An Assessment of Heme Oxygenase-1 and Glutathione-S-transferase in Obese Iraqi Patients

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Abstract

This study aimed to detect the relation of Heme oxygenase-1(HO-1) and Glutathione-S-transferase (GST) in obese patients, against oxidative stress. The study included 139 samples of people with age range of 35-65 years divided in two groups according gender (female and male) groups while four gropes according body mass index (BMI). Blood samples were collected from AL-Yarmouk Hospital at the period between September/2022 to January/2023. Some biochemical parameters were measured for all study groups, which include: -determination of serum HO-1 levels by using the ELISA-technique, lipid profile, GST activity, and fasting serum glucose (FSG) assessed manual. The BMI, waist hip ratio(WHR) and low density lipoprotein (LDL) were found to be significant [(38.83 ± 0.44^b) (32.03 ± 0.26^a) (26.40 ± 0.22^d) (23.10 ± 0.28^c)],,[(1.01 ± 0.03^b) (0.92 ± 0.02^{ab}) (0.93 ± 0.01^{ab}) $(0.88\pm0.01^{a)}$] ,[$(80,42\pm7.47^a)$ (99.74 ± 6.63^{ab}) (112.93 ± 10.12^b) (83.94 ± 8.95^{ab})]] respectively and not significant with other parameters of lipid profile, FSG, and Atherogenic index. Also, it appeared that the HO-1 levels were higher in obese groups compared with healthy groups [$(2.06 \pm 0.09^b)(1.95 \pm 0.04^{ab})$ $(1.88 \pm 0.04^{ab})(1.75 \pm 0.05^a)$] ng/ml respectively . It was shown that the increasing of GST level in healthy compared with obese groups [$(6.10\pm 0.57^c)(4.120 \pm 0.69^b)(2.44 \pm 0.21^a)(2.71\pm 0.30^{ab})$]IU/L respectively .The result showed that the increase in BMI,WHR and HO-1 in obese female and BMI,TG,HO-1 and VLDL in obese male ,compared with female and male healthy groups ,while the results showed an increase in GST levels in healthy female and male groups .We conclude from this study that high levels of heme oxygen-1 synchronized with low of glutathione-S- transferase , clearly indicating their primary role in detoxification, protection of the against oxidative stress, and prevention of the development of metabolic diseases.

Keywords: Body mass index (BMI), Glutathione s transferase, Heme oxygenase, Lipid profile, Obesity, Oxidative stress (OX).

Introduction

Obesity is a complex combination of genetics, it is a complicated and diverse disorder. But no one provides a precise explanation of the process that underlies obesity. The significance of genetics in obesity has been clearly shown by research on ethnic prevalence, family aggregation, twins and adoption [1,2]. The etiopathology of obesity has been linked to a number of risk factors, including both

genetic and environmental ones[3]. One of these factors is oxidative stress, which by promoting the buildup of white adipose tissue and altering food intake, can promote obesity and its accompanying comorbidities[4].There is a significant direct correlation between oxidative stress indicators and Body Mass Index (BMI).Numerous *in vitro* studies have demonstrated that elevated oxidative stress and reactive oxygen species stimulate adipocyte proliferation, differentiation and growth as well as control hunger and satiety responses[5].Oddly, obesity and oxidative stress are linked because too much fat accumulation can lead to an inflammatory and oxidative state via a number of cellular and metabolic pathways [6].When (BMI) is 30 or more, the person is considered obese[7,8].A diagnosis of overweight is indicated by a BMI between 25 and 30. With 62% of adults being categorized as obese or overweight[9] , obesity raises inflammatory cytokines and the likelihood of endothelial cell dysfunction .Patients' risk for diabetes and cardiovascular disease (CVD) is increased by metabolic syndrome[10]. Reactive oxygen species (ROS) cause oxidative stress, which advances disease and has a number of detrimental effects[11].The adipose tissue (AT) is the most efficient site for storing extra fat calories. In humans, adipose tissue, a substantial and active endocrine organ that is involved in energy storage, accounts for between 20 to 25% of total body mass [12]. The only cells in the body that are designed to securely store significant amounts of fat are the fat cells of adipose tissue. Adipose tissues (mainly white adipose tissue) are distributed in the subcutaneous fat (located under the skin) and the visceral fat (located intra-abdominally, adjacent to internal organs)[13].When excessive amounts of nutrients are consumed, the fat tends to build up in the visceral and subcutaneous depots, enlarging these depots through adipocyte cell hypertrophy and hyperplasia and making them unhealthy[14].One enzyme that has proved crucial in managing metabolic diseases is heme oxygenase (HO). The isozymes HO-1 (inducible form) and HO-2 are present in humans (constitutive form). In terms of their processes, cofactor and substrate needs and susceptibility to activation or inhibition by artificial metalloporphyrin, in which the iron atom at the center of heme has been substituted by other elements including tin, zinc, cobalt and chromium, isozymes are comparable. By generating equimolar amounts of carbon monoxide (CO), iron, and biliverdin, the pro-oxidant HO which is also a strong inducer of HO-1 contributes to the breakdown of heme. Biliverdin reductase subsequently transforms the biliverdin into bilirubin. Superoxide dismutase and catalase are two examples of antioxidant enzymes that are improved when HO-1 is activated[15].The secretion of large amounts of HO-1increases the resistance of cell injury by heme. The primary protective role of HO-1 against the development of chronic diseases such as metabolic disorders and type II diabetes is attributed to many processes that include reducing the levels of protein that are mainly dependent on heme, which raise the level of formation of oxidative stress and bilirubin. In addition to the role of heme iron in increasing the accumulation of fats generated in adipose / visceral tissues[16].Cells may be protected by increasing HO-1 expression. Other defensive enzymes, like as glutathione S-transferase (GST) and NAD(P)H: Quinone oxidoreductase (NQO1), which detoxifies oxidative stress by products, shares chromosomal regulatory mechanisms with HO-1 enhancing cellular antioxidant defenses via inducing glutathione (GSH) synthesis enzymes. Glutathione (GSH) is able to prevent damage of cellular components by (ROS) reactive oxygen species[17,18]. The Glutathione-S-Transferase (GST) groups of multifunctional proteins is essential for the liver's clearance of potentially hazardous hydrophobic molecules from blood as well as the detoxification of electrophilic substances [19] This study aimed to detect the relation of Heme oxygenase-1(HO-1) and Glutathione-S-transferase (GST) in obese patients, against oxidative stress.

Materials and Methods

Serum Sample Preparation

Seven milliliters of blood samples were collected in gel tube (serum separated tube) from obese and healthy individuals for heme oxygenase test: GST activity, and lipid profile. The circumference of the waist to the hip (WHR) was measured by tape, and the body mass index was also measured by the formula (weight / (Height^2). The

blood tubes were left for 20-25 minutes at room temperature 25 °C, blood samples then were centrifuged at 2000-3000 rpm for 10 minutes, the separated serum was kept in in deep freeze at- 20°C, in AL-Yarmouk Teaching Hospital.

Analysis of the Samples

This study was conducted at the College of Science for Women, University of Baghdad. The samples were collected from AL- Yarmouk Teaching Hospital. A number of 139 subjects including obese patients (84) who were divided in 2 groups according to BMI. The first group was obese class **I** (G3) (30-35) (n= 41) and the second group was obese class **II** (G4) with BMI ≥35 (n= 43), and control group 55 also was divided in 2 groups according BMI, the first group was healthy BMI (19-24) normal weight (G1), (n= 32) and the second

group was healthy BMI (25-28) over weight (G2) (n= 23). In this study, thyroid patients, diabetes, pregnant women, people with heart disease and other diseases were excluded. The blood serum was used for checking blood for glucose levels, and lipid profile that measured manually by using humane Germane kit, and Heme oxygenase -1(HO-1) by ELISA (My Bio Source, USA). GST activity was measured by colorimetric enzyme method.

Statistical Analysis

All statistical analyses were performed using SPSS software version 26.0. Data with normal distribution was presented as mean ±SE and analysis of variance (ANOVA) as post hoc pairwise comparison (between more than 2 groups), and ROC analysis. A p-value ≤ 0.05 was put as a statistical signal refer to (significant difference).

Results and Discussion

The age for all studied groups in the current study were matched. The body mass index (BMI) and WHR exhibited a significant difference $p \leq 0.05$

between the studied groups, as shown in the (mean ± SE) of BMI and WHR in Table 1, which were [(23.10 ± 0.28 [c])].

Table 1. Comparison of biochemical parameters between study groups.

Groups / Parameters	Normal weight Group (G1) No. (32)	Overweight Group (G2) No. (23)	Obesity class I Group (G3) No. (43)	Obesity class II Group (G4) No. (41)	P-value
Age (year)	46.68 ± 1.58 [a] (46.5)	48.13± 2.13 [a] (46)	46.65 ± 1.32 [a] (45)	46.66± 1.30 [a] (46)	0.915
BMI (kg/m^2)	23.10 ± 0.28 [c] (23.4)	26.40 ± 0.22 [d] (26)	32.03 ± 0.26 [a] (31.9)	38.83± 0.44 [b] (38)	0.0001*
Waist/Hip ratio	0.88 ± 0.01[a] (0.89)	0.93 ± 0.01 [ab] (0.94)	0.92 ± 0.02[ab] (0.90)	1.01 ± 0.03 [b] (0.96)	0.008**

- The data were shown as Mean± SE (Median)
** ANOVA-test results showing a significant difference between means at the 0.005 level.
- Significant variants are denoted by different small letters.
- Non-significant variations are denoted by identical small letters

Anthropometric data of the participants is shown in Table 2. The study showed that the mean± SE of lipid profile is significant in p ≤0.05 in LDL-C, low

density lipoprotein [(83.94 ± 8.95[ab])], but not significant with other parameters of lipid profile, FSG, and Atherogenic index p>0.05.

Table 2. (mean ± SE) of lipid profile between study groups.

Groups Parameters	Normal weight Group (G1) No. (32)	Overweight Group(G2) No. (23)	Obesity Class I Group(G3) No. (43)	Obesity Class II Group(G4) No. (41)	P-value
FSG (mg/dL)	99.11 ± 2.69 [a] (97.85)	98.45 ± 3.04 [a] (96)	98.29 ± 3.13 [a] (94)	94.36 ± 2.85 [a] (90.5)	0.644
TC (mg/dL)	160.2 ± 10.34 [a] (153)	186.62 ± 10.5 [a] (178.7)	178.84 ± 41.10 [a] (184)	160.35 ± 7.04 [a] (162)	0.073
TG (mg/dL)	174.61 ± 16.89 [a] (156)	151.90 ± 15.15 [a] (156)	173.98 ± 16.22 [a] (153)	170.01± 14.98 [a] (151)	0.811
HDL-C (mg/dL)	42.70 ± 1.47[a] (43.8)	43.30 ± 1.82 [a] (43.1)	44.30 ± 1.184 [a] (44.5)	45.92 ± 1.72 [a] (45.6)	0.449
LDL-C (mg/dL)	83.94 ± 8.95[ab] (89.4)	112.93 ± 10.12 [b] (110.7)	99.74 ± 6.63 [ab] (103)	80.42 ± 7.47 [a] (75.4)	0.033*
VLDL-C (mg/dL)	34.92 ± 3.37[a] (31.2)	30.38 ± 3.03 [a] (31.2)	34.79 ± 3.24 [a] (30.6)	34.0± 2.99 [a] (30.2)	0.811
Atherogenic index	0.55 ± 0.05[a] (0.56)	0.50 ± 0.04[a] (0.53)	0.51± 0.04 [a] (0.56)	0.50± 0.04[a] (0.55)	0.889

- Data were presented as Mean ± SE (Median)
*Significant difference between means using ANOVA -test at 0.05 level.
- Significant variants are denoted by different small letters.
- Non-significant variations are denoted by identical small letters.

The current study involved people who were divided in two groups according to the gender (healthy and obese) female and male groups, as shown in Tables 3, 4. The result showed an increase in BMI, WHR and HO-1 in obese compared with healthy female groups at p≤0.05. While the result showed an increase in GST activity in healthy compared with obese female groups at P≤0.05 as shown in Table 3.

Table 3. (Mean ± SE) for all parameters between healthy and obese female groups.

Groups Parameters	Healthy group female No. (39)	Obese group female No.(70)	P-value
BMI	24.49±0.36	35.85±0.51	0.0001**
Age	46.66±1.49	46.78±1.06	0.948
WHR	0.89±0.01	0.94±0.01	0.007**
FSG (mg/dL)	100.88± 2.38	95.66±2.25	0.140
TC (mg/dL)	169.02±9.78	172.46±5.46	0.740
TG (mg/dL)	165.29±15.21	156.18±10.09	0.607
HDL-C (mg/dL)	43.61±1.38	44.75±1.09	0.526
LDL-C (mg/dL)	93.38±8.46	96.47±5.48	0.751
VLDL-C (mg/dL)	33.05±3.04	31.23±2.01	0.607
Atherogenic index	0.52±0.04	0.47±0.03	0.472
GST	5.49±0.56	2.64±0.21	0.0001**
HO-1	1.83±0.04	2.01±0.06	0.047*

Also, the result showed increase in BMI, TG and VLDL in obese groups compared with healthy male groups at p≤0.05. While the result showed an increase in GST activity and LDL levels in healthy compared with obese male groups at P≤0.05 as shown in Table 4.

Table 4. (Mean ± SE) for all parameters between healthy and obese male groups

Groups Parameters	Healthy male Group No.(16)	Obese male Group No. (14)	P-value
BMI	24.45±0.46	34.18±0.86	0.0001**
Age	48.81±2.49	46±1.58	0.365
WHR	0.93±0.01	1.11±0.12	0.315
FSG (mg/dL)	93.85±3.50	99.93±6.01	0.376
TC (mg/dL)	176.75±10.98	156.61±8.27	0.163
TG (mg/dL)	164.68±16.22	251.36±36.78	0.032*
HDL-C (mg/dL)	41.21±1.84	46.81±2.97	0.112
LDL-C (mg/dL)	102.60±12.14	59.52±9.86	0.012*
VLDL-C (mg/dL)	32.93±3.24	50.27±7.35	0.032*
Atherogenic index	0.56±0.06	0.68±0.051	0.146
GST	4.75±0.76	2.23±0.27	0.007**
HO-1	1.81±0.07	1.95±0.08	2.222

The result indicated a statistically significant difference in groups (p≤0.05) in the serum heme oxygenase in unit (ng/ml) between obese groups (G3 and G4) and healthy groups (G1, G2) which were [(1.88 ± 0.04[ab]) (1.75 ± 0.05 [a]) (2.06 ± 0.09 [b]), (1.95 ± 0.04 [ab])] ng/ml respectively. Also, the current study showed a significant difference P≤ 0.05 in GST activity by unit (IU/L) between all study obese patients' groups (G1, G2) and healthy groups (G3, G4) were [(6.10 ± 0.57[c]), (4.120 ± 0.69 [b]), (2.44 ± 0.21 [a]), (2.71± 0.30 [ab])] IU/L respectively as shown in Table 5 and Fig. 1.

Table 5. (Mean ± SE) of Levels of heme oxygenase-1 and GST between study group

Groups Parameters	Normal weight Group(G1) No. (32)	Overweight Group(G2) No. (23)	Obesity Class-I Group(G3) No. (43)	Obesity Class II Group(G4) No. (41)	P-value
GST activity (U/mL)	6.10 ± 0.57[c] (5.46)	4.120 ± 0.69 [b] (3.12)	2.44 ± 0.21 [a] (2.08)	2.71± 0.30 [ab] (2.08)	0.0001**
Heme oxygenase-1 (ng/mL)	1.88 ± 0.04[ab] (1.94)	1.75 ± 0.05 [a] (1.77)	2.06 ± 0.09 [b] (1.99)	1.95 ± 0.04 [ab] (1.89)	0.044*

- Data were presented as Mean ± SE (Median)
*Significant difference between means using ANOVA -test at 0.05 level.
**Significant difference between means using ANOVA -test at 0.05 level.
- Significant variants are denoted by different small letters- Non-significant variations are denoted by identical small letters
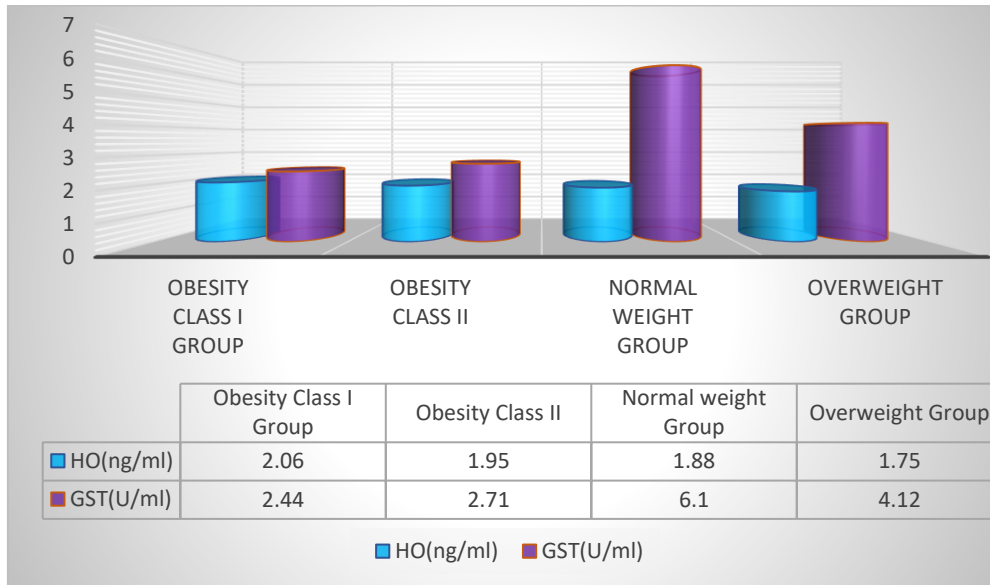
	Obesity Class I Group	Obesity Class II	Normal weight Group	Overweight Group
HO(ng/ml)	2.06	1.95	1.88	1.75
GST(U/ml)	2.44	2.71	6.1	4.12

Figure 1. Comparison between patients and control Groups HO-1 and GST.

The ROC curve analysis was employed in this study to differentiate between the obese groups and the control group. The findings from the ROC curve analysis of the 4 groups using the following criteria are shown in Table 6: - (HO=0.611), and (GST=0.767) show a large variation in obese and control, as shown in Figs. 2 and 3. The value of area under the curve of ROC curve for GST in obese person groups (0.767). Also, the cut off value for GST was (>93.27). The higher sensitivity and specificity were estimated for GST (58.73 %, 66.4 %, respectively) in obese patients, Fig. 3. The value of area under the curve of ROC curve for OH-1 in obese person groups (0.658). Also, the cut off value for HO-1 was (>80.36). The higher sensitivity and specificity were estimated for HO-1 (91.83 %, 26.7 %, respectively) in obese patients, Fig. 2.

Table 6. The ROC curve analysis of test variables for patients and control groups in HO-1and GST.

| Area Under the Curve					
Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
OH-1	.611	.048	.027	.518	.705
GST Activity	.767	.042	.000	.684	.849
The test result variable(s): OH-1, GST activity has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.					
a. Under the nonparametric assumption					
b. Null hypothesis: true area = 0.5					
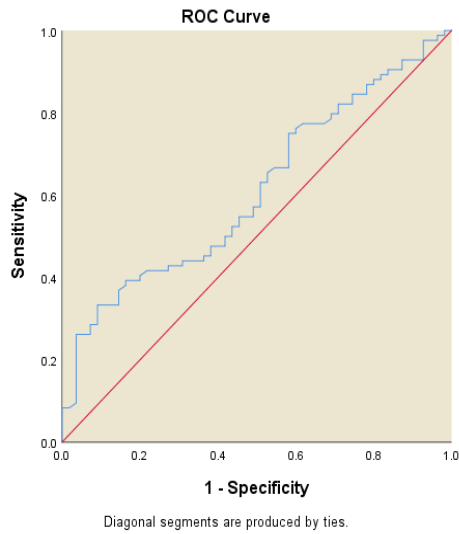
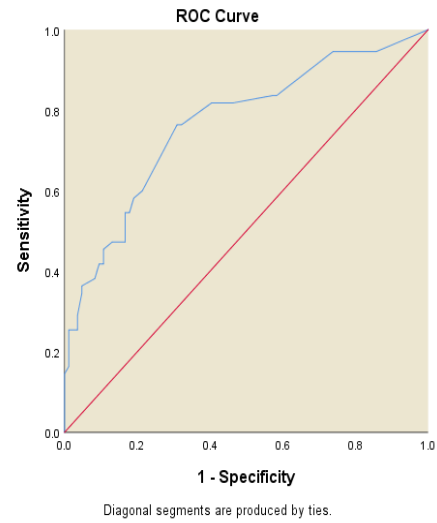**Figure 2. ROC curve analysis of HO for patients and control groups.**

Figure 3.ROC curve analysis of GST for patients and control groups.

Discussion

The present study showed significant differences in BMI and WHR between study groups at (P≤0.05)[(38.83±0.44[b]) (32.03±0.26[a]) (26.40±0.22[d]) (23.10±0.28[c])] ,[(1.01±0.03[b]) (0.92±0.02[ab]) (0.93±0.01[ab]) (0.88±0.01[a]] respectively ,the BMI refers to weight gain that can be caused by increasing muscle mass, bone density, or fat mass and WHR refers to the more belly fat available , the more likely it is that you may acquire through diseases like high cholesterol, diabetes, high blood pressure, or atherosclerosis . This agrees with Nadeem et al.,[20] who showed that BMI and WHR can be used to identify metabolic disorders in clinical and epidemiological investigations since they are straightforward and non-invasive, this also agrees with German adult men and women, as well as Australian adults aged 20 to 69, in whom found a high association between WC and BMI and a poor association between WHR and BMI[21]. Also, this study agrees with Tutunchi, H, et, al.,[22] who support our findings that WHR exhibited significant differences between subgroups but WHR revealed no significant difference between obese class I and obese class II, as seen in Table 1. The results also agree with Janjić J. et.al., [23]. Due concerning variations in body composition and the fact that obesity significantly affects body mass index, various body fat percentage (BF%) metrics produce more accurate results. The result showed

significance values with LDL-C, [(80,42±7.47[a]) (99.74±6.63[ab]) (112.93±10.12[b]) (83.94±8.95[ab])] respectively and non-significance values with other parameters. During times of high oxidative stress, a higher rate of reactive oxygen species (ROS) production in the mitochondria can lead to the formation of oxidized LDL. This result agrees with a study by Hazart et. al[24], who showed that there is non-significant value with (HDL-c, TG, TC, VLDL. and the results also agree with other study by Malaguarnera, L. et. al.,[25]. who found that here is a significant relationship between obesity and high bad fats (LDL), which cause stimulation of large amounts of enzyme HO-1 secretion in endothelial cells, smooth muscles, and connective tissue. The results are shown in the Table 4, 5. The result showed increase in BMI, WHR and HO-1in obese female [(24.49±0.36) (35.85±0.51)], [(0.89±0.01) (0.94±0.01)] [(1.83±0.04) (2.01±0.06)] respectively, and BMI, TG and VLDL in obese male [(24.45±0.46) (34.18±0.86)] [(164.68±16.22) (251.36±36.78)] [(32.93±3.24) (50.27±7.35)] compared with healthy group. Obesity in women is higher than that of men, especially in the waist-to-hip area. This may be due to the association of obesity with age and hormonal changes (postmenopausal age). While the result showed an increase in GST levels in healthy females and male group compared with obese groups. These results agree with

Rezaeipour M and Apanasenko G,[26]. who showed significant difference between BMI and obesity in female [26]. Also, the result showed increase LDL levels in healthy male group compared with obese male groups. Also, in the present study, HO-1level increased in obese groups (G3, G4) compared with healthy groups (G1, G2), because in higher obesity, the level of HO-1 increase becomes more active. As a regulator of cellular and tissue homeostasis, a modulator of immunological response and host defense. (HO-1), a stress protein and metabolic enzyme these results are shown in Table 5 and Fig. 1 [(2.06 ± 0.09^b) (1.95 ± 0.04^{ab}) (1.88 ± 0.04^{ab}) (1.75 ± 0.05^a)] ng/ml respectively. These results agree with Abraham, N.G. Junge , J.M and Drummond ,G.S (2016) [27]. HO-1 is related to an increase in cellular heme as well as inflammatory diseases such as atherosclerosis, hypertension, and stroke. The results also agree with Tirado, R. *et al.*, [28] who said that heme oxygenase (HO-1) is an anti-inflammatory enzyme that may become more active in morbid obesity, an enzyme with anti-inflammatory characteristics.The results are also in agreement with McClung, *et. al.*,[29]. who found the HO-1 also exerts positive effect by decomposing peroxiding heme, which is higher in obesity. The HO-1 antiadipogenic action is also mediated via CO, BV, and Fe^{+2}. Unfortunately, increasing ROS generation does not induce endogenous HO-1 expression, and as a result, obesity progresses unhindered. Therefore, increasing ROS generation causes HO-1 to be down regulated, which raises the likelihood of developing the metabolic syndrome caused by obesity. By inducing HO-1, a number of pharmaceutical substances that are currently employed in human or animal clinical trials can reduce inflammation. When inflammation starts, increasing HO-1 has no helpful anti-inflammatory benefits, but it has protective effects in myeloid and endothelial cells before inflammation begins. There is an increasing in level of GST in healthy groups (G1, G2) compared with obese groups (G3, G4). Obesity leads to an increase in the generation of oxidative stress and thus leads to the consumption of larger amounts of GST. The Glutathione–S- transferase (GST) is detoxification enzyme that is essential against oxidative damage in cellular. It is a powerful protective and antioxidant enzyme that works to remove toxins due to the effects of obesity and is consumed by the bodyas shown in Table 5 and Fig. 1[(6.10± 0.57^c) (4.120 ± 0.69^b) (2.44 ± 0.21^a) (2.71± 0.30^{ab})] IU/L respectively. The results are in line with a previous study conducted by Yilmaz, C (2020) [30.]who found that GST activity measured in patient with obesity in their family and with possible genetic background were lower compared to the other groups.

Conclusion

We conclude from this study that high levels of heme oxygenase-1 synchronized with low of Glutathione-S- transferase, clearly indicate their primary role in detoxification, protection against oxidative stress, and prevention of the development of metabolic diseases.

Acknowledgment

Authors' Declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for re-publication, which is attached to the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at University of Baghdad.

Authors' Contribution Statement

N.A.M. performed the acquisition of data analysis, interpretation, and drafting the manuscript while F.M.K. did the analysis, design of interpretation, revision and proof reading of the manuscript.

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تقييم الهيم اوكسيجينيز-1 والجلوتاثيون – اس-ترانسفيراز في مرضى عراقيين بدناء

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الخلاصة

هدفت الدراسة إلى الكشف عن العلاقة بين الهيم أوكسيجينيز-1 (HO-1) والجلوتاثيون- S- ترانسفيراز (GST) في مرضى السمنة ضد الاجهاد التأكسدي. اشتملت الدراسة على 139 عينة أعمارهم 35-65 سنة مقسمة إلى مجموعتين حسب الجنس مجموعات (نساء ورجال)، وأربع مجاميع حسب مؤشر كتلة الجسم (BMI)، وتم جمع عينات الدم في مستشفى اليرموك في الفترة من سبتمبر / 2022 إلى يناير / 2023. تم قياس بعض المتغيرات الكيميائية الحيوية لجميع مجموعات الدراسة، والتي تشمل: - تحديد مستويات HO-1 في الدم باستخدام تقنية ELISA, ملف الدهون GST activity بالطريقة الانزيمية، والسكر الصائم FSG بالطريقة الانزيمية. تم العثور على BMI نسبة الخصر إلى الورك WHR والبروتين الدهني منخفض الكثافة (LDL) زيادة معنوية بشكل ملحوظ 32.03 (38.83±0.44^b)[(23.10±0.28^c) (26.40±0.22^d) و±0.26^a) (0.88±0.01^a)] (0.93±0.01^{ab}) (0.92±0.02^{ab}) (1.01±0.03^b)] و [(83.94±8.95^{ab}) (112.93±10.12^b) (99.74±6.63^{ab}) (80.42±7.47^a)] على التوالي. بينما لا يوجد فرق معنوي مع المتغيرات الاخرى في ملف الدهون والسكر الصائم ومؤشر تصلب الشرايين. أن مستويات HO-1 كانت أعلى في المجموعة السمنة مقارنة بمجموعة الاصحاء ng/ml (1.75 ± 0.05^a)] (1.88 ± 0.04^{ab}) (1.95 ± 0.04^{ab}) (2.06 ± 0.09^b)]على التوالي. أظهر أن زيادة مستوى GST في الأصحاء مقارنة مع مجموعات السمنة (2.71±0.30 ^{ab}) (2.44±0.21^a) (4.120±0.69^b) (6.10± 0.57^c)] IU /L [على التوالي، اظهرت النتيجة زيادة في مؤشر كتلة الجسم، WHR وHO-1 في النساء البدينات ومؤشر كتلة الجسم، TG و VLDL في الذكور البدينين، بينما أظهرت زيادة في مستويات GST في النساء الأصحاء، أيضًا مستويات GST و LDL في مجموعة الذكور الأصحاء. نستنتج من هذه الدراسة. ان ارتفاع مستويات الهيم اوكسيجينيز-1 المتزامن مع انخفاض الجلوتاثيون- S- ترانسفيراز ، تبين دورهما الأساسي في إزالة السموم، والحماية من الإجهاد التأكسدي، والوقاية من تطور الأمراض الأيضية.

الكلمات المفتاحية: مؤشر كتلة الجسم، الجلوتاثيون-اس-ترانسفيراز، الهيم اوكسيجينيز-1,الدهون، السمنة، الاجهاد التأكسدي.