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Effect of maternal body mass index on inflammatory biomarkers and dynamic thiol-disulfide homeostasis during pregnancy

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Abstract

Background The aim of this study was to investigate the relationship between maternal body mass index (BMI), a modifiable factor during the reproductive period, and inflammation and oxidative stress by assessing dynamic thiol-disulfide homeostasis (TDH) in both the mother and fetus.

Method This prospective cohort study was conducted between May and June 2024 at a tertiary obstetric care center. The inclusion criteria consisted of healthy pregnant women aged over 18 years, between 37 and 41 weeks of gestation, who had not used medications other than iron and folic acid supplements, with newborns birth weight between 2,500 grams (g) and 4,500 g, and Apgar scores ≥ 7 at the 5th minute after birth. Maternal peripheral blood (5 mL) was collected at delivery admission, and 3 mL of fetal blood was obtained from the umbilical cord after delivery. Participants ($n = 125$) were categorized into three BMI-based groups: (1) non-obese at both pre-pregnancy and delivery (BMI < 30 kg/m^2, $n = 72$); (2) non-obese at pre-pregnancy but gained weight to a BMI classified as obese at delivery (BMI < 30 kg/m^2 pre-pregnancy, ≥ 30 kg/m^2 at delivery, $n = 29$); and (3) obese at both pre-pregnancy and delivery (BMI ≥ 30 kg/m^2, $n = 24$).

Results Maternal serum native thiol (SH) (306.21 ± 49.19 µmol/L vs. 270.9 ± 60.12 µmol/L vs. 276.9 ± 59.18 µmol/L, $p = 0.004$) and total thiol (SH + SS) (337.88 ± 52.43 µmol/L vs. 303.8 ± 62.13 µmol/L vs. 306 ± 58.01 µmol/L, $p = 0.006$) levels were significantly higher in the non-obese at both pre-pregnancy and delivery group compared to the other groups. Disulfide (SS) levels and thiol-disulfide ratios (SS/SH, SS/total thiol, and SH/total thiol) showed no significant differences among groups ($p > 0.05$, for all). In fetal cord blood, SH, SS, SH + SS levels, and thiol-disulfide ratios were not significantly different among the groups ($p > 0.05$, for all).

Conclusion Maternal obesity, whether longstanding or newly developed during pregnancy, disrupts TDH and reduces antioxidant capacity, increasing susceptibility to oxidative damage and may affect maternal and fetal health.

Clinical trial number Not applicable.

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Keywords Inflammation, Oxidative stress, Body mass index, Obesity, Pregnancy

Introduction

The worldwide prevalence of overweight and obesity has become a significant public health concern, posing a substantial burden on global health [1]. Treating obesity remains a challenge, despite extensive efforts and substantial expenses. According to the 2022 World Health Organization (WHO) report, 1 in 8 people ise classified as obese [2]. The increasing prevalence of obesity, especially among women of reproductive age, is alarming [3]. Obese pregnant women are at higher risk of experiencing various adverse pregnancy outcomes and birth complications [4]. A meta-analysis has determined that pre-pregnancy weight loss is beneficial for achieving optimal pregnancy outcomes [5]. One possible explanation for the connection between maternal obesity and health problems in newborns and children is the rise in oxidative stress (OS) and inflammation that is typically seen in non-pregnant individuals who are obese [6–8]. Maternal obesity has been proposed to contribute to negative effects in offspring through various mechanisms, such as inflammation, insulin resistance, lipotoxicity, increased production of adipocytokines, and OS [9, 10]. Recent data also indicate that OS is linked to the disruption of adipocytokines and lipotoxicity in these mechanisms [9, 11]. Given the role of OS in the disease processes associated with obesity, it has been proposed that similar alterations may occur in the umbilical cord blood of offspring born to obese mothers [12]. Multiple studies have reported an imbalance between the generation of reactive oxygen species (ROS) and the protective mechanisms against OS in the maternal and feto-placental units of pregnancies in obese women [13–16].

OS results in the accumulation of reactive oxygen and nitrogen species, which attack the sulfhydryl groups of many molecules, leading to the formation of disulfide bonds [17]. Thiols are organic compounds that contain sulfhydryl (-SH) groups, which play a critical role in protecting against OS. Under oxidative stress, sulfhydryl groups oxidize to form disulfide (-SS) bonds, but these bonds can revert to thiol groups when oxidative stress subsides [18–20]. This dynamic cycle maintains a balanced state known as dynamic thiol-disulfide homeostasis (TDH) [21]. TDH, a marker of OS, can be assessed using a spectrophotometric method developed by Erel and Neselioglu, which is both simple and cost-effective [22]. Disruptions in thiol-disulfide homeostasis have been strongly associated with obesity [23, 24].

Life course epidemiology is a valuable method that can analyze factors before conception and their impact on the health of both mothers and children. This approach considers the timing and duration of exposures, as well as their potential long-term or cumulative effects. From this viewpoint, adolescence can be considered a critical period for predicting future pregnancy outcomes, as unhealthy behaviors like smoking and poor diet often begin during the teenage years [25]. Despite its significance, there is limited knowledge about the relationship between body mass index (BMI) and its influence on subsequent pregnancy outcomes [4]. The aim of this study is to evaluate how BMI, one of the modifiable maternal factors during the reproductive period, affects the relationship between inflammation and oxidative balance via quantifying the balance of thiol and disulfide compounds in the mother and fetus.

Materials and methods

This prospective cohort study was conducted between May and June 2024 at the Health Sciences University Etlik Zubeyde Hanim Maternity, Teaching and Research Hospital in Ankara, Turkey, a tertiary obstetric care center. Ethical approval was obtained from the Ankara Etlik City Hospital Ethics Committee (approval number: AESH-BADEK-2024-418). Written informed consent was obtained from all participants prior to their inclusion in the study. The research was carried out in full compliance with the principles outlined in the Declaration of Helsinki.

Women aged over 18 years who were admitted to the delivery room at the Health Sciences University Etlik Zubeyde Hanım Maternity, Teaching and Research Hospital were eligible for inclusion. The inclusion criteria consisted of healthy pregnant women aged over 18 years, between 37 and 41 weeks of gestation, who had not used any medications other than iron and folic acid supplements, with newborns birth weight between 2,500 grams (g) and 4,500 g, and Apgar scores ≥ 7 at the 5th minute after birth.

The exclusion criteria included type I or II diabetes mellitus, gestational diabetes mellitus, hypertensive disorders, intrahepatic cholestasis of pregnancy, premature rupture of membranes (PROM) lasting more than 24 hours, thyroid dysfunction (hyperthyroidism or hypothyroidism), chronic renal or liver disease, cardiovascular diseases, autoimmune disorders, and rheumatologic diseases. Additional exclusions included fetal growth restriction, polyhydramnios, oligohydramnios, multiple pregnancies, and a maternal history of smoking or alcohol consumption. Mothers who underwent CS due to maternal/fetal emergency indications —such as preeclampsia, eclampsia, placental anomalies, placental abruption, cord prolapse, or fetal distress—were

also excluded. Participants were excluded if they had received labor induction medication, undergone operative vaginal delivery (e.g., vacuum or forceps extraction), or received anesthesia during vaginal delivery. Furthermore, pregnant women with labor-related complications, including fever, meconium-stained amniotic fluid, chorioamnionitis, or intrauterine fetal demise, were excluded. Neonates requiring admission to the neonatal intensive care unit (NICU) were also not included in the study.

Gestational age for all participants was confirmed through routine ultrasonographic examination conducted during the first trimester. During the study period, 138 eligible pregnant women were identified, of whom 13 mother-infant pairs were excluded due to hemolysis in their serum samples. The remaining 125 mother-infant dyads were categorized into three groups based on maternal body mass index (BMI) at pre-pregnancy and at delivery: (Group 1) non-obese at both pre-pregnancy and delivery (BMI < 30 kg/m², $n = 72$); (Group 2) non-obese at pre-pregnancy but gained weight to a BMI classified as obese at delivery (pre-pregnancy BMI < 30 kg/m² and delivery BMI ≥ 30 kg/m², $n = 29$); and (Group 3) obese at both pre-pregnancy and delivery (BMI ≥ 30 kg/m², $n = 24$).

Data collected included maternal BMI at delivery, weight at delivery, height, weight gained during pregnancy, pre-pregnancy weight, and pre-pregnancy BMI. The change in BMI during pregnancy was calculated as Delta BMI = BMI at delivery – pre-pregnancy BMI. Additionally, detailed obstetric and medical histories were documented for all participants.

Sample collection and analysis

In addition to routine hemogram and biochemistry blood samples, 5 mL of peripheral venous blood was taken from the mothers admission to the delivery room. Immediately after delivery, the umbilical cord was clamped and 3 mL of fetal blood serum was collected from the umbilical cord. The samples were processed within 10 min of collection by centrifugation at 5,000 revolutions per minute (2236×g) for 10 min and subsequently stored at -80 °C until further analysis. Serum levels of native thiol (SH) (µmol/L), disulfide (SS) (µmol/L), and total thiol (SH + SS) (µmol/L) were measured using the method described by Erel and Neselioglu [22]. Ratios of SS/SH (%), SS/total thiol (%), and SH/total thiol (%) were also calculated for further analysis.

Complete blood count (CBC) was performed using an automated hematology analyzer (Mindray BC-6000, Shenzhen, China). Albumin and total protein levels were measured using a clinical chemistry analyzer (Beckman Coulter AU 680, California, USA).

Statistical analysis

The data were analyzed using IBM® SPSS® Statistics version 29.0 (Statistical Package for the Social Sciences) in New York, USA. The normality of the distributions was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests prior to analysis. The numerical data ara presented either as the median with the interquartile range (IQR) or as the mean ± standard deviation (SD). Categorical variables are presented as numbers (percentages), and the Chi-square test was applied for their analysis. In this study consisting of three independent groups (non-obese at both pre-pregnancy and delivery, non-obese at pre-pregnancy but obese at delivery, and obese at both pre-pregnancy and delivery group), one-way ANOVA test was used for parametric numerical variables and Kruskal Wallis-H test was used for non-parametric variables. Post-hoc analyses were performed with the Bonferroni and Tamhane T2 test for comparisons between subgroups for significant variables. The Spearman's correlation test was used to assess the relationship between independent variables. A significance level of $p < 0.05$ was considered statistically significant. A power analysis was performed using G*Power version 3.1.9.7 (Franz, Universität Kiel, Germany). The study determined that a sample size of at least 21 patients in each group achieved approximately 80% power to detect a difference in thiol-disulfide homeostasis between pregnant women with and without obesity with a medium effect size (Cohen's d = 0.5).

Results

Maternal and newborn characteristics and perinatal outcomes of the participants are shown in Table 1. No statistically significant differences were observed in terms of maternal age, gravida, parity, gestational age at delivery, newborn gender, birth weight, body length of newborns, 1st and 5th minute Apgar scores of the newborns. In addition, white blood cell (WBC), hemoglobin, hematocrit, neutrophil, lymphocyte, platelet, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), mean corpuscular volume (MCV), red cell distribution width (RDW), mean platelet volume (MPV), albumin, and total protein were similar between the groups ($p > 0.05$, for all). The mode of delivery differed significantly among the groups, with cesarean section rates being notably higher in the non-obese at pre-pregnancy but obese at delivery group and the obese at both pre-pregnancy and delivery group compared to the non-obese at both pre-pregnancy and delivery group ($p < 0.001$). (Table 1)

The analysis of maternal and fetal thiol-disulfide levels is shown in Table 2. Maternal serum native thiol (SH) (306.21 ± 49.19 µmol/L vs. 270.9 ± 60.12 µmol/L vs. 276.9 ± 59.18 µmol/L, $p = 0.004$) and total thiol (SH + SS) (337.88 ± 52.43 µmol/L vs. 303.8 ± 62.13 µmol/L vs.

Table 1 Maternal and newborn characteristics and perinatal outcomes of the participants

	Non-obese at both pre-pregnancy and delivery (*n* = 72, 57.6%)	Non-obese at pre-pregnancy but obese at delivery (*n* = 29, 23.2%)	Obese at both pre-pregnancy and delivery (*n* = 24, 19.2%)	*p*-value
Maternal age (year) (mean ± SD)	27.3 ± 5.6	29 ± 4.9	28.6 ± 4.6	0.302
Gravida (n) median (Q1-Q3)	2 (2–3)	3 (2–3)	3 (2–3)	0.162
Parity (n) median (Q1-Q3)	1 (1–2)	2 (1–2)	1 (1–2)	0.101
BMI at delivery (kg/m^2) median (Q1-Q3)	27.26 (25.44–28.33)	32.34 (31.22–33.51)	38 (35.01–42.80)	**< 0.001**[a]
Maternal height (m) median (Q1-Q3)	1.65 (1.6–1.68)	1.64 (1.56–1.65)	1.66 (1.55–1.67)	0.081
Maternal weight at delivery (kg) median (Q1-Q3)	74 (68–78)	84 (79.5–90)	96 (91.7–109)	**< 0.001**[a]
Weight gained during pregnancy (kg) median (Q1-Q3)	10 (8–14)	12 (9.5–17.5)	10 (6.25-12)	**0.039**[b]
Pre-pregnancy weight (kg) median (Q1-Q3)	61 (56.5–68)	70 (64.5–80)	90 (79.7–99.5)	**< 0.001**[a]
Pre-pregnancy BMI (kg/m^2) median (Q1-Q3)	22.8 (20.99–24.6)	27.3 (26.1–29)	33.9 (31.6–37.7)	**< 0.001**[a]
Delta BMI (kg/m^2) median (Q1-Q3)	3.86 (2.81–5.16)	4.84 (3.56–6.73)	3.92 (2.61–5.08)	**0.011**[b]
Gestational age at delivery (week) median (Q1-Q3)	39 (38–39)	39 (38–39)	39 (38–39)	0.901
Newborn gender (n,%)				0.919
Female	30 (41.7)	13 (44.8)	11 (45.8)	
Male	42 (58.3)	16 (55.2)	13 (54.2)	
Mode of delivery (n, %)				**< 0.001**[c]
Vaginal delivery	47 (65.3)	9 (31)	5 (20.8)	
Cesarean section	25 (34.7)	20 (69)	19 (79.2)	
Birth weight (g) median (Q1-Q3)	3260 (2972–3427)	3360 (3140–3515)	3400 (3160–3670)	0.107
Body length of newborns (cm) median (Q1-Q3)	51 (50–52)	51 (50–52)	52 (51–52)	0.228
APGAR Score at 1stminute median (Q1-Q3)	9 (9–9)	9 (9–9)	9 (9–9)	0.692
APGAR Score at 5th minute median (Q1-Q3)	10 (10–10)	10 (10–10)	10 (10–10)	0.692
WBC (*10^3/mm^3) (mean ± SD)	10.82 ± 2.4	10.51 ± 2.71	9.49 ± 2.67	0.090
Hemoglobin (g/dL) (mean ± SD)	11.6 ± 1.2	11.3 ± 1.3	11.8 ± 1.3	0.444
Hematocrit (%) (mean ± SD)	36.23 ± 3.39	35.5 ± 3.22	36.3 ± 3.07	0.561
Neutrophil (*10^3/mm^3) median (Q1-Q3)	7.53 (6.35–9.84)	7.46 (6.09–8.81)	7.19 (5.4–8.48)	0.195
Lymphocyte (*10^3/mm^3) median (Q1-Q3)	1.87 (1.58–2.28)	1.96 (1.56–2.47)	1.88 (1.2–2.27)	0.531
Platelet (*10^3/mm^3) median (Q1-Q3)	250 (197–297)	212 (201–259)	233 (216.5-259.5)	0.326
Neutrophil-to-lymphocyte ratio median (Q1-Q3)	3.99 (3.21–5.29)	3.77 (3.15–4.62)	3.59 (3.02–4.67)	0.494
Platelet-to-lymphocyte ratio median (Q1-Q3)	124.7 (97.2-163.7)	124.1 (84.61-164.45)	134.6 (105.9-185.12)	0.433
MCV (fL) (mean ± SD)	85 ± 7	83.8 ± 6.5	81.9 ± 4.9	0.141
RDW (%) median (Q1-Q3)	15.5 (15.1–16.5)	15.3 (14.75–16.6)	16 (15-16.8)	0.729
MPV (fL) median (Q1-Q3)	8.1 (7.5–8.9)	8.1 (7.1–8.4)	7.9 (7.2–8.5)	0.273
Albumin (g/dL) median (Q1-Q3)	3.7 (3.4-4)	3.6 (3.2–3.7)	3.6 (3.2–3.9)	0.096
Total protein (g/dL) median (Q1-Q3)	6.4 (6.1–6.7)	6.2 (5.8–6.6)	6.4 (5.7–6.8)	0.204

Abbrevations: BMI: Body mass index, WBC: White blood cell, MCV: Mean corpuscular volume, RDW: Red cell distribution width, MPV: Mean platelet volume. Bold *p* values are statistically significant ($p < 0.05$). [a] The difference between all groups is significant. [b] The difference between Group 1 vs. Group 2 and Group 2 vs. Group 3 is significant. [c] The difference between Group 1 vs. Group 2 and Group 1 vs. Group 3 is significant

306 ± 58.01 μmol/L, $p = 0.006$) levels were significantly higher in the non-obese at both pre-pregnancy and delivery group compared to the other groups. Disulfide (SS) levels and thiol-disulfide ratios (SS/SH, SS/total thiol, and SH/total thiol) in maternal serum showed no significant differences across the groups ($p > 0.05$, for all). In fetal cord blood, SH, SS, SH + SS levels, and thiol-disulfide ratios were not significantly different among the groups ($p > 0.05$, for all). (Table 2; Fig. 1, and Fig. 2)

The relationship between maternal and fetal thiol-disulfide profiles and parameters using Spearman's correlation test presented in Table 3. Maternal serum native thiol (SH) and total thiol (SH + SS) levels showed a significant negative correlation with maternal age, BMI at delivery, maternal weight at delivery, pre-pregnancy weight, pre-pregnancy BMI and showed a significant positive correlation with WBC, neutrophil, NLR, albumin, and total protein ($p < 0.05$, for all). In maternal serum, NLR exhibited a significant negative correlation with SS/SH and SS/total thiol ratios, and a positive correlation with SH/total thiol ratio ($p < 0.05$, for all). In fetal cord blood, NLR was positively correlated with disulfide (SS) levels, SS/SH and SS/total thiol ratios, but negatively correlated with SH/total thiol ratio ($p < 0.05$, for all). Positive correlations were observed between maternal serum

Table 2 Comparison of maternal and fetal cord blood thiol and disulphide levels

	Non-obese at both pre-pregnancy and delivery (*n* = 72, 57.6%)	Non-obese at pre-pregnancy but obese at delivery (*n* = 29, 23.2%)	Obese at both pre-pregnancy and delivery (*n* = 24, 19.2%)	*p*-value
Maternal serum samples				
Native thiol (SH) (µmol/L) (mean ± SD)	306.21 ± 49.19	270.9 ± 60.12	276.9 ± 59.18	**0.004**[a]
Disulphide (SS) (µmol/L) median (Q1-Q3)	15.65 (11.95–19.38)	16.5 (12.22–19.55)	13.5 (11.71–18.58)	0.374
Total thiol (SH + SS) (µmol/L) (mean ± SD)	337.88 ± 52.43	303.8 ± 62.13	306 ± 58.01	**0.006**[a]
SS/SH (%) median (Q1-Q3)	5.4 (4.08–6.30)	5.8 (4.49–7.78)	5.5 (3.62–6.51)	0.208
SS/Total thiol (SH + SS) (%) median (Q1-Q3)	4.57 (3.77–5.59)	5.2 (4.12–6.73)	4.9 (3.37–5.76)	0.208
SH/Total thiol (SH + SS) (%) median (Q1-Q3)	90.85 (88.79–92.44)	86.4 (86.54–91.75)	90 (88.47–93.24)	0.208
Fetal cord blood samples				
Native thiol (SH) (µmol/L) (mean ± SD)	349.3 ± 70.56	329.7 ± 51.99	327.7 ± 42.96	0.194
Disulphide (SS) (µmol/L) median (Q1-Q3)	14.87 (10.84–18.69)	13.1 (9.14–19.16)	12.4 (9.06–16.6)	0.301
Total thiol (SH + SS) (µmol/L) (mean ± SD)	383.58 ± 66.4	358.4 ± 50.83	356.6 ± 52.55	0.065
SS/SH (%) median (Q1-Q3)	4.16 (3.1–5.8)	3.7 (2.59–5.53)	3.82 (2.49–5.89)	0.866
SS/Total thiol (SH + SS) (%) median (Q1-Q3)	3.84 (2.92–5.2)	3.5 (2.46–4.81)	3.5 (2.36–5.27)	0.864
SH/Total thiol (SH + SS) (%) median (Q1-Q3)	92.31 (89.61–94.16)	93 (90.36–95.06)	92.9 (89.46–95.26)	0.861

Abbrevations: SH: Native thiol, SS: Disulphide, SH + SS: Total thiol. Bold *p* values are statistically significant ($p < 0.05$). [a] The difference between Group 1 vs. Group 2 and Group 1 vs. Group 3 is significant

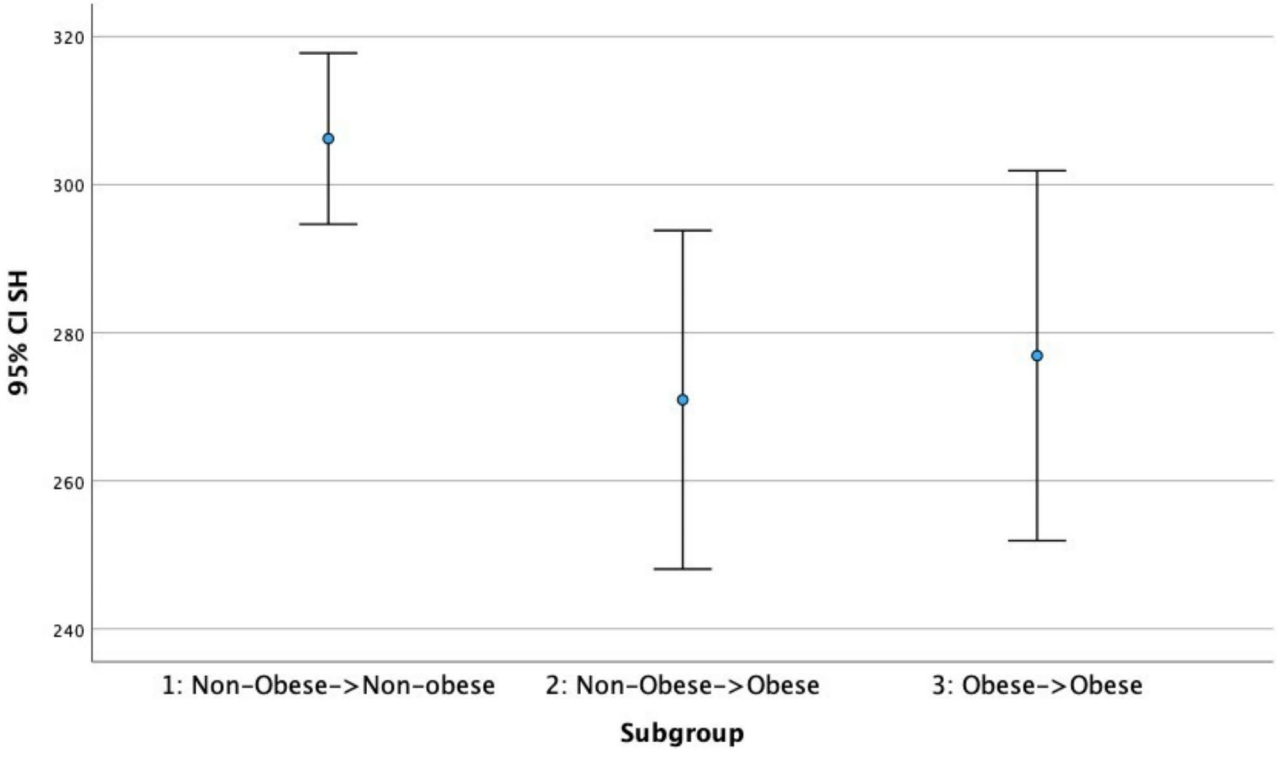

Fig. 1 Box plot of maternal native thiol according to groups, displaying the mean ± SD

disulfide (SS) levels with albumin and total protein levels ($p < 0.05$, for both). (Table 3)

Discussion

Plasma thiols are potent antioxidants that play a crucial role in neutralizing free radicals, thereby maintaining cellular redox homeostasis. TDH is important for cellular redox regulation, and shifts toward disulfides indicate acute OS, while decreases in SH and total thiol levels indicate decreased antioxidant capacity [22, 26]. As a novel marker of OS, TDH is integral to the antioxidant defense system, detoxification processes, and regulation of apoptosis. In this study, we simultaneously assessed thiol-disulfide balance in maternal serum and fetal cord

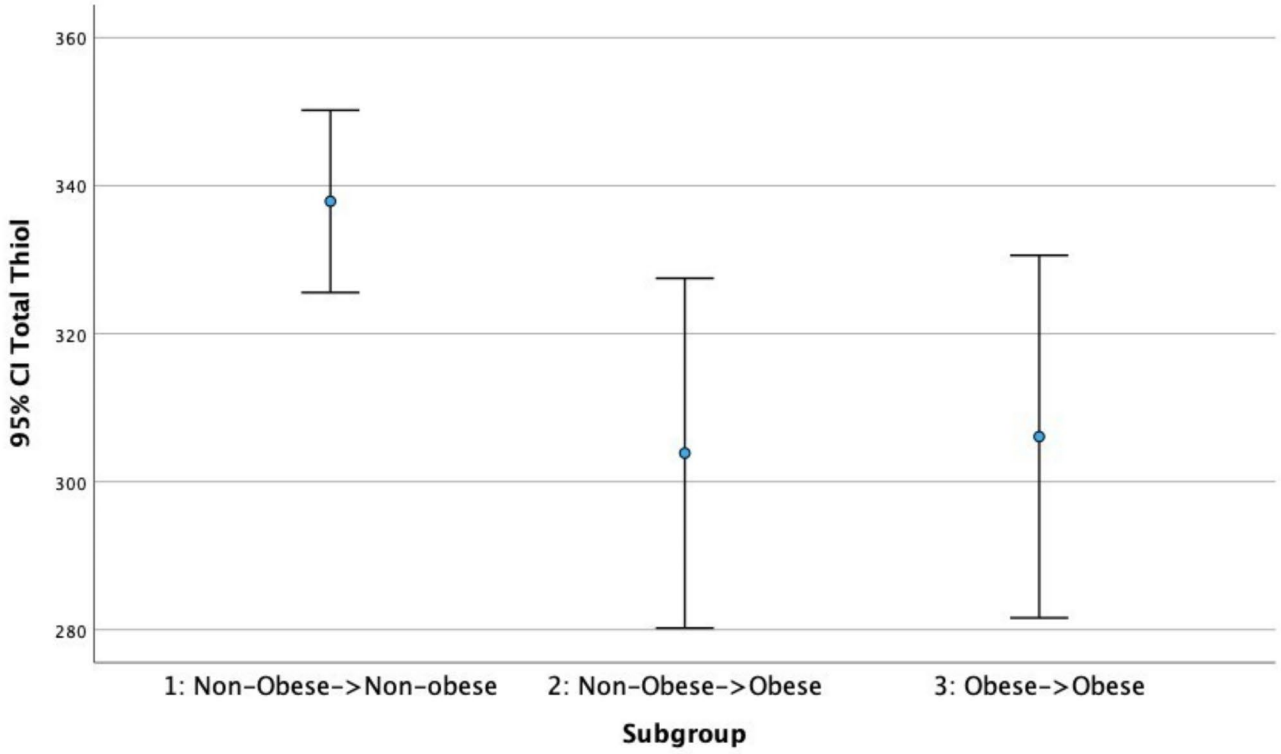

Fig. 2 Box plot of maternal total thiol according to groups, displaying the mean ± SD

blood samples across groups classified by maternal BMI at pre-pregnancy and delivery. Our results demonstrated that maternal serum levels of SH and total thiol were significantly higher in the group that remained non-obese throughout pregnancy compared to both the group that developed obesity during pregnancy and the group that was obese at both pre-pregnancy and delivery. Conversely, SS levels in maternal serum were similar across all groups, as were SH, SS, and total thiol levels in fetal cord blood. Ratios reflecting thiol-disulfide interactions (SS/SH, SS/total thiol, and SH/total thiol) also showed no significant differences between the groups in either maternal or cord blood. Correlational analyses revealed several notable relationships. Maternal serum SH and total thiol levels were negatively correlated with maternal age, BMI at delivery, maternal weight at delivery, pre-pregnancy weight, and pre-pregnancy BMI. In contrast, these antioxidant markers were positively correlated with inflammatory markers, including WBC, neutrophil, NLR, albumin, and total protein levels. These findings underscore the intricate interplay between OS and inflammation, particularly in the context of maternal obesity. Our study provides evidence that maternal obesity, whether longstanding or associated with excessive gestational weight gain leading to a BMI classified as obese at delivery, disrupts TDH and increases OS. The reduction in antioxidant capacity observed in obese women highlights the vulnerability of this population to oxidative damage, which may have implications for both maternal and fetal health.

The role of OS and inflammation in obesity is well established. It is widely accepted that OS contributes to the progression of metabolic disorders, neurodegenerative diseases, and aging. Vincent et al. [27] showed that obesity increases lipid peroxidation in direct proportion to the level of adiposity, leading to an increase in OS and inflammation, and also reduces antioxidant defense mechanisms. Similarly, multiple studies have confirmed that pregnant women with obesity experience elevated OS, often accompanied by a pro-inflammatory state [28, 29]. Prior research has shown that maternal obesity is linked to an elevation in the expression of inflammatory cytokines IL-1, IL-6, and TNF-α in $CD14^{+}$ resident cells obtained from the placenta [30]. Our findings align with these studies and extend them by showing that maternal antioxidant system capacities are significantly reduced in both women who develop obesity during pregnancy and those who remain obese throughout. The group that was non-obese both at pre-pregnancy and delivery demonstrated significantly higher antioxidant levels, suggesting that sustained non-obesity is protective against OS and inflammation.

Despite the higher BMI among obese participants in our study, no significant differences in birth weight were

Table 3 The relationship between maternal and fetal cord blood thiol-disulfide profiles and parameters using Spearman's correlation test

		Maternal serum samples						Fetal cord blood samples					
		Native thiol (SH)	Total thiol (SH + SS)	Disulphide (SS)	SS/SH	SS/ Total thiol	SH/ Total thiol	Native thiol (SH)	Total thiol (SH + SS)	Disulphide (SS)	SS/SH	SS/ Total thiol	SH/ Total thiol
Maternal age	**r**	**-0.292**	**-0.333**	-0.173	-0.028	-0.028	0.028	-0.070	-0.063	0.033	0.056	0.055	-0.055
	p	**< 0.001**	**< 0.001**	0.054	0.756	0.753	0.758	0.437	0.483	0.715	0.538	0.544	0.543
Gravida	**r**	-0.093	-0.109	-0.070	-0.065	-0.065	0.065	-0.031	-0.023	0.102	0.098	0.098	-0.097
	p	0.302	0.227	0.439	0.473	0.469	0.474	0.735	0.800	0.258	0.276	0.277	0.279
BMI at delivery	**r**	**-0.229**	**-0.235**	-0.072	0.060	0.060	-0.061	-0.159	-0.150	-0.077	-0.001	-0.002	0.002
	p	**0.010**	**0.008**	0.425	0.503	0.504	0.501	0.077	0.096	0.390	0.988	0.986	0.978
Maternal height	**r**	0.033	0.030	-0.045	-0.085	-0.085	0.085	-0.013	-0.011	-0.027	-0.003	-0.002	0.002
	p	0.716	0.736	0.615	0.348	0.348	0.349	0.890	0.906	0.768	0.977	0.981	0.980
Maternal weight at delivery	**r**	**-0.222**	**-0.229**	-0.101	0.015	0.015	-0.016	-0.155	-0.146	-0.091	0.000	0.000	0.001
	p	**0.013**	**0.010**	0.263	0.864	0.867	0.864	0.085	0.104	0.312	0.998	0.998	0.991
Weight gained during pregnancy	**r**	-0.037	-0.017	0.108	0.105	0.104	-0.104	-0.027	-0.024	-0.033	-0.044	-0.043	0.044
	p	0.684	0.850	0.230	0.246	0.248	0.248	0.764	0.791	0.714	0.629	0.634	0.628
Pre-pregnancy weight	**r**	**-0.192**	**-0.209**	-0.143	-0.036	-0.036	0.035	-0.143	-0.137	-0.063	0.032	0.032	-0.031
	p	**0.032**	**0.019**	0.111	0.693	0.691	0.696	0.111	0.128	0.484	0.722	0.723	0.728
Pre-pregnancy BMI	**r**	**-0.192**	**-0.206**	-0.111	0.011	0.011	-0.011	-0.131	-0.130	-0.059	0.018	0.018	-0.017
	p	**0.032**	**0.021**	0.219	0.907	0.906	0.904	0.146	0.147	0.511	0.842	0.845	0.850
Delta BMI	**r**	-0.039	-0.019	0.108	0.107	0.107	-0.107	-0.032	-0.018	0.005	-0.009	-0.009	0.009
	p	0.663	0.830	0.232	0.233	0.235	0.234	0.723	0.842	0.956	0.919	0.925	0.918
Gestational age at delivery	**r**	-0.058	-0.044	0.060	0.091	0.091	-0.091	0.057	0.067	0.018	0.009	0.009	-0.009
	p	0.519	0.625	0.507	0.310	0.313	0.312	0.525	0.460	0.845	0.917	0.920	0.922
Newborns weight	**r**	-0.020	-0.010	0.046	0.067	0.069	-0.069	0.093	0.079	-0.042	-0.063	-0.089	0.089
	p	0.822	0.908	0.614	0.456	0.443	0.442	0.302	0.381	0.644	0.484	0.325	0.324
Body length of newborns	**r**	-0.076	-0.069	0.061	0.103	0.102	-0.102	0.020	0.001	-0.096	-0.079	-0.078	0.080
	p	0.399	0.446	0.498	0.255	0.258	0.258	0.824	0.989	0.289	0.381	0.384	0.377
WBC	**r**	**0.202**	**0.187**	0.019	-0.069	-0.069	0.069	-0.004	0.026	0.122	0.115	0.115	-0.115
	p	**0.024**	**0.037**	0.835	0.442	0.442	0.443	0.964	0.777	0.175	0.203	0.202	0.200
Hemoglobin	**r**	0.112	0.076	-0.125	-0.139	-0.138	0.138	-0.102	-0.123	-0.150	-0.104	-0.105	0.105
	p	0.212	0.397	0.165	0.123	0.125	0.124	0.258	0.172	0.094	0.248	0.245	0.243
Hematocrit	**r**	0.052	0.018	-0.164	-0.137	-0.136	0.136	-0.081	-0.097	-0.119	-0.081	-0.081	0.082
	p	0.568	0.839	0.067	0.129	0.130	0.130	0.371	0.280	0.184	0.369	0.368	0.364
Neutrophil	**r**	**0.280**	**0.263**	0.011	-0.118	-0.118	0.118	-0.002	0.041	0.147	0.144	0.144	-0.144
	p	**0.002**	**0.003**	0.908	0.193	0.193	0.194	0.983	0.654	0.105	0.113	0.113	0.112
Lymphocyte	**r**	-0.008	0.013	0.082	0.098	0.098	-0.098	0.011	0.011	-0.070	-0.082	-0.081	0.081
	p	0.932	0.890	0.367	0.281	0.280	0.279	0.902	0.903	0.440	0.370	0.373	0.375

Table 3 (continued)

		Maternal serum samples						Fetal cord blood samples					
		Native thiol (SH)	Total thiol (SH + SS)	Disulphide (SS)	SS/SH	SS/ Total thiol	SH/ Total thiol	Native thiol (SH)	Total thiol (SH + SS)	Disulphide (SS)	SS/SH	SS/ Total thiol	SH/ Total thiol
Platelet	r	-0.079	-0.072	0.032	0.030	0.030	-0.030	-0.110	-0.127	-0.062	-0.013	-0.012	0.012
	p	0.380	0.422	0.720	0.742	0.738	0.737	0.223	0.157	0.493	0.886	0.893	0.892
Neutrophil-to-lymphocyte ratio	r	**0.293**	**0.255**	-0.066	**-0.216**	**-0.216**	**0.217**	0.015	0.055	**0.192**	**0.183**	**0.182**	**-0.182**
	p	**0.001**	**0.005**	0.472	**0.018**	**0.018**	**0.017**	0.868	0.551	**0.036**	**0.046**	**0.046**	**0.046**
Platelet-to-lymphocyte ratio	r	-0.049	-0.072	-0.059	-0.082	-0.081	0.082	-0.096	-0.101	0.035	0.076	0.076	-0.076
	p	0.592	0.434	0.519	0.376	0.377	0.376	0.297	0.273	0.701	0.409	0.410	0.412
MCV	r	0.047	0.023	-0.058	-0.059	-0.059	0.059	-0.126	-0.133	-0.067	-0.053	-0.054	0.054
	p	0.607	0.798	0.527	0.518	0.519	0.519	0.163	0.143	0.462	0.560	0.553	0.554
RDW	r	-0.001	0.028	0.064	0.065	0.064	-0.064	0.180	0.159	-0.024	-0.078	-0.077	0.078
	p	0.991	0.762	0.481	0.473	0.478	0.480	0.056	0.079	0.790	0.391	0.394	0.393
MPV	r	0.072	0.063	-0.004	-0.005	-0.005	0.005	0.168	0.170	0.132	0.056	0.055	-0.055
	p	0.427	0.487	0.964	0.960	0.958	0.956	0.063	0.060	0.146	0.540	0.547	0.543
Albumin	r	**0.472**	**0.486**	**0.218**	-0.101	-0.100	0.101	-0.010	0.007	0.107	0.111	0.112	-0.112
	p	**< 0.001**	**< 0.001**	**0.018**	0.280	0.282	0.281	0.911	0.944	0.250	0.233	0.231	0.229
Total protein	r	**0.455**	**0.479**	**0.210**	-0.089	-0.089	0.088	0.056	0.067	0.105	0.096	0.096	-0.097
	p	**< 0.001**	**< 0.001**	**0.023**	0.340	0.342	0.343	0.547	0.475	0.260	0.301	0.303	0.300

Abbrevations: BMI: Body mass index, WBC: White blood cell, MCV: Mean corpuscular volume, RDW: Red cell distribution width, MPV: Mean platelet volume, SH: Native thiol, SS: Disulphide, SH + SS: Total thiol. Bold *p* values are statistically significant ($p < 0.05$)

observed between the groups. Additionally, oxidative capacities in fetal cord blood were were similar among all groups. These findings suggest a potential adaptive role of the placenta in mitigating the adverse effects of maternal obesity on fetal development. The placenta, the critical interface between mother and fetus, may employ compensatory mechanisms to ensure normal fetal growth despite adverse maternal metabolic conditions. Functional studies have demonstrated reduced OS in the placentas of obese women, even in the context of increased nitrative stress, suggesting the activation of antioxidant pathways [31]. Additionally, maternal obesity has been associated with reduced placental activity of sodium-dependent neutral amino acid transporters (SNAT), which are crucial for fetal growth and development [32]. This reduction, coupled with maternal hyperleptinemia and leptin resistance, may represent another layer of placental adaptation aimed at regulating nutrient transfer and maintaining fetal growth under suboptimal conditions. Such mechanisms may involve altered signaling pathways that prioritize fetal homeostasis while minimizing the detrimental effects of maternal metabolic stress.

Despite its important findings, our study has limitations. First, the single-center design may restrict the generalizability of the results to broader populations. The second limitation of our study is its design, which precludes establishing causal relationships between maternal obesity, OS, and inflammation. Longitudinal studies are needed to assess how the process evolves throughout pregnancy and the long-term effects on maternal and neonatal health. The third limitation is the lack of data on dietary intake, physical activity, and other lifestyle factors that may influence OS and inflammation. Fourth, our participant classification was based on BMI at pre-pregnancy and at delivery. However, BMI at delivery alone may not fully capture maternal weight gain patterns, as it is influenced by factors such as fetal growth, placental weight, and amniotic fluid volume, rather than directly reflecting maternal fat accumulation. While BMI remains a widely used and accessible measure, gestational weight gain (GWG) exceeding recommended limits based on pre-pregnancy BMI may provide a more precise classification. Future studies should consider incorporating GWG into their analysis to enhance the accuracy of weight-related assessments and should aim to include these variables to provide a more comprehensive understanding of the mechanisms involved.

Conclusion

This study highlights the significant impact of maternal obesity, both pre-existing and newly developed during pregnancy, on OS and inflammation, as evidenced by disruptions in TDH. Maternal obesity was associated with reduced antioxidant capacity. These findings emphasize the importance of effective weight management strategies before and during pregnancy to mitigate the adverse effects of obesity on OS and inflammation. Preventing maternal obesity, a modifiable risk factor, is crucial for improving pregnancy outcomes and promoting the health of future generations.

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Author contributions
Design: BDC; Planning: BDC, BB, AA, YEU; Data Acquisition: BDC, BB, AA, MAS; Data analysis: BDC, AA; Manuscript writing: BDC, BB, MAS, AA, YEU, HLK, OE; Final Approval: BDC, BB, MAS, AA, YEU, HLK, OE.

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Data availability
The dataset analyzed during the current study is available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
Ethical approval was obtained from the Ankara Etlik City Hospital Ethics Committee (approval number: AESH-BADEK-2024-418). Written informed consent was obtained from all participants prior to their inclusion in the study. The research was carried out in full compliance with the principles outlined in the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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