ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Research Article DOI: 10.62425/pharmata.1541951

# Investigation of the Relationship Between Antioxidant Levels and Body Mass Index and Obesity

Elif Zeynep ÖZTÜRK[1]
Fatmagül DÜZENLİ[2]
Neslihan YÜCE[3]
Mehlika ALATAŞ[4]
İlayda HAVUZ[2]
Ufuk UZUN[2]
Özgür ARAZ[2]
Nurcan KILIÇ BAYGUTALP[5]

[1]Department of Fundamental Sciences, Faculty of Engineering and Natural Sciences, Kütahya Health Science University, Kutahya, Türkiye
[2]Department of Medical Laboratory Techniques, Vocational School of Health, Artvin Coruh University, Artvin, Türkiye
[3]Department of Biochemistry, Faculty of Medicine, Ataturk University, Erzurum, Türkiye
[4]Department of Physiotherapy and Rehabilitation, Vocational School of Health, Artvin Coruh University, Artvin, Türkiye
[5]Department of Biochemistry, Faculty of Pharmacy, Ataturk University, Erzurum, Türkiye

## ABSTRACT

**Objective:** Today, it is well known that obesity plays a role in the pathogenesis of many diseases, and one of these mechanisms of action is oxidative stress. There are many studies on the formation of oxidative stress in obesity, but its effect on young adults (18-35 years old) is not fully known.

**Methods:** In this context, we investigated oxidative stress levels by measuring oxidant (MDA) and antioxidant (SOD) parameters in young adult obese and overweight individuals with ELISA method. Our hypothesis is that the oxidant parameter (MDA) is high and the antioxidant parameter (SOD) is low in young adult obese individuals due to oxidative stress.

**Results:** According to the study results, a statistically significant difference was found in the SOD and MDA values of overweight individuals ($P<.001$) and a statistical difference was found in the SOD and MDA values of obese individuals ($P<.05$). According to the results of our study, oxidative stress is high not only in obese but also in overweight individuals. In fact, a higher rate of oxidative stress was observed in overweight individuals compared to obese individuals.

**Conclusion:** These data show us the importance of weight control in preventing the development of diseases by preventing the formation of oxidative stress. With further research, the effect of oxidative stress in young individuals will be understood more clearly.

**Keywords:** MDA, Obesity, Overweight, Oxidative Stress, SOD

## INTRODUCTION

Obesity, characterized by an increase in body weight resulting in excessive fat accumulation, represents a worldwide social problem[1] and has been recognized as a key factor in the pathogenesis of various diseases.[2] Unfortunately, obesity affects an increasing number of individuals in developed countries. Recently, obesity has also been found to be associated with low-grade chronic systemic inflammation in adipose tissue. This is influenced by the activation of the innate immune system, which promotes a pro-inflammatory state and Oxidative Stress (OS) in adipose tissue, triggering a systemic acute phase response. Various chronic diseases are also the result of obesity (e.g., metabolic syndrome, diabetes mellitus, liver and cardiovascular diseases, and cancer) and are associated with OS.[2] Therefore, it has been suggested that adipose tissue inflammation in obese patients plays a critical role in the pathogenesis of obesity-related complications.[3]

Adipose tissue is an endocrine and storage organ necessary for energy homeostasis. This tissue, consisting mainly of adipocytes, also contains other cells (e.g. fibroblasts, fibroblastic pre-adipocytes, endothelial and immune cells)[4] that secrete hormones and cytokines (adipokines or adipocytokines) that exert endocrine, paracrine and autocrine effects. In physiological and pathological conditions, adipokines also induce the production of Reactive Oxygen Species (ROS), producing OS and, in turn, driving massive, dysregulated production of other adipokines.[5] Various mechanisms play a role in creating OS in obesity. OS and proinflammatory processes are strongly associated.[5,6] Upon activation, many immune cells produce free radicals and likewise ROS synthesis promotes an inflammatory state. Adipose tissue is not just a fat depot. It is integral to various physiological functions, including metabolic regulation and energy storage.[6]

Received 12.10.2024
Accepted 24.07.2025
Publication Date 25.07.2025
Corresponding author: Elif Zeynep ÖZTÜRK
E-mail: elifzeynep.ozturk@ksbu.edu.tr
Cite this article: Öztürk EZ, Düzenli F, Yüce N, et al. Investigation of the Relationship between Antioxidant Levels and Body Mass Index and Obesity *Pharmata*. 2025;5(2):46-50.

ROS (Reactive Oxygen Species) occur under physiological conditions and in many diseases, causing direct or indirect damage to different organs; Therefore, Oxidative Stress (OS) is known to be involved in pathological processes such as obesity, diabetes, cardiovascular disease and atherogenic processes. It has been reported that obesity can induce systemic OS, and OS, in turn, is associated with dysregulated adipokine production, which contributes to the development of metabolic syndrome.[4] The sensitivity of C-Reactive Protein (CRP) and other biomarkers of oxidative damage is higher in individuals with obesity and is directly related to Body Mass Index (BMI) and body fat percentage, LDL oxidation and Trigliseride (TG) levels[5]; On the contrary, antioxidant defense markers are lower compared to the amount of body fat and central obesity.[5,6] One study showed that a diet high in carbohydrates caused a significant increase in OS stress and inflammation in people with obesity.[7]

OS biomarkers such as Malondialdehyde (MDA) and F-2 isoprostanes (F2-IsoPs) are products of peroxidation of polyunsaturated fatty acids. One study showed that BMI was significantly associated with the concentration of F2-IsoPs. Additionally, dietary factors were analyzed and it was observed that fruit consumption was inversely proportional to the lipid peroxidation level. This same study found that women showed a higher level of peroxidation compared to men, which may be due to the higher percentage of fat they have. Additionally, another study found a positive relationship between lipid peroxidation level and plasma cholesterol concentration.[8] When obesity persists for a long time, antioxidant resources can be depleted by reducing the activity of enzymes such as Superoxide Dismutase (SOD).[9] The activity of SOD in individuals with obesity is significantly lower compared to healthy individuals, with implications for the development of obesity-related health problems.[10] Our hypothesis in our study, based on the data in the literature, is that obesity creates oxidative stress, increasing oxidant parameters and decreasing antioxidant parameters. Additionally, in our study, overweight individuals will be included and the oxidative stress status in overweight individuals will be examined. The results of our study will contribute to the literature by confirming previous research or presenting different results.

Obesity occurs when the amount of energy taken from food exceeds the amount of energy consumed by metabolism and physical activity. Oxidative stress can be defined as the disruption of the balance between oxidant and anti-oxidant systems in favor of oxidant systems, resulting in the release of lipid peroxidation and reactive oxygen products (ROS), causing cellular damage in the organism. The balance between free radical formation and excretion is important. As a result of a certain increase in the formation of free radicals in the cell or a certain decrease in their excretion, oxidative stress occurs and damages the organism. Increased oxygen consumption leads to an increase in free radical production. These free radicals produce enzymatic (superoxide dismutase, catalase, glutathione peroxidase) or nonenzymatic (vitamins A, C, E, folic acid, glutathione, alpha lipoic acid, mixed carotenoids, coenzyme Q10, various bioflavonoids, uric acid, albumin, antioxidant minerals) antioxidants. It is neutralized by a defense system containing Oxidative stress is a critically important event in the pathogenesis of many diseases. One of the reasons put forward for the mechanism of obesity-related increase in oxidative stress is that obesity increases the metabolic and mechanical workload of the myocardium. As a negative result of increased oxygen consumption in the myocardium, mitochondrial respiration increases and this may lead to the emergence of ROS.

In the light of these data the aim of our study is,

1. To examine the oxidant (MDA) change between groups with different Body Mass Index,
2. To examine the antioxidant (SOD) change between groups with different Body Mass Index,
3. To examine the effects of obesity and overweight on young adults.

Our aim is to seek an answer to the question of how serum oxidant/antioxidant levels change as body mass index increases. According to our hypothesis, we expect oxidants (MDA) to increase and antioxidants (SOD) to decrease in serum**.**

## METHODS

### Study Design

Our study included 30 people with a Body Mass Index between 18-25 kg/m$^2$, 30 people with a Body Mass Index between 25-30 kg/m$^2$, and 30 people with a Body Mass Index between 30-35 kg/m$^2$, totaling 90 young healthy adults between the ages of 18-35 living in Artvin. Half of the individuals in the groups are women (15 people) and half are men (15 people). In our study, a comparison of 3 groups will be made. People with chronic diseases (acute renal failure, chronic respiratory failure, nephrotic syndrome, malignancy), end-stage liver disease, acute infection, those with any health problems requiring urgent intervention, smokers, alcohol consumers, medication users, pregnant women, and those who consume food supplements were included in the study. Users were excluded from the study. Ethical approval was received from Atatürk University Faculty of Medicine Clinical Research Ethics Committee with number B.30.2.ATA.0.01.00/205 dated 30.03.2023.

**Sample collection**

Blood samples will be taken after 8 hours of fasting and serum will be separated by centrifuging at 1250 x g for 10 minutes. Aliquoted sera were stored in the freezer at -20°C. Frozen samples were gradually thawed just before analysis on the day of the study. All chemicals and serums were brought to room temperature (18-26 °C) before use. Serum samples were analyzed on the same day and according to standard laboratory methods.

**Biochemical Analyzes**

**MDA Analysis**

First, the MDA standard and sample are pipetted into the Epondorf. After incubating at 96°C for 1 hour, it is cooled, centrifuged at 16,000 g for 10 minutes, 200 µL of the upper phase is taken and added to all microplate wells, with standard solutions of different concentrations prepared from 1.1.3.3 tetraethoxypropane (200 µmol/L, 100 µmol/L). L, 50 µmol/L, 25 µmol/L, 12.5 µmol/L, 6.25 µmol/L, 3.125 µmol/L) MDA standard graph will be drawn from the device at 532 nm (Benchmark Plus Microplate Reader (Biorad, Segrate- Milan, Italy) and serum MDA concentrations are expressed in µmol/L for all samples.

**SOD Analysis**

The samples and the Assay reagent are pipetted into the relevant wells of the microplate, mixed and incubated at 25 °C for 20 minutes. The absorbance of the purple complex formed after 20 minutes is measured at 560 nm in a microplate reader (Benchmark Plus Microplate Reader (Biorad, Segrate-Milano, Italy). The greater the coloration, the less SOD (since it is inhibited).

- Calculation: SOD activity is calculated with the formula below and the results are expressed as U/mL

1U: % 50 NBT inhibition

$$\% \text{ inhibition: } \frac{(\text{A blanck} - \text{A sample})}{\text{A blanck}} \times 100$$

SOD (U/mL): % inhibition / (50x 0,01)

**Statistical analysis**

Determination of the study sample number and power analysis were performed using the G-Power package program (Faul, Erdfelder, Lang,; Buchner). The power calculated as 1-β was taken as minimum 85% in our clinical study. Data analysis was done in GraphPad Prism 10.3.0 program. Shapiro-Wilk test was performed to examine the normal distribution of the parameters. The p value was found to be greater than .05 in both groups (normal distribution. The results were evaluated within the 95% confidence interval and the p value <.05 was considered statistically significant. One Way ANOVA test was used to compare the groups has been used.

**RESULTS**

Half of the individuals included in the study are female and half are male. Demographic information of the individuals participating in the study is given in Table 1.

Table 1. Demographic information of individuals

| Group | Age (Mean) | BMI (Mean± SD) | Number of Males (n) | Number of Females (n) |
|---|---|---|---|---|
| Control | 21.7 | 21.81±1.712 | 15 | 15 |
| Overweight | 21.4 | 27.02±1.433 | 15 | 15 |
| Obese | 23.3 | 32.39±1.894 | 15 | 15 |

In overweight individuals, SOD levels were significantly lower (*P*<.01) while MDA levels were significantly higher (*P*<.01). In similar, in obese individuals, SOD levels were significantly lower (*P*<.05) while MDA levels were significantly higher (*P*<.05). SOD and MDA values of the groups are given in Table 2.

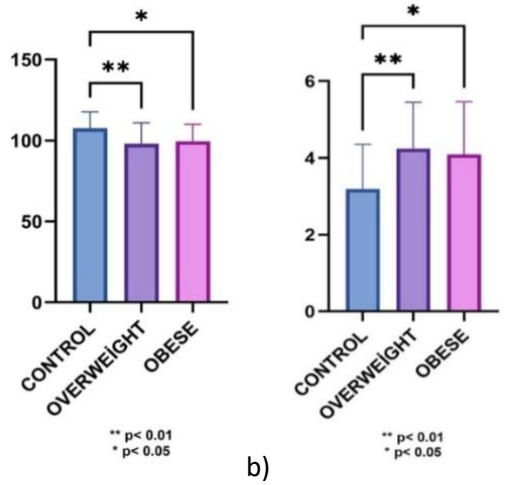
Table 2. Serum SOD and MDA levels in overweight and obese control group

| Group | SOD Levels (Mean (U/mL) ± SD) | *P* value | MDA Levels (Mean (µmol/L)±SD) | *P* value |
|---|---|---|---|---|
| Control | 107.7 ± 9.917 | - | 3.192 ± 1.164 | - |
| Overweight | 97.97 ± 12.98[a] | <.001 | 4.246 ± 1.208[a] | <.001 |
| Obese | 99.56 ± 10.45[b] | <.05 | 4.090 ± 1.377[b] | <.05 |

a: Statistically highly significant difference at the *P*<.01 level

b: Statistically significant difference at the *P*<.05 level

When SOD values were compared between groups, a significant relationship was found between BMI and SOD values. Again, when MDA values were compared between groups, a significant relationship was found between BMI and MDA values. Similar to obese, SOD and MDA values were observed to change significantly in overweight individuals. SOD and MDA distributions of the groups are given in Figure 1.




a) b)

Figure 1. a) SOD values of individuals (U/mL) b) MDA values of individuals (µmol/L)

## DISCUSSION

Our study evaluated the relationships between BMI and oxidative stress markers and antioxidant enzyme activity in a sample of individuals with different body mass indexes (normal, overweight and obese) in late healthy individuals. There were higher levels for superoxide dismutase (SOD) and lower levels for malondialdehyde (MDA) in normal weight individuals compared to individuals with higher BMI. Increased MDA and decreased SOD values observed together with increased BMI in young adults may be related to oxidative stress and metabolic disorders. The relationship between serum antioxidant levels, body mass index (BMI) and obesity is an area of increasing research interest, especially due to the effects of oxidative stress in metabolic disorders. Several studies have identified that lower serum antioxidant levels are generally associated with higher BMI and obesity, suggesting that antioxidants may play a protective role against the metabolic consequences of excess body fat.[5,10–12]

One of the key findings in this area is that serum antioxidant capacity is inversely related to BMI. For example, lower serum antioxidant status has been shown to be associated with metabolic syndrome, which is generally characterized by obesity and increased BMI.[12] Research suggests that individuals with higher BMI tend to have lower antioxidant levels, which may contribute to the oxidative stress observed in obese populations. This is consistent with the findings of a study reporting a significant decrease in total antioxidant capacity in serum among older adults, which was associated with increasing BMI.[13] These findings suggest that the body's ability to resist oxidative stress decreases as body weight increases.

The relationship between serum antioxidant levels, particularly superoxide dismutase (SOD) and malondialdehyde (MDA), and body mass index (BMI) is a complex interaction that has received significant attention in recent studies. This relationship is particularly important in the context of obesity, which is known to cause oxidative stress and alter the balance between oxidants and antioxidants in the body.[14]

Oxidative stress occurs when the oxidative component is favored in an oxidant and antioxidant system. Reactive oxygen species, the primary oxidant component in the human body, are produced during normal metabolic processes. However, it has been documented that in excess adiposity, reactive oxygen species levels increase and exceed the capacity of the antioxidant system, where the antioxidant enzymes superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT) play an important role.

Preclinical, clinical and epidemiological studies have reported the association between metabolic diseases and oxidative stress through the accumulation of lipid and protein oxidation products and decreased activity of antioxidant systems.[5]

Oxidative stress may result from an imbalance between increased production of reactive oxygen species (ROS) and/or decreased antioxidant defenses.[5,11] Malondialdehyde (MDA), the main end product of lipid oxidation caused by ROS, can cause cross-linking polymerization of proteins, nucleic acids, and other living macromolecules and cause great damage to the activities of the mitochondrial respiratory chain complex and key enzymes in mitochondria. At present, numerous evidences have confirmed that MDA in blood is an important indicator of the degree of oxidative damage *in vivo* and *in vitro*.[11]

Coversely, SOD, an important antioxidant enzyme that catalyzes the conversion of superoxide radicals into hydrogen peroxide and oxygen, tends to be lower in individuals with higher BMI. This decrease in SOD activity may exacerbate oxidative stress as the body loses the ability to neutralize harmful free radicals.[11,15] Studies have highlighted that lower SOD levels are associated with increased MDA levels and that there is a negative correlation between these two biomarkers.[14,15] When we look at the literature, we see that BMI and serum antioxidant levels are related, similar to the results of our study; they stated that oxidative stress and decreased antioxidant parameters are associated with hyperinsulinemia, hypertension and dyslipidemia findings and that this oxidative stress state may contribute to metabolic syndrome and cardiovascular diseases.[11,14]

Oxidative stress contributes to the pathology of cancer, cardiovascular diseases, diabetes, neurological disorders (Alzheimer's and Parkinson's diseases, Down syndrome), psychiatric diseases (depression, schizophrenia, bipolar disorder), kidney disease, lung disease (chronic pulmonary obstruction, lung cancer), and aging.[5,16] The amelioration/reversal of the harmful effects of oxidative stress occurs through the coordinated action of antioxidants. When the literature is examined, it is stated that the amelioration of the harmful effects of oxidative stress is carried out by antioxidant enzymes (Superoxide dismutases-SODs, catalase, glutathione peroxidase-GPx) and small molecular weight antioxidants (vitamins C and E, flavonoids, carotenoids, melatonin, ergothioneine, and others).[16]In our study, overweight individuals were included as well as obese individuals, and serum MDA and SOD values were significantly higher than those of normal weight individuals ($P$<.001).

Therefore, it can be said that serum antioxidant levels and oxidative stress levels in overweight individuals are parallel to those in obese individuals, and this oxidative stress status may contribute to the pathology of metabolic syndrome, cardiovascular diseases, diabetes, neurological disorders, and aging.

In summary, the relationship between serum antioxidant levels of SOD, MDA, and BMI is characterized by a consistent pattern of increased oxidative stress in individuals with higher BMI. In our study conducted in a healthy young adult sample, MDA and SOD levels were significantly different in overweight individuals compared to healthy individuals, parallel to those in obese individuals. Elevated MDA levels serve as a marker of oxidative damage, while SOD levels generally reflect the body's antioxidant defense capacity. Oxidative damage and antioxidant levels that play a role in repairing damage in overweight individuals as well as in obese patients may lead to adverse health outcomes. The interaction between these markers is influenced by a variety of factors, including physical activity, inflammation, and metabolic health. Future research should continue to explore these relationships to better understand the health consequences associated with increased BMI and the implications for potential therapeutic interventions.

**Ethics Committee Approval:** Ethical approval was received from Atatürk University Faculty of Medicine Clinical Research Ethics Committee with number B.30.2.ATA.0.01.00/205 dated 30.03.2023.
**Informed Consent:** The consent form was read and approved by the individuals participating in the study.
**Peer-review**: Externally peer-reviewed.
**Author Contributions:** Concept - E.Z.O,F.D; Design - E.Z.O; Supervision - N.B; Resources - F.D,I.H,U.U,O.A; Data Collection and/or Processing - E.Z.O,F.D,I.H,U.U,O.A; Analysis and/or Interpretation - N.Y, E.Z.O,F.D,I.H,U.U,O.A; Literature Search - E.Z.O,M.A; Writing Manuscript - E.Z.O,M.A; Critical Review - N.K.B,N.Y; Statistics – M.A.
**Conflict of Interest:** Nurcan Kılıç Baygutalp, who is featured in this article, is also the associate editor of the journal. This situation is considered a relationship that may create a conflict of interest. In order to ensure an impartial and transparent refereeing process, the process was carried out without assigning associate editors and without transferring the author's editorial position to the referees. In addition, in order to prevent conflicts of interest, all stages of this process were managed in accordance with the journal's ethical rules and international ethical guidelines such as COPE and ICMJE.
**Financial Disclosure:** This project was supported by TÜBİTAK 2209 Student Projects.

## REFERENCES

1. Lingvay I, Cohen R V, Roux CW le, Sumithran P. Obesity in adults. *The Lancet*. 2024;404(10456):972-987. [CrossRef]
2. Sarma S, Sockalingam S, Dash S. Obesity as a multisystem disease: Trends in obesity rates and obesity-related complications. *Diabetes Obes. Metab*. 2021;23(S1):3-16. [CrossRef]
3. Kunz HE, Hart CR, Gries KJ, et al. Adipose tissue macrophage populations and inflammation are associated with systemic inflammation and insulin resistance in obesity. *Am. J. Physiol Endocrinol. Metab*. 2021;321(1):E105-E121. [CrossRef]
4. Ahmed B, Sultana R, Greene MW. Adipose tissue and insulin resistance in obese. *Biomed. Pharmacother.* 2021;137:111315. [CrossRef]
5. Jakubiak GK, Osadnik K, Lejawa M, Kasperczyk S, Osadnik T, Pawlas N. Oxidative Stress in Association with Metabolic Health and Obesity in Young Adults. *Oxid. Med. Cell Longev*. 2021;2021(1):9987352. [CrossRef]
6. García-Sánchez A, Gámez-Nava J, Díaz- De la Cruz E, et al. The Effect of Visceral Abdominal Fat Volume on Oxidative Stress and Proinflammatory Cytokines in Subjects with Normal Weight, Overweight and Obesity. *Diabetes Metab. Syndr. Obes.* 2020;13:1077-1087. [CrossRef]
7. Zhao L, Liang J, Chen F, et al. High carbohydrate diet induced endoplasmic reticulum stress and oxidative stress, promoted inflammation and apoptosis, impaired intestinal barrier of juvenile largemouth bass (Micropterus salmoides). *Fish Shellfish Immunol*. 2021;119:308-317. [CrossRef]
8. Shabalala SC, Johnson R, Basson AK, et al. Detrimental effects of lipid peroxidation in type 2 diabetes: Exploring the neutralizing influence of antioxidants. *Antioxidants*. 2022;11(10):2071. [CrossRef]
9. Gusti AMT, Qusti SY, Alshammari EM, Toraih EA, Fawzy MS. Antioxidants-related superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPX), glutathione-S-transferase (GST), and nitric oxide synthase (NOS) gene variants analysis in an obese population: a preliminary case-control study. *Antioxidants*. 2021;10(4):595. [CrossRef]
10. Panic A, Stanimirovic J, Sudar-Milovanovic E, Isenovic ER. Oxidative stress in obesity and insulin resistance. *Explor Med*. 2022;3(1):58-70. [CrossRef]
11. Huang Y, Chen H, Liu Q, et al. Obesity difference on association blood malondialdehyde level and diastolic hypertension in the elderly population: a cross-sectional analysis. *Eur J Med Res*. 2023;28. [CrossRef]
12. Monserrat-Mesquida M, Quetglas-Llabrés M, Capó X, et al. Metabolic syndrome is associated with oxidative stress and proinflammatory state. *Antioxidants*. 2020;9(3):236. [CrossRef]
13. Zhang M, Liu N, Liu H. Determination of the Total Mass of Antioxidant Substances and Antioxidant Capacity per Unit Mass in Serum Using Redox Titration. *Bioinorg. Chem. Appl*. 2014;2014(1):928595. [CrossRef]
14. Cota-Magaña AI, Vazquez-Moreno M, Rocha-Aguado A, et al. Obesity Is Associated with Oxidative Stress Markers and Antioxidant Enzyme Activity in Mexican Children. *Antioxidants*. 2024;13(4). [CrossRef]
15. Vincent HK, Innes KE, Vincent KR. Oxidative stress and potential interventions to reduce oxidative stress in overweight and obesity. *Diabetes Obes Metab*. 2007;9(6):813-839. [CrossRef]
16. Jomova K, Raptova R, Alomar S, et al. Reactive oxygen species, toxicity, oxidative stress, and antioxidants: chronic diseases and aging. *Arch Toxicol*. 2023;97:1-76. [CrossRef]