# Impact of body mass index stratification on immunological and biochemical profiles in adult males: A comparative study

TABARAK J. TALAB[1], MARYAM M. BAKHEET[1], HIBA Q. MAHMOUD[1], YUSUR F. FARAJ[2] and MARYAM Q. MOHAMMED[1]

[1]Department of Biology, College of Science, Mustansiriyah University, Baghdad 10061, Iraq; [2]National Center of Hematology, Mustansiriyah University, Baghdad 10015, Iraq

Received July 29, 2025; Accepted October 27, 2025

DOI: 10.3892/wasj.2025.415

**Abstract.** Body mass index (BMI) is a standard monitoring tool used to assess the health of an individual. The present study aimed to examine the effects of BMI values on the levels of blood parameters, lipid profiles, leptin and TNF-α levels in 120 adult male participants with varying BMI categories. The age of the study subjects ranged between 30-60 years. The results revealed that the level of hemoglobin and the number red blood cells were significantly decreased in the obese and underweight groups, while the number of the white blood cells was significantly increased (P<0.003) in the obese compared to the normal BMI group. Moreover, the platelet count was significantly increased (P<0.05) in the obese II and obese III groups. Furthermore, as regards the lipid profiles and according to the BMI categories, the cholesterol levels, triglycerides and low-density lipoprotein level were increased in the obese I, II and III groups compared with the normal BMI group. However, high-density lipoprotein levels (mg/dl) were decreased compared to the normal values in the obese I, II and III groups. Moreover, leptin hormone levels were highly significantly increased (P<0.003) in the obese II and obese III groups in comparison with the other BMI category groups. In addition, the TNF-α levels was significantly increased (P<0.025) in the obese I, II and obese III groups. On the whole, the present study highlights the need for evaluating the levels of these parameters. This can help to identify individuals who are at risk at an early stage and guide focused medical interventions.

*Correspondence to:* Dr Tabarak J. Talab, Department of Biology, College of Science, Mustansiriyah University, Highway 6, Baghdad 10061, Iraq
E-mail: tabarak.jalil96@uomustansiriyah.edu.iq

*Key words:* body mass index, obesity, underweight, immunological, biochemical

## Introduction

According to the World Health Organization (WHO), body mass index (BMI) is the most effective marker for measuring overweight and obesity in the population; BMI is determined in the same manner in both sexes and adults of all ages (1). A BMI of 25-29.9 kg/m$^2$ is classified as overweight, while and a BMI >30 kg/m$^2$ is classified as obese in adults, regardless of sex or age to predict body fat percentage in the population (2).

Obesity is defined as the abnormal or excessive accumulation of fat that can affect the health of an individual (3). It is caused by a disruption in the homeostatic control of food intake, which results in increased energy intake in relation to the metabolic demands of the body and consequently, increased energy intake in relation to energy expenditure, and hence weight gain (4). Previous epidemiological studies have demonstrated that body weight is related to a risk of developing infections and disease (5-7). However, in children and adolescents, being underweight increases the chances of developing a variety of infections. It has been shown that being both obese or underweight is associated with an increased U-shaped risk of acquiring infections in adults, suggesting that a normal weight is associated with the lowest risk of acquiring infections in the majority of participants (7).

In previous research, there are variations in population and environmental variables, such as diet and lifestyle conditions (8). A previous study demonstrated that the underweight population had a 19.7% higher risk of developing cardiovascular diseases (CVDs) compared with the normal weight population, whereas the overweight and obese populations had a 50% and a 96% elevated risk, respectively (9).

Concentrations of cholesterol and serum triglycerides (TGs) are closely related to obesity (10). A higher BMI has been conclusively linked to higher levels of total cholesterol, low-density lipoprotein cholesterol (LDL) and TGs, and is inversely associated with high-density lipoprotein cholesterol (HDL), according to epidemiological and clinical research. It has been suggested that the association between BMI and lipoprotein levels, particularly LDL, plays a role in the development of CVDs linked to obesity (11,12).

Human fat cells are a major source of endogenous TNF-α production. TNF-α is a cytokine released mainly by macrophages in response to inflammation, endotoxemia and

cancer (13). Research has consistently demonstrated that serum TNF-α levels are markedly elevated in overweight and obese individuals compared to those with a normal BMI. A positive association exists between the TNF-α concentration and BMI, waist-to-hip ratio and other anthropometric indicators of obesity (14,15).

The aim of the present study was to determine whether any change in the normal value of BMI has an effect on biochemical and immunological parameters in adult males. This was analyzed by measuring the complete blood count, lipid profiles, leptin levels and TNF-α levels. In order to evaluate and improve therapies for obesity-related diseases, the present study emphasizes that it is crucial to evaluate these markers and their association with BMI. The present study also provides key insight that may aid in the development of more focused and efficient therapeutic approaches.

## Patients and methods

*Patient selection*. The present study was carried out on 120 Iraqi male participants of various BMI categories (16 to >43 kg/m$^2$) with an age range of 30-60 years and a median age of 42 years, who attended the obesity medical center at Al-Kindy Teaching Hospital and the Nutrition Research Institute (Baghdad, Iraq) during the period extended between June, 2024 to May, 2025. These sites were selected as they are major centers for handling the target samples. The BMI of the volunteers was calculated by dividing the weight by square height; the participants were then divided into six groups according to the BMI value. The study participants were categorized as 'apparently healthy' based on a comprehensive clinical screening process. This included: i) A detailed medical questionnaire to exclude individuals with self-reported chronic diseases; ii) the measurement of vital signs (Blood Pressure); and iii) routine blood tests (including fasting blood glucose and a lipid profile) to objectively exclude individuals with subclinical metabolic abnormalities. Only individuals with all screening results within normal clinical ranges were included. The exclusion criteria included individuals with major metabolic diseases, chronic or acute infections, those who recently underwent major surgery, and the use of medications that could alter body composition or metabolic profiles. All volunteers gave their informed consent for the use of their clinical data for scientific purposes. The Research Ethics Committee of Mustansiriyah University (Baghdad, Iraq) reviewed and approved the present study (approval no. BCSMU/162 4/00070Z). Ethics approval was obtained from the authors' institution as the governing document. This approval was presented to the administrations of Al-Kindy Teaching Hospital and the Nutrition Research Institute. As these are affiliated teaching hospitals with established inter-institutional collaboration protocols, they granted administrative site permission for sample collection based on the university's central ethics clearance.

*Blood samples*. A total of 5 ml peripheral venous blood was collected from each of the 120 male participants with varying BMI categories using a sterile syringe. A total of 2 ml blood samples were transferred into anticoagulated tubes containing ethylene diamine tetra acetic acid (EDTA) and used immediately to determine the complete blood count (CBC) using a Sysmex XP-300 automated hematology analyzer (Sysmex Corporation). The remaining 3 ml blood samples were transferred into a Gel Activator tube and left for 15 min, followed by centrifugation at 1,006 x g at 4˚C for 10 min to separate serum. The serum obtained from each individual was dispended into several sterilized Eppendorf tubes and stored at -20˚C until use for the analysis of various parameters using the Reader automated ELISA system and an automated microplate reader at 450 nm (Human GmbH) according to the manufacturer's instructions. Each sample was designated by a serial number and the name of the individual.

*Statistical analysis*. Data are presented as the mean ± standard error (SE). All analyses were performed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp). The normality of data distribution was assessed using the Shapiro-Wilk test. For normally distributed data, differences between the six BMI groups were analyzed using one-way analysis of variance (ANOVA), followed by the Tukey's honestly significant difference (HSD) test post hoc test for multiple comparisons to identify significant differences among group means. A P-value <0.05 was considered to indicate a statistically significant difference.

## Results

The distribution of the 120 male participants in the present study according to BMI categorization is demonstrated in Table I.

The level of hemoglobin (g/dl) was significantly increased in the overweight, obese I and obese II groups; however, the lowest value was observed in the underweight and obese III group. The values for red blood cells (RBCs, $10^6/\mu$l) were decreased in the underweight and obese (class I-III) groups; these values differed significantly from those of the normal weight group (Fig. 1).

The values for white blood cells (WBCs, $10^3/\mu$l) were significantly increased (P<0.003) in the obese II and obese III groups in comparison to the WBC counts in the underweight and normal weight groups (Fig. 2A). Lymphocyte counts exhibited highly significant differences across all study groups (P=0.029). Lymphocyte counts were significantly decreased in the underweight group (1516.49±4.2) in comparison with the obese II and obese III groups (2437.11±4.1 and 3068.58±3.3, respectively). In the normal, overweight and obese I groups, the lymphocyte counts were 1649.80±2.1, 2017.13±2.1 and 2097.31±3.1, respectively (Fig. 2B). However, for the monocyte and granulocyte levels, the post hoc analysis indicated no significant differences between any of the study groups (P>0.05) (Fig. 2C and D). Furthermore, the platelet count was significantly increased (P<0.05) in the obese II and obese III groups (274±9.15 and 278±8.12, respectively) in comparison with the underweight group (198±11.13) (Fig. 2E).

The results of the analyses of lipid profiles are presented in Fig. 3. The cholesterol level (mg/dl) was significantly increased (P<0.003) in the obese (classes I-III) groups in comparison to the underweight and normal weight groups. The levels of TGs (mg/dl) exhibited a highly significant (P<0.0031) increase in the

Table I. Distribution of the study participants according to BMI categories.

| BMI groups | BMI (kg/m$^2$) | No. of participants | Range of BMI sample (kg/m$^2$) |
|---|---|---|---|
| Underweight | <18.5 | 20 | 16-18.2 |
| Normal | 18.5-24.9 | 22 | 18.8-24.5 |
| Overweight | 25-29.9 | 21 | 25.1-29.2 |
| Obese I | 30-34.9 | 19 | 30.2-34.2 |
| Obese II | 35-39.9 | 20 | 35.3-38.5 |
| Obese III | ≥40 | 18 | 40.3-42.9 |

BMI, body mass index.

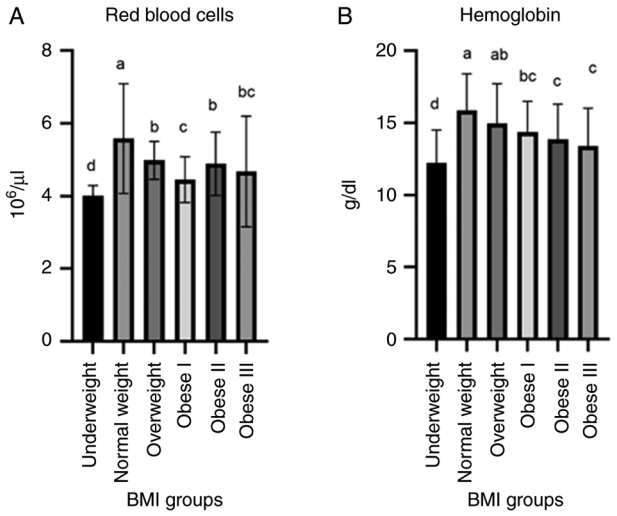


Figure 1. Levels of (A) red blood cell counts and (B) hemoglobin according to the BMI categories of the study participants. In the graphs, bars marked by different lowercase letters (a, b, c, d) indicate statistically significant differences (P<0.05). The different BMI categories are explained in Table I. BMI, body mass index.

obese III group (152.55±5.65) and obese II group (145.50±4.65) in comparison to the underweight group (81.20±2.48) and normal group (113.30±6.87). The LDL levels (mg/dl) also exhibited a significant (P<0.04) increase in the obese III group (159.24±6.55) and obese II group (153.90±5.70) in comparison to the underweight group (82.54±3.24), normal weight group (93.18±6.62) and overweight group (129.45±5.45). The levels of HDL (mg/dl) were decreased in the obese III (30.45±1.85), obese II (32.50±1.85) and obese I (36.85±1.95) groups in comparison to the underweight (41.42±1.85) and normal weight (53.56±2.23) groups.

Leptin hormone levels (ng/ml) exhibited a highly significant (P<0.003) increase in the obese III group (36.6±2.41) in comparison to the underweight and normal BMI groups (3.6±0.67, 6.2±1.12 respectively). In addition, the obese II (23.4±1.92) group exhibited a high level of leptin followed by the obese I (18.5±1.41) and overweight groups (Fig. 4).

As demonstrated in Fig. 5, the TNF-α levels were significantly increased in the obese I, obese II and obese III groups compared to other groups of study participants. The mean level of this cytokine was 48.43±3.34, 53.14±1.91 and 59.69±3.83 pg/ml in the obese I, obese II and obese III groups, respectively, compared to the other groups.

## Discussion

The present study revealed significant associations between BMI and the examined immunological and biochemical variables of the study participants, highlighting the importance of monitoring these markers in high-risk BMI groups to prevent potential metabolic and immune-related complications. From these results, it was noted that the underweight group had the lowest mean hemoglobin concentration; however, this was increased in the overweight, obese I and obese II groups, with low values also observed in the obese III group. Hemoglobin is an iron-rich protein in the blood that imparts a red hue. It constitutes 95% of an RBC, fulfilling the average adults requirement of 250 oxygen each minute (16). In the condition of anemia, there is a lack of hemoglobin, and this deficiency reduces the number of RBCs carrying oxygen, which prevents the fat burning process in an obese state (17). Therefore, an increased in the amount of body fat may be interpreted as a sign of a decreased hemoglobin level (18). Moreover, compared to individuals with a normal weight, those who are overweight or obese are at a higher risk of developing anemia (19). In the present study, the lowest values of hemoglobin were found in the obese II and obese III groups (Fig. 1). This result is in accordance with the findings reported in the study by Alshwaiyat *et al* (20), who demonstrated that an increased in BMI had an adverse effect on the iron status. The etiology of iron deficiency anemia in obese individuals may stem from an inadequate iron intake due to an unbalanced diet. Additionally, other hypothesized factors contributing to iron deficiency in individuals with an elevated BMI include diminished iron absorption in the small intestine, heightened iron demands resulting from an increased blood volume, and the association of obesity with a chronic low-grade inflammatory state. Moreover, Alshwaiyat *et al* (21) further supported the hypothesis that hemoglobin levels and a high BMI are negatively associated. As obese individuals may consume a higher amount of fats or carbohydrates in their diet rather than nutrient-dense foods high in vitamins and minerals, their hemoglobin levels decrease (i.e., more macronutrients and less micronutrients) (22). In addition, the association of obesity with a chronic low inflammatory condition may therefore be one of the proposed causes of iron deficiency in the obesity

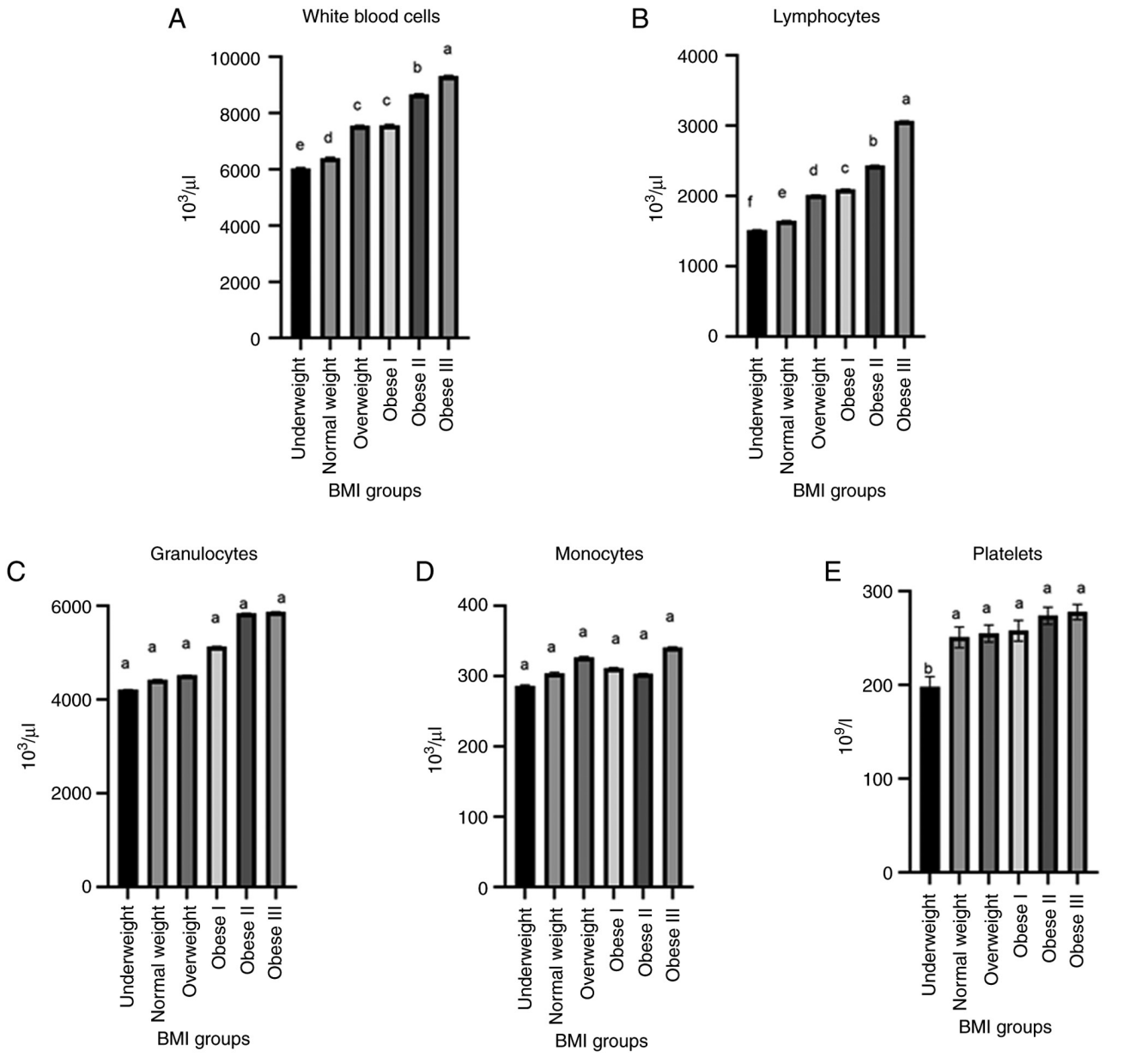


Figure 2. Comparison between (A) white blood cells, (B) lymphocytes, (C) granulocytes, (D) monocyte, and (E) platelets according to the BMI categories of the study participants. In the graphs, bars marked by different lowercase letters (a, b, c, d, e, f) indicate statistically significant differences ($P<0.05$). The different BMI categories are explained in Table I. BMI, body mass index.

state through inflammatory-mediated mechanisms (23). Furthermore, Nasif *et al* (24) demonstrated that obesity is an excessive accumulation of energy as adipose tissue, which adversely affects health.

The decrease in the level of hemoglobin in the present study observed in both the underweight and obese III groups may be related to the low level of RBCs, as the RBCs are comprised of hemoglobin, which is a metal protein that contains heme groups whose atoms are temporarily bounds to oxygen molecules in the lungs. A reduction in the hemoglobin content in RBBs results in a diminished oxygen-carrying ability of the blood, leading to insufficient cardiac output. Consequently, individuals with anemia experience dyspnea, palpitations and angina-like symptoms following intensive activity (25). Moreover, iron deficiency anemia is prevalent among urban populations due to inadequate dietary habits and diminished physical activity. Conversely, nutritional anemia is observed in medical students who possess superior nutritional understanding and reside in healthier environments (26).

Furthermore, in present study, it was found that the levels of WBCs were increased in the obese groups compared to the normal group (Fig. 2). These results are in accordance with those reported by Li *et al* (27) who found strong evidence of an association between obesity and WBCs counts. Obesity is a chronic inflammatory condition, and an elevated WBC count has widely recognized associations with inflammatory conditions (27). There is mounting evidence to indicate that obesity is a chronic inflammatory disease, and that the inflammatory features of the condition may be connected to certain comorbidities and risks associated with being overweight (28). There is also evidence to indicate that weight loss may cause the WBC count to decrease (29).

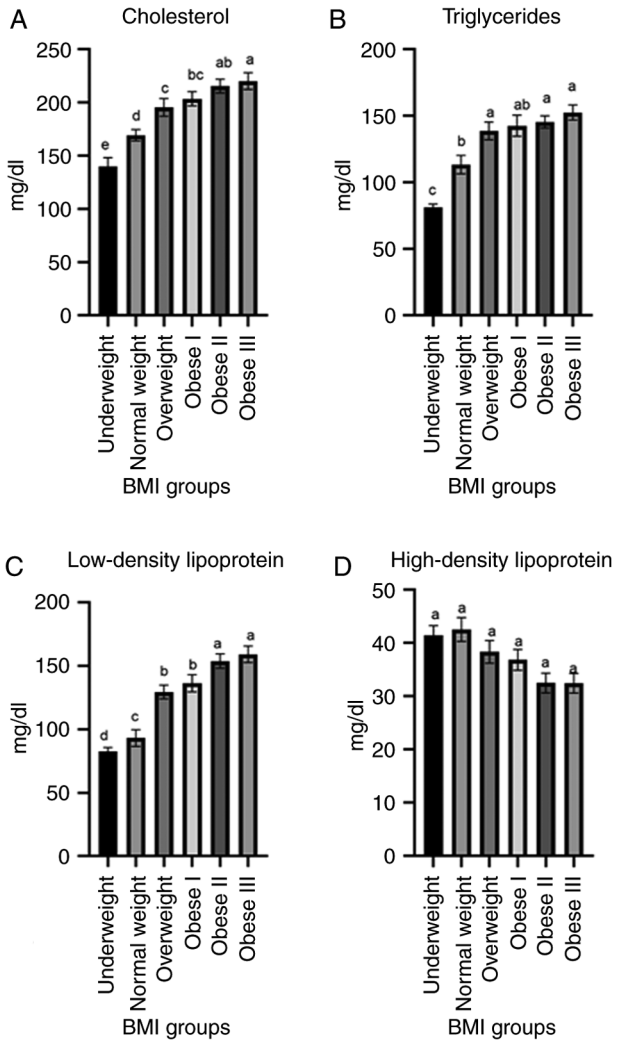


Figure 3. Lipid profile (mg/dl) levels according to according to the BMI categories of the study participants. (A) Cholesterol, (B) triglycerides, (C) low-density lipoprotein, and (D) high-density lipoprotein. In the graphs, bars marked by different lowercase letters (a, b, c, d) indicate statistically significant differences (P<0.05). The different BMI categories are explained in Table I. BMI, body mass index.

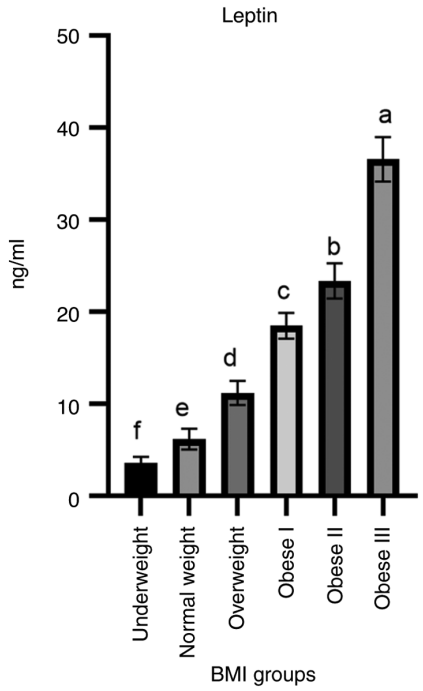


Figure 4. Levels of Leptin according to according to the BMI categories of the study participants. In the graphs, bars marked by different lowercase letters (a, b, c, d, e, f) indicate statistically significant differences (P<0.05). The different BMI categories are explained in Table I. BMI, body mass index.

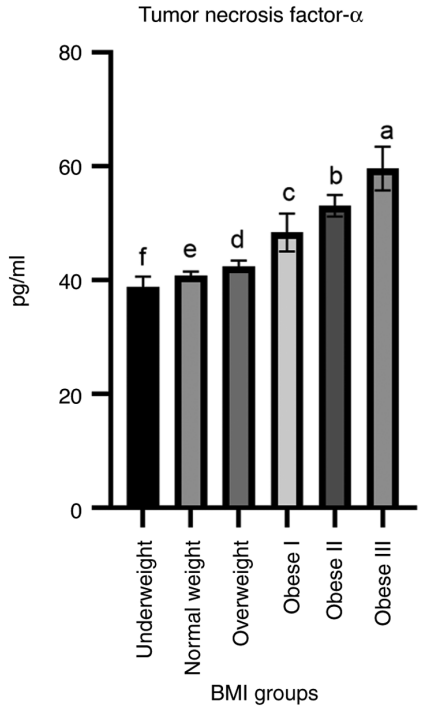


Figure 5. Measurement of tumor necrosis factor-α levels according to the BMI categories of the study participants. In the graphs, bars marked by different lowercase letters (a, b, c, d, e, f) indicate statistically significant differences (P<0.05). The different BMI categories are explained in Table I. BMI, body mass index.

Indeed, a study conducted on an Indian population indicated an association among body fat mass, leptin and WBC counts; however, the association between total WBC counts and leptin levels was more pronounced than that with fat mass (30). Naturally, there is a substantial association between elevated levels of pro-inflammatory substances and obesity, particularly central obesity. Numerous bioactive proteins, or adipokines, including cytokines that encourage inflammation such as TNF-α, are secreted by adipocytes (31). Furthermore, baseline WBC, neutrophils and lymphocyte counts increase with an increasing BMI and decrease with age (32). In addition, as granulocytes and monocytes may release substances into the bloodstream, including free radicals and proteolytic enzymes that can be detrimental to the health of an individual, the increase in leukocyte counts associated with obesity is clinically significant (33).

In the present study, lymphocyte counts were significantly lower in the underweight group (Fig. 2B). Lymphocyte counts are used as indicators of nutritional status or potential malnutrition (34). When BMI decreases, the lymphocyte counts also decrease; as demonstrated in a previous study among young Japanese women, the mean lymphocyte count

was higher in the obese group and lower in the underweight group than in the normal weight group (35). Another study by Rumińska *et al* (36), demonstrated that compared to individuals whose body fat was distributed naturally, obese individuals had a higher levels of inflammatory markers, including a 17% higher WBC count.

The results of the present study demonstrated that although the number of platelets was increased in the obese II and obese III groups, there were no significant differences between the obese II and obese III groups as regards the number of platelets compared to the normal, overweight and obese I group. However, the lowest value of platelets was observed in the -underweight group (Fig. 2E). A previous study by Anık *et al* (37), demonstrated an association between obesity and higher platelet counts in patients used as an inflammatory factor. However, the majority of diseases may be prevented by resolving behavioral risk factors by national campaigns, such as tobacco use, an unhealthy diet, obesity, the lack of physical exercise and alcohol intake. In fact, the continuous exposure of obese individuals to a pro-inflammatory and pro-thrombotic condition, in which platelets play a crucial role, has been linked to CVDs on numerous occasions (38). Nevertheless, the exact mechanisms via which obesity results in platelet dysfunction are not yet fully understood (39).

In the present study, an analysis was carried out to compare lipid profiles, including cholesterol, TGs, HDL and LDL levels among the participants belonging to the different BMI categories. The results revealed the existence of significant variations between the different groups of BMI categories (Fig. 3). The term dyslipidemia refers to altered lipid profiles. The increase in the cholesterol, TC and LDL levels in the overweight and obese groups in the present study was in agreement with the findings of a previous study (40), in which obesity was linked to numerous detrimental alterations in lipid metabolism, characterized by elevated serum levels of total cholesterol, LDL and TGs, alongside a decrease in the serum HDL concentration.

The elevation of TGs in lipid particles alters their metabolism; TG-rich HDL particles are hydrolyzed more swiftly, resulting in a decrease in HDL levels (41). Moreover, Turki *et al* (42) revealed that individuals who were obese had lower serum levels of HDL than subjects who were not, while subjects who were overweight had greater levels of total cholesterol and LDL. In addition, Shamai *et al* (43) demonstrated that an elevated BMI exhibited an inverse correlation with HDL and a direct correlation with TGs. In the previous study by Kawamoto *et al* (44), it was suggested that a high BMI status attenuated the associations of alcohol intake with lower LDL, cholesterol, a higher HDL cholesterol and a lower LDL/HDL ratio in Japanese males. Moreover, Raja *et al* (45) found an association of BMI with cholesterol and TGs in obese and non-obese subjects; they found a significant association between cholesterol and TGs with BMI. Cholesterol and TGs levels were strongly associated with obesity; therefore, obesity may be considered a risk for the development of hypertension, CVDs (45). Weight gain is caused by the consumption of junk food, poor nutrition, a sedentary lifestyle and the lack of physical activity. Hundreds of individuals suffer from coronary heart disease, hypertension and diabetes as a result of elevated lipid and blood sugar levels, which are caused by obesity. Insulin resistance may be the cause of the association between BMI, and both HDL and TGs; however, in the present study, the lack of a significant association between BMI and LDL is an unexpected finding that warrants for further research.

The present study demonstrated a higher level of leptin in the obese II and obese III groups (Fig. 4); this finding was in agreement with that observed in the study by Obradovic *et al* (10), who demonstrated that the serum leptin levels significantly increased with BMI, particularly with a BMI ≥30 $kg/m^2$, which aligns with obesity categories II and III. Obese patients have excessive levels of leptin; while leptin is known as the satiety hormone, and its levels are supposedly low in obese patients, leptin stimulates the release of gonadotropin releasing hormone; in obesity, excess levels of leptin cause a resistance later in on life (46). Moreover, Alaamri *et al* (47) examined the serum leptin levels of young Saudi students and demonstrated a substantial and independent association between elevated serum leptin levels and BMI. The higher prevalence of infertility in obese males would undoubtedly be caused by the large amounts of leptin released by the adipose tissue and its higher circulating levels (48).

Serum leptin concentrations are significantly associated with body fat percentage and weight. Obesity is linked to various lifestyle-related diseases and is frequently regarded as a contributing factor to male infertility. While adipocytes produce numerous other adipokines, previous research has indicated that leptin may serve as a crucial connection between obesity and obesity-related diseases (49). The finding of the present study are in agreement with those of the study by Kazmi *et al* (50), who demonstrated a positive association between BMI and leptin levels in obese subjects. Additionally, the structural and functional resemblance between leptin and its receptor with inflammatory cytokines suggests that leptin can be classified as a cytokine. Despite its significance in the normal immune response, leptin deficiency heightens vulnerability to infectious and inflammatory stimuli, potentially resulting in the dysregulation of cytokine production (51). Leptin also has immunological effects; its signaling can control innate inflammatory responses, adaptive immunity and even suppress regulatory T-cell differentiation (52). In addition to its metabolic and endocrine functions, leptin controls energy consumption and food intake by a direct effect on the hypothalamus (53). It may also modulate hematopoiesis, innate and adaptive immunological responses and inflammation, particularly in the presence of pro-inflammatory activities (54). Hyperleptinemia, characterized by an excess amount of leptin, is frequently associated with obesity and may greatly contribute to the development of severe health issues, including CVDs and rheumatoid arthritis (55).

The present study demonstrated that the serum levels of TNF-α were significantly increased in parallel with the increase in the BMI value (Fig. 5). Previous research has confirmed a positive association between the serum TNF-α concentration and BMI, particularly in overweight and obese patients. A previous study on an obese population revealed that increased BMI values were associated with increased serum TNF-α concentrations (56). Another clinical study (14), demonstrated an association between the amount of fat tissue and obesity and the increase in the TNF-α concentration in blood. TNF-α leads to brown adipocyte regression and insulin

resistance in adipocytes, which results in an aberrant energy consumption (57).

Furthermore, in a large comparative study on obese adults, the serum TNF-α levels were found to be considerably higher in overweight and obese individuals, and strongly correlated with BMI (r=0.65, P<0.001), alongside other metabolic parameters such as glucose and lipids (14). In addition, the plasma concentrations of TNF-α have been positively associated with elevated TG levels, which may be explained by its ability to induce the overproduction of very-low-density lipoprotein particles (58). The mechanistic basis lies in TNF-α being a proinflammatory cytokine secreted predominantly by macrophages in adipose tissue. It contributes to metabolic disturbances by promoting insulin resistance and altering lipid metabolism, which is reflected by high serum TNF-α levels in parallel with an increased BMI (59). Nonetheless, it is undeniable that optimal immune functions are inextricably related to nutritional status; this may be as a dietary fatty acid can affect cytokine formation (60). This has given rise to the alternative, but not exclusive notion that, at least in diet-induced obesity, dietary components, particularly fats, may be crucial in controlling the inflammatory profile of adipose tissue (61).

In conclusion, the present study demonstrates that deviations from normal BMI significantly affect hematological, metabolic and inflammatory pathways in men, underscoring the critical role of BMI assessment in developing targeted therapeutic strategies for obesity-related comorbidities. The present study performed a power analysis test which confirmed that the sample size (n=120/group) provided adequate statistical power (99.9%) to detect clinically meaningful effects for the primary endpoints, based on an effect size of 0.5 and α=0.05. It is important to emphasize that the present study wa exploratory in nature and did not intend to establish a diagnostic tool. The findings presented herein, should be regarded as a preliminary foundation for future, more comprehensive research. However, the present study has several limitations that should be mentioned. Firstly, as the present study was cross-sectional, it was impossible to establish a causal association between the variables that were observed. Second, the results are limited in their capacity to be applied to women as they are based solely on a cohort of adult men. Additionally, even when key variables were taken into account, the results may still have been affected by confounding variables that were not taken into account, such as dietary habits, specific amounts of physical activity and genetic predispositions. Despite these limitations, however, the present study provides new information about the association between adult male obesity, and indicators of immune function and systemic physiology. In order to validate these associations and clarify their underlying causative mechanisms, further longitudinal studies that involve a variety of populations and include more accurate assessments of adiposity and potential confounders are warranted.

## Acknowledgements

The authors would like to thank Dr Ali A. Mahdi, at the College of Health and Medical Technology, Middle Technical University in Baghdad, Iraq, for providing academic assistance and guidance.

## Funding

No funding was received.

## Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

## Authors' contributions

TJT and MMB contributed to the conception and design of the study, and wrote and edited the manuscript. HQM and YFF collected and analyzed data. MQM reviewed and edited the manuscript, and was also involved in data curation, and supervised the study All authors have read and approved the final manuscript. TJT and MMB confirm the authenticity of all the raw data.

## Ethics approval and consent to participate

The present study was reviewed and approved by the Research Ethics Committee of Mustansiriyah University (approval no. BCSMU/162 4/00070Z), and subsequently authorized by Obesity Medical Center at al-Kindy Teaching Hospital, and Nutrition Research Institute administration per standard institutional collaboration procedures. These sites were selected as they are major centers for handling the target samples. All patients who participated in the present study provided written informed consent for the publication of their data. The consent form emphasized that participation was entirely voluntary, and the participants could withdraw at any time without facing any repercussions. Anonymity and confidentiality were safeguarded by assigning coded identifiers rather than names or medical record numbers. In adherence to international ethical guidelines, including the Declaration of Helsinki, the study maintained strict ethical standards.

## Patient consent for publication

Not applicable.

## Competing interests

The authors declare that they have no competing interests.

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