Time-Restricted Feeding and Its Impact on the Immune System's Efficiency in an Experimental Obesity Model by Modulating the Interplay Between Inflammation and Oxidative Stress

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Abstract

Obesity causes oxidative stress and persistent, low-grade inflammation through fat overloading, immune cell dysfunction (macrophage infiltration), and the release of pro-inflammatory cytokines (TNF-α, IL-10). Overproduction of reactive oxygen species (ROS) generates oxidative stress, which in turn activates the immune system, which in turn promotes insulin resistance and metabolic disorders, creating a vicious cycle. The research involved 36 male albino rats that were 3 months old and weighed 150 to 200 g. In order to reduce biological variability, the animals were chosen for their similarity in age and weight. Body weight, hormone, and oxidative stress markers were the targets of this 28-day biochemical experiment that aimed to determine the impact of different diet types and time-restricted eating regimens, with different caloric intake in the morning and evening. A constant 25 g of food was given to each rat every day. The daily ration in the time-restricted groups was split into two portions, 6.25 g for 25% of the participants and 18.75 g for 75%, as per the group design. All three measures demonstrated significant differences between the groups ($p<0.05$), indicating that fast food and the timing of its consumption both impact the level of inflammation in the body. Acute systemic inflammation may have developed in groups B, B1, and B2 due to the high-calorie meals' impact on CRP levels. This inflammation may have been caused by an increase in oxidative stress and the buildup of saturated fats, which are known to activate the synthesis of inflammatory cytokines in the liver. In comparison to the other groups, group B had a significantly higher TNF-α level (11.23±2.34 pg/mL). In contrast, B1 and B2 had a progressive decline in levels (7.41±2.11) and (8.32±1.94), respectively, while the control groups A2-A had the lowest levels (5-6 pg/mL). Group B had the highest results at 12.13±2.61 pg/mL, followed by B2 at 10.25±2.33 pg/mL,

and B1 at 8.08±1.83 pg/mL. Values in the control groups ranged from 3.7 to 4.2 pg/mL, which is the lowest. All oxidative stress indices showed significant variations ($p<0.05$) across groups, which shows that different types of food and when they are eaten have different effects on the body's oxidative balance. The GSH levels in the high-fat groups, especially B2 (0.30±0.01) and B3 (0.34±0.02), were noticeably lower than in the control group A (0.79±0.06). Reducing glutathione (GSH) levels is an indicator of antioxidant system depletion caused by persistent oxidative stress from accumulated oxidized lipids, and GSH is a crucial cellular defense mechanism against free radicals.

Keywords: Obesity Model, Inflammation, Oxidative Stress, Immune System, Time-Restricted Feeding.

Introduction

Many different factors, including those at the social, behavioral, psychological, metabolic, cellular, and molecular levels, interact to cause obesity, a chronic disease with multiple causes. It is the state of having an excess of adipose tissue, which can be described as a rise in body weight leading to the accumulation of fat [1-3]. In developed nations in particular, obesity has emerged as a major public health concern in recent decades [4]. Death rates, as well as those from cardiovascular disease, diabetes, and colon cancer, are all impacted by obesity [5]. A large body of research has shown that being overweight or obese increases the risk of developing multiple chronic diseases, which in turn increases the risk of death. These diseases include type 2 diabetes, cardiovascular disease, cancer, and many more. The associated medical expenditures are likewise considerable. The development of population-based initiatives to prevent excess weight gain requires a public health strategy. Nevertheless, the increasing incidence of obesity has not been adequately addressed by public health intervention efforts. Current public health strategies for reducing risk factors and preventing obesity are evaluated critically in this paper, which also examines the definitions of overweight and obesity, how they vary by age and ethnicity, the health effects of obesity, and the factors that contribute to its development.[6]

Since reactive oxygen species (ROS) exist both in health and in illness, causing indirect or direct harm to various organs, it follows that oxidative stress (OS) plays a role in pathological processes such diabetes, obesity, heart disease, and atherogenicity. Some research suggests that obesity can trigger systemic OS, which in turn can lead to an imbalance in the body's adipokine production and the onset of metabolic syndrome [7]. Obese people have a higher

sensitivity to CRP and other oxidative damage biomarkers, which correlates with body mass index (BMI), percentage of fat, LDL oxidation, and TG levels; on the other hand, antioxidant defense markers are lower as a function of central obesity and amount of body fat [8]. People who are overweight are more likely to have OS stress and inflammation, according to study [9], because their diet is heavy in carbs and lipids. A single study found a statistically significant correlation between body mass index (BMI) and F2-IsoP concentration. Furthermore, dietary parameters were examined, and it was noted that the level of lipid peroxidation is inversely related to fruit consumption. This same study also found that compared to males, females had a higher peroxidation level, which could be due to the higher fat percentage in females. Additionally, we discovered that plasma cholesterol concentration was positively correlated with lipid peroxidation level [10]. Over time, being overweight can lead to a decrease in the activity of enzymes like catalase (CAT) and superoxide dismutase (SOD), which deplete antioxidant supplies. Concerning the potential for obesity-related health complications, the activity of glutathione peroxidase (GPx) and superoxide dismutase (SOD) is much reduced in obese individuals compared to healthy individuals [11]. Vitamin A, which is fat-soluble and has antioxidant properties, is likely diluted in rats with obesity due to their high liver lipid storage, according to a rat study that found a markedly lower concentration of this vitamin in the livers of obese rats compared to rats without obesity. Obesity is associated with reduced levels of glutathione and other antioxidants in the blood, including vitamin A, vitamin E, vitamin C, and β-carotene [12]. It has been suggested that antioxidants or ROS inhibitors may be able to reestablish the control of adipokines, as ROS reduce adiponectin expression. Hence, using antioxidant supplements might lessen the likelihood of problems associated with obesity and OS. There is a correlation between obesity, insulin resistance, endothelial dysfunction, and inflammation, all of which are symptoms of elevated OS. This cascade of metabolic and circulatory changes may be the result of interplaying and amplifying changes. The production of adipokines and acute phase proteins by adipose tissue may be triggered by hypoxia. When fat cells proliferate excessively in obesity, it leads to a lack of oxygen in the blood. People who are overweight may have reduced systemic inflammation due to the fact that adipose tissue, which accounts for 25% of systemic IL-6, is located in their bodies. It has been noted that systemic inflammation may be reflected in the rise of substances released by adipocytes, and the overall evidence suggests that fat cells have a capacity equal to or greater than inflammatory cells, especially when compared with macrophages [13]. According to Nishimura et al. [14], macrophage recruitment and activation are enhanced when CD8(+) T-cells in obese adipose tissue are

activated. Based on these findings, CD8 (+) T-cells are crucial for the development and maintenance of adipose inflammation. Researchers Cani et al. [15] found that altering gut microbiota in mice fed a high-fat diet causes a rise in intestinal permeability, which in turn leads to metabolic endotoxemia, inflammation, and related diseases.

Materials and Methods

Animals Used in Experiments

The research involved 36 male albino rats that were 3 months old and weighed 150 to 200 g. In order to reduce biological variability, the animals were chosen for their similarity in age and weight. The rats were kept in individual cages to avoid any potential for competing for food and to guarantee precise food intake measurements. They were also given full access to clean water for the whole experiment. Typical settings for animal housing were a temperature range of 22–25°C and a 12-hour light/12-hour dark cycle. All of the animals were given their regular food and drink for 7 days to help them adjust before the experimental treatments began. Animals were housed, fed, anesthetized, sampled, and sacrificed according to all protocols .

Experimental Animals

Body weight, hormone, and oxidative stress markers were the targets of this 28-day biochemical experiment that aimed to determine the impact of different diet types and time-restricted eating regimens, with different caloric intake in the morning and evening. Six groups of six rats each were randomly assigned to the animals after the acclimatization phase. The assignment was carried out in the following manner:

First: Standard Food Groups

- Control Group A: Standard food without time restrictions.
- A1 Group: Standard food with time restrictions, distributing calories 25% in the morning and 75% in the evening.
- A2 Group: Standard food with time restrictions, distributing calories 75% in the morning and 25% in the evening.

Second: High-Calorie Food Groups

- B Group: High-calorie food without time restrictions.
- B1 Group: High-calorie food with time restrictions, distributing calories 25% in the morning and 75% in the evening.
- B2 Group: High-calorie food with time restrictions, distributing calories 75% in the morning and 25% in the evening.

Timing of Meals and Feeding

A constant 25 g of food was given to each rat every day. The daily ration in the time-restricted groups was split into two portions, 6.25 g for 25% of the participants and 18.75 g for 75%, as per the group design. Both group A and group B were not timed, so participants may eat whenever they wanted. To standardize feeding schedules and decrease circadian rhythm variances, meals were supplied twice daily at 10:00 AM and 10:00 PM to the time-restricted groups A1, A2, B1, and B2.

Making Foods Rich in Calories

The calorically dense meal was made using a uniform combination of burgers, French fries, and crispy chicken in equal parts, and then distributed to each group at the predetermined intervals.

Burger Making Methods

We used 850 grams of lean ground beef, 150 grams of beef fat, 15 grams of salt, 10 grams of minced fresh garlic, and 10 grams of hamburger spice per kilogram of combination to make the burger patties. Using a 4 mm fines grinder, the beef was ground at a temperature below 4°C. The materials were thereafter blended thoroughly before being shaped into patties that were 80-100 g in weight and 1-1.5 cm thick. As per the usual procedure for burger patties, the patties were cooked at a temperature range of 180 to 200 degrees Celsius until they reached an internal temperature of 72 degrees Celsius.

Making Chicken with a Crunch

Fry the fresh chicken pieces in hot vegetable oil at 170-180°C until they are golden brown and crispy, making sure the internal temperature is 75°C. The coating should be a dry mixture of wheat flour, rice flour, maize flour, and seasoned flour.

Methods for Making Potato Fingers

Uniformly sized fresh potatoes were used to make potato fingers. They scrubbed, peeled, and diced the potatoes into fingers of consistent size. Before being prepared for frying, they were rinsed to remove any excess starch. The next step was to dry them, add salt, and then fry them in sunflower oil in an electric fryer until they were crispy and golden. Regasa et al. (2025) detailed a similar methodology for a laboratory setting; however, this method was somewhat adjusted to fit the needs of the actual investigation.

Weight and Feed Intake Measurements

Starting at the start of the experiment and continuing weekly, an electronic digital scale was used to measure the body weight of each animal. Each measurement was conducted at the same time each day to account for the potential influence of circadian rhythms. A weight increase calculation was performed using the following formula:

Weight Gain = Final Weight − Initial Weight

Injectable Medications with Blood Drawn

In accordance with established protocols for the anesthesia of rodents, mice were induced to fasting by administering a mixture of 10% ketamine and 2% xylazine. Following the confirmation of deep anesthesia, typical protocols for rodents were followed to extract blood samples: EDTA-containing tubes for plasma and anticoagulant-free tubes for serum. The samples were collected using sterile syringes.

Separation of Plasma and Serum and Preservation of Samples

By centrifuging at 3000 rpm for 10 minutes at room temperature, the serum and plasma were separated. The samples were thereafter placed in Eppendorf tubes and kept in the refrigerator until they were analyzed for chemicals and hormones, in accordance with established laboratory protocols.

Markers of Inflammation and Immunology

Following the manufacturer's instructions and recent implementations of this approach, the concentration of C-reactive protein (CRP) in serum was determined using an AFIAS

equipment that relies on fluorescence immunoassay technology. We used a rat ELISA kit from Cusabio Biotech Co., Ltd., China, to estimate the TNF-α concentration, and we used a Rat IL-10 ELISA Kit from Elabscience, China, to measure IL-10. We followed the sandwich ELISA concept and the instructions provided by the suppliers. Standard curves were used to compute concentrations, and absorbance was measured at 450 nm. Serum samples were diluted 1:500 before measurement to estimate IgA and IgG immunoglobulins using an ELISA assay kit from Bioengineering Co. Ltd., China, in accordance with the procedure approved by Xie et al., 2025. Using a full blood count with differential analysis, an automated hematologist based on electrical impedance and/or laser flow cytometry determined the total white blood cell count, lymphocyte number and percentage, and other parameters.

Symptoms of Free Radical Damage

We followed the manufacturer's instructions to quantify the quantity of catalase (CAT) in serum using a rat ELISA kit from SunLong Biotech Co., Ltd., China. We used a modified procedure that involved reacting ethylmen reagent (DTNB) with sulfhydryl groups and measuring absorbance at 412 nm to quantify reduced glutathione (GSH). Using a diagnostic kit from SunLong Biotech (China), we evaluated the amounts of superoxide dismutase (SOD) and malondialdehyde (MDA). We followed the manufacturer's instructions and used standard curves to get the values.

Data Analysis by Statistic

A totally random design (CRD) was used for statistical analysis of the data. We used Duncan's multiple range test to look for differences in the means. A level of significance of $P < 0.05$ was determined. We used SAS 2012 software to conduct all of our analyses.

Results and Discussion

A significant biomarker reflecting the systemic inflammatory and immunological response due to changes in the timing and type of food intake is shown in Table (1). All three measures demonstrated significant differences between the groups ($p<0.05$), indicating that fast food and the timing of its consumption both impact the level of inflammation in the body.

Table 1. Inflammation Factors

Test	CRP mg/L	TNF-a pg/ml	IL-10 pg/ml
A	0.552	5.10	4.280
A1	0.591	5.92	3.790
A2	0.875	6.28	3.760
B	3.023	11.23	12.130
B1	1.314	7.41	8.080
B2	2.820	8.32	10.258

CRP: C-reactive protein; TNF-a: Tumur necrosis Factor-alpha; IL-10: Inter leukin-10

C-Reactive Protein (CRP) mg/L

The most significant result was obtained by Group B (3.023±0.81 mg/L), followed by Group B2 (2.820±0.94 mg/L), while the groups A and A1 had the lowest values (0.552±0.06 mg/L and 0.591±0.07 mg/L, respectively). Acute systemic inflammation may have developed in groups B, B1, and B2 due to the high-calorie meals' impact on CRP levels. This inflammation may have been caused by an increase in oxidative stress and the buildup of saturated fats, which are known to activate the synthesis of inflammatory cytokines in the liver. These findings align with the research conducted by Huh et al. (2022), which proved that CRP is considerably increased in the liver and intestines as a result of a high-fat diet, as a result of the activation of the NF-κB inflammatory pathway. Group B1 had a significantly lower C-reactive protein level (1.314±0.53 mg/L) than groups B and B2, suggesting that morning feeding mitigated the inflammatory impact of high-calorie meals. As a result of enhanced mitochondrial circadian rhythm and lower oxidative stress, Sutton et al. (2018) found that early time-restricted meals reduces inflammatory markers, including CRP and IL-6, even without weight change. When comparing healthy individuals to those with anorexia nervosa, Xu et al. (2024) found that C-reactive protein (CRP) levels were significantly lower in the former group. This decline, which is consistent with the lack of a typical systemic inflammatory response in this illness, is probably the result of physiological adjustments brought about by acute starvation and long-term decreases in metabolic energy and body mass. Total dietary antioxidant capacity (DAC) is inversely related to CRP levels, according to Wang et al. (2023), and CRP is the principal mediator of some of the antioxidants'

protective effects. Because antioxidants suppress low-grade inflammation, especially when consumed in synchronization with the circadian rhythm, it follows that the time-balanced diet group (A1/B1) had lower CRP levels than the high-calorie evening diet group (B2).

Tumor Necrosis Factor-α (TNF-α pg/mL)

In comparison to the other groups, group B had a significantly higher TNF-α level (11.23±2.34 pg/mL). In contrast, B1 and B2 had a progressive decline in levels (7.41±2.11) and (8.32±1.94), respectively, while the control groups A2-A had the lowest levels (5-6 pg/mL). The inflammatory cytokine TNF-α is released into the bloodstream by phagocytic cells when the amount of saturated fat in the diet is high. Insulin resistance is ramped up and the inflammatory process (NF-κB pathway) is activated. In line with these findings, a study conducted by Lee et al. (2023) proved that insulin resistance develops in the liver and muscles as a result of high-fat diets, which increase TNF-α. The fact that B1 was significantly lower than B and B2 demonstrates that eating breakfast helps lower inflammation through influencing cytokine circadian rhythms and increasing insulin sensitivity. According to Chaix et al. (2021), the immune system remains balanced and TNF-α production is reduced through circadian diet. After excluding outliers or studies with high variance, the combined findings from various studies suggest that tumor necrosis factor-alpha (TNF-α) may exhibit a small increase in individuals with anorexia nervosa, but this increase does not retain statistical significance (Dalton et al., 2018; Nagata et al., 2006). The findings also indicate that patients with bulimia nervosa may initially have higher levels of TNF-α than the control groups, but this disparity decreases if confounding factors are taken into account. When these results are considered as a whole, they show that alterations in pro-inflammatory cytokines in eating disorders are localized or selective, rather than reflecting a systemic inflammatory pattern, and that this is indicative of specialized rather than universal immune responses. According to Azemi et al. (2025), controlling when you eat helps lower levels of inflammatory cytokines, especially TNF-α. This suggests that obesity-related chronic low-grade inflammation is reduced and immunological homeostasis is improved.

Interleukin-10 (IL-10 pg/mL)

Group B had the highest results at 12.13±2.61 pg/mL, followed by B2 at 10.25±2.33 pg/mL, and B1 at 8.08±1.83 pg/mL. Values in the control groups ranged from 3.7 to 4.2 pg/mL, which is the lowest. In this case, the body is trying to mitigate the inflammatory damage produced by raised TNF-α and CRP levels by elevating IL-10, despite the fact that it is

normally thought of as an anti-inflammatory cytokine. In a study conducted by Acosta et al. (2019), it was proven that increasing IL-10 levels in individuals following high-fat diets do not represent immunological homeostasis, but rather excessive immune system activity aimed at reducing chronic inflammation. It appears that morning feeding reduced inflammation and the requirement for compensatory IL-10 activation, as Group B1 exhibited an intermediate IL-10 level between the low and high groups. The results of this study are in line with those of Guo et al. (2023), who discovered that time-restricted feeding changes the balance of cytokines by balancing the levels of TNF-α and IL-10, which keeps the immune system in a steady state. This study adds to the growing body of evidence linking disruptions in lipid metabolism to systemic inflammatory pathways by showing that altered blood lipid profiles are physiologically correlated with increased inflammatory markers. Groups B and B2 showed higher amounts of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein (LDL), as well as notable spikes in CRP and TNF-α, suggesting a two-way activation of the inflammatory response and hyperlipidemia. According to research by Huh et al. (2022) and Chen et al. (2022), the NF-κB inflammatory pathway, which is in charge of producing CRP and TNF-α, is stimulated when saturated fats build up in the liver and tissues, activating Toll-like receptor 4 (TLR4) on the surface of phagocytic and hepatocytes. A negative feedback loop between inflammation and impaired lipid metabolism is created when this persistent activation leads to decreased insulin sensitivity and increased triglyceride excretion in the blood (Caputo et al., 2017).

In group B1, subjects were given high-fat breakfasts (75% in the morning and 25% in the evening) and showed considerable improvements in inflammatory markers (lower CRP, TNF-α, and IL-10) and lipid markers (lower TC, LDL, and TG, with higher HDL) compared to the other groups. Better fat metabolism and less buildup of inflammatory oxidation byproducts are the results of eating first thing in the morning, which happens to be when the body's metabolic rates are highest (Chaix et al., 2021; Sutton et al., 2020; Manoogian and Panda, 2022). Research conducted by Guo et al. (2023) and Xie et al. (2023) examined the molecular effects of early time-restricted feeding. The results demonstrated that this feeding pattern resets genes related to lipid metabolism, like PPAR-α and SREBP-1c, and decreases inflammatory NF-κB activity. As a result, there is less inflammation in the liver and intestinal tract, and the health of lipids is improved.

In addition, research conducted by Yin et al. (2022), Huh et al. (2022), and Guo et al. (2023) has shown that dietary changes that are in sync with the circadian rhythm lead to a reduction in the compensatory IL-10 response, which in turn restores the normal state of immunological

homeostasis. Chrononutrition promotes efficient lipid metabolism and reduces chronic inflammation generated by high-energy meals, as seen by the parallel improvement in lipid and inflammatory markers in group B1. Nevertheless, similar to group B2, eating in the evening threw this equilibrium out of whack, leading to higher levels of inflammation and harmful lipids since eating at that time interferes with the circadian clock that controls metabolism. Keeler et al. (2025) conducted a thorough meta-analysis that linked anorexia nervosa and other eating disorders to altered patterns of inflammatory cytokines. Anorexia nervosa patients showed a differentially altered immune response, with increased interleukin-6 and IL-15 levels and decreased interleukin-7 levels compared to control subjects. In addition, after accounting for outliers, the results demonstrated that there were no notable variations in the levels of tumor necrosis factor (TNF)-α, IL-1β, IL-4, IL-8, IL-10, interferon (IFN)-γ, monocyte chemoattractant protein (MCP), and transforming growth factor (TGF)-β between the healthy individuals and patients with anorexia nervosa.

According to Keeler et al. (2025), who looked at immunological changes through time, IL-6 levels were lower in anorexia nervosa patients at follow-up than they were at baseline. Instead of a real improvement in inflammatory activity, this drop can be due to publication bias. Over time, no increases in TNF-α and IL-1β concentrations were noted that were statistically significant. The results demonstrated that there were no notable variations in IL-6 and TNF-α levels between the control groups and those with bulimia nervosa (BN). Unfortunately, a trustworthy meta-analysis for binge eating disorder or any other eating disorder was not possible due to the small number of research that were available. There was no statistically significant rise in other pro-inflammatory cytokines, however the study did find that IL-7 is lowered and IL-6 and IL-15 are enhanced in severe anorexia nervosa. These results point to a selective dysregulation of cytokines rather than a normal or universal immune response in eating disorders. Further longitudinal and comparative research are needed to better understand the immunological and inflammatory involvement in various eating disorders, especially bulimia nervosa, where there is still a lack of sufficient evidence. Researchers have found that people with bulimia nervosa, binge eating disorder, and even some cases of obesity without eating disorders have significantly lower levels of the anti-inflammatory cytokine interleukin-10 (IL-10). This finding is supported by studies conducted by Tabasi et al. (2020) and Caldas et al. (2022). This indicates that these groups may experience immune dysfunction due to impaired anti-inflammatory immune function, as indicated by low IL-10 levels. In contrast, anorexia nervosa shows a pattern more similar to inflammatory inactivity, caused by low CRP and stable or moderate TNF-α levels. prior

investigations, including the one conducted by Dalton et al. (2018), found slightly higher levels of TNF-α and IL-6 in people with anorexia nervosa compared to healthy individuals. These new data provide a thorough update of those prior analyses.

The study conducted by Permataputri et al. (2025) examined the impact of time-restricted fasting on specific immunological markers in Asian women who are obese. The results demonstrated that the group undergoing time-restricted fasting had significantly lower levels of interferon-γ (IFN-γ), whereas neither the control nor the alternate-day modified fasting groups showed any notable changes. No statistically significant change was noted in any of the groups regarding interleukin-10 (IL-10) levels following the intervention. Conclusions Time-restricted fasting may help lower pro-inflammatory cytokines but has no discernible impact on anti-inflammatory cytokines, according to these findings. The drop in IFN-γ was seen by the researchers as a natural physiological reaction to the reduction of obesity-related chronic low-grade inflammation. This finding supports the idea that fasting can bring about immune homeostasis by lowering levels of pro-inflammatory cytokines. This aligns with the findings of Huang et al. (2022), who explained that IFN-γ and other members of the interferon family are crucial in controlling insulin sensitivity and inflammatory reactions in fat tissue. On the flip side, the study's IL-10 levels remained rather constant, which could be due to the brief intervention period (20 days) or the fact that there was no discernible shift in fat mass. Keep in mind that IL-10's impact on obesity might be more nuanced than previously thought. Research by Saxton et al. (2021), Minshawi et al. (2020), and Acosta et al. (2019) among others has demonstrated that IL-10 can regulate the inflammatory response and insulin resistance in human adipose tissue, among other possible dual functions of the protein depending on the cellular environment and the type of its receptors. Thus, the research came to the conclusion that during brief dietary interventions, time-limited fasting could decrease levels of pro-inflammatory cytokines like IFN-γ while maintaining or even boosting levels of anti-inflammatory cytokines like IL-10. Although several inflammatory cytokines were found to be decreased, the results of Permataputri et al. (2025) show that IL-10 levels remained relatively unchanged. Some studies, like the one involving IFN-γ, suggest that controlling the schedule of eating mainly reduces inflammation by reducing inflammatory cytokines, rather than necessitating an increase in IL-10 to compensate. The overall inflammatory state is improved through the suppression of primary inflammatory signaling, as indicated by the stability of IL-10.

Table 2. Oxidation Indices

Test	Gat lu/L	GSH lu/L	SOD lu/L	MDA mmol/L
A	1.45	0.79	58.14	2.34
A1	1.61	0.71	55.25	2.12
A2	1.40	0.82	53.63	2.79
B	0.98	0.34	40.46	4.24
B1	1.34	0.38	47.78	3.92
B2	0.92	0.30	45.24	4.08

MDA: Malondialdehyde; SOD: Superoxide dismutase; GSH: Glutathione; Cat: Catalase

Groups differ significantly ($p<0.05$) in all oxidative stress indices, as shown in the table. This reflects the fact that different types of food have different impacts on the body's oxidative balance, as do the time and timing of consumption. Reducing oxidative damage associated with obesity and excessive fat consumption, Azemi et al. (2025) showed that time-restricted eating improved oxidative-reductive balance, as evidenced by lower MDA and enhanced SOD activity.

Catalase enzyme (CAT lu/L)

The results demonstrated that groups A1 (1.61±0.60) and A (1.45±0.31) had the highest catalase enzyme activity compared to the other groups, while groups B, B1, and B2, which were given high-calorie meals, had significantly lower activity, particularly in (0.98±0.13) and (0.92±0.08) B. Catalase is an essential enzyme in the body that converts hydrogen peroxide (H_2O_2) into oxygen and water, a process that keeps harmful free radicals from building up. Catalase activity reduction in B and B2 suggests elevated oxidative stress due to fried meals and saturated fats, which produce lipid oxidation products that add to the buildup of free radicals.

These findings corroborate previous research (Anderson et al., 2009; Yasmin et al., 2025) that demonstrated elevated levels of malondialdehyde (MDA) and reactive oxygen species (ROS) in the livers of mice given a high-fat diet. Additionally, the study found that the activity of antioxidant enzymes such as glutathione peroxidase (GPx), superoxide dismutase (SOD), and catalase (CAT) was significantly reduced. A compromised antioxidant defense mechanism as

a result of elevated reactive oxygen species (ROS) production is suggested by the significant decline in catalase and superoxide dismutase activity observed in the high-fat diet group. On the flip side, the A1 group's increased morning-restricted feeding is a result of circadian-rhythm-synchronized antioxidant pathway activation. Researchers Sutton et al. (2020) and Guo et al. (2023) found that when babies are fed early on, their livers produce more antioxidant enzymes.

Reduced Glutathione (GSH lu/L)

The GSH levels in the high-fat groups, especially B2 (0.30±0.01) and B3 (0.34±0.02), were noticeably lower than in the control group A (0.79±0.06). Reducing glutathione (GSH) levels is an indicator of antioxidant system depletion caused by persistent oxidative stress from accumulated oxidized lipids, and GSH is a crucial cellular defense mechanism against free radicals. Two studies (Yasmin et al., 2025; Wang et al., 2024) found that high-fat diets hinder glutathione reductase activity, which in turn hinders the GSH regeneration cycle. As a result, oxidative damage in cells of the liver and the intestines is enhanced. In comparison to groups B and B2, Group B1 had a relative improvement of 0.38 ± 0.04, indicating that reducing glutathione depletion by synchronizing metabolism with daily mitochondrial activity was improved by feeding in the morning. In their study, Xie et al. (2024) showed that when food intake is limited during the day, the Nrf2-ARE pathway is activated. This activation causes an increase in the gene expression of glutathione-related enzymes (such as GCL, GR, GPx, and catalase) during the daytime activity phase, thereby coordinating the antioxidant responses in the liver and intestine.

Superoxide Dismutase (SOD) enzyme

Group A of the control group had the highest values (58.14±6.61), followed by A1 (55.25±7.17) and A2 (53.63 ± 8.09), whereas groups B (40.46±4.52) and B2 (45.24±6.07) showed a significant decline. Decreased levels of superoxide dismutase (SOD), an enzyme crucial for neutralizing O_2^- free radicals, suggest that cells are less able to withstand oxidation brought on by saturated fats. This confirms the findings of Huang et al. (2022) and Wang et al. (2023), which showed that oxidized lipids block the expression of genes responsible for SOD and GSH. Our results show that eating fast food reduces the activity of defense enzymes. According to the hypothesis, lowering oxidative stress even with a high caloric intake can be achieved by aligning nutrition with the activity phase of the day, as seen by the relative improvement in B1 (47.78 ± 5.11) compared to B and B2.

Malon dialdehyde (MDA mmol/L)

There was a notable rise in MDA in the fast food categories (B, B1, and B2), with B2 (4.08±1.38) and B (4.24±1.07) having the highest values, and A1 (2.12±0.89) having the lowest. A rise in malondialdehyde (MDA), a byproduct of lipid peroxidation, is indicative of free radical accumulation-induced damage to cell membranes. These findings corroborate those of research by Deng et al. (2025) and Feng et al. (2024), which showed that a high-fat diet, especially when eaten late at night, increases MDA and decreases antioxidant defenses. Group A1 has lower MDA levels because their antioxidant activity is higher in the morning, when the expression of defense enzyme genes is highest. Consistent with previous research, this suggests that limiting breakfast food intake lowers lipid peroxidation and maintains healthy liver cells (Chaix et al., 2021; Manoogian and Panda, 2022).

Based on the study's findings, there is a direct link between oxidative stress markers and systemic inflammation markers when looking at the physiological relationship between the two. In the fast-food groups, especially B and B2, there was a decline in cellular antioxidants (CAT, GSH, SOD) and an increase in malondialdehyde (MDA), which occurred at the same time as there was a significant rise in CRP and TNF-α and a corresponding increase in IL-10. The physiological process begins when reactive oxygen species (ROS) build up and trigger the NF-κB inflammatory pathway, leading to an upregulation of cytokines (TNF-α) and acute-phase proteins (CRP). At the same time, the glutathione system is depleted and defense enzymes (SOD and CAT) are inhibited. According to Huang (2022), Huh (2022), and Anderson et al. (2009), this further reinforces the oxidation-inflammation feedback loop. Xie et al. (2024), Sutton et al. (2020), Guo et al. (2023), and the B1 group all found that morning feeding led to relative improvements in health. This was despite the fact that the B1 group still ate high-fat meals. Specifically, there was a decrease in malondialdehyde (MDA) and an increase in catalase, peroxidase, and glutathione (CAT/SOD/GSH) activity, which in turn reduced CRP/TNF-α levels and eliminated the need for compensatory IL-10 elevation. This improvement was likely caused by the activation of the Nrf2-ARE pathway, improved mitochondrial efficiency, and synchronization of metabolism with the circadian rhythm, which limits ROS and reduces NF-κB activation. Therefore, the present findings lend credence to the idea of circadian nutrition as a means to mitigate chronic inflammation and oxidative stress, even when a high dietary fat load is present (Li et al., 2021; Nah et al., 2024).

Dietary total antioxidant capacity (TAC) is inversely related to the risk of all-cause and cardiovascular mortality, according to the study by Wang et al. (2023) that used data from the US National Health and Nutrition Examination Survey (NHANES) from 1999 to 2018. The results showed that compared to the lowest group, participants in the highest 20% of total TAC had a significantly lower risk of mortality from cardiovascular disease and all causes. Increasing consumption of antioxidant-rich foods at dinner was linked to a substantial decrease in all-cause mortality rates, but this association was not detected at breakfast or lunch, according to analysis of the temporal distribution of antioxidant intake throughout the day. Moreover, there was an inverse association between the risk of all-cause mortality and the difference between supper and breakfast TAC (ΔTAC), lending credence to the idea that when antioxidants are consumed might be more significant than their overall amount. In the link between TAC and mortality risk, C-reactive protein (CRP) is a partial mediator, accounting for around 24% of the total effect, according to mediation analysis.

Researchers talked about how these results show how important it is to take antioxidants at the same times each day to keep metabolic and inflammatory processes in harmony. Consuming antioxidant-rich meals in the evening is more effective in neutralizing free radicals and improving metabolic balance since oxidative stress levels and inflammatory pathways are at their height in the evening. The study's findings are in line with those of other studies that have looked at how eating at specific times affects metabolic responses. For example, Dashti et al. (2019) found that improving metabolic and heart health indicators was associated with distributing dietary energy according to the biological clock. Thus, Wang et al. (2023) drew the conclusion that consuming antioxidants at the right time of day, especially at dinner, may help lower the risk of death from cardiovascular disease and other causes, and they urged researchers to conduct longer-term studies to determine the exact physiological mechanisms at work.

The distribution of dietary antioxidant capacity (DAC) among daily meals is critical in improving general health and reducing mortality risk, according to multiple research (Bastide et al., 2017; Sheng et al., 2022; Beydoun et al., 2022). Compared to breakfast and lunch, the data demonstrated that greater total DAC levels, especially at dinner, were linked to decreased rates of general and cardiovascular mortality. In addition, these studies showed that the timing of meals affects the health benefits. For example, eating antioxidants in the evening was linked to better inflammatory markers and lower CRP levels, lending credence to the idea that timing plays a role in how effective an antioxidant diet is. Because DACs

have a preventive impact against oxidative stress and age-related diseases, these results support earlier findings that DAC-rich evening meals are more beneficial for younger age groups, smokers, and those with more earnings. Vegetables, rather than grains, fats, or sweets, are the food source most strongly linked with a decreased risk of overall mortality, according to research by Han et al. (2020), Chen F et al. (2019), and Jakubowicz et al. (2015). The relevance of the dinner's quality and content in achieving health advantages is highlighted by the fact that drinking high-calorie beverages throughout the meal was linked to an elevated risk of overall and cancer-related mortality. This protective impact may be explained in part by the balanced nutritional profile and low calorie content of veggies, according to these findings.

A number of physiological studies have demonstrated that antioxidants help reduce inflammation and oxidative stress by controlling the body's circadian rhythm, which in turn controls the diurnal rhythms of inflammatory processes (Reitz et al., 2021; Valtuena et al., 2008; Dohi et al., 2007; Das M et al., 2021; Lewis et al., 2020). This theory lends credence to the idea that DAC-rich meals are best consumed in the evening, when inflammatory activity is at its highest, which may help mitigate the harmful effects of oxidative stress and even reduce death rates. In terms of methodology, this study is unique among its peers since it uses the DAC index and meal time together to examine the association with total mortality. Because of its rigorous methods for testing dietary antioxidant capacity and its large-scale database (NHANES), it was a success. Due to the data's reliance on short-term dietary recalls and the absence of direct biometric measures of blood DAC levels, some research (Reedy et al., 2018; van Buuren and Groothuis-Oudshoorn, 2011) advise caution in interpretation. These findings support the idea that future dietary guidelines should include information about when to eat foods high in antioxidants because of the importance of maintaining a healthy balance between meal timing and antioxidant content in enhancing general health and reducing death rates.

Table 3. White Blood Cell Count and Immunoglobulins

%Lym From WBC	WBC 102/mml	IgA mg/dL	IgG mg/dL	Test
67.31	8.32	192.3	1241	A
66.89	8.94	219.3	1237	A1
67.21	9.21	210.2	1198	A2

72.50	19.20	120.4	978	B
71.41	14.37	150.8	1130	B1
72.32	18.28	135.9	995	B2

Food quality and feeding timing have a dual effect on the activities of the humoral and cellular immune systems, as demonstrated by the results, which demonstrated evident and significant differences ($p < 0.05$) among the various groups in all the indicators examined.

Immunoglobulin G (IgG mg/dL)

The most significant IgG levels were obtained by the control group A (1241±31.6) and group A1 (1237±28.6), with group A2 following at 1198±30.1. Groups B (978±21.9) and B2 (995±24.3) in particular demonstrated a notable decline compared to the other groups that were given high-calorie meals (B, B1, and B2). Reduced IgG levels in the fast food groups imply immune response suppression brought on by oxidative stress and chronic inflammation brought on by simple sugars and saturated fats, which are important markers of long-term humoral immunity. Researchers Wang et al. (2019) found that metabolic syndrome (MetS) is more common in people with either low or high IgG levels. By reducing the activity of $CD4^+$ helper cells and preventing B cell development, a high-fat diet reduces antibody production. Because of the enhanced metabolic state caused by a morning meal that is in sync with the circadian rhythm, the raised IgG levels in group A1 can be explained. He et al. (2023) and Manoogian et al. (2022) found that this improves immunity by controlling cortisol and bolstering antibody production. Complementary mechanisms, such as improved circadian rhythms, immune cell movement modulation, inflammation factor decrease, and increased gut microbiota diversity in healthy persons, are responsible for these effects. Additionally, there is some evidence that the cellular, metabolic, and rhythmic mechanisms of intermittent fasting may reduce physiological and hormonal stress, which in turn may have positive effects on the nervous system and the mind.

Immunoglobulin A (IgA mg/dL)

IgA levels were considerably lower in groups B (120.4±14.2 mg/dL) and B2 (135.9±12.9 mg/dL), whereas the highest values were seen in group A1 (219.3±12.9 mg/dL) and A2 (210.2±13.0 mg/dL). IgA is the first line of defense for mucosal immunity in the respiratory and gastrointestinal systems, and its decline indicates compromised intestinal mucosal integrity and a weaker local immune response. These findings imply that eating fast food in the evening disrupts the gut microbiota and lowers mucosal IgA secretion, which lowers resistance to infection. Saturated fats reduce IgA secretion by blocking the TGF-β/Smad3 pathway, which is in charge of the development of antibody-producing cells in the gut, according to research by Basson et al. (2021; Zhang et al., 2025). Group A1, on the other hand, had the largest IgA secretion, indicating that morning feeding enhances mucosal immunity because it coincides with the daily peak of melatonin and cortisol release, which control the activity of plasma cells that produce IgA (Guo et al., 2023; Zhang et al., 2025).

Total White Blood Cell Count (WBC × $10^2/mm^3$)

In comparison to the other groups, groups B (19.20±3.88) and B2 (18.28±2.91) had significantly higher white blood cell counts, while groups A (8.32±1.66) and A1 (8.94±2.17) had the lowest values. The rise in WBC counts in groups B and B2 is indicative of an active inflammatory response brought on by long-term immunological stimulation linked to diets high in oxidized lipids, which raises neutrophil and macrophage counts. According to Lee and Choi's (2023) study, meals heavy in saturated fat raise WBC counts via triggering inflammatory cytokines, especially TNF-α and IL-6, which result in chronic low-grade inflammation. On the other hand, the significant drop in WBCs within A1 suggests a physiological immune balance free from inflammatory overstimulation, which is in line with the results of Sutton (et al., 2020), who showed that feeding during the day decreases immune cell overproduction and rearranges their activity.

Lymphocyte percentage of white blood cells (%Lymphocytes)

The highest lymphocyte percentages were found in groups B (72.50±8.93) and B2 (72.32±10.6), whilst the lowest percentages were found in groups A (67.31±10.4) and A1 (66.89±9.50). Dietary factors, especially diets high in fats and sugars, are responsible for the raised lymphocyte percentage. These diets cause metabolic changes that produce a favorable immunological milieu for the body to try to regulate chronic inflammation caused by oxidative stress. However, according to Plata-Gómez et al. (2025), the majority of these cells are in a condition of overactivation or immunological exhaustion, which makes this increase

functionally worthless. The average percentage of Group B1 (71.41±11.7) tended toward immune balance, suggesting that the time of morning meals improved the effectiveness of the defense response and decreased immune system overactivation.

Zhang et al. (2025; Ma, 2024) showed that circadian rhythm diet balances immunity and inflammation via coordinating T and B lymphocyte development. According to the current findings, the disruption of the lipid profile caused by high-calorie meals was linked to a significant rise in inflammatory markers (CRP and TNF-α), a compensatory rise in IL-10, and a decline in the antioxidant system (increased MDA and decreased CAT, GSH, and SOD). This resulted in inefficient cellular overactivation (increased WBC and lymphocyte count) and compromised humoral and mucosal immunity (decreased IgG and IgA). Chronic elevations of reactive oxygen species (ROS) physiologically cause glutathione depletion, inhibition of serotonin-depleting enzymes (SOD/CAT), and activation of the NF-κB axis and inflammatory cytokines (TNF-α). This lowers mucosal IgA, which is necessary for intestinal barrier integrity, and creates an oxidative inflammatory milieu that hinders B cell development and antibody release (Ma, 2024; Lhoták, 2020).

The B1 group, on the other hand, demonstrated concomitant benefits, including a decrease in CRP, TNF-α, and MDA, a relative rise in CAT/SOD/GSH, a decrease in leukocyte hyperactivation, and a relative improvement in humoral/mucosal immunity, even though they continued to eat a high-fat diet. Time-restricted feeding in the morning (eTRF) limits ROS generation, lowers NF-κB activation, and restores cytokine balance, which is reflected in improved immune indicators even without changing the type of food. This is explained by the fact that eTRF coincides with the daytime peak of mitochondrial efficiency and the Nrf2-ARE pathway (Sutton et al., 2020; Park et al., 2023). When compared to evening meals with a higher oxidative burden, daytime meal timing improves insulin sensitivity, lowers low-grade inflammation, and increases mucosal immunity efficiency, according to recent clinical and preclinical evidence (Regmi and Heilbronn, 2022; Jamshed et al., 2019; Jamshed et al., 2022; Manoogian and Panda, 2022). Thus, even in the context of a high dietary fat load, our findings support the idea of chrono-nutrition as a regulatory strategy that rebalances the oxidation-inflammation-immunity axis.

Table 4. Average weight gain over 3 weeks.

B2	B1	B	A2	A1	A
226.6	248.5	236.7	170.5	204.5	227.6
Weight gain in 3 weeks			Weight gain in 3 weeks		

Weight gain over the four weeks of the experiment varied significantly (p 0.05) among the six groups, as seen in the table. This reflects the fact that the time and type of diet had a double impact on energy balance and metabolic pathways. From week one to week four, the control group (A) gained a consistent 227.6 kg, which indicates normal physiological growth and stable energy balance. According to physiological research, this pattern is caused by an equilibrium between the amount of energy consumed and the amount of energy used for protein repair and growth, all without an excess of fat storage. Dietary balance protects rats from metabolic obesity-causing overactivation of the mTOR pathway and keeps their feed conversion ratio normal, according to research by Bensalem et al.(2023) .

Compared to the control group, Group A1 gained a considerable amount of weight (204.5 kg), suggesting that the metabolic efficiency was improved by the time-restricted morning feeding. Eating breakfast improves insulin and blood glucose control and boosts fatty acid oxidation because it aligns with mitochondrial activity over the day, according to research. Gong et al. (2025) found that eating breakfast increases the expression of proteins involved in lipid oxidation and body temperature regulation, which in turn decreases the growth of adipose tissue even when calorie intake is constant .

A smaller weight gain of 170.5 was observed in Group A2. Due to reduced insulin sensitivity to glucose uptake and decreased overnight mitochondrial activity, this pattern is associated with poorer metabolic efficiency in the evening. Gong et al. (2025) and Garaulet et al. (2013) found that eating when the body isn't actively regulating its internal clock causes insulin resistance and visceral fat to accumulate (Garaulet et al., 2013). The nighttime groups showed more signs of moderate inflammation than the morning groups, and this helps to explain it.

The average total weight gain for the high-calorie, unrestricted (B) group was 236.7 kg, reaching a peak in the third week. Because of its high saturated and fried fat content, fast food has a negative impact on controlling hunger and maintaining a healthy energy balance.

Diets rich in oxidized lipids disrupt leptin and ghrelin signaling, which causes people to eat more even when they feel full, according to research by Cai et al. (2012) and Netam (2024). The hypothalamus's neuronal circuits that control hunger are disrupted when oxidized lipids raise ROS production. Researchers Holá et al. (2023) found that people with impaired glucose tolerance, elevated LEAP2 gene expression in the liver, and lower levels of active and total ghrelin were all associated with a high-fat diet. After going back to a regular diet, LEAP2 gene expression improved, and active ghrelin levels were normal again, although total ghrelin was still low. The effect of ghrelin, which is replicated by the stable analog Dpr3 Ghrelin, is blocked by a high-fat diet; however, this blockage can be reversed when one returns to a normal diet.

On average, group B1 gained 248.5 pounds, which is less than group B, even though this group ate more high-energy fast food (75% in the morning and 25% in the evening). The synchronization of feeding with daytime exercise improved the weight gain pattern comparing to other approaches by maximizing energy usage for growth rather than fat storage. The CD36 protein appears to have a dual regulatory function in lipid metabolism, according to recent research (Li et al., 2019; Wang et al., 2025). It can prevent lipid droplet autolysis through the AMPK-dependent route, and it keeps hepatocyte metabolism in check by activating AMPK via phosphorylation, which increases fatty acid oxidation and decreases their production. Research has demonstrated that consuming a high-fat diet during the day triggers this metabolic pathway, resulting in a decrease of 18-25% in visceral fat storage and an improvement in the efficiency of fatty acid oxidation in the liver. In addition to lowering the risk of fatty liver disease and improving metabolic performance, synchronizing eating times with the body's intrinsic circadian rhythm decreases low-grade inflammation that hinders normal metabolic activity. A notable but imbalanced weight gain of 226.6 kg was observed in group B2, whose members consumed 25% of their daily calories in the morning and 75% in the evening. This suggests that there was more fat formation than true growth in lean mass.

The SREBP-1c pathway, which is involved in fatty acid production and storage in tissues, is more active throughout the night, which is associated with this. Reytor-González et al. (2025) and Gu et al. (2020) showed that the circadian rhythm affects digestion, absorption, and oxidation. Triglyceride buildup and reduced tissue energy sensitivity are symptoms of late-night eating, which disrupts insulin and cortisol output. In addition, a reduced metabolic rate and different preferences for nutrition use can be the outcome of daily metabolism regulation.

The ghrelin-to-leptin ratio, which regulates hunger, is disturbed and hunger levels are dramatically raised by late-night eating, according to a study conducted by Vujović et al. in 2022. In addition to lowering basal metabolic rate, it lowered core body temperature and reduced energy consumption during waking.

Based on the results reported by Sa'ari et al. (2024) and Davis et al. (2022), these findings can be understood. According to reviews, eating late at night increases the consumption of high-calorie, sugary, and fatty foods and decreases energy expenditure efficiency during the night because of a lower basal metabolic rate and less thermic effect of food. Adipose tissue growth and weight gain are promoted by a positive energy balance that results from the interplay of excess energy intake and circadian rhythm disruption, even when food consumption remains relatively steady. Consequently, unlike eating during the day, eating at night contributes independently to weight increase and the onset of obesity.

In the body, the hypothalamic-pituitary axis controls hunger, fat oxidation, and metabolic efficiency in response to peripheral hormone signals like ghrelin and leptin (Panda, 2016). This axis is part of a larger network that regulates energy balance and body weight. The study found that high-calorie fast food was the main cause of weight gain within this physiological context. This is because fat oxidation is inhibited, hormones that regulate hunger are disrupted, and fat storage increases. Hall et al. (2019) found that ultra-processed diets boost energy expenditure and regulate appetite, and our results are in line with that. On the other side, eating at specific times became clear as a key regulator of metabolic response. Those whose food intake was most concentrated in the morning (A1 and B1) had a faster metabolism and less fat storage, whereas those whose food intake was most concentrated in the evening (A2 and B2) had a slower metabolism and more fat storage.

As pointed out by Garaulet et al. (2013), this can be explained physiologically by the fact that the timing of evening feedings does not correspond with the daily activity of metabolic enzymes and insulin sensitivity. These results provide credence to the idea of chrononutrition as a physiological mechanism for preventing metabolic diseases; they also show that circadian nutrition helps promote energy balance and reduce metabolic obesity, even on high-calorie diets.

Table 5. Inflammatory Factors.

IL-10 pg/ml	TNF-a pg/ml	CRP mg/L	Test
4.280 ± 0.76 d	5.10 ± 0.88 d	0.552 ± 0.06 d	A
3.790 ± 0.91 d	5.92 ± 1.06 d	0.591 ± 0.07 d	A1
3.760 ± 0.73 d	6.28 ± 1.82 cd	0.875 ± 0.09 c	A2
12.130 ± 2.61 a	11.23 ± 2.34 a	3.023 ± 0.81 a	B
8.080 ± 1.83 c	7.41 ± 2.11 bc	1.314 ± 0.53 b	B1
10.258 ± 2.33 b	8.32 ± 1.94 b	2.820 ± 0.94 a	B2

CRP: C-reactive protein; TNF-a: Tumur necrosis Factor-alpha; IL-10: Inter leukin-10

Table 6. Oxidation Indices.

MDA mmol/L	SOD lu/L	GSH lu/L	Gat lu/L	Test
2.34 ± 0.91 b	58.14 ± 6.61 a	0.79 ± 0.06 a	1.45 ± 0.31 b	A
2.12 ± 0.89 b	55.25 ± 7.17 b	0.71 ± 0.15 a	1.61 ± 0.60 a	A1
2.79 ± 0.68 b	53.63 ± 8.09 b	0.82 ± 0.07 a	1.40 ± 0.44 b	A2
4.24 ± 1.07 a	40.46 ± 4.52 d	0.34 ± 0.02 b	0.98 ± 0.13 c	B
3.92 ± 1.16 a	47.78 ± 5.11 c	0.38 ± 0.04 b	1.34 ± 0.50 b	B1
4.08 ± 1.38 a	45.24 ± 6.07 c	0.30 ± 0.01 b	0.92 ± 0.08 c	B2

MDA: Malondialdehyde;SOD: Superoxide dismutase;GSH: Glutathione;Cat: Catalase

Table 7. White Blood Cell Count and Immunoglobulins

%Lym From WBC	WBC 102/mml	IgA mg/dL	IgG mg/dL	Test
67.31 ± 10.4 b	8.32 ± 1.66 c	192.3 ± 13.8 c	1241 ± 31.6 a	A
66.89 ± 9.50 b	8.94 ± 2.17 c	219.3 ± 12.9 a	1237 ± 28.6 a	A1
67.21 ± 6.94 b	9.21 ± 2.91 c	210.2 ± 13.0 b	1198 ± 30.1 b	A2
72.50 ± 8.93 a	19.20 ± 3.88 a	120.4 ± 14.2 a	978 ± 21.9 d	B
71.41 ± 11.7	14.37 ± 2.67	150.8 ± 13.7	1130 ± 27.6	B1

a	b	d	c	
72.32 ± 10.6 a	18.28 ± 2.91 a	135.9 ± 12.9 e	995 ± 24.3 d	B2

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