Submitted: 31 July, 2023 Accepted: 04 September, 2023 Published: 30 December, 2023 DOI:10.22514/jomh.2023.136

ORIGINAL RESEARCH

JOMH Journal of Men's Health

Association between resting heart rate and pulmonary function in adult men

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Abstract

Elevated resting heart rate (RHR) is known to be associated with increased mortality in patients with chronic obstructive pulmonary disease (COPD) whose pulmonary function is reduced. On the other hand, no study has reported on the association between RHR and pulmonary function in healthy individuals without COPD. Thus, this study investigated the association between RHR and pulmonary function in healthy adult males. This study was conducted on 3351 adult males aged 30–60 years, who visited the National Fitness Center (located in Seoul, South Korea) for health examination from Jan 2015 to Dec 2017. The RHR uses data measured using a standard 12-lead electrocardiogram, in the interquartile range (>60 bpm, 60–69 bpm, 70–79 bpm, ≥80 bpm). To test the pulmonary function, a spirometer was used to measure the forced vital capacity (FVC) and forced expiratory volume in the first second (FEV1) and the FEV1/FVC. The diagnostic criterion of COPD was FEV1/FVC ≤70%. While FVC, FEV1 and FEV1/FVC showed no significant variation according to RHR, a correlation was found between RHR and FVC ($p < 0.05$). However, no correlation was found between FEV1 and FEV1/FVC. The relative risk of COPD according to RHR showed no significant variation. RHR and pulmonary function in healthy adult males, the indicator of lung capacity, FVC, showed a significant correlation. To more accurately determine the cause-effect relationship between RHR and pulmonary function, a prospective cohort study should be conducted in the future.

Keywords

Adult males; Resting heart rate; Pulmonary function

1. Introduction

The human body is constantly exposed to variations in the external environment or internal conditions that cause the functional status of the cardiovascular system to deviate from the normal range. For this, the autonomic nervous system (ANS) switches on the sympathetic and parasympathetic nerves with mutual antagonistic roles to maintain homeostasis. However, if the sympathetic response far exceeds the parasympathetic response, cardiovascular disease may result. This is because parasympathetic nerves show a protective effect on cardiac function [1, 2].

Resting heart rate (RHR) is regulated through the sympathetic and parasympathetic nerves distributed in the sinus nodes of the heart, and it is thus used as a functional marker of the ANS. Notably, an increase in RHR upon parasympathetic hyperactivation negatively affects the heart and vascular functions [3, 4]. Numerous previous studies also reported an effect of elevated RHR on the prognosis and mortality of cardiovascular diseases [5–7].

Respiration, as the basis of vitality, is also regulated through the ANS. The main goal of respiration is to supply oxygen (O_2) to the tissues where the cells constantly require oxygen and to discharge carbon dioxide (CO_2) to maintain homeostasis. The lungs are an essential respiratory organ, and to test pulmonary function, the ability of respiration, ventilation and gas exchange are objectively evaluated [8]. Of note in pulmonary function testing is that chronic obstructive pulmonary disease (COPD) due to airflow limitation is diagnosed at FEV1/FVC ≤70% [9, 10]. Previous studies reported an association between elevated RHR with mortality in COPD [11–13]. The heart rate variability (HRV) as an indicator of the ANS activities in the cardiovascular system in healthy adults was also shown to be associated with pulmonary function [14, 15].

Thus, it may be inferred that RHR as an ANS marker is associated with pulmonary functions in healthy adults. However, no study has yet clearly identified the relationship between RHR, which is relatively easy to estimate in clinical practice, and FVC and FEV1, which indicate pulmonary functions. Therefore, the purpose of this study is to determine the association between RHR and pulmonary functions in healthy adult males.

2. Methods

2.1 Participants

This study was designed as a cross-sectional study. The participants in this study were 3351 adult males aged 30–60 years, who visited the National Fitness Center (located in Seoul, South Korea) for health examination (RHR measurement, treadmill test and pulmonary function test) from January 2015 to December 2017.

The exclusion criteria were patients with inadequate data collected from health examination and patients with medical history indicating cardiovascular or cerebrovascular disease, endocrine disease, COPD or asthma that may influence the pulmonary test results. In addition, the participants in this study were divided based on demographics into (i) the group performing no exercise and the group performing three sessions of 30 min exercise a week, for physical activity, and (ii) non-smokers and smokers, for smoking status.

2.2 Measurement variables

The procedure of this study was performed in the order of physical measurement, blood test, blood pressure test, electrocardiogram test, pulmonary test and treadmill test so as not to have a minimal effect on the results. The measurement method is as follows, and all tests were conducted in the morning. In this study, it was conducted in a fasting state for more than 10 hours, and the sleep state of the previous day was not checked. In addition, smoking was prohibited for at least 4 hours before all tests.

Subject's height and weight were measured using an automatic height measuring instrument (BSM330, Inbody, Seoul, Korea). Body mass index (BMI) was calculated as body weight (kg)/height $(m)^2$. RHR uses data measured using a standard 12-lead electrocardiogram (EKG, Cardiocare, Korea). A 12-lead EKG was recorded at rest in supine position.

Blood pressure was measured by using an electronic blood pressure monitor (FT-500R plus, Jawon, Busan, Korea) for more than 10 min to measure the systolic blood pressure (SBP) and diastolic blood pressure (DBP).

Blood tests were performed with a biochemical analyzer (Selecta XL, Vital scientific, Newton, MA, USA) after 10 hours fasting in the brachial vein. Blood analysis items include total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and fasting blood glucose.

The pulmonary function test was performed based on the Digital Computed Spirometry (1022 Digital Computed Spirometry: Sensomedic, Yorba Linda, CA, USA), and according to the standard methods in the guidelines of the American Thoracic Society and European Respiratory Society [16]. The tested items were FVC, FEV1 and FEV1/FVC. The diagnostic criterion of COPD was FEV1/FVC $\leq$0.7 [17].

The treadmill test was performed according to the Balke protocol using the treadmill and the automatic gas analyzing system (Q4500, Quinton, Bothell, WA, USA). The test was discontinued upon request by the participant showing respiratory exchange rate $\geq$1.15 and RHR $\geq$90%. The measured item was VO_2max, an indicator of cardiopulmonary fitness.

2.3 Data analysis

To analyze the general characteristics of study participants, the mean and standard deviation were estimated for the following continuous variables: physical measurements, pulmonary function, cardiovascular risk factors and cardiopulmonary fitness, while the frequency and percentage were estimated for the following categorical variables: exercise status, smoking status and drinking status. The RHR was measured in the interquartile range. To examine the variation in pulmonary function according to RHR, the Analysis of Covariance (ANCOVA) was performed with age, BMI, exercise status, smoking status and drinking status as covariates. The association between RHR and pulmonary function was analyzed through Pearson's correlation coefficient. In addition, for the cross-sectional correlation of COPD according to RHR, the odds ratio was estimated through logistic regression analysis. SPSS (version 23.0, IBM, NY, USA) was used for all statistical analyses, with the level of significance set to $p < 0.05$.

3. Results

3.1 Physical characteristics according to RHR

While FVC and FEV1 decreased and FVC/FEV1 increased according to the level of RHR, no significant difference was found. In addition, the prevalence of COPD showed no significant difference according to RHR. In contrast, the risk factors of cardiovascular disease; SBP ($p < 0.001$), DBP ($p < 0.001$), TG ($p < 0.001$), fasting blood glucose ($p < 0.001$), and VO_2max ($p < 0.001$), showed significant differences. However, no significant difference was found for BMI, TC, LDL-C and HDL-C (Table 1).

3.2 Correlation between pulmonary function and RHR

A significant negative correlation was found between RHR and FVC ($r = -0.048$, $p < 0.5$), but no correlation was found between RHR and FEV1 ($r = -0.030$) or FVC/FEV1 ($r = 0.025$) (Table 2, Fig. 1).

3.3 Relative risk of COPD according to RHR

No significant difference was found for the relative risk of COPD according to RHR. Even after adjusting for age, BMI and exercise, smoking and drinking status, no significant difference was found (Table 3).

4. Discussion

This study investigated the association between RHR and pulmonary function in healthy adult males. As a result, a negative correlation was found between RHR and FVC, but no correlation was found between RHR and FEV1 or FEV1/FVC. In addition, the relative risk of COPD according to RHR was not significant.

RHR shows a repeating pattern of periodic increase and decrease as it is reflected in the sympathetic and parasympathetic nerves of the ANS. Elevated RHR is used as a marker

TABLE 1. Differences in physical characteristics according to RHR.

RHR (bpm)	>60[a] (n = 1482)	60–69[b] (n = 1384)	70–79[c] (n = 399)	≥80[d] (n = 86)	*p*	*post hoc*
	54.01 ± 3.96	63.92 ± 2.77	73.10 ± 2.57	86.33 ± 6.04		
Age (yr)	46.23 ± 6.44	46.11 ± 6.62	46.48 ± 6.57	46.52 ± 6.76	0.749	-
Height (cm)	170.59 ± 5.50	170.28 ± 5.52	169.74 ± 5.39	169.56 ± 5.90	0.144	-
Weight (kg)	71.23 ± 8.83	71.91 ± 9.32	71.98 ± 9.28	71.18 ± 8.27	0.176	-
Exercise status (%)	702 (50.6)	532 (63.5)	124 (60.5)	29 (63.0)	0.349	-
Smoking status (%)	589 (39.7)	537 (38.8)	168 (42.1)	28 (32.6)	0.364	-
Drinking status (%)	1206 (81.4)	1112 (80.3)	297 (74.4)	67 (77.9)	0.199	-
Lung capacity						
FVC (L)	4.09 ± 0.73	4.06 ± 0.73	4.02 ± 0.70	3.95 ± 0.66	0.124	-
FEV1 (L)	3.17 ± 0.65	3.17 ± 0.61	3.13 ± 0.62	3.09 ± 0.61	0.490	-
FVC/FEV1 (%)	77.50 ± 9.47	78.23 ± 8.77	77.93 ± 8.86	78.34 ± 8.95	0.186	-
COPD status (%)	266 (17.9)	218 (15.8)	63 (15.8)	14 (16.3)	0.473	-
CAD risk factors						
Body mass index	24.45 ± 2.56	24.77 ± 2.71	24.96 ± 2.84	24.75 ± 2.63	0.125	-
SBP (mmHg)	121.30 ± 14.30	125.70 ± 14.58	130.26 ± 14.76	131.85 ± 13.83	0.001	a < b < cd
DBP (mmHg)	75.12 ± 10.85	78.52 ± 11.14	82.05 ± 11.56	83.10 ± 9.68	0.001	a < b < cd
T-Chol (mg/dL)	184.34 ± 32.60	185.93 ± 33.82	188.07 ± 33.86	189.15 ± 33.84	0.142	-
TG (mg/dL)	127.07 ± 62.42	138.36 ± 68.16	147.96 ± 67.63	143.24 ± 75.48	0.001	ab < cd
LDL (mg/dL)	105.42 ± 30.17	104.77 ± 31.02	105.37 ± 31.59	108.08 ± 29.10	0.773	-
HDL (mg/dL)	53.68 ± 11.15	53.58 ± 11.04	53.11 ± 11.36	52.44 ± 11.26	0.638	-
FBG (mg/dL)	86.55 ± 12.19	88.41 ± 12.83	91.52 ± 15.01	92.07 ± 17.30	0.001	ab < cd
VO_2max	42.41 ± 7.07	40.54 ± 6.63	39.30 ± 6.19	36.37 ± 6.12	0.001	a > bc > d

Data shown as Mean ± SD or n (%).
Exercise status; ≥30 min/day for ≥3 days/week, smoking status; ≥1 cigarettes daily, drinking status: ≥once/month.
RHR: resting heart rate; FVC; forced vital capacity; FEV1; forced exploratory volume in one-second percent; CAD: cardiovascular disease; SBP: systolic blood pressure; DBP: diastolic blood pressure; TG: triglycerides; HDL: high-density lipoprotein cholesterol; FBG: fasting blood glucose; VO_2max: maximal oxygen uptake; COPD: chronic obstructive lung disease; LDL: low-density lipoprotein cholesterol.
Tested by ANOVA or chi-square test.

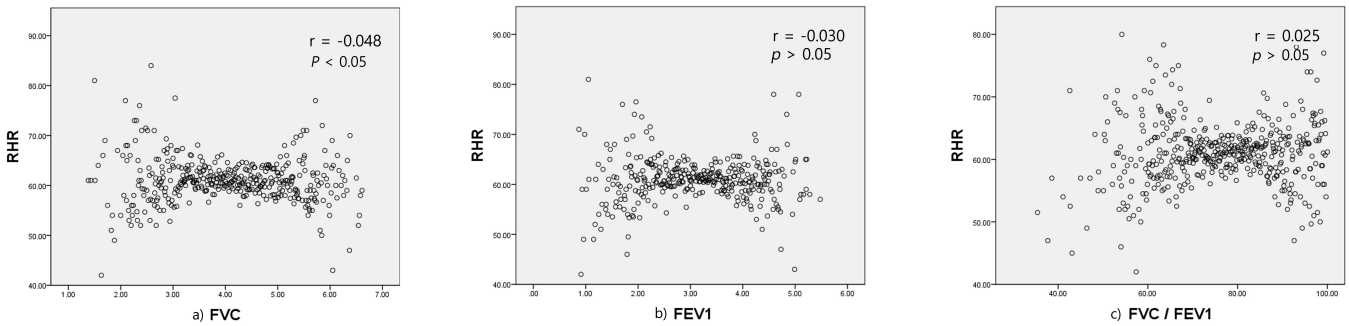

FIGURE 1. Scattergram of RHR and FVC, FEV1, FVC/FEV1. (a) RHR and FVC. (b) RHR and FEV1. (c) RHR and FVC/FEV1. RHR: rest heart rate; FVC: forced vital capacity; FEV1: forced exploratory volume in one second percent.

TABLE 2. Correlation coefficients between RHR and lung function.

	FVC	FEV1	FVC/FEV1
RHR			
r	−0.048	−0.030	0.025
p	0.028	0.172	0.253

RHR: resting heart rate; FVC: forced vital capacity; FEV1: forced exploratory volume in one second percent. Adjusted for age, body mass index, exercise, smoking, drinking.

TABLE 3. Odds ratio of having COPD according to the level of RHR.

RHR (bpm)	COPD			
	Unadjusted OR	(95% CI)	Adjusted OR	(95% CI)
>60	1		1	
60–69	1.17	(0.96–1.42)	1.01	(0.79–1.28)
70–79	1.16	(0.86–1.58)	0.92	(0.62–1.36)
≥80	1.13	(0.63–2.03)	1.79	(0.69–4.64)

RHR: resting heart rate; COPD: chronic obstructive pulmonary disease; OR: odds ratio; CI: confidence interval. Adjusted for age, body mass index, exercise, smoking, drinking.

of ANS dysfunction in the heart [18]. Like the cardiovascular system, respiration is also controlled by the ANS. Among the items of the pulmonary function test that functionally evaluates the respiratory process, FVC and FEV1 reflect the lung capacity and pulmonary obstruction, respectively. Notably, FEV1/FVC is useful in the determination of COPD caused by nonreversible airflow limitation [19]. The ANS dysfunction of the respiratory system has been shown to cause airway obstruction due to tracheal stenosis and excessive mucosal secretion [20, 21].

In this study, the RHR was measured using an electrocardiogram in the interquartile range for adult males without COPD. The result showed that FVC and FEV1 decreased as RHR increased with no significant variation. On the other hand, there was a significant difference in SBP, DBP, TG, FBG and VO_2max, which are risk factors for cardiovascular disease (CAD) according to RHR level. Previous studies [22, 23] have also reported that a high resting heart rate affects the risk of CAD, which is consistent with the results of this study.

For the association between RHR and pulmonary function, a significant negative correlation was found between RHR and FVC, whereas no correlation was found between RHR and FEV1 or FEV1/FVC. These results can be seen as a decrease in FVC as the RHR increases. However, there is no close relationship between RHR and airway obstruction. With different RHR measurement methods, Bianchim *et al.* [14] reported that, in the heart rate variability (HRV) test of the ANS activity in healthy adults, the mean HR and FVC and the HF as a high-frequency indicator of parasympathetic activity and FVC and FEV1 showed negative correlations. On the contrary, the LF as a low-frequency indicator of sympathetic activity and FVC and FEV1 showed a positive correlation. Sperandio *et al.* [15] also reported a significant correlation between HRV and pulmonary function in adults. These results partially agree with this study. However, the key difference is that, while the RHR is an indicator of cardiac activity based on electrocardiography, the HRV reflects the ANS activity by measuring and quantifying the temporal variation of the cardiac cycle. Thus, this study is significant in providing the basic data regarding the association between pulmonary function and RHR as an ANS marker that can be relatively easily estimated in clinical practice, while it is difficult to accurately assess the level of ANS dysfunction in the respiratory system. Notably, the inverse relationship between RHR and FVC as shown in this study may highlight the importance of RHR regulation in enhancing pulmonary function.

Meanwhile, Ricci *et al.* [24] through a cohort study with a 32-year follow-up monitoring, reported an association between elevated RHR in the initial examination and future incidence of COPD with decreased FVC and FEV1. The risk factors of COPD have been reported to include smoking, air pollution and occupational exposure to hazardous materials that increase oxidative stress [25, 26]. The diagnostic criterion of COPD was set as FEV1/FVC ≤70% in this study to determine the relative risk of COPD according to the level of RHR [17]. The result showed that, after the adjustments for age, BMI and smoking/drinking/exercise statuses, the risk of COPD showed no significant difference according to the level of RHR. This indicated that the RHR did not influence the incidence of COPD. However, there is almost no large-scale epidemiological study reporting a direct correlation between elevated RHR and the incidence of COPD in healthy adults. Moreover, the number of participants in this study showing RHR ≥80 bpm was relatively small. Thus, to accurately identify the role of RHR in pulmonary functions and the incidence of COPD, an additional study with a greater sample size should be conducted.

The limitations of this study were as follows. First, as a cross-sectional study analyzing the data of a single, regional health examination institution, this study could not clearly define the cause-effect relationship between RHR and COPD. In addition, as the participants in this study were adult males who received health examinations in the institution, it is difficult to generalize the results. Notably, the factor of occupational exposure to hazardous materials that may affect pulmonary functions could not be considered in this study. In addition, while the standard diagnostic criterion of COPD involves the use of a bronchodilator, the criteria in this study relied on data that did not report on bronchodilator measurements. Nevertheless, this study has a significant contribution in determining the association between RHR and pulmonary functions in healthy adults.

5. Conclusions

RHR in healthy adults was shown to be correlated with the FVC as an indicator of lung capacity among the items of the pulmonary function test. However, no correlation was found between the FEV1 as a useful indicator of airway obstruction and FEV1/FVC. Thus, a greater number of studies should be conducted to accurately define the role of RHR as an ANS marker related to pulmonary functions.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

SJK, STP and KJK—designed the study and the methodology. KJK and GCH—provided the software, contributed to the resource and data curation, supervised the study. STP, GCH and CGY—contributed to the formal analysis, revised the manuscript. SJK, STP, GCH and CGY—contributed to data curation. SJK—drafted the manuscript. KJK—contributed to data visualization. SJK, STP, KJK, GCH and CGY—were responsible for project administration. All authors contributed to the editorial changes in the manuscript. All authors have read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was approved by the Ethics Committee of the Changwon National University, Changwon-si, Republic of Korea (No. 1040271-201816-HR-013) and conformed to the standards set by the latest revision of the Declaration of Helsinki. Written informed consent was obtained from all study participants.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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How to cite this article: Seol-Jung Kang, Seung Tae Park, Kwang-Jun Ko, Gi-chul Ha, Cheol-Gyu Yoo. Association between resting heart rate and pulmonary function in adult men. Journal of Men's Health. 2023; 19(12): 107-112. doi: 10.22514/jomh.2023.136.