Evaluation Melatonin and some biochemical parameters of obese in Baiji City

Iman Abdul Karim Huwaish*[1], Enas Daoud Armid[2]
1,2- Salah al-Din Education Directorate -Baiji Education Department
Corresponding Author : Aymanalfyhan975@gmail.com

Abstract

A contemporary perspective on obesity recognizes the various factors contributing to weight gain and the health advantages associated with weight reduction. Central to any weight loss initiative are lifestyle modifications, dietary adjustments, and enhanced physical activity. The present study aimed to evaluated level of glucose, cholestrole and iron in obese, normal weight and overweight. The study samples were collected from private laboratories in the Baiji district. (90) samples were collected from participants. The participants were divided according to BMI into three groups as follows, depending on the classification of WHO. 30 samples were from normal weight, 32 of them were overweight, and 28 were obese. The ages were 15-60 for both sexes from October 4, 2024, to February 12, 2025. Result: The results show that mean melatonin levels were significantly higher in the normal-weight group (285.7 ± 56.12 U/ml) compared to the overweight (201.3 ± 44.5 U/ml) and obese (157.1 ± 24.2 U/ml) groups. The difference was statistically significant (P = 0.03). A significant in malondialdehyde levels decreased (3.1 ± 0.05 ng/ml) in the normal-weight group, while increase in the overweight and obese group (P = 0.001). Glutathione levels were lower in the overweight and obese groups compared to the normal-weight group, with a statistically significant difference (P = 0.01).The present study showed no significant differences (p-value >0.05) in fasting blood glucose between study groups. While significant increase cholesterol level as BMI increased (p-value<0.01), highly increased in obese patients (176.50±23.25), then decreased in overweigh patients (162.67±16.44) as compared with normal-weight (146.80±16.05). A significant increase (p-value <0.05) in the level of glucose in female as compared to male (80.17± 12.91,116.53±12.9) mg/dl respectively. Cholestrol level also increased in female (181.5±26.20) mg/dl as compared with male (157.1±13.45) mg/dl at (p-value <0.05). While no statistical differences in the Fe level between male and female (p-value >0.05). The current study concluded increase malondialdehyde, cholesterol in obese individual, while no differences in the level of iron and glucose. Glutathione and melatonin decreased in obese individual. In obese participants Glucose level increased in female as compared with male but no differences in iron and cholesterol. According ages, only cholesterol level increased in as ages increased. As residence, only iron increased in Urban residence.

Keywords: BMI, ,melatonin, antioxidant, MDA blood sugar, cholesterol, iron

تقييم الميلاتونين وبعض المعايير الكيميائية الحيوية لدى السمنة في مدينة بيجي

ايمان عبدالكريم هويش عبدالله *[1]، ايناس داود ارميض فريح [2]

1,2- مديرية تربية صلاح الدين - قسم تربية بيجي

الايميل:Aymanalfyhan975@gmail.com

الخلاصة:

يُقر المنظور المعاصر للسمنة بالعوامل المختلفة التي تُسهم في زيادة الوزن والمزايا الصحية المرتبطة بإنقاص الوزن. يُعد تعديل نمط الحياة والتعديلات الغذائية وزيادة النشاط البدني من العناصر الأساسية لأي مبادرة لإنقاص الوزن. هدفت الدراسة الحالية إلى تقييم مستوى الجلوكوز والكوليسترول والحديد لدى المصابين بالسمنة والوزن الطبيعي وزيادة الوزن. جُمعت عينات الدراسة من مختبرات خاصة في منطقة بيجي. جُمعت (90) عينة من المشاركين. قُسّم المشاركون وفقًا لمؤشر كتلة الجسم إلى ثلاث مجموعات على النحو التالي، بناءً على تصنيف منظمة الصحة العالمية: كانت 30 عينة من ذوي الوزن الطبيعي، و 32 منهم يعانون من زيادة الوزن، و 28 يعانون من السمنة. تراوحت أعمار المشاركين بين 15 و 60 عامًا لكلا الجنسين من 4 أكتوبر 2024 إلى 12 فبراير 2025.

أظهرت النتائج أن متوسط مستويات الميلاتونين كان أعلى بشكل ملحوظ في المجموعة ذات الوزن الطبيعي (285.7 ± 56.12 وحدة/مل) مقارنةً بمجموعتي زيادة الوزن (201.3 ± 44.5 وحدة/مل) والسمنة (157.1 ± 24.2 وحدة/مل). وكان الفرق ذا دلالة إحصائية (P = 0.03). انخفضت مستويات MDA بشكل ملحوظ (3.1 ± 0.05 نانوغرام/مل) في المجموعة ذات الوزن الطبيعي، بينما ارتفعت في المجموعة ذات الوزن الزائد والسمنة (P = 0.001). وكانت مستويات GSH أقل في المجموعتين ذات الوزن الزائد والسمنة مقارنةً بالمجموعة ذات الوزن الطبيعي، مع وجود فرق ذي دلالة إحصائية (P = 0.01).

لم تُظهر الدراسة الحالية أي فروق ذات دلالة إحصائية (القيمة الاحتمالية > 0.05) في نسبة الجلوكوز في الدم أثناء الصيام بين مجموعات الدراسة. بينما ارتفع مستوى الكوليسترول بشكل ملحوظ مع زيادة مؤشر كتلة الجسم (القيمة الاحتمالية <0.01)، حيث ارتفع بشكل كبير لدى مرضى السمنة (176.50 ± 23.25)، ثم انخفض لدى مرضى زيادة الوزن (162.67 ± 16.44) مقارنةً بالوزن الطبيعي (146.80 ± 16.05). كما ارتفعت نسبة الجلوكوز بشكل ملحوظ (القيمة الاحتمالية <0.05) لدى الإناث مقارنةً بالذكور (80.17 ± 12.91، 116.53 ± 12.9) ملغ/ديسيلتر على التوالي. كما ارتفع مستوى الكوليسترول لدى الإناث (181.5 ± 26.20) ملغ/ديسيلتر مقارنةً بالذكور (157.1 ± 13.45) ملغ/ديسيلتر عند (القيمة الاحتمالية <0.05). في حين لم تظهر فروق إحصائية في مستوى الحديد بين الذكور والإناث (القيمة الاحتمالية > 0.05). خلصت الدراسة الحالية إلى ارتفاع مستوى المالونديالدهيد والكوليسترول لدى الأفراد المصابين بالسمنة، مع عدم وجود فروق في مستويات الحديد والجلوكوز. انخفض مستوى الجلوتاثيون والميلاتونين لدى الأفراد المصابين بالسمنة. أما لدى المشاركين المصابين بالسمنة، فقد ارتفع مستوى الجلوكوز لدى الإناث مقارنةً بالذكور، مع عدم وجود فروق في مستويات الحديد والكوليسترول. وحسب الأعمار، ارتفع مستوى الكوليسترول فقط مع تقدم العمر. أما بالنسبة لسكن السكان، فقد ارتفع مستوى الحديد فقط لدى سكان المناطق الحضرية.

الكلمات المفتاحية: مؤشر كتلة الجسم ، الميلاتونين،المالون ثنائي الالديهايد،مضادات الاكسدة، ، سكر الدم، الكوليسترول، الحديد

Introduction:

Body mass index (BMI) is calculated by dividing body weight (in kilograms) by the square of height (in metres). It is one of the methods for assessing body fat and was discovered by the Belgian scientist Adolphe Quetelet. Overweight can be expressed as a BMI of 25.0-29.9 kg/m2 [1].

Obesity is a major health problem worldwide and can be defined as “a disease of widespread fat accumulation that has a significant impact on human health” [2].

However, the amount of excess fat distribution within the body, its duration, and the health effects associated with it can vary greatly among obese individuals [3]. Obesity may facilitate the onset of type 2 diabetes mellitus (T2DM) and cardiovascular illnesses[4]. The interaction among many metabolic systems, including the gut, liver, adipose tissue, and skeletal muscle, significantly regulates energy metabolism and influences metabolic health. T2DM is becoming increasingly prevalent and is a significant concern both nationally and worldwide[5].

Melatonin (N-acetyl-5-methoxytryptamine), the primary hormone regulating circadian rhythms, is secreted by the pineal gland during the evening when blue light wanes, in reaction to a circadian pacemaker situated in the suprachiasmatic nuclei of the hypothalamus, referred to as "the master clock. The diverse pleiotropic effects of melatonin in regulating cellular metabolism are linked to the differential distribution of its receptors, chiefly Melatonin receptor 1 (MT1) and Melatonin receptor 2 (MT2) [7]. Melatonin has been shown to influence carbohydrate metabolism through the regulation of the glucose transporter (Glut-4) and to contribute to immune system regulation[8]. Oxidative stress refers to an imbalance between oxidants and antioxidants, characterised by the excessive generation of reactive oxygen species (ROS). Polyunsaturated fatty acids (PUFA) readily compromise cell membranes when exposed to oxidising radicals. This process is referred to as lipid peroxidation, with malondialdehyde (MDA) being a significant end result of this phenomenon[9]. It is associated with the extent of lipid peroxidation and is consequently one of the most prevalent indicators employed to evaluate oxidant status [10]. Oxidative stress has been identified as a characteristic trait of obesity [11]. Glutathione (GSH), a tripeptide, is an essential element in its reduced form inside the cellular antioxidant defence mechanism. Glutathione peroxidase (GPx), a selenium-dependent enzyme, catalyses a mechanism in which GSH donates hydrogen to highly reactive reactive oxygen species (ROS), transforming them into more stable molecules. In these reactions, two GSH molecules dimerize via a disulphide link to produce oxidised glutathione (GSSG)[12].

A positive link is considered to exist between random blood glucose levels and obesity. Nonetheless, racial characteristics appear to have a significant role in the association between body mass index (a measure of adiposity) and glucose intolerance[13]. An elevation in total cholesterol is induced by multiple factors, including obesity. Obesity comprises peripheral obesity and central obesity. Peripheral obesity is characterized by an excessive accumulation of fat in the body's extremities, whereas central obesity involves a greater accumulation of fat in the waist and hips. Obesity is frequently assessed using the BMI. BMI is an economical, straightforward, and rapid metric for evaluating overall body size; nevertheless, it fails to analyze body fat distribution and morphology[14]. In humans, iron is crucial for erythropoiesis, myoglobin synthesis for oxygen transport to muscles, immune defense, DNA replication and repair, various metabolic enzymes, and mitochondrial energy production; both iron deficiency and excess adversely impact mitochondrial oxidative

phosphorylation. In addition to extreme obesity, the distribution of body fat is also correlated with iron measurements[15]

Materials and Method

Subject

The study samples were collected from private laboratories in the Baiji district. (90) samples were collected from participants. The participants were divided according to BMI into three groups as follows, depending on the classification of WHO[16]: 30 samples were from normal weight, 32 of them were overweight, and 28 were obese. The ages were 15-60 for both sexes from October 4, 2024, to February 12, 2025.

Sample collection

Specimens Venous blood samples for biochemical examination were obtained from participants following a minimum fasting time of 12 hours. Five milliliters were transferred to gel tube and permitted to coagulate at room temperature. After that, serum was extracted from blood samples by centrifuging them at 3000 rpm for 10 minutes. The serum has been kept at -20°C. The samples were permitted to thaw at ambient temperature prior to the measurement of biochemical markers, which include total cholesterol (TC), fasting blood glucose (FBG), and iron (Fe).

Assessment of biochemical parameters

a. The fasting blood glucose levels were determined using the BIOLABO reagents kit, which is designed for glucose analysis.

b. In vitro cholesterol CHOD PAP testing was performed using the BIOLABO reagents kit. Basically, the value that AL-OMARI et al. [17] suggest is less than 200 mg/dl.c. The concentration of iron in blood serum was assessed using the Colorimetric method, employing pre-prepared solutions from the French company BIOLABO[18].

Assessment of Melatonin, MDA, and GSH by ELISA

The ELISA technique was utilised to assess the serum levels. The dish was pre-treated with an antibody that targets human melatonin, MDA, and GSH. The specimen is enhanced with melatonin, MDA, and GSH levels. The levels of human Melatonin, MDA, and GSH showed a favourable relationship with the development of colour in the substrate solution. The process is halted by the addition of an acidic stop solution, after which absorbance is measured at a wavelength of 450 nm.

Statistical analysis

The data was analysed using the ANOVA test via Minitab software. The average was determined to be statistically significant based on the Duncan multiple range test with a significance threshold of 0.01.

Results

Table(1) showed the impact of increasing BMI on melatonin, MDA, and GSH. Level of melatonin were significantly increased in the normal-weight group (285.7 ± 56.12

U/ml) compared to the overweight (201.3 ± 44.5 U/ml) and obese (157.1 ± 24.2 U/ml) groups. The difference was statistically significant (P = 0.03).
A significant increase in MDA levels was observed with higher BMI, that was (3.1 ± 0.05 ng/ml) in the normal-weight group, (5.9 ± 0.4 ng/ml) in the overweight group and (6.4 ± 0.8 ng/ml) in the obese group (P = 0.001). GSH levels lower in the overweight (0.11 ± 0.04 nmol/ml) and obese (0.19 ± 0.08 nmol/ml) groups compared to the normal-weight group (0.23 ± 0.3 nmol/ml), with a statistically significant difference (P = 0.01). The present study showed no significant differences (p-value >0.05) in fasting blood glucose between study groups. While significant increase cholesterol level as BMI increased (p-value <0.01),highlyincreased in obese patients(176.50±23.25),then decreased in overweigh patients(162.67±16.44) as compared with normal weight(146.80±16.05).

Table (1): Level of parameters in study groups.

Parameters	**Normal weight 20-25**	**Over weight 26-30**	**Obese 31- >22**	**P- Value**
	Mean±SD			
Melatonin U/ml	285.7±56.12 a	201.3±44.5 b	157.1±24.2 c	0.03
MDA ng/ml	3.1±0.05b	5.9±0.4 a	6.4±0.8 a	0.001
GSH nmol/ml	0.23±0.3 b	0.11±0.05 a	0.19±0.03 a	0.01
Glugose	86.67±14.03 a	97.60± 14.20 a	99.10± 21.12 a	0.523
Cholesterol	146.80±16.05c	162.2±16.44 b	176.4±23.25 a	0.001
F e	68.6±16.4 a	69.6±10.9 a	70.5±10.3 a	0.06

According to gender, the present study showed no significant differences in the level of melatonin, MDA, GSH and Fe according to sex. While a significant increase (p-value <0.05) in the level of glucose in female as compared to male (80.17± 12.91,116.53±12.9)mg/dl respectively. Cholestrol level also increased in female(181.5±26.20) mg/dl as compared with male(157.1±13.45) mg/dl at (p-value <0.05). As shown in Table (2).

Table (2): Level of parameters in obese and overweight participants according sex

Parameters	Male	Female	P-value

	Mean±SD		
Melatonin U/ml	177.5± 17.5	180.9±15.1	0.1
MDA ng/ml	5.6±0.4	6.7±0.5	0.06
GSH nmol/ml	0.16 ± 0.05	0.14±0.07	0.37
Glugose	80.17± 12.91	116.53±12.9	0.047
Cholesterol	157.1±13.45	181.5±26.20	0.011
F e	69.02 ± 10.0	71.08±12.8	0.526

The participant divided into three age groups as A1 (Age groups **15-30**), A2 (Age groups **31-45**), A3 (Age groups **46-60**). According to age, the results of the present study showed non-significant differences in melatonin, MDA, GSH, glucose and Fe levels between study groups (p-value >0.05). Cholesterol level increased in group A3, and decrease in age group A2, while lowest level in age group A1, at (p-value <0.05). As shown in Table (3).

Table (3): Level of parameters in obese and overweight participants according ages

Parameters	**A1** 7	**A2** 11	**A3** n:	**P- value**
	Mean±SD			
Melatonin U/ml	182.0 ± 14.9 a	180.5±16.3 a	175.1± 15.9 a	0.68
MDA ng/ml	5.9 ±0.7 a	6.2 ± 0.6 a	6.35 ±0.5 a	0.9
GSH nmol/ml	0.14±0.02 a	0. 13±0.03 a	0.18±0.03 a	0.2
Glugose	92.61 a ± 15.91	99.10± 12.70 a	103.24±19.30 a	0.447
Cholesterol	151.5±19.9 c	175.1 ± 22.7 b	181.3±23.3 a	0.01
Fe	76. a 17.5	68.13 a 15.8	66.02 a 19.3	0.4

According to the residence, melatonin, MDA , GSH level showed non significant differences between rural residents and urban residents (P> 0.05). while significant increase (p-value <0.05) in the level of glucose and cholesterol in participants from rural as compared with participant from city. While Fe decrease in participants from rural at (p-value <0.05). As shown in Table (4).

Table(4): Level of parameters in obese and overweight participants according residence

Parameters	Participant from rural	Participant from city	

	Mean±SD		
Melatonin U/ml	175.1±11.80	183.3 ± 15.11	0.28
MDA ng/ml	6.1±0.4	6.2±0.5	0.1
GSH nmol/ml	0.17 ± 0.06	0.13± 0.05	0.08
Glugose	91.7±11.80	105.0 ± 15.11	0.010
Cholesterol	157. 6 ± 16.74	181.30 ± 20.40	0.005
Fe	79.4 ± 18.6	60.7± 16.6	0.04

Discussion

The current investigation revealed a reduction in melatonin levels concomitant with an increase in body weight. This finding can be attributed to the disruption of circadian rhythms and diminished antioxidant capability, as melatonin is recognised for its robust free radical scavenging abilities. Melatonin may influence insulin as a protective mechanism against hypoglycemia during sleep, a phase characterised by elevated melatonin levels and diminished metabolic demands [19]. Moreover, animal studies indicate that melatonin supplementation leads to decreased body weight, enhanced lipid profile, normalised glucose tolerance, and increased oocyte maturation [20,21].

The correlation between the traditional energy balance cycle and the hypothesised mechanisms of melatonin action. A prerequisite for life is the ability to equilibrate energy intake, storage, and expenditure, with the nett outcome of this equilibrium dictating the ultimate body weight. Excessive energy intake relative to energy expenditure results in overweight and obesity. The proposed anti-obesogenic effect of melatonin is partially attributed to its regulatory function in energy balance, primarily influencing the regulation of energy flow to and from reserves and in energy expenditure. Furthermore, its correlation with the physiological processes characteristic of the daily wakefulness/rest sleep cycle may influence body weight[22].

This outcome indicated no variations in melatonin levels based on age, sex, or residence. Melatonin treatment in rats and mice reduces body weight growth and fat tissue in many experimental scenarios. The effects are likely mediated by central and peripheral melatonin target tissues, leading to the synchronisation of circadian rhythms with the activity-feeding-rest-fasting cycle and the enhancement of glucose uptake in adipocytes. Nonetheless, both MT1-knockout and MT2-knockout mice exhibit insulin resistance, indicating that the receptors are involved in the regulation of other metabolic processes in nocturnal species rather than in body weight regulation[23.

The current study demonstrated an increase in MDA levels, which may imply heightened oxidative stress and cellular damage—conditions commonly linked to obesity resulting from fat storage and low-grade chronic inflammation. A study

conducted by [24] assessed the levels of MDA and GSH in rats, revealing a raised level of MDA and a decreased level of GSH.

Obesity elevates the mechanical and metabolic burden on the heart, hence augmenting myocardial oxygen demand. The increased myocardial oxygen consumption leads to the generation of reactive oxygen species (ROS), including superoxide, hydroxyl radicals, and hydrogen peroxide, due to heightened mitochondrial respiration. Fifteen Electron leakage from the mitochondrial electron transport chain facilitates a one-electron reduction of molecular oxygen, leading to the generation of superoxide radicals[25].

GSH is an essential intracellular antioxidant that preserves redox equilibrium, and consistent levels among groups may signify sufficient antioxidant defence irrespective of location. Decreased GSH levels may hinder the body's capacity to mitigate oxidative stress, potentially leading to obesity-related problems[26]. The lack of substantial differences in melatonin, MDA, and GSH indicates that, within the study sample, living in rural or urban regions did not significantly affect oxidative stress levels or antioxidant capacity.

The current study demonstrated increase glucose level according to sex and residence, which highest in female and Participant from rural. This result indicate increase incidence of T2DM in obesity patients especially in women and rural residence. The rationale for these findings is that males serve as the primary earners and are inherently more engaged in physical activities to fulfill their daily obligations as heads of households. The females, conversely, are typically restricted to their marital residences and may only leave with the explicit consent of their husbands or male guardians. As a result, they lead a predominantly sedentary lifestyle. Obesity is likely the most significant modifiable acquired risk factor in the etiology of T2DM, as evidenced by numerous cross-sectional and longitudinal studies[27]. A prospective study of normoglycaemic Swedish men monitored for the onset of T2DM revealed that the incidence of diabetes increased by a factor of twenty-two when comparing individuals with the highest BMI to those with the lowest BMI[28].

The current investigation demonstrated a correlation between cholesterol levels and BMI, sex, age, and residence. The research demonstrated a notable elevation in cholesterol levels correlated with an increase in body mass index. The elevation in cholesterol levels is attributable to dietary patterns, which contribute to heightened fat concentrations in blood plasma, thereby resulting in increased cholesterol levels, alongside the body's capacity for rapid cholesterol synthesis and elimination. This investigation corroborated the findings of [29], which indicated that adipocytes clustered in the abdominal region significantly contribute to lipid imbalance in obese persons, more so than peripheral fat. Visceral fat significantly contributes to insulin resistance and exhibits a larger capacity for fatty acid recycling during lipolysis[30]. Numerous investigations have demonstrated that elevated cholesterol levels are

influenced by dietary composition and genetic susceptibility, including familial hypercholesterolemia and obesity [31,32]. A study revealed that elevated fat mass around the waist significantly contributes to increased total cholesterol levels due to metabolic abnormalities[33]. Disruptions in the physiological processes of fat metabolism in obese persons are indicated by elevated lipid levels, especially total cholesterol [34]. The direct connection between blood cholesterol level and age was documented in two genders. [35 In individuals aged 25 to 64, hypertension prevalence rose with age, with a higher incidence in men compared to women in the 24 to 49 age range [36]. Veghari et al. [37] identified a correlation between abdominal obesity and blood cholesterol levels, which was less pronounced in older individuals and diminished in women. Lifestyle significantly influenced the alteration of blood cholesterol levels in individuals [38]. The studies referenced in [39]yielded varying results, indicating no differences in cholesterol levels between obese and non-obese persons. This is due to the multitude of factors that affect cholesterol production. Age and gender influence blood cholesterol levels. In individuals under 20 years of age, plasma total cholesterol levels typically decline between the ages of 10 and 20. Post-20 years of age, plasma total cholesterol levels rise steadily, peaking in men between 50 and 60 years, and in women between 60 and 70 years. Cholesterol levels in females are elevated during childhood compared to males[40]. Conversely, during adolescence, males exhibit a consistent reduction in cholesterol levels attributable to the influence of increasing testosterone hormone. Generally, adult men over the age of 20 have significantly elevated cholesterol levels in comparison to women. Post-menopausal women sometimes have increased cholesterol levels attributable to diminished estrogen action [41, 42]. A plausible explanation is that metropolitan locations exhibit lower cholesterol levels than rural regions, potentially attributable to women engaging in external employment that typically necessitates physical exercise, in contrast to labor in rural agricultural environments.

The current study indicated that iron levels increased among rural individuals, but no differences were seen concerning sex and age. The present study concurs with [43]; nevertheless, it identifies considerable disparities in anemia prevalence between urban and rural locations. Another study indicate that transferrin saturation exhibits a negative correlation with BMI and is diminished in both men and women with obesity and increased waist circumference compared to those with obesity and a reduced waist circumference[15].

Conclusion

The current study concluded malondialdehyde, cholesterol level increase as BMI increased, while no differences in the level of iron and glucose as BMI. Glutathione and melatonin decreased as BMI increased. In obese participants Glucose level increased in female as compared with male but no differences in iron and cholesterol.

According ages, only cholesterol level increased in as ages increased. As residence, only iron increased in Urban residence.

References

1. Boccellino M, D'Angelo S. Anti-obesity effects of polyphenol intake: Current status and future possibilities. International journal of molecular sciences. 2020;21(16):5642.
2. Ärnlöv J, Collaborators GRF. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet. 2020;396(10258):1223-49.
3. Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, et al. The global obesity pandemic: shaped by global drivers and local environments. The lancet. 2011;378(9793):804-14.
4. Mittendorfer B. Origins of metabolic complications in obesity: adipose tissue and free fatty acid trafficking. Current Opinion in Clinical Nutrition & Metabolic Care. 2011;14(6):535-41.
5. Petersen KF, Shulman GI. Etiology of insulin resistance. The American journal of medicine. 2006;119(5):S10-S6.
6. Samanta, S. Physiological and pharmacological perspectives of melatonin. *Archives of physiology and biochemistry*, 2022, 128.5: 1346-1367.
7. Boiko, Dmytro I., et al. Melatonergic receptors (Mt1/Mt2) as a potential additional target of novel drugs for depression. *Neurochemical Research*, 2022, 47.10: 2909-2924.
8. Gombert, Marie, et al. Melatonin levels in children with obesity are associated with metabolic risk and inflammatory parameters. *Nutrients*, 2021, 13.10: 3629.
9. Uçkan, Kazım, et al. Role of oxidative stress in obese and nonobese PCOS patients. *International journal of clinical practice*, 2022, 2022.1: 4579831.
10. Ghonimi, Nesma AM, et al. Serum malondialdehyde as a lipid peroxidation marker in multiple sclerosis patients and its relation to disease characteristics. *Multiple sclerosis and related disorders*, 2021, 51: 102941.
11. Jakubiak, Grzegorz K., et al. Oxidative stress in association with metabolic health and obesity in young adults. *Oxidative medicine and cellular longevity*, 2021, 2021.1: 9987352.
12. Alkazemi, Dalal; Rahman, Abdur; Habra, Banan. Alterations in glutathione redox homeostasis among adolescents with obesity and anemia. *Scientific reports*, 2021, 11.1: 3034.

13. Janghorbani M. Is the association between blood glucose and all causes and cardiovascular mortality risk dependent on body mass index. Medical Journal of the Islamic Republic of Iran. 1992;6(3):205-12.
14. Larasati S, Alvina A. Rasio lingkar pinggang panggul berhubungan dengan kadar kolesterol total pada dewasa. Jurnal Biomedika dan Kesehatan. 2018;1(2):126-32.
15. Volani C, Doerrier C, Demetz E, Haschka D, Paglia G, Lavdas AA, et al. Dietary iron loading negatively affects liver mitochondrial function. Metallomics. 2017;9(11):1634-44.
16. Gutiérrez-Fisac J, Guallar-Castillón P, León-Muñoz L, Graciani A, Banegas J, Rodríguez-Artalejo F. Prevalence of general and abdominal obesity in the adult population of Spain, 2008–2010: the ENRICA study. Obesity reviews. 2012;13(4):388-92.
17. Omari A. Relation between Serum Homocysteine Level and Lipid Profile in Patients with Dyslipidemia. Education. 2002;2004.
18. Hennessy DJ, Reid GR, Smith FE, Thompson SL. Ferene—a new spectrophotometric reagent for iron. Canadian journal of chemistry. 1984;62(4):721-4.
19. Tuomi, T. et al. Increased melatonin signaling is a risk factor for type 2 diabetes. Cell Metab. 23, 1067–1077 (2016).
20. Pai SA and Majumdar AS. Protective effects of melatonin against metabolic and reproductive disturbances in polycystic ovary syndrome in rats. *Journal of Pharmacy and Pharmacology*, 2014, 66.12: 1710-1721.
21. Nikmard F, Hosseini E, Bakhtiyari M, Ashrafi M, Amidi F, Aflatoonian R. Effects of melatonin on oocyte maturation in PCOS mouse model. *Animal Science Journal*, 2017, 88.4: 586-592.
22. Owino, S., Sánchez-Bretaño, A., Tchio, C., Cecon, E., Karamitri, A., Dam, J., and Tosini, G. Nocturnal activation of melatonin receptor type 1 signaling modulates diurnal insulin sensitivity via regulation of PI3K activity. J. Pineal Res. 64, e12462 (2018).
23. Lima, F. B. et al. The regulation of insulin action in isolated adipocytes. Role of the periodicity of food intake, time of day and melatonin. Braz. J. Med. Biol. Res. 27, 995–1000 (1994). 73. Contreras-Alcantara, S., Baba, K. & Tosini, G. Removal of melatonin receptor type 1 induces insulin resistance in the mouse. Obesity 18, 1861–1863 (2010).
24. WU, Peixian, et al. Serum TNF-α, GTH and MDA of high-fat diet-induced obesity and obesity resistant rats. *Saudi Pharmaceutical Journal*, 2016, 24.3: 333-336.

25. Amirkhizi, F., Siassi, F., Minaie, S., Djalali, M., Rahimi, A., & Chamari. Is obesity associated with increased plasma lipid peroxidation and oxidative stress in women?. *ARYA Atherosclerosis Journal*, 2010, 2.4.
26. He, L., He, T., Farrar, S., Ji, L., Liu, T., & Ma, X. Antioxidants maintain cellular redox homeostasis by elimination of reactive oxygen species. *Cellular Physiology and Biochemistry*, 2017, 44.2: 532-553.
27. Aliyu IS, Aliyu TM, Bakari AG, Hassan SS, Onyemelukwe GC, Sani BG. Relationship between random blood sugar and body mass index in an African population. International Journal of Diabetes and metabolism. 2006;14(3):144-5.
28. Skarfors ET, Selinus KI, Lithell HO. Risk factors for developing non-insulin dependent diabetes: a 10 year follow up of men in Uppsala. British medical journal. 1991;303(6805):755-60.
29. Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ, et al. Abdominal adiposity and coronary heart disease in women. Jama. 1998;280(21):1843-8.
30. Bergman RN, Ader M. Free fatty acids and pathogenesis of type 2 diabetes mellitus. Trends in Endocrinology & Metabolism. 2000;11(9):351-6.
31. Naoumova R, Cummings M, Watts G, Rendell N, Taylor G, Sönksen P, et al. Acute hyperinsulinaemia decreases cholesterol synthesis less in subjects with non-insulin-dependent diabetes mellitus than in non-diabetic subjects. European journal of clinical investigation. 1996;26(4):332-40.
32. Maghrani M, Lemhadri A, Zeggwagh N, El Amraoui A, Haloui M, Jouad H, et al. Effect of Retama raetam on lipid metabolism in normal and recent-onset diabetic rats. Journal of ethnopharmacology. 2004;90(2-3):323-9.
33. Taskinen M-R, Adiels M, Westerbacka J, Söderlund S, Kahri J, Lundbom N, et al. Dual metabolic defects are required to produce hypertriglyceridemia in obese subjects. Arteriosclerosis, thrombosis, and vascular biology. 2011;31(9):2144-50.
34. Pinnick KE, Nicholson G, Manolopoulos KN, McQuaid SE, Valet P, Frayn KN, et al. Distinct developmental profile of lower-body adipose tissue defines resistance against obesity-associated metabolic complications. Diabetes. 2014;63(11):3785-97.
35. Costa J, Borges M, Oliveira E, Gouveia M, Carneiro AV. Incidence and prevalence of hypercholesterolemia in Portugal: a systemic review. Part II. Revista portuguesa de cardiologia: orgao oficial da Sociedade Portuguesa de Cardiologia= Portuguese journal of cardiology: an official journal of the Portuguese Society of Cardiology. 2003;22(5):683-702.
36. Gostynski M, Gutzwiller F, Kuulasmaa K, Döring A, Ferrario M, Grafnetter D, et al. Analysis of the relationship between total cholesterol, age, body

mass index among males and females in the WHO MONICA Project. International journal of obesity. 2004;28(8):1082-90.
37. Veghari G, Sedaghat M, Maghsodlo S, Banihashem S, Moharloei P, Angizeh A, et al. The association between abdominal obesity and serum cholesterol level. International Journal of Applied and Basic Medical Research. 2015;5(2):83-6.
38. Hubert HB, Eaker ED, Garrison RJ, Castelli WP. Life-style correlates of risk factor change in young adults: an eight-year study of coronary heart disease risk factors in the Framingham offspring. American Journal of Epidemiology. 1987;125(5):812-31.
39. Hastuty YD. Perbedaan kadar kolesterol orang yang obesitas dengan orang yang non obesitas. AVERROUS: Jurnal Kedokteran dan Kesehatan Malikussaleh. 2015;1(2):47-55.
40. Wanggung TED, Setianingrum EL, Artawan IM, Rante SDT. Analysis of Waist-to-Hip Ratio with Total Cholesterol Levels in Obese and Non-Obese Employees at El Tari Airport Kupang. Age. 2024;18(19):4.
41. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. Endocrine reviews. 2000;21(6):697-738
42. Ujani S. Hubungan antara usia dan jenis kelamin dengan kadar kolesterol penderita obesitas rsud abdul moeloek provinsi lampung. Jurnal kesehatan. 2016;6(1).
43. Zou Y, Zhang R-H, Xia S-C, Huang L-C, Fang Y-Q, Meng J, et al. The rural-urban difference in BMI and anemia among children and adolescents. International Journal of Environmental Research and Public Health. 2016;13(10):1020.