ISSN 2515-8260 Volume 9, Issue 7, Summer 2022

ORIGINAL RESEARCH

To study the elevated resting heart rate (RHR) represents a separate risk factor for mortality or only a sign of physical fitness

[1]Dr Prabhjot Singh, [2]Dr Manoranjan Kumar, [3]Dr.Gowtham Raj

[1]Assistant Professor, Department of Physiology, Government Medical College and Associated Hospital, Rajouri, J&K, India
[2]SR Medicine, Department of Medicine, Sardar Vallabhbhai Patel Hospital, Patel Nagar, Delhi, India
[3]Resident Medical Officer, Pentamed Hospital, Derawal Nagar, Delhi, India

Correspondence:
Dr Prabhjot Singh
Assistant Professor, Department of Physiology, Government Medical College and Associated Hospital, Rajouri, J&K, India
Email: dr.prabhjot7373@gmail.com

ABSTRACT

Aim: To study the elevated resting heart rate (RHR) represents a separate risk factor for mortality or only a sign of physical fitness.

Material and methods: The sample size for this research was 410 males. All men were given a clinical examination by a doctor (FG) that included a discussion of their responses to a questionnaire and a test of their aerobic capacity (VO2Max) on a bicycle ergometer. Heart rate and labour load data from a bicycle ergometer were used to calculate estimates of physical fitness using the strand nomogram. Using a manometer created by the London School of Hygiene and Tropical Medicine, BP was taken on the right arm of sitting subjects after at least 10 minutes of rest. The survey's questions on hypertension care led to the collection of useful data.

Results: An elevated resting heart rate has been linked to decreased fitness, hypertension, total cholesterol, triglyceride, and body mass index. Everyone got along, regardless of age or status. Smoking prevalence was greater among participants with lower resting heart rates than among those with higher resting heart rates. The VO2Max and HR at rest correlated extremely significantly (R=0.29, p0.001). Higher fitness levels were associated with decreased resting heart rates. Using heart rate as a continuous variable in an adjusted model, we find that for every 10 bpm beyond 60, the chance of death rises by 16%. Resting heart rate, smoking, and mortality all interacted to a nearly statistically significant degree. Resting heart rate was included as a continuous variable in the final model, and an increase of 10 beats per minute was associated with a 25% increase in risk for smokers and a 15% increase in risk for non-smokers.

Conclusion: Resting heart rate was shown to be an independent risk factor for death in this research of 410 healthy middle-aged and older people, regardless of physical fitness (VO2Max) as measured by a bicycle ergometer, recreational physical activity, or other traditional risk factors.

Keywords: resting heart rate, risk factor, mortality, physical fitness.

INTRODUCTION

Life expectancy decreases with increasing resting heart rate (RHR). [1] Epidemiological research has shown that high RHRs are associated with an increased risk of death, especially from cardiovascular disease (CVD). This correlation has been proven across a wide range of populations, from the general public to hypertensives to those with preexisting coronary artery disease (CAD). [2] Previous research has provided evidence that suggests a correlation between resting heart rate and numerous health outcomes. It has been shown that there is a significant and gradated association between the two in males. Experimentally lowering heart rate has been shown to lessen atherogenesis in animal experiments. [3] A faster heart rate will necessarily impose more shear stresses than a slow one, and this is just one of several biologically plausible mechanisms for the effect of elevated RHR. A low heart rate has been shown to have antiischemic and antiarrhythmic benefits, while an elevated heart rate has been shown to have atherogenic hemodynamic effects. There are a few discrepancies, however, that need to be resolved, especially in terms of the impact on women.

However, women were often excluded from the sample in the first investigations of the general population.[4] Only three prospective studies in female participants showed an independent association between RHR and CHD or CVD end points; in two of these studies, the association was only significant within a narrow age range. RHR was significantly associated with CHD incidence in another large prospective cohort of healthy women. However, since systolic blood pressure was not controlled for as a continuous variable, the study cannot be deemed to have shown a genuinely independent impact. [5] There is still no solid foundation for independence. After include systolic blood pressure in the model, RHR was no longer an independent significant risk factor in several of the bigger studies. [5] Despite the fact that several research found independence from exercise, only binary variables were employed. [6] Adjustment for these possible confounders has been inadequate in prior research, even though RHR elevations may be related with comorbid illnesses. Due to left ventricular dysfunction brought on by subclinical heart disease, a higher heart rate may be a result of the temporal sequence.

MATERIAL AND METHODS

The sample size for this research was 410 males. All men were given a clinical examination by a doctor (FG) that included a discussion of their responses to a questionnaire and a test of their aerobic capacity (VO2Max) on a bicycle ergometer. Heart rate and labour load data from a bicycle ergometer were used to calculate estimates of physical fitness using the strand nomogram. Using a stopwatch and a stethoscope, we determined the subjects' heart rates as they rode a stationary bicycle at a submaximal intensity. Three different loads (100 W, 150 W, and 200 W) were tested. Numerous loads, ranging from one to three, were used. In each example, the load was selected based on the subject's age, weight, and/or heart rate after the first minute of the test. The procedure used has been extensively discussed before. Seven different measurements were obtained: height, weight, and blood pressure; a venous blood sample was taken for the assessment of serum lipids; and a urine sample was taken for the determination of glucosuria. The questionnaire measured medical background, smoking, drinking, and recreational exercise. The males ranked themselves as either never smokers, ex-smokers, or current smokers. Tobacco use at the time of the survey was quantified by asking respondents how many cigarettes, cheroots, cigars, or grammes of pipe tobacco they smoked on a daily basis. Tobacco was weighed at 1 g each cigarette, 3 g per cheroot, and 4 g per cigar. Serum cotinine assays supported the conclusion that self-reported cigarette use had a high level of accuracy. The formula for determining body mass index (BMI) is as follows: 8 kg / (height in metres squared). Enzymatic assays were used to measure total cholesterol, HDL-C, and TG levels in the blood (Boehringer-Mannheim Biochemica, Mannheim,

Germany).[9] Fasting lipids were measured only once in each subject. Definitions of high serum TG (≥1.70 mmol/l), low HDL-C (≤1.03 mmol/l), high systolic BP (>130 mm Hg) and high diastolic BP (>85 mm Hg) were based on criteria for definition of the metabolic syndrome according to the Adult Treatment Panel III.[10] Low density lipoprotein cholesterol (LDL-C) was calculated using the indirect method as developed by Friedewald.[11] Urine samples were examined for glucosuria using a glucose kinase method. Using a manometer created by the London School of Hygiene and Tropical Medicine, BP was taken on the right arm of sitting subjects after at least 10 minutes of rest. The survey's questions on hypertension care led to the collection of useful data.

RESULTS

Characteristics at the outset are broken out in Table 1 by RHR groupings. An elevated resting heart rate has been linked to decreased fitness, hypertension, total cholesterol, triglyceride, and body mass index. Everyone got along, regardless of age or status. Smoking prevalence was greater among participants with lower resting heart rates than among those with higher resting heart rates. The VO2Max and HR at rest correlated extremely significantly (R=0.29, p0.001). Higher fitness levels were associated with decreased resting heart rates.

As a whole, a high resting heart rate was a reliable indicator of early death. Table 2 shows that an increased risk of death was seen in all models for participants with a higher resting heart rate (1–5). A resting heart rate between 50 and 80 beats per minute was associated with an increased risk of 42-52% in the fully adjusted model, while a resting heart rate between 80 and 90 beats per minute was associated with a risk increase of twofold, and a resting heart rate above 90 beats per minute was associated with a risk increase of threefold, relative to subjects in the lowest heart rate category (below 50 bpm).

Using heart rate as a continuous variable in an adjusted model, we find that for every 10 bpm beyond 60, the chance of death rises by 16%. Resting heart rate, smoking, and mortality all interacted to a nearly statistically significant degree. Resting heart rate was included as a continuous variable in the final model, and an increase of 10 beats per minute was associated with a 25% increase in risk for smokers and a 15% increase in risk for non-smokers.

Table 1 demographic profile

Parameter	**200**	**100**	**50**	**30**	**20**	**10**	
Resting heart rate (bpm)	48.25± 3.58	57.85±3.69	66.85±4.58	75.85±3.67	85.85±5.52	101.58±5.66	
Leisure-time physical activity (%)							
Low	8	7	10	10	15	10	<0.001
Medium	32	34	40	36.67	55	60	
High	60	59	50	53.33	30	30	
Smoking (%)	72	58	52	50	42	40	<0.001
Alcohol, beverages/week	12	12	14	16.67	15	10	0.33
Clinical and metabolic risk factors							
Systolic BP	115.85 ±10.63	118.97±12.64	121.58±13.55	126.21±12.58	129.89±11.33	133.12± 14.52	<0.001
Systolic BP >130 (%)	20	21	26	30	50	50	<0.001
Diastolic BP	66.36±	71.85±7.3	73.63±5.6	78.25±6.3	79.85±5.2	79.96±4.	<0.001

	6.58	6	3	6	5	63	
Diastolic BP >85 (%)	5	10	12	20	30	30	<0.001
Hypertension (%)	13	12	10	10	10	20	0.22
TG (mmol/l)	1.41±0.36	1.51±0.44	1.52±0.54	1.70±0.25	1.85±0.55	1.70±0.25	<0.001
HDL-C (mmol/l)	1.36±0.21	1.37±0.15	1.38±0.36	1.36±0.14	1.40±0.22	1.51±0.36	0.07
Total cholesterol, mmol/l	6.66±1.36	6.51±1.69	6.68±1.57	6.78±1.62	6.85±1.66	6.74±1.74	0.03
LDL-C (mmol/l)	4.81±1.11	4.87±1.63	4.98±1.87	4.92±1.36	4.93±1.41	4.92±1.36	0.41
Age (years)	63.58±5.69	62.58±5.96	62.85±5.99	62.58±5.25	63.58±7.63	64.55±6.36	0.04

Table 2 HRs for all-cause mortality according levels of resting heart rate

	below 50	**50–60**	**60–70**	**70–80**	**80–90**	**above 90**
HR, adjustment for						
Age	1	1.35 (1.04 to 1.74)	1.41 (1.09 to 1.82)	1.52 (1.14 to 2.01)	2.05 (1.48 to 2.83)	3.01 (2.01 to 4.51)
Age+VO_2Max	1	1.28 (0.98 to 1.66)	1.28 (0.99 to 1.67)	1.33 (0.98 to 1.79)	1.76 (1.26 to 2.46)	2.56 (1.69 to 3.87)
Age+lifestyle	1	1.46 (1.12 to 1.91)	1.58 (1.21 to 2.06)	1.67 (1.25 to 2.24)	2.45 (1.47 to 2.91)	3.38 (2.24 to 5.08)
Age+clinical factors	1	1.41 (1.08 to 1.84)	1.48 (1.14 to 1.93)	1.59 (1.19 to 2.14)	2.07 (1.47 to 1.91)	3.12 (2.06 to 4.73)
Age+all potential confounders	1	1.41 (1.07 to 1.86)	1.47 (1.11 to 1.94)	1.52 (1.12 to 2.07)	2.05 (1.44 to 2.93)	3.07 (1.98 to 4.76)

DISCUSSION

Resting heart rate was revealed to be a risk factor for death in this research of healthy middle-aged men, even after controlling for VO2Max and other significant potential confounders.

The role of resting heart rate as a predictor of mortality has garnered a lot of research interest in recent years. However, it has been questioned if an increased resting heart rate is just a proxy sign of low physical fitness, which is in turn linked with a bad prognosis. The current research also shows that a lower heart rate is related with a higher degree of physical fitness, making fitness a powerful predictor of longevity. Therefore, resting heart rate research relies heavily on participants' levels of physical fitness. After accounting for activity time, researchers in the Paris Prospective Study found that resting heart rate was still a strong predictor of mortality, and in particular, unexpected death.[12-14] However, the majority of research rely on self-reported physical activity levels or provide no data at all.[15-16] Despite the fact that all participants in the current study completed a physical exercise test, an estimate of their VO2Max, and an evaluation of their physical activity during their free time, we discovered that those with higher resting heart rates performed less well than those with

lower heart rates, regardless of their fitness levels. This indicates that a high resting heart rate is an independent risk factor and not only a measure of poor fitness.

Low heart rate groups had more smokers than high heart rate categories. Smoking causes a rapid heart rate and a rise in blood pressure (BP) almost immediately after starting, although healthy smokers are more likely to have lower BP than non-smokers, as established in a number of studies [17–19]; the same processes may explain this finding in the current research.

Firstly, it represents a counter- regulatory physiological response to the acute rise in BP and heart rate following smoking; and secondly, the healthy worker effect probably plays a role smokers in the present study are healthier, leaner, and more fit than non-smokers; they can 'endure' the smoking.

A high resting heart rate is linked with a higher risk in smokers than in non-smokers, and we discovered a marginally significant interaction between resting heart rate, smoking, and mortality. Consistent with previous research, we found that in this sample, resting heart rate was a more accurate predictor of all-cause death in smokers than in non-smokers. The fact that this correlation is true across two large-scale studies strengthens the case that it is real and not just coincidental. Since there are about 1 billion smokers in the world,[20] this finding has therapeutic implications for the monitoring and possible modification of heart rate in smokers.

The association between a high resting heart rate and mortality has been explained in several ways from a pathophysiological standpoint.[21,22] The correlation between resting heart rate and animal lifespan is well established. High resting heart rates have been linked to an increased risk of death, and research by Levine23 found that this association held true across mammalian species. High heart rate may promote the development of atherosclerosis and plaque rupture through an increase in cardiac work, decreased artery compliance, and increased arterial wall stress[24, 25]; smoking is known to promote inflammatory pathways and induce alterations in metabolism, vessel walls, haemostatics, and impaired blood flow. [26] Together, these processes may explain the correlation between this research and its results. The link between an increased resting heart rate and increased lifespan is not fully understood, although it may be clarified by studies focusing on cause-specific mortality, such as those focusing on cardiovascular causes, cancer, or other disease entities.

Prospective researchers need to think about the potential shortcomings of their study. All research participants were employed, and hence, the study population may have been healthier than the overall population due to the 'healthy worker' effect. There may have been a prejudice in favour of the survivors as well. Only participants who were able to attend both assessments, when resting heart rate and VO2Max were assessed, were included in the analysis. Because of this, the observed HRs may be lower than the real HRs, and the findings may have been skewed towards the null hypothesis.

Between the first and second evaluations, there might have been shifts in physical fitness. However, subjects were free of overt disease and survived to participate in the second examination, suggesting they were healthier than the general population. Moreover, subjects with cardiovascular disease, diabetes, or an absence of sinus rhythm were excluded, suggesting that no major health-related event would have occurred to change general fitness levels substantially. The current study's finding of a negative association between resting heart rate and fitness ($r=0.29$) is consistent with the negative correlation reported between resting heart rate and fitness in the Troms Study, which was conducted at the same time on a male population. However, misclassification of heart rate owing to a single assessment would bias the data towards the null hypothesis and cannot explain our findings since heart rate varies throughout the day. Finally, it's worth noting that our results may not necessarily apply to other demographic groups since they are based exclusively on findings among healthy, middle-aged, and elderly Caucasian males.

CONCLUSION

Resting heart rate was shown to be an independent risk factor for death in this research of 410 healthy middle-aged and older people, regardless of physical fitness (VO2Max) as measured by a bicycle ergometer, recreational physical activity, or other traditional risk factors. These findings indicate that an increased resting heart rate in healthy adults is a risk factor in its own right and not only a measure of poor overall fitness.

REFERENCES

1. Levine H. Rest heart rate and life expectancy. JACC 1997;30:1104-6.
2. Diaz A, Bourassa M, Guertin M, et al. Long-term prognostic value of resting heart rate in patients with suspected or proven coronary artery disease. Eur Heart J 2005;26:967-74
3. Beere P, Glagov S, Zarins C. Retarding effect of lowered heart rate on coronary atherosclerosis. Science 1984;226:180-2.
4. Dyer A, Persky V, Stamler J, et al. Heart rate as a prognostic factor for coronary heart disease and mortality: findings in three Chicago epidemiologic studies. Am J Epidemiol 1980;112:736-49.
5. Hsia J, Larson L, Ockene J, et al. Resting heart rate as a low tech predictor of coronary events in owmen: prospective cohort study. BMJ 2009;b219:338
6. Tverdal A, Hjellvik V, Selmer R. Heart rate and mortality from cardiovascular causes: a 12 year follow-up study 0f 379,843 men and women aged 40-45 years. Eur Heart J 2008;29:2772-81
7. Gyntelberg F. Physical fitness and coronary heart disease in Copenhagen males aged 40–59. II. Dan Med Bull 1973;20:105–12.
8. Suadicani P, Hein HO, Gyntelberg F. Serum validated tobacco use and social inequalities in risk of ischaemic heart disease. Int J Epidemiol 1994;23:293–300.
9. Stähler F, Gruber W, Stinshoff K, et al. A practical enzymatic cholesterol determination. Med Lab (Stuttg) 1977;30:29–37.
10. Stone NJ, Bilek S, Rosenbaum S. Recent National Cholesterol Education Program Adult Treatment Panel III update: adjustments and options. Am J Cardiol 2005;96:53E–9E.
11. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499–502.
12. Batty GD, Shipley MJ, Kivimaki M, et al. Walking pace, leisure time physical activity, and resting heart rate in relation to disease-specific mortality in London: 40 years follow-up of the original Whitehall study. an update of our work with professor Jerry N. Morris (1910–2009). Ann Epidemiol 2010;20:661–9.
13. Jensen MT, Marott JL, Jensen GB. Elevated resting heart rate is associated with greater risk of cardiovascular and all-cause mortality in current and former smokers. Int J Cardiol 2011;151(2):148–54. doi:10.1016/j.ijcard.2010.05.003
14. Jouven X, Empana JP, Schwartz PJ, et al. Heart-rate profile during exercise as a predictor of sudden death. N Engl J Med 2005;352:1951–8.
15. Shaper AG, Wannamethee G, Macfarlane PW, et al. Heart rate, ischaemic heart disease, and sudden cardiac death in middle-aged British men. Br Heart J 1993;70:49–55.
16. Palatini P, Thijs L, Staessen JA, et al. Predictive value of clinic and ambulatory heart rate for mortality in elderly subjects with systolic hypertension. Arch Intern Med 2002;162:2313–21.
17. Green MS, Jucha E, Luz Y. Blood pressure in smokers and nonsmokers: epidemiologic findings. Am Heart J 1986;111:932–40.
18. Primatesta P, Falaschetti E, Gupta S, et al. Association between smoking and blood pressure: evidence from the health survey for England. Hypertension 2001;37:187–93.

19. Gyntelberg F, Meyer J. Relationship between blood pressure and physical fitness, smoking and alcohol consumption in Copenhagen males aged 40–59. Acta Med Scand 1974;195:375–80.
20. WHO. Tobacco facts. WHO. http://www.who.int/tobacco/mpower/tobacco_facts/en/index.html (accessed 20 May 2012).
21. Palatini P. Heart rate as an independent risk factor for cardiovascular disease: current evidence and basic mechanisms. Drugs 2007;67(Suppl 2):3–13.
22. DiFrancesco D, Camm JA. Heart rate lowering by specific and selective If current inhibition with ivabradine. A new therapeutic perspective in cardiovascular disease. Drugs 2004;16:1757–65.
23. Levine H. Rest heart rate and life expectancy. J Am Coll Cardiol 1997;30:1104–6.
24. Leone A. Smoking, haemostatic factors, and cardiovascular risk. Curr Pharmaceutical Design 2007;13:1661–7.
25. Newby DE, Wright RA, Labinjoh C, et al. Endothelial dysfunction, impaired endogenous fibrinolysis, and cigarette smoking. A mechanism for arterial thrombosis and myocardial infarction. Circulation 1999;99:1411–5.
26. Beere PA, Glagov S, Zarins CK. Retarding effect of lowered heart rate on coronary atherosclerosis. Science 1984;226:180–2.