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Investigating the Role of Irisin in Metabolic Health: A Study on Obese Patients with and without Diabetes

Soha M. Hamdy[1], Shimaa Rabie Saber[1*], Khaled G. Abdel-Wahhab[2]

[1]*Department of Chemistry, Faculty of Science, Fayoum University, Fayoum.*

[2]*Department of Medical Physiology, National Research Centre, Giza, Egypt.*

Abstract

The objective of this study was to investigate the association between circulating irisin and various anthropometric and biochemical parameters in obese patients. The study comprised 60 participants, categorized into three distinct groups: group I, designated as "control" (N = 20), group II, referred to "obese" (N = 20), and group III, referred to as "obese with diabetic " (N = 20It should be clarified that the diagnoses of BMI and weight Inex[H1] are based on the measurements of body mass index and weight. They demonstrated a non-significant variation in the Body Mass Index (BMI) measures and a non-significant diffing of the obese group from the control group, followed by a non-significant increment of the diabetic group. However, alongside these the obese and diabetic categories had significant increases compared to the control group, as well as the obese and diabetic condition had significant increases following the control.The acquired data demonstrated that, when both subjects were compared to the control group, obese subjects had a significant increase in serum Alanine Aminotransferase(ALAT) and Aspartate Aminotransferase (ASAT)activity (6.7%, -19.7%, respectively), whereas obese subjects with diabetes showed variation in serum ALAT and ASAT activity (26.3%, -10%).When compared to the control group, the kidney function parameters for both the obese and the obese with diabetes showed a significant increase (7.1%, -5.3%) for urea and a slight increase (12.5%, 25%) for creatinine. The groups showed variation (3.5%, 5.4%) for Fasting Blood Sugar (FBS) and 2 hour Postprandial Sugar (2PPBS) for the obese groups, but an increase (123.5%, 143.5%) for the obese with diabetes groups. However, there was a notable increase in Hemoglobin A1C (HbA1C) (26.2% vs. 140.5%) as well when compared to control groups in the obese and obese with diabetes groups. Furthermore, oxidative stress marker data showed a significant increase in obese subjects compared to controls (65.9%,-16.7%,-24.3%,-3.5%, -7.0%). However, when these groups were compared to the control, there were highly significant differences for Irisin and Tumor necrosis Factor (TNF-α) (-3.1%, 207.1%) in the obese group and (-18.1%, 407.1%) in the obese with diabetes group..

***Keywords*:** Obese patients, Obese with diabetic patients, Irisin, HBA1C. TNF-α.

1. Introduction

Over time, abnormal or excessive fat tissue accumulation in particular body parts that compromise health is a component of obesity, a complex and long-term (chronic) health condition. According to studies by (Bakhtiyari et al., 2022; La Sala & Pontiroli, 2020; Piché et al., 2020), obesity is linked to an increased risk of developing diabetes mellitus, hyperlipidemia, and hypertension. The causes of obesity are multifactorial. Individual level causes can include socioeconomic factors such as poverty, genetics and family history, and behavioural factors like exercise and eating habits (Sørensen et al., 2022; Vazquez & Cubbin, 2020). Undoubtedly, a family history of obesity represents a significant risk factor for obesity and hyperlipidemia, conditions that are closely associated with adult obesity (Chatterjee et al., 2020; Drozdz et al., 2021). However, a number of drugs, such as antidepressants, seizure inhibitors, diabetes medications, arthritic medications, and particular forms of contraception, as well as a number of illnesses, including cushing's syndrome, hypothyroidism, and Prader-Willi syndrome, can trigger obesity (Bays et al., 2022; Gungor et al., 2022; Mohajan & Mohajan, 2023; Prabhakar, 2024; Sridhar et al., 2022). A myokine known as irisin is secreted by the skeletal muscle of both humans and rodents, and it enters the bloodstream during or shortly after physical activity (Ma et al., 2021; Severinsen & Pedersen, 2020; Yano et al., 2021). Because of its capacity to increase brown white adipose tissue, lower insulin resistance, promote liver lipid accumulation, and improve glucose homeostasis, irisin is becoming recognized, as a critical molecule for metabolic illnesses and other conditions that are known to improve with exercise(Aladag et al., 2023; Li et al., 2021; Yano et al., 2021). Irisin influences the function of skeletal muscle, the pancreas, the liver, the brain, and the bone by increasing insulin sensitivity, metabolism, cognition, and osteogenesis. Additionally, according to(Korta et al., 2019; Liu et al., 2022; Rabiee et al., 2020), it suppresses adipogenic differentiation and promotes osteogenic differentiation. Irisin is primarily secreted by white subcutaneous adipose tissue and plays a significant regulatory role in the conversion of white fat into brown fat (Arhire et al., 2019). This implies that irisin

*Corresponding author e-mail: shimaarabee61@gmail.com, (**Shimaa Rabie Saber**)

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may play a part in improving the metabolic status and decreasing obesity and fat accumulation. Irisin has been suggested to be a key factor in determining the state of bone mineralization, making it a potential biomarker for bone metabolism (Kornel et al., 2021). Whether or not there were issues with the obesity management strategy, the purpose of this study was to assess the circulating irisin level and its relationship to anthropometric and biochemical measurements of obese subjects .

2. Methods

The present study was carried out on 60 female human bodies including obese cases (with and without diabetes mellitus type 2) ~~in~~ compared to healthy ones; the patients were recruited from University Hospital, Faculty of Medicine, Fayoum University in 2020. On behalf of the Scientific Research of Ethics Committee of the faculty of Science, Al-Azhar University, Assiut, this study was approved replace on March 21 with approval number AZHAR 18/2021.

Inclusion criteria

Female human bodies complaining of obesity, both uncomplicated and complicated obese, defined as a BMI equal 30 kg/m^2 or higher. With no psychotherapy or any other medications. Psychiatric conditions other than depression, like bipolar disorder, schizophrenia, psychotic disorders, and suicidal thoughts. In addition to any substance, including alcohol. Cases who are not receiving type 2 diabetes treatment but are taking any medication. They've all been left out.

Furthermore, healthy female volunteers from the same zone who matched in age and social status were also included. The cases were split into three groups: group 1 consisted of control subjects who were in good health, group 2 consisted of obese subjects who had no complications, and group 3 consisted of obese subjects who were diabetic. Every case in this study underwent three blood pressure readings, a comprehensive gynecology and obstetric history, a general examination, and a detailed medical history recording.

Additionally, a variety of anthropometric measurements, such as body fat percentage, fat free mass (FFM), body mass index (BMI), weight, length, waist, height, and basal metabolic rate (BMR), were evaluated using the body composition scale. Moreover, blood samples were collected from all subjects to determine the values of liver function [serum alanine aminotransferase (ALAT), and aspartate aminotransferase (ASAT)], kidney function [serum urea and creatinine], diabetic markers [fasting blood sugar (FBS), post prandial blood sugar (PPBS), and glycosylated hemoglobin (Hb A1C)], complete blood picture (CBC), oxidative stress markers [lipid-peroxidation end product (malondialdehyde, MDA), and nitric oxide (NO) levels], antioxidative markers [reduced glutathione (GSH) level, superoxide dismutase (SOD), and catalase (CAT)], lipid profile [total cholesterol, triglycerides, LDL-cholesterol, and HDL-cholesterol] obesity marker (leptin), myokine marker (irisin), cytokine inflammatory marker (tumor necrosis factor alpha (TNF-α).

Measurement of CBC

Automated hematology analyzer KX-21N (Sysmex Corporation, Kobe 651-0073, Japon) was used to assess hemoglobin (Hb), red blood cells (RBCs), hematocrit (HCT), white blood cells (WBCs), and platelets (PLTs) described by (Cohen et al., 2021).

Measurement of diabetic markers

Serum glucose level was determined colorimetrically using reagent kits obtained from GOD-POD kits purchased from Biomed Diagnostic, Germany described by (Trinder, 1969); HbA1c concentration was evaluated by using kits purchased from Biomed Diagnostic, Germany described by (Boye et al., 2021).

Assessment of liver and kidney functions

Serum ALAT and ASAT activity was measured spectrophotometrically using laboratory diagnostic kits purchased from Human Gesellschaft für Biochemica und Diagnostica mbH, Germany described by (Schumann & Klauke, 2003) . Serum level of urea and creatinine was measured using laboratory diagnostic kits obtained from BioSystems, Pennsylvania, USA described by (Young, 2001).

Assessment of lipid profile

Serum level of total cholesterol, triglycerides, LDL-cholesterol, and HDL-cholesterol was determined spectrophotometrically using reagent kits purchased from Roche Diagnostics, Indianapolis, USA described by (Berth & Delanghe, 2004; Naito, 2003)

Assessment of oxidative stress markers

Serum levels of GSH, NO and MDA as well as activities of SOD and CAT were assayed spectrophotometrically using reagent kites obtained from Biodiagnostic, Giza, Egypt, were estimated according to the methods of (Beutler et al., 1963; Montgomery, 1971; Nishikimi et al., 1972; Ruiz-Larrea et al., 1994).

Assessment of leptin (obesity marker

Serum leptin level was measured using a human ELISA reagent kit purchased from Diagnostics Biochem, Ontario, Canada. *Described by* (Sung & Heo, 2020)

Assessment of irisin (myokine)
Serum irisin was measured using human reagent elisa kit purchased from SimoGeneclon, Hang Zhou, China *Described by.*(Zhang et al., 2020)

Assessment of TNF-α (proinflammatory marker)
The level of serum TNF-α was measured using a human reagent elisa kit purchased from SimoGeneclon, Hang Zhou, China described by (Brouckaert et al., 1993).

Statistical analysis
The obtained data were subjected to one-way ANOVA, followed by post hoc (Duncan) test at $p \leq 0.05$ level using Package Program (SPSS, 2008) 17, released version carried out according to(Washington et al., 2020).

3. Results

The anthropometric results of this study demonstrated that the group of obese cases had a significant increase in body weight (54.4%), BMI (51.4%), BF (161.9%), and free-fat mass (FFM) (30%), which was associated with a discernible decrease in basal metabolic rate (BMR) (-27.03%); on the other hand, the group of obese diabetics showed an additional increase in body weight, BMI, BF, and FFM (57.4, 55.7, 162.4, and 301.6%, respectively) and paired with an additional decrease in BMR (-34.57%) when compared to the corresponding values of control, healthy subjects (Table 1).

Table 1. Anthropometric markers of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
Weight (kg)	M±SD	64.6±4.7^{b}	99.8±15.9^{a}	101.7±12.5^{a}
	%		54.4	57.4
BMI (kg/m^2)	M±SD	25.3±1.9 ^{b}	38.3±6.1 ^{a}	39.4±2.8 ^{a}
	%		51.4	55.7
Age(years)	M±SD	45.5±8.4 ^{a}	46.2±9.2 ^{a}	46±8.2 ^{a}
	%		1.5	1.1
BF (%)	M±SD	19.7±0.8 ^{b}	51.6±4.9 ^{a}	51.7±6.2 ^{a}
	%		161.9	162.4
FFM (kg)	M±SD	12.7±0.5 ^{b}	50.8±2.6 ^{a}	51±2.1 ^{a}
	%		300	301.6
BMR (kkal)	M±SD	1990±102.7 ^{b}	1452±49.2 ^{c}	1302±83.6 ^{b}
	%		-27.03	-34.57

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at $p \leq 0.05$. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group. BMI (body mass index), BF (body fat), FFM (free fat mass), BMR (body metabolic rate).

The current study also revealed a significant difference between the obese group and the healthy control group in terms of inflammatory markers and obesity. The obese group had significantly higher serum levels of leptin (242.6%) and tumour necrosis factor alfa (207.1%), along with a marked decrease in irisin (-13.54%) (Table 2). cases with diabetes and obesity.

Table 2. Obese and inflammatory markers of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
Leptin (ng/ml)	M±SD	13.6±3.5 ^{b}	46.6±19.5 ^{a}	63.4±31.8 ^{a}
	%		242.6	366.2
Irisin (pg/ml)	M±SD	25.1±1.2 ^{a}	21.7±4.9 ^{b}	18.5±1.8 ^{c}
	%		-13.54	-26.29
TNF-α (ng/ml)	M±SD	4.2±1.9 ^{c}	12.9±1.6 ^{b}	21.3±3.1 ^{a}
	%		207.1	407.1

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at $p \leq 0.05$. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group.

In comparison to the healthy control group, the obese-diabetic group demonstrated a significant increase (123.5, 143.48, and 140.47%) in serum concentration of FBS, PPBS, and HbA1c, respectively. Table 3 shows the values of diabetic markers among the study groups. The non-diabetic obese subjects did not show any notable changes in the measured diabetic markers. According to the current study, the obese group experienced a significant increase in the blood levels of oxidative stress markers, such as MDA (66.1%) and NO (16.71%), along with a significant decrease in GSH (24.43%); however, the antioxidant enzymes' activity (CAT and SOD) showed a slight decrease (-9.7, -7.0%, respectively).

Table 3. Diabetic markers of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
FBS (mg/dl)	M±SD	88.4±7.8 [b]	91.5±6.2 [b]	197.6±64.4 [a]
	%		3.5	123.5
PPBS (mg/dl)	M±SD	113.6±13.3 [b]	119.8±7.1 [b]	276.6±72.3 [a]
	%		5.45	143.48
HbA_1C (%)	M±SD	4.2±0.8 [b]	5.3±0.3 [b]	10.1±0.5 [a]
	%		26.2	140.47

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at $p \leq 0.05$. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group.

When comparing the two groups to the healthy control group, the obese-diabetic group also displayed an exceptionally marked increase in the serum levels of MDA and NO (157.5 and 71.16%, respectively), which was accompanied by an even more notable decrease (-44.45, -27.8 & -33.0%) in the serum values of GSH, CAT, and SOD. (Table 4).

Table 4. Oxidative stress markers of control, obese and obese-diabetic subjects

		Control	Obese	Obese/diabetic
MDA (nmol/L)	M±SD	128.6±11.2 [c]	213.6±31.4 [b]	331.1±44.4 [a]
	%		66.1	157.5
NO (µmo1/L)	M±SD	65.2±2.5 [c]	76.1±3.1 [b]	111.6±4.7 [a]
	%		16.71	71.16
GSH (mg/ml)	M±SD	110.9±10.36[a]	83.8±7.09 [b]	61.6±7.50 [c]
	%		-24.43	-44.45
CAT (µ mo1/L)	M±SD	77.3±3.23 [a]	69.8±2.82 [a]	55.8±2.21 [b]
	%		-9.7	-27.8
SOD (µ mo1/L)	M±SD	41989±3523 [a]	39049±3279 [a]	28132±2360 [b]
	%		-7.0	-33.0

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at $p \leq 0.05$. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group.

The serum lipid profile of control, obese, and obese-diabetic cases are displayed in Table 5. Both the obese and obese-diabetic groups had significantly higher serum levels of cholesterol (34.8 & 49.03%), triglycerides (45.66 & 67.98%), and LDL (28.0 & 58.4%) when compared to the control group. This was accompanied by a sharp decline in HDL levels (-14.34 & -17.67%), but the obese-diabetic group showed the greatest deterioration.

Table 5. Serum lipid profile of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
Chol (mg/dl)	M±SD	150.7±16.9 [c]	203.2±20.1 [b]	224.6±17.5 [a]
	%		34.8	49.03
Trigl (mg/dl)	M±SD	78.4±16.9[c]	114.2±39.5 [b]	131.7±29.5 [a]
	%		45.66	67.98
LDL (mg/dl)	M±SD	107.1±35.3 [c]	137.1±25.5 [b]	169.7±37.4 [a]
	%		28.0	58.4
HDL (mg/dl)	M±SD	48.1±5.1 [a]	41.2±8.4 [b]	39.6±4.03 [b]
	%		-14.34	-17.67

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at $p \leq 0.05$. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group

The results of the present study in Table 6 declared that obese non-diabetic cases recorded liver aminotransferase (ALAT & ASAT) activity and kidney function (urea & creatinine) values near to those of healthy control cases, while obese-diabetic cases showed a significant elevation in the activity of ALAT (72.67%) and ASAT (17.57%) while as kidney function declare minute changes in urea (6.63%) and creatinine (6.74%) in compare to control subjects.

Table 6. Serum liver functions (ALAT & ASAT) and kidney functions (urea & creatinine) of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
ALAT (IU/ml)	M±SD	18.3±4.7 [b]	20.1±3.5 [b]	31.6±4.7 [a]
	%		9.8	72.67
ASAT (IU/ml)	M±SD	23.9±2.8 [b]	24.3±4.4 [b]	28.1±3.9 [a]
	%		1.67	17.57
Urea (mg/dl)	M±SD	22.6±3.8 [a]	23.0±3.8 [a]	24.1±7.3 [a]
	%		1.77	6.63
Creatinine (mg/dl)	M±SD	0.89±0.2 [a]	0.9±0.1 [a]	0.95±0.2 [a]
	%		1.12	6.74

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at p≤0.05. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group.

Regarding the hematological findings in Table 7, the results of obese subjects group showed notable decrease in hemoglobin (-8.5%), hematocrit (-7.13%), and WBCs (-15.8%) values; however, the other blood indices slightly changed. In addition, the obese-diabetic group performed stronger hematological deteriorations; however, a remarkable decrement was observed in Hb (-17.1), RBCs count (-11.11%), HCt (-14.7%), MCV (-13.5%), MCH (-11.11%), and WBCs (-22.8%) however MCHC and platelets values never significantly changed when both groups were compared with the control group.

Table 7. Complete blood count of control, obese and obese-diabetic subjects

		Control	Obese	Obese-diabetic
Hb (g/dl)	M±SD	12.9±0.9 [a]	11.8±0.8 [b]	10.7±0.9 [c]
	%		-8.5	-17.1
RBCs (10^6/mm^3)	M±SD	4.5±0.3 [a]	4.3±0.3 [a]	4.0±0.3 [b]
	%		-4.4	-11.11
HCt (%)	M±SD	40.7±2.9 [a]	37.8±1.9 [b]	34.7±3.3 [c]
	%		-7.13	-14.7
MCV (fL)	M±SD	89.2±1.1 [a]	82.1±1.1 [a]	77.1±1.2 [b]
	%		-7.96	-13.5
MCH (pg/cell)	M±SD	28.8±0.4 [a]	27.6±0.4 [a]	25.6±0.4 [b]
	%		-4.17	-11.11
MCHC (g/dl)	M±SD	32.2±0.5 [a]	31.1±0.5 [a]	30.3±0.5 [a]
	%		-3.4	-5.9
WBCs (10^3/mm^3)	M±SD	5.7±0.83 [a]	4.8±0.64 [b]	4.4±0.61 [b]
	%		-15.8	-22.8
PLT (10^3/mm^3)	M±SD	254.1±52.9 [a]	251.9±43.9 [a]	245±51.5 [a]
	%		-0.87	-3.58

Data are presented as mean ±standard deviation. Data were subjected to one-way ANOVA followed by post hoc (Duncan) test at p≤0.05. Within the same raw, means with different superscript letters are significantly different. % was calculated from control group.

The results in Table 8 show the correlation of the serum myokine, irisin, with the anthropometrical measurement, diabetic, immune-inflammatory, and lipid profile, as well as hepatic oxidative stress, and markers among the study subjects. The results revealed that irisin is positively correlated with.

Table 8. Correlation of irisin with the measured anthropometric, diabetic, inflammatory, oxidative stress, and lipid markers among healthy, obese, and obese diabetic subjects.

Parameter	R	*p* value	Parameter	R	*p* value
BMI	-0.093	0.798	MDA	0.659	0.038
BF	0.221	0.539	NO	0.067	0,854
FFM	0.054	0.883	CAT	0.479	0.161
BMR	0.206	0.806	SOD	0.043	0.905
FBS	0.362	0.304	Cholesterol	-0.102	0.779
PPBS	-0.179	0.621	Triglycerides	0.135	0.711
HbA1c	-0.213	0.555	LDL-c	0.089	0.808
Leptin	0.256	0.475	HDL-c	0.436	0.208
TNF-α	-0.109	0.764			

R: correlation coefficient and
P value is non-significant when > 0.05

4. Discussion

Obesity that has collected to an excessive degree to the point that it could be dangerous to health is referred to as obesity, which is a medical condition that is occasionally referred to as a disease. A person is deemed obese if their body mass index (BMI), which is determined by dividing their weight by height squared, is higher than 30 kg/m2; between 25 and 30 kg/m2 is regarded as overweight (Nam *et al*., 2020)**.** Obesity can cause complications or indirectly through factors like poor diet or sedentary lifestyle. Excess body fat is linked to type 2 diabetes, with 64% of cases in males and 77% in women. Increased fat mass can lead to negative health effects, including insulin resistance, inflammation, and prothrombotic states (Gonzalez Ramirez & Bolaños Muñoz, 2023)**.**

The study found a significant increase in obese and obese with complications compared to the control group, with no significant variation in weight measurements or BMI measurements. This aligns with previous studies; (Bo *et al*., 2020; Hall *et al*., 2022; Kayser & Verges, 2021)demonstrating obesity is caused by positive energy balance and is a major public health issue worldwide.
Irisin levels in obese individuals showed no significant difference compared to the control group, but a significant decrease in those with complications which in agree with(Wang & Liu, 2021). Studies have linked irisin levels to BMI, obesity, and lipid and glucose homeostasis impairment in metabolic syndrome and obesity (Crujeiras *et al*., 2014).

Leptin, a hormone that controls appetite, weight, reproductive health, and immune responses, is produced by the obese gene and activated by fat cells in white adipose tissue. Its pleiotropic actions are enabled by LEP-R distribution. Results show significant increases in leptin levels in obese and obese with complications, contradicting previous findings, which agrees with (Izquierdo *et al*., 2019).
Studies show leptin levels are higher in obese patients, inversely correlated with abdominal fat index and waist-hip ratio, and directly correlated with subcutaneous fat, suggesting leptin has no clinical utility in obesity, these findings are supported ~~with~~(Kumar *et al*., 2020). Also, Obesity poses health risks, with Leptin, a protein secreted from adipose tissue, strongly linked to weight loss and obesity, regulated by the ob gene (Obradovic *et al*., 2021).

The study found significant increases in TNFa, MDA, and NO levels in obese and obese with complications groups in agree with agree ~~with~~(Bruun *et al*., 2002). However, IL-8 and BMI were not linked. Weight loss and maintenance led to a 30% increase in IL-8 and a 40% decrease in TNF-a, respectively. This suggests that obesity is associated with increased TNF-α expression in adipose tissue.
Our study confirms (Yesilbursa *et al*., 2005) finding that obesity increases lipid peroxide levels, while our findings show MDA decreases with orlistat-induced weight loss. Reduced free radical production in obesity and diabetes is linked to oxidative mechanisms, with nitric oxide (NO) playing a key role in insulin sensitivity and body fat regulation.

Serum NO levels are positively linked with BMI in both male and female groups, with obesity patients potentially having higher levels due to increased NO production. Obese females have 3.5-fold higher NO production than obese males, accompanied by a 40% decrease in this ~~as~~ (Wierzchowska-McNew *et al*., 2022) findingObesity and type 2 diabetes are global public health challenges, with rising prevalence and significant health consequences. Obesity is linked to higher chances of acquiring T2DM due to high BMI and WC. The study found no significant difference in body mass index (BMI) between obese and control groups, but a significant increase in obese with complications for FBS and PPs, and a significant increase in HbA1c, confirming a close relationship this agrees with (Al-Goblan *et al*., 2014).

Obesity increases insulin resistance, leading to impaired pancreatic -islet cells, causing blood glucose regulation, and is the aetiology of diabetes (Leitner *et al*., 2017; Martyn *et al*., 2008). Diabetes, a non-communicable disease, is strongly associated with obesity, leading to the term "diabesity." BMI is still used to categorize overweight and obesity, and measuring body composition is strongly recommended due to diminished muscle mass.

Superoxide dismutase and catalase are key enzymes in the cell's antioxidant defense system, with glutathione playing a vital role in DNA synthesis, vitamin C's antioxidant properties, amino acid transport, and toxic substance detoxification. The study found a significant decrease in GSH, SOD, and CAT levels in obese individuals and those with complications, indicating an increase in oxidative stress, and that these parameters gradually declined after bariatric surgery, regardless of metabolic syndrome this is supported by (Gusti *et al*., 2021).

Oxidative stress significantly influences obesity by altering biological components and obesogenic pathways, with catalase being a key antioxidant enzyme that catabolizes hydrogen peroxide.
(Goth & Nagy, 2013) suggest that catalase deficiency may be linked to obesity, as patients adopting healthy lifestyles avoid consequences like diabetes. Adipose tissue excess leads to excessive ROS production, causing metabolic abnormalities like insulin resistance and cardiovascular complications. High-fat meals reduce superoxide dismutase activity, but not in normal individuals.

Obesity patients have elevated serum triglyceride, VLDL, apolipoprotein B, and non-HDL-C levels due to increased hepatic production of VLDL particles and decreased clearance of triglyceride-rich lipoproteins. Cholesterol levels increased significantly in obese and obese with complications groups compared to control, but non-significant variation was observed in obese with complications groups. HDL levels also increased significantly. The 2020 research by Shabana *et al.* found that obese and CHD groups in Pakistan have dyslipidemic profiles compared to healthy controls. The obese group had a higher proportion of CHD in blood relations. 50.7% of cases had mixed lipid abnormalities, with TC, LDL-C, TG, and HDL-C levels out of range. This suggests Pakistani cases are lipid hyperlipidemic, with irisin secreted from muscles in response to exercise potentially mediating beneficial effects**.**.ALT and AST enzymes are vital for liver function, with ALT levels increasing in liver damage and AST levels often seen in low concentrations. Obesity and obesity with complications are linked to a higher risk of developing steatosis, causing abnormal liver function. These enzymes, along with liver enzymes like ALT and AST, are valid indicators of liver injury this agree with (Jalili *et al*., 2022). Studies show no significant difference in serum AST levels between overweight and obese women. Serum GGT is more connected to obesity than other liver enzymes. Obesity increases the risk of metabolic problems like elevated fasting blood sugar and insulin resistance. Factors like dietary intake may affect liver function, potentially linked to weight-related hormonal disorders ~~according to~~(Knight, 2011).

Urea and creatinine are nitrogenous end products of metabolism. Obesity results show non-significant variation in urea and creatinine levels in obese and obese with complications groups compared to the control group that agree with A Ude,*et al*,2022. Obesity affects renal function, but adult obesity or weight growth does not affect the number of nephrons. Excessive weight gain increases single nephron perfusion and renal plasma flow, leading to compensatory nephron hypertrophy, intraglomerular capillary pressure, glomerular hyperfiltration, and eventually, loss of GFR over time (Cortinovis *et al*., 2022).

A complete blood count (CBC) is a vital tool for assessing general health and identifying illnesses like leukaemia, anemia, and infections. It measures red blood cell oxygen-carrying proteins, hematocrit, and platelets(John & Bell, 2022). Results show a significant decrease in hemoglobin levels and WBCs in obese and obese with complications, but no significant varation that agree with (Al-Attar *et al*., 2020), BMI negatively correlates with serum iron, a link between adult obesity and low iron reserves or anemia. Platelet number and size increase with rising white blood cell (WBC) count, which can lead to thrombi development and increased risk of myocardial infarction or stroke. Platelet counts are also increased in inflammation in agreement with (Chen *et al*., 2020).

5. **Conclusion**

The study underscores obesity's health impact, notably on diabetes and oxidative stress. It links obesity to dyslipidemia, liver issues, and kidney function changes. The research echoes prior studies on obesity's effects on various biomarkers. Urgent public health efforts are needed to combat obesity globally**.**

6. Conflict of interest

There are no conflicts of interest.

7. Acknowledgment

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