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Original

Physiological changes of some oxidative stress during different pregnancy trimesters

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Abstract

Background: Pregnancy triggers a number of physiological and metabolic adjustments that may upset the equilibrium of reactive oxygen species (ROS) production and the body's antioxidant defenses. We compared serum catalase (CAT), malondialdehyde (MDA), peroxynitrite ($ONOO^-$), and reduced glutathione (GSH) between pregnant women assessed across all three trimesters and non-pregnant controls.

Methods: This comparative observational study enrolled 96 women aged 18–40 years: 40 non-pregnant controls and 56 women with normal singleton pregnancies (20 first, 20 second, 16 third trimester). Serum CAT, MDA, $ONOO^-$, and GSH were measured by validated spectrophotometric or fluorescence-based assays. Between-group comparisons used t-test and one-way ANOVA with Tukey's HSD post hoc test; Cohen's d with 95% CIs was computed. $p < 0.05$ was considered significant.

Results: Compared with controls, pregnant women had lower CAT (101.3 ± 12.3 vs. 120.7 ± 8.1 KU/L; $d = -1.81$; $p < 0.001$) and GSH (6.1 ± 1.1 vs. 6.9 ± 1.1 µmol/L; $d = -0.6$; $p = 0.002$), and higher MDA (7.7 ± 1.4 vs. 6.7 ± 1.1 µmol/L; $d = 0.8$; $p < 0.001$) and $ONOO^-$ (73.4 ± 15.6 vs. 64.3 ± 15.1 µmol/L; $d = 0.6$; $p = 0.005$). Across trimesters, CAT ($F = 28.0$, $p < 0.001$), GSH ($F = 3.9$, $p = 0.026$), and MDA ($F = 12.3$, $p < 0.001$) differed significantly, whereas $ONOO^-$ did not ($p = 0.096$).

Conclusion: Pregnancy is associated with a measurable shift in maternal redox balance toward lower antioxidant defenses and higher oxidative-damage markers, generally amplifying with gestation. Longitudinal studies with repeated within-person measurements are needed to confirm these trajectories.

Keywords: Catalase, Glutathione, Malondialdehyde, Oxidative stress, Peroxynitrite, Pregnancy

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Introduction

Pregnancy is a physiological state characterized by substantial metabolic and hemodynamic adaptations that support fetal growth and development (1, 2). These adaptations are accompanied by increased oxygen consumption, elevated mitochondrial activity in maternal and placental tissues, and a corresponding rise in the generation of reactive oxygen species (ROS). Under normal conditions, integrated antioxidant defense mechanisms; enzymatic (catalase, superoxide dismutase, glutathione peroxidase) and non-enzymatic (glutathione, vitamins C and E), maintain redox homeostasis by regulating ROS production and clearance. Oxidative stress arises when ROS generation exceeds the buffering capacity of these defense systems, potentially contributing to cellular damage, lipid peroxidation, protein modification, and DNA injury. Excessive or sustained oxidative stress has been implicated in the pathophysiology of several pregnancy-related complications, including preeclampsia, gestational diabetes mellitus, fetal growth restriction, and uteroplacental insufficiency (3).

Oxidative stress can be assessed using biomarkers that reflect distinct aspects of redox regulation. Catalase (CAT) is an antioxidant enzyme that protects cells from oxidative damage by catalyzing the decomposition of hydrogen peroxide into water and molecular oxygen. Malondialdehyde (MDA) is a stable end-product of polyunsaturated fatty acid peroxidation and is widely used as an indicator of oxidative damage to lipids. Peroxynitrite ($ONOO^-$), a reactive nitrogen species formed through the rapid reaction of nitric oxide with superoxide anion, is a potent oxidizing and nitrating agent that can damage proteins, lipids, and DNA. Reduced glutathione (GSH), a low-molecular-weight tripeptide and non-enzymatic antioxidant, plays an essential role in maintaining intracellular redox balance, neutralizing reactive electrophiles, and supporting the activity of glutathione-dependent enzymes (4-6).

Changes in oxidative stress and antioxidant biomarkers have been reported during normal pregnancy. However, findings regarding their patterns across the three trimesters remain inconsistent (7). Although some studies suggest that oxidative stress increases as pregnancy progresses (7), this pattern is not consistently observed across all biomarkers (8). Differences in the biological functions of individual markers, in the timing of sampling within each trimester, in study populations, and in assay methodologies may all contribute to the observed discrepancies. Consequently, examining both oxidative-damage markers and antioxidant defenses concurrently is essential for obtaining a more comprehensive understanding of redox changes during uncomplicated pregnancy.

Despite growing interest in oxidative balance during pregnancy, evidence describing the concurrent variation of multiple oxidative stress and antioxidant biomarkers across all three trimesters with a non-pregnant comparator group remains limited. The simultaneous assessment of CAT, MDA, $ONOO^-$, and GSH in pregnant women at defined gestational stages, compared with non-pregnant women of reproductive age, may help clarify the trajectory of these biomarkers during normal pregnancy. Accordingly, this study aimed to compare serum CAT, MDA, $ONOO^-$, and GSH levels among non-pregnant fertile women and pregnant women in the first, second, and third trimesters, and to quantify the magnitude of between-group differences using effect-size estimates with 95% confidence intervals (8, 9).

Materials & Methods

This was a comparative observational study conducted at the College of Medicine, University of Mosul, Iraq. The study included 96 women aged 18–40 years, comprising 40 healthy non-pregnant women and 56 pregnant women with normal singleton pregnancies. Participants were recruited by consecutive sampling, with all eligible women presenting during the study recruitment period enrolled consecutively until the predetermined sample size was reached. The pregnant participants were classified according to gestational trimester: first trimester (n = 20), second trimester (n = 20), and third trimester (n = 16). Gestational age was estimated based on the last menstrual period and confirmed by ultrasonographic assessment. The first, second and third trimesters were defined in weeks of pregnancy: 1–12, 13–26, and 27–40 respectively, which are months of pregnancy: 1–3, 4–6, 7–9, respectively. The sample size was determined based on feasibility and on effect-size estimates from comparable published studies; with n = 40 per comparator group and assuming a pooled standard deviation of approximately 10 KU/L for CAT, the study had > 80% power to detect a between-group difference of 7 KU/L or larger at a two-sided α of 0.05.

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The study was approved by the Medical Research Ethics Committee of the College of Medicine, University of Mosul (Approval No. UOM/COM/MREC/25(1104), dated 3 June 2025). Written informed consent was obtained from all participants before enrollment. Participant confidentiality was maintained in accordance with applicable institutional procedures and the principles of the Declaration of Helsinki.

Eligible participants were women aged 18–40 years who met the criteria for their respective study groups. The control group consisted of healthy, non-pregnant women of reproductive age, whereas the pregnancy groups comprised women with normal singleton pregnancies. Women were excluded if they had chronic medical conditions, including diabetes mellitus, hypertension, cardiovascular disease, renal disease, hepatic disease, or thyroid disorders; pregnancy-related complications, including preeclampsia, gestational diabetes, eclampsia, or hemorrhage; a history of smoking, alcohol consumption, or drug abuse; or multiple pregnancies, including twin or higher-order gestations. Women with a body mass index (BMI) below 18.5 or above 30 kg/m² were also excluded. Use of antioxidant supplements, including vitamins C and E or selenium, during the three months preceding enrollment was an additional exclusion criterion.

Following an overnight fast of 8–10 hours, 5 mL of venous blood was collected from each participant into serum-separator tubes containing a clotting agent. Samples were allowed to clot at room temperature for 30 minutes and were then centrifuged at 3,000 rpm for 15 minutes at room temperature to separate the serum. The separated serum was aliquoted into sterile, nuclease-free polypropylene tubes, immediately frozen at −20 °C for short-term storage (within 48 hours), and transferred to −80 °C for long-term storage until biochemical analyses were performed. All samples were protected from prolonged light exposure and were subjected to a single freeze–thaw cycle before assay.

Serum CAT activity, MDA concentration, $ONOO^-$ level, and GSH concentration were measured using previously described spectrophotometric and fluorescence-based methods (4-6, 10, 11). All measurements were performed in duplicate, and the mean of the two readings was used for analysis.

Serum CAT activity was determined using Aebi's spectrophotometric method (4, 10), which monitors the decomposition of hydrogen peroxide (H_2O_2) by following the decrease in absorbance at 240 nm. Catalase activity was calculated from the rate of change in absorbance per unit time and was expressed in kilounits per liter (KU/L).

Serum MDA was measured using the thiobarbituric acid reactive substances (TBARS) method (5). Briefly, serum was mixed with thiobarbituric acid (TBA) and trichloroacetic acid (TCA), and the mixture was heated at 95 °C for 30 minutes. After cooling, the mixture was centrifuged, and the absorbance of the resulting supernatant was measured at 532 nm against a reagent blank. MDA concentrations were expressed in micromoles per liter (µmol/L). Serum peroxynitrite was assessed using a fluorescence-based assay involving the oxidation of dihydrorhodamine 123 (DHR 123) to fluorescent rhodamine 123 (11). Fluorescence intensity was measured at an excitation wavelength of 500 nm and an emission wavelength of 536 nm. Peroxynitrite concentrations were expressed in micromoles per liter (µmol/L).

Serum reduced GSH was measured using Ellman's method (6), which is based on the reaction of 5,5′-dithiobis (2-nitrobenzoic acid) (DTNB) with free sulfhydryl groups to produce a yellow-colored complex. Absorbance was measured at 412 nm, and GSH concentrations were expressed in micromoles per liter (µmol/L).

Statistical Analysis

Statistical analyses were carried out using IBM SPSS Statistics, version 26 (IBM Corp., Armonk, NY, USA). Continuous variables were described using means and standard deviations reported as mean ± SD. To check if the data followed a distribution the Kolmogorov–Smirnov test was applied for each continuous variable. All four biomarkers showed distribution within each of the study groups. The assumption of variances was tested using Levene's test and this assumption was satisfied for all comparisons.

For comparing groups one-way analysis of variance (ANOVA) was used across the four study groups: - pregnant controls and three trimester groups. When the overall ANOVA showed a significant difference Tukey's honestly significant difference (HSD) test was used for pairwise comparisons.

Effect sizes were calculated using Cohen's d with pooled deviations. The 95% confidence intervals for Cohen's d were estimated using the large-sample

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method (12). Effect size interpretations followed guidelines: a value of |d| around 0.2 was considered small around 0.5 was medium and around 0.8 was large. The significance level was set at two-sided $p < 0.05$. Exact p-values were reported in values less, than 0.001 were written as $p < 0.001$.

Results

The study included 96 women: 56 pregnant women and 40 non-pregnant controls. Compared with non-pregnant controls, pregnant women had significantly lower mean serum CAT activity (101.3 ± 12.2 vs. 120.7 ± 8.1 KU/L; t = 8.7; $p < 0.001$; Cohen's d = −1.8) and lower mean GSH concentrations (6.1 ± 1.1 vs. 6.85 ± 1.0 µmol/L; t = 3.2; p = 0.002; d = −0.6). In contrast, pregnant women had significantly higher mean MDA concentrations (7.7 ± 1.3 vs. 6.7 ± 1.0 µmol/L; t = −3.9; $p < 0.001$; d = 0.8) and higher mean $ONOO^-$ concentrations (73.3 ± 15.58 vs. 64.3 ± 15.1 µmol/L; t = −2.8; p = 0.005; d = 0.5) than non-pregnant controls. All four between-group differences were statistically significant and were associated with medium-to-large effect sizes (Table 1).

Table 1 Oxidative stress and antioxidant biomarkers in pregnant and non-pregnant women

Biomarker	Pregnant (n = 56) mean ± SD	Control (n = 40) mean ± SD	t	p	Cohen's d (95% CI)
Catalase (KU/L)	101.3 ± 12.2	120.7 ± 8.1	8.7	< 0.001	−1.8 (−2.3 to −1.3)
GSH (µmol/L)	6.1 ± 1.1	6.8 ± 1.0	3.2	0.002	−0.6 (−1.0 to −0.2)
MDA (µmol/L)	7.7 ± 1.3	6.7 ± 1.0	−3.9	< 0.001	0.8 (0.3 to 1.2)
$ONOO^-$ (µmol/L)	73.3 ± 15.5	64.3 ± 15.1	−2.8	0.005	0.5 (0.1 to 1.0)

Values are mean ± standard deviation. Pregnant group: n = 56 (aggregated across trimesters). Control group: n = 40. Differences assessed by independent-samples t-test. Cohen's d computed using pooled standard deviations; positive d indicates higher values in pregnant women, negative d indicates lower values in pregnant women. CAT = catalase; GSH = reduced glutathione; MDA = malondialdehyde; $ONOO^-$ = peroxynitrite.

Table 2 Oxidative stress and antioxidant biomarkers across pregnancy trimesters.

Biomarker	T1 (n = 20), mean ± SD	T2 (n = 20), mean ± SD	T3 (n = 16), mean ± SD	TukeyHSD	F	p	d (T1 vs T3)
Catalase (KU/L)	112.4 ± 8.7	98.1 ± 8.3	91.3 ± 9.1	a / b / c	28.0	< 0.001	2.3 (1.5 to 3.2)
GSH (µmol/L)	6.6 ± 1.0	6.0 ± 1.0	5.6 ± 1.1	a / ab / b	3.9	0.026	0.9 (0.2 to 1.6)
MDA (µmol/L)	6.9 ± 1.0	7.6 ± 1.1	8.8 ± 1.3	a / ab / b	12.3	< 0.001	−1.6 (−2.3 to −0.8)
$ONOO^-$ (µmol/L)	68.0 ± 14.2	74.1 ± 15.4	79.2 ± 16.1	ns	2.4	0.096	−0.7 (−1.4 to −0.06)

Values are mean ± standard deviation. T1 = first trimester (n = 20); T2 = second trimester (n = 20); T3 = third trimester (n = 16). Overall, between-group differences assessed by one-way ANOVA (F-statistic with df = 2, 53). Pairwise comparisons performed using Tukey's HSD post hoc test; groups sharing the same letter (a, b, c) are not significantly different at $p < 0.05$; "ns" indicates non-significant pairwise comparisons. Cohen's d reported for the T1-vs-T3 comparison (positive d indicates higher T1 values, negative d indicates higher T3 values). CAT = catalase; GSH = reduced glutathione; MDA = malondialdehyde; $ONOO^-$ = peroxynitrite.

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Among pregnant women, mean CAT activity decreased progressively from the first to the third trimester (112.4 ± 8.7, 98.1 ± 8.3, and 91.3 ± 9.1 KU/L in the first, second, and third trimesters, respectively; F = 28.0; p < 0.001), with Tukey's HSD indicating that all three trimester groups differed significantly from one another (Table 2). Mean GSH concentrations also declined across gestation (6.6 ± 1.0, 6.0 ± 1.0, and 5.6 ± 1.1 µmol/L; F = 3.9; p = 0.026), with the first-trimester and third-trimester groups differing significantly (d = 0.9).

Mean MDA concentrations increased across trimesters (6.9 ± 1.0, 7.6 ± 1.1, and 8.8 ± 1.3 µmol/L; F = 12.30; p < 0.001), with significant differences between the first and third trimesters (d = −1.6). Mean $ONOO^-$ concentrations showed a numerical increase across trimesters (68.0 ± 14.2, 74.1 ± 15.4, and 79.2 ± 16.1 µmol/L), but the overall ANOVA did not reach statistical significance (F = 2.4; p = 0.096). Although the first-versus-third-trimester effect size was in the medium-to-large range (d = −0.7, 95%), pairwise Tukey's HSD comparisons for $ONOO^-$ did not achieve statistical significance (Table 2).

Taken together, antioxidant defenses (CAT, GSH) declined and oxidative-damage markers (MDA, $ONOO^-$) increased with advancing gestation. The trimester-dependent pattern was statistically robust for CAT, MDA, and GSH, whereas the $ONOO^-$ trend did not reach the conventional threshold for significance.

Discussion

We found that all four stress and antioxidant defense biomarkers differed between pregnant and non-pregnant women and the biomarkers varied across the three trimesters of pregnancy. Pregnant women had serum MDA and ONOO- concentrations and significantly lower serum CAT and GSH concentrations compared to non-pregnant controls (effect size for all comparisons medium-to-large: |d|= 0.6–1.8). GSH and GSH activity fell across gestation whereas MDA and ONOO- rose (significant trimester-dependent differences demonstrated for GSH, CAT and MDA but not for ONOO-; p = 0.096). These findings suggest that normal pregnancy is associated with a shift in the relationship between oxidant production and antioxidant defenses with these differences typically getting more pronounced with gestational age. The pattern is consistent with reports of increased oxidative activity during gestation due to growing/upregulating metabolic and physiological needs of the growing placenta and fetus (13, 14).

The rise in MDA during pregnancy in the third trimester is in line with greater lipid peroxidation. MDA, an useful marker of oxidative damage of membrane lipids has been found to be high in pregnancy in several studies (15-17). While the TBARS method is commonly used it is not completely specific to MDA and also can identify TBA-reactive aldehydes; hence the elevated response as noted can be interpreted as a saw-tooth increase in lipid peroxidation-related activity rather than as a precise quantitative measure of MDA-specific damage. Elevated $ONOO^-$ concentrations reflect increased nitrogen species activity. Peroxynitrite is produced via the fast reaction of nitric oxide and superoxide and as a result may cause protein tyrosine nitration, lipid oxidation and interfere cellular signaling. However, these biomarkers cannot identify the origin of species or demonstrate damage to the tissue level. Our current data are consistent with studies demonstrating variations in oxidative stress markers in pregnancy (15-17) with inter-study variation most likely the result of differences in study populations, assay methods, gestational timing and sample handling. Plausibly several physiological mechanisms could contribute to the pattern observed. Oxygen's consumption and mitochondrial ROS production throughout pregnancy is increased due to development, fetal growth changes in maternal basal metabolic rate and cardiometabolic system demands (16, 17). Xanthine oxidase and the isoforms of NADPH oxidase have also been identified as contributors to changes in oxidant-generating enzymatic pathways (17). It is important to keep in mind that oxidative signaling has a role in placental development, trophoblast invasion and angiogenesis. Thus, the presence of concentrations of oxidative biomarkers in uncomplicated pregnancy cannot automatically be considered simply as a biomarker of oxidative stress. The findings of decreased CAT and GSH are consistent with changes in defenses. CAT is a contributor for hydrogen peroxide. The reductions in CAT and GSH provide evidence of changes in antioxidant defenses. CAT contributes to hydrogen peroxide detoxification whereas GSH participates in redox regulation the neutralization of reactive electrophiles and the recycling of other antioxidants. The progressive fading in their serum concentrations may denote reinforced antioxidant usage, altered synthesis, shift between compartments or hormonal modulation of antioxidant gene expression with

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increasing pregnancy (17, 18). Hormonal changes including rising estrogen, progesterone and placental lactogen and increasing maternal and fetal metabolic requirements may all influence antioxidant systems (19, 20). The present study cannot determine whether the lower serum values resulted from depletion, altered synthesis or redistribution and serum CAT and GSH should not be interpreted as measures of antioxidant status in all maternal tissues. The balance, between processes and antioxidant defenses is complex and cannot be fully characterized using four biomarkers alone.

An important distinction is that this study talks about biomarker differences across pregnancy not that oxidative stress surely causes pregnancy outcomes. Although high MDA and changed defenses have been seen in pregnancies with preeclampsia, gestational diabetes and fetal growth restriction those links do not prove that the biomarker changes in this healthy group predict or cause those problems. In this study the researchers did not look at those complications as outcomes. Did they check how good the biomarkers were at predicting them. So, it should not be taken to mean that women in the trimester are clinically improving while still at risk. Instead, the study shows that simple biochemical markers vary during pregnancy, which matters for redox health.

The main contribution of this study is that it looks at two oxidative-damage markers (MDA, $ONOO^-$) and two antioxidant defense markers (CAT, GSH) in pregnant women from all three trimesters and also includes a non-pregnant comparison group. By looking at these markers' researchers can see a fuller picture of how the mother's redox state changes, better than using one marker alone. The study also gives effect-size estimates, with 95 % confidence intervals so readers can get a sense of how big and precise the differences.

There are limitations to this cross-sectional observational study. First because the study compares women at one point in time for each participant it cannot show changes that happen within a person during pregnancy. This means differences between trimesters might be influenced by differences between individuals than actual changes over time. Second the sample size is quite small in the third trimester where only 16 women were included. This limits how accurate the estimates are and reduces the ability to find effects. For example, the difference in concentration did not reach statistical significance ($p = 0.096$) but the effect size was medium to large ($d = 0.7$) which suggests a meaningful difference that the study might have missed due to limited power. Third a few biomarkers were used to look at oxidative and antioxidative pathways. This means some important factors were not measured, which could introduce bias. Fourth there might be confounding factors such as diet, nutrition, physical activity and other characteristics that were not controlled. These could influence the results. As a result, while the findings may be useful, for research they are not yet ready to be used in practice.

Conclusion

Pregnancy changes the balance of substances that protect the body from oxidation. Women who are pregnant have levels of CAT and GSH in their blood and higher levels of MDA and $ONOO^-$ compared to women who are not pregnant. As pregnancy moves through the trimesters the average amounts of CAT and GSH fall while the average amount of MDA rises. These changes are significant when the trimesters are compared. The rise in $ONOO^-$ across trimesters is not statistically significant; it should be seen as a trend of a proven effect.

These findings show biochemical changes that happen when a pregnancy is uncomplicated. They do not explain the reasons for these changes or how they might affect health. The results do not prove that oxidative stress causes problems during pregnancy nor do they suggest that regular monitoring of these biomarkers should become routine in care.

The study is limited by its design the small number of participants – especially in the third trimester – and the lack of health outcome data. Future research should use longitudinal studies, validated tests, repeated measurements, in the same individuals and real health outcomes to explore these questions further.

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Conflicts of Interest

The author declares no conflicts of interest.

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