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ORIGINAL RESEARCH

# Exploring the Relationship Between Inflammatory Biomarkers and Anthropometric Measures of Obesity in Healthy Adults: A Case Control Study

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**Background:** Obesity is a critical public health issue, marked by an accumulation of adipose tissue that presents numerous health hazards, with recent focus on the link between obesity and inflammation, especially in the context of inflammatory biomarkers as indicators of metabolic abnormalities related to obesity.

**Objective:** This study aimed to investigate the correlation between inflammatory biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and hematological indicators of inflammation with anthropometric obesity measures in obese and non-obese healthy adults.

**Methods:** The study employed a case control design involving both obese and non-obese healthy adults. The research evaluated anthropometric indicators. Blood samples were collected from participants to measure complete blood count (CBC), CRP and ESR. Platelet /Lymphocyte ratio (PLR), Neutrophil/Lymphocyte ratio (NLR), Monocyte/Lymphocyte ratio (MLR), and the systemic immune-inflammation index (SII) were calculated.

**Results:** A significant association exists between familial obesity, lack of physical activity, and constipation persistence in obese individuals compared to non-obese individuals. In the obese cohort, mean values of white blood cells (TWBCs), neutrophils, monocytes, platelet count, NLR, PLR, MLR, and SII were elevated compared to the non-obese group. Statistically significant differences in ESR and CRP were noted between the two populations. A positive correlation exists between TWBCs and WHR, whereas lymphocytes correlate positively with WHR and negatively with body fat percentage. Eosinophils exhibited negative associations with obesity metrics, while ESR and CRP had significant positive correlations with obesity indicators like BMI and body fat percentage; PLR showed a negative relationship with WHR and a positive one with body fat percentage, lacking significant correlations with BMI, WC, HC, or abdominal obesity.

**Conclusion:** The research indicates significant elevations in hematological biomarkers among obese individuals, highlighting a probable link between obesity and inflammation, with specific inflammatory markers potentially serving as predictors of obesity-related health risks.

**Keywords:** obesity, BMI, C-reactive protein, erythrocyte sedimentation rate, inflammatory markers, anthropometric measures, PLR

## Introduction

Obesity, a chronic disease marked by excessive white adipose tissue accumulation, is a complex condition with considerable health implications, currently affecting over 6 million Canadians and 22% of adolescents in the US.[1] Obesity is more prevalent in developed countries, but developing nations are also experiencing rising rates, often alongside malnutrition.[2] Since 1975, global obesity rates have surged nearly threefold, with 1.9 billion adults identified



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as overweight and 650 million as obese in 2016, and projections indicate that by 2025, 3 billion adults will be classified as overweight, with over 1 billion categorized as obese.[3,4]

Adipose tissue plays a vital role in energy homeostasis and interacts with the brain and other organs; the excessive fat accumulation in obesity markedly increases morbidity and mortality risks, constituting a growing health crisis that detrimentally influences both physical and mental health, with implications for global healthcare costs, while obesity is a notable risk factor for various cancers, contributing to 4–8% of cases, and is linked to cardiovascular diseases, type 2 diabetes, and hypertension, driven primarily by energy imbalances and socioeconomic barriers to nutritious food and physical activity.[5–7] The obesity induces an expansion of adipose tissue infiltrated by immune cells, predominantly macrophages, which secrete pro-inflammatory cytokines like TNF-α, IL-6, and MCP-1, thereby exacerbating systemic inflammation and correlating with increased CRP levels, as evidenced by studies on North Indian and Korean populations demonstrating higher CRP in obese individuals relative to non-obese counterparts.[8,9]

Systemic inflammation can be assessed using various biochemical markers from routine blood tests, offering a cost-effective alternative to traditional inflammation assessments. These markers facilitate the establishment of ratios for enhanced stability and predictability in measuring inflammation and immune status.[10]

NLR and PLR are established biomarkers for evaluating inflammatory processes in obesity, with research demonstrating notable correlations between these ratios and obesity, thereby suggesting their utility as indicators of subclinical inflammation in this population.[11,12]

Therefore, this study aimed to evaluate hematological inflammatory markers, CRP and ESR in both healthy individuals with and without obesity, alongside their correlation with potential risk factors.

# Materials and Methods

## Subjects and Methods

### Study Design and Settings

This case-control study, conducted from November 2022 to April 2023, involved sixty-nine healthy obese and sixty-nine non-obese individuals aged 19 to 45 years within the Department of Medical Microbiology and Immunology at Sana'a University, Yemen.

### Inclusion and Exclusion Criteria

Individuals eligible for inclusion in the study consist of those aged 19 years or older, non-smokers, and non-pregnant females. Conversely, individuals excluded from the study are those who have undergone bariatric or significant surgical procedures in the preceding three months, those classified as underweight, individuals currently experiencing any form of infection or inflammatory condition, as well as participants with endocrine, hepatic, renal, cardiac, or autoimmune pathologies, those administered weight-modulating pharmacological agents such as corticosteroids, and minors with genetic disorders or syndromes associated with obesity.

### Sample Size

Sample size was calculated using Open Epi, with a confidence level of 95%, a margin of error of 5%, 80% power, and a 1:1 ratio of unexposed to exposed, based on a 56.5% prevalence, yielding 69 participants for both obese and non-obese cohorts.[13]

### Socio-Demographic Characteristics

Socio-demographic and lifestyle information (eg, age, gender, educational attainment, etc.) was obtained through a self-administered questionnaire.

### Anthropometric Measurements

Anthropometric markers were quantified, including bodyweight, height, waist, hip, neck, and wrist circumferences using standardized methodologies. Body weight and height were assessed while participants wore light clothing and no footwear, followed by BMI calculation. Waist circumference was measured horizontally to the nearest millimeter at the midpoint between the costal margin and iliac crest during normal expiration, with the subject standing. Hip

circumference was determined at the level of the greater trochanters, with the legs positioned closely together. The WHR was computed as an indicator of central obesity by dividing waist circumference by hip circumference. Neck circumference was measured horizontally just below the larynx, ensuring perpendicular alignment to the neck's long axis. Wrist circumference was assessed bilaterally at the wrist crease distal to the styloid processes, and an average was derived. All circumferences were measured in a fasting state with subjects in an upright position and relaxed shoulders. The measuring tape was applied firmly against the skin without causing compression. Circumference measurements were recorded to the nearest 0.1 cm. The analysis utilized the average of the two measurements obtained.[14]

Participants were categorized into two distinct cohorts: individuals with a normal BMI (non-obese) and those with an abnormal BMI (obese). The classification of normal BMI is established as a BMI ranging from 18.5 to 29.9 kg/m$^2$; and the designation of obesity is attributed to a BMI of 30 kg/m$^2$ or greater.[15]

### Sample Collection

Venipuncture was executed to procure 10 mL of venous blood. Three milliliters were gathered in a lavender EDTA tube for CBC assessment utilizing the auto analyzer Diamond DF55 (Dymind, China), which was conducted within two hours post-collection. An additional three milliliters were collected in a lavender EDTA tube for ESR analysis. Furthermore, 2 mL was transferred into plain gel tubes for centrifugation, which was subsequently employed in the CRP assay.

### Hematological Inflammatory Markers

The NLR was derived by calculating the neutrophil count divided by the lymphocyte count, while the PLR was obtained by dividing the platelet count by the lymphocyte count. The MLR was determined by dividing the monocyte count by the lymphocyte count. The SII was defined as the product of the total count of neutrophils and platelets, divided by the lymphocyte count.[10,11]

### Qualitative and Semi-Quantitative CRP Assay

The CRP latex agglutination assay represents a qualitative and semi-quantitative diagnostic methodology. The latex particles employed in the CRP latex agglutination assay are functionalized with anti-human CRP antibodies that exhibit agglutination upon interaction with patient serum that contains CRP.[16]

## Statistical Analysis

Statistical analysis entailed the verification, coding, and data entry into SPSS Version 26 (IBM Corporation, New York, NY, USA). The statistical evaluation encompassed descriptive statistics, including frequency, percentage, mean, median, and standard deviation (SD), as well as components of inferential statistics. The Chi-square test and the Fisher exact test were implemented to evaluate the relationship between qualitative variables. The means were compared by *t*-test. Linear correlation was estimated using Pearson's correlation coefficient. The predetermined significance thresholds for the p-values were established at 0.05 and 0.01.

## Ethics Approval

This study was approved by the Research Ethics Committee of the Faculty of Medicine and Health Sciences at Sana'a University, Yemen, adhering to the principles of the Helsinki Declaration, whereby participants were fully informed, provided written informed consent.

# Results

## Socio-Demographic Characteristics

Table 1 illustrates the socio-demographic characteristics of the populations under examination. Among the 138 participants in this research, 69 were identified as obese. Of this cohort, 26.1% were male while 73.9% were female, with the predominant age bracket being 28–36 years, comprising 40.6% of the cases. Conversely, the control group consisted of 69 non-obese individuals. Within this group, 60.9% were male and 39.1% were female, with a significant majority (72.5%) falling within the age range of 19–27 years. Regarding residential locality, 69.6% of the obese participants and 60.9% of the non-obese participants resided in rural areas, while 30.4% and 39.1% lived in urban areas, respectively.

**Table 1** Socio-Demographic Characteristics Among Obese and Non-Obese Study Participants

| Character | Obese Cases No (%) | Non-Obese Cases No (%) | p |
|---|---|---|---|
| **Gender** | | | <0.001 |
| Male | 18 (26.1) | 42 (60.9) | |
| Female | 51(73.9) | 27 (39.1) | |
| **Age (years)** | | | <0.001 |
| 19-27 | 23 (33.3) | 50 (72.5) | |
| 28-36 | 28 (40.6) | 18 (26.1) | |
| 37-45 | 18 (26.1) | 1 (1.4) | |
| **Status** | | | <0.001 |
| Single | 22 (31.9) | 37 (53.6) | |
| Married | 47 (68.1) | 32 (46.4) | |
| **Area of Residence** | | | 0.284 |
| Rural | 48 (69.6) | 42 (60.9) | |
| Urban | 21 (30.4) | 27 (39.1) | |
| **Levels of education** | | | 0.566 |
| No formal | 2 (2.9) | 2 (2.9) | |
| Primary | 15 (21.7) | 13 (18.8) | |
| Secondary | 22 (31.9) | 16 (23.2) | |
| Graduated and post- graduated | 30 (43.5) | 38 (55.0) | |
| **Have a job** | | | 0.058 |
| Yes | 24 (34.8) | 35 (50.7) | |
| No | 45 (65.2) | 34 (49.3) | |
| **Type of job** | | | 0.854 |
| Full-time | 17 (24.6) | 18 (26.1) | |
| Part-time | 52 (75.4) | 51 (73.9) | |
| **Income** | | | 0.238 |
| Low | 15 (21.7) | 9 (13) | |
| Moderate | 52 (75.4) | 55 (79.7) | |
| High | 2 (2.9) | 5 (7.2) | |

**Note:** p is significant at value ≤ 0.05.

A statistically significant disparity was observed among the groups (obese and non-obese) concerning age, gender and marital status ($p<0.05$). Conversely, no statistically significant differences were noted between the groups regarding residential area, educational attainment, employment type, and income ($p>0.05$). Table 1

## Anthropometric and Body Composition Parameters of the Study Participants

In this study, the obese cohort exhibited significantly elevated mean values for all anthropometric measurements relative to the control group. These parameters encompassed weight, body fat, BMI, wrist, WC, HC, WHR, and abdominal obesity ($P < 0.001$). Table 2

## Comparison of Risk Factors in Obese and Non-Obese Individuals

A considerable correlation was identified between familial obesity, inadequate physical activity, and the persistence of constipation in individuals categorized as obese, in comparison to their non-obese participants ($p<0.05$). No significant association was found between dietary habits, meal frequency, and food allergies in obese individuals compared to non-obese individuals ($p>0.05$). Table 3

**Table 2** Comparative Analysis of Anthropometric and Body Composition Parameters Between Obese and Non-Obese Participants

| Anthropometry Parameters | Obese (n=69) Mean ±SD | Non-Obese (n = 69) Mean ±SD | Independence *T* Test | p |
|---|---|---|---|---|
| Weight (kg) | 86.03±12.36 | 57.03±7.92 | −16.14 | <0.001 |
| Height (mm) | 157.54±8.44 | 164.12±9.24 | 4.37 | <0.001 |
| Body fat % | 44.63±11.73 | 19.21±7.73 | −15.03 | <0.001 |
| BMI (kg\m$^2$) | 34.54±4.17 | 21.16±2.02 | −23.97 | <0.001 |
| Wrist (cm) | 16.06±1.24 | 14.94±1.34 | −5.09 | <0.001 |
| WC (cm) | 107.72±9.16 | 74.59±5.48 | −25.79 | <0.001 |
| HC (cm) | 116.70±8.32 | 90.33±6.87 | −20.14 | <0.001 |
| WHR (cm) | 0.92±0.069 | 0.83±0.044 | −9.99 | <0.001 |
| Abdominal Obesity (cm) | 107.25±9.20 | 77.28±7.52 | −20.95 | <0.001 |

**Notes:** p is significant at value ≤ 0.05.
**Abbreviations**: BMI, Body mass index; WC, Waist Circumference; HC, Hip Circumference; WHR, Waist hip ratio.

**Table 3** Comparative Analysis of Risk Factors in Obese versus Non-Obese Participants

| Risk Factor | Obese Cases No (%) | Non-Obese Cases No (%) | p |
|---|---|---|---|
| **Family history of obesity** | | | <0.001 |
| Yes | 49 (71) | 7 (10.1) | |
| No | 20 (29) | 62 (89.9) | |
| **Physical activity** | | | <0.001 |
| Low | 27 (39.1) | 5 (7.2) | |
| Moderate | 8 (11.6) | 16 (23.2) | |
| High | 34 (49.3) | 48 (69.6) | |
| **Food Habit** | | | 0.409 |
| Qat chewing | 22 (31.9) | 23 (33.3) | |
| Pepsi drinking | 7 (10.1) | 9 (13.0) | |
| Qat and Pepsi | 26 (37.7) | 30 (43.5) | |
| Nothing | 14 (20.3) | 7 (10.1) | |
| **No. of meals daily** | | | 0.120 |
| 1 | 1 (1.4) | 7 (10.1) | |
| 2 | 20 (29.0) | 15 (21.7) | |
| 3 | 48 (69.6) | 45(65.2) | |
| 4 | 0 (0.0) | 1 (1.4) | |
| 5 | 0 (0.0) | 1 (1.4) | |
| **Food allergy** | | | 0.060 |
| Yes | 19 (27.5) | 10 (14.5) | |
| No | 50 (72.5) | 59 (85.5) | |
| **Persistent of diarrhea or constipation** | | | <0.001 |
| Diarrhea | 8 (11.6) | 4 (5.8) | |
| Constipation | 40 (58.0) | 22 (31.9) | |
| Nothing | 21(30.4) | 43 (62.3) | |

**Notes:** p is significant at value ≤ 0.05.

## Comparison of Hematological Inflammatory Markers in Obese and Non-Obese Individuals

The mean values of TWBCs, neutrophils, monocytes, platelet count, NLR, PLR, MLR, and SII were elevated in the obese population compared to the non-obese cohort, yet no statistically significant differences were observed (p>0.05). There were statistically significant differences in ESR and CRP between obese and non-obese groups (p<0.05). Table 4

**Table 4** Comparative Analysis of Inflammatory Markers in Obese versus Non-Obese Participants

| Inflammatory Markers | Obese Cases N=69 Mean ±SD | Non-Obese Cases N=69 Mean ±SD | *p* |
|---|---|---|---|
| **Total WBCs ($10^9$/L)** | 5.72±1.65 | 5.54±1.58 | 0.512 |
| **Neutrophil (x$10^3$/μL)** | 3.04±1.14 | 2.99±1.10 | 0.774 |
| **Lymphocyte (x$10^3$/μL)** | 2.26±0.64 | 2.32±0.69 | 0.601 |
| **Monocyte (x$10^3$/μL)** | 0.18±0.36 | 0.12±0.31 | 0.303 |
| **Platelets count ($10^9$/l)** | 315.64±56.17 | 300.51±54.64 | 0.111 |
| **NLR** | 1.47±0.69 | 1.39± 0.61 | 0.491 |
| **PLR** | 151.56 ±50.67 | 142.3± 51.92 | 0.293 |
| **MLR** | 0.086± 0.19 | 0.061±0.17 | 0.428 |
| **SII** | 462.53±232.75 | 419.60±192.66 | 0.240 |
| **ESR (mm/hour)** | 14.0±10.08 | 6.65±5.64 | <0.001 |
| **CRP** | | | |
| Positive n (%) | 18 (26.1) | 2 (2.9) | |
| Negative n (%) | 51 (73.9) | 67 (97.1) | <0.001 |

**Notes:** p is significant at value ≤ 0.05.
**Abbreviations**: T WBCs, Total White Blood Cells; NLR, Neutrophil/Lymphocyte ratio; PLR, Platelets /Lymphocyte ratio; MLR, Monocyte/Lymphocyte ratio; SII, systemic immune-inflammation index; ESR, Erythrocyte Sedimentation Rate.

## Correlation Between Inflammatory Biomarkers and Anthropometric Metrics

A positive significant correlation was observed between TWBCs and WHR (r = 0.266, P = 0.002), whereas no correlations were found between TWBCs and other anthropometric parameters. A significant positive correlation was observed between lymphocytes and WHR (r = 0.249, P = 0.003), whereas a significant negative correlation was noted between lymphocytes and body fat (r = −0.197, P = 0.021), with no significant correlations found between lymphocytes and other anthropometric parameters. Table 5

The study found significant negative correlations between eosinophils and various obesity metrics, including BMI, body fat, HC, and abdominal obesity (r = −0.178, P = 0.037; r = −0.206, P = 0.015; r = −0.183, P = 0.031; r = −0.197, P = 0.020), respectively. Table 5

**Table 5** Correlation Between Inflammatory Biomarkers and Anthropometric Metrices

| Inflammatory Parameters | Pearson Correlation | BMI | WHR | % Body Fat | WC (cm) | HC (cm) | Abdominal Obesity (cm) |
|---|---|---|---|---|---|---|---|
| **TWBCs ($10^9$/L)** | *r* | 0.035 | 0.266 | −0.042 | 0.128 | 0.010 | 0.034 |
| | *p* | 0.685 | 0.002** | 0.627 | 0.135 | 0.910 | 0.694 |
| **Lymphocytes (x$10^3$/μL)** | *r* | −0.053 | 0.249 | −0.197 | 0.019 | −0.129 | −0.072 |
| | *p* | 0.537 | 0.003* | 0.021* | 0.827 | 0.132 | 0.398 |
| **Eosinophils (x$10^3$/μL)** | *r* | −0.178 | −0.034 | −0.206 | −0.146 | −0.183 | −0197 |
| | *p* | 0.037* | 0.691 | 0.015* | 0.088 | 0.031* | 0.020* |
| **ESR (mm/hour)** | *r* | 0.497 | 0.015 | 0.583 | 0.343 | 0.480 | 0.373 |
| | *p* | <0.001 ** | 0.862 | <0.001 ** | <0.001 ** | <0.001 ** | <0.001 ** |
| **CRP** | *r* | 0.445 | 0.166 | 0.450 | 0.342 | 0.368 | 0.360 |
| | *p* | <0.001 ** | 0.052 | <0.001 ** | <0.001 ** | <0.001 ** | <0.001 ** |
| **PLR** | *r* | 0.060 | −0.195 | 0.244 | −0.001 | 0.125 | 0.114 |
| | *p* | 0.485 | 0.022* | 0.004** | 0.995 | 0.146 | 0.148 |

**Notes**: *Correlation is significant at the 0.05 level. **Correlation is significant at the 0.01 level.
**Abbreviations**: T WBCs, Total White Blood Cells; PLR, Platelets /Lymphocyte ratio.

This study identified significant positive correlations between ESR and obesity metrics, such as BMI, body fat, WC, HC, and abdominal obesity. This research also revealed significant positive correlations between CRP and obesity indicators, including BMI, body fat, WC, HC, and abdominal obesity, as shown in Table 5.

PLR exhibited a significant negative correlation with WHR ($r = -0.195$, $P = 0.022$) and a positive correlation with body fat ($r = 0.244$, $P = 0.004$), whereas no significant correlations were observed with BMI, WC, HC, and abdominal obesity, as illustrated in Table 5.

No significant correlations were found between NLR, MLR, SII, neutrophils, monocytes, and platelet counts with anthropometric parameters. (Data not shown)

## Discussion

This research identifies a significant gender disparity in obesity among 69 participants, with 26.1% male and 73.9% female, predominantly within the 28–36 age range (40.6%). Such findings corroborate existing literature illustrating a higher prevalence of obesity in females, as demonstrated by studies indicating increased mean BMI and obesity rates among women compared to men.[17,18] Obesity is notably prevalent among adults aged 28–36, aligning with research indicating elevated obesity rates in middle-aged individuals, especially those below 65, while younger adults with obstructive sleep apnea exhibit a higher propensity for obesity compared to their older counterparts, further corroborated by findings from China highlighting significant obesity rates in adults aged 40–69 linked to specific lifestyle choices.[19,20] This demographic distribution underscores significant trends in obesity research, emphasizing the pivotal influence of gender and age on obesity prevalence.

The current study revealed no significant differences in residential area, educational attainment, employment type, or income between obese and non-obese individuals. In contrast to this study, previous studies have indicated varying health statuses linked to residential regions and a notable relationship between obesity and educational performance, as well as income disparities favoring non-obese individuals.[21,22] These discrepancies highlight the complexity of obesity's relationship with socioeconomic factors and suggest that further research is needed to understand these dynamics fully.

The present investigation establishes that individuals with obesity exhibit markedly elevated anthropometric indices, including BMI, WC, and HC, which correlate with heightened susceptibility to metabolic and cardiovascular pathologies. Recent literature confirms that such anthropometric factors, particularly BMI, WC and HC, are pivotal indicators of visceral fat accumulation, a notable risk determinant for metabolic disorders and cardiovascular health.[23–28] Although emphasis is frequently placed on elevated metrics in individuals with obesity, it is essential to acknowledge that anthropometric variables can significantly vary due to factors such as age, sex, and ethnicity, with females typically displaying greater body fat percentages than males, thereby influencing evaluations of obesity-related health risks.[25,26] The link between these measurements and health outcomes varies among populations, highlighting the need for customized obesity management strategies.

The current research identifies a notable link between familial obesity, low physical activity, and persistent constipation in obese individuals compared to non-obese individuals. A recent study found that familial obesity history adversely affects physical activity levels, with children from such backgrounds displaying reduced activity and heightened sedentary behavior; however, role models promoting physical activity can mitigate these negative outcomes and encourage healthier habits.[29] Physical activity levels in obese individuals, both children and adults, are markedly lower than in their non-obese counterparts, leading to increased sedentary lifestyles.[30] Environmental and psychological impediments, including lack of motivation and inadequate familial support, aggravate this inactivity. The significance of physical activity in reducing genetic obesity risk is particularly relevant for those with a familial background of obesity. Consequently, advocating for physical activity as a preventive measure against obesity requires focused interventions that tackle both individual and environmental obstacles.[31,32] Recent studies demonstrate that familial obesity is often linked to shared lifestyle choices, including dietary practices and physical activity, which may result in both obesity and constipation, with genetic predispositions further exacerbating these issues.[30,33] Additionally, a recent study suggests that physical inactivity is prevalent among obese individuals, leading to increased constipation rates, thus highlighting the necessity of addressing dietary habits, psychological stressors, and socioeconomic factors in comprehensive obesity prevention strategies.[34] Familial obesity and inadequate physical activity are closely linked to obesity; nonetheless, it is

essential to assess dietary patterns, psychological stressors, and socioeconomic factors, as these elements significantly influence obesity outcomes and should be integrated into comprehensive obesity prevention and intervention strategies.

The present investigation indicates that mean TWBC, neutrophil, monocyte, and platelet counts were elevated in the obese cohort compared to their non-obese counterparts, although these differences did not achieve statistical significance; this aligns with prior findings that associate obesity with heightened inflammatory markers such as leukocytes and neutrophils, as well as increased monocyte levels, thereby underscoring obesity's inflammatory pathology.[35,36] Higher platelet levels in obese cohorts may be associated with elevated inflammatory cytokines such as IL-6, though the lack of statistical significance in these variations likely results from individual variability and the overlap in hematological parameters between obese and non-obese subjects, with subclinical inflammation in obesity indicating that trends in inflammatory markers often lack significance; thus, individual differences, comorbidities, and biomarker sensitivity affect outcomes, highlighting the need for more accurate biomarkers for effective evaluation.[35–39]

The study indicates a significant positive association between TWBCs and WHR. The research also found a significant positive association between lymphocyte levels and WHR. Obesity, particularly central adiposity measured by WHR, is associated with low-grade systemic inflammation as indicated by elevated TWBC counts; however, research reveals inconsistent reliability of WHR as an inflammation marker across different populations.[40] Other recent studies identified no significant relationship between WHR and inflammatory markers such as NLR in obese women, challenging WHR's role as a consistent inflammation indicator), while additional findings suggest a negative yet non-significant correlation between WHR and TWBCs, highlighting methodological and population-based variability.[41,42] Lymphocytes are integral to numerous health conditions, including sepsis and rheumatoid arthritis, where they are associated with inflammation and disease progression.[43,44] While current literature identifies a link between TWBCs, lymphocytes, and WHR in obesity, it highlights the complex involvement of lymphocytes in obesity-related inflammation, warranting deeper exploration to clarify their relationship and health ramifications for effective intervention strategies.

This study delineated a significant negative correlation between eosinophil counts and several obesity metrics, such as BMI, body fat percentage, HC, and abdominal obesity, alongside notable positive correlations between ESR and diverse obesity indicators, including BMI, body fat, WC, HC, and abdominal obesity. A Study by Sunadome et al, 2020; indicates a notable inverse relationship between eosinophil counts and obesity metrics, with higher eosinophil levels correlating to lower BMI in certain populations.[45] Furthermore, eosinophils are implicated in metabolic homeostasis, as evidenced by their role in regulating inflammation and glucose metabolism, potentially alleviating obesity-related dysfunctions.[46] Higher BMI in certain populations correlates with elevated eosinophil levels, indicating potential influences from inflammation or genetic factors.[47] A comprehensive analysis of medical records revealed a significant association between BMI and ESR, especially in individuals with a BMI of 40 kg/m² or higher, indicating that elevated BMI correlates with increased systemic inflammation.[48] WC is a common measure of abdominal obesity and has been linked to various health outcomes. It is a significant predictor of metabolic disorders, which are often accompanied by inflammation, potentially affecting ESR levels.[49] Although HC is less commonly examined regarding inflammation, it is typically analyzed alongside WC to derive the WHR, a significant metric for assessing fat distribution linked to metabolic disorders that may affect inflammatory markers such as ESR.[50] Abdominal obesity is associated with heightened inflammatory markers that may elevate ESR.[51] The relationship between eosinophils and obesity metrics is modulated by environmental factors like diet and lifestyle, potentially impacting eosinophil functionality and adipose tissue distribution; thus, the documented link between ESR and specific obesity indicators, alongside the interplay of obesity, inflammation, and health outcomes, suggests that abdominal obesity might influence ESR through systemic inflammation, necessitating deeper exploration of this connection.

This study identified significant positive correlations between CRP levels and various obesity metrics, such as BMI, body fat, WC, HC, and abdominal obesity. A recent study indicates a robust positive association between BMI and CRP levels, with evidence showing that an increase in BMI correlates with rise in elevated CRP risk among adult women, and other research within a North Indian cohort demonstrated that individuals with a BMI exceeding 23 kg/m² exhibited significantly elevated CRP levels compared to those with lower BMI.[52,53] Body fat percentage positively correlates with CRP levels, especially in obese women, with a nonlinear relationship evident beyond 30% body fat. While WC correlates positively with CRP, this association is weaker than that of BMI, and although HC and WHR show some positive

correlations with CRP among obese populations, these relationships exhibit variability across different studies.[52–55] Despite these findings, some studies suggest that while CRP is a reliable marker of inflammation, other factors such as lifestyle and genetic predispositions may also play critical roles in the inflammatory response associated with obesity. This highlights the complexity of obesity-related inflammation beyond mere metrics.

In this study, the mean values of NLR, PLR, MLR, and SII were higher in obese individuals than in non-obese ones, although no statistically significant differences were noted. A comprehensive analysis indicated no significant disparity in NLR between obese and non-obese Egyptian subjects, despite elevated levels of CRP and PLR in the former, which reflect subclinical inflammation.[56] Similarly, in pediatric populations, NLR appeared non-discriminatory between obese and non-obese children, notwithstanding increased leukocyte counts in the former, suggesting its limited utility in gauging pediatric inflammatory status.[37] Conversely, in adults, NLR was elevated in those with metabolic syndrome, underscoring its potential relevance in assessing metabolic syndrome severity in individuals with obesity.[57] The obesity-inflammation nexus is intricate, influenced by adipokines and metabolic dyslipidemia, affecting PLR variably across demographics.[58] Variability in PLR significance between obese and non-obese cohorts may stem from differences in study design, demographics, and sample sizes.[59] The absence of significant differences in some studies may reflect the multifactorial nature of obesity-related inflammation, influenced by lifestyle and genetic factors.[60]

Research demonstrates that the MLR reveals no significant disparities between obese and non-obese groups.[34] As a relevant biomarker for inflammation, the MLR has been examined in various contexts, such as obesity and related conditions like cardiovascular diseases.[61] In obesity, the MLR is typically increased, yet studies have not consistently demonstrated statistically significant discrepancies between obese and non-obese individuals.[62]

The present study revealed that PLR demonstrates a notable negative relationship with WHR and a positive association with adiposity. A recent study identified the inverse correlation between PLR and WHR suggests that heightened inflammation, indicated by higher PLR, may correlate with a reduced WHR, potentially due to divergent fat distribution patterns or metabolic conditions that variably affect these indicators.[63] Other investigation observed that the correlation between PLR and adiposity indicates that rising body fat correlates with heightened systemic inflammation, corroborated by research connecting increased adiposity to elevated inflammatory markers.[64] Furthermore, WHR quantifies fat distribution, with elevated ratios signaling increased abdominal fat, a precursor to metabolic diseases.[65] The interplay between PLR, WHR, and adiposity elucidates the relationship between inflammation and body composition, yet it is essential to account for additional factors such as genetic predispositions, lifestyle choices, and comorbidities, alongside the influence of various inflammatory markers on metabolic health for a thorough comprehension of these interrelations.[66,67]

A comprehensive analysis of NHANES data from 2017–2020 revealed a significant correlation between SII and metrics such as BMI, WC, and obesity rates, while acknowledging the potential moderating effect of physical activity on this relationship.[68] Another study highlighted the crucial involvement of inflammation in obesity by analyzing the SII as a potential inflammatory marker, revealing a non-significant associative trend, while in pediatric populations, the SII showed a relationship with BMI, suggesting a correlation between inflammation and body weight in younger individuals, though with differing levels of significance.[40,69] This complexity is reflected in the diverse findings across different studies, emphasizing the importance of considering multiple factors when evaluating inflammatory markers in obesity.

## Limitations of the Study

The study design restricts our ability to investigate causal and temporal relationships among the variables, compounded by a limited sample size and the exclusion of inflammatory cytokines; thus, further research with larger cohorts incorporating inflammatory processes is warranted.

## Conclusions

The study indicates significant increases in hematological biomarkers among obese individuals, highlighting a potential link between obesity and inflammation. It identifies specific inflammatory markers such as TWBCs, Lymphocytes, Eosinophils and, PLR associated with various obesity parameters, suggesting that these markers could serve as predictors for health risks related to obesity.

## Data Sharing Statement

The datasets employed and examined in this study can be obtained from the corresponding author upon request.

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The authors have no conflicts of interest to disclose.

## References

1. Warnick J, Darling KE, Jelalian E, Goldschmidt AB. Overweight/Obesity. In: Troop-Gordon W, Neblett E, editors. *Encyclopedia of Adolescence*. New York: Academic Press; 2024:379–390. doi:10.1016/b978-0-323-96023-6.00054-3
2. Lafia AT, Ketounou TR, Honfoga JNB, Bonou SI, Zimé AKB. Dietary habits, prevalence of obesity and overweight in developed and developing countries. *Research Society Development*. 2022;11(10):e249111032769. doi:10.33448/rsd-v11i10.32769
3. Manasa HS, Mahadevaswamy RK. Obesity and its Effects on Health. *Saudi J Nurs Health Care*. 2023;6(1):16–17. doi:10.36348/sjnhc.2023.v06i01.004
4. Isaieva IM, Karmazina I. Excessive Weight Gain: the Problems and Solutions. *Ukraïns'kij Žurnal Medicini, Biologìï Ta Sportu*. 2022;7(5):28–34. doi:10.26693/jmbs07.05.028
5. Luo L, Liu M. Adipose tissue in control of metabolism. *J Endocrinol*. 2016;231(3):R77–R99. doi:10.1530/JOE-16-0211
6. Nagasri MT, Sanath P, Pasupuleti A, Akula S. Obesity and Lifestyle Changes: a Global Risk Factor for Cancer. *Obesity Diabet Res*. 2023. doi:10.47275/2692-0964-123
7. Singh Y. Consequences of obesity and Cardiovascular risk. *Int J Soc Sci*. 2023;12(1). doi:10.46852/2249-6637.01.2023.5
8. Săvulescu-Fiedler I, Mihalcea R. The Interplay between Obesity and Inflammation. *Reproduct Development Biol*. 2024;14(7):856. doi:10.3390/life14070856
9. Engin A. Reappraisal of Adipose Tissue Inflammation in Obesity. *Adv Exp Med Biol*. 2024;1460:297–327. doi:10.1007/978-3-031-63657-8_10
10. Menzel A, Samouda H, Dohet F, Loap S, Ellulu MS, Bohn T. Common and Novel Markers for Measuring Inflammation and Oxidative Stress Ex Vivo in Research and Clinical Practice-Which to Use Regarding Disease Outcomes? *Antioxidants*. 2021;10(3):414. doi:10.3390/antiox10030414
11. Thavaraputta S, Dennis JA, Ball S, Laoveeravat P, Nugent K. Relation of hematologic inflammatory markers and obesity in otherwise healthy participants in the National Health and Nutrition Examination Survey, 2011–2016. *Baylor Univ Med Center Proceedings*. 2021;34(1):17–21. doi:10.1080/08998280.2020.1799482
12. Radkhah H, Alirezaei A, Parhizkar P, et al. Insights into the clinical impact of complete blood cell inflammatory markers on body composition variations and fatty liver grading in Iranian adults undergoing bariatric surgery: a retrospective longitudinal study. *Ann Med Surg*. 2024;86(9):4990–4998. doi:10.1097/ms9.0000000000002257
13. Faam B, Zarkesh M, Daneshpour MS, Azizi F, Hedayati M. The association between inflammatory markers and obesity-related factors in Tehranian adults: tehran lipid and glucose study. *Iran J Basic Med Sci*. 2014;17(8):577–582. doi:10.22038/ijbms.2014.3189
14. Dave C, Agarwal N, Patel R, Shukla R, Bajpai A. Predictors of Metabolic Complications in Obese Indian Children and Adolescents. *Indian J Pediatr*. 2021;88(3):252–256. doi:10.1007/s12098-020-03515-5
15. Weir CB, Jan A. BMI Classification Percentile And Cut Off Points. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing;2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541070/. Accessed September 5, 2025.
16. Alhabbab RY. C-Reactive Protein (CRP) Latex Agglutination Test. In: *Basic Serological Testing. Techniques in Life Science and Biomedicine for the Non-Expert*. Cham: Springer; 2018. doi:10.1007/978-3-319-77694-1_9.
17. Djalalinia S, Yoosefi M, Shahin S, et al. The levels of BMI and patterns of obesity and overweight during the COVID-19 pandemic: experience from the Iran STEPs 2021 survey. *Front Endocrinol*. 2022;13. doi:10.3389/fendo.2022.1043894.
18. Ginting JB, Suci T. The Relationship between Age, Gender, and Physical Inactivity on the Incidence of Obesity at Puskesmas Johar Baru, Central Jakarta. *Kesmas Uwigama*. 2024;10(1):2597. doi:10.24903/kujkm.v10i1.2597
19. Esmaeili N, Gell L, Taranto-Montemurro L, et al. Prevalence of Obesity in Obstructive Sleep Apnea Within a Large Community-based Cohort of Middle-aged/Older Adults. *Sleep*. 2024;67:866. doi:10.1093/sleep/zsae067.0866
20. Feng X, Zhu J, Hua Z, Zhou J, Luo P. The prevalence and determinant of overweight and obesity among residents aged 40–69 years in high-risk regions for upper gastrointestinal cancer in southeast China. *Dental Sci Rep*. 2023;13(1):1. doi:10.1038/s41598-023-35477-x
21. Liu H. Socioeconomic Status and Health Disparities: a Comprehensive Study of Rural Communities in China. *Am J Health Behavior*. 2024. doi:10.5993/ajhb.48.3.9
22. Hammadi H, Alaslawi HA, Hewitt A, Reilly JJ. Differences in educational attainment between obese and non-obese Kuwaiti female university students. *J Nutr Sci*. 2020;9. doi:10.1017/JNS.2020.24.
23. Venkatesh D, Vinodha R, Shanmugapriya C. Study of Comparison of Anthropometric Parameters Among The Non-Obese, Overweight and Obese - subjects. *Int J Physiol*. 2023;11(1):3370. doi:10.37506/ijop.v11i1.3370
24. Saxena A, Jain A. To perform various anthropometric measurements in clinical cases of obesity. *Int J Med Biomed Stud*. 2020;4(2). doi:10.32553/IJMBS.V4I2.913
25. Stein MJ, Fischer B, Sedlmeier AM. Differences in Anthropometric Measures Based on Sex, Age, and Health Status: findings From the German National Cohort (NAKO). *Deutsches Arzteblatt International*. 2024. doi:10.3238/arztebl.m2024.0016
26. Bhatti R, Warshow U, Joumaa M, ElSaban M, Nawaz FA, Khamis AH. Relevance of Anthropometric Measurements in a Multiethnic Obesity Cohort: observational Study. *J Med Res*. 2021;10(2):27784. doi:10.2196/27784

27. de Medeiros PR, Del Duca GF, Streb AR, Heberle I, Leonel LDS, Gerage AM. Anthropometric indicators associated with blood pressure elevation in adults with obesity. *Acta Scientiarum Health Sci*. 2020;42:e50918. doi:10.4025/ACTASCIHEALTHSCI.V42I1.50918
28. Jang SY, Kim J, Kim S, Lee ES, Choi EJ. Impact of Anthropometric Indices of Obesity on the Risk of Incident Hypertension in Adults with Prehypertension: a Secondary Analysis of a Cohort Study. *J Korean Acad Nurs*. 2024;54(1):18–31. doi:10.4040/jkan.23067
29. Batish R, Oberoi S, Singh H, et al. Influences of role models and other factors on physical activity amongst 6 to 16 years overweight and obese students. *J Family Med Primary Care*. 2024;13(8):3238–3244. doi:10.4103/jfmpc.jfmpc_188_24
30. Sigmund E, Voráčová J, Dygrýn J, Vorlíček M, Sigmundová D. Comparative Analysis of 24-h Movement Behaviours in Non-Overweight and Overweight/Obese Children: findings from the FAMIly Physical Activity, Sedentary Behaviour, and Sleep (FAMIPASS). *Children*. 2024;11 (11):1298. doi:10.3390/children11111298
31. Chen J, Bai Y, Ni W. Reasons and promote strategies of physical activity constraints in obese/overweight children and adolescents. *Sports Med Health Sci*. 2023;6(1):25–36. doi:10.1016/j.smhs.2023.10.004
32. Specht IO, Heitmann BL, Larsen SC. Physical Activity and Subsequent Change in Body Weight, Composition and Shape: effect Modification by Familial Overweight. *Front Endocrinol*. 2022;13. doi:10.3389/fendo.2022.787827.
33. Sun X, Zhang S, Zhou X. A causal association between obesity and constipation: a two-sample bidirectional Mendelian randomization study and meta-analysis. *Front Nutrit*. 2024;11. doi:10.3389/fnut.2024.1430280.
34. Cui J, Yue H, Ma J, Han H, Fang M, Yao F. Physical activity and constipation: a systematic review of cohort studies. *J Global Health*. 2024;14. doi:10.7189/jogh.14.04197.
35. Takaya J, Tanabe Y, Nomura N, et al. Platelet and white blood cell counts correlate with leptin and body mass index in Japanese adolescents. *Clin Pediatr Endocrinol*. 2024;33(4):207–213. doi:10.1297/cpe.2024-0045
36. Yenigurbuz FD, Söbü E, Akbeyaz BB. The comparison of inflammatory hematological parameters in obese and non-obese children. *Family Pract Palliative Care*. 2023;8(1):24–29. doi:10.22391/fppc.1197997
37. Ningrum FH, Riwanto I, Sukmaningtyas H, et al. Differences Platelets Count, IL-6, Aldosterone Level and Fatty Liver Occurrence Between Obese and Non-Obese Adult Young Women. *Modern Medicine*. 2024;31(3):243–247. doi:10.31689/rmm.2024.31.3.243
38. Anık A, Çelik E, Anık A. The relation of complete blood count parameters with metabolic and clinical parameters in overweight and obese children. *J Pediatr Res*. 2021;8(2):161–170. doi:10.4274/jpr.galenos.2020.74824
39. Dushina TS, Суплотов СН, Klyashev SM, Суплотова ЛА. Features of changes in nonspecific factors of immunological reactivity in obesity. Медицинский Совет. 2024;16:257–264. doi:10.21518/ms2024-325
40. Liu F, Li Y, Li W, et al. The role of peripheral white blood cell counts in the association between central adiposity and glycemic status. *Nut Diabetes*. 2024;14(1). doi:10.1038/s41387-024-00271-9
41. Meher A, Mohapatra D, Behera M, Mishra T, Panda P. Association of neck circumference and waist-Hip ratio with total leukocyte count in healthy Indian adolescents. *J Basic Clin Physiol Pharmacol*. 2023;34(6):761–765. doi:10.1515/jbcpp-2023-0125
42. Aulia KR, Hendrianingtyas M, Limijadi EKS, Pramono D. Anthropometric Measurements and Inflammatory Marker in Obese Women. *Jurnal Gizi Indonesia*. 2022;10(2):88–94. doi:10.14710/jgi.10.2.88-94
43. Chen H, Xiong C, Shao X, et al. Lymphocyte To High-Density Lipoprotein Ratio As A New Indicator Of Inflammation And Metabolic Syndrome. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy*. 2019;12:2117–2123. doi:10.2147/DMSO.S219363
44. Lange A, Kostadinova L, Damjanovska S, et al. Red Cell Distribution Width and Absolute Lymphocyte Count Associate With Biomarkers of Inflammation and Subsequent Mortality in Rheumatoid Arthritis. *J Rheumatol*. 2022;50(2):166–174. doi:10.3899/jrheum.211411
45. Sunadome H, Izuhara Y, Nagasaki T, et al. Correlation between eosinophil count, its genetic background and body mass index: the Nagahama Study. *Allergol Int*. 2020;69(1):46–52. doi:10.1016/J.ALIT.2019.05.012
46. de Oliveira MC, Silveira ALM, de Oliveira ACC, et al. Eosinophils protect from metabolic alterations triggered by obesity. *Metabolism*. 2023;146:155613. doi:10.1016/j.metabol.2023.155613
47. Nishi K, Matsumoto H, Tashima N, et al. Impacts of lipid-related metabolites, adiposity, and genetic background on blood eosinophil counts: the Nagahama study. *Sci Rep*. 2021;11(1):15373. doi:10.1038/S41598-021-94835-9
48. Carroll MB. Impact of BMI on Erythrocyte Sedimentation Rate and C-Reactive Protein in Patients Without A Rheumatic Autoimmune Disorder: a Medical Record Analysis. *Br J Healthc Med Res*. 2023;10(3):291–302. doi:10.14738/bjhmr.103.14900
49. Dong J-Z, Ni Y-Q, Chu X, Zhao J, Yang Y, Yan Y. Association between the abdominal obesity anthropometric indicators and metabolic disorders in a Chinese population. *Public Health*. 2016;131:3–10. doi:10.1016/J.PUHE.2015.08.001
50. Singh N, Bairwa P, Bairwa P, Bairwa P, Bairwa P, Bairwa P. Waist circumference and waist-to-Hip ratio as indicators of abdominal obesity. *Int J Life Sci Res Archive*. 2022;2(2):102–105. doi:10.53771/ijlsra.2022.2.2.0055
51. Uutela T, Kautiainen H, Järvenpää S, Hakala M, Häkkinen A, Häkkinen A. Waist circumference based abdominal obesity may be helpful as a marker for unmet needs in patients with RA. *Scand J Rheumatol*. 2014;43(4):279–285. doi:10.3109/03009742.2013.858769
52. Li X, Yang H, Xiao J, et al. Association between different obesity measurement indexes and serum C-reactive protein in adult women. *Zhonghua liu xing bing xue za zhi = Zhonghua liuxingbingxue zazhi*. 2023;44(8):1251–1256. doi:10.3760/cma.j.cn112338-20221122-00992
53. Mraovic T, Radakovic S, Ristic-Medic D, et al. The relationship between adiposity parameters and C-reactive protein values in overweight and obese women. *Vojnosanit Pregl*. 2017;75(2):185–190. doi:10.2298/VSP160419208M
54. Shukla N, Singh S, Singh S, Shukla A. Association of High-sensitivity C-reactive Protein Level with Obese Normal individuals in North Indian Population. *Ann Afr Med*. 2024;24(1):45–50. doi:10.4103/aam.aam_140_23
55. Nagy M-A, El Shayeb MAR, Farres MN, Ahmed EE, Abdel Rahman SMM, Mahmoud MM. Study of Neutrophil-Lymphocyte Ratio (NLR), Platelet-Lymphocyte Ratio (PLR) and C Reactive Protein (CRP) as Systemic Biomarkers of Inflammation among Apparently Healthy Egyptian Obese Subjects. *QJM: Int J Med*. 2024;2024:2. doi:10.1093/qjmed/hcae175.251
56. Sartorio A, Sartorio A, Sartorio A, Sartorio A, Sartorio A. The neutrophil to lymphocyte ratio (NLR) positively correlates with the presence and severity of metabolic syndrome in obese adults, but not in obese children/adolescents. *BMC Endocr Disord*. 2023;23(1). doi:10.1186/s12902-023-01369-4
57. Rahimlou M, Mirzaei K, Keshavarz SA, Hossein-Nezhad A. Association of circulating adipokines with metabolic dyslipidemia in obese versus non-obese individuals. *Diabetes Metab Synd*. 2016;10(1):S60–S65. doi:10.1016/J.DSX.2015.09.015

58. Bukhari IA, Habib SS, Alnahedh A, et al. Relationship of Body Adiposity with Platelet Function in Obese and Non-obese Individuals. *Cureus*. 2020;12(1):6815. doi:10.7759/CUREUS.6815
59. Keye SA, Pindus DM, Walk AM, Burd NA, Holscher HD, Khan NA. Moderate-to-vigorous Physical Activity Is Related To Pre-motor Planning In Persons With Obesity. *Med Sci Sports Exercise*. 2022;54(9S):187–188. doi:10.1249/01.mss.0000877412.02387.20
60. Vakhshoori M, Nemati S, Shakarami M, et al. Prognostic impact of monocyte-to-lymphocyte ratio in coronary heart disease: a systematic review and meta-analysis. *J Int Med Res*. 2023;51(10):3000605231204469. doi:10.1177/03000605231204469
61. Soesilo N, Hendrianingtyas M, Rachma DE, Limijadi EKS. Monocyte to HDL Cholesterol Ratio (MHR) and Monocyte to Lymphocyte Ratio (MLR) in Overweight and Obese Women. *J Modern Pharmacy*. 2023;2023:1. doi:10.31689/rmm.2023.30.4.335
62. Maravi P, Kushwah SS, Shakya M, Rure D, Prajapati J, Rawat MSM. Relationship of PLR, NLR and LMR with Health Section Metabolic Syndrome in Schizophrenic Patients on Antipsychotics: a Longitudinal Case-control Study. *J Clin Diagn Res*. 2023. doi:10.7860/jcdr/2023/58357.17551
63. Moreira AD, Velasquez-Melendez G, Ladeira RM, da Silva Junior GB, de Fonseca MJ, Barreto SM. Association between Adiposity Indexes and Kidney Disease: findings from the Longitudinal Study of Adult Health (Elsa-Brazil). *J Am Coll Nutr*. 2021;40(1):1–7. doi:10.1080/07315724.2021.1878968
64. Arab-Zozani M, Joukar F, Hasanipour S, Abbasi Ranjbar Z, Naghipour M, Mansour-Ghanaei F. The association between waist-to-Hip ratio (WHR) with diabetes in the PERSIAN Guilan cohort study population. *BMC Endocr Disord*. 2024;24(1):1. doi:10.1186/s12902-024-01641-1
65. Artzberger B. *Environment-Wide and Epigenome-Wide Association Study of Adiposity in "Children of 1997" Birth Cohort*. 2022. doi:10.1101/2022.09.12.507623
66. Schell RC, Dow WH, Fernald LCH, Bradshaw PT, Rehkopf DH. Does educational attainment modify the causal relationship between adiposity and cardiovascular disease? *A Mendelian Randomization Study*. 2023;21:101351. doi:10.1016/j.ssmph.2023.101351
67. Yang S, Deng J, Zhang J, Jia E. Association of systemic immune-inflammation index with body mass index, waist circumference and prevalence of obesity in US adults. *Dental Sci Rep*. 2024;14(1):2. doi:10.1038/s41598-024-73659-3
68. Yu Y, Tan T, Yang W, Xu Z, Barua M. Association between the systemic immune-inflammation index and obesity among adults: insights from the NHANES 2017–2018. *PLoS One*. 2024;19(8):e0308288. doi:10.1371/journal.pone.0308288
69. Luo L, Wang X, Wang X, Wang X, Wang X. Association between systemic immune-inflammatory index and systemic inflammatory response index with body mass index in children and adolescents: a population-based study based on the National Health and Nutrition Examination Survey 2017-2020. *Front Endocrinol*. 2024;15. doi:10.3389/fendo.2024.1426404.