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Research Article

Association of Systemic Inflammatory biomarkers with Resting Heart Rate and Blood Pressure in Adults with Obesity: A Multivariable regression Analysis

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Article Info

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Abstract

Background: Obesity is associated with chronic low-grade systemic inflammation, which may contribute to alterations in cardiovascular regulation. Inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α) have been implicated in cardiovascular dysfunction. However, their independent associations with resting heart rate and blood pressure remain unclear. This study aimed to investigate the relationship between systemic inflammatory biomarkers and cardiovascular parameters among adults with obesity.

Methods: A cross-sectional analytical study was conducted 220 adults aged 18–65 years with a body mass index (BMI) of ≥30 kg/m². The associations of hs-CRP, IL-6, and TNF-α with resting heart rate, systolic blood pressure, and diastolic blood pressure were evaluated using separate multivariable regression models. The models were adjusted for age, sex, BMI, smoking status, physical activity, diabetes mellitus, and antihypertensive medication use. Multiple-comparison adjustment was applied to assess the statistical robustness of the observed associations.

Results: Nominal associations were observed between IL-6 and systolic blood pressure and between hs-CRP and diastolic blood pressure. However, these associations did not remain statistically significant after adjustment for multiple comparisons. TNF-α showed no statistically significant association with resting heart rate, systolic blood pressure, or diastolic blood pressure. Overall, none of the inflammatory biomarkers demonstrated statistically robust independent associations with the cardiovascular parameters following multivariable adjustment.

Conclusion: The findings indicate that hs-CRP, IL-6, and TNF-α are not independently associated with resting heart rate or blood pressure among adults with obesity after accounting for potential confounding factors and multiple comparisons. Further large-scale longitudinal studies are warranted to clarify the relationship between systemic inflammation and cardiovascular regulation in obesity.

Keywords: Obesity; Systemic inflammation; hs-CRP; Interleukin-6; Tumour necrosis factor-alpha; Resting heart rate; Blood pressure; Multivariable regression.

1. Introduction

Obesity is a pathological state where adipose tissue expansion activates multiple pathways, e.g. inflammatory, metabolic, and vascular, which promote the development of cardiometabolic morbidity. Adipose tissue is a specialized tissue with the potential to secrete many cytokines, adipokines, and chemokines. The adverse effect of obesity on cardiovascular health is partly explained by the proinflammatory state associated with adipose tissue and obesity [1,2,3]. Inflammatory processes may vary in intensity and type, and different markers may demonstrate variations in the level of inflammatory activity. High sensitivity C-reactive protein (hs-CRP) reflects the acute phase response in the liver, especially downstream of IL-6 signaling. IL-6 is a pleiotropic cytokine and may regulate the immune response and mediate metabolic and vascular adaptations. Tumor Necrosis Factor-alpha (TNF-α) also plays a role in inflammatory processes and has the potential to alter endothelial function [4,5,6]. Resting heart rate may reflect cardiovascular adaptations to obesity. The relationship between inflammatory markers (especially high sensitivity CRP) and obesity is well known. However, it has not been established whether a particular cytokine is responsible for increasing heart ate in individuals with obesity [7,8,9]. Blood pressure is the result of cardiac output and the peripheral vascular system. Additional factors that impact blood pressure include blood volume, the stiffness of arteries, and the control of the cardiovascular system by hormones. Activation of the immune system alters the levels of substances that regulate blood pressure and causes inflammation in blood vessels, especially in the kidneys. Some of these factors are depicted in the reviews, but the authors warn that determining the biological pathways and using biomarkers to identify those pathways may not always be the same [10,11,12]. Some population studies have related inflammatory indices to hypertension, and studies on adiposity have found significant variation by distribution of body fat and sex [13,14,15]. Only a few examples demonstrate the adjustment of biomarkers for resting heart rate and blood pressure, and/or confounding factors, and their effect on correlated dependent variables. This methodological study aims to address whether each biomarker has an adjusted association with the outcome measures. It also aims to determine whether at least one of the biomarkers has an adjusted positive association with resting heart rate and whether the biomarker associations with the two components of blood pressure differ.

Materials and Methods

Study Design and Study Population

A hospital-based cross-sectional analytical study was conducted in the Department of Patholog collaboration with general medicine, Government Medical College (GMC), Chhattisgarh. A total of 220 adults aged 18–65 years with obesity (BMI ≥30 kg/m²) were recruited. Written informed consent was obtained from all participants before enrolment.

Data Collection and Clinical Assessment

Demographic and clinical details, including age, sex, smoking status, physical activity, history of diabetes mellitus, and use of antihypertensive medication, were recorded using a structured proforma. Anthropometric measurements, including BMI and waist circumference, were documented. Resting heart rate and systolic and diastolic blood pressure were recorded using standard measurement procedures.

Biochemical Assessment

Blood samples were collected from the participants for the estimation of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α). The concentrations of these inflammatory biomarkers were determined using appropriate laboratory methods.

Statistical Analysis

Data were analysed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, and categorical variables as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test.

Spearman's correlation and the Welch t-test were used for correlation analysis and comparison of resting heart rate between sexes, respectively.
Multivariable linear regression models were developed separately for resting heart rate, systolic blood pressure, and diastolic blood pressure. Log-transformed hs-CRP, IL-6, and TNF-α were included simultaneously and adjusted for potential confounders. HC3 robust standard errors and 95% confidence intervals were calculated. Multiple comparisons were corrected using the Benjamini–Hochberg procedure. Statistical significance was set at an adjusted p-value of less than 0.05.

Results

Table 1. Demographic, clinical, and inflammatory characteristics (n = 220).

Characteristic	Mean ± SD or n (%)	Median (IQR)
Age (years)	40.64 ± 14.50	40.00 (27.00–54.00)
BMI (kg/m²)	34.61 ± 3.22	34.56 (32.11–36.62)
Waist (cm)	117.85 ± 7.45	117.74 (113.70–123.09)
Resting HR (beats/min)	73.92 ± 7.05	73.45 (69.38–77.96)
Systolic BP (mmHg)	125.79 ± 14.74	125.86 (116.79–135.65)
Diastolic BP (mmHg)	79.95 ± 8.62	79.91 (75.34–85.11)
hs-CRP (mg/L)	2.95 ± 1.69	2.53 (1.80–3.52)
IL-6 (pg/mL)	4.24 ± 2.28	3.77 (2.77–4.97)
TNF-α (pg/mL)	13.21 ± 4.84	12.30 (9.97–15.42)
Female, n (%)	126 (57.3%)	—
Current smoker, n (%)	49 (22.3%)	—
Diabetes, n (%)	30 (13.6%)	—
Antihypertensive use, n (%)	63 (28.6%)	—

Positive unadjusted Spearman's coefficients were found between hs-CRP and heart rate ($\rho = 0.28$), systolic pressure ($\rho = 0.24$) and diastolic pressure ($\rho = 0.25$). The crude IL-6 relationship with heart rate was stronger ($\rho = 0.41$). Relationships for TNF-α were less strong. The heatmap depicts the covariation among all six continuous biomarker-outcome measures. The unadjusted hs-CRP scatterplot should not be misconstrued as representing an independent effect. A Welch comparison showed that there was no sex difference in mean resting heart rate ($p = 0.400$). The three original biomarker scales all deviated from normality ($p < 0.001$) on the Shapiro-Wilk test.

Table 2. Unadjusted Spearman correlations between biomarkers and cardiovascular outcomes.

Biomarker	Resting HR (ρ)	Systolic BP (ρ)	Diastolic BP (ρ)
hs-CRP	0.28 (p <0.001)	0.24 (p <0.001)	0.25 (p <0.001)
IL-6	0.41 (p <0.001)	0.22 (p 0.001)	0.20 (p 0.003)
TNF-α	0.24 (p <0.001)	0.23 (p <0.001)	0.11 (p 0.094)

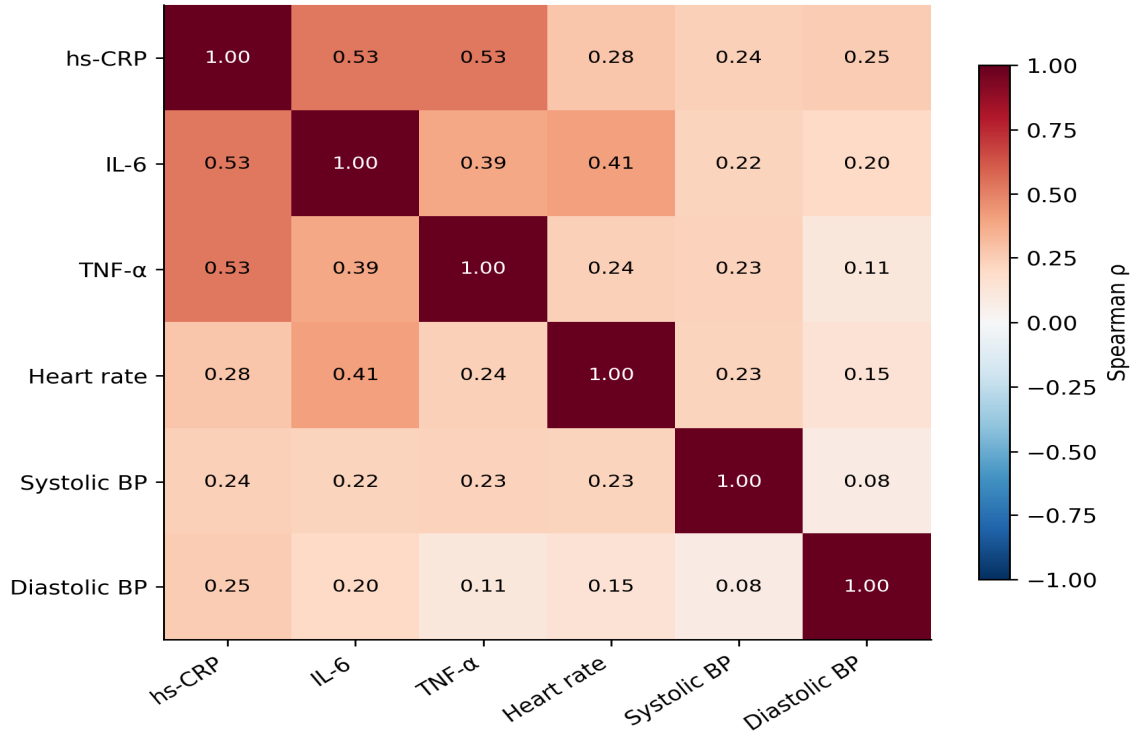


Figure 1. Spearman correlation matrix across biomarkers and outcomes; colours encode strength and direction.

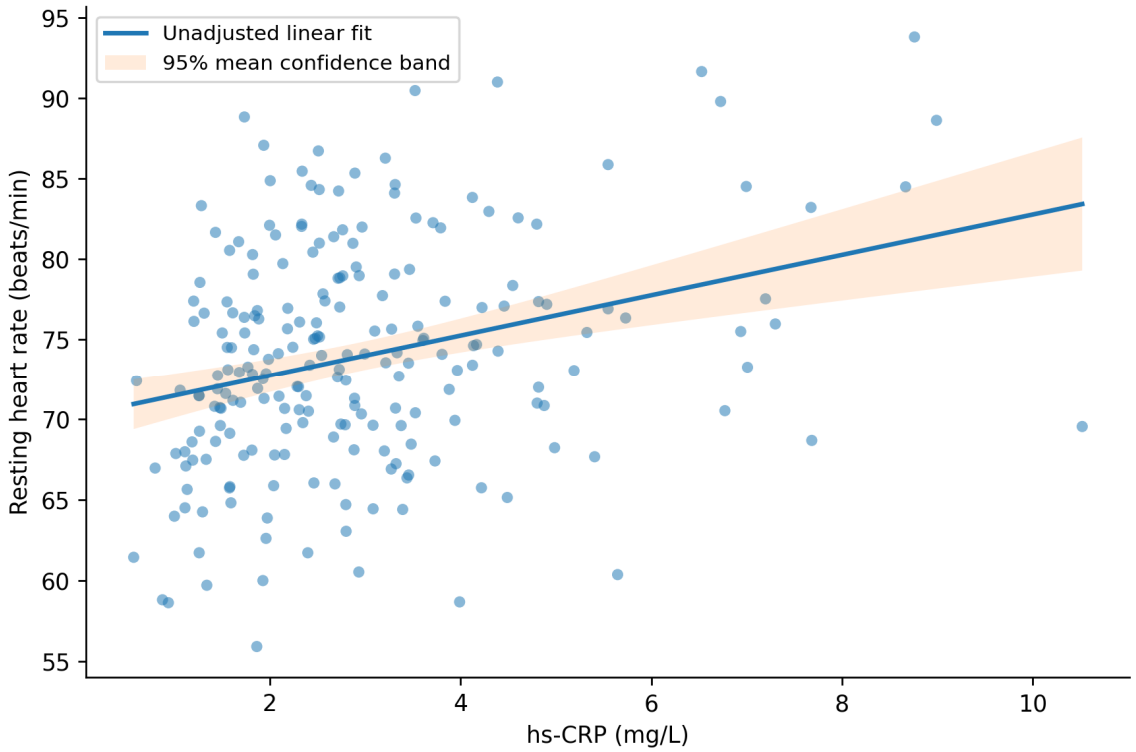


Figure 2. Unadjusted hs-CRP–heart-rate relationship and mean confidence band; not a causal or adjusted association.

According to the fully adjusted heart-rate model, IL-6 remained positively correlated with heart rate: β = 4.95 beats/min per log unit (95% CI 2.94 to 6.95; $p < 0.001$). The Benjamini–Hochberg-adjusted p-value was <0.001. In the model, hs-CRP and TNF-α showed no independent correlation with heart rate (adjusted p = 0.555 and 0.515, respectively). Adjusted R^2 was 0.268, leaving a considerable amount of variability at the record level, which was unexplained.

Table 3. Resting heart rate: multivariable regression with HC3 robust standard errors.

Predictor	β	SE	95% CI	p
ln(hs-CRP)	0.66	1.11	-1.51 to 2.83	0.552
ln(IL-6)	4.95	1.02	2.94 to 6.95	<0.001
ln(TNF-α)	1.21	1.44	-1.62 to 4.04	0.401
Age (years)	0.13	0.03	0.07 to 0.19	<0.001
Female	-0.96	0.91	-2.74 to 0.81	0.288
BMI (kg/m²)	0.19	0.16	-0.13 to 0.51	0.241
Current smoking	0.99	1.02	-1.00 to 2.99	0.329
Activity: moderate	-0.77	1.00	-2.74 to 1.19	0.440
Activity: high	-2.65	1.21	-5.02 to -0.29	0.028
Diabetes	-0.12	1.44	-2.95 to 2.70	0.931
Antihypertensive use	-1.88	1.17	-4.17 to 0.40	0.106

The systolic-pressure model yielded an IL-6 coefficient of 4.20 (95% CI 0.09 to 8.30) mmHg per log unit, with nominal p = 0.045. Its adjusted p = 0.136 did not meet the multiplicity criterion. For diastolic pressure, hs-CRP yielded 3.15 (95% CI 0.24 to 6.06) mmHg (nominal p = 0.034, adjusted p = 0.136). No other biomarker coefficient was distinguishable from zero with respect to the primary adjustment. Therefore, with respect to the nominal associations of pressure, we cannot make strong claims about different inflammatory pathways.

Table 4. Systolic and diastolic blood pressure: adjusted HC3 regression coefficients.

Predictor	SBP β	SBP 95% CI	p	DBP β	DBP 95% CI	p
ln(hs-CRP)	1.59	-2.03 to 5.21	0.389	3.15	0.24 to 6.06	0.034
ln(IL-6)	4.20	0.09 to 8.30	0.045	0.76	-1.77 to 3.29	0.555
ln(TNF-α)	3.57	-2.76 to 9.90	0.269	-2.37	-6.20 to 1.45	0.224
Age (years)	0.54	0.43 to 0.65	<0.001	0.18	0.10 to 0.26	<0.001
Female	-1.41	-4.63 to 1.82	0.393	0.15	-2.14 to 2.43	0.899
BMI (kg/m²)	0.39	-0.23 to 1.01	0.213	0.41	-0.01 to 0.83	0.056
Current smoking	0.40	-3.13 to 3.93	0.825	0.56	-2.00 to 3.11	0.669
Activity: moderate	-3.53	-7.10 to 0.04	0.053	-1.84	-4.50 to 0.81	0.174
Activity: high	-1.76	-6.74 to 3.22	0.489	-0.44	-3.64 to 2.76	0.789
Diabetes	3.46	-2.08 to 8.99	0.221	1.07	-2.30 to 4.43	0.534
Antihypertensive use	-4.21	-8.23 to -0.20	0.040	-4.05	-6.84 to -1.26	0.004

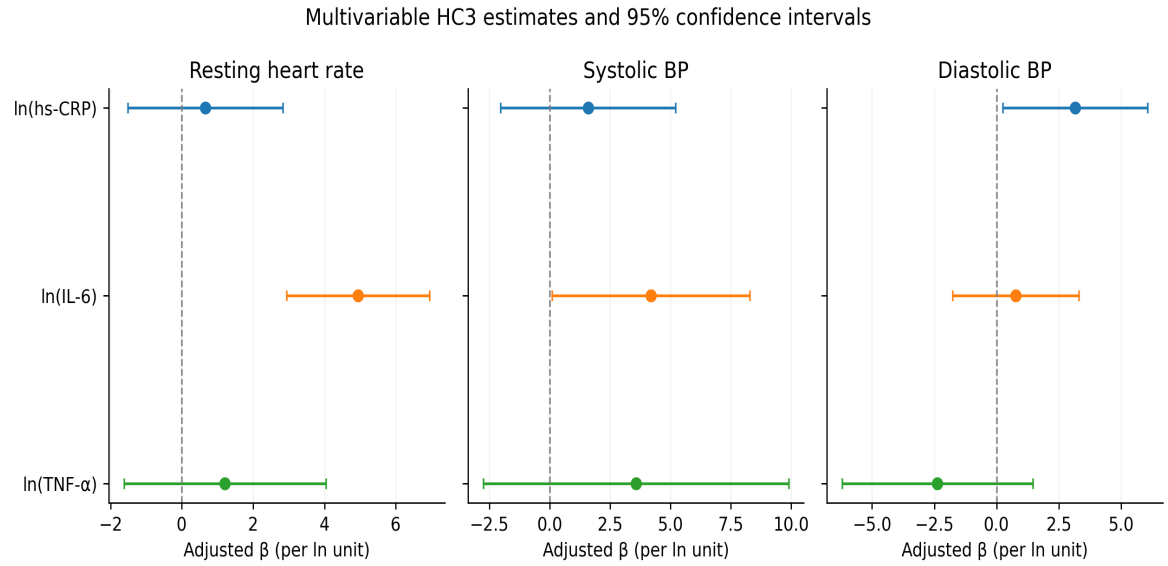


Figure 3. Adjusted biomarker estimates and 95% robust confidence intervals for the three outcomes.

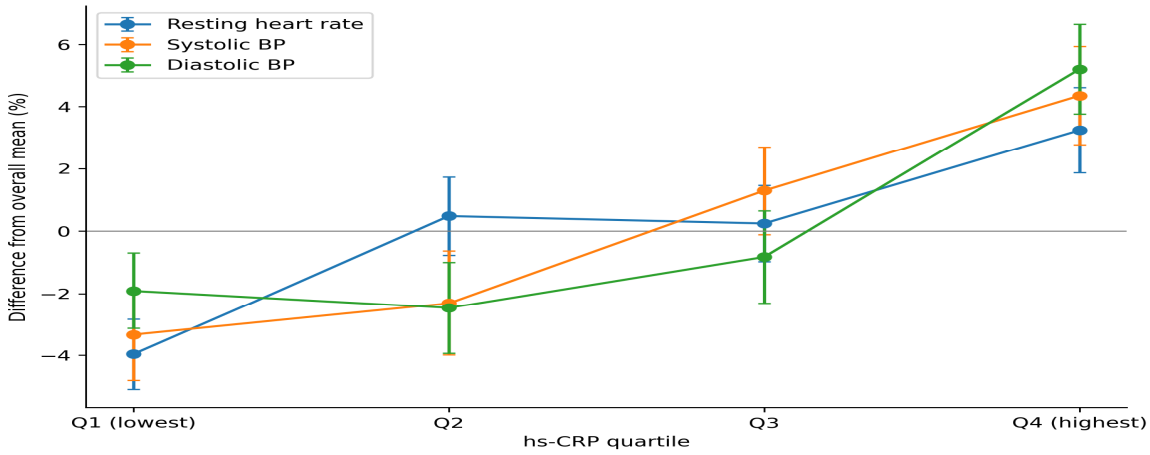


Figure 4. Outcome means across hs-CRP quartiles, shown as percentages relative to each outcome overall mean; bars denote standard errors.

The adjusted R^2 was 0.348 for systolic and 0.132 for diastolic blood pressure. The maximum predictor VIF was 1.85, suggesting no severe linear collinearity. The normality of the residuals of heart rate was tested using the residual-based-F test and the Breusch-Pagan test. The p-value for both tests was 0.945 and 0.199, respectively. The cook's distance screening threshold for 157 nonusers of antihypertensives was 4, and 13 records crossed the threshold, indicating these records require special attention. The heart-rate coefficient of hs-CRP was -0.19, p=0.893.

Table 5. Model fit, residual diagnostics, and nonuser sensitivity analysis.

Outcome	Adj. R^2	Shapiro p	BP test p	Cook's >4/n	Nonuser hs-CRP β (p)
Resting HR	0.268	0.945	0.199	13	-0.19 (p=0.893)
Systolic BP	0.348	0.566	0.258	10	0.71 (p=0.768)
Diastolic BP	0.132	0.101	0.259	14	4.66 (p=0.016)

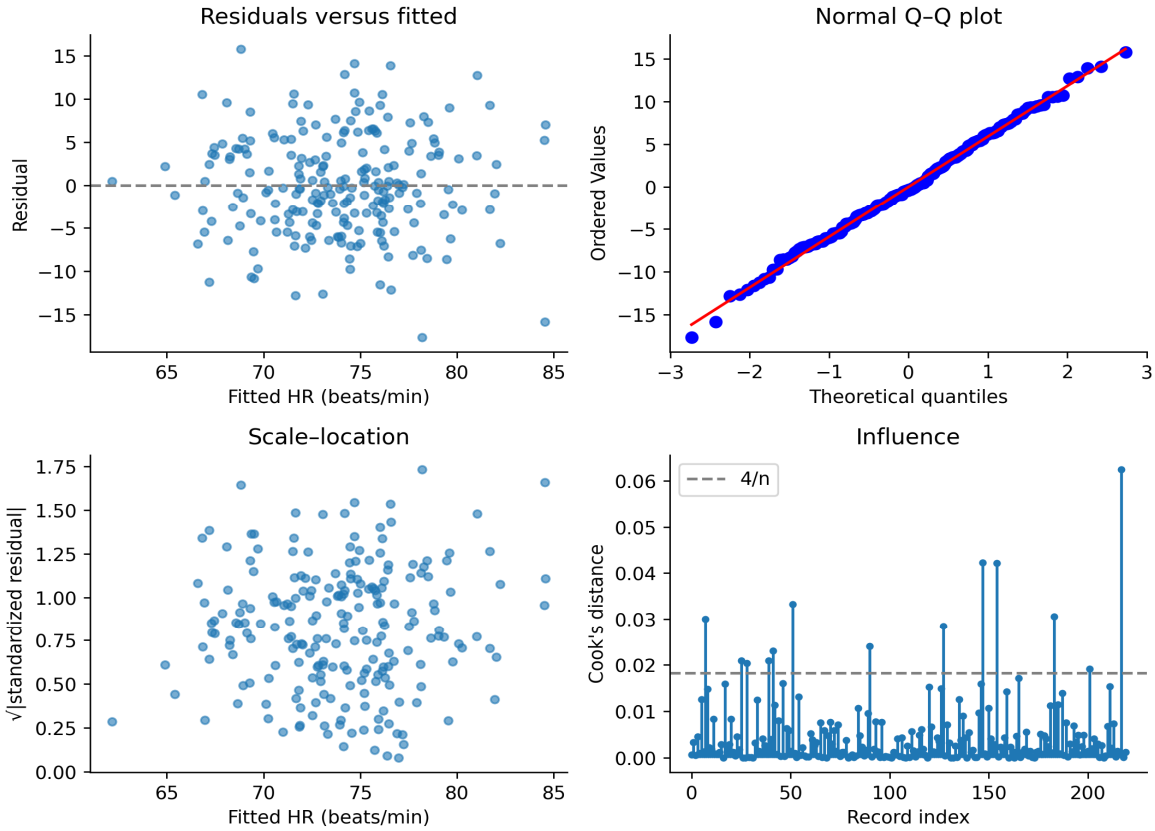


Figure 5. Heart-rate model residual, normality, scale–location, and influence diagnostics.

Note on Tables 3-5: Models account for all covariates listed. β values relate to a change in the natural log of each biomarker. The primary multiplicity correction applies to the nine biomarker coefficients. Abbreviations: BP, blood pressure; HC3, heteroscedasticity-consistent variance estimator; SE, standard error; IQR, interquartile range.

4. Discussion

This exercise illustrates that three inflammatory indicators should not be used interchangeably. All three, to some degree, had positive crude correlations with cardiovascular outcomes. However, after joint adjustment, the independent contribution of hs-CRP and TNF-α to resting heart rate was absent. In the heart rate model constructed, IL-6 was the only indicator which was significantly associated after the primary multiplicity adjustment. Since the mechanism also included IL-6, this demonstrates recovery of a specified signal, rather than a new human biological relationship.

One of the biological relationships that could be investigated in primary populations is the relationship between inflammation and neuro-autonomic activity. Resting heart rate can be impacted by a variety of factors, such as fitness, glucose regulation, sleep, and treatment. Evidence linking inflammation to increased heart rate in obesity, presents a case for testing rather than identifying a cause in a single observation [7,9]. Future studies could incorporate repeated measurements of resting pulse, heart rate variability, the timing of medication in relation to pulse measurements, objective activity, and sleep apnea, among other factors, to help discern the true inflammatory signal from the autonomic signal. Systolic and diastolic pressure yielded different nominal biomarker patterns. However, neither survival pressure finding was to be considered a false discovery. Reporting a pressures finding and assuming $p<0.05$ has created false certainty. Large observational studies have examined the prevalence and incidence of inflammation and hypertension, including the REGARDS study, while formal guidelines have prioritized the use of validated blood pressure measurement and conventional risk reduction [13,16,17]. A cytokine regression test should not be considered a hypertension screening test. Adipose inflammation may impact the cardiovascular system through circulating inflammatory mediators and endothelial nitric oxide, as well as dysfunction of perivascular adipose tissue [11,10]. Observational studies have reported that excess adiposity is associated with inflammation and have described sex-based differences and variation based on metabolic health [18,15,19]. Likewise, mechanistic reviews have described protective, partly immune responses and urged caution regarding uniform assumptions regarding all measured cytokines in different pathological conditions [5,20]. Additional literature provides context for the epidemiology of the estimated coefficients. Studies describe systemic inflammatory profiles associated with adiposity and hypertension and protein signatures associated with cardiovascular outcomes [14,21,22]. Studies related to obesity-related hypertension describe metabolic and vascular pathways, and change in cytokines due to an intervention suggest that some biomarkers are modifiable [23,24,25]. This diversity supports prespecified causal pathways, rather than post hoc selection of biomarkers. Log transformation was chosen because most acute phase reactants and cytokines are skewed. It also simplifies interpretability of regression coefficients. For example, a one-unit change in the log transform of a biomarker means that the biomarker level is multiplied by 2.71, not adding one milligram or microgram. The different reporting of BMI and waist circumference accounts for body-fat distribution in the reported data, while simultaneously avoiding redundant predictors in the main equations. Standard errors primarily account for uncertainty in the main equation estimation; they do not eliminate bias from confounders, nonlinearity, selection, and measurement error. Also, a negligible coefficient in a multiple regression does not prove an absence of biological association in the presence of a host of noisy and correlated biomarkers. The records provide a basis to examine the output of data processing software, to determine the consistency of interpretations

and calculations, and to instruct on the reproducibility of results, but they cannot estimate the prevalence of obesity-related hypertension in the population.

5. Conclusion

IL-6 was associated with heart rate in the adjusted model with the obesity-range data; however, no biomarker-pressure associations were significant after multiplicity adjustment. The goal of the analysis is to show the potential of joint biomarker adjustment and outcome inference. The goal of the research should not be to estimate patients' cardiovascular risk and make diagnosis and treatment decisions based on these coefficients. Studies that test inflammatory biomarkers along with other robust standards should be performed to determine if inflammation provides additional information beyond traditional clinical indicators of cardiovascular disease.

Declarations

Ethics Approval and Consent to Participate

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of Government Medical College, Chhattisgarh, India. Written informed consent was obtained from all participants before enrolment in the study.

Availability of Data and Materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

Competing Interests

The authors declare that they have no competing interests.

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Use of Artificial Intelligence

The authors declare that no artificial intelligence tools were used to generate or analyse the research data.

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