



## Effects of physical activity and obesity on hypertension: A narrative mini-review of the main effective mechanisms

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### ABSTRACT

Physical activity (PA) and obesity are effective interventions for hypertension. The current study is a review to explain possible mechanisms related to the effects of PA and obesity on hypertension. To this end, several scientific databases were searched using the keyword "hypertension" and also some English articles related to obesity and PA were investigated. Then, the mechanisms of obesity and PA associated with hypertension were extracted from the collected articles. Overall, obesity causes an increase in renin-angiotensin-aldosterone (RAA) systems, harmful changes in lipid and lipoprotein profile, a decrease in insulin sensitivity, as well as harmful changes in adipokines, oxidative stress, and inflammatory factors. PA improves the above-mentioned changes caused by obesity. Overall, PA mainly via an effect on oxidative stress, inflammation, endothelial cells, the elasticity of arteries, body weight, the activity of the RAA system, activity of parasympathetic and renal function as well as improve the insulin sensitivity has positive effects on hypertension. It should be noted that the effects of PA against hypertension is highly variable and they are related to PA modes, environmental and genetic factors.

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### Introduction

Blood pressure (BP) is the blood-exerted force against the walls of the vessels.

BP depends on the blood vessels' resistance and ability of heart work.

Hypertension is elevated BP to a high level. Hypertension is a medical condition

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that is prevalent all around the world. Long-term hypertension increased cardiovascular risk factors and causes heart problems such as heart attack and stroke, heart failure, and aneurysm.

Obesity is a risk factor for having hypertension. Several studies have shown a significant positive relationship between overweight/obesity and hypertension (1). It is demonstrated that people have an increase in BP along with an increase in body weight (BW) (2). It has also been shown that individuals with the highest body mass index (BMI) have a 16 mmHg higher systolic BP and a 9 mmHg higher diastolic BP compared to individuals who have the lowest BMI (2). It showed that the systolic BP increases by about 4 mmHg/4.5 kg of BW (2). On the other hand, it has been shown that the loss of BW in overweight people lowers BP (3). Another investigation (meta-analysis study) showed that 3%-9% of BW loss reduced systolic (3 mmHg) and diastolic (3 mmHg) BP (4). It showed that BW loss (3.5 kg) reduced BP (4.0/1.1 mmHg) in hypertensive aged people (5).

There appears to be a significant negative relationship between having a physical activity (PA) and hypertension. It previously showed that PA has critical roles in hypertension prevention and treatment. PA has beneficial effects on both systolic and diastolic BP and maybe induced a reduction in BP about 5–7 mmHg in hypertension condition (6-10). PA may immediately cause a reduction in systolic BP. It noted that the BP reduction followed almost 24 hours post of PA event with the most positive effects seen in subjects with higher baseline BP (7). It showed that chronic PA has more sustained reductions in BP (11).

As mentioned above, PA and obesity are effective interventions for hypertension. The present study is a narrative review to explain possible mechanisms related to the effects of PA and obesity on hypertension.

### Method

In the current review study, a variety of scientific databases were searched using the keyword "hypertension" and some English articles related to obesity and PA were investigated. Then, the mechanisms of obesity and PA concerning hypertension were extracted from the collected articles.

### Mechanisms of obesity-related to hypertension

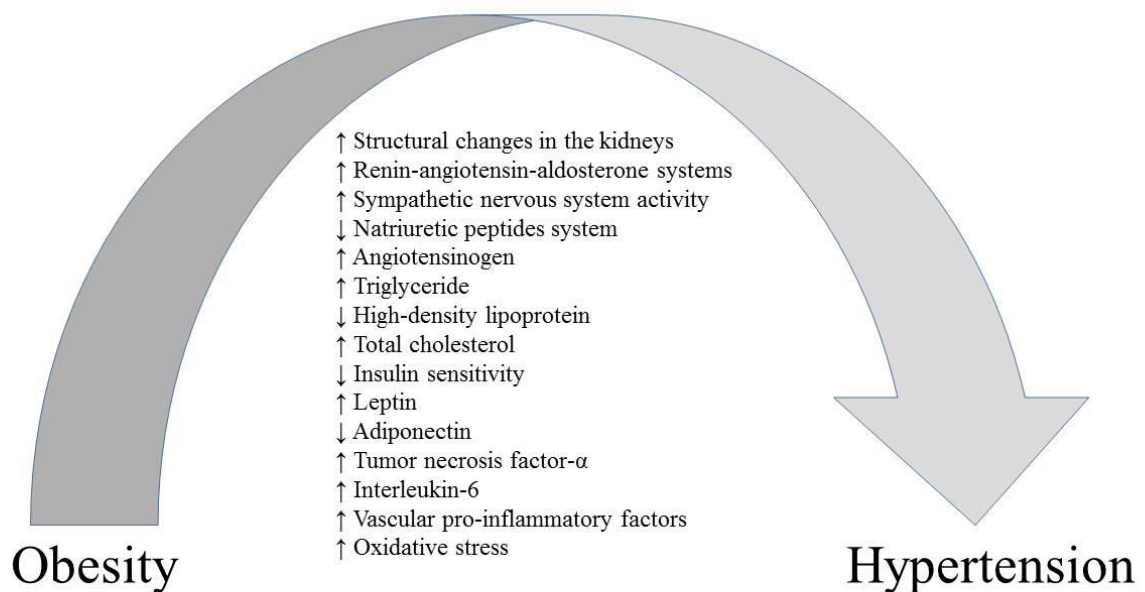
Lobato *et al.* reported that obese mice have higher BP and impairment in the relaxation of the aorta in response to acetylcholine. They reported that the dysfunction in endothelial cells, reduction in antioxidant defense, and nuclear factor  $\kappa$ B (NF $\kappa$ B) pathway activation are related to hypertension (12). Thus, it seems that vascular pro-inflammatory factors and oxidative stress have effects on endothelial dysfunction and consequently hypertension. Ferro *et al.* reported that respiratory quotient value is related to hypertension prevalence. They reported that subjects with high respiratory quotient and BMI have a higher risk of having hypertension (13). It has been reported that loss of BW (even a modest reduction about 5%) can cause a significant reduction in the activity of the RAA systems -in blood circulation and fat tissue- and reduces BP (14). On the other hand, BW gain increases the incidence of hypertension significantly (15). Becque *et al.* reported that the most of obese adolescents with elevated BP, showed elevated blood triglyceride (TG) concentration, decreased high-density lipoprotein (HDL), and increased total cholesterol (TC) concentration along with elevated systolic or diastolic BP (16).

Obesity and overweight may induce hypertension via impairing renal pressure (RP) natriuresis in hypertension conditions (17). In obesity conditions, the kidneys sodium reabsorption increased and RP natriuresis was impaired by activation of RAA and sympathetic nervous systems and also by altering intrarenal physical forces (17). Obesity condition also induced changes in the structure of kidneys and loss of the nephron function (17). The BW loss probably lowering BP via insulin sensitivity improvement and sympathetic nervous system activity reduction (18). The decreased activity of sympathetic caused by reduced activation of the RAA system, natriuresis, contracted plasma volume, and return of the high cardiac output state are mechanisms for the decrease

of BP by loss of BW in overweight hypertensive people (3).

As mentioned above, the obesity condition induces an overactivated RAA system. The sympathetic nervous system activity is increased in obese subjects with hypertensive, which could induce obesity-related renal effects. On the other hand, there might be inhibition of the natriuretic peptides system. A decrease in insulin sensitivity and hyperinsulinemia (metabolic syndrome element) is a critical link between obesity and hypertension (19). Adipose

tissue secretions such as leptin, tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin (IL)-6, angiotensinogen, and nonesterified fatty acids may contribute to the development of hypertension (20), for example, leptin-mediated sympathetic activation and consequently hypertension (21). On the other hand, adiponectin has a protective effect against hypertension through an endothelial-dependent mechanism (22). Figure 1, presents some main mechanisms related to the effects of obesity on hypertension.



**Figure 1.** Main mechanisms related to the effects of obesity on hypertension.

### Mechanisms of PA concerning hypertension

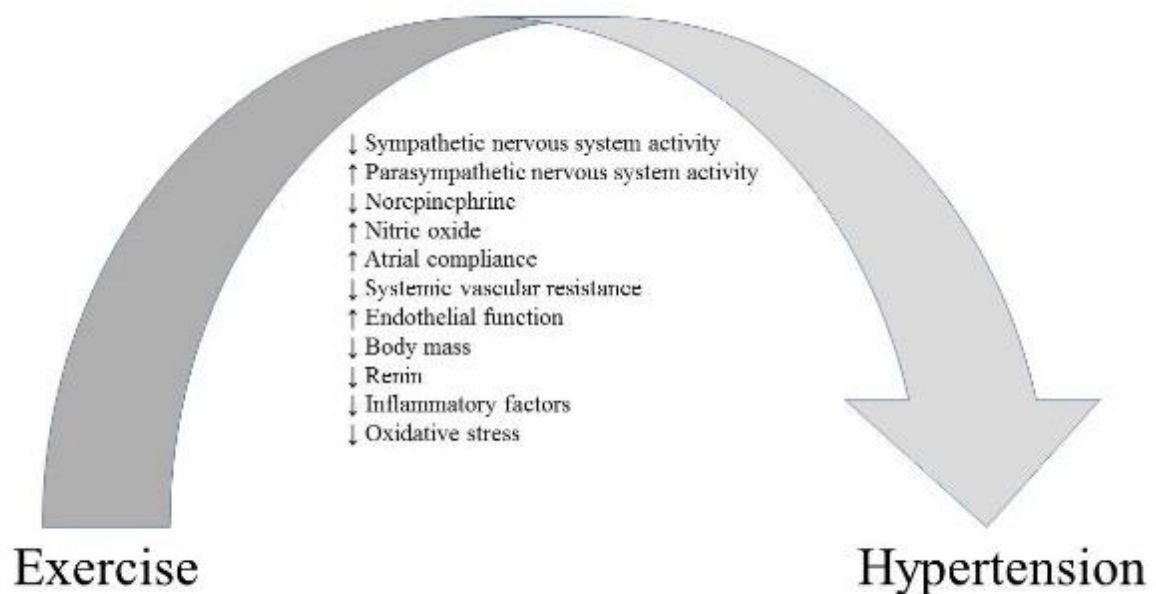
A meta-analysis suggests that PA reduces BP independent of changes in the BW (23). According to a study, PA was reduced but did not remove the effects of obesity on hypertension risk (24).

It reported that (a meta-analysis study), systemic vascular resistance dysfunction, norepinephrine, and renin activity are key factors regarding the decrease in BP after PA (25). The BP reduction following PA is probably due to changes in the resistance of

peripheral vascular. The changes in the resistance of peripheral vascular may be due to neurohormonal (sympathetic nerve activity) and structural (arterial lumen diameters) (26). Changes in oxidative stress and inflammation, endothelial cells, compliance of arterial, BW, activity of the RAA system, the activity of the parasympathetic, renal function, and sensitivity of insulin are other mechanisms regarding the effect of PA (6). The mechanisms underlying the beneficial effect of PA on BP are not certain and are still

under investigation (27). Improved endothelial function is a key possible mediator of the hypotensive response observed with PA. The 24 hours post of PA blood pressure-lowering is mainly due to the stroke volume reduction or the sympathetic nervous tone modulation. PA affects BP via endothelium vasodilatation and nitric oxide release. PA as shear stresses endothelium nitric oxide and enhances the endothelial velocity (24, 28). Shear stress enhances the nitric oxide expression and synthase in endothelial cells and induces up-regulation of free-radical scavengers such as cytosolic copper-and zinc-containing superoxide dismutase. Shear-stress induced of angiotensin-converting enzyme suppression and creates relaxation by activation of bradykinin(29, 30).

The PA anti-hypertensive response must be highly variable due to differences in PA regimens, environmental factors, and genetic related factors (31). In one study, 20–25% of subjects with hypertension were non-responders and had no reduction in BP by using PA (32,33). Studies regarding the impact of genetic and clinical factors on response/no response of patients to PA is still going on (31). Figure 2, presents some main mechanisms related to the effects of PA on hypertension.



**Figure 2.** Main mechanisms related to the effects of PA on hypertension.

### Conclusion

There are many mechanisms related to the effects of PA and obesity on hypertension. Overall, obesity causes an increase in RAA systems, harmful changes in lipid and lipoprotein profile, a decrease in insulin sensitivity as well as harmful changes in adipokines, oxidative stress, and

inflammatory factors. PA improves the above-mentioned changes caused by obesity. Overall, PA mainly via critical changes in oxidative stress, inflammation, the function of endothelial cells, compliance of arterial, BW, the activity of RAA system, the activity of parasympathetic, renal function, and sensitivity of insulin has positive effects on hypertension. It should be noted that the

effects of PA against hypertension are highly variable and they are related to PA modes, environmental and genetic factors.

### Declaration of conflicting interests

The authors declare no potential conflicts of interest concerning the research, authorship, and/or publication of this article.

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