International Journal of Scientific Advances
ISSN: 2708-7972
Volume: 6 | Issue: 1 | Jan – Feb 2025 Available Online: www.ijscia.com
DOI: 10.51542/ijscia.v6i1.6

Effects of Stress on Blood Pressure: A Literature Review

Eugenia Richita Oei[1], Dewi Ratna Sari[2*], Hayuris Kinandita Setiawan[3], Damba Bestari[4,5]

[1]Medical Study Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[2]Department of Anatomy, Histology, and Pharmacology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[3]Department of Physiology, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[4]Department of Psychiatry, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[5]Department of Psychiatry, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

E-mail: eugenia.richita.oei-2021@fk.unair.ac.id; dewi-r-s@fk.unair.ac.id; hayuris-k-s@fk.unair.ac.id; damba.bestari@fk.unair.ac.id

*Corresponding author details: Dewi Ratna Sari; dewi-r-s@fk.unair.ac.id

ABSTRACT

Background: Stress disrupts homeostasis, triggering adaptive responses that can become maladaptive with prolonged exposure, leading to conditions like hypertension. Chronic stress elevates sympathoadrenal activity and stress hormones, contributing to vascular tone and hypertension, a major global health concern. In Indonesia, hypertension prevalence rose to 34.1% in 2018, particularly among young adults. ***Objective:*** This review explores stress effects and mechanisms linking stress to blood pressure. ***Method:*** Collecting the data and information from Google Scholar, PubMed, and ScienceDirect with a total of 22 pieces of literature using keywords like “effects”, “stress”, and “blood pressure”. ***Result:*** The review highlights that distress and sustress disrupt homeostasis, adversely affecting blood pressure. Acute stress temporarily raises blood pressure through the "fight or flight" response involving the sympathetic nervous system and HPA axis, while chronic stress causes sustained hypertension via elevated cortisol, inflammation, and circadian disruption. Gender differences, with women showing oxytocin-mediated buffering and men's heightened sympathoadrenal reactivity, underscore the need for effective stress management to reduce cardiovascular risks.

Keywords: blood pressure; effect; stress.

INTRODUCTION

Stress is a condition in which the body's homeostatic state faces a challenge or threat that requires adjustment [1]. In a state of stress, the body responds with physical reactions to the source of stress or stimuli that attack [2]. This response is adaptive, where the body attempts to cope with the circumstances triggered by the stressor. However, when exposure to a stressor is intense, recurrent (repeated acute stress), or long-term (chronic stress), the stress response can become maladaptive and negatively impact the physiology. For example, exposure to chronic stressors can trigger maladaptive reactions such as depression, anxiety, cognitive impairment, and cardiovascular disease [3]. It is known that elevated sympathoadrenal activity, increased secretion of norepinephrine and epinephrine, and heightened vascular tone contribute to the development of hypertension induced by stress [4]. Hypertension is one of the major risk factors that occupies the first position as a preventable cause of death worldwide. Despite this, hypertension has a very high prevalence and is also ranked third as the factor that most negatively affects the quality of life [5].

In Indonesia, the prevalence rate of hypertension has dramatically increased from 25.8% in 2013 to 34.1% in 2018 [5]. The prevalence of hypertension among young and middle-aged adults has been increasing, posing a significant and urgent public health concern [6]. This literature review aims to explore the effects of stress and pathophysiological mechanisms linking stress response to blood pressure.

METHODS

To review the effects of stress on blood pressure, the researcher used several valid databases from Google Scholar and Pubmed to retrieve information from articles, journals, and books. Keywords used were “stress”, “effects”, and “blood pressure”. While the thematic search consists of “effects of stress on blood pressure”, “types of stress and its effect”, “pathophysiology of stress-induced blood pressure”, and “acute and chronic stress effects on blood pressure”.

RESULTS AND DISCUSSION

The initial search across Google Scholar and PubMed yielded a total of 3,756 articles using the keywords "stress," "blood pressure," and "effects.

With the thematic searches to refine the search were performed using phrases such as "effects of stress on blood pressure," "pathophysiology of stress-induced blood pressure," and "acute and chronic stress effects on blood pressure". These were screened based on their titles and abstracts to assess relevance to the research, resulting in the exclusion of 3,734 articles that did not relate to the topic and the aim of the review. In total, 22 articles were included in this review, covering a range of topics such as the evolution and types of stress, physiological mechanisms in blood pressure changes induced by stress, and the comparisons between acute and chronic stress effects.

The findings of this review highlight the significant role of stress in influencing blood pressure through distinct mechanisms, depending on its type and duration. Stress can be categorized into eustress, distress, and sustress, each exerting unique effects on homeostasis [1,7]. While eustress promotes resilience and benefits overall health, distress, and sustress are associated with disrupted physiological functions that can lead to pathological conditions. Acute stress activates the "fight or flight" response, mediated by the sympathetic nervous system and HPA axis, resulting in temporary increases in blood pressure through mechanisms such as vasoconstriction and elevated secretion of stress hormones. Chronic stress, however, represents prolonged exposure to stressors, leading to cumulative physiological burdens that exacerbate cardiovascular risks [8,9].

Chronic stress mechanisms, in particular, show maladaptive outcomes through sustained activation of the HPA axis and sympathetic nervous system. This includes heightened cortisol levels, inflammatory activity, and altered circadian rhythm, which collectively contribute to systemic effects such as metabolic syndrome, obesity, and persistent hypertension. Gender differences further shape stress responses, with women showing a tendency to buffer stress through oxytocin-mediated attachment behaviors, while men exhibit heightened sympathoadrenal reactivity. These insights emphasize the necessity of managing both acute and chronic stress to mitigate its detrimental effects on blood pressure and overall cardiovascular health [8,10].

TYPES OF STRESS

The concepts and definitions of stress have evolved and expanded significantly over the past decades. We can define stress as the state where the body's homeostasis condition is disrupted by a challenge or threat that requires the body to adapt [1]. The stress system framework categorizes stress into three types: eustress, distress, and sustress [1,7]. Eustress is a type of stress representing good stress. It is a state in which moderate levels of stressors challenge the state of homeostasis mildly, triggering a mild stress response that strengthens homeostatic buffering capacity which promotes and benefits the overall health. Distress is a type of stress representing bad stress. It is a condition where homeostasis is significantly disrupted by excessive levels of stressors, potentially triggering an intense stress response, compromising homeostasis, and posing a risk to health. Sustress is a type of stress representing inadequate stress. It is a condition where homeostasis remains unchallenged due to the absence or insufficiency of stressors, which can diminish the buffering capacity of homeostasis and threaten health [7].

Sustress and distress can disrupt normal physiological functions and potentially result in pathological conditions, whereas eustress may promote health by optimizing homeostasis adapting. Thus, maintaining an optimal level of stress is crucial for developing biological defenses that support normal life processes [1].

Studies have shown different responses and pathways of stress according to its duration of exposure. It is divided into two types which are acute stress and chronic stress [4,11,12]. Acute stress is a condition of short-term exposure to stressors. Acute stress can arise in daily life due to factors such as anger, fear, intense work pressure, or stressful sports events, as well as from natural disasters like earthquakes and hurricanes, or man-made events such as industrial accidents and terrorist attacks. Chronic stress refers to long-term, repetitive stress exposure. Chronic stress is strongly associated with factors such as depression, anxiety, low self-esteem, loneliness, job-related stress, retirement, low socioeconomic status, marital problems, etc [11,12].

Acute stress refers to a type of stress in which a “fight or flight” response occurs due to the activation of the sympathetic nervous system. This response involves increased heart rate, contractility, vasoconstriction, and secretion of epinephrine and norepinephrine by the adrenal medulla and sympathetic nerves, respectively. Meanwhile, ongoing stress in daily life can lead to chronic stress, which adversely affects the body through the accumulation of load, surpassing the “fight or flight” response mechanism of acute stress [4,13].

GENDER DIFFERENCES IN STRESS RESPONSE

There are differences in stress responses in men and women. The response of the HPA-axis and autonomic nervous system is higher in men, which affects their response to stressors. In addition, sex hormones in women will decrease the sympathoadrenal response which can reduce the negative feedback of cortisol to the brain so that women tend to be less stressed [14].

From an evolutionary perspective, men have typically responded to stressors like predators by either confronting them or escaping. In contrast, women tend to cope with stress by nurturing their offspring and seeking support within social groups, strategies that enhance species survival during challenging times. While the physiological stress response in both genders involves activation of the sympathetic nervous system and the HPA axis, the female stress response is more likely to incorporate attachment and caregiving behaviors.

These processes, often influenced by oxytocin, help to mitigate sympathetic activation and HPA axis arousal [14,15].

ACUTE STRESS RESPONSE MECHANISM ON BLOOD PRESSURE

The brain's response to stress includes activation of the autonomic nervous system, hypothalamic-pituitary-adrenal (HPA) axis, and neural-hematopoietic-arterial pathways. These mechanisms give rise to pathophysiological effects that can trigger cardiovascular events, such as increased sympathetic nervous system activity, decreased parasympathetic nervous system activity, and stimulation of neurohormonal production [11,16].

Amygdala utilizes past experiences to form long-term memory through its connections with the hippocampus and anterior cingulate cortex (ACC). This enables emotion modulation and predictive evaluation of stimuli to produce appropriate responses to stress. Efferent projections from the amygdala to the cortico-limbic region play a role in regulating the activity of the autonomic nervous system and the HPA axis [12]. The hypothalamus combines autonomic afferent signals from the brainstem with influences from cortical and subcortical limbic areas, including the hippocampus and amygdala involved in evaluation and memory. The hypothalamus plays a role in regulating autonomic responses to stress, through the paraventricular nucleus, as well as being the prime mover of endocrine stress responses by releasing corticotropin releasing hormone (CRF) that activates the HPA axis [11,12].

When faced with stress, the hypothalamus is activated to synthesize corticotropin releasing factor (CRF) and vasopressin. CRF stimulates the anterior pituitary gland to release adrenocorticotropin hormone (ACTH), which in turn triggers the adrenal cortex to produce glucocorticoids, namely cortisol [17]. When cortisol levels surpass the capacity of the enzyme 11β-hydroxysteroid dehydrogenase type 2 (HSD2) to convert it into cortisone, cortisol binds to mineralocorticoid receptors (MR) with an affinity comparable to that of aldosterone. This interaction enhances sodium reabsorption and potassium excretion in the kidneys, resulting in an expansion of blood volume and a subsequent rise in blood pressure [18]. Cortisol activates the renin-angiotensin system (RAS) indirectly by enhancing hepatic production of angiotensinogen, it also suppresses renin levels due to negative feedback from its mineralocorticoid activity. Despite this suppression, cortisol-exposed individuals exhibit heightened sensitivity to angiotensin II's pressor effects, contributing to sustained hypertension[18].

Stressful situations increase the activation of the sympathetic nervous system and cause an increase in vascular resistance, blood pressure, pulse rate, and cause vasoconstriction. Activation of the sympathetic nervous system also stimulates the adrenal medulla to release the hormones epinephrine and norepinephrine, further increasing the sympathetic response [19]. The adrenal medulla is the main site for epinephrine synthesis. Epinephrine synthesis increases in response to corticosteroids and stress. Sympathetic nerves can absorb epinephrine from the bloodstream and produce small amounts of epinephrine. It has been suggested that this epinephrine enhances norepinephrine release and may play a role in the development of hypertension [20].

CHRONIC STRESS RESPONSE MECHANISM ON BLOOD PRESSURE

Chronic activation of the stress axis caused by long-term uncontrollable stress has been regarded as causing maladaptive and/or pathological effects. Chronic exposure to stress results in long-term cortisol exposure which becoming maladaptive and can lead to a broad range of problems including the metabolic syndrome, obesity, cancer, mental health disorders, cardiovascular disease and increased susceptibility to infections [12,21].

Chronic stress is consistently found to be associated with increased inflammatory activity, as measured mainly by plasma inflammatory markers such as interleukin 6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and c-reactive protein (CRP) [22]. In addition to its relationship with chronic inflammation, there is evidence that this is related to glucocorticoid response to stress and is also associated with elevated baseline cortisol secretion and higher HPA-axis reactivity to psychological stress as well as physiological and pharmacological stimulation [9,22]. This HPA axis alteration is also found to be characterized by loss of circadian rhythm and overall cortisol output throughout the day [9].

CONCLUSIONS

Stress is an adaptive physiological response aimed at maintaining homeostasis during challenges or threats. However, when stress exposure becomes recurrent or chronic, it can transition into a maladaptive state, adversely affecting physical and mental health. Chronic stress, in particular, has been linked to numerous health issues, including cardiovascular diseases like hypertension. The increasing prevalence of hypertension, especially among younger populations, underscores the urgency of understanding and mitigating stress-related health impacts.

Acute and chronic stress responses differ in their mechanisms and effects on blood pressure. Acute stress activates the sympathetic nervous system and the HPA axis, leading to temporary increases in blood pressure through mechanisms such as vasoconstriction and heightened release of stress hormones like cortisol, norepinephrine, and epinephrine. Chronic stress, however, results in sustained activation of these systems, causing long-term maladaptive effects such as persistent inflammation, dysregulated cortisol secretion, and heightened sensitivity to pressor hormones, all contributing to sustained hypertension and broader systemic health problems.

Addressing stress-related hypertension requires a holistic approach, considering both biological and behavioral aspects. Understanding the role of gender, social dynamics, and adaptive coping mechanisms can provide insights into targeted interventions. Future efforts should focus on strategies to balance stress levels, such as promoting resilience through lifestyle modifications, stress management techniques, and addressing socioeconomic determinants of health, ultimately reducing the burden of stress-induced hypertension.

ACKNOWLEDGMENT

We would like to acknowledge the contributions of our supervisors for the guidance and the invaluable feedback that was provided to this review study. The author also like to acknowledge the gratitude towards all of the researchers and authors of the previous studies, whose work has influenced this literature review study.

REFERENCES

[1] Lu S, Wei F, Li G. The evolution of the concept of stress and the framework of the stress system. Cell Stress 2021;5:76–85. https://doi.org/10.15698/CST2021.06.250.

[2] Lumban Gaol NT. Teori Stres: Stimulus, Respons, dan Transaksional. Buletin Psikologi 2016;24:1. https://doi.org/10.22146/bpsi.11224.

[3] Ketchesin KD, Stinnett GS, Seasholtz AF. Corticotropin-releasing hormone-binding protein and stress: from invertebrates to humans. Stress 2017;20:449–64. https://doi.org/10.1080/10253890.2017.1322575.

[4] Caylen A, Toru Ü, Korkut Y. The relationship of stress and blood pressure effectors. 2015.

[5] Indonesia Ministry of Health. Indonesia: Agency for Health Research and Development. Basic Health Research (RISKESDAS) 2018.

[6] Hinton TC, Adams ZH, Baker RP, Hope KA, Paton JFR, Hart EC, et al. Investigation and Treatment of High Blood Pressure in Young People: Too Much Medicine or Appropriate Risk Reduction? Hypertension 2020;75:16–22. https://doi.org/10.1161/HYPERTENSIONAHA.119.13820.

[7] Chrousos GP. Stress and disorders of the stress system. Nat Rev Endocrinol 2009;5:374–81. https://doi.org/10.1038/nrendo.2009.106.

[8] Cavigelli SA, Bao AD, Bourne RA, Caruso MJ, Caulfield JI, Chen M, et al. Timing matters: the interval between acute stressors within chronic mild stress modifies behavioral and physiologic stress responses in male rats. Stress 2018;21:453–63. https://doi.org/10.1080/10253890.2018.1459557.

[9] Rohleder N. Stress and inflammation – The need to address the gap in the transition between acute and chronic stress effects. Psychoneuroendocrinology 2019;105:164–71. https://doi.org/10.1016/j.psyneuen.2019.02.021.

[10] Golbidi S, Frisbee JC, Laher IX. Chronic stress impacts the cardiovascular system: Animal models and clinical outcomes. Am J Physiol Heart Circ Physiol 2015;308:H1476–98. https://doi.org/10.1152/ajpheart.00859.2014.

[11] Henein MY, Vancheri S, Longo G, Vancheri F. The Impact of Mental Stress on Cardiovascular Health—Part II. J Clin Med 2022;11. https://doi.org/10.3390/jcm11154405.

[12] Vancheri F, Longo G, Vancheri E, Henein MY. Mental Stress and Cardiovascular Health—Part I. J Clin Med 2022;11. https://doi.org/10.3390/jcm11123353.

[13] Artinian NT, Washington OGM, Flack JM, Hockman EM, Jen K-LC. Progress in Cardiovascular Nursing Depression, Stress, and Blood Pressure in Urban African-American Women. 2006.

[14] Wang J, Korczykowski M, Rao H, Fan Y, Pluta J, Gur RC, et al. Gender difference in neural response to psychological stress. Soc Cogn Affect Neurosci 2007;2:227–39. https://doi.org/10.1093/scan/nsm018.

[15] Farag NH, Vincent AS, McKey BS, Al'Absi M, Whitsett TL, Lovallo WR. Sex differences in the hemodynamic responses to mental stress: Effect of caffeine consumption. Psychophysiology 2006;43:337–43. https://doi.org/10.1111/j.1469-8986.2006.00416.x.

[16] Osborne MT, Shin LM, Mehta NN, Pitman RK, Fayad ZA, Tawakol A. Disentangling the Links Between Psychosocial Stress and Cardiovascular Disease. Circ Cardiovasc Imaging 2020;13:E010931. https://doi.org/10.1161/CIRCIMAGING.120.010931.

[17] Girod JP, Brotman DJ. Does altered glucocorticoid homeostasis increase cardiovascular risk? Cardiovasc Res 2004;64:217–26. https://doi.org/10.1016/j.cardiores.2004.07.006.

[18] Barbot M, Ceccato F, Scaroni C. The Pathophysiology and Treatment of Hypertension in Patients with Cushing's Syndrome. Front Endocrinol (Lausanne) 2019;10. https://doi.org/10.3389/fendo.2019.00321.

[19] Irwin MR, Cole SW. Reciprocal regulation of the neural and innate immune systems. Nat Rev Immunol 2011;11:625–32. https://doi.org/10.1038/nri3042.

[20] Ziegler MG, Milic M. Sympathetic nerves and hypertension in stress, sleep apnea, and caregiving. Curr Opin Nephrol Hypertens 2017;26:26–30. https://doi.org/10.1097/MNH.0000000000000288.

[21] Rohleder N. Stress and inflammation – The need to address the gap in the transition between acute and chronic stress effects. Psychoneuroendocrinology 2019;105:164–71. https://doi.org/10.1016/j.psyneuen.2019.02.021.

[22] McInnis CM, Thoma M V., Gianferante D, Hanlin L, Chen X, Breines JG, et al. Measures of adiposity predict interleukin-6 responses to repeated psychosocial stress. Brain Behav Immun 2014;42:33–40. https://doi.org/10.1016/j.bbi.2014.07.018.