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AN EXPERIMENTAL STUDY ON THE EFFECTS OF PSYCHOLOGICAL FACTORS AND LIFESTYLES ON CARDIOVASCULAR REACTIVITY AND RECOVERY FROM ACUTE STRESS AMONG COVENANT UNIVERSITY STUDENTS

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Abstract

Cardiovascular reactivity (CVR) and recovery are physiological responses that reflect the body's adaptation to stress. While these responses are typically adaptive, exaggerated or blunted cardiovascular reactions to acute stress have been linked to adverse health outcomes, including cardiovascular diseases (CVD). This study investigated the effects of acute stress on CVR and recovery among Covenant University students and examined the moderating roles of emotional regulation and physical activity. Sixty undergraduate students (42 female and 18 male) from Covenant University participated in an experimental study involving exposure to an acute stressor (serial subtraction task), during which systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse rate (PR) were measured across baseline, stress exposure, and recovery. Emotional dysregulation was assessed using the Brief Emotion Dysregulation Scale, while physical activity levels were self-reported. Results from paired samples t-tests and repeated-measures ANOVA showed significant increases in SBP, DBP, and PR during stress and significant reductions during recovery, confirming the physiological impact of acute stress. Moderation analyses revealed that emotional regulation, specifically the lability dimension, significantly moderated PR recovery, suggesting that emotional instability can impair autonomic recovery. Meanwhile, physical activity significantly moderated SBP reactivity, with more active individuals showing higher blood pressure responses to stress. However, neither emotional regulation nor physical activity significantly moderated cardiovascular recovery for SBP or DBP. These findings show that psychological and lifestyle factors can influence how the cardiovascular system responds to stress, but their effects are parameter-specific. The study emphasises the need for integrated interventions to improve cardiovascular resilience in Covenant University students within Nigeria.

Keywords: *Cardiovascular Reactivity, Cardiovascular Recovery, Acute Stress, Emotional Regulation, Physical Activity.*

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Introduction

Cardiovascular reactivity (CVR) can be seen as the dynamic changes within the cardiovascular system in response to stressful situations (Okoronta, 2002). The physiological changes in this system involve the heart, the blood itself, and the network of blood vessels, which allows for continuous flow of blood throughout the body. The cardiovascular system is always reactive and adaptive to the metabolic demands of the body. The heart is the major muscular organ that pumps blood throughout the body through rhythmic contractions. Blood flows through the vasculature or blood vessels, made up of the arteries, veins, and capillaries (Okoronta, 2002; Ogiri, 2020). Blood pressure, another major aspect of cardiovascular function, involves the rate of heartbeats, the resistance of the peripheral blood vessels, and the volume of blood emitted with each beat, which is the stroke volume (Okoronta, 2002). The process involving the transportation of blood to and from the heart is known as the cardiac cycle (Ogiri, 2020). Through the aorta, oxygenated blood exits the heart and travels via other arteries to the other parts of the body. The capillaries, the thinnest blood vessels, allow for the passage of nutrients and oxygen from the blood into the cells of the tissues, and also the passage of carbon dioxide into the veins, carrying deoxygenated blood to the heart. The arterial walls are elastic in their structure and function. The amount of blood pumped when the heart contracts, known as the cardiac output, causes the arterial walls to stretch to make room to accommodate the blood pressure and then returns to its diameter between the beats as the heart relaxes (Ogiri, 2020). Systolic blood pressure (SBP) is the pressure of the heart's contraction, while the diastolic blood pressure (DBP) is the relaxation of the heart between beats (Okoronta, 2002). The cardiovascular system communicates with the brain through the autonomic nervous system to facilitate maintenance of rest, or the fight-or-flight response to threats or stressors. These adaptations and responses are collectively referred to as cardiovascular reactivity (Ogiri, 2020). It refers to the physiological alterations in heart rate, blood pressure, or other measures of cardiovascular responses that may occur between the baseline state and the introduction of a stressor (Blascovich & Katkin, 1993). The most common measures of cardiovascular reactivity are heart rate and blood pressure. Heart rate is the number of pulses or heartbeats per minute, and blood pressure is measured by the systole

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and diastole (Okoronta, 2002). While cardiovascular reactivity involves acute alterations in cardiovascular function in response to stressors, cardiovascular recovery involves the process of returning to baseline physiological levels after the stressor has been removed (Forcier et al., 2006). Getting to understand how psychological factors influence cardiovascular responses is important in identifying individuals with higher susceptibility to cardiovascular problems and in developing targeted and effective interventions (Okoronta, 2002; Turner et al., 2020).

Cardiovascular reactivity (CVR) is a normal, adaptive physiological state, but exaggerated CVR to stress has been seen in the progression of cardiovascular diseases (Creaven, Higgins, Ginty, & Gallagher, 2020; Gallagher, O'Riordan, McMahon, & Creaven, 2018; McMahon, Creaven, & Gallagher, 2021). To buttress this, the reactivity hypothesis states that consistently high or elongated cardiovascular reactivity to stress could be germane in the progression of cardiovascular disorders (Gallagher et al., 2018). However, both excessively high and excessively low cardiovascular responses to acute stress can be harmful to health. Also, low levels of cardiovascular responses during stress, known as *blunted reactivity*, are seen to be connected with adverse health results (Carroll, Ginty, Whittaker, Lovallo, & De Rooij, 2017), including depression, poor subjective health, and obesity (McMahon et al., 2021). This shows that a high range of cardiovascular reactions to stress is cardinal in maintaining health.The connection between psychological and cardiovascular health is a major focus of investigation. To be specific, researchers have expressed interest in understanding how psychological stressors and individual differences in the management of emotions and the use of coping can influence cardiovascular reactivity and recovery. The idea that psychological factors influence cardiovascular reactivity dates back several centuries. Ancient perspectives stated that imbalances in bodily humours could influence mental states. This is typical of the humoral theory by Hippocrates and Galen. With time, this theory was overruled by the idea that personality traits are related to biological processes. As health psychology has expanded over the years, the focus has consequently expanded to look into the relationships between physical illness, mental illness, and physical well-being. (Okoronta, 2002).

Psychological factors have also been seen to significantly moderate how individuals

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deal with stressful situations. *Emotional regulation*, which is the capacity to identify, understand, oversee and respond to emotional experiences effectively, is germane in moderating responses to stress (Gallagher et al., 2018). This involves biological, behavioural, and social mechanisms, including automatic and conscious efforts to adjust emotional experiences (Calkins, Dollar, & Widemanet, 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. Problems with emotional regulation contribute 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). Lifestyle factors also have a significant moderating impact on the body's reaction to stress and cardiovascular health. Regular *physical activity* has been seen to have moderating protective effects against cardiovascular disease (Alves, Santos, Da Silva, & Cunha, 2024; Forcier et al., 2006; Nigade, Varshney, & Bedi, 2023), and this would improve cardiovascular function and enhance the body's resilience to stress. Regular exercise has been shown to improve cardiovascular responses to acute stress, and this lowers the magnitude of hyperarousal and enhances cardiovascular recovery after exposure to stress. Some stressful experiences are transient, lasting from seconds to hours, and this is known as *acute stress*. It has been established that acute stress activates the sympatho-medullary (SAM) system, which leads to disturbances in cardiovascular activity and the hypothalamic-pituitary-adrenal (HPA) axis, and this creates altered cortisol levels. In functional conditions, certain brain structures would gauge the situation and orchestrate a habituated form of autonomic and endocrine outrush to brace the physiological and behavioural responses that are necessary to restore homeostasis (Lovallo, 2016; McEwen & Gianaros, 2011). The reactions of the sympathetic nervous system to stress would employ the cardiovascular system and the adrenal medulla to create a "fight-or-flight" preparation. At the same time, the hypothalamic-pituitary-adrenocortical system circulates increased cortisol and adrenaline, which help in mobilising stored energy and regulate the stress response peripherally and within the central nervous system. Exercise has been seen to benefit the HPA axis by reducing stress-instigated

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cortical reactions and accelerating HPA recovery through a negative feedback cycle, thus reducing stress reactivity and improving cardiac autonomic control (Chen et al., 2022).

The relationship between stress and cardiovascular health is important in research, especially among Covenant University students. CVR and the subsequent return to baseline, cardiovascular recovery, are influenced by psychological and lifestyle factors. The motive of this study is to ascertain the outcome of acute stress on cardiovascular function among Covenant University students and the moderating effect of psychological and lifestyle factors. Having an understanding of the relationships can help with the understanding of cardiovascular problems and the appropriate interventions to promote cardiovascular health. The main purpose of this study was to look into the effects of psychological and lifestyle factors on the cardiovascular reactivity and recovery from acute stress among Covenant University students. The research objectives for the study are to investigate the effect of acute stress on cardiovascular reactivity, the effect of acute stress on cardiovascular recovery, the moderating effect of emotional regulation on cardiovascular reactivity to acute stress, the moderating effect of emotional regulation on cardiovascular recovery from acute stress, the moderating effect of physical activity levels on cardiovascular reactivity to acute stress, and the moderating effect of physical activity levels on cardiovascular recovery from acute stress in Covenant University students.

Research Hypotheses

H1: There will be no significant effect of acute stress on cardiovascular reactivity.

H2: There will be no significant effect of acute stress on cardiovascular recovery.

H3: Emotional regulation will not significantly moderate the effect of acute stress on cardiovascular reactivity.

H4: Emotional regulation will not significantly moderate the effect of acute stress on cardiovascular recovery.

H5: Physical activity levels will not significantly moderate the effect of acute stress on cardiovascular reactivity.

H6: Physical activity levels will not significantly moderate the effect of acute stress on cardiovascular recovery.

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Method

Design

This study adopted a within-subjects (repeated measures) experimental design. This design was chosen because there was a manipulation of the measure of the independent variable by the researcher, and the cardiovascular readings were obtained from the same set of participants across three conditions in this study, including baseline, stress exposure, and recovery. The dependent variables for this study were cardiovascular reactivity and cardiovascular recovery. The independent variable, acute stress, was induced using a serial subtraction task. The psychological moderating variables were measured using questionnaires. The lifestyle variables were measured by obtaining direct responses to questions about the number of times individuals engage in exercise per week.

Population of the Study: The target population for this study was students from Covenant University, Ota, Ogun State, with a population of approximately 9,000. Covenant University was chosen as the research setting for various reasons: access to the target population, feasibility, and potential opportunity for intervention application if needed.

Sample and Sampling Techniques: A sample size of 60 participants was recruited for this study from a total population of 9,000 through random sampling. A sample size of 35 was derived through the use of Cohen's formula for a correlational design, but the sample size was increased to ensure statistical accuracy and to account for exclusions in the screening:

Effect size (r): 0.50

Alpha level (α): 0.05

Statistical power (1 – β):0.80

Directionality: Two-tailed test

Population size (N): 9000

***Calculation*:** Cohen's formula for correlation sample size: n0 = (Zα/2 + Zβ /r) ^2 + 3

Where:

Zα/2 = 1.96 (for α = 0.05)

Zβ = 0.84 (for power = 0.80)

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r = 0.50

n0 = (1.96 + 0.84 / 0.50) ^2 + 3

n0 = (2.80 / 0.50) ^2 + 3 = (5.6) ^2 + 3 = 31.36 + 3

= 34.36

≈ 35 participants

Research Instruments

A questionnaire consisting of three sections was used to obtain data in this study, as seen in Appendix A. The first section (A) covers socio-demographic data. Section B covers health information that will be used in the screening phase and also in the measurement of the lifestyle moderating variables. Section C includes the items from the Brief Emotion Dysregulation Scale (BEDS). In the experimental phase, a structured log was used to collect data, as presented in Appendix B. A digital sphygmomanometer was used to obtain the cardiovascular readings, and a serial subtraction task was introduced as a stressor.

Brief Emotion Dysregulation Scale (BEDS): The Brief Emotion Dysregulation Scale (BEDS) was developed by Andrea Wycoff, Sarah Griffin, Ashley Helle, and Timothy Trull (2020). It measures the intensity and dysfunctionality of emotional regulation. BEDS assesses four components: sensitivity, lability, reactivity, and consequences. *Sensitivity* has to do with how easily an individual gets emotionally triggered, or how strongly they react to emotional stimuli. *Lability* refers to the degree of fluctuation in the emotional states of an individual, such as rapid changes in mood. *Reactivity* refers to how intensely an individual responds emotionally to external events. *Consequences* have to do with the impacts or negative outcomes of poorly regulated emotions, such as difficulties in functioning or in relationships.

The BEDS is brief but comprehensive, and it helps in obtaining a quick assessment of emotion dysregulation, especially in clinical settings. It comprises 8 items on a 4-point Likert scale, ranging from "False/Not true at all" to "Very true", rated on a scale of 1 to 4, with higher scores indicating greater emotional dysregulation. The scale has good psychometric properties, with the exploratory factor analysis on Bartlett's test of sphericity being significant (X^2= 2817.20 (28), p <

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0.001), with a sampling adequacy of 0.90. On a test of convergent validity against the Difficulty in Emotional Regulation Scale (DERS), a strong positive correlation was established (r = 0.59). The internal consistency was high across samples (sample 1: α = 0.89; sample 2: α = 0.88; sample 3: α = 0.90) (Wycoff, Griffin, Helle, & Trull, 2020).

Serial Subtraction Task: The Serial Subtraction Task can also be called "Serial Sevens" or "Serial Thirteens". It began in early psychiatric evaluations to assess an individual's concentration, attention span, and working memory. It gained recognition in experimental psychology in the mid-20th century, and researchers found it useful in examining dual-task interference, cognitive fatigue, and information processing. In the 1980s and 1990s, it gained recognition as a reliable stress-induction tool. The serial subtraction test can be used as a standalone or as a part of the Trier Social Stress Test (TSST). TSST was founded by Kischbaum, Pirke, & Hellhammer (1993), and it involves a 5-minute preparation period, a 5-minute public speaking test, and a 5-minute mental arithmetic task.

In a study by Morava, Shirzad, James, Nader, and Prapavessis (2024), serial subtraction was used as part of the Trier Social Stress Test (TSST) to induce acute stress responses in the participants, following a pattern of consecutive subtractions of thirteen from 1,022. Another study by Garrison, Coyle, Baggott, Mendelson, & Galloway (2010) used a subtraction stress task as part of the experimental procedure to induce stress. A study by Hunt (2017) used serial subtraction of sevens to induce acute stress while checking for cardiac function, and significant increases in systolic blood pressure and diastolic blood pressure were recorded.

Digital Sphygmomanometer: The digital sphygmomanometer is an automated, non-invasive device used in clinical practice, home monitoring, and psychophysiological research to measure blood pressure (BP). The measurement of BP can be traced by to Reverend Stephen Hales in 1733, who recorded BP in animals using invasive methods (Noh et al., 2024). With time, the manual sphygmomanometer was developed by Scipione Riva-Rocci in 1896, and the auscultatory method, which made use of Korotkoff sounds, was introduced in 1905 (O'Brien, 2001). Digital sphygmomanometers became famous in the 1970s. It functions through oscillometry,

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and does not require a stethoscope, unlike the manual sphygmomanometer.

As the cuff inflates, it temporarily blocks blood flow, and while it deflates, it detects oscillations in the arterial wall. The microprocessor calculates systolic and diastolic values based on the amplitude of the oscillations, often displaying results within a minute (Parati, Ulian, Santucciu, Omboni, & Mancia, 1995). Digital sphygmomanometers are useful in psychological and cardiovascular research, in cases where continuous or repeated BP monitoring is required. In studies measuring stress reactivity, they are used to monitor cardiovascular changes before, during, and after tasks like serial subtraction or the Trier Social Stress Test (Gerin, Davidson, Christenfeld, Goyal, & Schwartzet., 2006). For this study, the Yasee Arm-type Blood Pressure Monitor (model JN-163B) was used.

Procedure for Data Collection

An informed consent document was presented to the participants before participation. Participants completed a standardised questionnaire to assess emotional dysregulation. The scale used was the Brief Emotional Dysregulation Scale (BEDS).

Experimental Procedure: The experimental procedure involved a stress-inducing task while monitoring cardiovascular parameters, including blood pressure and pulse rate. The participants filled out the questionnaire and then proceeded to the experimental phase. The experiment was carried out individually, not in groups. The experiment for each participant was about 10-15 minutes per person. The baseline cardiovascular reading for each person was taken, and then a task (stressor) was introduced. The task required the participants to carry out serial subtraction for a specified duration (1 min) to induce acute stress. Blood pressure and pulse rate were measured at different points, including baseline, during the stressor, and 1 minute after the stressor. These measurements helped capture the cardiovascular reactivity and recovery patterns in response to the stressors. All the data were collected between 8:00 am and 12:00 pm to control for physiological changes due to fatigue. Data was collected from the participants in paper-and-pencil form. Data collection took place between 2nd – 6th June 2025.

Ethical Consideration

Ethical approval was obtained from the Covenant University Research Ethics Committee, with the HREC protocol

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assigned number, CHREC/1100/2025, as seen in Appendix C. Participants were fully informed about the study's purpose, procedures, potential risks, and their right to withdraw at any time without penalty. Informed consent was obtained before participation. Measures were taken to ensure that all data were anonymised, and participant information was securely stored to maintain confidentiality. Participants were monitored closely for any adverse reactions during the stress tasks. Participants were debriefed at the conclusion of the experiment to alleviate any potential psychological discomfort. This ethical approach ensured the protection and well-being of all participants throughout the research process.

Demographic Data of Participants

Table 1 **:** *Demographic Data of Participants*

	FREQUENCY (*N= 60*)	**PERCENTAGE (*100*)**
AGE		
18 - 29	*60*	*100*
GENDER		
Male	*18*	*30*
Female	*42*	*70*
RELIGION		
Christianity	*60*	*100*
Islam	*0*	
Other	*0*	
EMPLOYMENT	*55*	*91.7*
Student	*4*	*6.7*
Employed	*1*	*1.6*
Self-employed		
TOTAL	*60*	*100*

Table 1 displays the demographic information of the sample for the study. 30% of the respondents were male while 70% were female. All of the participants fell into the 18 – 29 age range, with a mean age of 20.07, and with 18 having the most participants (40%). 91.7% of the participants were only students, 6.7% were employed alongside being students, and 1.6% was self-employed, alongside being a student.

Results

Data Analysis

Hypothesis 1

There will be no significant effect of acute stress on cardiovascular reactivity among Covenant University students.

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Table 2a

Paired Samples T-Test table showing the significant effect of acute stress on cardiovascular reactivity among Covenant University students.

Variable	M	SD	df	t	p	Cohen's d
SBP1	*121.30*	*20.67*	*59*	*2.10*	*.040*	*0.27*
SBP2	*125.93*	*18.13*	*59*			
DBP1	*69.10*	*9.09*	*59*	*5.69*	*<.001*	*0.73*
DBP2	*75.07*	*11.89*	*59*			
PR1	*74.32*	*12.28*	*59*	*9.69*	*<.001*	*1.25*
PR2	*84.22*	*13.14*	*59*			

A paired samples t-test was conducted to examine the effect of the experimental condition on participants' cardiovascular functioning, specifically systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse rate (PR), as indicated in Table 4.2a.

For systolic blood pressure, participants recorded a significantly higher mean at post-test (SBP2: M = 125.93, SD = 18.13) compared to pre-test (SBP1: M = 121.30, SD = 20.67), t (59) = 2.10, p = .040, Cohen's d = 0.27. This result suggests a small but statistically significant increase in systolic blood pressure after the stressor. The increase may reflect a mild physiological response such as arousal or Similarly, for pulse rate, there was a statistically significant increase from pre-test (PR1: M = 74.32, SD = 12.28) to post-test (PR2: M = 84.22, SD = 13.14), t (59) = 9.69, p < .001, Cohen's d = 1.25. This indicates a strong physiological response, such as heightened alertness, anxiety, or acute stress, leading to increased heart rate.

stress activation. For diastolic blood pressure, the post-test mean (DBP2: M = 75.07, SD = 11.89) was significantly higher than the pre-test mean (DBP1: M = 69.10, SD = 9.09), t (59) = 5.69, p < .001, Cohen's d = 0.73. This indicates an increase in diastolic blood pressure following the stressor, suggesting a substantial cardiovascular response.

Together, these findings suggest that the stressor administered had a measurable impact on participants' cardiovascular functioning, consistent with an arousal or stress-inducing effect.Thus, the null hypothesis was rejected.

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Hypothesis 2

There will be no significant effect of acute stress on cardiovascular recovery among Covenant University students.

Table 2b

ANOVA table showing the significant effect of acute stress on cardiovascular recovery among Covenant University students.

Variable	SS	df	MS	F	p	Partial η²
Systole (Time)	*2,727.78*	*3*	*909.26*	*10.11*	*<.001*	*.146*
Error (Systole)	*15,913.72*	*177*	*89.91*			
Diastole (Time)	*2,917.82*	*3*	*972.61*	*21.53*	*<.001*	*.267*
Error (Diastole)	*7,996.18*	*177*	*45.18*			
Pulse (Time)	*3,827.25*	*3*	*1,275.75*	*47.81*	*<.001*	*.448*

A repeated-measures ANOVA was conducted to examine changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse rate across three time points: baseline (Time 1), post-stressor (Time 2), and the recovery point (Time 3), as seen in Table 4.2b. While all three time points were included in the ANOVA to determine whether significant changes occurred over time, the primary focus of the present analysis was to evaluate cardiovascular recovery, operationalised as changes in value at Time 3.

As shown in Table 2b, the effect on systolic blood pressure was significant, $F(3, 177) = 10.11$, $p < .001$, indicating that SBP significantly varied across the time points. There was also a statistically significant effect on diastolic blood pressure as well, $F(3, 177) = 21.53$, $p < .001$ (see Table 3). For pulse rate, the effect was highly significant, $F(3, 177) = 47.81$, $p < .001$ (see Table 3). Thus, the null hypothesis was rejected.

Hypothesis 3

Emotional regulation will not significantly moderate the effect of acute stress on cardiovascular reactivity among Covenant University students.

Table 2c

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Summary of moderated regression analysis table showing the moderating effect of emotional regulation on cardiovascular reactivity among Covenant University students.

Moderator (W)	Predictor	B	T	p	95% CI Lower	95% CI Upper
TOTAL	Constant	*4.194*	*2.253*	*.0281*	*0.466*	*7.921*
	SBP1 (X)	*-0.405*	*-4.392*	*<.001*	*-0.594*	*-0.217*
	Total Dysreg (W)	*-0.899*	*-0.872*	*.3871*	*-2.926*	*1.128*
	X * W	*-0.073*	*-1.269*	*.2103*	*-0.188*	*0.042*
TOTAL	Constant	*6.135*	*5.635*	*<.001*	*3.954*	*8.316*
	DBP1	*-0.019*	*-0.151*	*.881*	*-0.274*	*0.236*
	TOTAL	*-0.023*	*-0.110*	*.913*	*-0.435*	*0.390*
	X * W	*0.018*	*0.786*	*.435*	*-0.028*	*0.065*
TOTAL	Constant	*10.099*	*9.887*	*<.001*	*8.054*	*12.144*
	PR1	*-0.100*	*-1.205*	*.233*	*-0.265*	*0.066*
	TOTAL	*-0.780*	*-1.285*	*.204*	*-1.993*	*0.432*
	X * W	*-0.069*	*-1.315*	*.194*	*-0.173*	*0.035*

Note. *SBP = Baseline Systolic Blood Pressure; DBP = Diastolic Blood Pressure; PR = Pulse Rate; TOTAL = Total Emotional Dysregulation.*

Moderation analysis was conducted using Hayes' PROCESS macro (Model 1) for SPSS (version 4.2), with emotional dysregulation entered as the moderator. Emotional dysregulation was analysed both as a total construct and across its individual dimensions - sensitivity, reactivity, lability, and consequences. The moderating effects were not statistically significant in any of the models, as seen in Table 2c. When total emotional dysregulation was entered as the moderator, the outcome was not significant (B = -0.073, p = .210), indicating that the degree of emotional regulation difficulties did not meaningfully moderate the effect of acute stress on systolic blood pressure (SBP).

When the analysis was broken down into the four sub-dimensions of emotional dysregulation, a similar pattern emerged. For sensitivity, the moderation did not reach statistical significance (B = -0.127, p = .082). The reactivity subscale also did not yield a significant moderation effect (B = -0.113, p = .135). For lability, the moderation was positive but nonsignificant (B = 0.034, p = .678), and for consequences, the moderation effect remained nonsignificant (B = -0.073, p = .339). Together, these findings suggest that emotional dysregulation, whether

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considered globally or in terms of its specific components, does not significantly alter the effect of acute stress on systolic blood pressure reactivity. Therefore, the results support the null hypothesis: emotional dysregulation does not significantly moderate the effect of acute stress on cardiovascular reactivity. These findings imply that individual differences in emotional regulation do not necessarily translate into differential physiological reactivity to acute stress, at least in terms of systolic blood pressure changes. For the diastolic blood pressure (DBP), in all models examined, the results demonstrated that emotional dysregulation did not significantly alter the effect of acute stress on cardiovascular reactivity. For the total emotional dysregulation score (TOTAL), the interaction between pre-stress DBP (DBP1) and emotional dysregulation (X * W) was not statistically significant (B = 0.018, t = 0.786, p = .435). The main effects of DBP1 (B = -0.019, p = .881) and total emotional dysregulation (B = -0.023, p = .913) were also not significant. This indicates that overall emotional dysregulation did not significantly moderate DBP reactivity following stress. When considering the specific dimensions, a similar non-significant pattern was observed. For Sensitivity (SENSI), the interaction was B = 0.047, t = 0.577, p = .566. Neither the main effect of DBP1 (p = .872) nor Sensitivity (p = .775) reached statistical significance. Reactivity (REACT) also showed no significant interaction with DBP1 (B = 0.007, t = 0.080, p = .937), and the moderator effect was likewise non-significant (p = .773 and .648, respectively). In the Lability (LABILI) model, the interaction term approached a greater magnitude (B = 0.126), but still failed to reach significance (t = 1.161, p = .251). The main effects of DBP1 and Lability remained non-significant (p = .915 and .825). Lastly, for Consequences (CONSE), the interaction term was B = 0.071, t = 1.027, p = .309, with the confidence interval crossing zero (-0.068, 0.210), indicating no moderating effect. The main effects of both DBP1 (p = .765) and Consequences (p = .837) were also not significant. When put together, emotional dysregulation, whether considered as a total score or in terms of its dimensions (Sensitivity, Reactivity, Lability, and Consequences), did not significantly moderate the effect of acute stress on cardiovascular reactivity as measured by DBP. None of the interaction terms approached statistical significance, and their confidence intervals all included zero. This suggests that among the

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Covenant University students studied, variations in emotional dysregulation levels did not significantly moderate how their cardiovascular system responded to acute stress in terms of diastolic blood pressure reactivity. For Pulse Rate, the overall model using the total score of emotional dysregulation was not statistically significant, p = .1246, accounting for approximately 9.7% of the variance in pulse rate change (R^2 = .0967). None of the individual moderators were significant: PR1 (B = -0.100, p = .233), total dysregulation score (B = -0.780, p = .204), or the interaction term (B = 0.018, p = .435). This suggests that the overall emotional dysregulation level did not significantly moderate the relationship between baseline pulse rate and subsequent change.

Similarly, sensitivity did not significantly moderate the relationship between PR1 and PR2 – PR1. The interaction between PR1 and sensitivity was non-significant (B = 0.047, p = .566). Neither PR1 (B = -0.021, p = .872) nor sensitivity (B = -0.187, p = .775) was a significant moderator of pulse rate change. This indicates that individuals with varying levels of emotional sensitivity did not differ significantly in how their initial pulse rate predicted reactivity. Using reactivity as a moderator also did not yield significant results. The interaction between PR1 and reactivity was not significant (B = 0.007, p = .937). PR1 itself was not a significant moderator of change in pulse rate (B = -0.036, p = .773), nor was reactivity (B = 0.319, p = .648). This suggests that emotional reactivity does not meaningfully alter the relationship between initial and during-task pulse rate.

In the model including lability, the interaction between PR1 and lability did not reach statistical significance (B = 0.126, p = .251). PR1 (B = 0.015, p = .915) and lability (B = -0.173, p = .825) were also non-significant. Consequences also did not moderate the effect of acute stress on reactivity (PR2 – PR1). The interaction term (B = -0.077, p = .179) was not statistically significant. Neither PR1 (B = -0.108, p = .200) nor the consequences score (B = -0.972, p = .136) significantly moderated pulse rate recovery. This indicates that the degree to which individuals experience negative consequences from emotional dysregulation does not meaningfully change the link between initial and during-task pulse rates. Overall, the moderation analyses did not provide evidence that emotional dysregulation significantly moderates the relationship between baseline pulse rate and pulse rate recovery

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following a stressor. Thus, the null hypothesis was not rejected.

Hypothesis 4

Emotional regulation will not significantly moderate the effect of acute stress on cardiovascular recovery among Covenant University students.

Table 2d:*Summary of moderated regression analysis table showing the moderating effect of emotional regulation on cardiovascular recovery (SBP) among Covenant University students.*

Moderator	Predictor	B	T	P	95% CI Lower	95% CI Upper
TOTAL	Constant	*6.364*	*4.07*	*.0002*	*3.229*	*9.499*
	SBP1	*0.038*	*0.50*	*.617*	*-0.114*	*0.191*
	TOTAL	*-0.032*	*-0.11*	*.915*	*-0.633*	*0.569*
	X * W	*-0.016*	*-0.81*	*.420*	*-0.057*	*0.024*
TOTAL	Constant	*8.411*	*4.904*	*<.001*	*4.975*	*11.847*
	DBP1	*0.277*	*1.379*	*.173*	*-0.125*	*0.678*
	TOTAL	*0.061*	*0.188*	*.851*	*-0.588*	*0.710*
	X * W	*-0.001*	*-0.017*	*.987*	*-0.074*	*0.072*
TOTAL	Constant	*9.592*	*8.770*	*<.001*	*7.401*	*11.783*
	PR1	*0.101*	*1.090*	*.280*	*-0.085*	*0.288*
	TOTAL	*-0.363*	*-1.690*	*.097*	*-0.793*	*0.067*
	X * W	*-0.041*	*-2.383*	*.021**	*-0.076*	*-0.007*
LABILI	Constant	*9.411*	*8.581*	*<.001*	*7.214*	*11.609*
	PR1	*0.066*	*0.722*	*.473*	*-0.117*	*0.248*
	Lability (LABILI)	*-0.972*	*-1.285*	*.204*	*-2.488*	*0.544*
	X * W	*-0.125*	*-2.057*	*.044**	*-0.246*	*-0.003*

Note. *SBP = Baseline Systolic Blood Pressure; DBP = Diastolic Blood Pressure; PR = Pulse Rate; TOTAL = Total Emotional Dysregulation.*

Moderation analysis was conducted using Hayes' PROCESS macro (Model 1) for SPSS (version 4.2). A series of moderated regression analyses was conducted to investigate whether different dimensions of emotional dysregulation moderated the

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relationship between baseline systolic blood pressure (SBP1) and systolic blood pressure measured 1 minute after recovery, as seen in Table 2d. The moderation effects of the total emotional dysregulation score (TOTAL) and its four subcomponents, Sensitivity (SENSI), Reactivity (REACT), Lability (LABILI), and Consequences (CONSE), were individually tested.

The overall model (TOTAL) was not statistically significant, F (3, 56) = 0.34, p = .797. The interaction term between SBP1 and TOTAL was not significant (B = -0.004, t = -0.81, p = .420), indicating that overall emotional dysregulation did not moderate the effect of baseline SBP on SBP after 1 minute of recovery. The second model evaluated the Sensitivity (SENSI) subscale as a moderator. The interaction term (SBP1 × SENSI) did not reach statistical significance (B = -0.005, t = -0.94, p = .352), suggesting that sensitivity did not significantly moderate the effect of acute stress on cardiovascular recovery. In the third model, Reactivity (REACT) was examined as the moderator. The interaction term was not significant (B = -0.002, t = -0.30, p = .765), indicating no moderation. The fourth model tested lability (LABILI) as a moderator, and it was not statistically significant, F(3, 56) = 0.97, p = .415. The interaction between SBP1 and LABILI also was not significant (B = -0.007, t = -1.48, p = .146). Finally, the Consequences (CONSE) yielded the weakest results. The interaction term was not significant (B = -0.001, t = -0.13, p = .893). Together, none of the emotional dysregulation dimensions significantly moderated the relationship between baseline systolic blood pressure and systolic blood pressure recorded 1 minute into recovery.

For diastolic blood pressure (DBP), in the first model, the interaction between DBP1 and total emotional dysregulation was not statistically significant (B = 0.025, t = 0.512, p = .611. This suggests that emotional dysregulation does not significantly moderate the effect of acute stress on diastolic blood pressure one minute after recovery. It also implies that emotional dysregulation does not amplify or weaken the effect of acute stress on short-term recovery, at least within the first minute. When examining the sensitivity dimension of emotional dysregulation, the interaction with DBP1 was again non-significant (B = 0.017, t = 0.364, p = .717, and sensitivity (B = 0.092, t = 0.556, p = .580) did not independently moderate DBP2_3. This result suggests that high emotional sensitivity does not appear to interfere with cardiovascular recovery in the immediate aftermath of stress. The reactivity model also yielded a non-significant interaction (B = 0.010, t = 0.190, p = .850). This implies that being emotionally reactive does not appear to

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hinder or trigger immediate blood pressure recovery. In the lability model, the interaction term remained non-significant (B = 0.014, t = 0.346, p = .731. This suggests that emotional instability may not have a short-term impact on physiological recovery post-stress. Finally, in the consequences model, the interaction between DBP1 and emotional consequences (for instance, rumination, regret, guilt after emotional episodes) was not significant (B = 0.027, t = 0.548, p = .586). This result implies that ruminative or consequence-focused tendencies do not meaningfully moderate immediate blood pressure regulation following a stressor. Together, the findings consistently indicate that emotional dysregulation does not moderate the level of diastolic blood pressure one minute after recovery. Across all models, PR (Pulse Rate) demonstrated a small positive effect on the outcome, but these main effects were not statistically significant, such as B = 0.101, p = .280, indicating that PR1 alone did not significantly moderate the outcome. However, significant moderation effects were observed when emotional dysregulation dimensions were considered. The interaction between PR1 and the total emotional dysregulation score was significant (B = -0.041, p = .021), indicating that higher overall emotional dysregulation weakens the positive moderation between acute stress and the outcome. Similarly, the interaction with the Liability dimension was statistically significant (B = -0.125, p = .044). This implies that individuals who experience rapid emotional fluctuations exhibit a diminished recovery. The moderating effects of Sensitivity (B = -0.096, p = .079) and Reactivity (B = -0.128, p = .052) were not statistically significant. The interaction involving the Consequences dimension was not significant (B = -0.103, p = .103). Thus, the null hypothesis was rejected in the measure of Pulse Rate.

Hypothesis 5

Physical activity levels will not significantly moderate the effect of acute stress on cardiovascular reactivity among Covenant University students.

Table 2e:*Summary of moderated regression analysis table showing the moderating effect of physical activity levels on cardiovascular reactivity among Covenant University students.*

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Regression Model	Predictor	B	T	P	95% CI Lower	95% CI Upper
SBP2_1	Constant	*3.4603*	*2.04*	*.0463*	*0.0581*	*6.8625*
	SBP1	*-0.4945*	*-5.97*	*<.001*	*-0.6605*	*-0.3284*
	EXERCISE	*1.5951*	*0.79*	*.4307*	*-2.4309*	*5.6211*
	X * W	*0.3165*	*3.44*	*.0011*	*0.1319*	*0.5010*
DBP2_1	Constant	*6.4007*	*6.12*	*<.001*	*4.3046*	*8.4968*
	DBP1	*-0.0257*	*-0.21*	*.8365*	*-0.2738*	*0.2224*
	EXERCISE	*2.7384*	*2.13*	*.0378*	*0.1601*	*5.3167*
	X * W	*-0.1876*	*-1.91*	*.0612*	*-0.3844*	*0.0091*
PR2_PR1	Constant	*10.1354*	*9.86*	*<.001*	*8.0753*	*12.1954*
	PR1	*-0.1211*	*-1.44*	*.1564*	*-0.2900*	*0.0478*
	EXERCISE	*0.9188*	*0.80*	*.4270*	*-1.3814*	*3.2190*
	X * W	*0.1158*	*1.16*	*.2509*	*-0.0841*	*0.3157*

Note. *SBP = Baseline Systolic Blood Pressure; DBP = Diastolic Blood Pressure; PR = Pulse Rate; TOTAL = Total Emotional Dysregulation.*

Moderation analysis was conducted using Hayes' PROCESS macro (Model 1) for SPSS (version 4.2). The interaction between baseline SBP (SBP1) and physical activity was significant (B = 0.3165, t = 3.44, p = .001), indicating that the relationship between baseline SBP and post-stressor SBP was moderated by physical activity levels, as seen in Table 2e.

In contrast, the interaction between baseline DBP and physical activity was not significant (B = -0.1876, t = -1.91, p = .0612). Neither the main effect of baseline pulse rate (PR1), nor physical activity, nor their interaction (B = 0.1158, p = .2509) significantly moderated during-stress pulse rate. This suggests that physical activity does not significantly moderate pulse rate reactivity, supporting the null hypothesis for this measure. Specifically, physical activity levels significantly moderated the effect of acute stress on systolic blood pressure reactivity, such that higher physical activity buffered individuals from reactive increases or decreases in SBP during stress. Therefore, the hypothesis that *physical activity does not significantly moderate cardiovascular reactivity* was rejected for systolic reactivity, but not rejected for diastolic blood pressure and

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pulse rate. Thus, the null hypothesis was rejected in the measure of SBP.

Hypothesis 6

Physical activity levels will not significantly moderate the effect of acute stress on cardiovascular recovery among Covenant University students.

Table 2f : *Summary of moderated regression analysis table showing the moderating effect of physical activity levels on cardiovascular recovery among Covenant University students after 1 min.*

Outcome	Predictor	B	T	P	95% CI LL	95% CI UL
SBP2_3	Constant	*6.1369*	*3.9697*	*.0002*	*3.0400*	*9.2339*
	SBP1	*0.0182*	*0.2411*	*.8104*	*-0.1330*	*0.1693*
	EXERCISE	*1.0788*	*0.5897*	*.5578*	*-2.5859*	*4.7436*
	X * W	*0.1159*	*1.3826*	*.1723*	*-0.0520*	*0.2839*
DBP2_3	Constant	*9.0958*	*5.4456*	*<.001*	*5.7498*	*12.4418*
	DBP1	*0.3986*	*2.0165*	*.0486*	*0.0026*	*0.7947*
	EXERCISE	*0.7915*	*0.3852*	*.7015*	*-3.3244*	*4.9073*
	X * W	*-0.2936*	*-1.8725*	*.0664*	*-0.6077*	*0.0205*
PR2_PR3	Constant	*9.3654*	*8.1248*	*<.001*	*7.0563*	*11.6745*
	PR1	*0.0126*	*0.1338*	*.8941*	*-0.1767*	*0.2020*
	EXERCISE	*-1.6875*	*-1.3111*	*.1952*	*-4.2658*	*0.8908*
	X * W	*0.0322*	*0.2875*	*.7748*	*-0.1919*	*0.2563*

Note. *SBP = Baseline Systolic Blood Pressure; DBP = Diastolic Blood Pressure; PR = Pulse Rate; TOTAL = Total Emotional Dysregulation.*

Moderation analysis was conducted using Hayes' PROCESS macro (Model 1) for SPSS (version 4.2). The overall regression model for SBP2_3 was not statistically significant, $F(3, 56) = 1.12$, $p = .348$, as seen in Table 2f. Physical activity (EXERCISE) did not significantly moderate recovery. Specifically, the interaction between SBP1 and EXERCISE was not significant, $B = 0.1159$, $t = 1.38$, $p = .172$. This suggests that physical activity did not significantly moderate the effect of acute stress on systolic recovery after 1 minute. For DBP, the overall model was not significant $F(3, 56) = 1.95$, $p = .132$, with $R^2 = .095$. The interaction between DBP and

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EXERCISE was not significant, $B = -0.2936$, $t = -1.87$, $p = .066$. Since the interaction did not meet the significance criterion ($p < .05$), the moderation is not statistically supported. The model moderating pulse rate recovery after 1 minute was also not significant, $F(3, 56) = 0.65$, $p = .585$. None of the interactions were significant. The interaction term had a coefficient of $B = 0.0322$, $t = 0.29$, $p = .775$. These results indicate that physical activity levels did not moderate the effect of acute stress on pulse rate recovery after 1 minute. In total, this supports the hypothesis that physical activity levels do not significantly moderate the effect of acute stress on cardiovascular recovery. Thus, the null hypothesis was accepted.

Moderation Analyses

Two moderation models were tested to examine the roles of psychological and lifestyle factors in cardiovascular responses to acute stress. The first model showed that emotional regulation, specifically the lability dimension, significantly moderated pulse rate recovery, such that higher emotional instability was associated with slower autonomic recovery. The second model revealed that physical activity significantly moderated systolic blood pressure reactivity, with more physically active individuals demonstrating higher blood pressure responses under stress.

Summary of Findings

1. There was a significant effect of acute stress on cardiovascular reactivity.
2. There was a significant effect of acute stress on cardiovascular recovery, as significant changes in systolic blood pressure, diastolic blood pressure, and pulse rate were observed over time, with full recovery occurring only by the final time point.
3. Emotional regulation did not significantly moderate the effect of acute stress on cardiovascular reactivity, as none of the moderation effects, whether using the total emotional dysregulation score or its individual dimensions, were statistically significant for systolic blood pressure, diastolic blood pressure, or pulse rate.
4. Emotional regulation (lability) significantly moderated the effect of acute stress on cardiovascular recovery (pulse rate), while showing no significant moderation effects on systolic and diastolic blood pressure.
5. Physical activity significantly moderated systolic blood pressure reactivity to acute stress while showing

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no significant moderation effects on diastolic blood pressure or pulse rate; thus, the null hypothesis was rejected.

6. Physical activity did not significantly moderate the effect of acute stress on cardiovascular recovery, whether systolic or diastolic blood pressure or pulse rate. Thus, the null hypothesis was not rejected.

Discussion

The findings revealed that there was a significant increase in systolic blood pressure, diastolic blood pressure, and pulse rate during the stress condition compared to baseline. This means that the stressor led to physiological arousal, and this activated the cardiovascular system. This then led to a measurable stress response across all three measures, and it was concluded that acute stress significantly had an effect on cardiovascular reactivity. The finding that acute stress had a significant effect on cardiovascular reactivity goes with a large body of literature that shows that psychological stressors bring about measurable changes in cardiovascular parameters such as systolic and diastolic blood pressure and heart rate (Chen et al., 2022; McMahon et al., 2021). This aligns with the reactivity hypothesis, which states that heightened reactivity to stress would predict cardiovascular disease (Creaven et al., 2020; Gallagher et al., 2018). Similar to Matthews and his colleagues (2004) and De Rooij (2013), the present results show the association between higher blood pressure reactivity and hypertension risk in the future. The reactivity and the repeated activation of such responses over time can add to vascular remodelling and higher strain on the cardiovascular system (Chauntry et al., 2022). This study supports the idea that acute stress triggers significant cardiovascular responses that could increase long-term cardiovascular risk if sustained or frequent. Significant changes in systolic and diastolic blood pressure and pulse rate were observed over time, with full recovery only at the final time point. This supports Chida and Steptoe's (2010) conclusion that delayed recovery is a stronger predictor of poor cardiovascular outcomes than reactivity alone. Steptoe and Marmot (2005) also found that slower blood pressure recovery predicted coronary artery disease risk years later. The delayed recovery pattern observed here may reflect prolonged sympathetic activation or impaired parasympathetic reactivation (Pierpont et al., 2000), both of which have been linked to negative cardiovascular results. This shows how important it is to measure both the peak responses and the return to baseline. These findings show that

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interventions should aim to reduce peak stress responses and also to hasten recovery. Emotional regulation also did not significantly moderate the relationship between acute stress and cardiovascular reactivity, and this is against studies suggesting that effective emotional regulation can reduce cardiovascular responses (Griffin & Howard, 2022). For example, Jentsch and Wolf (2020) reported that reappraisal techniques were associated with lower heart rate reactivity and enhanced heart rate variability after stress. The lack of significant moderation in this study may be due to sample characteristics, the acute nature of the stressor, or the possibility that emotional regulation has a greater influence on the recovery processes than on the initial reactivity. This result shows that while emotional regulation may play a role in stress physiology, the effects on reactivity may be less consistent or more dependent on the context than was previously thought.

On the contrary, emotional regulation lability significantly moderated pulse rate recovery, and this shows the role of emotional stability in autonomic recalibration after stress. The first model showed that emotional regulation, specifically the lability dimension, significantly moderated pulse rate recovery, such that higher emotional instability was associated with slower autonomic recovery. Individuals with greater emotional lability may experience difficulty disengaging from stress, and this would result in longer elevations in heart rate (Whiston et al., 2025). This goes with evidence that individuals who regulate emotions effectively show faster heart rate recovery and greater heart rate variability after stress (Griffin & Howard, 2022). The effect of this on pulse rate, rather than blood pressure, shows that emotional regulation may have a stronger influence on cardiac vagal reactivation than on vascular tone. Physical activity significantly moderated systolic blood pressure reactivity, and this supports evidence that fitness levels would influence cardiovascular responses to stress. Hamer and his colleagues (2006) found that individuals who were physically active showed smaller increases in systolic blood pressure during stress compared to those who were not. This can be as a result of improved vascular elasticity, reduced peripheral resistance, and enhanced cardiac efficiency that is associated with regular physical activity (Chen et al., 2022). The absence of moderation effects for diastolic blood pressure or pulse rate in this study goes with prior mixed findings (De Geus et al., 1993; Crews & Landers, 1987), and this

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may show the greater sensitivity of systolic measures to both cardiac output and vascular changes during stress. These results mean that exercise interventions could be an effective strategy for reducing systolic blood pressure reactivity to stress. However, physical activity did not significantly moderate cardiovascular recovery, and this is contrary to research showing that regular exercise can accelerate autonomic recovery (Borresen & Lambert, 2008; Sloan et al., 2008). The benefits of physical activity for recovery may be more obvious under conditions of physical stress rather than psychological stress. It is also possible that recovery dynamics are more strongly influenced by emotional factors than by baseline fitness levels in the context of acute psychological stress. This is seen in the emotional regulation findings.

Implications of the Study

The need for early therapy to help students manage stress has been made clear by the significant effect of acute stress on cardiovascular reactivity and recovery. The changes in blood pressure and pulse rate during stress mean that even brief exposure to stressors can place strain on the cardiovascular system, and this would increase long-term health risks if those responses are frequent and unresolved. Also, the absence of a moderating effect of emotional regulation on cardiovascular reactivity, in contrast with its moderating effect on pulse rate recovery, could mean that emotional dysregulation may not alter immediate stress responses but may interfere with the body's recovery from the stress. This would impact psychological interventions, such as emotion regulation training, mindfulness, and Acceptance and Commitment Therapy, which may help improve post-stress physiological regulation and reduce autonomic strain over time. Also, the moderating role of physical activity on just systolic blood pressure reactivity, but not on recovery or other cardiovascular measures, shows the need for targeted intervention plans. Encouraging regular physical activity could be useful in increasing the body's resistance to stress-related cardiovascular spikes, even if the activity does not directly speed up the recovery process. This shows that it would be important to introduce physical activity programmes in comprehensive health and wellness plans for youths as a preventive measure against cardiovascular stress.

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Conclusion

According to the findings of this research, acute stress has an effect on cardiovascular responses, and this would result in higher physical arousal and a partial recovery to the baseline level after the stress passes. This illustrates why stress must be regarded not only as a mental concern but also as a factor that impacts the body. Although emotional regulation did not seem to affect how the body initially responded to stress, it did play a role in how quickly the pulse rate returned to normal. This means that people who struggle with emotional regulation might stay physiologically aroused for longer, which could make them more prone to stress-related health issues. Likewise, physical activity showed a protective moderating effect on systolic blood pressure reactivity but had no significant effect on recovery, meaning that while exercise may buffer initial stress responses, it may not alter recovery processes in the short term. In all, there is a need for comprehensive approaches that would promote both emotional and physical health to manage stress and support cardiovascular wellbeing. Therefore, more effective prevention and intervention strategies can and should be designed to support holistic health in stressful environments.

Limitations of the Study

The type of acute stressor used may not have been adequate to induce a strong and uniform physiological stress response across all participants. The stressor, serial subtraction, relied primarily on cognitive effort, particularly mathematical ability, and this varies among individuals. Participants with stronger mathematical skills may have found the task less challenging or stressful, and this would result in a diminished stress response. On the other hand, participants with lower mathematical proficiency may have experienced more pronounced stress, and this would have introduced variability in reactivity that could have affected the reliability of the findings. While a psychological stressor involving emotional or interpersonal content might have been more effective in simulating real-world stress, such stressors are difficult to standardise in experimental settings. Psychological stress responses are often subjective and influenced by personal history, meaning that identical scenarios may cause different responses in different individuals. On the other hand, experimental designs would require uniform conditions to ensure internal validity. This makes the use of

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psychological stressors problematic because maintaining consistency and fairness across participants would be challenging. Also, relying on self-report measures to assess emotional regulation introduces the potential for bias, such as social desirability or inaccurate self-perception. The sample size and demographic scope, limited to a single university, also restrict the generalizability of the findings.

Suggestions for Further Studies

Future studies should consider using a broader range of stress-inducing tasks that combine both cognitive and emotional components. Incorporating emotionally charged or socially evaluative stressors (for instance, public speaking simulations or interpersonal conflict scenarios) may cause a more generalised stress response. Nevertheless, it is acknowledged that standardising psychological stressors is challenging in experimental settings due to the subjective nature of emotional experiences. Hybrid stress models that are emotionally engaging but still allow for experimental control may be helpful. Also, recommendations should be based on each cardiovascular index observed in the study. The findings revealed that acute stress significantly increased SBP, DBP, and pulse rate. While SBP reactivity was moderated by physical activity, DBP and pulse rate were not. This means that future research should look into specific strategies for managing each index. Future research should also investigate whether certain emotional regulation strategies are more effective for managing heart rate recovery compared to blood pressure regulation. This could lead to more personalised interventions that match individuals' emotional profiles with the most effective coping strategies for physiological regulation.

The use of self-report measures of emotional regulation should be complemented by more objective, behavioural, or physiological assessments to reduce the risk of response bias. Including tools such as heart rate variability (HRV) monitoring or observer-based emotion regulation tasks would enhance the validity of the data and deepen our understanding of the relationship between emotional functioning and physiological responses. Also, future studies should control for cognitive ability when using math-based tasks. Researchers could use stressors that are equally demanding across a broad population, regardless of academic background, to reduce variability in task-induced stress. Larger and more diverse

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samples that span different institutions, regions, and sociodemographic groups would also improve how generalisable the findings are. Future research could also adopt ecological momentary assessment (EMA) or wearable monitoring technologies to examine cardiovascular responses to stress in real-life contexts. Approaches like these would enhance ecological validity and capture the nuanced fluctuations in SBP, DBP, and pulse rate as individuals navigate daily stressors.

Contributions to Knowledge

Within psychophysiology, the study provides some experimental data on how acute stress affects cardiovascular responses (systolic blood pressure, diastolic blood pressure, and pulse rate). This is important, as much of the existing literature on stress physiology is based on Western populations. By demonstrating that even brief, non-physical stressors can significantly impact cardiovascular functioning in a Nigerian sample, this study helps to localise global theories of stress and physiology to the Nigerian context, where sociocultural, environmental, and healthcare factors may differ. In the field of health psychology, the study shows the role of physical activity as a buffer against stress-related increases in systolic blood pressure. In a country where sedentary lifestyles are on the rise due to the fast-growing world of remote jobs, particularly among university students and urban youth, these findings provide evidence-based support for integrating physical activity programmes into university health promotion efforts. This adds to the local discourse on preventive health measures and lifestyle modification, which is important in addressing Nigeria's growing burden of non-communicable diseases. In clinical psychology, the study contributes to an understanding of emotional regulation as a factor in physiological stress recovery, particularly in pulse rate regulation. By identifying emotional lability as a barrier to autonomic recovery, the findings show the relevance of psychological interventions, like emotion regulation training, Acceptance and Commitment Therapy (ACT), Dialectical Behaviour Therapy (DBT), and other evidence-based therapies, in supporting mental and physical well-being among Nigerian youths. This is useful for clinicians working with populations vulnerable to emotional instability or stress-related disorders.

The study provides an interdisciplinary model that combines self-report psychological measures with experimental physiological monitoring. This integrative

approach is still rising in Nigeria, and the study serves as a practical example of how psychological and biological data can be meaningfully combined to inform interventions. It also opens the door for collaboration between psychologists, medical professionals, and public health experts in addressing stress and cardiovascular health more holistically.

References

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.sleepe.2024.100105

Baumeister RF, Vohs KD. Self-regulation, ego depletion, and motivation. *Soc Personal Psychol Compass.* 2007;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

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

Chauntry, 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. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status. *Hypertension.* 2010;55(4):1026–1032. doi:10.1161/hypertensionaha.109.146621

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. B*iological 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/3316910/

De Geus EJ, Van Doornen LJ, Orlebeke JF. Regular exercise and aerobic fitness in relation to psychological make-up and physiological stress reactivity. *Psychosom Med.* 1993;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

Engel GL. The need for a new medical model: A challenge for biomedicine. *Science.* 1977;196(4286):129–136. doi:10.1126/science.847460

Folkman S, Lazarus R. Stress: Appraisal and coping. *Encycl Behav Med.* 1984;1(1):1913–1915. doi:10.1007/978-1-4419-1005-9_21

Forcier, K., Stroud, L. R., Papandonatos, G. D., Hitsman, B., Reiches, M., Krishnamoorthy, J., & Niaura, R. (2006). Links between physical fitness and cardiovascular reactivity and

recovery to psychological stressors: A meta-analysis. *Health Psychology*, *25*(6), 723–739. doi:10.1037/0278-6133.25.6.723

Gallagher, S., O'Riordan, A., McMahon, G., & Creaven, A.-M. (2018). Evaluating personality as a moderator of the association between life events stress and cardiovascular reactivity to acute stress. *International Journal of Psychophysiology*, *126*, 52–59. doi:10.1016/j.ijpsycho.2018.02.009

Garrison KJ, Coyle JR, Baggott MJ, Mendelson J, Galloway GP. Imagery scripts and a computerized subtraction stress task both induce stress in methamphetamine users: A controlled laboratory study. *Subst Abus Res Treat.* 2010;4:S6019. doi:10.4137/sart.s6019

Griffin SM, Howard S. Individual differences in emotion regulation and cardiovascular responding to stress. *Emotion.* 2021. doi:10.1037/emo0001037

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. B*iological 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

Kirschbaum C, Pirke K-M, Hellhammer DH. The "Trier Social Stress Test" – A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology.* 1993;28(1-2):76–81. doi:10.1159/000119004

Lei M-K, Berg MT, Simons RL, Simons LG, Beach SRH. Childhood adversity and cardiovascular disease risk: An appraisal of recall methods with a focus on stress-buffering processes in childhood and adulthood. *Soc Sci Med.* 2020;246:112794. doi:10.1016/j.socscimed.2020.112794

Lovallo, W. R. (2016). *Stress and Health: Biological and Psychological Interactions*. doi:10.4135/9781071801390

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 BS, Gianaros PJ. Stress- and allostasis-induced brain plasticity. *Annu Rev Med.* 2011;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

Morava A, Shirzad A, James V, Nader EAJ, Prapavessis H. Acute stress negatively impacts on-task behavior and lecture comprehension. *PLoS One.* 2024;19(2):e0297711. doi:10.1371/journal.pone.0297711

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

Noh SA, Kim H-S, Kang S-H, Yoon C-H, Youn T-J, Chae I-H. History and evolution of blood pressure measurement. *Clin Hypertens.* 2024;30(1). doi:10.1186/s40885-024-00268-7

Ogiri, S. O. (2020). Cardiovascular stress reactivity in Covenant University students: The role of personal characteristics.

Okoronta B., (2002). Personality And Haemodynamic Patterns in Stress and Non-Stress Conditions.

Parati G, Ulian L, Santucciu C, Omboni S, Mancia G. Blood pressure variability, cardiovascular risk and antihypertensive treatment. *J Hypertens Suppl.* 1995;13(4):S27-34. doi:10.1097/00004872-199512002-00005

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

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

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

Tindle H, Davis E, Kuller L. Attitudes and cardiovascular disease. *Maturitas.* 2010;67(2):108–113. doi:10.1016/j.maturitas.2010.04.020

Turner, A. I., Smyth, N., Hall, S. J., Torres, S. J., Hussein, M., Jayasinghe, S. U., Ball, K., & Clow, A. J. (2020). Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. *Psychoneuroendocrinology, 114*, 104599. doi:10.1016/j.psyneuen.2020.104599

Warburton DER, Nicol CW, Bredin SSD. Health benefits of physical activity: The evidence. *CMAJ.* 2006;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

Wycoff AM, Griffin SA, Helle AC, Trull TJ. The Brief Emotion Dysregulation Scale (BEDS). *Psychol Assess.* 2020;32(7):686-696. doi:10.1177/10731911231161800

ABBREVIATIONS

α (Alpha): Level of significance

ANOVA: Analysis of Variance

B: Unstandardized Coefficient

BEDS: Brief Emotion Dysregulation Scale

BP: Blood Pressure

CI: Confidence Interval

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CVR: Cardiovascular Reactivity

CVD: Cardiovascular Disease

DBP: Diastolic Blood Pressure

DERS: Difficulties in Emotion Regulation Scale

df: Degrees of Freedom

DV: Dependent Variable

HPA: Hypothalamic-Pituitary-Adrenal Axis

HR: Heart Rate

HRR: Heart Rate Recovery

HRV: Heart Rate Variability

IV: Independent Variable

M: Mean / Moderator (context-dependent)

p: p-value

PR: Pulse Rate

SAM: Sympatho-Adrenal Medullary System

SBP: Systolic Blood Pressure

SD: Standard Deviation

SPSS: Statistical Package for the Social Sciences

t: t-statistic

TSST: Trier Social Stress Test

η^2 (Eta squared): Effect size

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APPENDICES

Appendix A

Research Questionnaire

Dear respondent,

This questionnaire is designed to gather information regarding your emotions and experiences, and all answers will be kept confidential and anonymous. Your honest and thoughtful input is greatly appreciated.

NOTE

- **There are no right or wrong answers**. Your responses are completely confidential and anonymous. Your name is not requested, therefore feel free to give your sincere responses. Your openness and honesty are key to ensuring an accurate evaluation.
- Your participation in this survey is entirely voluntary, and you have the option to stop at any point. Should you have any questions or concerns regarding the scale or your responses, please feel free to discuss them with your healthcare provider.
- If you are interested in the outcome of this study, kindly provide your email address below: _____

By filling out this questionnaire, you consent to your answers being used for research purposes.

Thank you for taking the time to complete this questionnaire.

SECTION A

Kindly provide the appropriate responses.

1. **Age**: ____ years
2. **Sex**: ☐ Male ☐ Female
3. **Marital status**: ☐ Single ☐ Married ☐ Divorced ☐ Separated ☐ Widowed
4. **Highest Level of Education**: ☐ Secondary/High school ☐ Bachelor's degree ☐ Master's degree ☐ Ph.D
5. **Employment status**: ☐ Student ☐ Employed ☐ Self-employed ☐ Unemployed
6. **Religion**: ☐ Christianity ☐ Islam ☐ Traditional worship ☐ Free-thinker

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SECTION B

Kindly answer these health-related questions.

1. Do you have a family history any of these cardiovascular diseases? ☐ Hypertension ☐ Heart disease ☐Arrhythmia ☐Diabetes ☐ Hypotension ☐ None
2. Do you have any diagnosed cardiovascular conditions ☐ Hypertension ☐ Heart disease ☐Arrhythmia ☐ Hypotension ☐ None
3. Have you used any of these substances within the last 72 hrs?
   ☐ Cocaine (Crack, powder) ☐ Ice/Crystal meth ☐ Ecstasy (MDMA, Molly) ☐ Heroin ☐Tramadol ☐ Codeine ☐ Marijuana (Loud, SK, Colorado, Edibles) ☐ LSD (Acid) ☐Magic mushrooms ☐ Valium
4. Are you on medications at the moment? ☐ Yes ☐ No (If yes, list: _____
   _____
   _____)
5. Do you have any sleep disorders (e.g., insomnia, sleep apnea/paralysis)? ☐ Yes ☐ No ☐ Not sure
6. Do you smoke or use tobacco products? ☐ Yes, regularly ☐ Occasionally ☐ No
7. Do you consume alcohol? ☐ Yes, regularly ☐ Occasionally ☐ No
8. Do you consume caffeine (e.g., coffee, energy drinks, tea)? ☐ Yes, regularly ☐ Occasionally ☐ No
9. How often do you engage in moderate to vigorous exercise (e.g., running, gym workouts, sports)?
   ☐ Daily ☐ 3–5 times per week ☐ 1–2 times per week ☐ Rarely ☐ Never
10. How many hours of sleep do you usually get per night?
    ☐ Less than 4 hours ☐ 4–6 hours ☐ 6–8 hours ☐ More than 8 hours
11. Is there anything else related to your health that you believe might influence your heart rate or blood pressure?

SECTION C

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Kindly tick the correct responses.

S/N	QUESTIONS	False/Not true at all	Slightly true	Mainly true	Very true
1	My moods are often intense.				
2	When I am upset, I have trouble controlling my behaviour.				
3	I feel angry frequently.				
4	When I am upset, I have trouble controlling my emotions.				
5	My moods change frequently				
6	My moods tend to cause problems for me.				
7	My moods are often unpredictable.				
8	Other people notice my mood changes.				

Thank you for taking the time to fill out this questionnaire.

If you are interested in the outcome of this study, kindly provide your email address below:

_____

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Appendix B

Log

1. **Weight**: _____
2. **Height**: _____

BASELINE

- **SBP:** _____
- **DBP**: _____
- **PR**: _____

DURING TEST

- **SBP:** _____
- **DBP**: _____
- **PR**: _____

POST-TEST RECOVERY

- **SBP:** _____
- **DBP**: _____
- **PR**: _____