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RESEARCH ARTICLE

Effect of body mass index on time taken to attain maximum post-exercise hypotension in healthy adult males

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ABSTRACT

Background: Post-exercise hypotension (PEH) denotes a physiological fall in blood pressure (BP) from resting value after exercise. It is mainly due to decrease in sympathetic outflow after exercise. As obese individuals are known to have high basal sympathetic tone, we wanted to find a correlation between obesity and the time taken to attain maximum PEH. **Aim and Objective:** The aim of the study was to compare the time taken to attain maximum PEH between healthy adult males with normal body mass index (BMI) with 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/mt^2 (n = 30 in each group) were recruited. The resting heart rate and BP were recorded. Then, the participants were made to walk on a treadmill at 50% of their VO_2 max for 20 min. BP was recorded at 5-min intervals for 30 min. **Results:** The time taken to attain maximum systolic PEH was significantly lesser ($P < 0.01$) in the normal BMI group (Median=20 vs. 25 min) than the higher BMI group. However, the time taken to attain maximum diastolic PEH was comparable between the groups. Spearman correlation test revealed a significant positive correlation between BMI and time taken to attain maximum fall in systolic BP after exercise ($r = 0.52$, $P < 0.01$). **Conclusion:** Therefore, it is concluded that as BMI increases the time taken to attain maximum systolic PEH also increases. Hence, when devising an exercise regimen, the BMI must be taken into consideration to attain maximum benefit.

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

INTRODUCTION

Physical fitness is not only most important to a healthy body but also the basis of dynamic and creative intellectual activity. However, due to scientific and technological advancements, the amount of physical activity has come down. As a result of this reduction, non-communicable diseases such as obesity, hypertension, and coronary heart diseases have increased in incidence, and treating them has become a huge public health issue. Physical exercise/activity is one of the easiest and earliest physiological ways of preventing and treating many such diseases. Particularly in patients with hypertension, lifestyle modification, which included regular physical activity is a well-known physiological method to bring down the blood pressure (BP).[1]

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Post-exercise hypotension (PEH) is "a phenomenon of prolonged decrease in resting BP in minutes and hours following exercise."[2] PEH is seen in pre-hypertensive, hypertensive individuals, and also in normotensive individuals.[3,4] There is sufficient evidence to prove that even a single bout of exercise (called acute exercise) itself

can cause sufficient PEH.[5] PEH is mainly seen in endurance (aerobic) exercises.[6] PEH can occur within minutes[7] to even an hour after exercise.[8] The maximum PEH occurs at around the 15th min[3] in the post-exercise period, but it was found to last for even 12 h[9] after exercise. The physiological basis behind PEH and the time taken to attain PEH has both central and peripheral mechanisms.[10] There is enough evidence to prove that during the exercise recovery period, there is a centrally mediated decrease in sympathetic nerve activity, and also activation of local vasodilator mechanisms.[10] The central mechanism is baroreceptor resetting to reduce the sympathetic outflow post-exercise[11] while the post-exercise release of metabolites and histamine[12] to cause vasodilatation is the peripheral mechanisms causing PEH. One of the major mechanisms for PEH is the centrally mediated baroreceptor resetting mechanism. After exercise, the baroreceptor reflex is reset to a lower BP level.[11] This causes a reduction in sympathetic outflow after exercise. During exercise, the myelinated and unmyelinated muscle afferents are activated in response to muscle contraction. At the caudal nucleus tractus solitarii (NTS), there is the release of substance P at neurokinin-1 receptors on gamma-aminobutyric acid (GABA) ergic interneurons due to the inputs from these afferent fibers. These GABAergic interneurons cause the release of GABA at $GABA_A$ receptors on second-order neurons within the NTS. The second-order neurons then convey the information from the baroreceptor afferents to the caudal ventrolateral medulla. The release of GABA reduces their excitability, which results in decreased inhibition of sympathetic neurons in the rostral medulla, which, in turn, leads to greater firing of sympathetic neurons during exercise and therefore the baroreflex is reset to a higher pressure during exercise.[10] As the person continues to exercise, due to the release of substance P from muscle afferent stimulation, neurokinin-1 receptors internalize the GABA interneuron, therefore after exercise, the neurokinin-1 receptors will be decreased in number for further binding.[13] As a result, there will be diminished GABAergic interneurons response to tonic inputs, and thus, the inhibitory effect on the second-order neurons will be decreased.

This, in turn, leads to an overall decrease in the sympathetic outflow from the rostral ventrolateral medulla after exercise. The second important mechanism underlying PEH is post-exercise vasodilatation. There are two vasodilatory phenomena which are recognized during the post-exercise recovery period: (1). Immediate post-exercise hyperemia and (2) sustained post-exercise vasodilatation.[12] The immediate post-exercise hyperemia lasts from seconds to minutes. This is not the same as PEH which will last longer and is sustained. There is sufficient evidence to prove that sustained post-exercise vasodilatation is dependent on the activation of histamine H_1 and H_2 receptors.[14] This was further supported by another study which showed that PEH following 60 min of moderate-intensity unilateral dynamic knee-extension exercise was abolished by H_1 and H_2 receptor antagonist.[15] Several possible mechanisms can cause an increase in intramuscular histamine during the post-exercise recovery period. The few exercise-related factors which are associated with mast cell degranulation include high temperature, reactive oxygen species, and a variety of cytokines.[16] Mast cells located in the connective tissue layer around the skeletal muscle fascicles will degranulate, releasing histamine locally.[17] In addition to these mechanisms, there is antigen-dependent and antigen-independent mechanism which causes mast cell degranulation, but with regard to exercise, the antigen-independent methods predominate. These are some of the major physiological principles put forward to explain PEH.

Obesity denotes a condition with excessive fat accumulation in the body to the extent that health and well-being are adversely affected (WHO). Obesity in India is prevalent in both the urban and in the rural population.[18] The major causes of obesity are a sedentary lifestyle and unhealthy eating patterns.[19] The first and most important step in treating obesity is lifestyle modification which includes regular physical activity and healthy dietary modifications.

The problem associated with obesity is that it is almost always associated with other diseases such as diabetes[20] and hypertension[21] which makes treatment even more difficult. Physical activity is a common remedy for both hypertension and obesity as it helps to reduce weight and BP.[2] The hemodynamic changes due to exercise and post-exercise are different between normal and obese individuals.[22] This study shows that body mass index (BMI) has a confounding effect on PEH. To the best of our knowledge, studies on the Indian population to evaluate the effect of BMI on time taken to attain maximum PEH are scarce. Hence, this study was done to investigate the effects of BMI on time taken to attain maximum PEH among healthy Indian adult males.

MATERIALS AND METHODS

Ethical Consideration

Ethical Committee Clearance (IEC: RC/13/103) from the Institute 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 for the study.

Sample Size

Based on an earlier study,[22] 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%.

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/mt^2 (n = 30)

Group II – Participants with higher BMI (25 and above) kg/mt^2 (n = 30)

Exclusion Criteria

The following criteria were excluded from the study:

- Participants practicing yoga or trained athletes.
- Prehypertensives.
- Participants with previous h/o 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 the exercise physiology lab. Each participant was asked to report at around 9 A.M. They were instructed to refrain from rigorous physical activity for 48 h before the test and not to consume tea/coffee as well as use tobacco for 12 h before the study. After obtaining the medical history, 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 after 5 min rest.

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 using the Uth-Sørensen-Overgaard-Pedersen formula.[24] The value obtained was divided by two 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 maximum was then calculated by dividing the obtained O_2 consumption by 0.2.[25] This was the speed in meters/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 time taken to attain maximum fall in systolic BP (Systolic PEH) below the resting value was noted. Similarly, the time taken to attain maximum fall in diastolic BP was also noted.

Statistical Analysis

The data were checked for normality and 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 significant difference between the groups in age, anthropometric parameters, basal heart rate, basal BP, and time taken to attain maximum systolic and diastolic PEH. Spearman correlation test was used to find out the association between the BMI and time taken to attain maximum PEH values. All the data were analyzed using SPSS version 21. $P < 0.05$ was considered to be statistically significant.

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 ones ($P < 0.05$) and had significantly lower body weight ($P < 0.01$) [Table 1].

The basal BP and heart rate values of the participants in each group were expressed as median and interquartile range. 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 a similar trend, 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].

While analyzing the time taken to attain the maximum fall in systolic and diastolic BP in the post-exercise period among the two groups, it was noted that normal BMI individuals have taken lesser time to attain maximum fall in systolic BP compared with the above-normal BMI group and the difference was found to be statistically significant using

Mann–Whitney U-test ($P < 0.01$). Although the time taken to attain maximum fall in diastolic BP in normal BMI group was also lower when compared with the above-normal BMI group, the difference was not statistically significant [Table 3].

The correlation between BMI and the time taken to attain maximum fall in systolic BP was analyzed using the Spearman correlation test. It was found that there was a significant positive correlation between BMI and the time taken to attain maximum fall in systolic BP in the post-exercise period ($r = 0.52$, $P < 0.01$) [Figure 1].

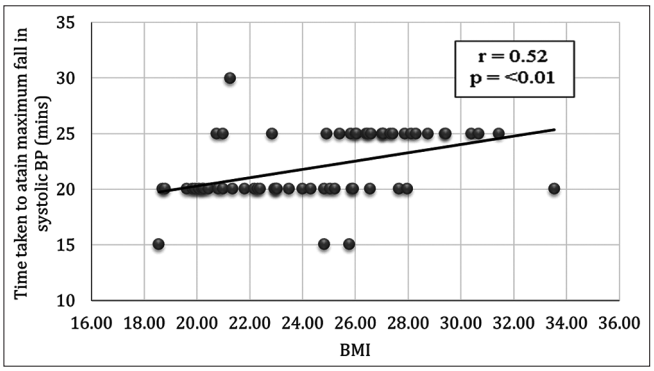

Figure 1: Scatter plot showing the correlation between body mass index and time taken to attain maximum fall in post-exercise systolic blood pressure using Spearman's test

DISCUSSION

The normal BMI group was significantly younger than the obese group ($P < 0.05$) and as to be expected their weight was considerably lower than the higher BMI group which was statistically significant ($P < 0.01$). The height variation between the two groups was minimal.

There was a significant difference in the basal systolic BP with the normal BMI group having a lower systolic BP than the above-normal group. The trend was similar in diastolic BP also, but it was not statistically significant. The basal heart rate of the above-normal BMI group was significantly higher than the normal BMI group. These findings are consistent with that of Nageswari *et al*.[26] who stated that obese individuals have a higher basal sympathetic tone when compared with non-obese individuals, whereas the peripheral resistance is similar between the two groups. The extra adipose tissue in obese individuals requires extra blood flow, which in turn leads to an increase in cardiac output. Thus, the high basal BP could be due to an increase in cardiac output in the obese individuals.

Landsberg *et al*. in 1989[27] and Troisi *et al*.[28] in 1991 reported that diet intake also has a role to play in the activation of the sympathetic system. They postulated that fat and carbohydrate in the diet of obese individuals stimulated the sympathetic

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 (mts)	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*

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

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*

BMI: Body mass index, BP: Blood pressure. **P* value statistically significant for Mann–Whitney U test

Table 3: Comparison of time taken to attain maximum post-exercise hypotension among groups with normal BMI (18.5–24.9) and 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
Time taken to attain maximum fall in systolic BP (min)	20 (20–20)	25 (20–25)	<0.01*
Time taken to attain maximum fall in diastolic BP (min)	20 (15–20)	20 (20–25)	>0.05

BMI: Body mass index, BP: Blood pressure. **P* value statistically significant for Mann–Whitney U test

system. Insulin regulated glucose metabolism within the ventromedial hypothalamus plays an important role in the relationship between diet and sympathetic nervous system activity. These are the proposed mechanisms by which obesity is linked with increased activation of the sympathetic system and Grassi *et al*.[29] suggested that this increased sympathetic system activity is the cause of hypertension commonly associated with obesity. High sympathetic tone will also cause higher basal heart rate; hence, a high basal heart rate can be taken as a marker of sympathetic activity.[30] In an earlier study by us, we compared the maximum PEH between the two groups and found that the normal BMI group had a higher systolic PEH when compared with the diastolic PEH and that systolic PEH had a negative correlation with BMI.[31] This was postulated to be due to the higher sympathetic tone in obese individuals. In this study, we compared the time taken to attain maximum systolic as well as diastolic PEH between the two groups. The normal BMI group attained a maximum fall in systolic BP earlier than the above-normal BMI group ($P < 0.01$). The time taken to attain maximum fall in diastolic BP also followed the same trend, but, the difference was not statistically significant ($P > 0.05$).

The most important reason for PEH is the centrally mediated baroreceptor resetting which decreases sympathetic outflow following exercise and the diastolic was due to local vasodilator metabolites.[10,14] In the present study, normal BMI group attained maximum PEH (Systolic) faster than the above-normal BMI group. This could be due to the fact that normal BMI has a lower sympathetic tone than the obese group, so it will take longer time to reduce the sympathetic outflow after exercise in the obese group than the normal BMI group. Therefore, the systolic PEH is attained much quickly in the normal BMI group than the obese group. In case of the time taken for diastolic PEH, though the trend is similar it did not reach statistical significance which might be due to the fact that diastolic PEH is mainly due to the production of local metabolites which might be similar in both groups.

Scatter plot analysis also reveals than the majority of the men above BMI of 25 reached the maximum fall in systolic PEH around 25th min, and the majority of the normal BMI group reached around 20th min. This finding was similar with a previous study done by MacDonald *et al*. in 2000 who reported that the maximum reduction in BP in the post-exercise period has been shown to occur at around the 15th min after exercise.[3]

Nevertheless, our study shows that as the BMI increases the time taken to attain maximum fall in PEH also increases, particularly for the systolic BP.

Limitations

1. A greater sample size could have given a better idea about the time taken to attain maximum diastolic PEH also.
2. Whether there is any gender variation needs further investigation.
3. BP recording could have been done at shorter intervals (or even continuous BP monitoring if possible) rather than 5 min for more accurate time recordings.

CONCLUSION

From our study, it can be concluded that BMI significantly affects the time taken to attain maximum PEH, systolic PEH more than diastolic PEH. This could be due to a 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 for a greater duration of time or at higher intensity to attain the same beneficial effects of PEH as individuals with normal BMI. Therefore, while devising an exercise regime for the treatment of hypertension or obesity, BMI of the individual must be taken into consideration for the optimum benefit for the individual.

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