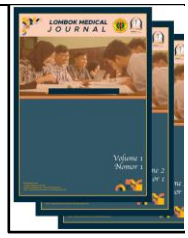
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Blood Pressure in Young Adults in Relation to Anxiety, Stress, and Cortisol: A Cross-Sectional Analysis of Mediation

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Abstract: Anxiety and perceived stress may be associated with blood pressure elevation in young adults. This study examined these associations and whether serum cortisol accounted for an indirect association. Methods: A cross-sectional study included 296 adults aged 18–40 years in Al-Najaf, Iraq. Blood pressure was measured using an automated monitor; anxiety and perceived stress were assessed using Arabic GAD-7 and PSS-10 questionnaires. Morning serum cortisol was assayed. Regression and PROCESS Model 4 mediation analyses used 5,000 bootstrap resamples. Results: Mean age was 31.4 ± 6.2 years; 57.8% were male. Mean SBP, DBP and MAP were 126.0 ± 16.3, 84.1 ± 12.2 and 98.1 ± 13.1 mmHg, respectively. The reported anxiety coefficients were 0.644 for SBP ($p<0.001$), 0.454 for DBP ($p = 0.002$) and 0.516 for MAP ($p<0.001$). Perceived stress was associated with SBP ($\beta = 0.421$, $p = 0.008$) and MAP ($\beta = 0.387$, $p = 0.014$). Cortisol correlated positively with SBP ($r = 0.312$) and MAP ($r = 0.298$), both $p<0.001$. All reported indirect-effect confidence intervals included zero. Conclusion: Anxiety and perceived stress were associated with higher blood pressure, but the analyses did not demonstrate mediation through a single morning cortisol measurement.

Keywords: Anxiety; Perceived stress; Cortisol; Blood pressure; Young adults.

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Introduction

Hypertension remains the leading modifiable risk factor for cardiovascular disease globally, and its prevalence among young adults has risen substantially over recent decades (World Health Organization, 2024). While traditional risk factors such as obesity, high sodium intake, and physical inactivity explain a significant portion of this burden, emerging evidence suggests that psychosocial distress—including anxiety and chronic stress—contributes independently to blood pressure elevation (Richardson et al., 2026; Mancia et al., 2026). Among young adults, the prevalence of anxiety disorders is particularly high, with an estimated 75% of cases diagnosed before age 21 (Kessler et al., 2005). This has prompted investigation into the

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physiological mechanisms linking psychological states to cardiovascular dysfunction.

The hypothalamic-pituitary-adrenal (HPA) axis, with cortisol as its primary effector hormone, has been proposed as a key mediator (Chrousos, 2009). Acute stress reliably activates the HPA axis, producing cortisol surges accompanied by transient blood pressure increases (Sapolsky et al., 2000). Chronic psychosocial distress, however, may dysregulate this system, leading to sustained cortisol exposure and downstream vascular effects (McEwen, 1998). Evidence from Mendelian randomization supports a possible causal contribution of cortisol to hypertension. A one-standard-deviation increase in the genetically predicted plasma cortisol concentration corresponded to an odds ratio of 1.12 for hypertension (95% CI 1.05–1.18), equivalent to a 12% increase in odds; central obesity was proposed as a potential mediator (Lee et al., 2024).

Studies of young adults have reached differing conclusions about whether cortisol helps explain the connection between psychological distress and blood pressure. In a Malaysian comparative study with a cross-sectional sample of 240 young adults, greater anxiety corresponded significantly to higher SBP, DBP, and MAP, with β coefficients of 0.644, 0.454, and 0.516, respectively. The analysis did not identify an indirect pathway through cortisol (Wan Omar et al., 2019). Conversely, other studies have reported inverse associations between anxiety scores and blood pressure in young adults, challenging the assumption of a straightforward positive relationship (Bhat et al., 2017). These discrepancies highlight the need for further investigation in diverse populations.

The experience of young adults in Iraq makes this question especially pertinent. Prolonged conflict and instability may have increased the psychological burden carried by this population (Al-Mohammed & Amin, 2022). A study of Iraqi medical students found that approximately 31% exhibited unhealthy anxiety levels, with females reporting significantly higher anxiety than males Alshiblawy, 2024). Cardiovascular adaptive potential among young Iraqis appears compromised, with obesity, physical inactivity, and emotional stress identified as significant risk factors (Marzoog, 2023). Whether cortisol provides an indirect link between psychological distress and blood pressure has not yet been investigated in this population.

We therefore pursued three objectives in young adults from Al-Najaf, Iraq: (1) to estimate how anxiety and perceived stress relate to blood pressure measures; (2) to assess the association of serum cortisol with blood pressure; and (3) to test a mediation model in which cortisol forms an intermediate link between psychological distress and blood pressure.

Materials and Methods

Study Setting and Design

Between January and December 2024, we carried out a cross-sectional investigation in Al-Najaf Governorate, Iraq, at the University of Kufa College of Medicine, Department of Physiology, and its affiliated primary health care centers. Ethical approval was granted by the University of Kufa Institutional Review Board under approval number 2024-089. The research followed the Declaration of Helsinki (World Medical Association, 2013).

Participants

A total of 296 young adults aged 18–40 years were recruited through purposive sampling from university campuses, primary health care centers, and community settings. Inclusion criteria were: (1) age 18–40 years; (2) no prior diagnosis of hypertension or use of antihypertensive medications; (3) no known chronic medical conditions (diabetes, renal disease, endocrine disorders); (4) not pregnant or lactating; (5) no current use of corticosteroid or psychotropic medications. Participants were excluded if they met any of the following criteria: inability or unwillingness to provide written informed consent; any physical, cognitive, or psychiatric impairment that would preclude reliable measurement of blood pressure or completion of study assessments; participation in another clinical trial or intervention study at the time of recruitment; acute illness (e.g., febrile illness, acute infection) at the time of assessment that could affect blood pressure measurement; and known secondary causes of hypertension (e.g., coarctation of the aorta, pheochromocytoma, Cushing's syndrome) or family history suggestive of hereditary hypertension syndromes, where relevant to the study aims. Every participant provided written consent after being informed about the study.

Measurements

Anthropometric and Demographic Data. A structured interview documented participants' age, sex, educational attainment, and smoking status. Calibrated instruments were used to obtain height and weight. For each participant, BMI was derived by dividing weight in kilograms by the square of height in meters.

Blood Pressure Measurement. After participants had rested while seated for 5 minutes, readings were obtained with the validated Omron HEM-7130 automated oscillometric monitor (Kyoto, Japan) (O'Brien et al., 2010). We recorded three readings, spaced 2 minutes apart, and analyzed their mean. SBP, DBP, and MAP (calculated as (SBP + 2 × DBP)/3) were the primary outcome variables (Sesso et al., 2000).

Anxiety Assessment. We evaluated anxiety symptoms with the validated Arabic GAD-7 (Generalized Anxiety Disorder-7). Its seven items yield a total score between 0 and 21 (Spitzer et al., 2006), with increasing totals reflecting more severe symptoms. The Arabic version has demonstrated good internal consistency (Cronbach's α = 0.84) in prior studies (El Khoury-Malhame et al., 2024).

Perceived Stress Assessment. Participants rated their perceived stress during the preceding month using the Arabic PSS-10 (Perceived Stress Scale-10) (Cohen et al., 1983). The total can take values from 0 to 40; larger totals denote a greater perceived stress burden. Validation studies support this Arabic instrument in several Middle Eastern populations (Chaaya et al., 2010).

Serum Cortisol Measurement. Following an overnight fast, participants underwent venous blood sampling from 8:00 to 9:00 AM. Samples were centrifuged to isolate serum, which was then kept at −80°C pending analysis. Cortisol concentrations were determined with a commercial enzyme-linked immunosorbent assay (ELISA) supplied by DRG International (Springfield, NJ, USA), following the supplier's protocol (DRG International, 2023). Sensitivity was 0.5 ng/mL. The coefficients of variation were 6.8% within assays and 8.5% between assays (Levine et al., 2007).

Statistical Analysis

We performed the analyses in IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA) (IBM Corp., 2019). Numerical variables were summarized by their means and standard deviations (SD); categorical data were presented as counts with corresponding percentages. Pearson correlation coefficients were used to examine bivariate relationships among anxiety, stress, cortisol, and blood pressure parameters (Pearson, 1920). Multiple linear regression models were constructed to assess the independent associations of anxiety and stress with each blood pressure parameter, adjusting for age, sex, BMI, and smoking status (Cohen et al., 2003).

To assess mediation, we fitted Model 4 in the SPSS PROCESS macro. Estimates of indirect effects and their bias-corrected 95% confidence intervals were obtained from 5,000 bootstrap resamples (Hayes, 2022). Anxiety and stress were entered as independent variables, cortisol as the mediator, and SBP, DBP, and MAP as dependent variables in separate models. Covariates included age, sex, and BMI. A statistically significant indirect effect was indicated by a 95% confidence interval that did not include zero (Preacher & Hayes, 2008). We used a two-tailed significance threshold of $p<0.05$.

Author verification: The covariate sets reported in the abstract, regression methods, mediation methods and Table 4 differ. The final model specifications must be confirmed against the original statistical output.

Result and Discussion

Results

Participant characteristics

The study included 296 participants, with a mean age of 31.4 ± 6.2 years; 171 (57.8%) were male and 125 (42.2%) were female. Mean BMI was 27.8 ± 4.6 kg/m². Current smokers accounted for 85 (28.7%) participants. Mean GAD-7 and PSS-10 scores were 9.4 ± 5.2 and 17.2 ± 6.8, respectively. Moderate or severe anxiety was reported by 93 participants (31.4%), while 39 (13.2%) were in the high perceived-stress category (Table 1).

Blood pressure and cortisol

Mean SBP, DBP and MAP were 126.0 ± 16.3, 84.1 ± 12.2 and 98.1 ± 13.1 mmHg, respectively. Mean serum cortisol was 153.5 ± 42.1 ng/mL (Table 2). The reported normal/elevated category counts require verification against the stated thresholds and have been retained as supplied.

Correlation analysis

Anxiety and perceived stress were positively correlated (r = 0.548). The reported correlations of anxiety with SBP, DBP and MAP were 0.412, 0.334 and 0.389, respectively; the corresponding stress correlations were 0.356, 0.287 and 0.321. Cortisol correlated with SBP (r = 0.312) and MAP (r = 0.298). Table 3 retains the supplied coefficients; its significance annotations require confirmation.

Regression and mediation analysis

The reported anxiety coefficients were positive for SBP (β = 0.644, p<0.001), DBP (β = 0.454, p = 0.002) and MAP (β = 0.516, p<0.001). Perceived stress was associated with SBP (β = 0.421, p = 0.008) and MAP (β = 0.387, p = 0.014), but its reported association with DBP was not statistically significant (β = 0.268, p = 0.089). None of the indirect effects through cortisol had a bootstrap 95% confidence interval excluding zero (Table 4). These estimates are reproduced from the source and require confirmation of coefficient type and covariate specification.

Table 1. Participant Demographics and Clinical Profile (n = 296)

Characteristic	Category	n	%	Mean ± SD
Age (years)	18–25	78	26.4	31.4 ± 6.2
	26–30	72	24.3	
	31–35	81	27.4	
	36–40	65	22.0	
Gender	Male	171	57.8	
	Female	125	42.2	
BMI (kg/m²)	Normal (18.5–24.9)	128	43.2	27.8 ± 4.6
	Overweight (25–29.9)	101	34.1	
	Obese (≥30)	67	22.6	
Smoking Status	Non-smoker	211	71.3	
	Current smoker	85	28.7	
Education	Secondary or below	94	31.8	
	University	172	58.1	
	Postgraduate	30	10.1	
GAD-7 Score	Minimal (0–4)	68	23.0	9.4 ± 5.2
	Mild (5–9)	135	45.6	
	Moderate (10–14)	62	20.9	
	Severe (15–21)	31	10.5	
PSS-10 Score	Low (0–13)	89	30.1	17.2 ± 6.8
	Moderate (14–26)	168	56.8	
	High (27–40)	39	13.2	

Note: GAD-7 = Generalized Anxiety Disorder-7; PSS-10 = Perceived Stress Scale-10; BMI = body mass index.

Table 2. Blood Pressure Parameters and Serum Cortisol in the Study Sample (n = 296)

Parameter	Mean ± SD	Median (IQR)	Range	Normal n (%)	Elevated n (%)
Systolic blood pressure (mmHg)	126.0 ± 16.3	124 (114–136)	98–168	198 (66.9)	98 (33.1)
Diastolic blood pressure (mmHg)	84.1 ± 12.2	83 (76–92)	62–112	212 (71.6)	84 (28.4)
Mean arterial pressure (mmHg)	98.1 ± 13.1	97 (89–106)	74–130	205 (69.3)	91 (30.7)
Serum cortisol (ng/mL)	153.5 ± 42.1	149 (124–180)	68–298	248 (83.8)	48 (16.2)

Note: IQR = interquartile range. Source classification thresholds: SBP<120 mmHg; DBP<80 mmHg; MAP<95 mmHg; morning cortisol 50–230 ng/mL. Category counts and thresholds require author verification; these are not diagnoses based on repeated visits.

Table 3. Pearson Correlation Matrix Among Anxiety, Stress, Cortisol, and Blood Pressure Parameters (n = 296)

Variable	1	2	3	4	5	6
1. GAD-7 (Anxiety)	—					
2. PSS-10 (Stress)	0.548**	—				
3. Serum cortisol	0.287**	0.264**	—			
4. Systolic BP	0.412**	0.356**	0.312**	—		
5. Diastolic BP	0.334**	0.287*	0.156	0.612**	—	
6. Mean arterial pressure	0.389**	0.321**	0.298**	0.842**	0.891**	—

Note: GAD-7 = Generalized Anxiety Disorder-7; PSS-10 = Perceived Stress Scale-10; BP = blood pressure. Source legend: *p<0.01; **p<0.001. Significance markings have been retained and require verification against the correlation output.

The correlation matrix showed a coherent pattern of small-to-medium positive associations among psychological distress, cortisol, and blood pressure. Anxiety and stress were strongly interrelated ($r = 0.548$, $p < 0.001$) but not redundant, and both correlated positively with serum cortisol ($r = 0.287$ and 0.264, respectively, both $p < 0.001$), accounting for only about 7–8% of shared variance. Blood pressure parameters were also positively associated with the psychological measures, with anxiety showing the strongest relationships (SBP $r = 0.412$; MAP $r = 0.389$; DBP $r = 0.334$), followed by stress (SBP $r = 0.356$; MAP $r = 0.321$; DBP $r = 0.287$). Cortisol correlated significantly with systolic BP ($r = 0.312$) and MAP ($r = 0.298$), but not with diastolic BP ($r = 0.156$). Intercorrelations among BP parameters were high (SBP–DBP $r = 0.612$; SBP–MAP $r = 0.842$; DBP–MAP $r = 0.891$), but this is mathematically expected because MAP is derived from systolic and diastolic pressure. Two points require verification: the significance legend (*$p < 0.01$; **$p < 0.001$) is non-standard, and the DBP–cortisol coefficient of 0.156 would reach conventional significance at $p < 0.05$ given the sample size (critical $r \approx 0.113$), so its classification as non-significant depends on the stricter $p < 0.01$ cutoff. Overall, the findings support a psychophysiological pathway in which anxiety and stress cluster together and relate positively to both cortisol and blood pressure, with anxiety generally showing the stronger associations.

Table 4. Multiple Linear Regression and Mediation Analysis: Associations of Anxiety, Stress, and Cortisol with Blood Pressure Parameters (n = 296)

Dependent Variable	Independent Variable	Direct Effect β (95% CI)	p-value	Indirect Effect via Cortisol β (95% CI)	Mediation
Systolic BP	Anxiety (GAD-7)	0.644 (0.412–0.876)	<0.001	0.018 (−0.012 to 0.056)	No
	Stress (PSS-10)	0.421 (0.112–0.730)	0.008	0.014 (−0.009 to 0.048)	No
	Cortisol	0.312 (0.142–0.482)	<0.001	—	—
Diastolic BP	Anxiety (GAD-7)	0.454 (0.198–0.710)	0.002	0.011 (−0.008 to 0.041)	No
	Stress (PSS-10)	0.268 (−0.042 to 0.578)	0.089	0.009 (−0.006 to 0.035)	No
	Cortisol	0.156 (−0.018 to 0.330)	0.078	—	—
Mean Arterial Pressure	Anxiety (GAD-7)	0.516 (0.298–0.734)	<0.001	0.014 (−0.010 to 0.048)	No
	Stress (PSS-10)	0.387 (0.081–0.693)	0.014	0.012 (−0.008 to 0.042)	No
	Cortisol	0.298 (0.126–0.470)	<0.001	—	—

Note: CI = confidence interval; BMI = body mass index. Source values retained. Indirect effects were assessed using PROCESS Model 4 and 5,000 bootstrap resamples. Covariates and whether coefficients are standardized or unstandardized must be confirmed.

Discussion

This study examined relationships among anxiety, stress, serum cortisol, and blood pressure in 296 young adults in Al-Najaf, Iraq. Anxiety and perceived stress were significantly and independently associated with higher blood pressure, higher cortisol

accompanied higher SBP and MAP, and mediation analysis found no significant indirect association through cortisol. This null indirect effect should not be read as evidence of a direct relationship; it indicates only that circulating cortisol did not statistically account for the association, not that the association is direct, unmediated, or causal. Unmeasured mediators, measurement limitations, and the cross-sectional design remain plausible explanations. Several cortisol-independent pathways could link distress to blood pressure, including sympathetic activation with increased catecholamine release, heart rate, cardiac output, and vasoconstriction; sustained sympathetic activation promoting vascular remodelling and arterial stiffness; distress-related behaviours such as poor sleep, inactivity, high sodium intake, and alcohol or tobacco use; single-time-point cortisol failing to capture tissue-level activity or diurnal secretion; and renin–angiotensin–aldosterone activation and endothelial dysfunction. These possibilities suggest the absent cortisol-mediated pathway reflects the complexity of psychophysiological regulation rather than the absence of a physiological link, warranting future studies with repeated cortisol sampling, sympathetic markers, and longitudinal designs. Two points require verification: "significant independent associations" implies multivariable adjustment, so confirm covariates such as age, sex, and BMI were included; and if only cortisol was tested as a mediator, state explicitly that the analysis was not designed to exclude other pathways.

Our finding that greater anxiety accompanies higher blood pressure agrees with the Malaysian cohort results. In that cohort, adjustment for sex, age, and cortisol yielded significant anxiety coefficients of 0.644 for SBP, 0.454 for DBP, and 0.516 for MAP (Wan Omar et al., 2019). The divergence between Pan et al. (2015) and Jones and Romeiser (2025) is directly pertinent to the present findings. The positive anxiety–blood pressure association observed here is consistent with Pan et al.'s pooled prospective estimate (adjusted HR 1.55, 95% CI 1.24–1.94), supporting the direction of the GAD-7 correlations, although that meta-analysis reported substantial heterogeneity ($I^2 \approx 84–85\%$) and publication bias in its cross-sectional pool. By contrast, Jones and Romeiser's Add Health follow-up of 9,283 participants found no association between earlier anxiety and objectively measured hypertension, and an inverse association when self-reported hypertension was included (aOR 0.76, 95% CI 0.63–0.92), showing that outcome definition alone can reverse the observed effect. Three factors plausibly explain this variability: blood pressure ascertainment, since clinic readings are susceptible to the white-coat effect that anxiety amplifies, so part of the present correlation may reflect situational arousal rather than sustained hypertension; outcome definition, since self-reported diagnosis depends on healthcare access and labelling, and anxious individuals may differentially avoid or over-utilize care; and age and exposure window, since participants here were aged 18–40 and those in Add Health approximately 29 and 37 years, when incidence remains low. Explicit acknowledgement of this contradiction strengthens interpretation and underscores the need for ambulatory measurement and repeated assessment. Finally, the reported identity of the anxiety coefficients with a published study requires verification against the original output before reproducibility can be interpreted. Similarly, a cross-sectional study of 1,014 young Australian adults reported inverse associations between depression/anxiety scores and SBP (Bhat et al., 2017). These discrepancies may reflect differences in study design, population characteristics, and measurement approaches, underscoring the complexity of the anxiety–blood pressure relationship.

The association between perceived stress and blood pressure observed here aligns with the broader literature implicating chronic stress in hypertension pathogenesis. The CARDIA study demonstrated that young adults exhibiting large blood pressure responses to psychological stress were at increased risk for developing hypertension over 13 years of follow-up (Matthews et al., 2004). Among Iraqi young adults specifically, a prior study identified emotional stress as a significant factor contributing to reduced cardiovascular adaptive potential (Marzoog, 2023).

The positive correlation between serum cortisol and blood pressure is consistent with genetic evidence supporting a causal relationship. A Mendelian randomization analysis of UK Biobank data linked higher genetically predicted plasma cortisol to a 12% increase in the odds of hypertension, as well as higher SBP and DBP; central obesity was identified as a possible intermediate factor (Lee et al., 2024).

However, the lack of significant mediation by cortisol in the present study suggests that the pathways linking anxiety and stress to blood pressure are more complex than a simple HPA axis–cortisol model would predict. The Malaysian investigation likewise detected significant direct associations of anxiety with blood pressure without evidence for an indirect effect through

cortisol (Wan Omar et al., 2019). Although those investigators suggested that a larger sample might reveal mediation, no such evidence emerged in our sample of 296 participants.

Several explanations may account for the absence of cortisol mediation. First, serum cortisol represents only a single time-point measurement and may not adequately capture HPA axis dynamics, including diurnal variation, cortisol reactivity, or tissue-level glucocorticoid sensitivity. Alternative measures such as salivary cortisol profiles, hair cortisol, or urinary free cortisol may provide more comprehensive assessments (DRG International, 2023; Levine et al., 2007). Second, the effects of psychosocial distress on blood pressure may be mediated through pathways other than the HPA axis, including sympathetic nervous system activation, inflammatory processes, or oxidative stress. The editorial by Richardson et al (2026) highlighted oxidative stress as a promising mechanistic target linking chronic anxiety to cardiovascular dysfunction, potentially independent of cortisol. Third, cortisol and blood pressure could influence one another, or adiposity could confound their association. The weaker genetic association observed after accounting for waist circumference supports consideration of this possibility (Lee et al., 2024).

The clinical implications of these findings are significant for hypertension prevention in young adults. The high prevalence of moderate-to-severe anxiety (31.4%) and perceived stress in this cohort, combined with their independent associations with blood pressure, suggests that psychological screening should be integrated into routine cardiovascular risk assessment. The Malaysian investigators similarly argued that care for young adults who have elevated blood pressure should address psychological needs alongside medication-based treatment (Wan Omar et al., 2019). In the Iraqi context, where young adults face unique stressors related to post-conflict reconstruction and economic uncertainty, mental health support may represent an underutilized strategy for cardiovascular risk reduction (Alabbood&Al-Rubaie, 2021).

Study Strengths and Limitations

The study examines psychological symptoms, morning serum cortisol and blood pressure in young Iraqi adults. The sample included 296 participants and used Arabic versions of the GAD-7 and PSS-10. The source reports adjustment for potential confounders, although the exact covariate sets require confirmation.

The findings should be interpreted in light of several limitations. Because the data were collected cross-sectionally, they cannot establish causation; follow-up studies are needed to determine the temporal order of the observed associations. Serum cortisol was measured at a single time point, which may not capture the complexity of HPA axis function. The study was conducted in a single governorate, potentially limiting generalizability to other regions of Iraq (Iraqi Ministry of Health, 2023). Ambulatory blood pressure monitoring was not described; therefore, white-coat effects could not be excluded. Purposive recruitment may also introduce selection bias, and a sample-size or power calculation was not provided. Finally, although the GAD-7 and PSS-10 are well-validated instruments, self-reported psychological measures are subject to reporting bias.

Conclusion

In this cross-sectional sample of 296 young adults in Al-Najaf, Iraq, the reported analyses associated anxiety and perceived stress with higher blood pressure. Cortisol correlated with systolic and mean arterial pressure, but the reported bootstrap estimates did not demonstrate mediation through cortisol. These findings cannot establish causal pathways or rule out a role for HPA axis activity. Longitudinal studies with repeated cortisol and blood pressure measurements are needed

Recommendations

1. Conduct prospective studies with repeated blood pressure and cortisol measurements to establish temporal relationships.
2. Use ambulatory or home blood pressure monitoring to assess sustained blood pressure elevation.
3. Evaluate psychosocial symptoms alongside established cardiovascular risk factors in future research.
4. Confirm the reported model specifications and statistical estimates using the original dataset before journal submission.

Declarations

Ethics and consent: The source reports University of Kufa Institutional Review Board approval 2024-089 and written informed consent from all participants.

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