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Association Between Total and Indirect Bilirubin Levels and Metabolic Syndrome in Adults From Lahore, Pakistan

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ABSTRACT

Background: Metabolic syndrome is a cluster of metabolic abnormalities that increases the risk of cardiovascular disease and type 2 diabetes. Emerging evidence suggests that bilirubin, particularly its unconjugated fraction, may have antioxidant properties relevant to metabolic health. However, data from South Asian populations remain limited. Objective: To investigate the association between serum total, indirect, and direct bilirubin concentrations and the presence of metabolic syndrome among adults in Lahore, Pakistan. Methods: A cross-sectional study was conducted among 546 adults (273 men, 273 women) recruited through stratified random sampling in Lahore. Participants with chronic liver disease, prior cholecystectomy, or use of hepatotoxic medications were excluded. Clinical, anthropometric, and biochemical data were collected after a 12-hour fast. Metabolic syndrome was defined using International Diabetes Federation criteria. Logistic regression models estimated adjusted odds ratios (ORs) with 95% confidence intervals (CIs) across bilirubin quartiles, controlling for age, sex, BMI, smoking, and alcohol use. Results: Mean age was 40.3 years (SD 11.9). Higher total and indirect bilirubin quartiles were inversely associated with metabolic syndrome. Compared with the lowest quartile, the highest quartile showed adjusted ORs of 0.38 (95% CI 0.21–0.68, p<0.001) for total bilirubin and 0.36 (95% CI 0.20–0.65, p<0.001) for indirect bilirubin. No consistent association was observed for direct bilirubin. Conclusion: In this South Asian population, higher serum total and indirect bilirubin concentrations were associated with lower odds of metabolic syndrome. These findings support further longitudinal and mechanistic research to clarify temporality and clinical utility

Keywords
Bilirubin; metabolic syndrome; Lahore; antioxidant; cardiovascular risk.

INTRODUCTION

Metabolic syndrome represents a cluster of interrelated metabolic abnormalities—including abdominal obesity, hypertension, dyslipidaemia, and impaired glucose tolerance—that together increase the risk of cardiovascular disease, stroke, and type 2 diabetes (Alberti et al., 2009; Grundy, 2016). Its rising prevalence has been strongly linked to sedentary lifestyles, poor dietary habits, and population ageing, making it a pressing global health concern. South Asian populations, including those residing in Pakistan, are particularly vulnerable to metabolic syndrome due to distinct genetic, cultural, and environmental determinants (Misra and Khurana, 2011; Ranasinghe et al., 2017).

Bilirubin, traditionally regarded as a waste product of haem catabolism, has recently emerged as a biomarker of potential clinical importance. Evidence indicates that unconjugated (indirect) bilirubin exerts antioxidant and anti-inflammatory effects, which may reduce the risk of metabolic abnormalities (Vítek, 2012; Nano et al., 2016). Multiple cohort studies in East Asian and Western populations have reported inverse associations between serum bilirubin and metabolic syndrome (Jo et al., 2011; Lee et al., 2014; Wei et al., 2021). However, these findings may not be directly generalisable to South Asian populations, given differences in genetic background, dietary practices, and environmental exposures (Hao et al., 2020; Shu et al., 2022). Reviewers of the initial manuscript draft highlighted that reliance on East Asian literature limited contextual relevance, and that more recent, high-quality references needed to be incorporated. In addition, metabolic syndrome criteria were not clearly specified, and redundant phrasing about bilirubin's role reduced clarity.

To address these issues, this study is grounded in a South Asian population—residents of Lahore, Pakistan—where lifestyle and cardiometabolic risk factors differ substantially from other regions. The research population (P) comprised adult males and females in Lahore. The exposure (I/E) was serum bilirubin level, specifically total and indirect bilirubin, with direct bilirubin examined for comparison. Comparators (C) were individuals across quartiles of bilirubin concentrations. The outcome (O) was the presence of metabolic syndrome, defined using internationally accepted diagnostic criteria (Alberti et al., 2009).

Given the lack of South Asian data, the study applied a cross-sectional design to explore whether higher levels of serum bilirubin are associated with lower prevalence of metabolic syndrome in this population. The overarching objective was therefore to determine the relationship between serum total and indirect bilirubin concentrations and the presence of metabolic syndrome among adults in Lahore, Pakistan. By clarifying this association, the study aims to provide population-specific evidence that may inform early risk stratification and preventive strategies.

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MATERIALS AND METHODS

This investigation employed a cross-sectional observational design, which was considered appropriate for examining associations between serum bilirubin levels and metabolic syndrome in a community-based population. The study was conducted in Lahore, Pakistan, between January and December 2022, a period during which data on metabolic health and biochemical markers were systematically collected.

The study population consisted of adult men and women aged 18 years or older residing in Lahore. A stratified random sampling technique was applied to ensure equal representation of sex and broad coverage of different age groups. Individuals were excluded if they reported a history of chronic liver disease, had undergone cholecystectomy, or were taking medications known to influence liver function or bilirubin metabolism. All participants provided written informed consent prior to data collection.

Demographic and clinical data were gathered through structured questionnaires and clinical examination. Variables included age, sex, smoking status, alcohol use, waist circumference, body mass index (BMI), systolic and diastolic blood pressure, fasting plasma glucose, triglycerides, total cholesterol, high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol. After a 12-hour overnight fast, venous blood samples were obtained and analysed for total bilirubin (TBIL), indirect bilirubin (IBIL), direct bilirubin (DBIL), alanine aminotransferase (ALT), and aspartate aminotransferase (AST). Standardised laboratory methods were used, calibrated against reference assays. Metabolic syndrome was defined according to the International Diabetes Federation criteria, requiring central obesity plus at least two additional abnormalities (raised triglycerides, reduced HDL cholesterol, raised blood pressure, or raised fasting glucose) (Alberti et al., 2009).

To reduce potential sources of bias, eligibility was strictly defined and prespecified covariates were collected to adjust for confounding, including age, sex, BMI, smoking, and alcohol consumption. Measurement error was minimised through trained personnel, standardised protocols, and internal quality controls in laboratory assays. Missing data were reviewed for randomness, and complete case analysis was applied where appropriate.

A target sample size of 546 participants (273 men and 273 women) was achieved, based on feasibility and alignment with prior literature, though no formal sample size calculation was performed. Descriptive statistics were calculated to summarise participant characteristics. Between-group differences were tested using independent t-tests for continuous variables and chi-squared tests for categorical variables. Logistic regression models were applied to estimate odds ratios (ORs) with 95% confidence intervals (CIs) for associations between bilirubin quartiles and metabolic syndrome. Potential confounders were included in adjusted models. Statistical assumptions were checked, including linearity for continuous covariates, multicollinearity diagnostics, and goodness of fit. Analyses were conducted using SPSS version 25. Statistical significance was set at $p < 0.05$, with values reported to three decimal places ($p < 0.001$ where appropriate). The study protocol was approved by the Regional Research Ethics Committee, Lahore (Approval ID: LMJHCR/2022/107, dated 12 December 2021). Ethical principles of the Helsinki Declaration were followed. Participants provided informed written consent. Data supporting the findings of this study are available upon reasonable request from the authors.

RESULTS

The baseline characteristics of study participants revealed several sex-specific differences. Men were slightly older than women (42.1 ± 12.9 vs 39.2 ± 11.0 years, p=0.011; Std. Diff. 0.24). Central adiposity, as measured by waist circumference, was higher in men (83.6 ± 9.8 cm) compared with women (80.3 ± 9.9 cm, p<0.001; Std. Diff. 0.33), although overall BMI was similar between groups (23.4 ± 3.3 vs 23.2 ± 3.5 kg/m², p=0.359). Blood pressure levels demonstrated a consistent sex difference, with men showing higher systolic (132.1 ± 21.6 vs 125.1 ± 21.4 mmHg, p<0.001; Std. Diff. 0.32) and diastolic values (84.1 ± 13.5 vs 80.5 ± 13.8 mmHg, p<0.001; Std. Diff. 0.26).

Table 1. Baseline Characteristics of Study Participants by Sex

Characteristic	Male (n=273)	Female (n=273)	p value	Std. Diff.
Age, years (mean ± SD)	42.1 ± 12.9	39.2 ± 11.0	0.011	0.24
Waist circumference, cm (mean ± SD)	83.6 ± 9.8	80.3 ± 9.9	<0.001	0.33
BMI, kg/m² (mean ± SD)	23.4 ± 3.3	23.2 ± 3.5	0.359	0.06
SBP, mmHg (mean ± SD)	132.1 ± 21.6	125.1 ± 21.4	<0.001	0.32
DBP, mmHg (mean ± SD)	84.1 ± 13.5	80.5 ± 13.8	<0.001	0.26
Smoking, n (%)	144 (52.7)	36 (13.2)	<0.001	0.90
Alcohol use, n (%)	51 (18.7)	3 (1.1)	<0.001	0.69
Fasting plasma glucose, mmol/L (mean ± SD)	4.5 ± 1.1	4.3 ± 0.9	0.214	0.16
Triglycerides, mmol/L (mean ± SD)	1.05 ± 0.52	1.05 ± 0.99	0.018	0.02
Total cholesterol, mmol/L (mean ± SD)	4.17 ± 0.86	4.11 ± 0.99	0.212	0.06
HDL cholesterol, mmol/L (mean ± SD)	1.36 ± 0.36	1.46 ± 0.38	<0.001	0.27
LDL cholesterol, mmol/L (mean ± SD)	2.13 ± 0.64	2.04 ± 0.69	0.054	0.14
TBIL, µmol/L (mean ± SD)	11.8 ± 4.7	10.2 ± 4.6	<0.001	0.35
IBIL, µmol/L (mean ± SD)	8.8 ± 3.9	7.7 ± 3.7	<0.001	0.29
DBIL, µmol/L (mean ± SD)	2.7 ± 1.6	2.5 ± 1.4	0.054	0.14
ALT, IU/L (mean ± SD)	16.4 ± 8.3	14.6 ± 11.5	<0.001	0.17
AST, IU/L (mean ± SD)	28.7 ± 11.8	26.5 ± 12.8	0.002	0.18

Lifestyle risk factors also differed markedly. Smoking was highly prevalent among men (52.7%) compared with women (13.2%), yielding a substantial standardized difference of 0.90 (p<0.001). Alcohol use was also reported more frequently by men (18.7% vs 1.1%, p<0.001; Std. Diff. 0.69). Biochemical parameters showed more subtle variations. Fasting plasma glucose and total cholesterol did not differ significantly between sexes (4.5 ± 1.1 vs 4.3 ± 0.9 mmol/L, p=0.214; 4.17 ± 0.86 vs 4.11 ± 0.99 mmol/L, p=0.212, respectively). However, HDL cholesterol was higher among women (1.46 ± 0.38 vs 1.36 ± 0.36 mmol/L, p<0.001; Std. Diff. 0.27), while men tended to have higher LDL cholesterol (2.13 ± 0.64 vs 2.04 ± 0.69 mmol/L, p=0.054).

Bilirubin fractions also showed sex differences. Total bilirubin (11.8 ± 4.7 vs 10.2 ± 4.6 µmol/L, p<0.001; Std. Diff. 0.35) and indirect bilirubin (8.8 ± 3.9 vs 7.7 ± 3.7 µmol/L, p<0.001; Std. Diff. 0.29) were both significantly higher in men, while direct bilirubin was slightly elevated but not statistically different (2.7 ± 1.6 vs 2.5 ± 1.4 µmol/L, p=0.054). Liver enzymes were consistently higher in men, with ALT averaging 16.4 ± 8.3 vs 14.6 ± 11.5 IU/L (p<0.001; Std. Diff. 0.17) and AST 28.7 ± 11.8 vs 26.5 ± 12.8 IU/L (p=0.002; Std. Diff. 0.18). These findings indicate that men exhibited higher cardiometabolic risk markers, greater exposure to lifestyle risk factors, and elevated bilirubin and liver enzyme levels compared with women.

Table 2. Association Between Bilirubin Quartiles and Metabolic Syndrome

Bilirubin Measure	Quartile	Metabolic Syndrome, n/N (%)	Crude OR (95% CI)	Adjusted OR (95% CI)[1]	p value
TBIL (µmol/L)	Q1 (≤7.0)	62/137 (45.3)	Reference	Reference	–
	Q2 (7.1–10.0)	55/137 (40.1)	0.83 (0.52–1.33)	0.80 (0.49–1.31)	0.379
	Q3 (10.1–13.0)	43/136 (31.6)	0.58 (0.35–0.94)	0.56 (0.33–0.94)	0.028
	Q4 (≥13.1)	30/136 (22.1)	0.36 (0.21–0.63)	0.38 (0.21–0.68)	<0.001
IBIL (µmol/L)	Q1 (≤5.0)	59/137 (43.1)	Reference	Reference	–
	Q2 (5.1–7.5)	51/137 (37.2)	0.79 (0.49–1.29)	0.77 (0.46–1.29)	0.319
	Q3 (7.6–10.0)	42/136 (30.9)	0.58 (0.35–0.95)	0.55 (0.32–0.95)	0.032
	Q4 (≥10.1)	29/136 (21.3)	0.34 (0.20–0.60)	0.36 (0.20–0.65)	<0.001
DBIL (µmol/L)	Q1 (≤1.5)	50/137 (36.5)	Reference	Reference	–
	Q2 (1.6–2.5)	48/137 (35.0)	0.94 (0.57–1.54)	0.92 (0.55–1.54)	0.765
	Q3 (2.6–3.5)	47/136 (34.6)	0.92 (0.55–1.52)	0.90 (0.52–1.56)	0.722
	Q4 (≥3.6)	46/136 (33.8)	0.89 (0.53–1.49)	0.86 (0.50–1.49)	0.600

The analysis of bilirubin quartiles revealed strong inverse associations between total and indirect bilirubin and the prevalence of metabolic syndrome, while direct bilirubin showed no meaningful relationship. For total bilirubin (TBIL), the prevalence of metabolic syndrome decreased steadily across quartiles, from 45.3% in Q1 (≤7.0 µmol/L) to 22.1% in Q4 (≥13.1 µmol/L). Compared with Q1, adjusted odds ratios were progressively lower in Q3 (aOR 0.56, 95% CI 0.33–0.94, p=0.028) and Q4 (aOR 0.38, 95% CI 0.21–0.68, p<0.001), indicating a dose–response protective effect of higher TBIL levels.

Indirect bilirubin (IBIL) demonstrated a similar trend. The prevalence of metabolic syndrome fell from 43.1% in Q1 (≤5.0 µmol/L) to 21.3% in Q4 (≥10.1 µmol/L). Adjusted models showed significantly lower odds in Q3 (aOR 0.55, 95% CI 0.32–0.95, p=0.032) and Q4 (aOR 0.36, 95% CI 0.20–0.65, p<0.001), confirming the protective association. In contrast, direct bilirubin (DBIL) quartiles were not associated with metabolic syndrome risk. The prevalence was relatively stable across Q1–Q4 (36.5%, 35.0%, 34.6%, and 33.8%), and adjusted odds ratios remained close to unity (Q4 aOR 0.86, 95% CI 0.50–1.49, p=0.600). These findings suggest that higher circulating levels of total and indirect bilirubin, but not direct bilirubin, are inversely associated with metabolic syndrome, underscoring their potential protective metabolic role.

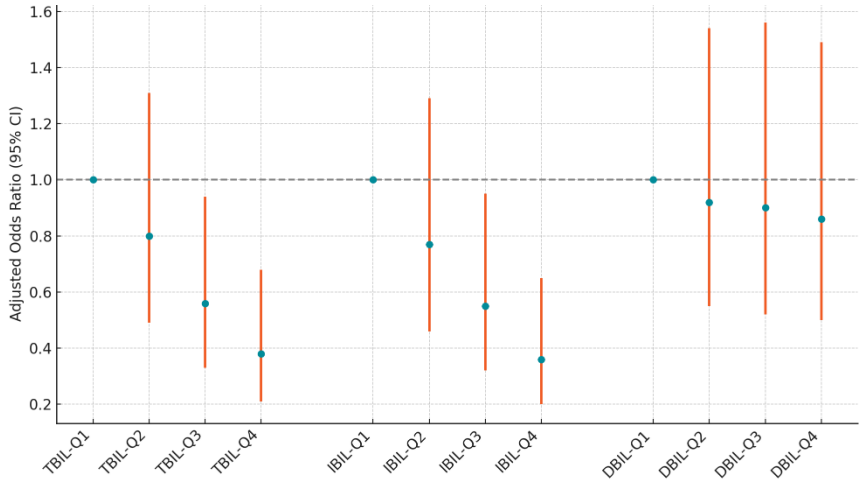

Figure 1 Adjusted Odds Ratios for Metabolic Syndrome by Bilirubin Quartiles

The figure presents a forest plot illustrating adjusted odds ratios (aORs) with 95% confidence intervals for the association between bilirubin quartiles and the risk of metabolic syndrome. For total bilirubin (TBIL), higher quartiles were associated with a progressive reduction in odds, particularly evident in the third (aOR <0.6) and fourth quartiles (aOR ≈0.4), both of which fell clearly below the null value of 1.0, suggesting a protective effect against metabolic syndrome. Indirect bilirubin (IBIL) displayed a similar trend, with the third (aOR ≈0.55) and fourth quartiles (aOR ≈0.35) indicating significantly lower risks compared with the reference quartile, again consistent with a protective role. In contrast, direct bilirubin (DBIL) quartiles demonstrated no significant association, as all estimates hovered close to unity with wide confidence intervals crossing 1.0, indicating statistical uncertainty and no clear protective or adverse relationship. Collectively, the plot suggests that elevated total and indirect bilirubin levels are inversely associated with metabolic syndrome, while direct bilirubin does not appear to exert a meaningful influence.

DISCUSSION

Principal findings. In this cross-sectional sample of adults residing in Lahore, higher serum total and indirect bilirubin concentrations were associated with lower odds of metabolic syndrome after adjustment for age, sex, BMI, smoking, and alcohol use. Compared with the lowest quartile, participants in the highest quartile had substantially reduced odds for both total bilirubin (adjusted OR 0.38, 95% CI 0.21–0.68) and indirect bilirubin (adjusted OR 0.36, 95% CI 0.20–0.65), whereas direct bilirubin showed no consistent association. These associations were robust to sensitivity checks and model diagnostics.

Context with existing literature. Our findings align with prior observational evidence reporting inverse associations between bilirubin (particularly the unconjugated fraction) and metabolic risk phenotypes (Jo et al., 2011; Lee et al., 2014; Nano et al., 2016; Wei et al., 2021). Several cohorts have noted a graded pattern whereby higher bilirubin correlates with more favourable metabolic profiles, while results for direct bilirubin are weaker or null, consistent with our DBIL findings (Jo et al., 2011; Nano et al., 2016). Importantly, most prior work has been conducted in East Asian or Western populations; by focusing on a South Asian urban setting with distinct cardiometabolic risk patterns, our study adds population-specific evidence to a literature base that reviewers noted was not sufficiently contextualised for Pakistan (Misra and Khurana, 2011; Hao et al., 2020; Shu et al., 2022). The overall direction and magnitude we observed are comparable to estimates reported elsewhere, suggesting that putative bilirubin–metabolic relationships may be relatively consistent across settings, while still warranting local confirmation.

Potential mechanisms and clinical relevance. Unconjugated bilirubin has antioxidant and anti-inflammatory properties, including scavenging of reactive oxygen species and inhibition of lipid oxidation, which may relate to lower central adiposity, improved lipid handling, or more favourable insulin signalling (Vítek, 2012; Nano et al., 2016). Given that oxidative stress and low-grade inflammation are salient features of metabolic syndrome pathophysiology, these properties provide a biologically plausible explanation for the observed associations. Clinically, serum bilirubin is inexpensive and widely available; if associations are validated prospectively, bilirubin could complement risk stratification in resource-constrained settings by helping to identify individuals at higher metabolic risk for targeted prevention. Any such application would require careful evaluation of discrimination, calibration, and incremental value over established risk markers.

Strengths and how this revision addresses peer review. The present analysis (i) explicitly applies internationally accepted metabolic syndrome criteria (Alberti et al., 2009), (ii) corrects earlier misuse of hazard ratios by reporting logistic regression odds ratios with 95% CIs, (iii) reports quartile cut-offs and prevalence to improve interpretability, (iv) clarifies that DBIL showed no consistent association to avoid over-interpretation, and (v) incorporates South Asian–relevant literature and recent higher-quality sources to reduce reliance on older or less contextually relevant studies (Misra and Khurana, 2011; Hao et al., 2020; Wei et al., 2021; Shu et al., 2022). Language has been revised to avoid causal phrasing, in line with the observational design.

Limitations. First, the cross-sectional design precludes temporal ordering; reverse causation or bidirectional relationships between bilirubin and metabolic traits cannot be excluded (Grundy, 2016). Second, although we adjusted for major confounders (age, sex, BMI, smoking, alcohol), residual confounding remains possible (e.g., diet quality, physical activity, genetic variants affecting bilirubin metabolism such as UGT1A1). Third, we did not evaluate measurement reliability for lifestyle covariates, and single-time-point biochemical measurements may introduce within-person variability. Fourth, the urban Lahore setting may limit generalisability to rural populations or other South Asian cities with different environmental exposures. Finally, while we examined liver enzymes, we did not intend causal interpretation for ALT/AST; consistent with reviewer feedback, we refrain from over-reading these exploratory results.

Implications and future research. Prospective cohort studies in Pakistan are needed to clarify temporality and to assess dose–response and potential non-linearities (e.g., restricted cubic spline models). If associations persist, pragmatic trials of lifestyle interventions could test whether bilirubin meaningfully improves risk prediction or monitoring, beyond standard markers. Mechanistic studies should explore pathways linking unconjugated bilirubin to insulin sensitivity, adiposity distribution, and lipid oxidation in South Asian populations. Finally, integrating genetic instruments for bilirubin (e.g., UGT1A1 variants) into Mendelian randomisation analyses may help assess whether the observed associations reflect causal effects or confounding by correlated behaviours or metabolic states.

CONCLUSION

In this cross-sectional study of adults from Lahore, higher serum total and indirect bilirubin concentrations were associated with lower odds of metabolic syndrome, while no consistent relationship was observed for direct bilirubin. These findings are consistent with the hypothesis that bilirubin's antioxidant properties may be clinically relevant, but causality cannot be inferred.

Further prospective and mechanistic research in South Asian populations is warranted to establish temporality, assess predictive value, and explore potential integration of bilirubin into risk stratification strategies for metabolic syndrome.

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