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Criteria for Gender Differences in Assessing the Indicators of the Metabolic Relationship Between the Formation of Obesity and the Severity of Oxidative Stress and Inflammation

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Abstract

Introduction. Obesity represents a multifactorial metabolic disorder linked to chronic inflammation and oxidative stress.
Purpose. To establish gender differences in the indicators of the combined assessment of the metabolic manifestation of oxidative stress and inflammation with different degrees of excess body weight in men and women.
Materials and methods. A cross-sectional study was conducted at Nineveh General Hospital, affiliated with the Medical Research Unit of Al-Nahrain University, including 234 participants divided equally among three BMI-based categories: normal weight (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (>30 kg/m^2). Each group comprised 26 control and 52 patient subjects. Blood samples were collected under aseptic conditions for measurement of CRP, superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase (CAT), and malondialdehyde (MDA) using ELISA techniques. Data were analyzed using SPSS 27 and GraphPad Prism with a significant threshold of $p<0.05$.
Results. The study included 78 subjects, with 26 assigned to the control subgroup and 52 to the patient subgroup. In the normal-weight category, serum CRP increased from 0.34±0.40 mg/dL in controls to 0.46±0.07 mg/dL in patients, while SOD activity rose from 1.2±0.07 to 2.6±0.05 U/mL and reached 6.39±0.10 U/mL in obese patients, indicating a marked oxidative imbalance. BMI in the normal-weight group showed a positive correlation with MDA ($r=0.294$, $p=0.035$), and GSH correlated strongly with CAT ($r=0.693$, $p<0.001$) and MDA ($r=0.608$, $p<0.001$). In contrast, the overweight group displayed weaker associations, with only a modest correlation between age and BMI ($r=0.295$, $p=0.034$).
Conclusion. The findings highlight obesity's strong association with inflammation and oxidative imbalance, underscoring the need for weight-management initiatives and regular biochemical screening to prevent chronic disease progression.
Keywords: oxidative stress, C-Reactive Protein, obesity, type 2 diabetes, cardiometabolic risk

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Критерии гендерных различий в оценке показателей метаболической связи формирования ожирения с состоянием выраженности оксидативного стресса и воспаления

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Резюме

Введение. Ожирение представляет собой мультифакторное метаболическое расстройство, связанное с хроническим воспалением и оксидативным стрессом.
Цель. Установить гендерные различия в показателях сочетанной оценки метаболического проявления оксидативного стресса и воспаления при разной степени избыточной массы тела у мужчин и женщин.
Материалы и методы. Исследование осуществлено на базе Больницы Ниневии в тесном сотрудничестве с представителями Научно-исследовательского медицинского подразделения Университета Аль-Нахраи. Обследованию подлежали 234 пациента, которые был разделены на три группы по индексу массы тела (ИМТ): с нормальными его показателями (18,5–24,9 кг/м^2), с избыточной массой тела (25–29,9 кг/м^2) и ожирением (>30 кг/м^2). Каждая группа включала 78 участников, из них 26 практически здоровых лиц, составивших подгруппу контроля, и 52 пациента с повышенной массой тела. Биохимический анализ крови включал в себя определение содержания С-реактивного белка (CRP), активности супероксиддисмутазы (SOD), глутатионпероксидазы (GPx), каталазы (CAT), уровня малонового диальдегида (MDA) и глутатиона (GSH) методами иммуноферментного анализа (ИФА, ELISA). Полученные данные анализировались в SPSS 27 и GraphPad Prism со статистической значимостью $p<0,05$.
Результаты. Уровень сывороточного CRP увеличивался с 0,34±0,40 мг/дл (у лиц подгруппы контроля) до 0,46±0,07 мг/дл у пациентов с ожирением, в то время как активность SOD возрастала с 1,2±0,07 до 2,6±0,05 Ед/мл у пациентов с избыточной массой тела и достигала 6,39±0,10 Ед/мл у лиц с ожирением, что указывает на причастность к его формированию воспалительного процесса и оксидативного стресса. В подгруппе лиц с нормальной массой тела показатели ИМТ демонстрировали положительную корреляцию с уровнем MDA ($r=0,294$, $p=0,035$), а уровень GSH значительно коррелировал с таковыми CAT ($r=0,693$, $p<0,001$) и MDA ($r=0,608$, $p<0,001$). Напротив, в группе пациентов с избыточной массой тела связи оказались менее выраженными, выявлена лишь умеренная корреляция между возрастом и ИМТ ($r=0,295$, $p=0,034$).

Заключение. Полученные данные иллюстрируют тесную связь ожирения с воспалением и состоянием оксидативного стресса, что служит основанием к реализации программ по снижению массы тела и осуществлению регулярного биохимического мониторинга с целью предотвращения прогрессирования в организме нарушений липидного обмена, влекущего за собой формирование сахарного диабета 2-го типа и кардиальной патологии.
Ключевые слова: окислительный (оксидативный) стресс, С-реактивный белок, ожирение, сахарный диабет 2-го типа, кардиометаболический риск

■ INTRODUCTION

Obesity has become one of the most critical global health challenges of the twenty-first century, affecting individuals across all age groups and socioeconomic backgrounds. It is a complex, multifactorial condition characterized by excessive accumulation of adipose tissue that results from an imbalance between caloric intake and energy expenditure. According to the World Health Organization (2024), the global prevalence of obesity has tripled since 1975, with more than 1.9 billion adults classified as overweight and over 650 million as obese. In Iraq, a rapid growing of obesity percentage became a great disaster that may threaten the community health status. This growing epidemic is strongly associated with numerous metabolic disturbances, including insulin resistance, type 2 diabetes mellitus, hypertension, dyslipidemia, and cardiovascular diseases. However, recent evidence suggests that the biological mechanisms underlying these complications may also involve chronic low-grade inflammation and increased oxidative stress, both of which play central roles in the pathogenesis of obesity-related disorders [1–5].

C-Reactive Protein (CRP) is a key biomarker of systemic inflammation synthesized primarily by hepatocytes in response to interleukin-6 and other pro-inflammatory cytokines. Elevated CRP concentrations are consistently linked to obesity and metabolic syndrome, reflecting the inflammatory burden imposed by adipose tissue dysfunction. Adipocytes, particularly in visceral fat depots, secrete cytokines such as tumor necrosis factor-α and interleukin-1β, which stimulate hepatic CRP production [6–8].

As well, the oxidative stress defined as an imbalance between reactive oxygen species (ROS) generation and antioxidant defense mechanisms exacerbates cellular damage and contributes to lipid peroxidation, protein modification, and DNA injury. The interplay between oxidative stress with different oxidative markers and inflammation forms a vicious cycle that amplifies metabolic disturbances, making their combined evaluation essential in understanding the biochemical landscape of obesity [9–13].

Gender differences play a significant role in modulating obesity-related metabolic pathways. Men and women exhibit distinct fat distribution patterns, hormonal profiles, and immune responses that can influence inflammatory and oxidative stress markers differently. Estrogen, for instance, possesses antioxidant properties and modulates lipid metabolism, potentially offering some protection to premenopausal women against oxidative damage. Conversely, the decline in estrogen after menopause is associated with increased adiposity and inflammation. Males, on the other hand, tend to accumulate more visceral fat, which is metabolically active and strongly linked to higher CRP levels and

oxidative stress indices. Therefore, investigating gender-specific biochemical variations provides deeper insight into the mechanisms linking obesity with systemic inflammation and oxidative imbalance [14–21].

Understanding comprehensive biochemical analysis that explores how obesity correlates with CRP levels and oxidative stress parameters across genders may reveal critical pathways underlying metabolic risk. Such data could guide clinicians in tailoring prevention and management programs based on gender-specific vulnerabilities, improving outcomes in obesity-related diseases [22–24].

■ PURPOSE OF THE STUDY

To establish gender differences in the indicators of the combined assessment of the metabolic manifestation of oxidative stress and inflammation with different degrees of excess body weight in men and women.

■ MATERIALS AND METHODS

Population and Setting of the Study

The study was carried out at Nineveh General Hospital, affiliated with the Medical Research Unit of Al-Nahrain University. The study included a total of 234 participants. The inclusion criteria was individuals who did not have Type 2 diabetes (T2DM) or any other chronic diseases. While subjects were removed if their waist and neck measurements could not be obtained by the researchers.

Body Mass Index Measurement and Interpretation

Each group consisted of 78 participants with different ages. There were three primary categories: the Normal Weight Group (BMI 18.5–24.9), the Overweight Group (BMI 25–29.9), and the Obese Group [BMI >30]. Participants in each BMI category were then classified into two subgroups: control and patients. The control subgroups consisted of 26 people each, whereas the patients' subgroups consisted of 52 persons of each group.

Antioxidant Assessment

The subjects' blood samples were obtained using aseptic syringes. The samples were extracted from the individuals' veins, usually from the arm. Every blood sample was obtained and placed into a properly labeled sterile tube. Following collection, blood samples were promptly stored in a refrigerator at a temperature of 4 °C.

The plate has been pre-coated with Malondialdehyde (MDA) protein. Samples are introduced into the suitable wells of a microtiter plate. Subsequently, Avidin is attached to HRP. Then TMB Solution was added. The resulting change in color is then quantitatively assessed using spectrophotometry at a specific wavelength of 532 nm ± 10 nm.

The Human SOD ELISA Kit functions based on the premise of a sandwich enzyme immunoassay. This technique utilizes a plate that has been previously coated with an antibody that specifically targets SOD. Subsequently, a biotin-conjugated antibody that targets SOD is introduced to create a complex between the antibody and the antigen. Subsequently, HRP conjugated to Avidin is introduced. Following incubation with the TMB Solution. The process is stopped using a stop solution, and the intensity of the color measured at 450 nm. The plate included in this kit was pre-coated with Human GPX protein. Standards or samples were introduced into the suitable wells, together with a

biotin-conjugated antibody that is specific to Human GPX. Subsequently, HRP conjugate was introduced. Then TMB Solution was added. The resulting change in color was then detected at 450 nm ± 10 nm.

The plate included in the kit was already coated with an antibody that specifically targets CAT. CAT-specific biotin-conjugated antibodies were introduced to the relevant wells. Subsequently, HRP conjugate was introduced. Upon the addition of the TMB Solution. The enzyme-substrate reaction was stopped, and the alteration in color was quantified using spectrophotometry at 450 nm±10 nm.

C-reactive Protein Assay

The CRP was evaluated by Human CRP [C-Reactive Protein] ELISA Kit (Cat: ELK1040), ELK Biotechnology CO., Ltd (Chian). The Human C-Reactive Protein ELISA Kit (Cat: ELK1040) is specifically developed for the precise and quantitative determination of CRP levels in humans. This kit employs a sandwich enzyme immunoassay method, in which a microtiter plate is coated in advance with an antibody that specifically targets human CRP. Additionally, a biotin-conjugated antibody is introduced, and the magnitude of the color is quantified at 450 nm.

It was calculated the average of the readings that were taken more than once for each Standard, Control, and Sample. The CRP content was plotted on the y-axis, and the absorption was plotted on the x-axis to make a standard curve (Fig. 1).

Statistical Analysis

The statistical analyses were performed utilizing SPSS (Statistical Package for the Social Sciences) version 27.0nd GraphPad Prism. A significant threshold of $p<0.05$ was set for all statistical tests. ANOVA tests were employed to find the correlations between and in groups.

■ RESULTS

From a total of 78 subjects for each weight group, divided into 62 for controls and 52 for patients. And upon assessing the obesity degrees, the BMI was measured (Fig. 2).

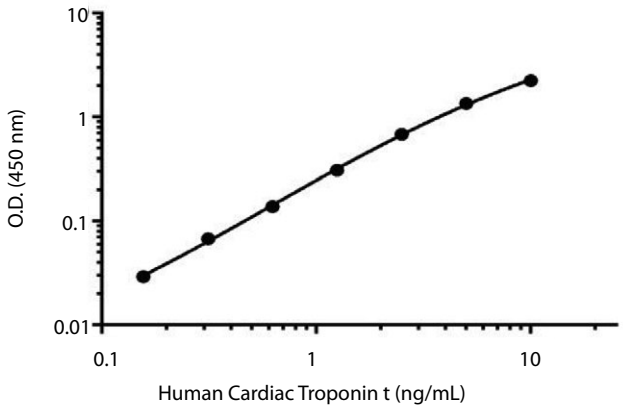

Fig. 1. The CRP standard curve

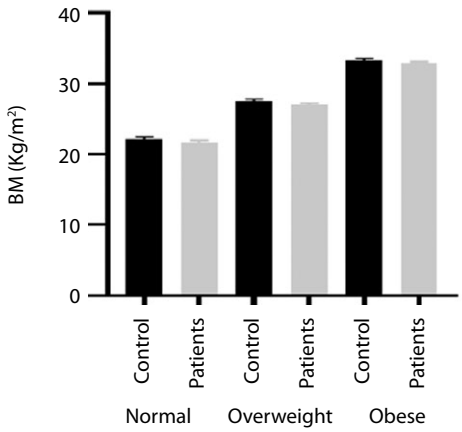

Fig. 2. The distribution of BMI among the weight-based groups

The gender and BMI correlation was investigated and set in the Table 1. The weight groups (normal, overweight and obesity) presented that there are a significant difference in their BMI ($p<0.05$). There are significant differences in gender variability between each group ($p>0.05$) while there is no significant difference between normal, overweight, and obesity groups ($p<0.05$).

CRP as Inflammatory Marker

Within the normal weight category, there is a notable and meaningful positive relationship between age and CRP. This suggests that as individuals get older, their CRP levels tend to increase, indicating a rise in inflammation. There is no significant correlation between age and other parameters in the normal weight group. Gender exhibits notable inverse connections with many factors among different weight categories. Within the normal weight group,

BMI exhibits strong positive relationships with several indicators, indicating its crucial significance in cardiovascular well-being. Within the normal weight category, there is

Table 1
Comparison of gender and BMI among three weight-based groups (n=78 for each, Controls=62 and Patients=52)

		Normal weight group		Overweight group		Obesity group	
		Control, n=26	Patient, n=52	Control, n=26	Patient, n=52	Control, n=26	Patient, n=52
Gender	Male, n [%]	14 (53.8%)*	27 (51.9%)	14 (53.8%)*	27 (51.9%)	14 (53.8%)*	27 (51,9%)
	Female, n [%]	12 (46.2%)	25 (48.1%)	12 (46.2%)	25 (48.1%)	12 (46.2%)	25 (48,1%)
BMI [kg/m²]		22.18±0.35	21.75±0.25	27.51±0.29	27.09±0.19	33.35±0.28	32.93±0.21

Notes: * p<0,05 compared of patients and control groups for each weight group using two-way ANOVA test.

Table 2
The correlation between CRP as marker and gender variability among different weight-based groups

		Normal weight group		Overweight group		Obesity group	
		Control	Patient	Control	Patient	Control	Patient
Gender	Male, n [%]	14 (53.8%)	27 (51.9%)	14 (53.8%)	27 (51.9%)	14 (53.8%)	27 (51,9%)
	Female, n [%]	12 (46.2%)	25 (48.1%)	12 (46.2%)	25 (48.1%)	12 (46.2%)	25 (48,1%)
CRP [mg/dl]		0.34±0.4	0.46±0.07	0.34±0.04#	0.59±0.11*#	0.42±0.06*	0.72±0.16*#

Notes: * p<0.05 compared of patients and control groups for each weight group using two-way ANOVA test; # p<0.05 compared of patients groups across the weight-based groups using two-way ANOVA test.

a positive correlation between BMI and CRP. This suggests that higher BMI is linked to heightened inflammation, cardiac strain, and an elevated risk of heart failure. Within the overweight group, there is a positive association between BMI and CRP. This suggests that greater BMI is linked to increased inflammation and cardiac stress. Within the obesity group, there are notable positive associations between BMI and CRP. These findings suggest that greater BMI in obese individuals is closely linked to heightened levels of inflammation, cardiac strain, and an elevated risk of heart failure.

CRP, a marker of inflammation, exhibits strong positive associations with several parameters. The higher levels of inflammation are linked to increased cardiac stress and a greater risk of heart failure. Within the overweight cohort, the increased inflammation is linked to heightened cardiac stress (Table 2).

Oxidative Stress Markers

In the normal weight control group, when comparing the biochemical parameters, we noticed that the most significant biochemical parameters is GPx. While in the normal weight Patient group, when comparing the biochemical parameters, we noticed that the most significant biochemical parameters is GPx and CAT. There may be a priority in importance and influence of GPx more than CAT by a small margin. The rest of the biochemical parameters were less significant, indicating that their influence was less compared to GPx and CAT (Table 3).

So, in the Overweight control group, when comparing the biochemical parameters, we noticed that the most significant correlations of biochemical parameters is GPx and CAT. There may be a priority in importance and influence of GPx more than CAT by a small margin. The rest of the biochemical parameters were less significant, indicating that their influence was less compared to GPx and CAT. While in the Overweight Patient group, when comparing the biochemical parameters, we noticed that the most significant biochemical parameters were GPx and CAT. There may be a priority in importance and influence of GPx more than CAT by a small margin. The rest of the biochemical parameters were less significant, indicating that their influence was less compared to GPx and CAT.

In the Obesity control group, when comparing the biochemical parameters, we noticed that the most significant biochemical parameters is GPx. While in the Obesity Patient group, when comparing the biochemical parameters, we noticed that the most significant biochemical parameters is GPx. An increase in the level of oxidative stress in patients with diabetes with an increase in body mass index was proven, as indicated by

Table 3
The correlation between oxidative stress markers marker and gender variability among different weight-based groups

		Normal weight group		Overweight group		Obesity group	
		Control	Patient	Control	Patient	Control	Patient
Gender	Male, n [%]	14 (53.8%)	27 (51.9%)	14 (53.8%)	27 (51.9%)	14 (53.8%)	27 (51,9%)
	Female, n [%]	12 (46.2%)	25 (48.1%)	12 (46.2%)	25 (48.1%)	12 [46.2%]	25 (48,1%)
SOD [U/mL]		1.2±0,07	2.6±0.05	1.34±0.08#	4.19±0.04*#	1.9±0.42#	6.39±0.10*#
GPx [nmol/mL]		22,5±0,54	35.5±0.42	24.1±0.26#	49.6±0.36*#	42.1±0.08*#	68.5±0.28*#
CAT [U/mL]		42.42±9.6	57.71±1.4	35.75±2.73*#	32.7±1.35*#	25.7±31*#	14.9±9.1*#
MDA [μM]		1.43±0.28	1.7±0.11	1.10±0.04*#	1.90±0.05*#	2.58±0.26*#	3.4±0.94*#

Notes: * p<0.05 compared of patients and control groups for each weight group using two-way ANOVA test; # p<0.05 compared of patients groups across the weight-based groups using two-way ANOVA test.

an increase in the content of lipid peroxidation products, superoxide dismutase activity, glutathione peroxidase.

It has been noted that there were no significant relationships between age and oxidative stress indicators GSH, SOD, CAT, or MDA. This implies that age may not exert a significant influence on oxidative stress measures in persons with normal weight. There was a strong negative relationship between gender and BMI, with a correlation coefficient of –0.431. The BMI exhibited a notable positive connection with the CAT, indicating that higher BMI is linked to elevated levels of CAT. The BMI exhibited a non-significant but positive connection with GSH and MDA, suggesting a possible inclination towards elevated oxidative stress as BMI increases. GSH exhibited strong positive associations with CAT and MDA, and a notable negative connection with SOD. The SOD enzyme showed strong negative associations with GSH and MDA, and a substantial positive connection with CAT. This suggests that elevated SOD levels are linked to decreased levels of GSH and

Table 4
Correlation between all oxidative stress parameters in normal wright group

Normal weight group							
		Age	BMI	GSH	SOD	CAT	MDA
Age	Pearson Correlation	1	0.27	0.136	–0.003	0.154	0.153
	Sig. (2-tailed)		0.053	0.335	0.981	0.277	0.278
BMI	Pearson Correlation	0.27	1	0.172	–0.057	0.294*	0.205
	Sig. (2-tailed)	0.053		0.224	0.69	0.035	0.145
GSH	Pearson Correlation	0.136	0.172	1	–0.431**	0.693**	0.608**
	Sig. (2-tailed)	0.335	0.224		0.001	0	0
SOD	Pearson Correlation	–0.003	–0.057	–0.431**	1	–0.287*	–0.390**
	Sig. (2-tailed)	0.981	0.69	0.001		0.039	0.004
CAT	Pearson Correlation	0.154	0.294*	0.693**	–0.287*	1	0.636**
	Sig. (2-tailed)	0.277	0.035	0	0.039		0
MDA	Pearson Correlation	0.153	0.205	0.608**	–0.390**	0.636**	1
	Sig. (2-tailed)	0.278	0.145	0	0.004	0	

MDA, as well as reduced levels of CAT. CAT enzyme exhibited strong positive associations with GSH and MDA, as well as a notable negative connection with SOD. The level of MDA showed strong positive associations with GSH and CAT, and a notable negative association with SOD (Table 4).

There was a clear and meaningful connection between age and BMI, with a positive correlation coefficient of 0.295 and a significant level of $p<0.05$. Gender demonstrated a significant inverse association with BMI, but did not exhibit significant relationships with GSH, SOD, CAT, or MDA. The overweight group did not exhibit any significant relationships between BMI and GSH, SOD, CAT, or MDA. This suggests that BMI may not be a reliable

Table 5
Correlation between all oxidative stress parameters in normal wright group

Overweight group							
		Age	**BMI**	**GSH**	**SOD**	**CAT**	**MDA**
Age	Pearson Correlation	1	0.295*	–0.1	0.081	–0.021	0.074
	Sig. (2-tailed)		0.034	0.64	0.568	0.885	0.602
BMI	Pearson Correlation	0.295*	1	0.05	0.1	0.014	–0.004
	Sig. [2-tailed]	0.034		0.72	0.48	0.923	0.979
GSH	Pearson Correlation	–0.07	0.05	1	0.204	–0.254	0.056
	Sig. (2-tailed)	0.644	0.724		0.146	0.069	0.694
SOD	Pearson Correlation	0.081	0.1	0.2	1	–0.183	–0.077
	Sig. (2-tailed)	0.568	0.48	0.15		0.193	0.587
CAT	Pearson Correlation	–0.02	0.014	–0.3	–0.183	1	0.01
	Sig. (2-tailed)	0.885	0.923	0.07	0.193		0.946
MDA	Pearson Correlation	0.074	–0.004	0.06	–0.077	0.01	1
	Sig. (2-tailed)	0.602	0.979	0.69	0.587	0.946	

Table 6
Correlation between all oxidative stress parameters in normal wright group

Obesity group							
		Age	**BMI**	**GSH**	**SOD**	**CAT**	**MDA**
Age	Pearson Correlation	1	0.131	–0.3	0.163	–0.137	–0.16
	Sig. (2-tailed)		0.356	0.05	0.247	0.334	0.257
BMI	Pearson Correlation	0.131	1	0.15	–0.004	0.059	–0.179
	Sig. (2-tailed)	0.356		0.29	0.979	0.677	0.205
GSH	Pearson Correlation	–0.27	0.151	1	–0.262	0.289*	–0.06
	Sig. (2-tailed)	0.054	0.287		0.061	0.038	0.67
SOD	Pearson Correlation	0.163	–0.004	–0.3	1	0.087	–0.049
	Sig. (2-tailed)	0.247	0.979	0.06		0.539	0.728
CAT	Pearson Correlation	–0.14	0.059	0.289*	0.087	1	–0.066
	Sig. (2-tailed)	0.334	0.677	0.04	0.539		0.64
MDA	Pearson Correlation	–0.16	–0.179	–0.1	–0.049	–0.066	1
	Sig. (2-tailed)	0.257	0.205	0.67	0.728	0.64	

Notes: * a significant correlation at $p<0.05$; ** a significant correlation at $p<0.01$.

predictor of oxidative stress in overweight persons. No significant associations were seen between the levels of GSH, SOD, CAT, or MDA in the overweight group. The obesity group did not exhibit significant associations between BMI and GSH, SOD, CAT, or MDA. The study found a strong positive association between GSH and CAT, indicating that higher levels of GSH are linked to higher levels of CAT. No significant associations were seen between the levels of SOD, CAT, or MDA in the obese group (Table 5, 6).

■ DISCUSSION

Obesity is increasingly recognized as a chronic inflammatory and oxidative disorder contributing to metabolic and cardiovascular diseases. The present study aimed to analyze biochemical variations between males and females in the correlation among obesity indices, C-reactive protein concentrations, and oxidative stress markers. The findings demonstrate that elevated body mass index was positively associated with higher CRP concentrations and increased oxidative stress markers such as superoxide dismutase, glutathione peroxidase, catalase, and malondialdehyde. These outcomes support the concept that excessive adiposity exacerbates both inflammation and oxidative imbalance regardless of gender.

In this study, it has been observed that CRP levels rose significantly with BMI agrees with several investigations linking obesity with chronic low-grade inflammation [1–4]. Adipocytes release tumor necrosis factor-α and interleukin-6 that stimulate hepatic CRP synthesis [5–7]. Mohamed-Ali et al. first described adipose tissue as a major cytokine source [8]. Visser et al. found a positive trend between CRP and adiposity even among healthy adults [9]. Similar relationships were demonstrated by Park et al. in Korean adults [10]. Ridker et al. N observed that inflammatory biomarkers predict cardiovascular risk independently of lipid profile [11].

The lack of marked gender differences in CRP within our data corresponds with results from Thorand et al. who reported that gender variance diminishes after adjusting for adiposity [12]. Khera et al. similarly found minimal sex-based differences in inflammatory response once fat distribution was considered [13]. These findings imply that obesity per se, rather than sex hormones, drives systemic inflammation.

The positive correlations between CRP observed in overweight and obese subjects in the present study underscore the cardiometabolic burden of obesity. Danesh et al. demonstrated a direct relation between CRP and vascular injury markers [14]. Bahrami et al. linked obesity-associated inflammation with subclinical myocardial dysfunction [15].

Elevated oxidative stress indices in our cohort agree with evidence that obesity enhances reactive oxygen species generation and reduces antioxidant reserves [16–19]. Furukawa et al. reported increased lipid peroxidation with higher BMI [20]. Marseglia et al. emphasized GPx activation as a compensatory antioxidant response [21]. Vincent et al. confirmed that chronic oxidative load in obesity elevates GPx while impairing SOD activity [22]. The reciprocal patterns of GPx and CAT observed here are consistent with the adaptive antioxidant cycle described by Dandona et al. wherein oxidative stress stimulates both enzymatic and inflammatory cascades [23].

The interrelation between CRP and oxidative stress in this study reflects the feedback loop highlighted by Roberts and Sindhu who proposed that reactive oxygen species induce cytokine expression, perpetuating inflammation [24]. Positive associations between CRP in obese individuals reinforce the cardiac oxidative-inflammatory hypothesis supported

by Wang et al. [25]. Our data therefore confirm that BMI is a principal determinant of both inflammatory and oxidative status.

Comparable biochemical disruptions have been reported regionally. Al-Zubaidi HJ, Al-Badr AA, Al-Hilli MA, Al-Zubaidi MJ and Al-Hamdani AA showed elevated MDA and reduced antioxidant enzymes in Iraqi adults with obesity [26]. Al-Fartosi et al. documented similar findings in Basrah populations [27]. These outcomes parallel international results by Khan et al. [28] and El-Shahat et al. [29]. Collectively, these studies affirm that oxidative stress and inflammation are universal consequences of adiposity.

■ CONCLUSIONS

1. Obesity in both genders was associated with higher inflammatory and oxidative indices, while gender had a minor influence compared with BMI.
2. The integration of CRP and antioxidant markers offers a valuable biochemical approach to assess cardiometabolic risk.
3. There was a strong and positive relationship between CRP levels and getting older in age and higher body mass index (BMI).
4. Glutathione peroxidase (GPx), as an oxidative stress marker, is the highest and most significant marker among normal weight groups.
5. Age was only correlated with GPx, not correlated with any of the oxidative stress markers (GSH, SOD, catalase, and MDA.

Future national initiatives in Iraq should emphasize large-scale awareness programs on the health hazards of obesity, promote weight-loss interventions, and encourage routine biochemical screening for CRP and oxidative markers to enable early detection of chronic disease risk. More health campaigns in Iraq is crucial to be conducted to declare about importance of weight loss and regular screening for population about their biochemical markers related to chronic diseases.

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