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ORIGINAL ARTICLE

Evaluation of Serum Zinc as a Potential Biomarker of Dyslipidemia among Obese Adults

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ABSTRACT

Background: *Dyslipidemia is a common metabolic disorder among obese adults, and emerging evidence suggests that micronutrient imbalance, particularly serum zinc deficiency, may be linked to altered lipid metabolism. This study aimed to evaluate serum zinc as a potential biomarker of dyslipidemia among obese adults.* ***Methods & Materials:*** *This comparative cross-sectional study was conducted in the Department of Physiology at Sylhet MAG Osmani Medical College, Sylhet, Bangladesh, from July 2022 to June 2023. A total of 60 individuals attending various Outpatient Departments of Sylhet MAG Osmani Medical College Hospital were enrolled. Participants were divided into two groups: Group A (Obese individuals with BMI ≥ 25 kg/m^2), and Group B (Healthy individuals with normal BMI 18.5-24.9 kg/m^2).* ***Results:*** *The demographic characteristics were comparable between the groups ($p > 0.05$). Obese participants exhibited significantly higher TC, TG, and LDL-C levels and lower HDL-C levels compared to healthy controls ($p < 0.01$). Serum zinc was significantly reduced in Group A (10.15 ± 1.03 µmol/L) compared to Group B (13.13 ± 1.34 µmol/L; $p < 0.001$). Regression analyses revealed moderate linear correlations between serum zinc and TC ($r = –0.503$), TG ($r = –0.591$), HDL-C ($r = 0.616$), and LDL-C ($r = –0.546$), all of which were statistically significant ($p < 0.001$). Each lipid parameter significantly predicted serum zinc levels, with HDL-C showing the strongest predictive association.* ***Conclusion:*** *Serum zinc levels are significantly associated with lipid abnormalities in obese adults and may serve as a potential biomarker for dyslipidemia. Further longitudinal studies are recommended to validate its predictive utility.*

Keywords: *Serum zinc, Dyslipidemia, Obesity, Lipid Profile, Biomarker*

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INTRODUCTION

Obesity, defined as excessive fat accumulation, has been among the top five global risk factors contributing to premature mortality since 1990 [1]. It has emerged as a major worldwide public health concern due to its strong association with multiple metabolic comorbidities, including dyslipidemia and cardiovascular diseases [2]. According to the World Health Organization, approximately 13% of the world's adult population was classified as obese in 2016, and the global prevalence of obesity has nearly tripled between 1975 and 2016 [3]. Currently, obesity ranks as the fifth leading cause of mortality and significantly reduces quality of life, imposing substantial economic burdens on societies [4].

The development of obesity is multifactorial, involving genetic predisposition, environmental influences, and nutritional factors such as intake of vegetables, fruits, and red meat [5–7]. Furthermore, increasing evidence suggests that obesity is closely linked to oxidative stress and chronic low-grade inflammatio [8,9]. Data from the most recent National Survey of Health and Nutrition in Mexico (ENSANUT, 2018) reported obesity and elevated serum triglyceride (TG) and total cholesterol (TC) levels in 36.1% and 19.5% of adults aged 20 years and older, respectively[10].

Dyslipidemia is typically characterized by elevated concentrations of TC, low-density lipoprotein cholesterol (LDL-C), and TG, accompanied by reduced levels of high-density lipoprotein cholesterol (HDL-C). Substantial evidence shows that dyslipidemia significantly increases the risk of atherosclerosis and cardiovascular disease in adults[11]. Elevated LDL-C levels, in particular, are recognized as key early-stage contributors to atherosclerosis and major risk factors for ischemic heart disease and stroke[12]. The primary environmental and biological contributors to dyslipidemia include high intake of fats and carbohydrates, inadequate physical activity, obesity, and certain genetic determinants[13]. With rapid lifestyle and dietary transitions worldwide, the

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prevalence of obesity and dyslipidemia has risen sharply over the past two decades, and it is estimated that 65% of the global population now lives in countries where overweight and obesity cause more morbidity than undernutrition[14].

Zinc is an essential trace mineral involved in numerous metabolic pathways, functioning as a catalytic, regulatory, and structural component in human physiology. It serves as a cofactor for more than 300 enzymes, including carbonic anhydrase, alcohol dehydrogenase, and alkaline phosphatase and is integral to over 2,500 transcription factors[15,16]. As an antioxidant micronutrient, zinc plays an important role in modulating oxidative stress and inflammation[8]. Several studies have explored differences in serum zinc levels between obese and normal-weight individuals, with many reporting significantly lower zinc levels among obese patients[8,17,18].

More recent evidence suggests that zinc status may be linked to cardiovascular risk through its influence on lipid metabolism, insulin secretion, and insulin sensitivity[13,19,20]. Notably, Cinar et al. demonstrated that zinc supplementation over six weeks resulted in increased HDL-C levels and reductions in TC, LDL-C, and TG concentrations[21]. These findings highlight a potential role for zinc as an important biomarker related to metabolic health, particularly among individuals with obesity.
Therefore, in the present study, we aimed to evaluate serum zinc as a potential biomarker of dyslipidemia among obese adults.

METHODS & MATERIALS

This comparative cross-sectional study was conducted in the Department of Physiology, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh, from July 2022 to June 2023. In this study, we included 60 individuals who attended different Outpatient Departments of Sylhet MAG Osmani Medical College Hospital. The study participants were categorized into two groups based on their BMI in accordance with the WHO guidelines for the Asian population:

- **Group A:** Obese individuals with a BMI ≥ 25 kg/m^2 (n = 30)
- **Group B:** Healthy individuals with a BMI between 18.5 and 24.9 kg/m^2 (n = 30)

These were the following criteria for eligibility as study participants:

Inclusion Criteria

- Adults aged 18–60 years
- Obese Individuals with Body Mass Index (BMI) ≥ 25 kg/m^2
- Normal Individuals with normal BMI
- Individuals willing to provide informed consent

Exclusion Criteria

- Presence of any acute infection
- Diagnosed cases of diabetes mellitus, hypertension & CKD
- Known or suspected malignancy
- Current or recent use of medications such as antipsychotics, antidepressants, hormonal contraceptives, corticosteroids & zinc supplementation
- History of malabsorption syndromes

Data Collection Procedure: Sociodemographic and clinical data were recorded using a structured questionnaire. Informative written consent was obtained after an explanation of the study procedure. After an overnight fast, 5 mL of venous blood was collected from each participant. Serum total cholesterol, serum triglyceride and serum high-density lipoprotein cholesterol were measured by enzymatic method with a fully automated biochemistry analyzer (Vitros 5600, USA). Friedewald's formula was used to calculate serum low-density lipoprotein cholesterol. Serum zinc was measured by photometric method with a fully automated biochemistry analyzer (Konelab Prime 60i, Thermo Fisher Scientific, USA).

Statistical Analysis: All data were recorded systematically in a pre-formatted data collection form. Quantitative data were expressed as mean and standard deviation, and qualitative data were expressed as frequency distribution and percentage. Group comparisons were performed using the independent samples t-test. Simple linear regression analyses were conducted to determine the predictive relationship between serum lipid parameters and serum zinc levels. Correlation coefficients (R), beta coefficients (β), coefficient of determination (R^2), F-statistics, and p-values were reported. A p-value <0.05 was considered significant. Statistical analysis was performed by using SPSS 26 (Statistical Package for Social Sciences). This study was ethically approved by the Institutional Review Committee of Sylhet MAG Osmani Medical College.

RESULTS

Table – I: Baseline Demographic Profile of the Study Participants (*n* = 60)

Age	Group A (n = 30)	Group B (n = 30)	P-value
21–30 years	8 (26.7%)	8 (26.7%)	1.000
31–40 years	11 (36.7%)	11 (36.7%)	
41–50 years	6 (20.0%)	6 (20.0%)	
51–60 years	5 (16.6%)	5 (16.6%)	
Mean age ± SD	39.73 ± 10.82	36.80 ± 6.42	0.212
Gender			
Male	13 (43.3%)	13 (43.3%)	1.000
Female	17 (56.7%)	17 (56.7%)	

Group A = Obese, Group B = Healthy Individual

Table I shows the distribution of age and gender among the study participants. The age categories were identical in both groups, with no significant difference observed (p = 1.000). The mean age was slightly higher in Group A (39.73 ± 10.82 years) compared to Group B (36.80 ± 6.42 years), although this difference was not statistically significant (p = 0.212). Gender distribution was also similar between the two groups, with 13 males (43.3%) and 17 females (56.7%) in both Group A and Group B. No statistically significant difference was found between the groups regarding gender (p = 1.000).

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Table – II: Comparison of Lipid Parameters and Serum Zinc Levels Between the Study Groups

Lipid Parameters	Group A (n = 30)	Group B (n = 30)	P-value
TC (mg/dL)	203.43 ± 36.49	175.90 ± 33.01	0.003
TG (mg/dL)	188.53 ± 36.93	145.30 ± 32.22	< 0.001
HDL-C (mg/dL)	39.43 ± 5.93	47.43 ± 7.93	0.001
LDL-C (mg/dL)	104.76 ± 33.78	86.17 ± 8.77	0.006
Serum Zinc Level			
Serum Zinc (µmol/L)	10.15 ± 1.03	13.13 ± 1.34	< 0.001

Table II shows that the mean total cholesterol (TC) was significantly higher in Group A (203.43 ± 36.49 mg/dL) compared to Group B (175.90 ± 33.01 mg/dL; t = 3.064, p = 0.003). Similarly, triglyceride (TG) levels were elevated in Group A (188.53 ± 36.93 mg/dL) relative to Group B (145.30 ± 32.22 mg/dL), with the difference being highly significant (t = 4.831, p < 0.001). In contrast, high-density lipoprotein cholesterol (HDL-C) was lower in Group A (39.43 ± 5.93 mg/dL) than in Group B (47.43 ± 7.93 mg/dL), and this difference was statistically significant (t = 3.319, p = 0.001). Low-density lipoprotein cholesterol (LDL-C) was also higher in Group A (104.76 ± 33.78 mg/dL) compared to Group B (86.17 ± 8.77 mg/dL), with a significant difference observed between the groups (t = 2.903, p = 0.006). Serum zinc levels were markedly lower in Group A (10.15 ± 1.03 µmol/L) than in Group B (13.13 ± 1.34 µmol/L), and this difference was highly significant (p < 0.001).

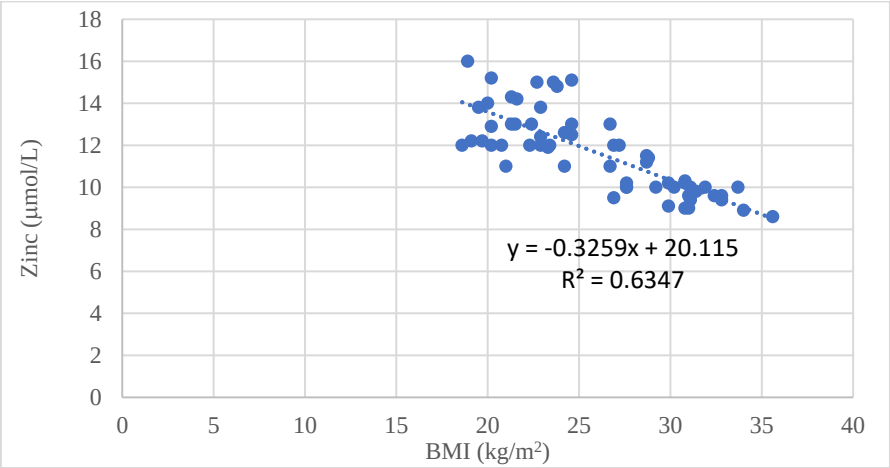

Figure – 1: Relationship between BMI and Serum Zinc Level

Figure 1 presents the results of a simple linear regression analysis conducted to evaluate the predictive relationship between body mass index (BMI) and serum zinc levels. A strong negative correlation was observed between BMI and serum zinc (r = −0.797). The fitted regression model for BMI was −0.3259x + 20.115. The overall model was statistically significant (r^2 = 0.6347, F = 100.782, p < 0.001), indicating that BMI is a significant predictor of serum zinc levels. Specifically, higher BMI values were associated with lower serum zinc concentrations (β = −0.797, p < 0.001).

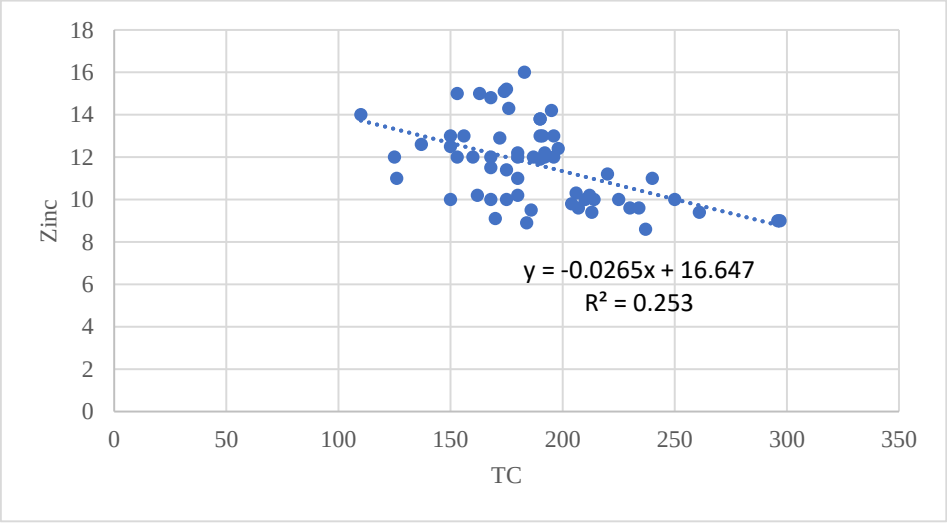

Figure – 2: Relationship between TC and Serum Zinc Level

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Figure 2 illustrates the results of a simple linear regression analysis conducted to assess the predictive relationship between serum total cholesterol (TC) and serum zinc levels. A moderate negative correlation was observed between serum zinc and TC (r = −0.503). The fitted regression model for serum TC was -0.0265x + 16.647. The overall regression model was statistically significant (r^2 = 0.253, F = 19.647, p < 0.001), indicating that serum TC is a significant predictor of serum zinc levels (β = -0.503, p < 0.001).

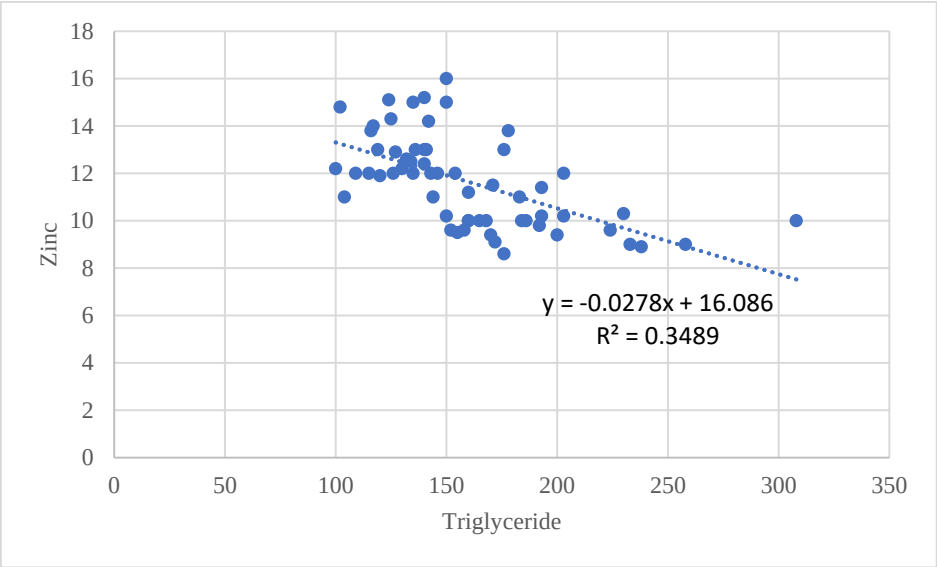

Figure – 3: Relationship between TG and Serum Zinc Level

Figure 3 illustrates the results of a simple linear regression analysis examining the predictive relationship between serum triglycerides (TG) and serum zinc levels. A moderate negative correlation was observed between serum zinc and TG (r = −0.591). The fitted regression model for serum TG was -0.0278x + 16.086. The overall regression model was statistically significant (r^2 = 0.349, F = 31.082, p < 0.001), indicating that serum TG is a significant predictor of serum zinc levels (β = -0.591, p < 0.001).

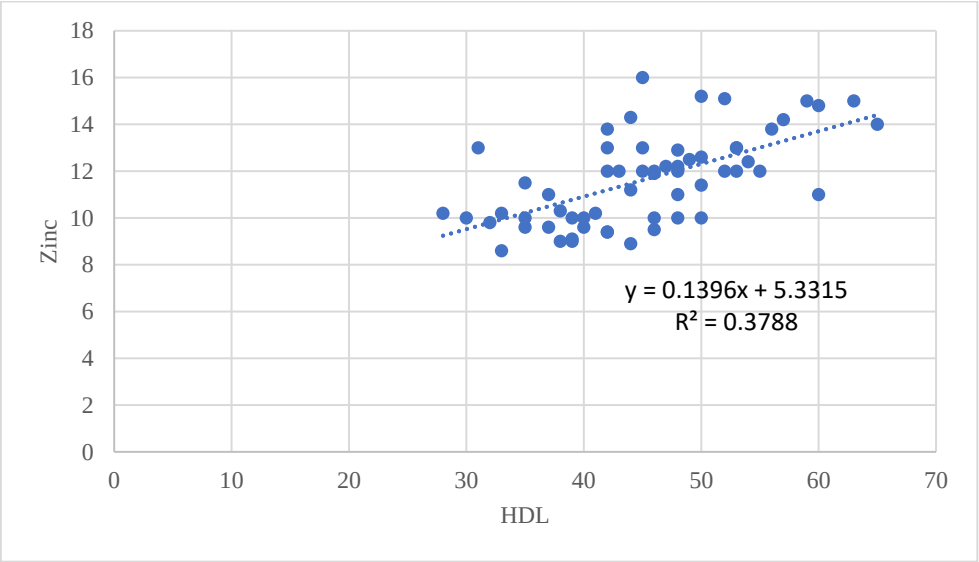

Figure – 4: Relationship between HDL and Serum Zinc Level

Figure 4 illustrates the results of a simple linear regression analysis examining the predictive relationship between serum high-density lipoprotein (HDL) and serum zinc levels. A moderate positive correlation was observed between serum zinc and HDL (r = 0.616). The fitted regression model for serum HDL was 0.1396x + 5.3315. The overall regression model was statistically significant (r^2 = 0.379, F = 35.375, p < 0.001), indicating that serum HDL is a significant predictor of serum zinc levels (β = 0.616, p < 0.001).

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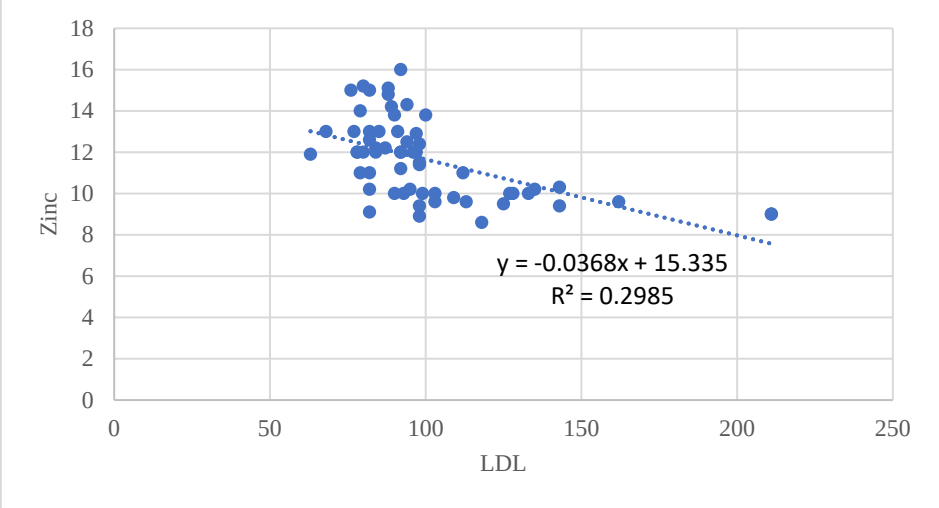

Figure – 5: Relationship between LDL and Serum Zinc Level

Figure 5 illustrates the results of a simple linear regression analysis examining the predictive relationship between serum low-density lipoprotein (LDL) and serum zinc levels. A moderate negative correlation was observed between serum zinc and LDL ($r = -0.546$). The fitted regression model for serum LDL was -0.0368x + 15.335. The overall regression model was statistically significant ($r^2 = 0.2985$, $F = 24.679$, $p < 0.001$), indicating that serum LDL is a significant predictor of serum zinc levels ($\beta = -0.546$, $p < 0.001$).

DISCUSSION

This comparative cross-sectional study was conducted in the Department of Physiology at Sylhet MAG Osmani Medical College, Sylhet, Bangladesh, from July 2022 to June 2023. A total of 60 individuals attending various Outpatient Departments of Sylhet MAG Osmani Medical College Hospital were enrolled. Based on WHO guidelines for the Asian population, participants were divided into two groups according to their BMI: Group A consisted of obese individuals with a BMI ≥ 25 kg/m^2 (n = 30), and Group B included healthy individuals with a BMI between 18.5 and 24.9 kg/m^2 (n = 30).

In the present study, the mean age of participants was slightly higher in the obese group (Group A: 39.73 ± 10.82 years) compared to healthy controls (Group B: 36.80 ± 6.42 years). Similar age distributions have been reported in earlier studies; for example, Knez et al. observed a mean age of 56 years among dyslipidemic adults, with values ranging from 39 to 72 years [22].

Serum zinc levels were markedly reduced in obese individuals compared with healthy participants, with Group A showing significantly lower zinc concentrations (10.15 ± 1.03 µmol/L) than Group B (13.13 ± 1.34 µmol/L, $p < 0.001$). Comparable findings were reported by Knez et al., who noted an average plasma zinc concentration of 0.75 ± 0.10 mg/L, with 26% of participants exhibiting zinc levels below 0.7 mg/L, indicating widespread zinc insufficiency in dyslipidemic populations [22].

A strong inverse relationship between BMI and serum zinc was also observed ($r = -0.797$), with the regression model demonstrating high statistical significance ($r^2 = 0.6347$, $F = 100.782$, $p < 0.001$). This suggests that increasing BMI is strongly predictive of declining zinc levels ($\beta = -0.797$, $p < 0.001$). These results align with previous studies reporting a negative association between zinc levels and anthropometric indicators such as BMI and waist circumference[13,23,24]. Rios-Lugo et al. also documented reduced serum zinc in overweight and obese Mexican adults[23], while Laillou et al. found zinc deficiency in 61.1% of overweight or obese Vietnamese women [25]. Similar reductions in zinc status among obese pediatric and adult populations have been reported across Egypt, Turkey, Italy, and the UK [8,9,26].

In this study, serum zinc showed moderate correlations with lipid parameters: negative correlations with total cholesterol (TC) ($r = -0.503$), triglycerides (TG) ($r = -0.591$), and LDL cholesterol ($r = -0.546$), and a moderate positive correlation with HDL cholesterol ($r = 0.616$). These findings indicate that dyslipidemic alterations are significantly associated with lower zinc levels.

However, the literature presents mixed evidence. Zhu et al. reported positive linear associations between serum zinc and TC, HDL-C, and LDL-C, along with a U-shaped relationship with triglycerides, suggesting that zinc–lipid interactions may vary across populations [27]. Similarly, a clinic-based US study involving 778 adults found that serum zinc positively correlated with TC, LDL-C, and TG [28]. An Iranian cross-sectional study also observed positive associations between serum zinc and TC and TG [20]. In contrast, a Korean population-based analysis reported decreased HDL-C with higher zinc levels, and elevated TG levels only among men [29]. Other studies found no significant associations between zinc status and lipid profiles [30,31].

Several studies support the trends observed in the present analysis. Zaky et al. reported significant negative correlations between serum zinc and both TG and LDL-C, along with a positive correlation with HDL-C [24]. Al-Sabaawy similarly identified significantly reduced zinc levels in hyperlipidemic adults and observed negative correlations with TC, LDL-C, and TG [32].

Zinc deficiency appears to be prevalent among individuals with dyslipidemia. According to Knez et al., 30% of dyslipidemic participants had low plasma zinc concentrations (with 50% below 0.75 mg/L), reflecting a considerable nutritional deficit[22,33]. Their findings are consistent with Obeid et al., who reported a high risk of zinc deficiency among Lebanese adults with metabolic syndrome[34]. Conversely, some studies have demonstrated direct correlations between zinc and HDL levels or other lipid ratios[35,36], further highlighting the inconsistency within existing literature.

Overall, the present study supports growing evidence of an inverse association between obesity, dyslipidemia, and serum

zinc status. However, inconsistencies across studies suggest that the relationship may be influenced by dietary patterns, ethnicity, metabolic status, and methodological variations.

Limitations of the study

This study has several limitations. The relatively small sample size and single-center design may limit the generalizability of the findings. Its cross-sectional nature prevents establishing causal relationships between serum zinc levels and dyslipidemia. Important confounding factors such as dietary intake, physical activity, and socioeconomic status were not assessed, which may have influenced both zinc status and lipid profiles. Additionally, serum zinc levels can be affected by acute phase responses or interactions with other micronutrients, which were not evaluated. These limitations should be considered when interpreting the results.

Conclusion and recommendations

The study findings demonstrate a significant association between serum zinc levels and lipid abnormalities among obese adults. Dyslipidemic individuals exhibited markedly lower serum zinc levels compared to their non-dyslipidemic counterparts. Furthermore, all major lipid parameters (TC, TG, HDL-C, and LDL-C) showed significant predictive relationships with serum zinc, suggesting its potential value as a biomarker of dyslipidemia. The strong negative correlations with TC, TG, and LDL-C, and the positive correlation with HDL-C, highlight the possible role of zinc in lipid metabolism.

Further multicenter and longitudinal studies with large sample sizes are needed to confirm the predictive utility of serum zinc and to explore its role in targeted interventions for dyslipidemia.

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Ethical approval: This study was approved by Ethical Committee of Sylhet MAG Osmani Medical College.

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