

Heart rate recovery in normal and obese males with and without parental history of cardiovascular disease

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ABSTRACT

Background: Parental history of cardiovascular disease (CVD) and obesity is associated with delayed parasympathetic nervous system reactivation after exercise. Heart rate recovery (HRR) after a minute of exercise is inversely related to cardiovascular events. **Aim:** To determine the effect of body mass index (BMI) and parental CVD history on HRR in apparently healthy young Indian males. **Method:** The present cross-sectional experimental study involved 100 males, aged 18–25 years. Subjects were divided into two equal groups based on the parental CVD history—(i) Parental CVD history present, and (ii) Parental CVD history absent. Each of these groups were further divided into two equal sub groups based on BMI—(a) BMI <23 kg/m², and (b) BMI ≥25 kg/m². Participants exercised on the treadmill at variable speeds and grades to achieve their target HR (THR). THR was calculated by adding 60–90% HR-reserve (HRR) in their basal HR (BHR). HRR was calculated by subtracting maximal HR (MHR) from BHR. MHR was estimated by the formula: 208–0.7 × age. The HRR was calculated by subtracting the immediate postexercise HR with the HR after a minute of rest postexercise. ANOVA with post-hoc Tukey was applied and a *P* value ≤0.05 was considered as statistically significant. **Results:** HRR value was significantly lesser in subjects having a positive parental history of CVD than the subjects with no parental history of CVD, irrespective of BMI. Also, HRR was inversely related to BMI. **Conclusion:** Not only obesity but also a family history of CVD impacts the recovery of HR after vigorous-intensity exercise.

Keyword: Body mass index, cardiovascular, family history, heart rate recovery

Introduction

Heart rate recovery (HRR) is the decrease in heart rate (HR) after 1 min (or up to 3 min) of submaximal to maximal intensity exercise cessation. HRR is a simple, reliable, easily accessible method to monitor and assess cardiovascular health and is an important predictor of all cause mortality and death associated with cardiovascular diseases (CVD). Attenuation in HRR is associated with adverse cardiovascular events.^[1]

The recovery in HR after exercise is affected more by parasympathetic reactivation than the sympathetic withdrawal.^[1,2] Autonomic nervous system dysfunction would result in attenuated HRR and predisposition to CVD.^[3] Family history of CVD affects parasympathetic reactivation.^[4] Obese individuals have vagus nerve dysfunction.^[5] Thus, we hypothesized that parental CVD history and increased BMI would attenuate the HRR.

To the best of the author's knowledge, none of the studies from India has reported the effect of obesity and family history of CVD on HRR. Hence, the present study aims to observe the effect of body mass index (BMI) and family history of CVD

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on HRRe. Furthermore, the study results would conclude that whether obesity or family history of CVD has more impact on HRRe.

Materials and Methods

Ethical clearance reference number was 71 ECM IIB Thesis/P9, December 2015. The present population-based cross-sectional study was conducted in the Department of Physiology, King George's Medical University (KGMU), Lucknow, Uttar Pradesh, India after approval from the Institutional Ethical Committee.

The diagnosed CVD patients visiting the Cardiology Department, KGMU were counseled to send their children to the Physiology Department, KGMU for HRRe test. A structured interview and systemic examination were done before the recruitment of the subject in the study. Informed written consent was taken from each participant. 'No' as an answer to all the questions in Physical Activity Readiness Questionnaire,^[6] resting HR ≤ 100 beats per minute (bpm) or blood pressure $\leq 140/90$ mmHg and a normal electrocardiogram (BPL Cardiart 108T-DIGI, India) were the inclusion criteria. Involvement in regular moderate-intensity physical activity^[7] in the past 6 months; history of any abnormalities like diabetes mellitus, valvular or congenital heart disease, hypertension, endocrine disorder, neuromuscular disorders (could have jeopardized the subject's health or study results); inability to follow exercise protocol were the exclusion criteria.

A total of 50 young (18–25 years), apparently healthy, male volunteers with a positive family history of CVD in their first-degree relative (mother, father or both) were involved in the study. Control group comprised of another 50 apparently healthy males that were recruited from KGMU with no history of CVD in the family. Subjects in both groups were matched by age and BMI. These groups were further divided into two equal sub-groups—(a) BMI: 18.5–22.9 kg/m² and, (b) BMI ≥ 25 kg/m² as per Asian-Pacific obesity classification by the World Health Organization.^[8]

Anthropometrical measurements

The subjects in minimal clothing stood still on the platform of a digital weighing machine to record the weight up to the nearest 0.1 kg. The subject stood barefoot on the platform of a rigid stadiometer with straight knees, buttocks, and shoulders touching the stadiometer, and head in Frankfort plane to record the height up to the nearest 0.1 cm. BMI was calculated by the formula: weight (kg)/height (m²).

Determination of the target heart rate

First, the maximum heart rate (MHR) was calculated by the formula proposed by Tanaka *et al.*: $208 - 0.7 \times \text{age}$ in years.^[9–11] Then, the Heart Rate Reserve (HRR) was calculated by subtracting basal heart rate (BHR) from MHR. Thereafter target HR (THR) was estimated by the equation: $(0.60 - 0.90) \times \text{HRR} + \text{BHR}$. This THR keeps the exercise intensity

in the vigorous range.^[11,12] For example, consider a 24-year-old adult who has a BHR about 75 bpm, then MHR is $208 - 0.7 \times \text{age}$, i.e. $208 - 17 = 191$. HRR is $\text{MHR} - \text{BHR}$, i.e. $191 - 75 = 116$. THR range is $0.60 \times \text{HRR} + \text{BHR}$ i.e. $0.60 \times 116 + 75 = 145$ bpm to $0.90 \times \text{HRR} + \text{BHR}$ i.e. $0.90 \times 116 + 75 = 179$ bpm.

Exercise testing and heart rate recovery

The test was done in the morning hours (9–10 AM) at least after 2 h of breakfast. One familiarization session and two trials were done, each separated by a week. Average value of the two trials was taken. All subjects were encouraged to walk on a motorized treadmill (Pro Bodyline Fitness 970) at variable speeds and grades (inclination or slope) to achieve their calculated THR. Subject walked/ran on the treadmill until achieved THR remained almost constant (± 5 bpm) for 2 min to achieve a steady-state.^[13] HR was assessed and monitored continuously before, during and after exercise test by 'Omron pulse oximeter 'MD300C20' whereas the subject was standing on the treadmill with hands placed on handrails. HRRe was calculated as the difference in the HR immediately after exercise to HR after a minute of rest postexercise.

Statistical analysis

Initial data entry was done in Microsoft Excel 2016. IBM SPSS Statistics for Windows, Version 25.0 was used to carry out further statistical analyses. Data are presented as either mean \pm standard deviation. Data have been rounded off to one decimal place. Shapiro–Wilk was used to test the normality of data. ANOVA with post-hoc Tukey was applied and a P value ≤ 0.05 was considered as statistically significant. The point-biserial correlation coefficient was calculated to correlate HRRe with a positive history of CVD in normal BMI subjects. The Pearson correlation coefficient was calculated to correlate HRRe with BMI in subjects without a parental history of CVD. Linear regression analysis was done to know the R-square value. The confidence interval was 95%. Correlation coefficient was considered mild, moderate and strong for values 0.3–<0.5, 0.5–<0.7, 0.7–<0.9, respectively.

Results

Table 1 represents the BHR, THR, and anthropometric indices of the subjects that were divided into four equal groups based on the presence or absence of parental CVD history and BMI. Subjects were comparable for age and height ($P > 0.05$) in all the groups. Weight and BMI of groups (both with and without CVD parental history) having BMI ≥ 25 were comparable ($P < 0.05$). BHR was non-significantly higher in groups with BMI ≥ 25 as compared with the groups with BMI < 23 kg/m². THR achieved after exercise was similar ($P \geq 0.97$) in all the groups.

Table 2 represents HRRe in the subjects divided into four groups. HRRe was significantly ($P < 0.05$) lowest in the group having a positive family history of CVD and BMI ≥ 25 , followed by the groups with — negative CVD history in the family and

Table 1: Characteristic of participants

	With parental CVD history (n=50)		Without parental CVD history (n=50)	
	BMI<23 (n=25)	BMI≥25 (n=25)	BMI<23 (n=25)	BMI≥25 (n=25)
Height (cm)	167.8±6.0	166.6±6.0	167.3±5.4	168.0±5.0
Weight (Kg)	58.9±5.1	77.6±6.2	59.2±4.6	79.2±6.3
BMI (Kg/m ²)	20.9 ±1.3	27.9 ±1.7	21.2±1.3	27.9±1.8
Age (years)	21.6±2.2	21.0±2.3	21.0±2.1	21.7±2.3
Basal HR (bpm)	74.7±6.5	78.3±6.1	74.1±5.3	76.2±6.1
Achieved target HR (bpm)	163.7±7.4	164.3±7.2	163.4±7.1	164.0±7.0

bpm: beats per minute

Table 2: Heart rate recovery after a minute of vigorous-intensity exercise

BMI	Family history of CVD		P
	Present	Absent	
<23	29.7±5.0	34.2±5.3	0.007
≥25	20.9±4.4	24.7±4.6	0.031
P	<0.001	<0.001	

ANOVA with post-hoc Tukey was applied

BMI ≥ 25, positive CVD history in the family and BMI < 23, and negative family history of CVD and BMI < 23, respectively.

The point-biserial correlation coefficient was 0.413 ($R^2 = 0.171$, $P = 0.003$) for the presence of parental CVD history and delayed HHRre in normal BMI subjects ($n = 50$). Pearson correlation coefficient was -0.598 ($R^2 = 0.358$, $P < 0.001$) for BMI and HHRre in subjects ($n = 50$) without a family history of CVD.

Discussion

The current study involved young and apparently healthy, normal weight and obese males with and without a family history of CVD. HHRre after 1 min of vigorous-intensity treadmill exercise test was least in the obese group with a positive family history of CVD. Thus, the family history of CVD has an additional impact on attenuating HHRre in comparison to obesity as an individual factor. Moreover, a significantly mild correlation of parental CVD history with delayed HHRre was obtained in our study result. BMI has shown a moderate negative correlation with HHRre, i.e. as BMI increases HHRre attenuates.

There are considerable inter individual differences in the extent and rate of HHRre after exercise whose mechanisms are not known exactly. However, polymorphisms in the genes encoding the autonomic nervous system (sympathetic and parasympathetic) could be a possible cause.^[14,15] Any genetic variation and inheritance of these genes in the offsprings of CVD patients could be a cause of delayed HHRre as obtained in our study results. Ingelsson *et al.*^[16] involved 2982 Framingham offspring participants^[17] to perform a submaximal exercise according to standard Bruce protocol and HHRre was accessed after 3 min of exercise cessation. Ingelsson *et al.* reported a heritability of 34% for slow HHRre. Nederend *et al.*^[18] involved 491 healthy adolescent twins and their siblings to perform a maximal intensity (till exhaustion) exercise testing and reported a heritability of

60% for HHRre after a minute of exercise cessation. Parental CVD history is an established risk factor for CVD development in offspring.^[19] However, Jha *et al.* reported that HHRre does not depends on the parental history of hypertension and concluded that the family history of hypertension is not accompanied by autonomic nervous system dysfunction.^[20] Our study results show that the 17% attenuation in HHRre can be explained by the positive family history of CVD. It is in accordance with the previous study results.^[16,18,19]

A moderate and negative correlation of BMI with HHRre found in our study is in accordance with the result reported by Barbosa *et al.*,^[21] Brinkworth *et al.*^[22], and Azam F *et al.*^[23] It was observed that a 36% change in HHRre can be explained by the presence of obesity. Barbosa *et al.* involved 2443 subjects of both genders, aged 20–59 years to analyze the effect of BMI on HHRre. They found that obese subjects (BMI > 30 kg/m²) had a higher basal HR compared with subjects with normal BMI which might be due to autonomic nervous system dysfunction.^[5] In our study results, basal HR was non-significantly higher in the obese group. Barbosa *et al.* concluded that higher BMI causes a delay in HHRre. Brinkworth *et al.* reported that weight loss improves the HHRre postexercise which further strengthens our study results. Azam F *et al.* involved 64 healthy males, aged 25–55 years and reported that the BMI is significantly and strongly correlated with slow HHRre.

The prevalence of CVD is rising in India.^[24,25] Family history has not been given much importance in traditional risk prediction tools. However, parental CVD history is particularly important in younger individuals that do not yet present with traditional risk factors.^[26] Thus, CVD patients visiting the hospitals must be counseled to have their children assessed for HHRre. Also, an epidemic of obesity has been established in the Indian youth.^[27-29] HHRre monitors cardiovascular health easily and can segregate high-risk individuals. Exercise restores sympathovagal balance^[30] and by prescribing an appropriate exercise protocol like eccentric exercises^[6] to these high-risk individuals would decrease the future risk of CVD morbidity and mortality and enhance the present health-related quality of life.

Limitation of Study

The study involved only young males hence results might not apply to the general population. However, as the Indian

youth (15–34 years) constitutes around 35% population of the country^[25] and recent data show that India is the third most obese country in the world^[26] with the second-highest number of obese children (<20 years of age),^[27] our study results become important. Only males were included in the study to maintain the homogeneity of data.^[31]

A convenient sample size was taken in our study rather than calculating it. However, on calculating the sample size retrospectively it was found that 16 subjects were required per group for 5% alpha error and 80% power of the study. This calculation was done by using G*Power calculator v3.1.9.4.^[32] The effect size f was calculated as 1.062 from partial $\eta^2 = 0.53$ (sum of squares for between groups in ANOVA for HRRe was 2541.88 and total sum of squares was 4791.56; $\eta^2 = \text{sum of squares for between groups} \div \text{total sum of squares}$).

Standardized exercise protocol should be used to estimate MHR rather than calculating it by various available equations. However, owing to limitation of resources we have to calculate the MHR by equation by Tanaka *et al.* Future studies at the molecular level with larger samples, which can directly explore the heritability of delayed HRRe in individuals with a positive family history of CVD are necessary for external validation of our study results.

Conclusion

Not only obesity but also a family history of CVD impacts the recovery of HR after exercise. Obese offspring of CVD patients are at a higher risk of CVD development than obese individuals with negative parental CVD history or normal BMI offspring of CVD patients. Delayed HRRe has been associated with poor cardiovascular health. Hence, obese subjects and offspring of CVD patients should be counseled to take HRRe test. HRRe could prove to be an effective tool in reducing CVD disease burden in the country.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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