



## A LITERATURE REVIEW ON THE EFFECTS OF PSYCHOLOGICAL FACTORS AND LIFESTYLE ON CARDIOVASCULAR REACTIVITY AND RECOVERY FROM ACUTE STRESS

**Samson F. Agberotimi<sup>1</sup>**

[samson.agberotimi@covenantuniversity.edu.ng](mailto:samson.agberotimi@covenantuniversity.edu.ng)

+234 803 489 1825

**Peace T. Akhibi<sup>2</sup>**

[peace.akhibipgs@stu.cu.edu.ng](mailto:peace.akhibipgs@stu.cu.edu.ng)

+2347081081759

**Benedict C. Agoha<sup>3</sup>**

[ben.agoha@covenantuniversity.edu.ng](mailto:ben.agoha@covenantuniversity.edu.ng)

+234 802 321 3131

<sup>1,2,3</sup> Department of Psychology, Covenant University, Ota, Ogun state, Nigeria.

### Abstract

*Cardiovascular reactivity (CVR) and recovery are important physiological processes that reflect how the cardiovascular system responds to and recovers from acute stress. Elevated or blunted CVR and delayed recovery have been associated with negative cardiovascular outcomes like hypertension and coronary heart disease. This literature review examines how psychological factors, such as emotional regulation and lifestyle factors, like physical activity levels, moderate CVR and recovery. The review states the biological mechanisms of stress responses, the roles of the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system, and how these interact with behavioural and environmental influences. Theoretical models like the Transactional Model of Stress and Coping, Allostatic Load Theory, Self-Regulation Theory, and the Biopsychosocial Model help in understanding these interactions. Together, the Transactional Model of Stress and Coping, Allostatic Load Theory, Self-Regulation Theory, and the Biopsychosocial Model provide an integrated framework showing that cardiovascular reactivity and recovery to acute stress are shaped not only by biological mechanisms, but also by psychological factors such as emotional regulation and coping, as well as lifestyle influences like physical activity. Emotional dysregulation is associated with exaggerated or blunted cardiovascular responses and delayed recovery. Whiston et al. (2025) found that individuals who struggle with emotional regulation show heightened cardiovascular reactivity and slower recovery following acute stress. On the other hand, effective emotion regulation and physical fitness are connected to more moderate reactivity and faster cardiovascular recovery. A study conducted by Agberotimi, Akhibi & Agoha (2025), found that effective emotion regulation (particularly lower lability) and physical activity contribute to healthier cardiovascular responses, with more moderate reactivity and faster recovery following acute stress. The findings show the need for better approaches in cardiovascular health that account for psychological resilience and healthy lifestyle behaviours in helping to reduce the effects of acute stress.*

**Keywords:** Cardiovascular reactivity, cardiovascular recovery, acute stress, emotional regulation, physical activity.



## Introduction

Cardiovascular reactivity and recovery from acute stress are important indicators of heart health. Psychological factors such as emotional regulation and lifestyle behaviours, including physical activity, may either reduce or heighten cardiovascular strain. Cardiovascular diseases (CVDs) persist as the predominant reason for death worldwide. Nigeria and other low-income countries are experiencing an increase in deaths related to CVD. A review of 20 years (2000 - 2020) shows a transition from CVDs related to malnutrition, like rheumatic heart disease, to an increase in CVDs like heart failure and hypertension. The high mortality rate shows the need for a holistic approach to both circumvention and treatment. Acute stress, tension, and frustration have been seen to be connected to a higher chance of CVD. While traditional risk factors like hypertension and hyperlipidaemia have been studied, the importance of psychological factors has been recognised in the growth and evolution of CVD (Mendez et al., 2018). Psychological factors, particularly emotional regulation and coping strategies, can regulate an individual's response to stress (Griffin & Howard, 2022; Lazarus & Folkman, 1984). Emotional distress in

reaction to stress can activate activity in the sympathetic nervous system, leading to alterations in the cardiovascular system. The modifications include increased heart rate, elevated blood pressure, and alterations in the flow of blood. The sympathetic hyperactivity, added to the parasympathetic hypoactivity, shows a potential pathological mechanism that can increase the risk of cardiovascular disease. This can also add to the growth and evolution of depressive and anxiety disorders. Emotional dysregulation contributes to problematic behaviours, and this is seen in various forms of psychopathology and may have direct or indirect consequences on physical health. Difficulties in emotional regulation have been linked to a range of mental and physical health issues, including cardiovascular disease (CVD).

Despite available studies, the extent to which emotional regulation can moderate the outcome of stress on cardiovascular responses needs to be explored. Lifestyle factors also significantly influence both cardiovascular health and the body's response to stress. Sedentary behaviour has also been identified as a risk for CVD, associated with higher resting blood pressure, systemic inflammation, and cortisol levels, and these are all linked to



stress response patterns. As a result of the significant global health burden of CVD and the factors potentially influencing cardiovascular responses to stress, further research in this area is important in helping to understand and develop effective preventive strategies. This review examines how psychological and lifestyle factors moderate cardiovascular reactivity and recovery.

### **Conceptual Framework**

Cardiovascular reactivity and recovery from acute stress are important indicators of heart health. Psychological factors such as emotional regulation and lifestyle behaviours, including physical activity, may either reduce or heighten cardiovascular strain. This review examines how psychological and lifestyle factors moderate cardiovascular reactivity and recovery.

### ***Cardiovascular Reactivity***

Cardiovascular reactivity (CVR) has to do with the physiological changes in cardiovascular function, primarily heart rate (HR) and blood pressure (BP), in response to a stressful stimulus (Creaven et al., 2020; McMahon et al., 2021; Whiston, Keogh, Howard, & Gallagher, 2025). It involves the dynamic changes in cardiovascular function in response to a

stressor, including changes in heart rate, blood pressure, cardiac output, and total peripheral resistance. It is the physiological alteration in heart rate (HR), blood pressure (BP), or more indices of cardiovascular function that occurs between a baseline state and the introduction of a stressor (Blascovich & Katkin, 1993). It refers to the short-term changes in HR and BP in response to physical or psychological stress (Chen, Mat Ludin, & Farah, 2022). The most common measures of cardiovascular reactivity are heart rate and blood pressure. Heart rate is measured as the number of pulses or heartbeats per minute and blood pressure is measured by the systole and diastole (Okoronta, 2002). The hypothalamus-pituitary-adrenal (HPA) axis and the autonomic nervous system and the autonomic nervous system are the primary systems responsible for cardiovascular reactivity (Chen et al., 2022). The early foundations were laid by Cannon and De la Paz (1911). Their work on the “fight-or-flight” bodily response laid emphasis on the role of the sympathetic nervous system in preparing the body for real or perceived threats. The response encompasses quick cardiovascular changes like increased heart rate and blood pressure, which enhance survival in the face of danger. Hans Selye



then brought about the general adaptation syndrome in 1936, focusing on how the body adapts to stress. This gave way to the cardiovascular reactivity hypothesis. The reactivity hypothesis states that consistently high or elongated cardiovascular reactivity to stress could be an important factor in the development of cardiovascular disorders (Krantz & Manuck, 1984). Cardiovascular reactivity can be elevated, normal, or blunted. Deviations from the normal range may signify poor systems integration and diminished homeostatic control. While elevated reactivity has negative health consequences, blunted responses may similarly signify poor homeostatic regulation and may lead to a distinct set of outcomes for health and behaviour (Carroll et al., 2017). Elevated reactivity is connected with the prevalence of hypertension (Carroll et al., 2012). Exaggerated response to stress can be termed as “hyper-responsive”. Frequent exposure to stressors can lead to tendencies to exhibit exaggerated cardiovascular reactivity, and individuals who show exaggerated cardiovascular reactions stand a greater chance of the untimely progression of hypertension and more antecedents to coronary heart disease, unfavourable cardiovascular-

related events, and untimely death due to these events (Chen et al. 2022).

A tentative model of blunted reactivity addresses different behavioural and health correlates like addiction, obesity, and poor subjective health. Blunted reactivity may signal poor homeostatic regulation, leading to the afore-mentioned consequences for health and behaviour. The model does not view blunted cardiovascular reactivity as a direct cause, but as a pointer to motivational dysregulation, and this is believed to be what ultimately determines adverse health and behavioural outcomes. An individual's CVR is determined by an interaction of genetic, physiological, environmental, behavioural, and psychological factors. Genetic factors generally influence the baseline cardiovascular function and may also influence an individual's response to stress at a biological level (Al'Absi, 2006). Environmental factors can also alter the development of regulatory systems, which would alter cardiovascular responses to stress, and may continue into adulthood (Lei, Berg, Simons, Simons, & Beach, 2020). Psychological factors also affect cardiovascular responses to stress. Individuals with high scores in traits like anxiety, hostility, and depressive symptoms may exhibit higher



cardiovascular responses to stress, while traits like optimism and perceived social support are associated with more adaptive cardiovascular responses (Tindle, Davis, & Kuller, 2010). Lifestyle choices like smoking, diet, and exercise may also modulate CVR (Al'Absi, 2006). Assessing CVR would involve monitoring changes in cardiovascular measures in response to standardised laboratory stressors, including tasks like mental tasks under time constraints, cold pressor test, public speaking tests, and so on (Ginty et al., 2022). Newer approaches include ambulatory monitoring of blood pressure and heart rate in real-life environments.

### ***Cardiovascular Recovery***

Cardiovascular recovery is the process by which the cardiovascular system reverts to the pre-stressor baseline state. It is the physiological process through which the cardiovascular system reverts to its pre-stressor baseline state, following the cessation of the stressful stimulus (McMahon et al., 2021). It is how quickly and effectively the heart and blood vessels return to the pre-activity or pre-event state. This includes the normalisation of the blood pressure, the slowing of heart rate, clearing of metabolic byproducts like lactate, and the rebalancing of autonomic nervous system activity (Eckelkamp,

2024). The process of cardiovascular recovery can be supported or hindered by a number of factors, some within our control and others not. Just like CVR, it is an indicator of an individual's cardiovascular health. A strong determinant of recovery is the baseline cardiovascular fitness. More physically fit individuals may experience faster and better recovery, because their cardiovascular systems are better conditioned to adapt to and recover from stress. Biological factors like age and the presence of chronic conditions may also make a significant difference (Eckelkamp, 2024). Psychological states like anxiety and emotional well-being may also affect cardiovascular recovery (Tessler & Bordoni, 2023). Individuals experiencing stress or low mood may recover more slowly because psychological strain affects the nervous system and impacts its ability to regulate heart function. In cases where individuals have major cardiac events like surgeries or heart attacks, the degree of damage to the tissue of the heart and the quality of the medical care received would also determine cardiovascular recovery.

Delayed recovery has been seen in the development of different cardiovascular diseases. Alongside having an exaggerated CVR, delayed recovery in response to stressors can disrupt homeostasis and



trigger or worsen pathophysiological changes involving the cardiovascular system (Chen et al., 2022). To keep track of recovery, clinicians may rely on the measure of Heart Rate Recovery (HRR). This measures the speed at which the heart slows down after the cessation of exercise, usually over the first minute. Faster HRR may be as a result of a well-regulated autonomic nervous system and shows a lower risk of future cardiovascular events. Apart from HRR, some measures like heart rate variability (HRV), blood pressure trends, and oxygen consumption may explain how well the cardiovascular system recovers.

### ***Acute Stress***

Acute stress refers to a short-term encounter with a challenging or threatening situation that prompts immediate physiological and psychological responses (McMahon et al., 2021). The body's responses to stress represent a systems-level response to threats to homeostasis. Consequently, maximum reactions to these threats would necessitate suitable fusion of the system at many levels, including circumferential physiology, the brainstem and hypothalamus, and the cortex and limbic system (Carroll et al., 2017). The response of the body to stress involves complex

mechanisms, involving the arousal of the sympathetic nervous system and also the release of hormones like adrenaline, cortisol, and glucagon, leading to increased glucose production for vital organs like the heart (Xue et al., 2024). Subjection to acute stress arouses the sympatho-medullary (SAM) system, leading to disturbances in cardiovascular activity and the hypothalamic-pituitary-adrenal (HPA) axis, resulting in altered levels of cortisol. Sympathetic nervous system reactions to stress employ the cardiovascular system and the adrenal medulla to create a synchronised “fight-or-flight” readiness. Concurrently, the hypothalamic-pituitary-adrenocortical system triggers an increment in cortisol production, and this helps in mobilising previously set-aside energy, and this regulates the stress reactions both peripherally and within the nervous system. In prime conditions, certain brain structures gauge the perceived threat and orchestrate a compliant model of autonomic and endocrine outburst to hold up the physiological and behavioural responses necessary to restore homeostasis (Lovallo, 2016; McEwen & Gianaros, 2011). The stress response typically resolves once the problem has been addressed. However, autonomic and





endocrine dominance over stress reactions can be changed in individuals with specific genetic predispositions, and also due to psychological factors. In these individuals, physiological, behavioural, and cognitive activity may be modified, and the health practices may be prejudiced toward maladaptive practices, resulting in worse overall health. Physiological responses to stress are meant to fall within the customary span for a specified homeostatic challenge, as stress is not just a biological event. Emotionally, acute stress may result in fear and anxiety, or anger, frustration, or even excitement, but this depends on how the individual interprets the situation (Lazarus & Folkman, 1984). Cognitively, stress may narrow our focus, which may be helpful in focusing on a particular task or threat, but may also affect performance on more complex tasks (Arnsten, 2009). Stress can also push people towards more impulsive or risk-averse choices, sometimes with significant consequences (Starcke & Brand, 2012).

On the other hand, individuals undergoing acute stress may seek out social support, especially if they believe that others will help them cope (Von Dawans, Fischbacher, Kirschbaum, & Heinrichs, 2012). Other times, acute stress may provoke withdrawal or aggression, laying emphasis

on individual differences. More resilient individuals may navigate stress more successfully and with fewer long-term consequences (Southwick, Martini, Charney, & Southwick, 2017). Exposure to moderate stress over time can sometimes build tolerance and foster effective coping skills. This process is called stress inoculation (Meichenbaum, 1985). However, frequent exposure to severe or unrelenting stress may have the opposite effect and may heighten a person's sensitivity to future stressors.

### ***Emotional regulation***

Emotional regulation refers to different strategies that individuals use to manage and respond to their emotional experiences (Griffin & Howard, 2022). It refers to the various complex ways people influence the emotions they feel, when they feel them, and how the emotions are expressed or experienced (Gross, 1998). The Process model by Gross (1998) differentiates between emotional regulation strategies used before emotions fully kick in (for instance, avoiding or reframing the situation) and those used after the intense feeling starts (for instance, deep breathing). Strategies in emotional regulation include (Griffin & Howard, 2022);



- **Reappraisal:** This involves reinterpreting a situation in order to alter the emotional impact. Reappraisal is generally considered an adaptive strategy, and it is related to positive psychological and physiological outcomes, characterised by either lower CVR or a challenge-oriented response profile.
- **Suppression:** This involves inhibiting the outward expression of emotions. This is usually considered a less adaptive strategy. Suppression is related to increased stress responses and negative health consequences.

More recent approaches include dual-process models, and these differentiate between conscious, deliberate efforts to manage emotion and the unconscious, automatic processes (Gyurak, Gross, & Etkin, 2011). An example of this is the Cognitive Emotion Regulation model, which focuses on how people use thoughts to control their feelings. Another model, the Experiential-Dynamic model, focuses on reconnecting with natural emotional processes. Yet another model, that of regulatory flexibility, involves the ability to choose the right strategy for the moment, and people who do this may be

more resilient and emotionally healthy (Aldao, Sheppes, & Gross, 2015). Difficulties in emotional regulation are commonly known as emotional dysregulation. Emotional dysregulation involves difficulties in identifying, understanding, and effectively managing emotional responses. This involves biological, behavioural, and social mechanisms, including automatic and conscious efforts to adjust emotional experiences (Calkins et al., 2019). Developing emotional regulation skills gradually begins in infancy, growing from learning to rely on co-regulation with caregivers to self-regulation. Some factors, such as parenting styles and individual temperaments, can influence this developmental course. Emotional dysregulation contributes to problematic behaviours, and this is seen in various forms of psychopathology and may have direct or indirect consequences on physical health (Calkins et al., 2019).

The process of emotional regulation begins in childhood, with babies and toddlers relying on caregivers to soothe and regulate their emotional states (Schor, 2019). As individuals grow, more emotional tools may develop, like distraction, self-talk, or perspective-taking. With time, most people get better at





managing their emotions, especially from experiences and relationships. Emotional regulation skills continue to evolve into adulthood (Charles & Carstensen, 2010). Culture and social norms may also shape how people feel and express emotions. In East Asia, suppressing one's emotions may be seen as a way to keep the peace, and it may not necessarily cause psychological harm. In more individualistic cultures, however, suppression might lead to emotional strain, because self-expression is more valued in those cultures (McRae & Gross, 2020; Ford & Mauss, 2015; Matsumoto, 2007).

### ***Physical activity levels***

According to Caspersen et al (1985), physical activity, from the perspective of exercise, is more than just movement; it is a purposeful and structured activity which aims at improving or maintaining physical fitness. Physical activity involves any bodily movement that is produced by skeletal muscles, which results in the expenditure of energy. It is how our bodies stay in motion across hours, blending sedentary behaviour, light physical activity, and more intense activities (Tremblay et al., 2017). This includes a wide range of activities, from walking to more structured forms of activities like sports and exercise. Exercise is a category

of physical activity that is purposeful, structured, planned, and repetitive, with the maintenance of physical fitness (one or more components) being an objective (Caspersen, Powell, & Christenson, 1985). Some components of physical fitness include muscle strength, flexibility, cardiorespiratory fitness, muscle endurance, and body composition (ACSM, 2025). An important aspect of physical activity levels is the concept of energy balance. A person's daily energy expenditure is shaped by the body's basic needs and the energy used to digest food, alongside exercise. An important aspect of physical activity levels is activity intensity. Sedentary behaviour requires very little energy, and is now recognised as a health risk, even for people who meet weekly exercise goals (Saunders et al., 2020; WHO, 2010). Light-intensity activity, which may include standing, slow walking, or gentle household tasks, plays a big role in reducing health risks among older adults (Diaz et al., 2017). Moderate-to-vigorous physical activity, like speed walking, running, or cycling, may have clear and consistent benefits for both physical and mental health (Piercy et al., 2018).

Exercise has many benefits. It has been seen to strengthen the muscle of the heart,



improve cholesterol levels, and lower blood pressure (Warburton, Nicol, & Bredin, 2006). It also increases the ability of the body to transport and use oxygen, and this would lead to higher stamina. Resistance training, which involves exercises like using resistance bands or even weightlifting, helps in building muscle mass, which helps in metabolism, strength, and functional independence as individuals get older. Physical activity levels may be measured through direct calorimetry, indirect calorimetry, doubly labelled water, or self-reports like questionnaires. Beyond physical health, exercise helps in mental and emotional well-being. Engaging in physical activity triggers a release of endorphins, and endorphins help boost the mood and relieve pain (Sharma, Madaan, & Petty, 2006). It provides an outlet for stress, increases self-esteem, and offers opportunities for social interaction (especially when done in public spaces), enhancing the overall well-being of an individual. Regular exercise interacts with the HPA axis by lowering stress-induced cortisol responses and hastening HPA recovery through a negative feedback loop (Chen et al., 2022).

## **Theoretical Framework**

This review revolves around theories that help explain how our minds, bodies, and everyday lives work together when we face stress. These theories show that it is not just the stressful event itself that matters, but how it is perceived and handled over time. By looking at stress from different angles, these theories give a fuller picture of what really happens inside the body.

### ***The Transactional Model of Stress and Coping***

This model was originated by Richard Lazarus and Susan Folkman in 1984. It is a psychological framework that explains stress as an evolving process involving an individual's comprehension and reaction to events. This accentuates the significance of cognitive appraisal in regulating how individuals recognise and react to stressors. According to Lazarus and Folkman (1984), stress is a consequence of interactions between a person and the environment. The important concepts of the transactional model include:

1. **Cognitive Appraisal:** This states that stress is not solely due to the stressor itself, but rather how an individual interprets and evaluates



the stressor. Cognitive appraisal is further divided into:

- **Primary Appraisal:** This involves assessing if an event, mostly significant, is irrelevant, threatening, challenging, positive, or stressful. Stressful events can be further classified as loss (past damage), threat (likely future harm), or challenge (capacity for growth). The initial lens through which stress is interpreted plays a role in determining emotional and physical responses.
- **Secondary Appraisal:** Once an event is concluded to be a stressor, the individual then evaluates the resources and coping options available to manage the stressful situation, to determine which ones will help. Resources might be external (for instance, financial help) or internal (confidence). A study by Van der Heijden and his colleagues (2019) showed that nurses who held the

belief that they had the right tool to cope with their demanding jobs had less chances of burn out, even when they faced high pressures from work. Knowles and his colleagues (2023) also found that people living with chronic illnesses coped better when they felt more equipped to handle the stress their condition brought.

2. **Reappraisal:** This is the ongoing process of reassessing the situation and one's coping strategies as new information becomes available.
3. **Coping:** This refers to the cognitive and behavioural attempts to control internal and external dictates that are recognised as burdensome. This is a result of the appraisal done by the individual. Coping is further divided into:

- **Problem-focused coping:** Using or channelling available resources directly addresses the problem to change the stressful situation. This works best when a person feels that



they have the capacity to influence a situation.

- Emotion-focused coping: Aiming to regulate the emotional distress that arises as a response to the stressor. This is helpful when a situation is beyond a person's control.

4. Outcomes: In response to coping, there are both immediate outcomes and long-term outcomes. The immediate outcomes include physiological and emotional responses, and the long-term outcomes include psychological well-being, social functioning, and health.

Within this framework, acute stress triggers cognitive appraisal processes that shape physiological outcomes. Primary and secondary appraisals influence cardiovascular reactivity by determining whether the stressor is perceived as threatening and the resources available to address it. Emotional regulation functions as a moderating variable by guiding the choice and effectiveness of coping strategies, thereby influencing both the magnitude of cardiovascular reactivity and the rate of cardiovascular recovery.

### *The Theory of Allostasis and Allostatic Load*

This theory was developed by Bruce McEwen in 1993. This model sees stability not as rigid control, but as stability through change. Allostasis involves acclimation to acute stress, involving the release of hormones like glucocorticoids and catecholamines, which help in restoring homeostasis. These hormones are essential for adaptation and survival in the short term. These stress mediators, while protective in the short run, can be damaging over longer periods. For instance, glucocorticoids help replenish energy reserves during acute stress, but this can contribute to obesity and insulin resistance when chronically elevated. Similarly, catecholamines are necessary for blood pressure regulation but can promote atherosclerosis when repeatedly elevated. Allostasis involves the brain anticipating what the body will need and adjusting ahead of time. This is coordinated across several systems in the body, including the nervous system, the stress hormone system, the cardiovascular system, the immune system, and metabolism (Logan & Barksdale, 2008).

On the other hand, allostatic load is the eventual wear and tear on the body due to chronic stress or inefficient stress



responses. It represents the physiological cost of prolonged or repeated adaptation to stress, which can amount to different health issues, including cardiovascular disease. It refers to the cumulative strain on the body that builds up over time, due to repeated or inefficient efforts to manage stress. Patterns of allostatic load include repeated stress, lack of adaptation, extended reactions due to delayed shutdown, and insufficient response. These patterns emphasise that it is not just the presence of stress, but also the body's ability to respond to and recover from stress that impacts health. Psychological factors like emotional regulation can influence the perception of stress and the physiological response to it, affecting the allostatic load. Lifestyle factors like physical activity levels are shown in the document to contribute to or influence allostatic load. Allostatic load can increase when the stress responses are triggered too often, the response does not shut off properly, or fails to adapt to repeated exposure to stress (Romero, Dickens, & Cyr, 2009). Scientists use markers like blood pressure, HRV, and inflammation-related substances to assess allostatic load. When these markers show problems across different systems, it could signal high allostatic load, which could mean that the

body is under significant physiological stress and may be at a greater risk for diseases (Juster, McEwen, & Lupien, 2010). This theory explains how acute stress leads to physiological adjustments to maintain stability through change. Effective emotional regulation moderates the efficiency of stress-response activation and deactivation, and this influences cardiovascular reactivity and recovery. Physical activity levels also moderate the process by enhancing cardiovascular resilience and reducing allostatic load. Prolonged or poorly regulated stress responses can increase cardiovascular reactivity and delay recovery.

### ***Self-Regulation Theory***

Self-Regulation Theory was developed by Roy Baumeister and his colleagues in the 1990s. Self-Regulation Theory (SRT) involves the manner in which individuals control and direct their thoughts, emotions, and behaviours to achieve goals or respond effectively to internal or external demands. Rooted in psychology and health behaviour research, the theory emphasises an individual's ability to monitor and modify responses to meet situational or long-term demands, especially under stress. SRT describes self-regulation as a process unfolding in cycles. Zimmerman (2000) divides the cycle into three phases:



forethought, performance, and self-reflection. In the forethought phase, people get ready for action. They set goals, plan how to achieve them, and believe in their own ability to excel. This is called self-efficacy (Schunk & Mullen, 2012). In the performance phase, plans are put into motion. People focus their attention, manage their time, and ask for help when necessary. In the self-reflection phase, people evaluate how things went with respect to their goals.

In the context of acute stress, self-regulation helps in determining physiological and behavioural outcomes. When exposed to a stressor, the body undergoes changes in cardiovascular function as part of the stress response. Following the stressor, cardiovascular recovery refers to the body's return to baseline physiological levels. According to SRT, an individual's ability to manage this process effectively hinges on self-regulatory capacities. Some components of self-regulation include emotional regulation and coping strategies, both of which serve as psychological moderators in stress reactivity and recovery. Effective self-regulation through adaptive emotion regulation and active coping strategies can reduce physiological arousal and enhance recovery, while poor regulation may

prolong stress responses, contributing to dysregulated cardiovascular patterns. SRT also recognises the influence of lifestyle factors on self-regulation. Behaviours such as physical activity contribute to or detract from the body's self-regulatory capacity. For instance, sleep deprivation impairs cognitive control and emotional regulation, while regular physical activity has been shown to enhance stress resilience and autonomic flexibility. Self-Regulation Theory helps in understanding how both psychological and lifestyle factors moderate the relationship between acute stress and cardiovascular outcomes. It supports the hypothesis that individuals with stronger self-regulatory capacities, through emotion regulation, effective coping, and healthy lifestyle habits, will exhibit more moderate cardiovascular reactivity and faster recovery following acute stress. Self-Regulation Theory shows how individuals control their responses during acute stress, with self-regulatory capacities that influence cardiovascular reactivity and recovery. Emotional regulation directly moderates the stress–cardiovascular link by reducing physiological arousal and supporting quicker return to baseline. Physical activity strengthens self-regulatory capacity, enabling more adaptive responses to acute





stress. Together, these moderators shape how the body responds to and recovers from cardiovascular strain during stress.

### ***Biopsychosocial Theory***

This model was propounded by Engel (1977). Engel argued for a shift from the traditional biomedical model, which was inadequate in addressing contemporary health challenges, to a biopsychosocial model. The Biopsychosocial Model appraises the interactivity of biological, psychological, and social factors in health and disease. The biological aspect includes genetics, immune responses, organ function, and risk for diseases. The psychological aspect deals with how we think, feel, and behave. It includes stress levels, mood, personality, and confidence. The social aspect involves relationships, communities, cultural beliefs, income, education, and access to healthcare. Engel illustrated the model by comparing diabetes and schizophrenia, stating that both conditions are influenced by biological, psychological, and social factors. This model supports including psychological (emotional regulation and coping strategies) and lifestyle (sleep quality and physical activity levels) factors as moderating variables in the association between acute stress and cardiovascular reactivity and recovery. It suggests that

cardiovascular reactivity and recovery in response to acute stress cannot be solely explained by biological mechanisms. Instead, psychological and lifestyle factors play significant roles in modulating these cardiovascular responses. For instance, emotional regulation and coping strategies may affect how individuals perceive and respond to stress, affecting cardiovascular reactivity. Similarly, lifestyle factors like sleep quality, physical activity levels, and BMI can affect one's physiological responses to stress and the rate of recovery.

The biopsychosocial models lay emphasis on a more compassionate, patient-centred approach to healthcare, going beyond just diagnosing and treating a disease to listening to patients' stories, understanding their personal and social circumstances, and designing treatment plans that would incorporate their whole experience. This model places acute stress within a connection of biological, psychological, and social factors. Cardiovascular reactivity and recovery are affected not only by biological mechanisms but also by psychological moderators like emotional regulation and lifestyle moderators such as physical activity levels. Emotional regulation affects how acute stress is perceived and managed, while physical activity supports cardiovascular efficiency



and faster recovery. The interaction of these moderators determines the overall impact of acute stress on cardiovascular outcomes.

### **Empirical Framework**

This empirical framework synthesises evidence on how stress influences cardiovascular functioning, focusing on reactivity and recovery processes. It considers studies demonstrating that both heightened and blunted cardiovascular responses to stress can have significant health implications. It also examines how acute stress, emotional regulation, and physical activity interact to shape cardiovascular outcomes.

### ***Cardiovascular Reactivity***

Cardiovascular reactivity (CVR) refers to changes in cardiovascular function, like the heart rate (HR) and blood pressure (BP), in response to a stressor (Chen et al., 2022; Creaven et al., 2020; McMahon et al., 2021). These changes can also include alterations in cardiac output and total peripheral resistance. Research has consistently exhibited that individuals exhibit considerable variability in their cardiovascular and cortisol responses to acute psychological stress, with the reactivity hypothesis stating that increased

cardiovascular reactions to acute stress elevate the chance of developing subsequent cardiovascular disease (CVD), hypertension, and overall poorer cardiovascular health (Chen et al., 2022; Creaven et al., 2020; Gallagher et al., 2018; McMahon et al., 2021). Research shows that frequent subjection to stressors can lead to a tendency to display high cardiovascular reactivity and retarded recovery, which can negatively affect the internal balance and potentially activate or worsen various pathological changes involving the cardiovascular system. Notably, individuals who display exaggerated cardiovascular reactions to a stressor face a heightened risk for the untimely progression of hypertension and more antecedents to coronary heart disease, as well as unfavourable clinical cardiovascular events and premature cardiovascular mortality (Chen et al., 2022). A longitudinal study of young adults by De Rooij (2013) showed that those who had heightened systolic blood pressure during mental stress were more likely to develop heightened resting blood pressure over time. However, not all concerns are about heightened reactivity. A study by Carroll and his colleagues (2001) showed that men with little cardiovascular or hormonal reaction to acute stress were



more likely to die earlier, countering the assumption that low reactivity was always a good thing. A meta-analysis by Chida and Steptoe (2010) showed that individuals with higher reactivity to acute stress had poorer recovery. In patients already diagnosed with heart disease, blunted reactions have been linked to worse outcomes, and this shows that both overreacting and underreacting to stress can be harmful.

### ***Cardiovascular recovery***

Cardiovascular recovery has been defined as the process by which cardiovascular functions return to baseline levels after a stressor has been removed (Chida & Steptoe, 2010). A review of studies by Pierpont, Stolpman, and Gornick (2000) showed that how quickly the heart rate slows down after activity can be a reliable sign of parasympathetic nervous system recovery, which is also a core component of cardiovascular recovery. Steptoe and Marmot (2005) carried out a study to check for cardiovascular recovery after stress among middle-aged British adults and they found that slower SBP was predictive of greater coronary artery disease three years later, showing that impaired recovery may be a warning sign for cardiovascular disease in the future, moving the focus from just reactivity to

the importance of the recovery phase too. Chida and Steptoe (2010) also conducted a meta-analysis on cardiovascular reactions to stress, and the findings showed that delayed recovery predicted poor cardiovascular outcomes, including hypertension and mortality. They also found that recovery had a stronger predictive value for health issues in the future, compared to reactivity. A study by Borresen and Lambert (2008) found that a slower heart rate recovery was associated with an increased risk of cardiovascular mortality. Sloan and his colleagues (2008) also found that individuals with higher heart rate variability (HRV) showed faster cardiovascular recovery following both acute physical and psychological stress. Recent studies show the significance of cardiovascular recovery as an indicator of autonomic and cardiovascular health. Leal-Menezes and his colleagues (2025) observed that heart rate variability recovery was slower in the first 10 minutes after high-intensity interval exercise compared with moderate-intensity exercise, although values returned to baseline within one hour. Yilmaz et al. (2024) also showed that patients with coronary artery disease experienced measurable improvements in heart rate recovery after a structured cardiac



rehabilitation program, and this shows the possibility of certain interventions to restore autonomic balance.

### ***Acute Stress and Its Effect on Cardiovascular Reactivity and Cardiovascular Recovery***

Acute stress, which is a short-term stressor, initiates immediate physiological and psychological responses within the body. The impact of stress on physiological health is documented, with cardiovascular reactivity (CVR) being a significant pathway through which this occurs (McMahon et al., 2021). Population studies have associated high cardiovascular responses to laboratory stress exposures with conditions such as hypertension, atherosclerosis (Barnett, Spence, Manuck, & Jennings, 1997), increased left ventricular mass, and higher cardiovascular disease mortality (Carroll et al., 2012).

A meta-analysis by Chida and Steptoe (2010) found that large cardiovascular responses to stress, including high reactivity and slow post-test recovery, are speculated to contribute to the increase in blood pressure (BP) and the rise of cardiovascular disease (CVD). Also, increased and elongated blood pressure reactions to psychological stress forecast

cardiovascular morbidity and mortality. Large psychobiological reactions to acute psychological stress can stimulate unfavourable cardiovascular events, and their regular occurrence over time increases the risk of CVD mortality and mishaps (Chauntry et al., 2022). A longitudinal study of healthy adults over thirteen years by Matthews and his colleagues (2004) found that individuals with stronger blood pressure reactions to acute stress were more likely to develop hypertension later on, even after accounting for their baseline blood pressure readings and other factors.

### ***Emotional Regulation as a Moderating Factor***

Difficulties in emotional regulation have been associated with a range of mental and physical health issues, with the inclusion of cardiovascular disease (CVD). Emotional regulation encompasses the strategies individuals employ to manage and respond to their emotional experiences, and these individual differences translate to varying patterns of cardiovascular response to stress (Griffin & Howard, 2022). A study by Whiston and his colleagues (2025) indicates that psychological factors, such as emotional



dysregulation, can influence how the cardiovascular system reacts to stress. It has been found that individuals who struggle with emotional regulation tend to exhibit heightened cardiovascular reactivity and a slower recovery period following acute stress. In support of this, the use of reappraisal as a stress regulation technique is associated with a more adaptive cardiovascular response, characterised by low cardiovascular reactivity (Griffin & Howard, 2022). A study by Jentsch and Wolf (2020) found that individuals using reappraisal showed greater HRV after a stressor, while those using suppression had elevated cortisol levels, which indicates a more intense stress response. Another study by Whiston and his colleagues (2025) found that anhedonia (absence of the ability to experience pleasure) symptoms were connected to blunted systolic blood pressure (SBP) and heart rate (HR) reactions to acute psychological stress, supporting the idea that anhedonia plays a significant role in the connection between depression and blunted CVR. The study by Jentsch and Wolf (2020) also discovered that individuals who struggle to control impulses when upset exhibited dampened heart rate and blood pressure in response to stress, and showed impaired

cardiovascular habituation over time. Recent experimental evidence among Nigerian university students further supports this link, showing that effective emotion regulation, particularly lower emotional lability, was associated with faster pulse rate recovery following acute stress (Agberotimi, Akhibi, & Agoha, 2025). Effective emotional regulation appears to reduce the impact of stress on the cardiovascular system, with individuals who can effectively manage their emotions showing lower cardiovascular reactivity and a quicker return to baseline (Griffin & Howard, 2022).

### ***Physical Activity Levels as a Moderating Factor***

According to Alves (2024), moderate to vigorous physical activity is positively associated with better HRV indices. Research indicates that regular exercise improves cardiovascular responses to acute stress by lowering the magnitude of hyperarousal and enhancing cardiovascular recovery after stress exposure. Also, studies have shown that exercise training can reduce stress reactivity and improve cardiac autonomic control. A single bout of exercise may also impact cardiovascular responses to stress (Chen et al., 2022). A



study by Nigade and his colleagues (2023) found that exercise affects the cortisol response to stress, suggesting that physical activity can influence cardiovascular reactivity and the cortisol response to stress. However, findings concerning the relationship between physical fitness and cardiovascular reactivity to stress have been mixed. A study by De Geus, Van Doornen, & Orlebeke (1993) showed that more physically fit individuals exhibited enhanced reactivity, while another study by Crews and Landers (1987) found blunted reactivity. This blunted reactivity was supported by a study by Hamer, Taylor, & Steptoe (2006), which found that physically active individuals (aerobics) tend to show a reduced increase in heart rate and blood pressure compared to sedentary individuals when faced with stressful tasks. Consistent with these findings, experimental evidence from Nigerian students revealed that higher physical activity significantly moderated systolic blood pressure reactivity, contributing to more adaptive cardiovascular responses under stress (Agberotimi et al., 2025).

### **Integrated Synthesis of Theories and Empirical Findings**

Understanding the impact of acute stress on cardiovascular functioning would need an integrated lens that merges theoretical frameworks, empirical findings, and moderating factors. This review shows that cardiovascular reactivity (CVR) and cardiovascular recovery are important physiological responses that mediate between stress and cardiovascular health (Creaven et al., 2020; Chen et al., 2022). The Reactivity Hypothesis states that exaggerated or blunted cardiovascular responses to acute stress would predict future cardiovascular disorders such as hypertension and coronary heart disease (Krantz & Manuck, 1984; Carroll et al., 2012; Chen et al., 2022).

The Transactional Model of Stress and Coping (Lazarus & Folkman, 1984) supports the idea that how an individual appraises and copes with stress would determine physiological outcomes. For instance, studies have shown that individuals who use reappraisal tend to have lower CVR and better recovery, while those who rely on suppression show higher physiological arousal and slower recovery (Griffin & Howard, 2022; Jentsch & Wolf, 2020). This aligns with the Self-Regulation Theory (Baumeister, 1990; Zimmerman, 2000), which shows emotional regulation and lifestyle choices





as critical self-regulatory strategies that regulate the impact of stress. More empirical support comes from studies that document individual variability in CVR and cardiovascular recovery. High reactivity and delayed recovery have been associated with increased risk of hypertension and cardiovascular mortality (Chida & Steptoe, 2010; Matthews et al., 2004; Steptoe & Marmot, 2005). Both exaggerated and blunted cardiovascular responses are maladaptive. Carroll et al. (2001) found that men with blunted stress responses were more likely to die earlier, while De Rooij (2013) observed that higher systolic blood pressure during stress predicted higher resting BP later in life.

These empirical findings are also contextualised by the Theory of Allostasis and Allostatic Load (McEwen, 1998), which provides a biological explanation of how repeated or poorly regulated stress responses lead to cumulative wear and tear on the body. The interaction between stress exposure and poor emotional regulation or low physical activity contributes to higher allostatic load, and this would impair cardiovascular health over time (Juster, McEwen, & Lupien, 2010; Romero, Dickens, & Cyr, 2009). Also, physical activity is an important lifestyle factor that moderates stress responses. Regular

exercise is associated with improved heart rate variability (HRV), reduced cortisol reactivity, and faster cardiovascular recovery (Chen et al., 2022; Alves et al., 2024). However, findings are mixed: while some studies state that physically fit individuals show reduced cardiovascular reactivity (Hamer, Taylor, & Steptoe, 2006), others show enhanced reactivity, especially under certain task conditions (De Geus et al., 1993; Crews & Landers, 1987).

Finally, the Biopsychosocial Model (Engel, 1977) shows the interactions among biological systems, psychological functioning, and social context, stating that cardiovascular health is influenced by not only genetic and physiological predispositions, but also by cognitive appraisals, coping styles, emotional regulation, and lifestyle behaviours. For instance, poor emotional regulation (e.g., impulsivity or anhedonia) may blunt or exaggerate stress responses (Whiston et al., 2025), while moderate-to-vigorous physical activity may reduce these responses, improving cardiovascular outcomes over time (Nigade et al., 2023). Conclusively, the review shows that the relationship between acute stress and cardiovascular outcomes is moderated by both psychological factors (e.g., emotional



regulation) and lifestyle behaviours (e.g., physical activity). These variables, in interaction with the individual's biopsychosocial context, determine the degree of vulnerability or resilience to cardiovascular morbidity and mortality.

## References

- Agberotimi, S. F., Akhibi, P. T., & Agoha, B. C. (2025). An experimental study on the effects of psychological and lifestyle factors on cardiovascular reactivity and recovery from acute stress among Covenant University students. *Nnadiebube Journal of Social Sciences*.
- Al'Absi, M. (2006). Hypothalamic–Pituitary–Adrenocortical responses to psychological stress and risk for smoking relapse. *International Journal of Psychophysiology*, 59(3), 218–227. doi:10.1016/j.ijpsycho.2005.10.010
- Aldao, A., Sheppes, G., & Gross, J. J. (2015). Emotion Regulation Flexibility. *Cognitive Therapy and Research*, 39(3), 263–278. doi:10.1007/s10608-014-9662-4
- Alves, S. F. L., Santos, T. A. B. P. de S., da Silva, M. L., & Cunha, K. da C. (2024). Heart rate variability, sleep quality and physical activity in medical students. *Sleep Epidemiology*, 5, 100105. doi:10.1016/j.sleep.2024.100105
- American College of Sports Medicine. (2025). *Physical Activity Guidelines*. ACSM. Retrieved from <https://acsm.org/education-resources/trending-topics-resources/physical-activity-guidelines/>
- Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10(6), 410–422. National Library of Medicine. doi:10.1038/nrn2648
- Barnett, P. A., Spence, J. D., Manuck, S. B., & Jennings, J. R. (1997). Psychological stress and the progression of carotid artery disease. *Journal of Hypertension*, 15(1), 49–55. doi:10.1097/00004872-199715010-00004
- Baumeister, R. F., & Vohs, K. D. (2007). Self-Regulation, Ego Depletion, and Motivation. *Social and Personality Psychology Compass*, 1(1), 115–128. doi:10.1111/j.1751-9004.2007.00001.x
- Blascovich, J. J., & Katkin, E. S. (1993). *Cardiovascular reactivity to psychological stress & disease*. American Psychological Association. doi:10.1037/10125-000
- Borresen, J., & Lambert, M. I. (2008). Autonomic Control of Heart Rate during and after Exercise. *Sports Medicine*, 38(8), 633–646. doi:10.2165/00007256-200838080-00002
- Calkins, S. D., Dollar, J. M., & Wideman, L. (2019). Temperamental vulnerability to emotion dysregulation and risk for mental and physical health challenges. *Development and Psychopathology*, 31(3), 957–970. doi:10.1017/s0954579419000415
- Cannon, W. B., & de la Paz, D. (1911). Emotional Stimulation of Adrenal Secretion. *American Journal of Physiology-Legacy Content*, 28(1),



- 64–70.  
doi:10.1152/ajplegacy.1911.28.1.64
- Carroll, D., Ginty, A. T., Der, G., Hunt, K., Benzeval, M., & Phillips, A. C. (2012). Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. *Psychophysiology*, 49(10), 1444–1448. doi:10.1111/j.1469-8986.2012.01463.x
- Carroll, D., Ginty, A. T., Whittaker, A. C., Lovallo, W. R., & De Rooij, S. R. (2017). The behavioural, cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress. *Neuroscience & Biobehavioral Reviews*, 77, 74–86. doi:10.1016/j.neubiorev.2017.02.025
- Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Reports*, 100(2), 126–131. Retrieved from <https://pubmed.ncbi.nlm.nih.gov/3920711/>
- Charles, S. T., & Carstensen, L. L. (2010). Social and Emotional Aging. *Annual Review of Psychology*, 61(1), 383–409. doi:10.1146/annurev.psych.093008.100448
- Chantry, A. J., Bishop, N. C., Hamer, M., Kingsnorth, A. P., Chen, Y.-L., & Paine, N. J. (2022). Sedentary behaviour is associated with heightened cardiovascular, inflammatory and cortisol reactivity to acute psychological stress. *Psychoneuroendocrinology*, 141, 105756. doi:10.1016/j.psyneuen.2022.105756
- Chen, W. J., Mat Ludin, A. F., & Farah, N. M. F. (2022). Can Acute Exercise Lower Cardiovascular Stress Reactivity? Findings from a Scoping Review. *Journal of Cardiovascular Development and Disease*, 9(4), 106. doi:10.3390/jcdd9040106
- Chida, Y., & Steptoe, A. (2010). Greater Cardiovascular Responses to Laboratory Mental Stress Are Associated With Poor Subsequent Cardiovascular Risk Status. *Hypertension*, 55(4), 1026–1032. doi:10.1161/hypertensionaha.109.146621
- Creaven, A. M., Higgins, N. M., Ginty, A. T., & Gallagher, S. (2020). Social support, social participation, and cardiovascular reactivity to stress in the Midlife in the United States (MIDUS) study. *Biological Psychology*, 155, 107921. doi:10.1016/j.biopsycho.2020.107921
- Crews, D. J., & Landers, D. M. (1987). A meta-analytic review of aerobic fitness and reactivity to psychosocial stressors. *Medicine and Science in Sports and Exercise*, 19(5 Suppl), S114–120. Retrieved from <https://pubmed.ncbi.nlm.nih.gov/316910/>
- De Geus, E. J., Van Doornen, L. J., & Orlebeke, J. F. (1993). Regular exercise and aerobic fitness in relation to psychological make-up and physiological stress reactivity. *Psychosomatic Medicine*, 55(4), 347–363. doi:10.1097/00006842-199307000-00003



- De Rooij, S. R. (2013). Blunted cardiovascular and cortisol reactivity to acute psychological stress: A summary of results from the Dutch Famine Birth Cohort Study. *International Journal of Psychophysiology*, 90(1), 21–27. doi:10.1016/j.ijpsycho.2012.09.011
- Diaz, K. M., Howard, V. J., Hutto, B., Colabianchi, N., Vena, J. E., Safford, M. M., Blair, S. N., & Hooker, S. P. (2017). Patterns of Sedentary Behavior and Mortality in U.S. Middle-Aged and Older Adults. *Annals of Internal Medicine*, 167(7), 465. doi:10.7326/m17-0212
- Eckelkamp, S. (2024). Your Heart Rate Recovery May Be a Significant Marker of Cardiovascular Health: Here's Why. *Peloton Interactive*. Retrieved from <https://www.onepeloton.com/blog/heart-rate-recovery>
- Engel, G. L. (1977). The Need for a New Medical model: a Challenge for Biomedicine. *Science*, 196(4286), 129–136. doi:10.1126/science.847460
- Folkman, S., & Lazarus, R. (1984). Stress: Appraisal and coping. *Encyclopedia of Behavioral Medicine*, 1(1), 1913–1915. doi:10.1007/978-1-4419-1005-9\_21
- Ford, B. Q., & Mauss, I. B. (2015). Culture and emotion regulation. *Current Opinion in Psychology*, 3(1), 1–5. <https://doi.org/10.1016/j.copsyc.2014.12.004>
- Ginty, A. T., Tyra, A. T., Young, D. A., Brindle, R. C., De Rooij, S. R., & Williams, S. E. (2022). Cardiovascular reactions to acute psychological stress and academic achievement. *Psychophysiology*, 59(10). doi:10.1111/psyp.14064
- Griffin, S. M., & Howard, S. (2021). Individual differences in emotion regulation and cardiovascular responding to stress. *Emotion*. doi:10.1037/emo0001037
- Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74(1), 224–237. doi:10.1037//0022-3514.74.1.224
- Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition & Emotion*, 25(3), 400–412. doi:10.1080/02699931.2010.544160
- Hamer, M., Taylor, A., & Steptoe, A. (2006). The effect of acute aerobic exercise on stress related blood pressure responses: A systematic review and meta-analysis. *Biological Psychology*, 71(2), 183–190. doi:10.1016/j.biopsycho.2005.04.004
- Jentsch, V. L., & Wolf, O. T. (2020). The impact of emotion regulation on cardiovascular, neuroendocrine and psychological stress responses. *Biological Psychology*, 154, 107893. doi:10.1016/j.biopsycho.2020.107893
- Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience &*



- Biobehavioral Reviews*, 35(1), 2–16.  
doi:10.1016/j.neubiorev.2009.10.002
- Knowles, S. R., Apputhurai, P., Jenkins, Z., O’Flaherty, E., Ierino, F., Langham, R., ... Castle, D. J. (2023). Impact of chronic kidney disease on illness perceptions, coping, self-efficacy, psychological distress and quality of life. *Psychology, Health & Medicine*, 1–14.  
doi:10.1080/13548506.2023.2179644
- Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. *Psychological Bulletin*, 96(3), 435–464. Retrieved from <https://pubmed.ncbi.nlm.nih.gov/6393178/>
- Leal-Menezes, R., Rodrigues-Krause, J., dos Santos, G. C., do Nascimento Queiroz, J., Silva da Silva, C., Umpierre, D., & Reischak-Oliveira, A. (2025). High-intensity interval aerobic exercise delays recovery from heart rate variability: a systematic review with meta-analysis. *Clinical Autonomic Research*. doi:10.1007/s10286-024-01103-7
- Lei, M.-K., Berg, M. T., Simons, R. L., Simons, L. G., & Beach, S. R. H. (2020). Childhood adversity and cardiovascular disease risk: An appraisal of recall methods with a focus on stress-buffering processes in childhood and adulthood. *Social Science & Medicine*, 246, 112794. doi:10.1016/j.socscimed.2020.112794
- Logan, J. G., & Barksdale, D. J. (2008). Allostasis and allostatic load: expanding the discourse on stress and cardiovascular disease. *Journal of Clinical Nursing*, 17(7b), 201–208. doi:10.1111/j.1365-2702.2008.02347.x
- Lovallo, W. R. (2016). *Stress and Health: Biological and Psychological Interactions*. doi:10.4135/9781071801390
- Matsumoto, D. (2007). Culture, Context, and Behavior. *Journal of Personality*, 75(6), 1285–1320. doi:10.1111/j.1467-6494.2007.00476.x
- Matthews, K. A., Katholi, C. R., McCreath, H., Whooley, M. A., Williams, D. R., Zhu, S., & Markovitz, J. H. (2004). Blood Pressure Reactivity to Psychological Stress Predicts Hypertension in the CARDIA Study. *Circulation*, 110(1), 74–78. doi:10.1161/01.cir.0000133415.37578.e4
- McEwen, B. S., & Gianaros, P. J. (2011). Stress- and Allostasis-Induced Brain Plasticity. *Annual Review of Medicine*, 62(1), 431–445. doi:10.1146/annurev-med-052209-100430
- McMahon, G., Creaven, A.-M., & Gallagher, S. (2021). Cardiovascular reactivity to acute stress: Attachment styles and invisible stranger support. *International Journal of Psychophysiology*, 164, 121–129. doi:10.1016/j.ijpsycho.2021.03.005
- McRae, K., & Gross, J. J. (2020). Emotion regulation. *Emotion*, 20(1), 1–9. doi:10.1037/emo0000703





- Meichenbaum, D. (1985). Stress inoculation training. New York Pergamon Press. *Scientific Research Publishing*. (n.d.). Retrieved from <https://www.scirp.org/reference/referencespapers?referenceid=1015711>
- Mendez, Y. P., Ralston, P. A., Wickrama, K., Bae, D., Young-Clark, I., & Ilich, J. Z. (2018). Lower life satisfaction, active coping and cardiovascular disease risk factors in older African Americans: outcomes of a longitudinal church-based intervention. *Journal of Behavioral Medicine*, 41(3), 344–356. doi:10.1007/s10865-017-9909-0
- Nigade, P. S., Varshney, V., & Bedi, M. (2023). Effect of acute exercise on Cardiovascular and Serum cortisol reactivity to mental stress in offspring of hypertensives. *MAMC Journal of Medical Sciences*, 9(1), 1–6. doi:10.4103/mamcjms.mamcjms\_2\_23
- Okoronta B., (2002). Personality And Haemodynamic Patterns in Stress and Non-Stress Conditions.
- Piercy, K. L., Troiano, R. P., Ballard, R. M., Carlson, S. A., Fulton, J. E., Galuska, D. A., ... Olson, R. D. (2018). The physical activity guidelines for Americans. *JAMA*, 320(19), 2020–2028. doi:10.1001/jama.2018.14854
- Pierpont, G. L., Stolpman, D. R., & Gornick, C. C. (2000). Heart rate recovery post-exercise as an index of parasympathetic activity. *Journal of the Autonomic Nervous System*, 80(3), 169–174. doi:10.1016/s0165-1838(00)00090-4
- Romero, L. M., Dickens, M. J., & Cyr, N. E. (2009). The reactive scope model — A new model integrating homeostasis, allostasis, and stress. *Hormones and Behavior*, 55(3), 375–389. doi:10.1016/j.yhbeh.2008.12.009
- Saunders, T. J., McIsaac, T., Douillette, K., Gaulton, N., Hunter, S., Rhodes, R. E., ... Healy, G. N. (2020). Sedentary behaviour and health in adults: An overview of systematic reviews. Sedentary behaviour and health in adults: an overview of systematic reviews. *Applied Physiology, Nutrition, and Metabolism*, 45(10 (Suppl. 2)), S197–S217. doi:10.1139/apnm-2020-0272
- Schore, A. (2022). Right brain-to-right brain psychotherapy: recent scientific and clinical advances. *Annals of General Psychiatry*, 21(1). doi:10.1186/s12991-022-00420-3
- Schunk, D. H., & Mullen, C. A. (2012). Self-Efficacy as an Engaged Learner. *Handbook of Research on Student Engagement*, 219–235. doi:10.1007/978-1-4614-2018-7\_10
- Sharma, A., Madaan, V., & Petty, F. D. (2006). Exercise for mental health. *Primary Care Companion to the Journal of Clinical Psychiatry*, 8(2), 106. doi:10.4088/pcc.v08n0208a
- Sloan, R. P., Huang, M.-H., McCreath, H., Sidney, S., Liu, K., Williams, O. D., & Seeman, T. (2008). Cardiac autonomic control and the effects of age, race, and sex: The CARDIA study. *Autonomic Neuroscience*,





- 139(1–2), 78–85.  
doi:10.1016/j.autneu.2008.01.006
- Southwick, F., Martini, B., Charney, D., & Southwick, S. (2017). (PDF) *Leadership and Resilience*. ResearchGate. Retrieved from [https://www.researchgate.net/publication/318034847\\_Leadership\\_and\\_Resilience](https://www.researchgate.net/publication/318034847_Leadership_and_Resilience)
- Starcke, K., & Brand, M. (2012). Decision making under stress: A selective review. *Neuroscience & Biobehavioral Reviews*, 36(4), 1228–1248.  
doi:10.1016/j.neubiorev.2012.02.003
- Steptoe, A., Wardle, J., & Marmot, M. (2005). Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *Proceedings of the National Academy of Sciences*, 102(18), 6508–6512.  
doi:10.1073/pnas.0409174102
- Tessler, J., & Bordoni, B. (2023). *Cardiac Rehabilitation*. PubMed; StatPearls Publishing.  
<https://www.ncbi.nlm.nih.gov/books/NBK537196/>
- Tremblay, M. S., Aubert, S., Barnes, J. D., Saunders, T. J., Carson, V., Latimer-Cheung, A. E., Chastin, S. F. M., Altenburg, T. M., & Chinapaw, M. J. M. (2017). Sedentary Behavior Research Network (SBRN) – terminology consensus project process and outcome. *International Journal of Behavioral Nutrition and Physical Activity*, 14(1), Article 75.  
doi:10.1186/s12966-017-0525-8
- Van der Heijden, B., Brown Mahoney, C., & Xu, Y. (2019). Impact of Job Demands and Resources on Nurses' Burnout and Occupational Turnover Intention Towards an Age-Moderated Mediation Model for the Nursing Profession. *International Journal of Environmental Research and Public Health*, 16(11).  
doi:10.3390/ijerph16112011
- Von Dawans, B., Fischbacher, U., Kirschbaum, C., Fehr, E., & Heinrichs, M. (2012). The Social Dimension of Stress Reactivity: Acute Stress Increases Prosocial Behavior in Humans. *Psychological Science*, 23(6), 651–660.  
doi:10.1177/0956797611431576
- Warburton, D. E. R., Nicol, C. W., & Bredin, S. S. D. (2006). Health Benefits of Physical activity: the Evidence. *Canadian Medical Association Journal*, 174(6), 801–809. doi:10.1503/cmaj.051351
- Whiston, A., Keogh, T. M., Howard, S., & Gallagher, S. (2024). Depression and cardiovascular reactions to acute psychological stress: Is anhedonia the driver? *International Journal of Psychophysiology*, 207, 112492.  
doi:10.1016/j.ijpsycho.2024.112492
- Whiston, A., Keogh, T. M., Howard, S., & Gallagher, S. (2024). Depression and cardiovascular reactions to acute psychological stress: Is anhedonia the driver? *International Journal of Psychophysiology*, 207, 112492.  
doi:10.1016/j.ijpsycho.2024.112492
- World Health Organization. (2010). *Global Recommendations on Physical Activity for Health*. Retrieved from <https://www.who.int/publications/item/9789241599979>



Xue, Q., Ji, S., Xu, H., & Yu, S. (2024). O-GlcNAcylation: a pro-survival response to acute stress in the cardiovascular and central nervous systems. *European Journal of Medical Research*, 29(1). doi:10.1186/s40001-024-01773-z

Yilmaz, H., Ozbilgin, N., Ipek, G., Bilir Kaya, B., Yilmaz, M., Karatas, M. B., & Bolca, O. (2024). Effect of cardiac rehabilitation on heart rate recovery in patients with coronary artery disease. *Journal of Surgery and Medicine*, 8(8). doi:10.28982/josam.7751

SRT: Self-Regulation Theory

TSST: Trier Social Stress Test

## ABBREVIATIONS

BEDS: Brief Emotion Dysregulation Scale

BP: Blood Pressure

CI: Confidence Interval

CVR: Cardiovascular Reactivity

CVD: Cardiovascular Disease

DBP: Diastolic Blood Pressure

DERS: Difficulties in Emotion Regulation Scale

DV: Dependent Variable

HPA: Hypothalamic-Pituitary-Adrenal Axis

HR: Heart Rate

HRR: Heart Rate Recovery

HRV: Heart Rate Variability

IV: Independent Variable

PR: Pulse Rate

SAM: Sympatho-Adrenal Medullary System

SBP: Systolic Blood Pressure