The Impact of Psychological Stress on Cardiovascular and Immune Function

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Abstract

Psychological stress is a response to perceived threats or challenges beyond an individual coping capacity. It affects both the cardiovascular and immune systems: acute stress results in immediate response to stressors and chronic stress is associated with developing long-term health consequences. The sympathetic nervous system (SNS) is activated by acute stress and increases heart rate, blood pressure, and vascular resistance (part of the body's 'fight or flight' response). Short-term effects of these stressors provide adaptive advantages but chronic stress leads to long-term SNS activation and high circulating cortisol levels and is associated with hypertension, endothelial dysfunction, inflammation, and increased predisposition to cardiovascular diseases (CVDs). Stress also has a profound impact on the immune system. Acute stress causes a temporary increase in immune responses, while chronic stress lowers immunity making individuals more susceptible to infection and poor wound healing. Besides pharmacological approaches, there are some management strategies, including cognitive behavioral therapy (CBT), mindfulness, and lifestyle interventions to counter the effects of stress. This review emphasizes that stress cardiovascular health and immunity are interconnected, thus integrated strategies and further research are needed to cope with it.

Keywords: Psychological stress, Cardiovascular function, Immune function, Chronic stress, Sympathetic nervous system, Cortisol

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Introduction

Physiological stress is a cognitive, emotional, and physiological response to stressors. It can vary depending on the individual's personality, coping mechanisms, and past experiences (Schneiderman et al., 2005a). It activates the hypothalamic pituitary adrenal (HPA) axis and SNS, releasing hormones such as cortisol and adrenaline. Chronic activation of these systems threatens to erode homeostasis and result in physical and mental health problems including cardiovascular, immune, and neurodegenerative disorders (Huberman, 2022).

1.1 Cardiovascular System

It is made up of the heart, blood vessels, and blood. It works as a whole to pump hormones, nutrients, and oxygen throughout the body and to discard waste (Mohrman & Heller, 2018). The blood mainly travels through a network of veins and arteries. However, sympatho-adrenomedullary system (SAM) activation blocks these functions, which in turn raises blood pressure and heart rate over time and causes atherosclerosis and hypertension (Cohen et al., 2007).

1.2 Immune System

The immune system also protects the body from infections and harmful chemicals. Two types of the immune response, innate and adaptive response, work in synergy to detect and dispose of the external threats (Parham, 2014). The adaptive immune system slowly comes to a more specific response, and the innate immune system provides a non-specific but immediate response. Persistent psychological stress can induce adverse effects on the immune cells, therefore, changes in the cytokine balance and subsequent changes to immune function, possibly even with changes in inflammation or immunological suppression (Segerstrom & Miller, 2004).

1.3 Interrelation of Cardiovascular and Immune Systems

The cardiovascular and immune systems are closely related to maintaining overall health as well as remaining responsive to injury, infection, and inflammation. Complex cellular and molecular mechanisms between these two systems dictate communication that is vital for immunity, tissue repair, and disease protection (Libby et al., 2009).

The interaction of two such key processes has been well demonstrated in atherosclerosis, an inflammatory disorder of the arteries. In this process, immune cells, such as monocytes and macrophages, accumulate in the arterial wall due to cholesterol accumulation and eventually form a plaque. First protective but can turn out to be chronic inflammation and plaque buildup leading to heart attack and stroke (Falk et al., 2012). Immune cells are now also known to play a key role in causing chronic low-grade inflammation that contributes to CVDs such as atherosclerosis and hypertension, myocardial infarction, and heart failure (Everett et al., 2018).

2 Understanding Psychological Stress

Chronic stress can harm both physical and mental health, and social interaction, overall decreases quality of life. Understanding psychological stress can be helpful for stress management.

2.1 Types of Psychological Stress

Psychological stress can be categorized broadly into two types: acute and chronic stress.

2.1.1 Acute stress

The acute stress response is a short-term physiological and psychological response to an immediate perception of a threat or challenge. It activates the autonomic nervous system, releasing hormones such as adrenaline and cortisol. This response will help people overcome the situation, but once the stressor is removed, the response subsides (Rosch, 1994; McEwen & Wingfield, 2010).

2.1.1.1 Impacts of Acute Stress

Acute stress may be felt by a person as anxiety, irritability, and fear, with physiological symptoms like muscle tension, headaches, and nausea (McEwen, 2007). Short-term, acute stress can improve focus and concentration, but excessive stress of any kind can hinder thinking, and decision-making and impair memory (LeBlanc, 2009).

2.1.2 Chronic Stress

Chronic stress is a state of prolonged and persistent stress due to frequent exposure to a stressor for a long time, which results in continuous activation of the HPA axis as well as the dysregulation of the physiological systems. This causes negative impacts on physical and mental health leading to compromised immunity, cardiovascular problems, and psychological disorders (Cohen et al., 2007; Lupien et al., 2009).

2.1.2.1 Impacts of Chronic Stress

Anxiety, depression, and burnout all point to chronic stress. It can compromise emotional regulation and leave one feeling helpless and less resilient (Chrousos, 2009a). Stress for a long period of time negatively impacts brain regions important for memory and learning, such as the hippocampus. It also affects the prefrontal cortex which is crucial for decision-making and executive function (Rosch, 1994).

People with persistent stress may use damaging coping ways, for example, eating, substance dependence, or social detachment, which will pave the way for further harmful effects (Folkman, 2013). However, chronic stress is one of the major risk factors for many serious illnesses, including CVDs, depression, gastrointestinal disorders, and immune dysfunction. It also raises the risk of infection and chronic inflammation (Cohen et al., 2007).

2.2 Examples of Stressor

Stressors are conditions or events that trigger a stress response and can vary in nature, intensity, and duration. Examples of stressors are typically classified as acute or chronic and can be physical, social, emotional, or environmental (McEwen & Wingfield, 2010).

Short-term, acute stressors like a job demand that must be met, an important exam, or a minor accident tend to activate short-term stress responses that resolve quickly after the stressful event has passed (Rosch, 1994). In contrast, chronic stressors are sustained as well as potentially more disruptive to health and well-being. Examples are ongoing financial hardship, long-term caregiving responsibilities, and long-term work place pressures (Cohen et al., 2007).

Other sources of stress include environmental stressors (pollution, noise, and overcrowding), and life events like divorce, migration, etc. can also have negative impacts on individuals (Schneiderman et al., 2005b). Different stressors affect people differently based on factors like resilience, social support, and methods of coping. Stress management strategies are desperately needed for all levels of society (Thoits, 2010).

2.3 Physiological Response to Stress

The body's response to stress is a complex process involving multiple systems, with two primary mechanisms: the HPA axis and the autonomic nervous system (ANS) (Figure 2).

2.3.1 Neuroendocrine Response (HPA axis)

The neuroendocrine system involves the interaction of the nervous system and the endocrine glands in response to stress. The body's stress response relies heavily on the HPA axis (Fig. 1). The first part is when the brain perceives a stressor and then stimulates the hypothalamus to release corticotropin-releasing hormone (CRH). CRH increases the release of the adrenal hormones, namely cortisol (major stress hormone) and epinephrine and norepinephrine, which are released from the adrenal glands in response to Adrenocorticotropic hormone (ACTH) stimulation by the pituitary gland. Specifically, cortisol is important in the mobilization of energy stores, in the increase of glucose availability, and in the suppression of non-essential functions (e.g., reproduction and digestion) for the purpose of coping with the stressor (Rosch, 1994).

2.3.2 ANS

The ANS, comprising the sympathetic and parasympathetic branches, is integral to the body's immediate response to stress.

2.3.2.1 SNS

The SNS is activated in response to acute stressors, resulting in the "fight or flight" response. The response consists of an increase in heart rate, blood pressure, and respiratory rate in preparation for an immediate activity. Blood also begins to reroute from less critical functions (like digestion) to muscle and vital organs. (Thayer et al., 2012).

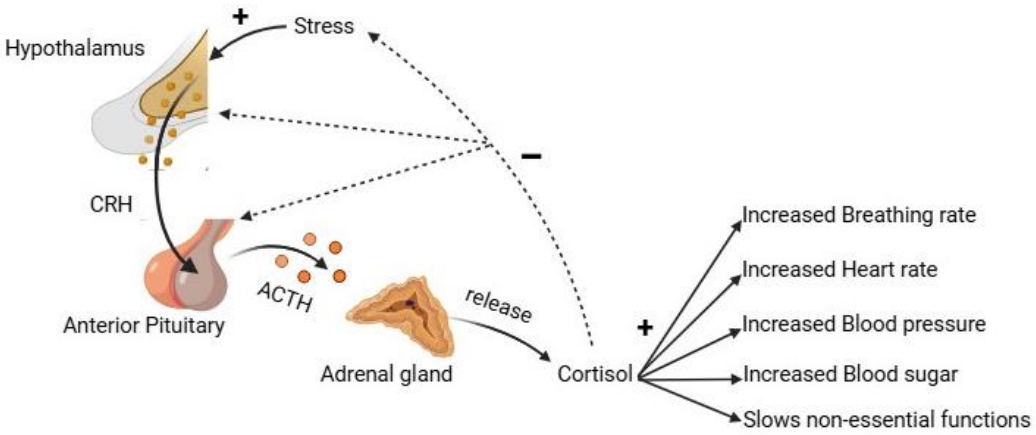

Fig. 1: Role of HPA axis in response to Stress (Retrieved from Biorender).

2.3.2.2 Parasympathetic Nervous System (PNS)

PNS counters the effects caused by SNS, thus promoting relaxation and recovery from the stress response. This helps lower your blood pressure, lower your heart rate, and improve digestion. Health depends on an optimal balance between SNS and PNS because chronic dominance of the SNS is linked to CVDs and PNS dysfunction associated with impaired stress recovery and mental health problems (Thayer & Lane, 2000).

2.3.3 Interaction between the HPA Axis and ANS

The HPA axis and ANS are joined and participate in controlling the body's reaction to stress. Both systems are activated at the same time during acute stress: the HPA axis provides sustained, mobilized energy through cortisol, and the ANS quickly prepares the body physically by releasing adrenaline and noradrenaline (Thayer & Lane, 2009).

2.3.3.1 Feedback and Regulation

There are feedback mechanisms connecting the HPA axis and ANS. The activity of the HPA axis is regulated by negative feedback to the hypothalamus and pituitary gland from cortisol, the end product of the HPA axis. In addition, cortisol modulates the sympathetic response to reduce the over-activation of this stress response (McEwen, 2007).

2.3.3.2 Health Implications

Chronic stress can dysregulate the interplay between the ANS and HPA axis, which can result in the over-activation of both the HPA axis and ANS. It causes prolonged sympathetic activation and increased release of cortisol. This dysregulation has been linked with mental health disorders (e.g., depression, PTSD), physical health disorders (e.g., hypertension, diabetes, immune suppression) (Chrousos, 2009a; Thayer et al., 2012).

2.3.4 Role of Gut-Brain Axis

Over the last couple of years, it has been argued that the response to stress is also mediated by the gut-brain axis. The gut microbiome has an important influence on the body's stress response. It modulates the HPA axis, immune function, and neurotransmitter production. Gut microbiota composition changes with stress may lead to gut dysbiosis, which is associated with gastrointestinal disease, anxiety, depression, and other mental health problems (Mayer et al., 2014).

3 Stress Effects on the Cardiovascular Function

Acute stress can cause a temporary increase in heart rate and blood pressure, whereas chronic stress may progress to several CVDs.

3.1 Effects of Acute Stress on Cardiovascular Function

Acute stress dramatically affects the cardiovascular system through SNS activation and through activation of the HPA axis. The SNS, when an individual experiences acute stress, causes a release of catecholamine (adrenaline and noradrenaline) through the body to increase heart rate (tachycardia) and blood pressure (hypertension) for a 'fight or flight' response (Chrousos, 2009a). The HPA axis also releases cortisol which amplifies vascular resistance and cardiac workload, all at the same time.

3.1.1 Heart Rate

An increase in heart rate is a physiological adaptation that permits the body to deal with immediate threats by increasing readiness, and motor performance, as well as mobilizing the energy. The ability of the body to respond to imminent stressors is a result of increased cardiac output and oxygen delivery to life-sustaining organs such as the brain, heart, and lungs (Lovallo, 2016).

Although adverse outcomes, such as arrhythmias, endothelial dysfunction, and plaque rupture, may arise from repetitive or extreme acute stress in predisposed individuals (Dimsdale, 2008). Transient conditions that have been associated with acute stress include stress induced cardiomyopathy (Takotsubo syndrome), characterized by reversible left ventricular dysfunction as result of excessive catecholamine release (Sharkey et al., 2007).

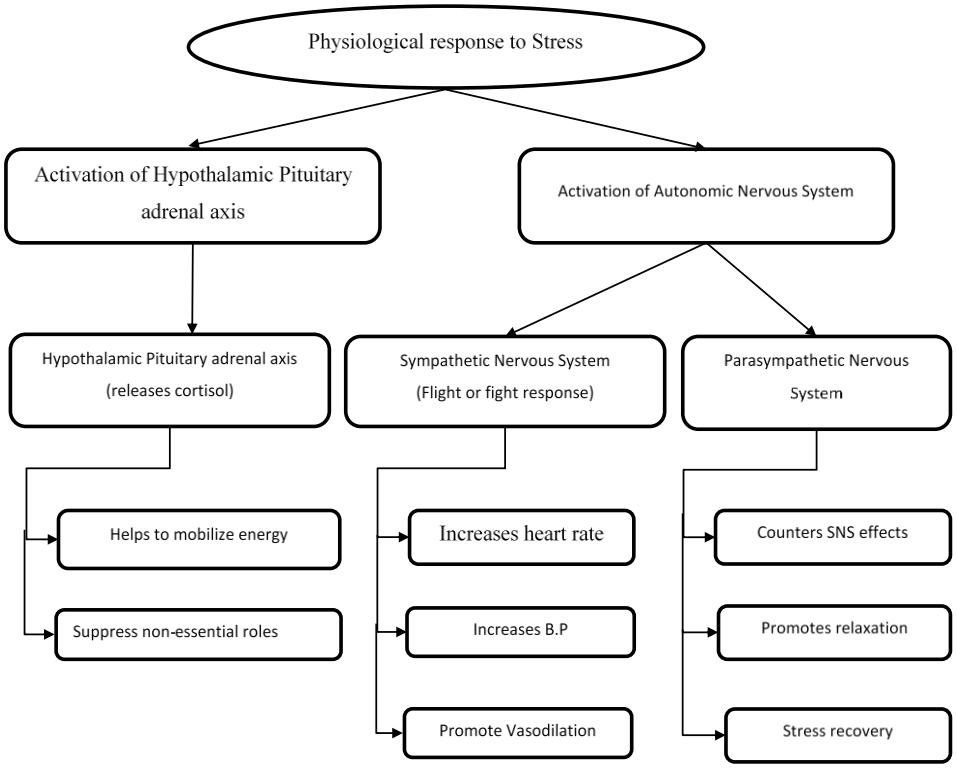

Fig. 2: Physiological response to Stress (Retrieved from Microsoft word 2016).

3.1.2 Blood Pressure

Acute stress-induced blood pressure elevation is a protective mechanism. Nevertheless, among people with preexisting cardiovascular disorders, these sudden increases can cause complications, including angina or myocardial infarction (Dimsdale, 2008).

3.1.3 Vascular Tone

The SNS is activated by psychological stress and releases the catecholamines (norepinephrine and epinephrine) that act upon α-adrenergic receptors in vascular smooth muscle cells. As a result, vasoconstriction occurs, and there is an increase in vascular resistance and elevated blood pressure (Chrousos, 2009a).

3.2 Effects of Chronic Stress on Cardiovascular Function

Persistent elevations in vascular tone resulting from chronic psychological stress are crucial factors in the progression of disease, including hypertension, atherosclerosis, and other CVDs (Steptoe & Kivimäki, 2013).

3.2.1 Endothelium dysfunction

Chronic high levels of cortisol can cause a dysfunction in the endothelium, the inner lining of blood vessels, by decreasing the level of nitric oxide (NO), a factor required for vascular relaxation. It often correlates with pro-inflammatory cytokines such as interleukin 6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which are associated with the mediation of endothelial dysfunction and abnormalities in vascular tone. This leads to an increase in vascular resistance and higher blood pressure (Chida & Steptoe, 2009).

This increased vascular tone over time can actually lead to vascular remodeling, increased arterial stiffness, and increased risk for cardiovascular events such as stroke and myocardial infarction (Steptoe & Kivimäki, 2013).

3.2.2 Hypertension, and Left Ventricular Hypertrophy (LVH)

From a physiological perspective, it has been shown that chronic stress may contribute to the development of hypertension, and hypertension over time can progress to LVH, which is defined as the left ventricular thickening due to work overload induced by high blood pressure. LVH is an important risk for heart failure, arrhythmias, and ischemic heart disease (Cohen et al., 2007).

3.2.3 Inflammation and Atherosclerosis

High levels of certain pro-inflammatory cytokines, such as IL-6 and C-reactive protein (CRP) have been linked with chronic stress and are capable of contributing to atherosclerosis (the buildup of plaque in the arteries). This contributes to arterial stiffening and blood vessel narrowing that raises the risk of coronary artery disease. (Kiecolt-Glaser et al., 2002). Constant SNS and HPA axis activation causes perpetuated high blood pressure and vascular resistance, placing an additional workload on the heart and propelling the atherosclerotic process.

3.2.4 Stress-Induced Dyslipidemia

Dyslipidemia, an imbalance of lipid profiles because of elevated low-density lipoprotein (LDL) cholesterol and reduced high-density

lipoprotein (HDL) cholesterol, can also be caused by chronic stress. It also intensifies the process of atherosclerosis with the narrowing and hardening of arteries (Rosmond, 2005).

4 Stress Effects on Immune Function

Acute stress temporarily enhances immune function, but chronic stress can have negative impacts on the immune system.

4.1 Effects of Acute Stress on Immune Function

When the body responds to acute stress, it regulates the stress response by temporarily raising immune function through the mobilization of immune cells to the sites of injury or infection. The stress hormones such as epinephrine and norepinephrine boost the circulation of immune cells like neutrophils, monocytes, and natural killer cells to help prevent a threat. They also contribute to the quick appearance and destruction of pathogens from the early phase of immune defense. In this state, pro-inflammatory cytokines such as IL6 and TNF-α also surge in the body to prepare the body's tissues to respond to injury or infection (Segerstrom & Miller, 2004).

4.2 Effects of Chronic Stress on Immune Function

Acute stress temporarily boosts immune function, but chronic stress results in continual HPA axis and SNS activation. Long-term stress produces elevated cortisol levels that have a knockdown effect on immunity. Chronic high cortisol levels have been shown to reduce the function of T lymphocytes and inhibit cytokine production (interleukins, TNF-α), essential for the coordination of immune response (Cohen et al., 2007). This suppression affects innate and adaptive immune systems. Natural killer cells, macrophages, and lymphocytes cannot work properly. This leads to increased susceptibility to infections and slow wound healing (Dhabhar, 2014).

Chronic stress also works to weaken the immune defense and promotes inflammatory pathways causing a surge in proinflammatory cytokines (IL-6, CRP, and TNFα). These inflammatory markers can lead to the development of chronic low-grade inflammation that can compromise immune system functioning and raise the risk of disease, for example, CVDs, autoimmune disorders, and some cancers (Kiecolt-Glaser et al., 2002; Glaser & Kiecolt-Glaser, 2005).

Autoimmune diseases are also linked to chronic stress. Stress causes immune dysregulation and increased proinflammatory cytokines, resulting in the onset of autoimmune conditions, including rheumatoid arthritis, multiple sclerosis, and systemic lupus erythematosus (Segerstrom & Miller, 2004). Epigenetic changes that include DNA methylation, and histone modifications to genes involved in immune response, are induced by stress. The latter has long-term implications for immune function (Zannas & Chrousos, 2017).

5 Interventions to Reduce Stress

Following interventions can help in stress reduction (Figure 3).

5.1 Psychological Interventions

Interventions like CBT, mindfulness practices, and relaxation techniques are crucial in stress management.

5.1.1 CBT and Mindfulness-based practices

Studies found that CBT and mindfulness practices are associated with increased mental well-being. CBT teaches us to restructure negative thought patterns (Regehr et al., 2013). Mindfulness-based practices such as meditation and deep breathing actually help to reduce stress by promoting relaxation and better emotional regulation. These techniques can reduce physiological markers of stress (Goyal et al., 2014).

5.1.2 Relaxation Techniques

Progressive muscle relaxation (PMR) and deep breathing exercises relax individuals by reducing physiological markers of stress (McCallie et al., 2006).

5.2 Lifestyle Interventions

These are non-pharmacological interventions and are defined as strategies that intend to decrease psychological stress by inducing healthier behaviors and expanding overall well-being. For example, regular diet, regular exercise, and sufficient sleep. Studies have shown that exercise, particularly aerobic exercise, reduces stress as it improves mood, improves resilience to stress, and produces endorphins (Salmon, 2001).

5.2.1 Physical Activity

Regular exercise reduces stress, lowers cortisol levels, improves mood (feel better) due to the release of endorphins, and helps with overall physical health. Aerobic exercises, yoga, and tai chi have proven useful in scaling down psychological stresses (Sharma, 2013).

5.2.2 Adequate Sleep

Stress reactions worsen over time because of the lack of sleep and result in cognitive problems (Meerlo et al., 2008). Quality sleep is crucial for emotional regulation and stress management Quality sleep is crucial for emotional regulation and stress management. Many things can reduce stress, such as getting a consistent sleep schedule and practicing good sleep hygiene (no electronics 1 hour before bed) (Irwin, 2015).

5.2.3 Healthy Diet

Eating a diet packed with whole grains, fruits, vegetables, and lean proteins keeps your brain healthy and may prevent inflammation related to stress. In particular, omega-3 fatty acids and antioxidants are capable of reducing stress (O'Neil et al., 2014).

5.2.4 Time Management

Time management reduces stress, which in turn helps you to focus properly, stop procrastinating, and set up a disciplined routine. Time blocking helps you to prioritize your tasks and allay your overwhelm and boosts productivity so you feel more accomplished and in control of your day (Verywell mind, 2023).

5.2.5 Social Support

Having friends, family, and a support group can give emotional comfort, and help to diminish solitary (loneliness), a key factor of stress. Social interactions can also release oxytocin, a counter to stress response (Uchino, 2006). It also involves the exchange of feelings and experiences of how to deal with the stressful situation of life together. By joining support groups and other community programs, you will find yourself less isolated and get practical stress-coping strategies (Mayo clinic, 2023).

5.3 Pharmacological Approaches

Severe or chronic stress symptoms are treated pharmacologically. Management of stress by pharmacological interventions consists of the use of selective serotonin reuptake inhibitors (SSRIs) for improving mood, benzodiazepines to alleviate transient anxiety, beta blockers to attenuate physical manifestations of stress (e.g., tachycardia), and adaptogens (e.g., ashwagandha) for stress-inducing higher cortisol levels. These medications are more helpful in conjunction with therapy and lifestyle modifications (Chrousos, 2009b). Sometimes medications like antipsychotics or mood stabilizers are added to different treatments for severe stress-related disorders, such as PTSD and bipolar disorder (Brady et al., 2000).

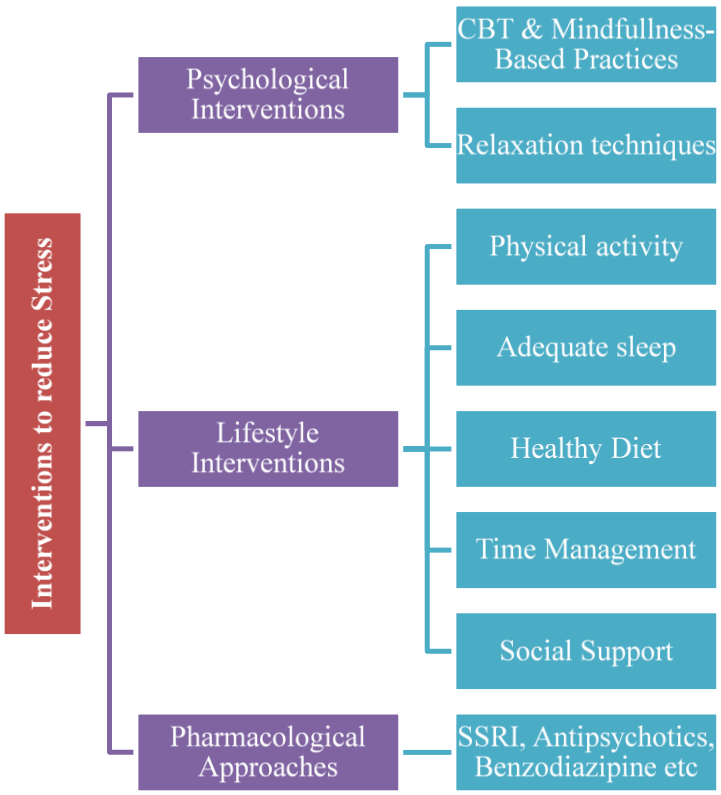

Fig. 3: Interventions to reduce Stress (Retrieved from Microsoft Word 2016).

6 Future research and insights

Future research in stress management is increasingly emphasizing the integration of advanced technologies, genetic insights, and personalized intervention. Accurately measuring stress through the use of wearable devices and real-time physiological monitoring is a promising field. Heart rate variability and skin conductance provide more objective stress data than self-reports, and these devices can track these markers (Sano & Picard, 2013).

Moreover, artificial intelligence (AI) and machine learning contribute to stress prediction, enabling earlier intervention by recognizing patterns in physiological responses (Healey & Picard, 2005). Genetic and epigenetic research is also progressing by exploring how different genetic variations determine an individual's susceptibility and stress response. Genes such as FKBP5 have variants that modulate stress sensitivity so that specific stress management strategies can be implemented on a person-by-person basis (Zannas et al., 2016).

Conclusion

In this chapter, we have looked at how psychological stress is linked to the cardiovascular system and immune function. To reflect that difference, we have distinguished between acute and chronic stress, focusing on their differing physiological impacts. Acute stress can cause a temporary increase in heart rate and blood pressure, whereas chronic stress will result in endothelial dysfunction, hypertension, and inflammation that will eventually contribute to the development of CVDs. Stress also affects immune function. Acute stress may increase certain immune responses, while chronic stress may depress the immune system, making an individual more susceptible to infections and disease. However, certain interventions can reduce stress, including CBT, mindfulness-based practices, physical activity, and a healthy diet. More research is still required concerning these critical interactions and developing targeted approaches to mitigate psychological stress, promoting cardiovascular and immune health.

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