigarette to smoke ad libitum. Participants' RSA was recorded before and after smoking. A more detailed description of laboratory procedures are published elsewhere (Conrad et al., 2015; Veilleux, Conrad, & Kassel, 2013).

Demographics and Potential Background Covariates

Demographic information, including race/ethnicity, education, gender. A number of other potential variables of interest in understanding smoking risk were also collected, including mother's highest education, father's highest education, the number of adult smokers in participant's household, how often around friends while they're smoking, how often around people while they're smoking, the number of days in the past week they exercised for 20 minutes and sweat, the number of days in the past week they exercised for 30 minutes without sweating, and the number of days in the past week they exercised to strengthen or tone was obtained through self-report questionnaires at baseline. Body mass index (BMI) was computed at baseline after study personnel measured participants' height and obtained weight using a scale. Participants completed the Beck Depression Inventory-II (BDI-II) and Beck Anxiety Inventory (BAI) to assess depression and anxiety symptoms at baseline. The seven item Modified Fagerstrom Tolerance Questionnaire (mFTQ; Prokhorov, Koehly, Pallonen, & Hudmon, 1998; Prokhorov, Pallonen, Fava, Ding, & Niaura, 1996), modified for use with adolescents, was used to assess nicotine dependence at baseline and at 5-years. The mFTQ has been found to be accurate and reliable in adolescent and young adult populations (Prokhorov et al., 1996; Wilens et al., 2008). Total sum score was used.

Cigarette Use

At baseline and 5 years, participants were asked to report the number of days they smoked in the past month (frequency of cigarette use) and the number of cigarettes they smoked on each day of use during the past month. These variables were used to calculate total cigarettes used over the past month and smoking rate (total cigarettes smoked in past month averaged over 30 days). Cigarette use frequency in the past month was measured using a single item question with 9 ordinal options coded as: 0= zero days; 1=one day; 3= 2-3 days; 5= 4-5 days; 7= 6-7 days; 9= 8-10 days; 16= 11-20 days; 25= 21-29 days; 30= every day. At baseline participants reported the total number of cigarettes they have smoked in their lifetime on a descending ordinal scale (1= 500 or more, 2= 100 or more, 3= 26-99, 4= 16-25, 5= 6-15, 6= 2-5, 7= 1, 8= 1 or more puffs, 9= never smoked) and the age they first smoked cigarettes. Additionally, at 5 years follow-up participants who endorsed smoking in the past month (n=60) reported how often they have tried to stop or cut-down, but have been unable to do so (1= not at all, 2= a little bit, 3= somewhat, 4= quite a bit), how many times they've seriously tried to quit (1= never tried to quit, 2= 1 time, 3= 2 times, 4= 3-5 times, 5= 6-9 times, 6= 10 or more times), and if they plan to quit smoking (1= yes, 2= no, 3= I don't smoke now).

Respiratory Sinus Arrhythmia (RSA) and Heart Rate (HR)

EKG data were recorded by electrodes placed on the subjects' inner forearms to record interbeat intervals (IBI) via a Schmitt trigger to interrupt the computer when a cardiac R-wave was detected. Data were recorded and initially processed using Acqknowledge 4.0 data acquisition software (BIOPAC Systems, Inc., Goleta, CA) installed on a Pentium IV computer, with a resolution of 1000 Hz. Artifacts were identified and manually corrected by hand. IBI data was then extracted and processed with CardioEdit (Brain-Body Center, University of Illinois at Chicago) for further artifact correction. Average RSA was calculated using CardioBatch based on methods developed by Porges and Bohrer (1990). The data were resampled into 30-second epochs and the IBI in each segment was calculated by averaging the time (in milliseconds) between R-peaks. RSA was then calculated by applying the adolescent CardioBatch filter to the data, which extracts the variance in the 0.12-1.0 Hz frequency band associated with heart-rate variability. Mean heart rate (HR) was calculated in the same manner using CardioBatch. RSA was computed as the natural logarithm of heart period variance and reported in units of ms squared, $\ln(\text{ms})^2$. Average RSA was obtained prior to smoking one cigarette (pre-smoke RSA) and after smoking one cigarette (post-smoke RSA). We also calculated the change in RSA pre- to post-smoking (pre-post smoke change in RSA; post-smoke RSA minus pre-post RSA).

General Statistical Procedures

All analyses were carried out using SPSS 20.0 (IBM). Participants' demographic and substance use variables were compared at baseline and 5 years using ANOVA or chi-square tests as appropriate. Data were inspected for non-normal distribution and outliers. Five year past month smoking rate (skewness= 0.83 (SE= 0.29), kurtosis = -0.50 (SE= 0.57)) and 5 year past month smoking frequency (skewness= -0.71 (SE= 0.29), kurtosis = -1.21 (SE= 0.57)) violated the Shapiro-Wilk test of normality (p -values < .05), so square-root transformations were used for these variables. To test our hypotheses, we conducted 6 hierarchical multiple regression analyses with baseline pre-smoke RSA, post-smoke RSA, and pre-post smoke change in RSA as separate independent variables in the first block and gender, as well as baseline frequency of past month cigarette use or baseline past month smoking rate as a covariate in the second block in each model. In addition, bivariate correlations between cigarette use measures, RSA measures, and several participant characteristics were run and any variables that were significantly associated with 5 year cigarette use measures or baseline RSA measures were included as a covariate in respective models. Cigarette use variables at 5 years follow-up, including frequency of past month cigarette use and past month smoking rate served as separate dependent variables. Furthermore, to examine whether cigarette use at 5 years follow-up is related to RSA or heart rate in general, we performed linear regressions with baseline pre-smoke mean HR, post-smoke mean HR, and pre-post smoke change in mean HR as separate independent variables and frequency of past month cigarette use and past month smoking rate served as separate dependent variables. All results were deemed statistically significant when p -values < .05.

Results

Participant Characteristics

Participant characteristics at baseline and at 5 years follow-up are shown in Table 1. As expected, participants had significantly higher symptoms of nicotine dependence at five years compared with baseline.

Relationships between Baseline RSA and Cigarette Use at 5 Years Follow-Up

Bivariate correlations found that age of initiated use was significantly associated with cigarette use measures, so age of initiated use was included as a covariate in all models (see Supplemental Table 1). In addition, race/ethnicity was significantly related to smoking rate at 5 years follow-up, so race/ethnicity was included as a covariate in models with smoking rate at 5 years follow-up (see Supplemental Table 1). Finally, baseline number of days in past week exercised to strengthen or tone was significantly associated with post-smoke RSA, so this variable was included as a covariate in analyses with post-smoke RSA, including pre-post smoke change RSA.

Results revealed that lower baseline pre-smoke RSA was related to a higher past month smoking rate at 5 years follow-up, even after controlling for baseline past month smoking rate, gender, and other relevant covariates (see Table 2). On the other hand, baseline pre-smoke RSA, post-smoke RSA, and pre-post change in RSA was not associated with frequency of past month cigarette use at 5-years follow-up (Table 2). Baseline post-smoke RSA or baseline pre-post smoke change RSA was not related to higher past month smoking rate at 5 years follow-up (Table 2). We also ran an additional hierarchical regression to examine the residualized difference score of pre-post smoking RSA (see Supplemental Table 2) and found similar results as the difference score.

Relationships between Baseline Heart Rate and Cigarette Use at 5 Years Follow-Up

Pre-smoke, post-smoke, and pre-post smoke change mean HR were not related to past month cigarette frequency at 5 years follow-up ($\beta = 0.10$, $p = .43$; $\beta = 0.14$, $p = .24$; and $\beta = 0.10$, $p = .42$, respectively). Similarly, pre-smoke, post-smoke, and pre-post smoke change mean HR were not related to past month smoking rate at 5 years follow-up ($\beta = 0.14$, $p = .26$; $\beta = 0.12$, $p = .35$; and $\beta = 0.02$, $p = .85$, respectively).

Exploratory Analyses

Due to the fact that many participants smoked everyday or close to everyday in the past month, we ran three follow-up, binary logistic regressions with individuals who smoked 25-30 days in the past month at 5 years follow-up ($n=40$; coded as 1) versus individuals who smoked less than 25 days in the past month at 5 years follow-up ($n=29$; coded as 0) as the dependent variable and pre-smoke RSA, post-smoke RSA, and pre-post smoking change RSA as separate independent variables, respectively, as a sensitivity analyses for frequency of cigarette use at 5 years follow-up. The covariates used in the primary analyses were also used in each of these respective models (see Table 2). None of the binary logistic models were significant: 1) pre-smoke RSA omnibus model ($\chi^2 = 5.56$, $p = .14$, Cox & Snell $R^2 =$

0.08); 2) post-smoke RSA omnibus model ($\chi^2 = 5.22$, $p = .27$, Cox & Snell $R^2 = 0.07$); and pre-post smoke RSA omnibus model ($\chi^2 = 3.17$, $p = .37$, Cox & Snell $R^2 = 0.05$).

Furthermore, given evidence that measures of distress and stress response are related to quit attempts and smoking relapse (al'Absi, 2006; al'Absi, Hatsukami, & Davis, 2005; S. Cohen & Lichtenstein, 1990; Shadel & Mermelstein, 1993; Slopen et al., 2013), we examined if baseline RSA measures were related to quit attempts at 5 years follow-up with Spearman's rho correlations. Among participants who reported smoking in the past month at 5 years follow-up ($n=60$), baseline pre-smoke RSA, post-smoke RSA, and pre-post smoking change in RSA were not related to how often participants have tried to stop or cut-down, but have been unable to do so ($r(56) = -0.17$, $p = .19$; $r(56) = -0.09$, $p = .52$; $r(56) = -0.09$, $p = .51$, respectively), how many times participants have seriously tried to quit ($r(56) = 0.004$, $p = .97$; $r(56) = -0.05$, $p = .73$; $r(56) = 0.08$, $p = .53$, respectively), or if participants plan to quit smoking ($r(56) = 0.20$, $p = .13$; $r(56) = 0.15$, $p = .26$; $r(56) = 0.01$, $p = .97$, respectively). When examining if baseline RSA measures were related whether participants had quit smoking at 5 years follow-up ($n=9$) or continued smoking ($n=60$), Spearman's rho correlations were not significant for pre-smoke RSA ($r(67) = -0.08$, $p = .49$), post-smoke RSA ($r(67) = -0.02$, $p = .85$), or pre-post smoking change in RSA ($r(67) = -0.04$, $p = .74$).

Discussion

In this study, we examined whether RSA among adolescents at high risk for developing nicotine dependence was related to their smoking behavior five years later during young adulthood. We found that although baseline RSA was not associated with cigarette frequency at five years, lower baseline pre-smoke RSA was related to a higher smoking rate in the past month five years later, even after controlling for baseline smoking rate and several relevant covariates including gender, race/ethnicity, age of initiated use, and frequency of exercise at baseline. The relationship between lower baseline pre-smoke RSA and higher smoking rate in the past month five years later had a medium effect size ($sr^2 = .068$ after controlling for baseline smoking rate and other relevant covariates), similar or larger to many reported psychosocial and substance use predictors of adolescent cigarette use (Collins et al., 1987; Everett et al., 1999; Flay, Hu, & Richardson, 1998; Roberts, Colby, & Jackson, 2015). In addition, baseline heart rate was not related to cigarette use at 5 years follow-up. This provides preliminary evidence that lower baseline RSA during adolescence can be used as a vulnerability factor in predicting smoking escalation, especially increased intensity of smoking in young adulthood, but it may not be a sensitive predictor of smoking frequency in young adulthood. Of note, many individuals in this sample smoked frequently at baseline, on average smoking 18 out of 30 days per month, so we were concerned that the lack of relationship with smoking frequency may be due to a ceiling effect. However, exploratory binary logistic regression analyses also did not find a relationship between baseline RSA measures and whether participants smoked at least 25 days out of the past 30 days at 5 years follow-up, supporting the notion that baseline RSA is not a sensitive predictor of smoking frequency in young adulthood. Further, we found that post-smoking RSA and change in pre-post smoking RSA were not associated with subsequent cigarette use.

Our findings support the notion that low RSA may be a trait or developmental vulnerability that increases adolescents' likelihood of escalating cigarette use, especially those who have already begun smoking, and that the acute effects of smoking on RSA do not connote risk for subsequent cigarette use in adolescence nor does it account for the low resting RSA seen in adult chronic smokers (Levin, Levin, & Nagoshi, 1992). This is in line with a recent study finding that RSA in children who had prenatal exposure to substances was predictive of subsequent substance use and risky sexual behavior during adolescence (Conradt et al., 2014), indicating that RSA may reflect trait or developmental vulnerabilities that increase adolescents' risk for substance use. It is also possible that adolescent cigarette use disrupts RSA, even with a limited history of use, which then contributes to an increased risk of smoking escalation. Due to the fact that we captured RSA during adolescence, when individuals had a limited history of cigarette use, and we controlled for baseline recent cigarette use, our findings suggest that chronic nicotine exposure does not fully account for the low RSA seen in chronic smokers. However, it may be that individuals who go on to smoke more have lower RSA to begin with and smoking may exacerbate possible deficits in physiological regulation and also perhaps emotional regulation over time. The acetylcholine neurotransmitter system is one neurotransmitter pathway for nicotine (see Henderson & Lester, in press), the same system involved in vagus nerve activity (Thayer, 2009). Indeed, decreased heart rate variability has been found in chronic, habitual smokers, suggesting nicotine use may decrease heart rate variability (Dinas, Koutedakis, & Flouris, 2013). As such, smoking in adolescence may attenuate individual differences in RSA through nicotine's affect on autonomic cardiac control, exacerbating the pre-existing vulnerability of low RSA in individuals who engage in cigarette use at an early age. On the other hand, it may be that we did not capture RSA early enough in this sample, as many individuals had already smoked over 100 cigarettes at baseline even though they were not daily or high rate (more than 5 day) smokers, and this exposure to nicotine may have altered RSA and increased individual's risk for smoking escalation. It will be important for longitudinal studies to examine how RSA, measures of emotional regulation, and other cognitive and physiological processes linked to RSA may predict subsequent substance use in individuals at an earlier age, prior to the onset of substance use.

The results suggest that low resting RSA may be a vulnerability factor that could help identify adolescents at risk for cigarette escalation for prevention and intervention efforts. For example, vagal enhancement treatments (e.g., mindfulness meditation; Krygier et al., 2013; Libby, Worhunsky, Pilver, & Brewer, 2012) may increase resting RSA in vulnerable individuals and therefore lessen the risk that they will escalate smoking. Future research is needed to better understand whether increases in resting RSA are related to change in subsequent cigarette escalation risk outcomes and if so, what may be the mechanisms of this treatment.

The current findings should be considered in the context of limitations. First, participants were current experimenters and regular adolescent smokers who smoked at least once a week in the past three months and had smoked recently, within two weeks of the session. Therefore this is a high-risk sample for cigarette smoking escalation, limiting the generalizability of our findings. Second, RSA was captured in this study during adolescence after most participants had already had exposure to cigarettes and this non-chronic exposure

may have disrupted their baseline RSA. Along these lines, nicotine use has been shown to raise sympathetic tone (Narkiewicz et al., 1998; Niedermaier et al., 1993), which may lead to reciprocal vagal withdrawal. Due to the fact that we did not assess a direct measure of sympathetic activity, we cannot rule out that this may have influenced the findings. Third, we did not have a measure of respiration in the current study, which may influence RSA measurements (see Denver, Reed, & Porges, 2007; Hatfield et al., 1998; Porges, 2007; Sargunraj et al., 1996; but also see Grossman, Karemaker, & Wieling, 1991; Grossman & Kollai, 1993; Grossman & Taylor, 2007; Houtveen, Rietveld, & de Geus, 2002). Furthermore, some evidence suggests that between subjects there is only a loose association between actual vagal tone and RSA due to residual inspiratory vagal tone (Grossman & Kollai, 1993), limiting our ability to interpret RSA as a direct measure of vagal activity. Lastly, RSA has also been reported as a risk factor in longitudinal studies of risk for depression, including measures prior to onset of illness or any substance use (Jacobs, Orr, Gowins, Forbes, & Langenecker, 2015). This suggests that RSA may, indeed, be a potential vulnerability factor, if somewhat nonspecific, for smoking risk.

Our study expands upon previously reported relationships between measures of RSA and cigarette use, finding that lower adolescent resting RSA is related to a higher smoking rate during young adulthood. Therefore, our results indicate that that low baseline RSA, which may be related to poorer emotion regulation, is an important vulnerability among adolescent smokers, who have a limited history of use, that increases their likelihood of escalating their in cigarette use in young adulthood. Low resting RSA may be a vulnerability factor that could help identify adolescents at risk for cigarette escalation for prevention and intervention efforts. Future longitudinal studies are needed to better understand if emotional regulation is a key factor involved in initiation and continued cigarette use, as well as other substance use.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Vulnerability factors may help to identify adolescents at risk for nicotine dependence
- We examined if RSA was associated with cigarette smoking 5 years later
- Lower resting RSA in adolescence was related to higher smoking rate 5 years later
- Resting RSA seems to be a unique factor related to cigarette escalation

Table 1

Participant Characteristics

	Baseline (n=69) <i>M</i> ± <i>SD</i> [<i>Range</i>]	5 Year (n=69) <i>M</i> ± <i>SD</i> [<i>Range</i>]	<i>p</i> -value
Age	15.7 ± 0.6 [14.5-16.7]	21.3 ± 0.8 [19.7-23.2]	<.001*
Gender (% female)	49%	--	
Years of Education	9.5 ± 0.5 [9.0-10.0]	12.8 ± 0.9 [11.0-16.0]	.25
Ethnicity/Race			
<i>Caucasian</i>	61%	--	--
<i>Black</i>	13%	--	--
<i>Hispanic</i>	17%	--	--
<i>Asian</i>	3%	--	--
<i>Other</i>	6%	--	--
Mother's Highest Education			
<i>No more than high school</i>	28%	--	--
<i>Some college</i>	14%	--	--
<i>College degree or higher</i>	49%	--	--
<i>Unknown</i>	9%	--	--
Father's Highest Education			
<i>No more than high school</i>	35%	--	--
<i>Some college</i>	12%	--	--
<i>College degree or higher</i>	38%	--	--
<i>Unknown</i>	15%	--	--
Body Mass Index (BMI)	25.3 ± 6.2 [16.6-45.8]	--	--
Number of adult smokers in household	0.7 ± 0.8 [0-3]		
How often around friends while they're smoking			
<i>Not at all</i>	7%	--	--
<i>A little</i>	29%	--	--
<i>A lot</i>	64%	--	--
How often around people while they're smoking			
<i>Not at all</i>	2%	--	--

	Baseline (n= 69) M ± SD [Range]	5 Year (n= 69) M ± SD [Range]	p-value
<i>A little</i>	44%	--	--
<i>A lot</i>	54%	--	--
Beck Depression Inventory (BDI)	10.3 ± 7.0 [0-28]	--	--
Beck Anxiety Inventory	10.2 ± 8.4 [0-39]	--	--
Number of Days in Past Week Exercised for 20 Minutes and Sweat	4.5 ± 2.2 [0-7]	--	--
Number of Days in Past Week Exercised for 30 Minutes Without Sweating	3.6 ± 2.3 [0-7]	--	--
Number of Days in Past Week Exercised to Strengthen or Tone	3.7 ± 2.3 [0-7]	--	--
Cigarette Use			
Smoking Rate in the Past Month	4.1 ± 3.4 [0.5-15]	6.4 ± 6.6 [0-20]	.95
Frequency of Past Month Cigarette Use	18.0 ± 10.2 [1-29]	20.2 ± 12.0 [0-30]	.21
Total Number of Cigarettes in Past Month	96.0 ± 107.2 [0.5-450]	192.6 ± 196.7 [0-600]	.87
% >100 Total Cigarettes in Lifetime	75%	96%	--
Modified Fagerstrom (mFTQ)	2.7 ± 1.4 [0-6]	3.1 ± 1.6 [0-7]	.02*
Age of Initiated Use	12.2 ± 1.8 [8-15]	--	--
Respiratory Sinus Arrhythmia (RSA; ln (ms)²)			
Pre-Smoke	6.8 ± 1.3 [3.1-9.4]	--	--
Post- Smoke	5.2 ± 1.6 [0.9-8.6]	--	--
Pre-Post Smoke Change	1.6 ± 1.4 [-1.5-6.0]	--	--

Note. "ln (ms)²", natural logarithm of heart period variance and reported in units of ms squared;

***, $p < .05$.

Table 2

Hierarchical Regression Models Predicting How Baseline RSA is Related to Cigarette Use at 5-years

Variable	<u>Pre-Smoke RSA</u>			<u>Post-Smoke RSA</u>			<u>Pre-Post Smoke Change in RSA</u>		
	R^2	β	p	R^2	β	p	R^2	β	p
5 Year Frequency of Past Month Cigarette Use (sqrt)									
<i>Block 1- RSA</i>	0.02	-.013	.30	0.02	-.012	.34	0.01	0.03	.84
<i>Block 2- RSA</i>	0.03	-.013	.29	0.06	-.007	.57	0.06	-.001	.91
Gender		0.10	.42		0.12	.32		0.12	.34
Baseline # Days in Past Week Exercised to Strengthen/Tone		--	--		-.020	.13		-.022	.09
Baseline Frequency of Past Month Cigarette Use		0.06	.62		0.06	.62		0.08	.55
5 Year Past Month Smoking Rate (sqrt)									
<i>Block 1- RSA</i>	0.10	-.032	.008*	0.04	-.021	.08	0.01	-.004	.78
<i>Block 2- RSA</i>	0.26	-.028	.018*	0.25	-.011	.37	0.25	-.011	.36
Gender		0.14	.20		0.17	.14		0.15	.20
Race/Ethnicity		0.21	.09		0.21	.10		0.24	.05
Age of Initiated Use		-.012	.28		-.017	.14		-.015	.19
Baseline # Days in Past Week Exercised to Strengthen/Tone		--	--		-.019	.12		-.023	.05
Baseline Past Month Smoking Rate		0.18	.14		0.16	.19		0.19	.13

Note. RSA, Respiratory Sinus Arrhythmia; sqrt, square root transformation;

, $p < .05$. Pre-smoke RSA refers to RSA measured prior to smoking one cigarette, post-smoke RSA refers to RSA measured after smoking one cigarette, and pre-post smoke RSA refers to the difference between pre-smoke RSA and post-smoke RSA (post-smoke RSA minus pre-post RSA).