National Journal of Physiology, Pharmacy and Pharmacology

RESEARCH ARTICLE

Effect of body mass index on post-exercise hypotension in healthy adult males

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Received: July 20, 2018; **Accepted:** August 07, 2018

ABSTRACT

Background: Post-exercise hypotension (PEH) is a physiological phenomenon of fall in blood pressure (BP) from basal value following exercise. Therefore, it can be used as a physiological method to treat hypertension. Obesity is known to be associated with hypertension. However, the association between body mass index (BMI) and PEH is unknown. **Aims and Objective:** The aim and objective are to compare the maximum PEH between healthy adult males with normal BMI and those with above normal BMI. **Materials and Methods:** Sixty healthy, young male participants with normal BMI (18.5–24.9) and BMI above >25 kg/m^2 (n = 30 in each group) were recruited. After recording basal heart rate and BP, the participants were made to walk on a treadmill at 50% of their VO_2 max for 20 min. Six post-exercise BP values at 5-min intervals were recorded. Data were analyzed using Mann–Whitney U-test. **Results:** The maximum systolic PEH was significantly greater ($P < 0.01$) in normal BMI group (median = 7 vs. 5) than the higher BMI group. However, the maximum post-exercise diastolic PEH was comparable between the groups. Spearman correlation test revealed a significant negative correlation between BMI and fall in systolic BP after exercise ($r = -0.52$, $P < 0.01$) for the pooled data. **Conclusion:** Therefore, it is concluded that BMI has a negative effect on the post-exercise systolic PEH. Hence, BMI must be taken into consideration while devising an exercise regimen for an individual as part of their lifestyle modification.

KEY WORDS: Post-exercise Hypotension; Body Mass Index; Blood Pressure; Exercise

INTRODUCTION

"Physical fitness is not only one of the most important keys to a healthy body but also it is the basis of dynamic and creative intellectual activity." However, due to career and profession taking up ones time, and aided by technological advancement, the amount of physical activity has come down. Due to this, non-communicable diseases such as obesity, hypertension, and coronary heart diseases are on the rise and treating these diseases is fast becoming a public health problem. Physical exercise/activity is an easy and early physiological way to prevent and treat many such diseases. In patients with hypertension, regular physical activity is a well-known physiological method to bring down the blood pressure (BP).[1]

Post-exercise hypotension (PEH) is "a phenomenon of a prolonged decrease in resting BP in the minutes and hours following exercise."[2] PEH is seen not only in normotensive individuals but also in pre-hypertensive and hypertensive individuals.[3,4] Research is going on to see if a single bout of exercise itself can cause sufficient PEH.[5] PEH is seen in endurance exercises and is not evident in resistance exercises.[6] PEH occurs within minutes[7] to a maximum of

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DOI: 10.5455/njppp.2018.8.0724808082018

1 h after exercise.[8] PEH was found to last for even 12 h[9] after exercise, but the maximum fall occurred at around the 15th min[3] in the post-exercise period. Both central and peripheral mechanisms are implicated in PEH. Baroreceptor resetting to reduce the sympathetic outflow post exercise is the central mechanism,[10] while post-exercise vasodilation due to release of metabolites and histamine[11] is the peripheral mechanisms causing PEH. During the exercise recovery period, there is a combination of centrally mediated decrease in sympathetic nerve activity, as well as activation of local vasodilator mechanisms.[12] These are some of the mechanisms put forward to explain the phenomenon of PEH. The centrally mediated phenomenon of baroreceptor resetting has been put forward as a major mechanism to explain PEH. The arterial baroreflex is reset to function at a lower BP after exercise.[10] This will result in a reduction in sympathetic outflow post exercise. During exercise, the thinly myelinated and unmyelinated (Groups III and IV) muscle afferents are activated in response to muscle contraction. Input from these afferents releases the neurotransmitter substance *P* at neurokinin-1 receptors on GABAergic interneurons in the caudal NTS. These GABAergic interneurons release GABA at $GABA_A$ receptors on second-order barosensitive neurons within the NTS. The second-order barosensitive neurons convey information from baroreceptor afferents to the caudal ventrolateral medulla. GABA reduces their excitability, resulting in less inhibition of sympathetic neurons in the rostral medulla, greater firing of sympathetic vasoconstrictor neurons during exercise, and the observed resetting of the baroreflex to higher pressures during exercise.[12] As exercise continues, neurokinin-1 receptors internalize the GABA interneuron (a result of substance *P* release from muscle afferent stimulation during exercise) so that after exercise the neurokinin-1 receptors are less available for binding.[13] As a result, the GABAergic interneurons show diminished responses to tonic inputs and thus exert less inhibitory influence on the second-order barosensitive neurons.

This results in an overall decrease in sympathetic outflow from the rostral ventrolateral medulla after exercise. Another important mechanism underlying PEH is post-exercise vasodilation. Two vasodilatory phenomena are recognized during recovery from exercise: (1) Immediate post-exercise hyperemia and (2) sustained post-exercise vasodilatation.[11] The immediate post-exercise hyperemia can last from seconds to minutes. This should not be confused with PEH which lasts longer and is sustained. Recent studies show that sustained post-exercise vasodilatation is dependent on the activation of histamine H_1 and H_2 receptors.[14] In another study PEH following 60 min of moderate intensity unilateral dynamic knee-extension exercise was abolished by H1 and H2 receptor antagonist.[15] Several possible mechanisms may increase intramuscular histamine during recovery from exercise. Mast cells located within the connective tissue layer surrounding skeletal muscle fascicles and those also found near blood vessels may degranulate releasing histamine locally.[16] Antigen-dependent and antigen-independent mechanisms cause mast cell degranulation. However, in exercise, the antigen-independent methods predominate. Exercise-related factors that have been associated with mast cell degranulation include reactive oxygen species, a variety of cytokines, and increase in temperature.[17] These are some of the major mechanisms put forward to explain PEH.

Obesity is defined as a condition with excessive fat accumulation in the body to the extent that health and well-being are adversely affected (World Health Organization). Obesity is a silent killer. Obesity in India is rising in both the urban and in the rural population.[18] Sedentary lifestyle combined with unhealthy eating patterns is the major cause of obesity.[19] The first step in treating obesity is lifestyle modification which includes increased physical activity and dietary modifications.

A major concern about obesity is that it is almost always associated with other diseases such as hypertension[20] and diabetes[21] which make treatment even more difficult. A common solution for both hypertension and obesity lies in physical activity as it helps to reduce weight and BP.[2] Hamer and Boutcher[22] using 10 normal and 6 obese individuals studied the post-exercise hemodynamics between the two groups and concluded that changes in body mass index (BMI) were associated with variations in hemodynamic patterns in the post-exercise period. India is going through great epidemiological transition and must face the burden of dealing with the problem of both undernutrition and obesity. To the best of our knowledge, no such studies have been reported so far in the Indian population to evaluate the effect of BMI on PEH. Hence, this study was done to investigate the effects of BMI on PEH.

MATERIALS AND METHODS

Ethical Consideration

Ethical committee clearance (IEC: RC/13/103) from the institutional ethical committee was first obtained before the study commenced. An informed written consent was obtained after explaining the protocol to each participant before the commencement of the study.

Selection of Subjects

Healthy adult male participants in the age group of 20–30 years with normal BMI and those with BMI above normal were included in the study.

Number of Groups

The participants were divided into two groups based on their BMI.

Group I - Participants with normal BMI (18.5–24.9) kg/m^2.
Group II - Participants with higher BMI (25 and above) kg/m^2.

Sample Size

Based on an earlier study, a sample size of 30 was calculated for each group to detect a significant difference in BP between normal and overweight groups, with 90% power and significance level of 5%.

Exclusion Criteria

Participants who came under the following criteria were excluded from the study:

- Participants practicing yoga or trained athletes.
- Pre-hypertensives.
- Participants with previous history of musculoskeletal injuries.

Equipment Used

Omron M10-IT (HEM–7080 IT-E) digital BP monitor was used to record the BP of the participants before and after exercise. A motorized treadmill manufactured by AFTON (ACP087) was used to make the participant exercise at the desirable VO_2 max.

The exercise test was conducted in a well-ventilated room. Each participant was asked to report to the physiology laboratory in the morning around 9 a.m. after having light breakfast. They were instructed to refrain from rigorous physical activity for 48 h before the test and not to consume tea/coffee for 12 h before the study. A detailed medical history was obtained, and general physical examination was done to assess the health status of the participant. Anthropometric measurements were also recorded, and the BMI of each individual was calculated using the Quetelet's formula. Omron M10-IT (HEM-7080 IT-E) digital BP monitor was used to record the baseline BP in both the groups.

The predicted maximum heart rate of the participant was calculated by subtracting their age from 220.[23] The participants' VO_2 max (the maximum amount of oxygen utilization by the tissues) was calculated by dividing the participant's maximum heart rate by the basal heart rate and multiplying it with 15.3[24] (Uth-Sørensen-Overgaard-Pedersen estimation). The value obtained was divided by 2 to calculate the 50% VO_2 max. To eliminate the resting oxygen consumption, 3.5 ml/kg/min was subtracted. The speed at which they must walk to make them exercise at 50% VO_2 max was then calculated by dividing the obtained O_2 consumption by 0.2.[25] This was the speed in m/min. To get it in km/h, we multiplied it with 60 and divided it by 1000. This was the speed at which they walked on the treadmill to exercise at 50% VO_2 max. The speed would vary depending on the participant's age and basal heart rate; however, the work done by each would be the same.

Exercise Test

Each participant was instructed to walk on the treadmill (AFTON ACP087) for 20 min at the specific speed which was calculated for them. At the end of 20 min, the participants were asked to sit on a chair and their sitting BP immediately after exercise was recorded within 30 s. Subsequently, their BP was recorded for the next 30 min at 5-min intervals. Therefore, one immediate BP value measurement and six post-exercise BP values at intervals of 5 min were totally recorded. The maximum fall in systolic BP was calculated by subtracting the lowest recorded systolic BP in the post-exercise period from the basal systolic BP value. Similarly, the maximum fall in diastolic BP was calculated by subtracting the lowest recorded diastolic BP from the basal diastolic value.

Statistical Analysis

All the data were analyzed using SPSS version 21. $P < 0.05$ was considered to be statistically significant. The data were found to be not normally distributed, and hence, the variables are expressed as median and interquartile range. Mann–Whitney U-test was used to compare the significance of any difference between the groups in age, anthropometric parameters, basal heart rate, pre- and post-exercise BP values (both systolic and diastolic). Spearman correlation test was used to find out the association between the BMI and PEH values.

RESULTS

The descriptive data of the study population demonstrated that there was no significant difference in height between the groups. However, the values of age, weight, and BMI showed that normal BMI group participants were significantly younger than the higher BMI group ($P < 0.05$) ones and had significantly lower body weight ($P < 0.01$) [Table 1]. The basal systolic BP of the normal BMI group was found to be lower than the above normal BMI group, and the difference was statistically significant ($P < 0.05$). Although the basal diastolic values were also of similar pattern, the difference was not statistically significant. The basal heart rate was also found to be lower in normal BMI group, and it was statistically significant ($P < 0.01$) [Table 2]. The maximum fall in systolic BP was found to be greater in normal weight subjects when compared with their overweight counterparts, and the difference was found to be statistically significant ($P < 0.01$) using Mann–Whitney U-test. Similarly, the maximum fall in diastolic BP was also higher in normal BMI group when compared with the higher BMI group in the post-exercise period, but it did not reach statistically significant values [Table 3]. Spearman correlation test showed that there was a significant negative correlation between BMI and fall

Table 1: Descriptive statistics of the study population

Variable	Normal BMI group median (25^{th}–75^{th} percentile)	Above normal BMI group median (25^{th}–75^{th} percentile)	*P* value
Age (years)	23 (20–26)	26 (23–28)	<0.05*
Height (m)	1.69 (1.65–1.72)	1.68 (1.64–1.72)	>0.05
Weight (Kg)	61 (55–69)	78 (72–81)	<0.01*
BMI (Kg/mt^2)	21 (20–23)	27 (26–28)	<0.05*

P value is for Mann–Whitney U-test between the two groups. **P* value statistically significant. BMI: Body mass index

Table 2: Comparison of cardiovascular parameters among the groups with normal BMI (18.5–24.9) and with above normal BMI (25 and above)

Variable	Normal BMI group median (25^{th}–75^{th} percentile)	Above normal BMI group median (25^{th}–75^{th} percentile)	*P* value
Basal systolic BP (mmHg)	111 (108–116)	116 (112–117)	<0.05*
Basal diastolic BP (mmHg)	74 (70–78)	77 (74–78)	>0.05
Basal heart rate (beats/min)	70 (64–74)	76 (70–80)	<0.01*

P value is for Mann–Whitney U-test. **P* value statistically significant. BMI: Body mass index

Table 3: Comparison of PEH values among the groups with normal BMI (18.5–24.9) and above normal BMI (25 and above)

Variable	Normal BMI group median (25th–75th percentile)	Above normal BMI group median (25^{th}–75^{th} percentile)	*P* value
Maximum fall in systolic BP after exercise (mmHg)	7 (6–8)	5 (4–6)	<0.01*
Maximum fall in diastolic BP after exercise (mmHg)	4 (3–5)	4 (3–4)	>0.05

P value for Mann–Whitney U-test between the two groups. **P* value statistically significant. PEH: Post-exercise hypotension. BMI: Body mass index

in systolic BP in the post-exercise period ($r = -0.52$, $P < 0.01$) [Figure 1].

DISCUSSION

Among the two groups, the normal BMI group was significantly younger than the obese group ($P < 0.05$) and their weight was considerably lower than the higher BMI group which was statistically significant ($P < 0.01$). The height of the participants between the two groups was similar. There were differences in the basal BP and heart rate values between the two groups. Analysis revealed that the basal systolic BP of normal BMI group was significantly lower than (median, 25^{th} and 75^{th} percentile - 111 [108–116]) the above normal BMI group (116 [112–117]) ($P < 0.05$). A similar trend was seen in diastolic BP also. However, the difference was not statistically significant ($P > 0.05$). The higher BMI group also showed higher basal heart rate ($P < 0.01$) when compared with the normal BMI group.

The results of our study are consistent with those of Nageswari *et al.*[26] who found similar high basal BP values in obese individuals. These findings indicate that the basal sympathetic tone in the obese group is higher than the normal BMI group, whereas the peripheral resistance is similar between the two groups. In long-standing obesity, the sustained and prolonged hemodynamic burden induces structural changes in the left ventricle.[27] Therefore, the high basal BP could be due increase in cardiac output (CO) because of high BMI. Landsberg and Krieger in 1989[28] and Troisi *et al.*[29] in 1991 reported that diet intake has an important role in the activation of sympathetic system. Obese individuals also have elevated levels of insulin which is also a stimulator of the sympathetic system. Increased sympathetic activity in turn will gradually increase the BP from normal values. Higher sympathetic tone will also cause an increase in the heart rate. Therefore, high basal heart rate can be taken as a marker of sympathetic activity.[30] After exercise for 30 min, we compared the post-exercise fall in BP between the two groups using Mann–Whitney U-test. The normal BMI group had a greater fall in both systolic and diastolic BPs when compared to the above normal BMI group; however, the difference was statistically significant only in case of systolic fall ($P < 0.01$), whereas the difference in diastolic BP fall was not statistically significant ($P > 0.05$). The most important reason for the fall in systolic BP is due to centrally mediated baroreceptor resetting to reduce the sympathetic outflow after exercise.[10] The diastolic fall is due to the release of metabolites and local vasodilator substances like histamine.[14] The normal BMI participants have statistically significant greater fall in systolic BP than the participants with above normal BMI. This could be due to the

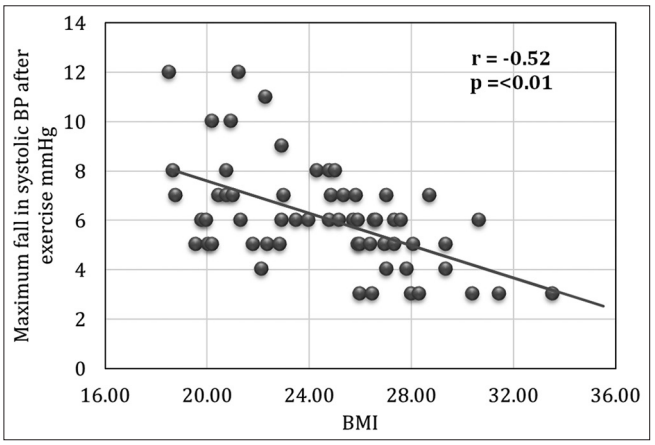

Figure 1: Scatter plot showing correlation between body mass index and maximum fall in systolic blood pressure in the post-exercise period

fact that obese individuals have higher sympathetic tone than non-obese individuals. Therefore, even after baroreceptor resetting, their sympathetic discharge would have been higher than the normal BMI individuals. Hence, normal BMI individuals have a statistically significant fall in systolic BP. In case of diastolic BP, the fall is due to the production of local metabolites which might be similar in both groups. Furthermore, Spearman correlation revealed a significant negative correlation between the maximum fall in systolic BP and BMI in the post-exercise period ($P < 0.01$, $r = -0.52$), but the correlation between maximum fall in diastolic BP and BMI was not statistically significant ($P > 0.05$).

Our study findings vary with the study findings done by Hamer and Boutcher.[22] They stated that BMI was associated with specific post-exercise hemodynamic responses between the normal BMI and obese group. Higher BMI was associated with greater reductions in CO and stroke volume but lower reductions in total peripheral resistance. Based on these findings, they concluded that BMI has no effect on PEH. However, they suggested that body composition has an impact on the mechanisms of PEH and should, therefore, be considered as an important confounding variable in future studies. This variation can be due to the fact that, in our study, a total of 60 subjects were recruited, whereas their study was done with only 16 participants in number. The inadequate sample size might be the reason their study did not show statistical significance.

Nevertheless, our study shows that BMI has a negative effect on post-exercise hypotension, particularly the systolic BP.

Limitations

1. Greater sample size might have given a better idea about the diastolic BP also.
2. Whether the effect is the same in women needs further investigation.
3. BP recording could have been done at 2-min intervals (or even continuous BP monitoring if possible) rather than 5 min for better knowledge about the BP fall after exercise.

CONCLUSION

From our study, it can be concluded that BMI significantly affects post-exercise hypotension, systolic PEH more than diastolic PEH, in the post-exercise period. This could be due to higher sympathetic tone in the higher BMI individuals. Clinically, by this study, it can be concluded that, an above normal BMI individual may have to exercise with greater intensity or duration to attain the same beneficial effects of PEH as individuals with normal BMI. Therefore, while devising an exercise regimen for the treatment of hypertension, BMI of the individual must be taken into consideration for optimum benefit for the individual.

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How to cite this article: Jeeva K, Bhattacharya P. Effect of body mass index on post-exercise hypotension in healthy adult males. Natl J Physiol Pharm Pharmacol 2018;8(10):1457-1462.

Source of Support: Nil, **Conflict of Interest:** None declared.