# Association of Cortisol and Adiponectin with Oxidative Stress Biomarkers in Obese Iraqi Adults

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Abstract: Obesity is a condition that directly impacts a variety of metabolic processes. The study investigated relationship between cortisol, adiponectin, and oxidative stress indicators in obese Iraqi adults. Between October 2025 and April 2026, a total of 88 Iraqi adults between the ages of 18 and 65 were recruited from Baqubah Teaching Hospital and specialist outpatient clinics in Baqubah City, Diyala Governorate. Participants were divided into three categories: healthy controls (n=28), overweight (n=30), and obese individuals (n=30). Venous blood samples were obtained under sterile circumstances, and cortisol, adiponectin (ADPN), catalase (CAT), and malondialdehyde (MDA) concentrations were determined using enzyme-linked immunosorbent assay (ELISA). The study observed significant anthropometric differences across groups. BMI increased sequentially among the three groups ($P < 0.001$). Additionally, cortisol levels were significantly higher in obese people than in overweight and healthy persons ($P < 0.001$). ADPN levels, on the other hand, gradually and dramatically decreased as body weight status increased ($P < 0.001$). Significant changes were also observed in oxidative stress markers, where CAT activity decreased and MDA levels increased in study groups from healthy to overweight and obese. According to ROC curve analysis, ADPN levels showed better diagnostic value in differentiating across study groups compared to cortisol levels, which only showed moderate diagnostic value. In conclusion, obesity is closely related to oxidative stress and hormonal imbalance, which are indicated by increased levels of cortisol, decreased levels of ADPN, antioxidant defenses, and MDA. It is therefore proposed that ADPN could be used as a biomarker for metabolic disorders related to obesity.

## 1 INTRODUCTION

Obesity is a disease that impacts many metabolic pathways directly. Obesity is now a threat to all, including industrialized nations, because of lifestyle changes, lack of physical activity, and overconsumption of high-calorie meals [1]. Especially in low- and middle-income nations, obesity has been rising in recent decades and is projected to become one of the most significant public health issues in the twenty-first century [2]. Obesity among Iraqi patients is a major health concern, mirroring other obesity-related health issues in the Arab region. Obesity levels have escalated in the country, mirroring global statistics on obesity. It is estimated that the number of overweight and obese persons in Iraq reflects a general increase in obesity levels worldwide. The prevalence of obesity in the world has almost doubled since 1980 [3]. There are worrying statistics on obesity among certain demographic groups. For example, obesity levels are as high as 54.5% among men and 45.5% among women [4]. In a study carried out in the Kurdistan Region between 2018 and 2023, hypertension, diabetes, dyslipidemia, and physical inactivity are identified as the most common modifiable risk factors for CVD among Iraqi patients. In the study, 86.17% of cardiac patients were found to have hypertension, whereas physical inactivity and dyslipidemia were observed in 60.59% and 56.31%, respectively [5]. These results are comparable to other findings in other places, and in addition to this, smoking and unhealthy food habits are also contributing to the rise in obesity levels [6], [7]. Besides this, Obesity also affects metabolic health in a significant way. Recent studies suggest that obesity may influence the release

and activity of many metabolic hormones, causing disruptions in energy and glucose metabolism [8], [9]. Obesity's effect on hormones such as insulin, leptin, ghrelin, and adiponectin (ADPN) are crucial for understanding the pathophysiology of obesity-related problems, including type 2 diabetes and cardiovascular disease [10]. The importance of hormonal imbalance in obesity management is further emphasized by the potential for a vicious cycle to arise from these hormonal imbalances to worsen the condition [11]. In addition to fat storage, WAT is a key component in metabolism and inflammation due to the secretion of adipokines, which are bioactive molecules that have hormone-like effects. Among these is adiponectin, a 30-kDa secretory hormone primarily secreted by adipocytes. It is known to increase insulin sensitivity and possesses anti-inflammatory effects. There are three major isoforms of adiponectin, which are classified according to molecular weight: low molecular weight (LMW), middle molecular weight (MMW), and high molecular weight (HMW). Among these variants, HMW adiponectin is thought to be the most physiologically active and a good indication of insulin sensitivity. Reduced HMW adiponectin levels have been linked to obesity-related insulin resistance [8]. Furthermore, adipose tissue produces hormones that can impact energy balance and help regulate metabolism. These hormones may encourage the buildup of fat in obese people and impede attempts to lose weight [12]. One of these hormones is cortisol, also known as the "stress hormone," which is synthesized by the adrenal glands in reaction to stress and is integral to numerous physiological processes, including metabolism and immunological response [13]. A study reported there is a correlation between chronic stress and obesity, which in turn activates the hypothalamic-pituitary-adrenal (HPA) axis and sets off the neuroendocrine response system. This activation leads to increased cortisol synthesis, which causes inflammatory responses that can harm the body's tissues [14]. Also, chronic stress raises cortisol levels, which can contribute to weight gain, especially around the abdomen, because of an increase in hunger [15]. Furthermore, obesity disrupts the balance of antioxidant enzymes like superoxide dismutase (SOD) and catalase (CAT), which exacerbates oxidative stress and lipid peroxidation and strengthens the connection between oxidative stress and obesity [16], [17]. As a result, understanding hormonal issues is crucial for treating obesity and related metabolic disorders. This study intends to evaluate the relationship between serum cortisol, adiponectin levels, and oxidative stress indicators in obese people in an Iraqi community.

# 2 MATERIALS AND METHODS

## 2.1 Participants

The current study involved 88 Iraqi individuals aged between 18 and 65 years. The participants were divided into three categories: healthy controls (n=28), those who are overweight (n=30), and those who are obese (n=30). They were recruited from Baqubah Teaching Hospital and the Specialized Outpatient Clinics in Baqubah City, Diyala Governorate, during the timeframe from October 2025 to April 2026.

All individuals involved shared their clinical and demographic data as per the study protocol. Venous blood, totaling five milliliters, was drawn from each individual under sterile conditions, transferred to gel tubes, and permitted to clot at room temperature. After a 10-minute centrifugation at 4000 rpm, the serum was isolated, placed into sterile Eppendorf tubes, and stored at -20 °C until further biochemical testing was conducted.

## 2.2 Determination of Serum Metabolic Hormones

Using commercially available kits (cortisol, ADPN, CAT and MDA, USCN. Comp.), the sandwich enzyme-linked immunosorbent assay (ELISA) was used to measure the serum concentrations of cortisol and adiponectin. 96-well microplates that had been pre-coated with particular antibodies were used for the assay. As directed by the manufacturer, serum samples, biotin-conjugated antibodies, and HRP-conjugated avidin were added one after the other. Absorbance was measured at 450 nm when the reaction was stopped with stop solution after incubation and the addition of TMB substrate.

## 2.3 Demographics/BMI

A questionnaire created specifically for this study was used to gather demographic data, such as age and sex. A member of the study team measured the participants' weight both at baseline and at follow-up. These weights and measured heights were used to calculate the body mass index (BMI) [18].

## 2.4 Exclusion Criteria

In order to ensure homogeneity among the participants and minimize confounding factors. Those taking medications like estrogens, inhaled corticosteroids, anabolic steroids by injection, or oral glucocorticoids, which are known to influence cortisol or adipokine concentrations, were excluded. Those suffering from psychopathology or other pathological disorders associated with energy metabolism were not selected either. Moreover, individuals diagnosed with heart, renal, hepatic, or pulmonary insufficiencies were excluded.

## 2.5 Data Analysis

The statistical analysis as well as the graphical representation of the data were conducted using IBM SPSS Statistics software as well as GraphPad Prism. In SPSS, the data were checked for normality (Shapiro-Wilk test) as well as homogeneity of variance (Levene's test). After checking the data, the comparison of the three groups of the study (mean ± standard deviations), namely healthy control, overweight, and obesity, was conducted using one-way analysis of variance (ANOVA) as well as multiple pairwise comparisons. In addition, Spearman's rank correlation coefficient. In GraphPad Prism, diagnostic performance of biomarkers was evaluated using receiver operating characteristic (ROC) curve analysis, with area under the curve (AUC), sensitivity, and specificity reported. A P value less than 0.05 was considered significant.

# 3 RESULTS AND DISCUSSION

Table 1 summarizes the participants' baseline demographics by BMI category (healthy control, overweight, and obesity). Participants were distributed fairly evenly across the three groups, with 30 people (34.1%) in each of the overweight and obese groups and 28 people (31.8%) in the healthy control group. The groups' BMI showed highly significant differences ($P < 0.001$), clearly increasing from the healthy control group to the overweight group and finally to the obese group. Males and females made up 46.4% and 53.6% of the healthy control group, 70.0% and 30.0% of the overweight group, and 60.0% and 40.0% of the obese group, respectively ($P > 0.05$). Similarly, there was no statistically significant difference ($P > 0.05$) in the mean age, which was 34.29 ± 13.78 years in the healthy control group, 34.80 ± 10.99 years in the overweight group, and 34.20 ± 9.46 years in the obesity group. Additionally, there was no discernible difference in the distribution of participants by age group ($P > 0.05$).

BMI, a widely used tool for assessing obesity, better predicts obesity risk [19]. When taken as a whole, these findings show that the demographic features of the three groups were well matched at baseline. This interpretation is in line with earlier studies on obesity that purposefully selected age- and sex-comparable groups to increase study efficiency and reduce confounding. Age affects the relationship between BMI and health outcomes, with younger individuals showing higher BMI values for the same adiposity levels compared to older adults [20].

Table 1: Baseline demographic characteristics of the study participants according to body mass index category (healthy control, overweight, and obesity).

| Variable | Healthy control (n=28) | Over-weight (n=30) | Obesity (n=30) | *P* value |
|---|---|---|---|---|
| Sample size, n (%) | 28 (31.8%) | 30 (34.1%) | 30 (34.1%) | |
| BMI (kg/m²) | 23.92 ± 1.37[a] | 27.41 ± 1.20[b] | 34.30 ± 2.88[c] | <0.001** |
| Sex, n (%) | | | | > 0.05[a] |
| Male | 13 (46.4%) | 21 (70.0%) | 18 (60.0%) | |
| Female | 15 (53.6%) | 9 (30.0%) | 12 (40.0%) | |
| Age (years), Mean ± SD | 34.29 ± 13.78 | 34.80 ± 10.99 | 34.20 ± 9.46 | > 0.05[b] |
| Age categories, n (%) | | | | > 0.05[a] |
| 18–19 | 4 (14.3%) | 3 (10.0%) | 2 (6.7%) | |
| 20–29 | 8 (28.6%) | 7 (23.3%) | 9 (30.0%) | |
| 30–39 | 8 (28.6%) | 10 (33.3%) | 11 (36.7%) | |
| 40–49 | 3 (10.7%) | 7 (23.3%) | 6 (20.0%) | |
| 50–65 | 5 (17.9%) | 3 (10.0%) | 2 (6.7%) | |
| ** p < 0.001, * p < 0.05 | | | | |

Table 2: Comparison of biochemical and oxidative stress biomarkers among healthy control, overweight, and obesity groups.

| Biomarker | Healthy control (n=28) | Over-weight (n=30) | Obesity (n=30) | *P* value |
|---|---|---|---|---|
| Cortisol ng/mL | $15.84 \pm 15.75^{a}$ | $17.34 \pm 12.11^{a}$ | $30.85 \pm 13.87^{b}$ | <0.001** |
| ADPN ng/mL | $5.28 \pm 1.06^{a}$ | $3.79 \pm 0.88^{b}$ | $2.11 \pm 1.03^{c}$ | <0.001** |
| CAT ng/mL | $0.072 \pm 0.032^{a}$ | $0.045 \pm 0.014^{b}$ | $0.035 \pm 0.010^{c}$ | <0.001** |
| MDA ng/mL | $108.38 \pm 50.90^{a}$ | $178.72 \pm 45.40^{b}$ | $228.13 \pm 40.61^{c}$ | <0.001** |

Additionally, gender plays a significant role in obesity-related health outcomes. Females generally exhibit higher complication rates post-surgery across all BMI categories [21]. Conversely, the association between BMI and mortality risk varies with age. Obesity class II consistently correlates with higher mortality risk across all ages, while the relationship for lower obesity classes changes with age [22].

All biochemical and oxidative stress markers in Table 2 indicated very significant differences between the three study groups; the P value for each variable was less than (< 0.001), demonstrating a strong relationship between the changes and the participants' weight status. The obese group had higher cortisol levels than the overweight and healthy groups, according to the findings. The obese group had cortisol levels of 30.85 ± 13.87, while the healthy and overweight groups had values of 15.84 ± 15.75 and 17.34 ± 12.11, respectively. The latter two groups did not differ significantly from one another. The results of our investigation are in consistent with a recent study that discovered that women who were obese had considerably higher total cortisol levels than their counterparts who were normal weight, with mean serum cortisol levels of 6.2 µg/dl versus 4.7 µg/dl, respectively [23]. About 20% of children and adolescents in another study had high cortisol levels, which may be connected to metabolic syndrome [24]. The metabolic activity of the enzyme 11β-HSD1 may be the cause of increased cortisol release in obesity. This enzyme is in charge of converting inactive cortisol into cortisol, the active form. Elevated 11β-HSD1 activity in adipose tissue raises local cortisol levels, which in turn stimulate fat storage, especially in the visceral area, and the differentiation of primary adipocytes into mature adipocytes [25]. The findings also showed that when weight increased and obesity developed, adiponectin levels significantly and steadily decreased. The healthy group's ADPN levels were 5.28 ± 1.06, the overweight groups were 3.79 ± 0.88, and the obese groups were 2.11 ± 1.03. All groups showed significant differences. The results of our investigation are in consistent with a recent study that indicated that healthy obese people had considerably lower serum ADPN levels than non-obese controls (2.35±0.77 vs. 8.10±2.98 µg/mL) [26]. The reduction in ADPN levels is explained by the hypertrophy of fat cells in obesity, which increases the production of inflammatory cytokines, including TNF-α and IL-6, which directly block the expression and secretion of the ADPN gene [27]. The antioxidant CAT, which registered 0.072 ± 0.032 and 0.045 ± 0.014 and 0.035 ± 0.010, respectively, showed a substantial and steady decline from the healthy group to the overweight group and finally to the obese group. This is attributed to a CAT overexpression in reaction to elevated oxidative stress in this particular setting [28]. this result consistent with Many studies show that obese people frequently have decreased catalase activity in a variety of tissues, including the liver and adipose tissue. This decline exacerbates oxidative stress and cell damage by reducing the body's capacity to neutralize hydrogen peroxide [29]. MDA showed a distinct and steady rise in all research groups, starting at 108.38 ± 50.90 in the healthy group, increasing to 178.72 ± 45.40 in the overweight group, and finally reaching 228.13 ± 40.61 in the obese group. This finding is in agreement with numerous adult and pediatric studies that show considerably greater MDA in obese compared to normal-weight individuals [30], [31]. This rise in MDA levels in the blood or urine is indicative of increased lipid and cell membrane damage brought on by the long-term oxidative stress linked to obesity. The significance of MDA as a sensitive clinical biomarker of oxidative stress and its interaction with lipid membranes in the setting of metabolic disorders has been validated by recent research [32], [33].

From Tables 3 and 4, (Fig.1) receiver operating characteristic (ROC) curve analysis was done to evaluate the diagnostic efficacy of both cortisol and ADPN in distinguishing between overweight and obese people and healthy persons. According to the data (AUC = 0.810) and significant statistical significance (P < 0.001), the current results demonstrated that cortisol has a moderate diagnostic ability in distinguishing between obesity and healthy individuals. Cortisol's capacity to distinguish between overweight and healthy people was clearly limited, according to the data (AUC = 0.586), and the findings were not statistically significant (P > 0.05). The limited specificity reported in most comparisons

indicates a significant proportion of false positives, which limits cortisol's utility as an independent biomarker for early detection. These findings are comparable with those of Abraham et al. (2013), who found that cortisol levels can be altered by a variety of physiological and psychological factors other than fat storage, reducing its discriminatory value [34]. Fan et al. (2021) back up this claim by finding inconsistent relationships between morning cortisol levels and several measures of obesity, implying that cortisol is not a sufficiently specific biomarker for detecting early-stage overweightness [35]. On the other hand, adiponectin showed remarkable diagnostic efficacy in every comparison, with AUC values ranging from 0.864 to 0.989, indicating extremely high case classification accuracy. Adiponectin was the best at discriminating between obesity and healthy people, with full sensitivity (100%) and good specificity (89.3%) as shown Table 5. These findings support the critical function of ADPN as an accurate biomarker, as revealed in a previous study by Liu et al. (2018), who found that adiponectin had strong predictive potential for metabolic disorders linked with obesity [36]. Agostinis-Sobrinho et al. (2022) supports these findings by demonstrating that ADPN surpasses many other biomarkers in predicting the likelihood of insulin resistance and fat mass disorders due to its strong and direct inverse connection with adipose tissue [37].

From Table 5, Spearman's correlation matrix in the overweight and obese groups indicated multiple significant relationships between anthropometric factors and biochemical markers. Weight and BMI showed a high positive connection ($r_s = 0.81$, $P < 0.01$), whereas weight and height showed a moderate positive correlation ($r_s = 0.53$, $P < 0.01$). At the biomarker level, weight was positively connected with cortisol ($r_s = 0.36$, $P < 0.01$) and MDA ($r_s = 0.32$, $P < 0.01$) but negatively correlated with ADPN ($r_s = -0.45$, $P < 0.01$) and CAT ($r_s = -0.36$, $P < 0.01$). BMI also correlated positively with cortisol ($r_s = 0.42$, $P < 0.01$) and MDA ($r_s = 0.54$, $P < 0.01$), but negatively with ADPN ($r_s = -0.66$, $P < 0.01$) and CAT ($r_s = -0.47$, $P < 0.01$). A positive connection was also discovered between height and ADPN ($r_s = 0.26$). The majority of the variables examined showed no significant associations with age, indicating that the changes seen in this cohort are more strongly associated with the obesity markers themselves than with age. The study's findings are consistent with BMI and weight having a high positive association, which is consistent with many studies that view BMI as a valid metric for classifying people based on their weight. Given the nature of physical growth, a positive link between height and weight is also anticipated [38]. Regarding the relation of cortisol, one study found that cortisol levels gradually increased with body weight growth. Individuals with high cortisol levels are 72% more likely to be fat, and obesity tends to persist over time in those with elevated cortisol [39]. Whereas another recent study found that increasing visceral fat (related to higher BMI and waist circumference) leads to a substantial drop in total ADPN levels [40]. In terms of the relationship between the CAT enzyme and MDA, a recent study found that as obesity increases, the body's antioxidant capacity declines, and that depletion of antioxidant defense mechanisms in situations of chronic obesity is caused by chronic oxidative stress. It also discovered that MDA was independently related to BMI, with MDA levels considerably higher in the overweight/obese group [41].

Table 3: Diagnostic performance of cortisol for discriminating overweight and obesity from healthy controls using receiver operating characteristic analysis.

| Cortisol | AUC | Std. Error | Sig. | Sensitivity | Specificity |
|---|---|---|---|---|---|
| Healthy *vs* Overweight-Obesity | 0.698 | 0.065 | < 0.01 * | 95% | 39.3% |
| Healthy *vs* Overweight | 0.586 | 0.078 | > 0.05 | 90% | 39.3% |
| Healthy *vs* Obesity | 0.810 | 0.061 | < 0.001** | 93.3% | 64.3% |

Table 4: Diagnostic performance of adiponectin for discriminating overweight and obesity from healthy controls using receiver operating characteristic analysis.

| Adiponectin | AUC | Std. Error | Sig. | Sensitivity | Specificity |
|---|---|---|---|---|---|
| Healthy *vs* Overweight-Obesity | 0.927 | 0.028 | < 0.001 ** | 95% | 75% |
| Healthy *vs* Overweight | 0.864 | 0.049 | < 0.001 ** | 90% | 75% |
| Healthy *vs* Obesity | 0.989 | 0.009 | < 0.001 ** | 100% | 89.3% |

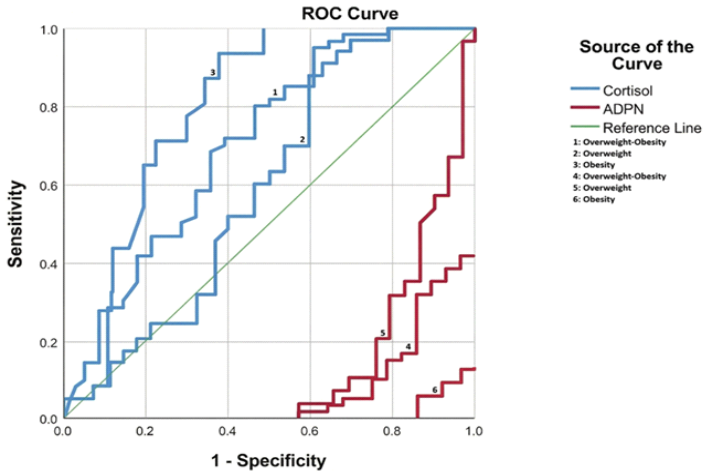


Figure 1: ROC curve of parameters (Cortisol and ADPN) under study for groups (Overweight-Obesity, Overweight and Obesity).

Table 5: Spearman correlation matrix among anthropometric variables and biochemical biomarkers in the combined overweight and obesity group.

| | Age | Weight | Height | BMI | Cortisol | ADPN | CAT | MDA |
|---|---|---|---|---|---|---|---|---|
| Age | 1.00 | 0.06 | -0.02 | 0.12 | -0.01 | 0.15 | -0.14 | 0.04 |
| Weight | 0.06 | 1.00 | 0.53 | 0.81 | 0.36 | -0.45 | -0.36 | 0.32 |
| Height | -0.02 | 0.53** | 1.00 | -0.01 | -0.02 | 0.26 | -0.03 | -0.24 |
| BMI | 0.12 | 0.81** | -0.01 | 1.00 | 0.42 | -0.66 | -0.47 | 0.54 |
| Cortisol | -0.01 | 0.36** | -0.02 | 0.42** | 1.00 | -0.33 | -0.08 | 0.23 |
| ADPN | 0.15 | -0.45** | 0.26* | -0.66** | -0.33 | 1.00 | 0.18 | -0.33 |
| CAT | -0.14 | -0.36** | -0.03 | -0.47** | -0.08 | 0.18 | 1.00 | -0.36 |
| MDA | 0.04 | 0.32* | -0.24 | 0.54** | 0.23 | -0.33 | -0.36 | 1.00 |

Consequently, it can be said that obesity is more than just being overweight; rather, it is linked to serious hormonal and metabolic problems, which are typified by increased stress and oxidative damage markers and decreased antioxidant and metabolic control markers. This lends credence to the idea that obesity is a complicated metabolic disorder that may make a person more vulnerable to long-term problems.

# 4 CONCLUSIONS

The findings of this study show a clear and statistically significant link between higher BMI and abnormalities in biochemical and oxidative stress indicators. The obesity group had significantly higher cortisol levels than the healthy and overweight control groups, indicating an elevated physiological stress response with growing obesity. In contrast, ADPN levels decreased gradually and significantly across all groups, showing a deterioration in its metabolic and anti-inflammatory protective effect as adipose tissue accumulation increased. The study also found a large decrease in the antioxidant enzyme CAT in overweight and obese people, which coincided with a considerable increase in MDA levels, a critical biomarker of lipid peroxidation. These findings indicate that obesity is linked to a significant imbalance in oxidative stress, as seen by decreased antioxidant defenses and increased cellular oxidative damage. As a result, obesity can be classified as a complex metabolic illness that includes hormonal, metabolic, and oxidative alterations that may raise the risk of developing chronic difficulties in the future. As a result, these indicators may be useful in detecting the pathogenic effects of obesity and promoting early intervention to reduce oxidative stress imbalances and their repercussions.

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# REFERENCES


[1] I. S. Elias and M. A. H. J. Mohammed, "Effect of obesity on several biochemical variables and vitamin D in the blood serum of infertile women in Mosul," Journal of Basic Science, vol. 9, no. 29, pp. 325-338, 2025, , [Online]. Available: https://doi.org/10.31185/bsj.Vol19.Iss29.1179.

[2] G. M. Abood, R. H. Kadhem, and A. A. B. Ali, "Childhood obesity: Causes, complications and preventive strategies," Thi-Qar Medical Journal, vol. 29, no. 1, pp. 119-123, 2025, doi: 10.32792/jmed.2025.29.14.

[3] B. Abuyassin and I. Laher, "Obesity-linked diabetes in the Arab world: A review," Eastern Mediterranean Health Journal, vol. 21, no. 6, pp. 420-439, 2015, doi: 10.26719/2015.21.420.

[4] S. N. Abed, S. K. S. Al-Zamali, and T. M. Muslim, "The epidemiological profile associated with lifestyle risk factors and nutritional status for COVID-19 patients in the Iraqi population," Journal of Public Health in Africa, vol. 14, no. 6, p. 2323, 2023, doi: 10.4081/jphia.2023.2323.

[5] N. S. Murad, S. S. Miro, V. A. H. Ismael, and D. M. Abdulah, "Modifiable risk factors for cardiovascular disease in Iraqi Kurdistan population: A large epidemiological study," Healthcare in Low Resource Settings, vol. 12, no. 2, 2023, doi: 10.4081/hls.2023.12087.

[6] T. M. J. Taher, "Public awareness of healthy lifestyle among Iraqi population," Al-Rafidain Journal of Medical Sciences, vol. 5, pp. 92-98, 2023, , [Online]. Available: https://doi.org/10.54133/ajms.v5i.170.

[7] M. W. Mahdi et al., "Significant iron and vitamin D deficiency and significant increase of hepcidin level in Iraqi patient with iron deficiency anemia," Biochemical & Cellular Archives, vol. 19, 2019, doi: 10.35124/bca.2019.19.1.1671.

[8] M. A. McArdle, O. M. Finucane, R. M. Connaughton, A. M. McMorrow, and H. M. Roche, "Mechanisms of obesity-induced inflammation and insulin resistance: Insights into the emerging role of nutritional strategies," Frontiers in Endocrinology, vol. 4, p. 52, 2013, doi: 10.3389/fendo.2013.00052.

[9] A. M. S. Almohaidi et al., "The association between rs2414096 of CYP19 gene polymorphism and polycystic ovary syndrome (PCOS) in Iraqi women," Journal of Obstetrics, Gynecology and Cancer Research, vol. 10, no. 12, pp. 917-923, 2025, doi: 10.24200/jogcr.10.12.917.

[10] B. Peters, J. Vahlhaus, and O. Pivovarova-Ramich, "Meal timing and its role in obesity and associated diseases," Frontiers in Endocrinology, vol. 15, p. 1359772, 2024, doi: 10.3389/fendo.2024.1359772.

[11] S. H. Kim et al., "Impact of socioeconomic status on health behaviors, metabolic control, and chronic complications in type 2 diabetes mellitus," Diabetes & Metabolism Journal, vol. 42, no. 5, p. 380, 2018, doi: 10.4093/dmj.2017.0102.

[12] A. Shoemaker, "Obesity and its association with endocrine disorders in metabolic syndrome," Endocrinology & Metabolic Syndrome, vol. 13, p. 426, 2024, doi: 10.35248/2161-1017.24.13.426.

[13] J. Yin, "Cortisol: The stress hormone's impact on health and well-being," American Journal of Physiology, Biochemistry and Pharmacology, vol. 13, no. 12, pp. 1-2, 2023.

[14] T. R. Chandan et al., "Cortisol imbalance and weight gain," Research Journal of Pharmacology and Pharmacodynamics, vol. 17, no. 4, pp. 319-326, 2025, doi: 10.52711/2321-5836.2025.00049.

[15] E. S. van der Valk, M. Savas, and E. F. C. van Rossum, "Stress and obesity: Are there more susceptible individuals?," Current Obesity Reports, vol. 7, no. 2, pp. 193-203, 2018, doi: 10.1007/s13679-018-0306-y.

[16] A. H. Alta'ee et al., "The balance between superoxide dismutase and catalase activities in sera of obese Iraqi men," EuroMediterranean Biomedical Journal, vol. 10, 2015, , [Online]. Available: https://doi.org/10.3269/1970-5492.2015.10.17.

[17] N. P. V. Widhiani, I. A. E. Widiastuti, and F. F. Zubaidi, "The difference in malondialdehyde (MDA) levels among normoweight, overweight, and obesity in student of medicine program faculty of medicine, Mataram University," Jurnal Biologi Tropis, vol. 23, no. 3, pp. 595-599, Aug. 2023, doi: 10.29303/jbt.v23i3.5429.

[18] L. Q. Abdulhameed, "Evaluation of serum hyperprolactinemia status among Iraqi infertile males in Diyala province," Journal of Krishna Institute of Medical Sciences (JKIMSU), vol. 12, 2023.

[19] S. B. Çelik, P. G. E. Bucaktepe, Ü. B. Batur, and İ. U. Bulut, "Obezite geliştirme riskini öngörebilecek bazı ölçüm parametrelerinin analizi: Klinik bir çalışma," Journal of Medicine and Palliative Care, Feb. 2023, doi: 10.47582/jompac.1199357.

[20] A. M. Nevill and G. S. Metsios, "The need to redefine age- and gender-specific overweight and obese body mass index cutoff points," Nutrition & Diabetes, vol. 5, no. 11, Nov. 2015, doi: 10.1038/NUTD.2015.36.

[21] E. L. Smith et al., "The obesity paradox: Body mass index complication rates vary by gender and age among primary total hip arthroplasty patients," Journal of Arthroplasty, vol. 35, no. 9, pp. 2658-2665, Sep. 2020, doi: 10.1016/j.arth.2020.04.094.

[22] M. A. Sánchez-Lastra et al., "Body mass index and mortality from middle age to old age," Medicine & Science in Sports & Exercise, vol. 54, no. 9S, p. 576, Sep. 2022, doi: 10.1249/01.mss.0000882292.04814.83.

[23] Z. A. Al-Safi et al., "Evidence for disruption of normal circadian cortisol rhythm in women with obesity," Gynecological Endocrinology, vol. 34, no. 4, pp. 336-340, Apr. 2018, doi: 10.1080/09513590.2017.1393511.

[24] A. Martens et al., "Clinical and biological correlates of morning serum cortisol in children and adolescents with overweight and obesity," PLOS ONE, vol. 16, no. 10, Oct. 2021, doi: 10.1371/journal.pone.0258653.

[25] Y. Han et al., "11β-HSD1 inhibitor alleviates lipid metabolism disorder by activating the AMPK signaling pathway," Scientific Reports, vol. 15, p. 41968, 2025, doi: 10.1038/s41598-025-25941-1.

[26] F. Tabassum et al., "Correlation of serum adiponectin level with insulin resistance in healthy obese individuals: A case-control study," National Journal of Laboratory Medicine, Jan. 2023, doi: 10.7860/njlm/2023/58015.2703.

[27] D. Banerjee and A. Mani, "Obesity's systemic impact: Exploring molecular and physiological links to diabetes, cardiovascular disease, and heart failure," Frontiers in Endocrinology, vol. 16, p. 1681766, 2025, doi: 10.3389/fendo.2025.1681766.

[28] J. Bausenwein et al., "Elevated levels of oxidized low-density lipoprotein and of catalase activity in follicular fluid of obese women," Molecular Human Reproduction, vol. 16, no. 2, pp. 117-124, Feb. 2010, doi: 10.1093/molehr/gap078.

[29] F. A. Alhumaydhi et al., "Catalase functions and glycation: Their central roles in oxidative stress, metabolic disorders, and neurodegeneration," Catalysts, vol. 15, p. 817, 2025, , [Online]. Available: https://doi.org/10.3390/catal15090817.

[30] alaa, M. I. Salman, "Obesity in western Iraqi patients: The involvement of glutathione peroxidase, catalase, superoxide dismutase, and malondialdehyde," Wasit Journal for Pure Sciences, vol. 2, pp. 224-234, 2023, , [Online]. Available: https://doi.org/10.31185/wjps.211.

[31] A. K. Fariya and B. F. Dias, "Study of oxidative stress biomarkers in obese children," International Journal of Research in Medical Sciences, vol. 6, p. 3335, 2018, doi: 10.18203/2320-6012.ijrms20184042.

[32] T. Choosong et al., "Correlation between obesity and urinary malondialdehyde levels in a rural Southern Thai population: A cross-sectional study," Clinical Epidemiology and Global Health, p. 102258, 2025, doi: 10.1016/j.cegh.2025.102258.

[33] L. Couture et al., "Malondialdehyde as a clinical indicator for oxidative stress and functional outcomes in acute ischemic stroke," Stroke: Vascular and Interventional Neurology, vol. 4, p. e12984-029, 2024, , [Online]. Available: https://doi.org/10.1161/SVIN.04.suppl_1.029.

[34] S. Abraham et al., "Cortisol, obesity, and the metabolic syndrome: A cross-sectional study of obese subjects and review of the literature," Obesity, vol. 21, pp. E105-E117, 2013, doi: 10.1002/oby.20083.

[35] K. Fan et al., "Negative associations of morning serum cortisol levels with obesity: The Henan rural cohort study," Journal of Endocrinological Investigation, vol. 44, pp. 2581-2592, 2021, doi: 10.1007/s40618-021-01558-9.

[36] Z. Liu et al., "Meta-analysis of adiponectin as a biomarker for the detection of metabolic syndrome," Frontiers in Physiology, vol. 9, p. 1238, 2018, doi: 10.3389/fphys.2018.01238.

[37] C. Agostinis-Sobrinho et al., "Is the leptin/adiponectin ratio a better diagnostic biomarker for insulin resistance than leptin or adiponectin alone in adolescents?," Children, vol. 9, p. 1193, 2022, doi: 10.3390/children9081193.

[38] N. R. Mayasari, I. Kumalasari, V. Indrawati, and S. A. Pratama, "Obesity and correlation of body mass index and body composition among sports sciences versus non-sport sciences students," Al-Rafidain Journal of Medical Sciences, vol. 8, no. 1, pp. 27-31, Jan. 2025, , [Online]. Available: https://doi.org/10.54133/ajms.v8i1.1549.

[39] K. L. Rodrigues et al., "Hair cortisol levels are associated with overweight and obesity in the ELSA-Brasil cohort," Frontiers in Endocrinology, vol. 15, p. 1361715, 2024, doi: 10.3389/fendo.2024.1361715.

[40] S. Gariballa et al., "Total adiponectin in overweight and obese subjects and its response to visceral fat loss," BMC Endocrine Disorders, vol. 19, p. 55, 2019, doi: 10.1186/s12902-019-0386-z.

[41] A. Klisic et al., "Malondialdehyde as an independent predictor of body mass index in adolescent girls," Journal of Medical Biochemistry, vol. 42, p. 224, 2023, doi: 10.5937/jomb0-39044.