Are smoking and passive smoking related with heart rate variability in male adolescents?

O tabagismo e o fumo passivo estão relacionados com a variabilidade da frequência cardíaca em adolescentes homens?

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ABSTRACT

Objective: To analyze the relation between smoking and passive smoking with heart rate variability parameters in male adolescents. **Methods**: The sample consisted of 1,152 males, aged 14 and 19 years. Data related to smoking and passive smoking were collected using a questionnaire. RR intervals were obtained by a heart rate monitor, on supine position, for 10 minutes. After collecting the RR intervals, time (standard deviation of all RR intervals, root mean square of the squared differences between adjacent normal RR intervals and the percentage of adjacent intervals over 50ms) and frequency domains (low and high frequency and sympathovagal balance) parameters of heart rate variability were obtained. **Results:** No significant differences between smoker and nonsmoker adolescents were observed in heart rate variability parameters ($p>0.05$). Similarly, heart rate variability parameters did not show significant difference between exposed and not exposed to passive smoking ($p>0.05$). **Conclusion**: Cigarette smoking and passive smoking are not related to heart rate variability in adolescence.

Keywords: Smoking; Tobacco smoking pollution; Heart rate; Adolescent; Questionnaires

RESUMO

Objetivo: Analisar a relação entre o tabagismo e o fumo passivo com os parâmetros da variabilidade da frequência cardíaca em adolescentes do sexo masculino. **Métodos:** A amostra foi composta por 1.152 adolescentes, do sexo masculino, com idade entre 14 e 19 anos. Dados referentes ao hábito de fumar e ao fumo passivo foram coletados por questionário. Os intervalos RR foram obtidos por meio de um cardio frequencímetro na posição supina durante 10 minutos. Após a coleta dos intervalos RR, os parâmetros da variabilidade da frequência cardíaca do domínio do tempo (desvio padrão de todos os intervalos RR, raiz quadrada da média do quadrado das diferenças entre os intervalos RR normais adjacentes e percentagem dos intervalos adjacentes com mais de 50ms) e da frequência (bandas de baixa e alta frequência e balanço simpatovagal) foram obtidos. **Resultados:** Os parâmetros da variabilidade da frequência cardíaca entre adolescentes tabagistas e não tabagistas não apresentaram diferenças significantes ($p>0,05$). Da mesma forma, a comparação entre expostos e não expostos ao fumo passivo em relação aos parâmetros da variabilidade da frequência cardíaca também não apresentou diferenças significantes ($p>0,05$). **Conclusão:** O tabagismo e o fumo passivo não estão relacionados com a variabilidade da frequência cardíaca na adolescência.

Descritores: Hábito de fumar; Poluição por fumaça de tabaco; Frequência cardíaca; Adolescente; Questionários

INTRODUCTION

Heart rate variability (HRV) is a non-invasive measurement method based on the analysis of the variation in time of successive heart beats (RR intervals) designed to evaluate the integrity of the autonomic nervous system.[1] Several studies demonstrated that a low HRV is both indicative of autonomic dysfunction and can be associated to several chronic diseases found in different population subgroups, including obesity and hypertension.[2-5]

In adults, smoking is known as a risk factor for hypertension. Previous studies demonstrated the harmful effects of smoking on HRV in adults,[6-10] suggesting that

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a decrease in HRV is one of the various mechanisms causing hypertension in smokers.[8] In fact, in adults, the decrease in HRV seems to precede the development of this disease.[2,4] However, the association of smoking and the development of cardiovascular diseases at the early stages of life is still not well known. Studies with adolescents failed to confirm an association between smoking and high blood pressure.[11-13] Nevertheless, it is possible that this risk behavior affects HRV, triggering the development of hypertension in adults.

It is interesting that the only three studies that evaluated the effects of smoking on HRV in adolescents obtaining inconclusive results due to both the limited sample size[14,15] and design.[15,16] Moreover, another important gap in these analyses has been the association between passive smoking and HRV considering that, just like smoking, passive smoking also affects the health of children and adolescents, and strongly influences the development of cardiovascular diseases later in the life.[17,18]

Considering that smoking is a risk behavior seen in 3% to 15.8%[19] of Brazilian adolescents, especially in the male population, studies aiming to evaluate the effects of both smoking and passive smoking on the HRV of adolescents need to be based on more representative samples in order to provide the subsidy needed to design health promotion policies aimed at this age group.

OBJECTIVE

To study the association between both smoking and passive smoking on the HRV of male adolescents.

METHODS

Study sample and design

This study is part of a cross-sectional statewide epidemiologic research project on students entitled "The practice of physical activities and health-related risk behaviors of high-school students living in the state of Pernambuco: a temporal trend study (2006-2011).

The target population included male adolescents aged between 14 and 19 years attending public high schools in the state of Pernambuco. To ensure the accuracy of HRV measurements, the following exclusion criteria were considered: adolescents who had ingested caffeine-based drinks 12 hours before assessment, had made use of alcohol or any form of tobacco and/or illegal drugs, had engaged in physical exercise 24 hours before assessment, and adolescents with *diabetes mellitus*, cardiovascular diseases and neurological or mental impairment.

Ethical considerations

This study was approved by the Ethics Committee on Research with Human Beings of the *Universidade de Pernambuco* (CAAE-0158.0.097.000-10). To be eligible to participate in the study, adolescents aged between 18 and 19 years old had to sign an Informed Consent Form, while the parents or guardians of adolescents fewer than 18 years old were asked to sign an Parental Passive Consent Form provided their sons had also agreed to participate in the study.

Demographic characteristics, level of physical activity, smoking and passive smoking and anthropometric assessment

To collect data on the demographic characteristics, level of physical activity and the amount of both active and passive smoking, an adapted version of the Global School-Based Student Health Survey, which was designed and validated for adolescents, was used. The adapted version is used in epidemiological studies.[20-22]

Data on sex, age, place of living (urban or rural area), skin color and school shift were collected. The level of regular physical activity was assessed by asking the following question: "In a typical week, how many days are you physically active for at least 60 minutes a day?" Adolescents who reported having exercised less than five times a week were considered not sufficiently active.[23]

The following question related to active smoking was asked: "In the last 30 days, how many days did you smoke?", and adolescents who had smoked one day in the last 30 days were defined as smokers.[22] To further investigate the issue, participants were also asked about the frequency of use: "In the last 30 days, how many days did you smoke cigarettes?" The answers were up to 10 days and 10 days or over in a month. The question about passive smoking was: "In the last 7 days, how many days did someone smoke in your presence?", and subjects who reported having been in the presence of someone who smoked at least one day a week were considered to have been exposed.

The anthropometric data of body mass and height were collected. Using an automatic scale with a precision error of no more than 0.1 kg, subjects were weighed without any shoes and coats on. Height was measured using a wooden stadiometer with a precision error of no more than 1cm. The body mass index was calculated based on the quotient between body mass and square of the height (kg/m^2).

Heart rate variability analysis

Prior to measuring the HRV, adolescents were asked if they had followed the recommendations. After

answering the questions, eligible subjects were taken to a silent room at the school where their HRV was measured. For measurement purposes, adolescents remained at rest in supine position for 10 minutes, during which time the RR intervals were captured using a heart rate monitor (Polar model RS800CX, Polar Electro Oy Inc., Kempele, Finland). However, to be considered as valid, a signal had to remain stationary for at least 5 minutes.

RR intervals were then exported to the Kubios HRV Analysis Software 2.0 for Windows (The Biomedical Signal and Medical Imaging Analysis Group, Department of Applied Physics, University of Kuopio, Finland), which performed time and frequency domain analyses. This software was ran by only one duly trained investigator who was blind to the other study variables.

Time domain measures, standard deviation of all NN intervals (SDNN), root mean square of the successive differences (RMSSD), and the percentage of the adjacent intervals higher than 50ms (PNN50) were calculated.[1]

Frequency domain measures were obtained employing spectral HRV analysis. Stationary periods of at least 5 minutes as recorded by the tachogram were divided into low frequency (LF) and high frequency (HF) ranges using a self-regressive model following order 12 of the Akaike criterion. Frequencies between 0.04 and 0.4Hz were considered physiologically significant, where the LF component was represented by oscillations between 0.04 and 0.15Hz and the HF component between 0.15 and 0.4Hz. The power of each spectral component was calculated in normalized terms (un). Normalization was performed by dividing the power of each range by the total power, from which the very LF band value (<0.04Hz) was subtracted, and the result was then multiplied by 100. The normalized LF and HF ranges were considered as the predominantly sympathetic and the parasympathetic modulations of the heart, respectively, and the ratio between these two ranges (LF/HF) as the cardiac sympathovagal balance.[1]

Statistical analysis

The data was tabulated with EpiData 3.1 software program using the double entry method to identify and correct possible typos. All typos were identified and corrected based on the questionnaires. Next, the data was exported to the Statistical Package for the Social Sciences software program, version 20, which ran the statistical analyses.

Intraobserver variability was measured by calculating the intraclass correlation coefficient. One single analyst studied the 27 HRV signals twice (data from these adolescents was not used in other study analyses) without, however, identifying the signals.[24] Normal HRV parameters were tested using the Kolmogorov-Smirnov test, while variance homogeneity was tested using the Levene test. The Mann Whitney U test was used to compare HRV parameters of adolescents who had engaged in active smoking or were exposed to passive smoking with those who had not or were not exposed. This same test was also employed to compare HRV parameters of adolescents who smoked cigarettes more than 10 days a month and those who smoked cigarettes fewer than 10 days a month. The effect size was calculated to estimate the magnitude of the differences between smokers and non-smokers. Because HRV parameters in the time domain (SDNN, RMSSD and PNN50) and frequency domain (LF, HF and LF/HF) shared interdependent variables, Bonferroni adjustment was utilized.[25] The data is presented as median and interquartile range.

RESULTS

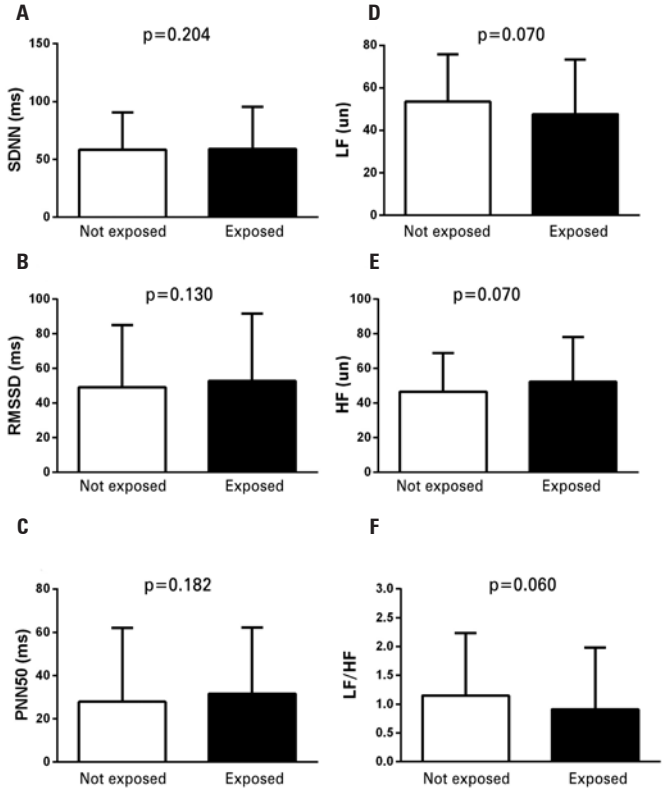
The intraclass intraobserver correlation coefficient ranged between 0.982 and 1.00.[24] In total, 1,212 adolescents participated in the study, but 60 had to be excluded due to the low quality of their HRV signal (stationary period under 5 minutes). Therefore, the final sample comprised 1,152 adolescents with a mean age of 16.6±1.2 years. Table 1 shows the demographic characteristics of the adolescents included in the study who were both smokers and non-smokers. Out of 1,152 adolescents, 66 (5.7%) referred to be smokers. And theywere older as compared to non-smokers ($p<0.05$), while no significant differences in skin color, place of living, school shift, level of physical activity and excess weight were observed ($p>0.05$).

Figure 1 illustrates the comparison between HRV parameters among adolescents who smoked and those who did not.

No significant differences were observed between adolescents who had been exposed to tobacco and those who had not in any of the variables (SDNN: 58.4±32.1ms *versus* 59.1±36.4ms; p=0.204; RMSSD: 49.2±35.9ms *versus* 52.9±38.9ms; p=0.130; PNN50: 27.9±34.2% *versus* 31.6±30.8%; p=0.182; LF: 53.4±22.4un *versus* 47.6±25.8un; p=0.070; HF: 46.6±22.4un *versus* 52.4±25.8un; p=0.70; LF/HF: 1.15±1.08 *versus* 0.91±1.07;

Table 1. Demographic characteristics of adolescents (n=1,152)

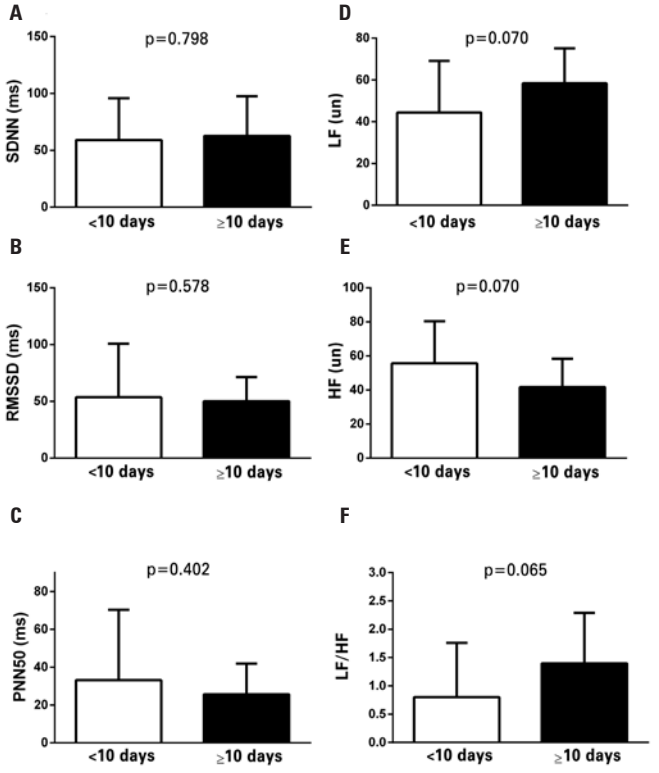
Variable	Smokers n (%)	Non-smokers n (%)	p value
Age group (years)			0.025
14-16	23 (4.2)	519 (95.8)	
17-19	43 (7.1)	563 (92.9)	
Skin color			0.490
Caucasian	19 (5.9)	301 (94.1)	
Non-caucasian	47 (5.7)	776 (94.3)	
Place of living			0.355
Rural area	12 (5.0)	227 (95.0)	
Urban area	54 (6.0)	853 (94.0)	
School shift			0.118
Day	44 (5.2)	802 (94.8)	
Evening	22 (7.3)	280 (92.7)	
Physical activity level			0.213
Active	20 (4.9)	388 (95.1)	
Not sufficiently active	46 (6.2)	691 (93.8)	
Overweight			0.412
Yes	12 (6.3)	178 (93.7)	
No	54 (5.6)	902 (94.4)	



SDNN: standard deviation of all RR intervals; LF: low frequency range; RMSSD: root mean square between adjacent normal RR intervals; HF: high frequency range; PNN50: percentage of adjacent intervals longer than 50ms; LF/HF: symphatovagal balance.

Figure 1. Comparison of heart rate variability parameters between adolescents who smoked and those who did not

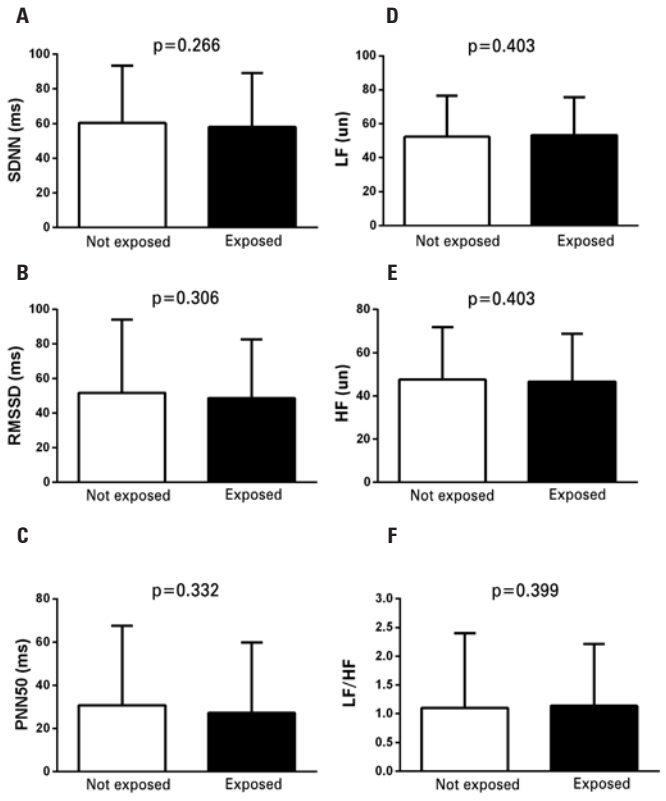
p=0.060) and the effect size of all the values ranged between 0.00 and 0.09. Among smokers, there was no difference between any HRV parameters and the use of tobacco in a month *i.e.*, <10 days *versus* ≥10 days (SDNN: 59.0±36.8ms *versus* 62.7±34.9ms; p=0.798; RMSSD: 53.5±47.2ms *versus* 50.1±21.2ms; p=0.578; PNN50: 33.2±37.2% *versus* 25.7±16.2%; p=0.402; LF: 44.4±24.8un *versus* 58.4±16.8un; p=0.070; HF: 55.6±24.8un *versus* 41.6±16.8un; p=0.070; LF/HF: 0.065) (Figure 2).



SDNN: standard deviation of all RR intervals; LF: low frequency range; RMSSD: root mean square between adjacent normal RR intervals; HF: high frequency range; PNN50: percentage of adjacent intervals longer than 50ms; LF/HF: symphatovagal balance.

Figure 2. Comparison between heart rate variability parameters of adolescents who smoked on more or fewer than 10 days/month

In terms of passive smoking, 73.4% referred that they had been exposed at home or when in the company of friends at social occasions. There were no significant differences, however, in the HRV parameters of adolescents who had been exposed to passive smoking and those who had not (SDNN: 58.1±31.1ms *versus* 60.3±33.1ms; p=0.266; RMSSD: 58.1±31.1ms *versus* 48.9±33.9ms; p=0.306; PNN50: 27.3±32.7% *versus* 30.8±36.6%; p=0.332; LF: 53.3±22.2un *versus* 52.3±24.2un; p=0.403; HF: 46.7±22.2un *versus* 47.7±24.2un; p=0.403; LF/HF 1.14±1.07 *versus* 1.10±1.13; p=0.399) (Figure 3).



SDNN: standard deviation of all RR intervals; LF: low frequency range; RMSSD: root mean square between adjacent normal RR intervals; HF: high frequency range; PNN50: percentage of adjacent intervals longer than 50ms; LF/HF: symphatovagal balance.

Figure 3. Comparison between heart rate variability parameters of adolescents who were exposed to passive smoking and those who were not

DISCUSSION

This was the first population-based study designed to investigate the association between HRV and both smoking and passive smoking among adolescents. Another strength of this study is that the methodology used to collect HRV parameters was carefully selected so as to include only adolescents who had gone at least 24 hours without physical exercise or alcohol and cigarettes, and 12 hours without ingesting caffeine. In other words, investigators took special care to minimize the influence of said substances which strongly affect HRV.[9,26,27] Also methodologically relevant was the fact that subjects were required to rest for at least one hour prior to data collection. Moreover, because the HRV was analyzed by only one investigator who had been blinded to all other study variables, results are highly reproducible and reliable.

HRV parameters were similar when smokers were compared to non-smokers. This was unexpected considering that it is a well-known fact that nicotine and carbon monoxide found in cigarettes may lead to release of noradrenalin and adrenalin,[28] which, in turn, increase the activation of the sympathetic nervous system, thereby reducing HRV and increasing heart rate and blood pressure even at rest.[29,30] Nevertheless, upon review of the literature, we found that Henje Blom et al. found similar results.[14] In their study with 71 adolescents aged 15 and 17 years old, they could not identify any association between smoking and HRV parameters. However, Baructu et al.,[15] showed in their study with 24 young smokers and 22 young non-smokers that, in smokers, SDNN and RMSSD values had come down while LF/HF values had gone up. The main reason that our findings were different from those of the Baructu[15] study is that the latter did not control for the variables that strongly affect HRV, including cigarette smoking.[9,31] Moreover, there were important differences between our subjects and those of the Baructu study, which included young people who had been smoking an average of 29 cigarettes a day, for approximately 11 years, whereas our study considered the number of days in a month on which the subject had smoked, as well as the number of cigarettes that were smoked in the previous 30 days.[22]

Regarding the HRV parameters according to the number of days in a month on which cigarettes were smoked, no significant difference was seen among the adolescents. To date, the only study that actually verified a lower HRV in adolescents who smoked more than 10 cigarettes a day was conducted by Kupari et al.[6] In their study, Kupari et al. analyzed the association between frequency of smoking and HRV in adults. This discrepancy suggests that HRV seems to be more affected by the number of cigarettes smoked than by the total number of days of smoking. This hypothesis, however, requires further testing.

The present study found that the prevalence rate of adolescents who had been exposed to passive smoking reached 73.4%, revealing a high exposure rate compared to other studies with prevalence rate ranging between 36% and 53%.[32,33] It is possible that this difference results from the different criteria employed to measure exposure to passive smoking. While other studies considered only exposure at home, this study considered exposure at home and at social gatherings.

Similarly to previous studies with adult populations,[31,34,35] no significant association was found between HRV parameters and passive smoking. It is worthwhile mentioning that this study did not control for the number of either hours per day or years of passive exposure, a fact which may partly explain our results. In fact, studies that demonstrated an association between passive smoking and HRV analyzed adults who had been exposed daily to at least 2 hours passive smoking.[34,35]

Moreover, because these subjects were adults, it is possible that they had already been exposed to passive smoking for many years, a fact which may be crucial when trying to establish an association between passive smoking and HRV indicators.

In practical terms, however, the results of this study should not lead to the misinterpretation that both smoking and passive smoking do not constitute risk factors for cardiovascular diseases in adolescents, but rather to the interpretation that exposure to these factors does not negatively affect the HRV parameters of this population group. This may be attributed to the time of exposure to both smoking and passive smoking, which was possibly shorter than the time needed to cause changes to the autonomic nervous system. In a way, our results may explain the reason why the literature,[11-13] has not been able to establish an association between smoking and high blood pressure, considering that a direct association between a smaller HRV and the development of high blood pressure has been established.[8]

Study limitations

When interpreting the results of this study, certain limitations should be taken into account. For example, no evaluation of the maturational stage was performed and, therefore, no analysis of this potential confounding factor was carried out. Notwithstanding this, because of the homogeneous nature of the study sample, we may presume that the adolescents were at similar maturational stages. Also, considering that it is a well-established fact that adolescents tend to underreport their smoking habits, a reporting bias may be present. Self-reporting on the use of tobacco at this age is, therefore, not entirely reliable, resulting in a small sample of smokers. The daily and annual frequencies of the use of tobacco, as well as time of exposure to passive smoking were not measured. Furthermore, it is possible that a large number of adolescents who smoke were unable to abstain from smoking during the 24-hour period prior to evaluation, a fact which automatically excluded them from the study.

CONCLUSION

In conclusion, no association was found between smoking or passive smoking and HRV in adolescents, suggesting that any eventual active or passive exposure to smoke does not lead to early changes in autonomic modulation.

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