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***Research Article***

# Oxidative Stress and Antioxidant Responses in Postpartum and Non-Postpartum Female Wistar Rats Exposed to Pesticides

**Onome Francisca Oghenevwaire** [1] **and Oyeyemi Adeyemi** [1*]

[1] *Department of Environmental Management and Toxicology, Federal University of Petroleum Resources, PMB 1221, Effurun, Delta State, Nigeria*
[*] *Corresponding author: adeyemi.oyeyemi @fupre.edu.ng*

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**Abstract**

Pesticide exposure, particularly from compounds such as dichlorvos, dimethoate, and cypermethrin, is a major environmental concern, leading to oxidative stress in biological systems. This study investigates the differential oxidative stress and antioxidant responses in postpartum and non-postpartum female Wistar rats exposed to these pesticides, individually and in combinations. Sixty-four female rats were randomly assigned to eight groups and exposed to varying pesticide mixtures over 28 days. Oxidative stress biomarkers, including malondialdehyde (MDA), glutathione (GSH), superoxide dismutase (SOD), catalase, and glutathione S-transferase (GST), were analyzed in uterine and ovarian tissues to assess oxidative damage and antioxidant defense mechanisms. The results revealed significant reductions in antioxidant enzyme activities and elevated MDA levels across all pesticide-exposed groups, with the most pronounced effects observed in groups exposed to combined pesticides. The triple combination of dichlorvos, dimethoate, and cypermethrin induced the highest oxidative burden, marked by severe reductions in antioxidant defenses and increased lipid peroxidation. Notably, postpartum rats exhibited relatively higher antioxidant capacities compared to non-postpartum counterparts, suggesting potential physiological adaptations that may mitigate oxidative insults during the postpartum stage. These findings highlight the compounded toxicological risks associated with pesticide mixtures and underscore the critical need to regulate pesticide exposure. The study provides valuable insights into the biochemical mechanisms underlying pesticide-induced oxidative stress and its implications for reproductive health, particularly during sensitive life stages such as postpartum. These results emphasize the importance of targeted interventions to reduce pesticide toxicity and protect reproductive health.

## 1. Introduction

Pesticide exposure is a major environmental concern with significant implications for human and animal health. Pesticides such as dichlorvos, dimethoate, and cypermethrin are widely used in agriculture to control pests, but their extensive use has raised concerns about their toxicity and environmental persistence [1, 2]. These chemicals, classified as organophosphates and pyrethroids, are known to induce oxidative stress, a condition characterized by an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defenses [3].

Oxidative stress has been implicated in the pathophysiology of various diseases, including neurodegenerative disorders, reproductive toxicity, and metabolic dysfunction [4]. In female mammals, the reproductive system is particularly vulnerable to oxidative damage due to its high metabolic activity and susceptibility to endocrine-disrupting chemicals [5]. Postpartum and non-postpartum states present

unique physiological conditions that may modulate the effects of oxidative stress, as hormonal fluctuations and metabolic demands differ significantly between these stages [6].

The glutathione-S-transferase (GST) enzyme system plays a critical role in detoxifying ROS and maintaining cellular redox homeostasis [7]. Altered GST activity has been reported in response to pesticide exposure, indicating its potential as a biomarker for oxidative damage [8]. Previous studies have demonstrated that exposure to pesticides such as dichlorvos and dimethoate can impair antioxidant responses, leading to tissue-specific oxidative damage in experimental models [2, 9].

Despite extensive research on pesticide toxicity, few studies have investigated the comparative effects of pesticide-induced oxidative stress in postpartum and non-postpartum female animals. The postpartum period is characterized by physiological adaptations to lactation and recovery from parturition, which may influence antioxidant defenses and susceptibility to toxicants [10]. In contrast, non-postpartum females maintain a more stable hormonal and metabolic profile, which could alter their response to oxidative challenges [11].

This study aims to elucidate the differential oxidative stress and antioxidant responses in postpartum and non-postpartum female Wistar rats exposed to a mixture of dichlorvos, dimethoate, and cypermethrin. By evaluating GST activity and other oxidative stress markers in reproductive tissues, this research seeks to provide insights into the reproductive toxicology of pesticides and their potential risks to female health during critical life stages. Understanding these mechanisms is essential for developing targeted interventions to mitigate pesticide toxicity [12, 13].

## 2. Materials and Methods

### Reagents and Solvents

All reagents and solvents used in this study were of analytical grade and procured from the British Drug House, Poole, England. This research was designed to evaluate the effects of a chemical mixture containing dichlorvos, dimethoate, and cypermethrin on serum lipid profiles in female rats during critical biological stages: exposure, mating, pregnancy, and lactation.

### Experimental Design

A total of 64 female and 16 male Wistar rats of uniform strain, age (2–3 months), and weight (190–200 g) were used. The animals were acclimatized for one week under optimal conditions to promote well-being and reduce stress-related variability. The female rats were randomly divided into eight groups (A–H), each comprising eight rats. Graded doses of the chemical mixture were administered to the experimental groups via a standardized protocol over 28 days to ensure precise and consistent exposure. Throughout this period, the health and behavior of the rats were carefully monitored.

At the end of the exposure phase, four rats per group (n=4) were randomly selected for haematological and histopathological evaluations, establishing a baseline for assessing the effects of the chemical mixture. The remaining rats in each group were paired with two males to initiate mating. Successful copulation was confirmed through behavioral observations, and the males were subsequently removed to minimize stress. Pregnant females were monitored for health and behavioral changes during gestation.

Following parturition, lactating dams were observed for maternal care behaviors, while the growth and health of their offspring were recorded to assess potential transgenerational impacts of pesticide exposure. At the conclusion of the 10-day lactation period, the lactating dams were sacrificed for comprehensive haematological and histopathological analyses. Data from both exposure and lactation phases underwent rigorous statistical analysis to identify dose-dependent trends, significant biological effects, and long-term health risks associated with pesticide mixtures.

### Animal Housing and Care

The animals were housed in groups of five per cage under controlled and hygienic conditions at the Animal House of the Federal University of Petroleum Resources, Effurun. Environmental parameters were strictly maintained, including a room temperature of 25 ± 2°C, relative humidity of 50 ± 10%, and a 12-hour light/dark cycle. The rats were fed standard pelleted rodent feed and provided with clean drinking water *ad libitum*. Cage bedding was regularly replaced to ensure cleanliness and minimize external stressors.

All procedures adhered to the ethical guidelines for the care and use of laboratory animals, ensuring the well-being of the subjects and the reliability of experimental outcomes. This meticulous approach ensured that the results were both valid and reproducible, providing a robust foundation for understanding the biochemical impacts of pesticide exposure during key biological stages.

### Dose Levels

The agrochemicals dichlorvos, dimethoate, and cypermethrin, sourced from Hubei Sanonda Co. Ltd, China, were utilized in this study. These chemicals were obtained from a certified agrochemical retailer and diluted with clean water according to domestic usage guidelines. Dose combinations were formulated in specific ratios for each experimental group. Groups B, C, and D received individual chemicals diluted at a 1:1 ratio. Groups E, F, and G were exposed to binary mixtures prepared in a ratio of 1:0.5:0.5, while Group H received a ternary mixture diluted at 1:0.33:0.33:0.33. Freshly prepared solutions were used daily throughout the 28-day exposure period.

To replicate real-world scenarios of domestic pesticide usage, the solutions were sprayed into a poorly ventilated compartment containing the animal cages. The control group (Group A) was exposed to water sprayed under the same conditions. This method closely simulated the confined spaces where animals might encounter pesticide residues.

Animal welfare was prioritized through daily monitoring in accordance with the *Guide for the Care and Use of Laboratory Animals* by the National Academy of Sciences and the National Institute of Health. These ethical practices ensured humane treatment and reliable data collection on the biochemical and toxicological impacts of chemical exposure.

The experimental groups were organized as follows:

• Group A (Control): Exposed to sprayed water.

• Group B: Exposed to dichlorvos.
• Group C: Exposed to dimethoate.
• Group D: Exposed to cypermethrin.
• Group E: Exposed to a mixture of dichlorvos and dimethoate.
• Group F: Exposed to a mixture of dichlorvos and cypermethrin.
• Group G: Exposed to a mixture of dimethoate and cypermethrin.
• Group H: Exposed to a mixture of dichlorvos, dimethoate, and cypermethrin.

### Mating and Fertility Assessment

On post-treatment day 29 (PTD 29), the remaining treated female rats (n = 4 per group) were paired with proven fertile adult males at a 2:1 female-to-male ratio to assess mating and fertility outcomes. Daily vaginal smears were collected from cohabiting females, and the presence of sperm in the smear was used as a definitive marker of successful copulation. The day of sperm-positive vaginal smears was recorded as gestational day zero (GD 0).

The interval from cohabitation to the first observation of sperm-positive smears was carefully documented to evaluate the impact of chemical treatments on mating efficiency and fertility. This method provided critical insights into reproductive performance and the potential disruptions induced by pesticide exposure.

### Anaesthetization of Animals and Tissue Isolation

At the conclusion of the 28-day treatment period, blood samples were collected from the treated female rats under light chloroform anesthesia. Blood was drawn from the dorsal aorta of four females per group and transferred into non-heparinized tubes. The samples were allowed to clot at room temperature before centrifugation at 3500 rpm for 15 minutes. The resulting serum was carefully separated and stored under appropriate conditions to preserve its integrity for lipid profile analysis and ensure accurate biochemical evaluations.

Following blood collection, the ovaries and uteri of the animals were excised and immediately placed in a beaker containing ice-cold normal saline solution to prevent enzymatic degradation and preserve tissue viability for subsequent analyses.

### Preparation of Tissue Homogenate

The isolated tissues were weighed, and portions were carefully sectioned into small pieces. These sections were homogenized using a pre-cooled pestle and mortar, maintained in a bowl of ice to minimize thermal degradation during processing. The tissue homogenates were diluted with normal saline solution to achieve a 1:30 dilution ratio, ensuring consistency and suitability for further biochemical assessments.

### Biochemical Analyses

The malondialdehyde (MDA) concentration in the serum and tissues of the experimental rats was determined using the method outlined by [14]. This method provides a reliable measure of lipid peroxidation, an indicator of oxidative stress. The glutathione (GSH) concentration in the tissues of the experimental rats was assessed using the procedure described by [15]. This method quantifies reduced glutathione, a critical antioxidant involved in cellular defense mechanisms.

Tissue protein concentration was determined following the method reported by Gornall et al. [16]. This technique ensures accurate protein quantification essential for normalizing enzyme activities and other biochemical measurements. Superoxide dismutase (SOD) activity in the tissues of experimental animals was measured using the method described by [17]. This assay evaluates the enzymatic ability to dismutate superoxide radicals, a vital antioxidant defense mechanism. The catalase activity of the tissue homogenate was determined using the method described by [18]. This method quantifies the enzyme's capacity to decompose hydrogen peroxide, a key reactive oxygen species. The cytosolic glutathione S-transferase (GST) activity was assessed spectrophotometrically at 37°C and 340 nm following the procedure described by [19]. This method measures the enzyme's ability to conjugate glutathione with electrophilic substrates, reflecting its detoxification role.

### Statistical Analysis

Numerical results were derived from the eight experimental groups (control and treated). Data were expressed as mean ± SEM and analyzed using one-way analysis of variance (ANOVA) followed by Duncan's Multiple Range Test, utilizing the SPSS 30.0 software (Statistical Package for Social Scientists, version 30.0). A p-value of $<0.05$ was considered statistically significant.

## 3. Results

Figure 1 illustrates the concentration of Glutathione (GSH) in the uterus and ovary of non-postpartum female rats across various pesticide exposure groups, highlighting the impact on antioxidant defenses.

**In the Uterus,** Group A (Control) shows the highest GSH concentration, significantly higher ($p < 0.05$) than in all other groups, indicating robust antioxidant activity in the absence of pesticide exposure. Group B (dichlorvos exposure) has significantly lower GSH levels than the control ($p< 0.05$) but still significantly higher than in Groups D, E, and F, suggesting a moderate reduction in antioxidant capacity. Group C (dimethoate exposure) also shows a significant decrease in GSH compared to the control, with levels similar to Group B, indicating comparable oxidative stress effects. Group D (cypermethrin exposure) displays significantly lower GSH concentration than in Groups A, B, and C, reflecting higher oxidative stress. Group E (dichlorvos and dimethoate exposure) has GSH levels lower than Groups A, B, and C but similar to Group D, demonstrating a strong combined toxic effect. Group F (dichlorvos and cypermethrin exposure) shows GSH concentrations higher than Group E but still significantly lower than in the control, indicating moderate oxidative stress with this combination.

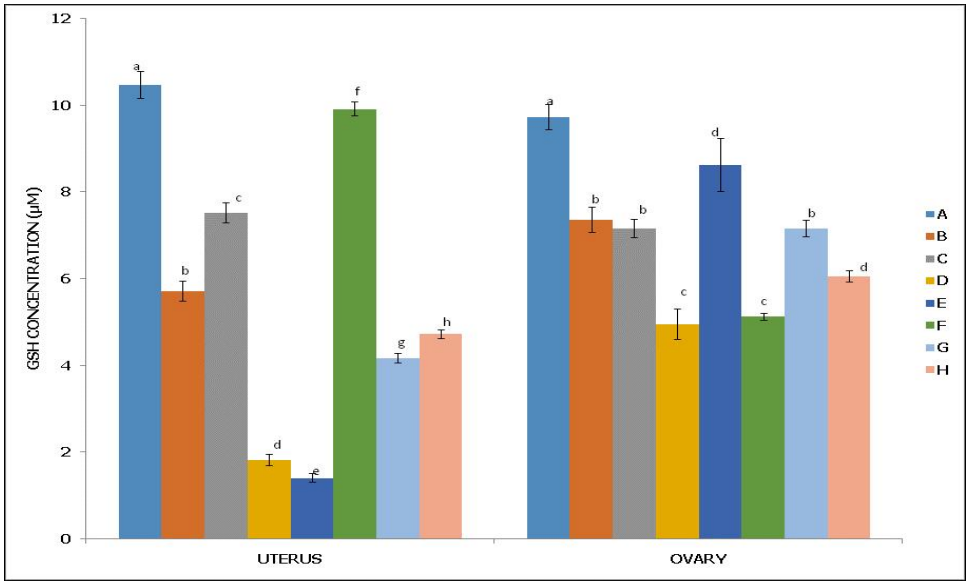


**Figure 1:** Concentration of reduced glutathione (GSH) of uterus and ovary of non-postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$).

Group G (dimethoate and cypermethrin exposure) has GSH levels significantly lower than in Group F but higher than in Group H, illustrating increased oxidative stress with combined pesticide exposure. Finally, Group H (dichlorvos, dimethoate, and cypermethrin exposure) exhibits the lowest GSH concentration, significantly lower than in all other groups, indicating severe depletion of antioxidant defenses due to the triple pesticide combination.

**In the Ovary,** Group A (Control) also has the highest GSH concentration, significantly higher ($p < 0.05$) than in all other groups, indicating low oxidative stress without pesticide exposure. Group B (dichlorvos exposure) shows a significant reduction in GSH compared to the control, though levels are not significantly different from Group C, suggesting moderate oxidative stress. Group C (dimethoate exposure) has significantly lower GSH than the control, with levels comparable to Group B, indicating similar oxidative impact. Group D (cypermethrin exposure) has GSH concentrations significantly lower than in Groups A, B, and C, highlighting stronger oxidative effects. Group E (dichlorvos and dimethoate exposure) shows significantly reduced GSH levels compared to Groups C and A, suggesting heightened oxidative stress with this combination. Group F (dichlorvos and cypermethrin exposure) displays GSH concentrations higher than Group E but still significantly lower than Groups B and C, reflecting a compounded impact on antioxidant function. Group G (dimethoate and cypermethrin exposure) has significantly lower GSH levels than Group F, indicating increased oxidative stress. Group H (dichlorvos, dimethoate, and cypermethrin exposure) has the lowest GSH concentration, significantly lower than in all other groups, underscoring the severe oxidative damage associated with the triple pesticide mixture.

GSH levels are highest in the control group for both uterus and ovary tissues, with significant declines observed across all pesticide-exposed groups. The triple combination in Group H shows the most pronounced reduction in GSH, indicating the highest oxidative stress. Single pesticide exposures result in moderate GSH depletion, while combined exposures, especially the triple mixture, lead to a severe decline in antioxidant defenses in non-postpartum female rats.

Figure 2 shows the Glutathione (GSH) concentration in the uterus and ovary of postpartum female rats across various pesticide exposure groups, highlighting the effects on antioxidant defenses in postpartum conditions.

**In the Uterus,** Group A (Control) has the highest GSH concentration, significantly higher ($p < 0.05$) than all other groups, indicating minimal oxidative stress in the absence of pesticide exposure. Group B (dichlorvos exposure) exhibits significantly lower GSH levels than the control ($p < 0.05$) but higher than in Groups C, D, and E, suggesting moderate oxidative stress. Group C (dimethoate exposure) has GSH levels similar to Group B but significantly lower than the control, showing a comparable reduction in antioxidant capacity. Group D (cypermethrin exposure) shows GSH concentrations lower than Groups A, B, and C, and significantly lower than Groups E and F, indicating increased oxidative damage from cypermethrin exposure. Group E (dichlorvos and dimethoate exposure) has GSH levels lower than Groups A and B but similar to Group D, indicating a combined toxic effect from these pesticides. Group F (dichlorvos and cypermethrin exposure) exhibits GSH concentrations higher than Group E but significantly lower than Groups A and B, showing moderate oxidative stress. Group G (dimethoate and cypermethrin exposure) has GSH levels lower than Group F and significantly lower than Groups A and B, suggesting further depletion of antioxidant defenses with combined pesticide exposure. Group H (dichlorvos, dimethoate, and cypermethrin exposure) has the lowest GSH concentration, significantly lower ($p < 0.05$) than all other groups, indicating the most severe oxidative stress effect due to the triple pesticide combination.

**In the Ovary,** Group A (Control) also exhibits the highest GSH concentration, significantly higher ($p < 0.05$) than in all other groups, reflecting strong antioxidant defenses in the absence of exposure. Group B (dichlorvos exposure) shows significantly lower GSH levels than the control but comparable to Group C, indicating moderate oxidative stress. Group C (dimethoate exposure) has GSH levels similar to Group B but significantly lower than the control, showing a comparable impact on antioxidant capacity. Group D (cypermethrin exposure) has GSH concentrations significantly lower than in Groups A, B, and C, indicating a more pronounced oxidative effect. Group E (dichlorvos and dimethoate exposure) shows a significant reduction in GSH compared to Groups B and C, though its levels are higher than in Group F, highlighting an increased toxic effect. Group F (dichlorvos and cypermethrin exposure) has significantly higher GSH levels than Group E but remains significantly lower than in Groups B and C, suggesting reduced antioxidant protection. Group G (dimethoate and cypermethrin exposure) shows significantly lower GSH levels than Group F, indicating further depletion of antioxidant capacity. Group H (dichlorvos,

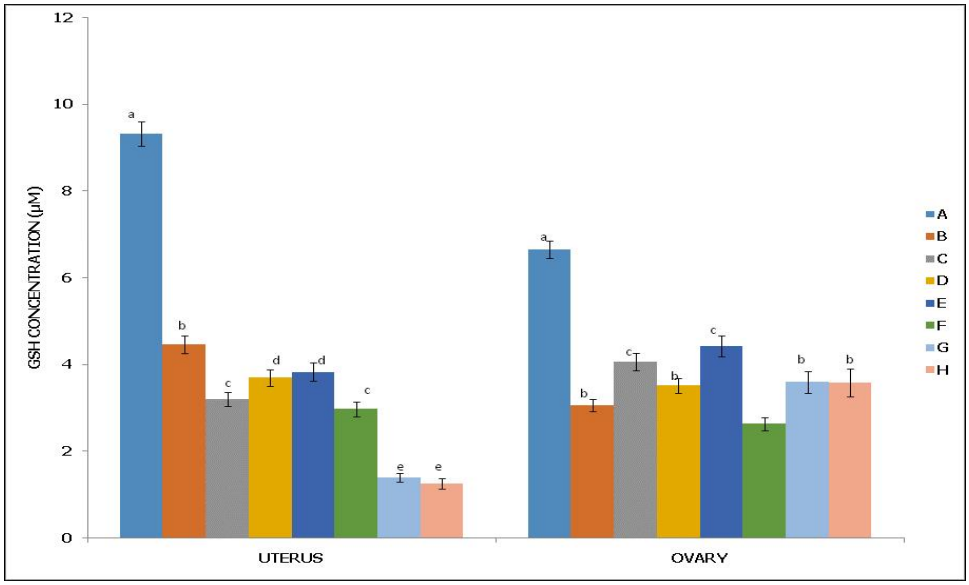


**Figure 2:** Concentration of reduced glutathione (GSH) of uterus and ovary of postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

dimethoate, and cypermethrin exposure) has the lowest GSH concentration, significantly lower than in all other groups, indicating the strongest oxidative impact from the triple pesticide exposure.

In summary, GSH levels are highest in the control group across both uterus and ovary tissues, with significant reductions observed in all pesticide-exposed groups. Combined exposures, particularly the triple mixture in Group H, result in the most substantial decrease in GSH, indicating severe oxidative stress. Single pesticide exposures lead to moderate GSH depletion, while combinations, especially those involving three pesticides, greatly reduce antioxidant defenses in postpartum female rats.

Figure 3 presents the Malondialdehyde (MDA) concentration in the uterus and ovary of nonpostpartum female rats across various pesticide exposure groups, revealing insights into lipid peroxidation levels and oxidative stress.

**In the Uterus,** Group A (Control) has the lowest MDA concentration, significantly lower than all other groups ($p<0.05$), indicating minimal oxidative stress in the absence of pesticide exposure. Group B, exposed to dichlorvos, shows significantly higher MDA levels than the control but remains lower than the levels seen in Groups D and E. Group C, exposed to dimethoate, has a similar MDA concentration to Group B, but its levels are also significantly lower than those in Groups D and E, suggesting that dimethoate and dichlorvos individually cause moderate increases in oxidative stress. Group D, with cypermethrin exposure, exhibits a significantly higher MDA concentration than Groups B and C, highlighting the stronger pro-oxidant effect of cypermethrin. Group E, a dichlorvos and dimethoate combination, has MDA levels comparable to Group D and significantly higher than Groups B and C, suggesting an additive effect of these pesticides on oxidative damage. Group F, with dichlorvos and cypermethrin exposure, shows MDA concentrations lower than Groups D and E but higher than Group C, indicating moderate oxidative stress. Group G (dimethoate and cypermethrin exposure) presents slightly lower MDA levels than Group F, though the difference is not statistically significant. Group H, exposed to the combination of dichlorvos, dimethoate, and cypermethrin, has an MDA concentration lower than Groups D and E but slightly higher than Group G, though this increase is not statistically significant.

**In the Ovary,** Group A (Control) also exhibits the lowest MDA concentration, significantly lower than in all other groups ($p<0.05$), reflecting minimal oxidative stress under control conditions. Group B (dichlorvos exposure) shows higher MDA levels than the control but lower than in Groups E and F, indicating moderate oxidative stress. Group C (dimethoate exposure) has lower MDA levels than Groups E, F, and H, suggesting a milder oxidative impact compared to those combinations. Group D (cypermethrin exposure) has MDA levels significantly lower than those in Groups E and F, reinforcing cypermethrin's relatively lower pro-oxidant effect in single exposure compared to combined exposures. Group E (dichlorvos and dimethoate exposure) displays significantly elevated MDA concentrations compared to Groups C and D, similar to Group H, pointing to an increased oxidative stress effect with this combination. Group F, combining dichlorvos and cypermethrin, has the highest MDA concentration among all groups, significantly higher than all except Group H, indicating severe lipid peroxidation. Group G (dimethoate and cypermethrin exposure) has lower MDA than Group F, though it remains higher than Group C, without a significant difference. Lastly, Group H (dichlorvos, dimethoate, and cypermethrin) has significantly elevated MDA levels, especially compared to Group C, and is similar to the levels in Group F, suggesting a compounded oxidative effect from the triple pesticide exposure.

Figure 4 displays the Malondialdehyde (MDA) concentration in the uterus and ovary of postpartum female rats across various pesticide exposure groups, highlighting lipid peroxidation and oxidative stress levels.

**In the Uterus,** Group A (Control) has the lowest MDA concentration, significantly lower ($p<0.05$) than all other groups, indicating minimal oxidative stress without pesticide exposure. Group B (dichlorvos exposure) shows MDA levels significantly higher than the control but lower than in Groups E and H, suggesting a moderate increase in oxidative stress from dichlorvos. Group C (dimethoate exposure) has an MDA concentration similar to Group B but significantly lower than in Groups E and H, indicating a comparable yet modest oxidative effect. Group D (cypermethrin exposure) exhibits MDA levels higher than Group B but lower than Groups E and F, showing increased oxidative stress, although less severe than some combined exposures. Group E (dichlorvos + dimethoate exposure) has the highest MDA concentration, significantly elevated compared to all other groups except Group H, pointing to severe oxidative stress. Group F (dichlorvos + cypermethrin exposure) shows MDA levels significantly lower than Group E but higher than Groups B and C, suggesting a considerable oxidative effect. Group G (dimethoate + cypermethrin exposure) has MDA levels significantly lower than Group E but higher than Groups B and C, reflecting

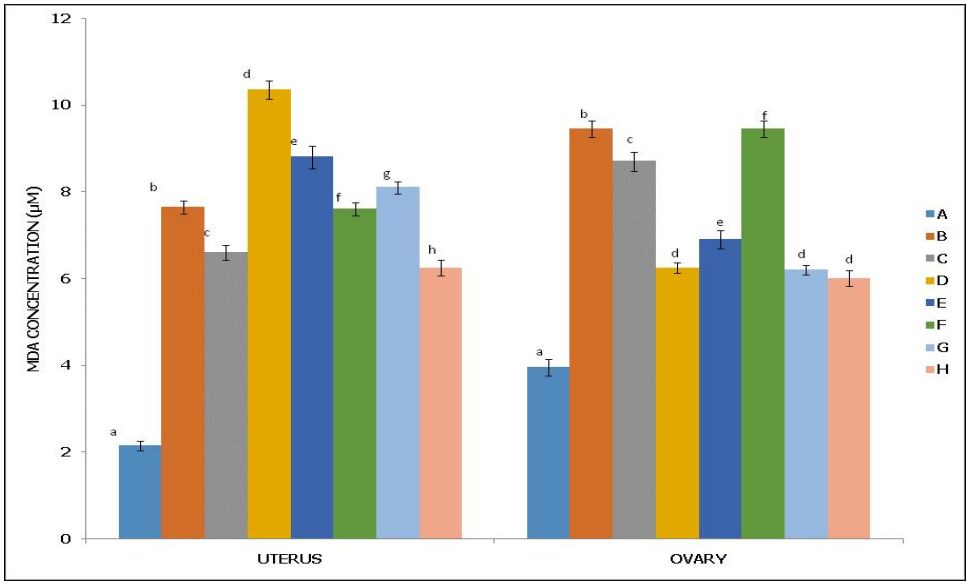


**Figure 3:** Concentration of malondialdehyde (MDA) of uterus and ovary of non-postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

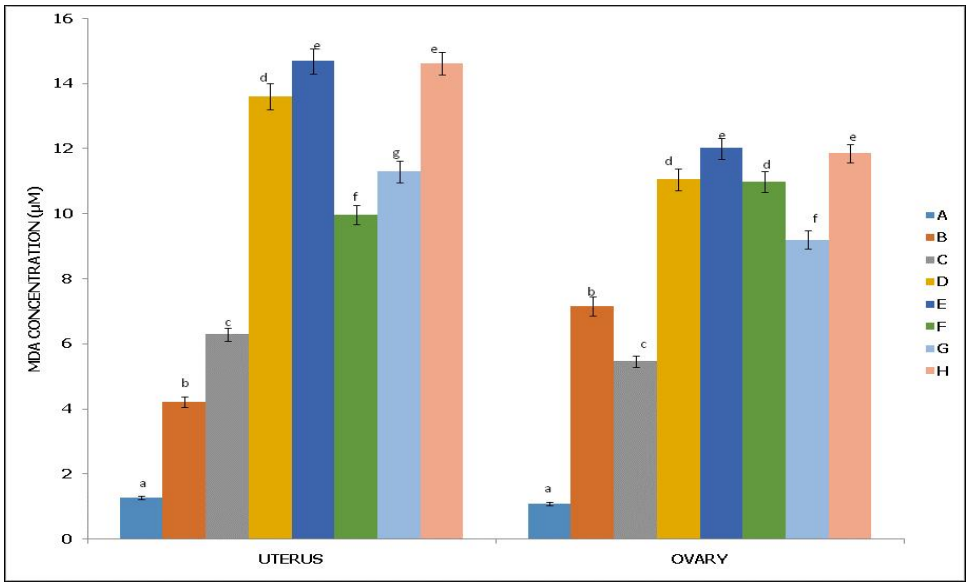


**Figure 4:** Concentration of malondialdehyde (MDA) of uterus and ovary of postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

moderate oxidative stress. Group H (dichlorvos + dimethoate + cypermethrin exposure) has an MDA concentration comparable to Group E and significantly higher than most other groups, underscoring the compounding toxic effects of the triple pesticide mixture.

**In the Ovary,** Group A (Control) again exhibits the lowest MDA concentration, significantly lower ($p<0.05$) than in all other groups, indicating low oxidative stress in the absence of pesticides. Group B (dichlorvos exposure) shows MDA levels significantly higher than the control but lower than in Groups F and E, reflecting moderate oxidative stress. Group C (dimethoate exposure) has an MDA concentration similar to Group B and significantly lower than Group E, showing a mild oxidative effect. Group D (cypermethrin exposure) displays MDA levels significantly lower than in Groups E and F, similar to Group C, indicating moderate oxidative stress. Group E (dichlorvos + dimethoate exposure) has the highest MDA concentration, significantly elevated compared to all groups except Group H, highlighting severe oxidative damage. Group F (dichlorvos + cypermethrin exposure) has MDA levels similar to Group E and significantly higher than in Group C, indicating marked lipid peroxidation. Group G (dimethoate + cypermethrin exposure) shows lower MDA levels than Group F but significantly higher than Groups C and D, reflecting moderate oxidative damage. Group H (dichlorvos + dimethoate + cypermethrin exposure) has MDA levels comparable to Group E, significantly higher than Groups C and D, suggesting a substantial oxidative effect from the combined pesticide exposure.

In summary, MDA levels are lowest in the control group across both uterus and ovary tissues, with substantial increases observed across all pesticide-exposed groups. The highest MDA concentrations occur in groups exposed to dichlorvos combined with dimethoate or cypermethrin, and the triple mixture, particularly in Groups E and H, demonstrates the strongest oxidative stress effects in both uterine and ovarian tissues. These findings emphasize the elevated oxidative stress risks associated with multiple pesticide exposures in postpartum female rats.

Figure 5 presents the Superoxide Dismutase (SOD) activity (U/mg protein) in the uterus and ovary tissues of different exposure groups.

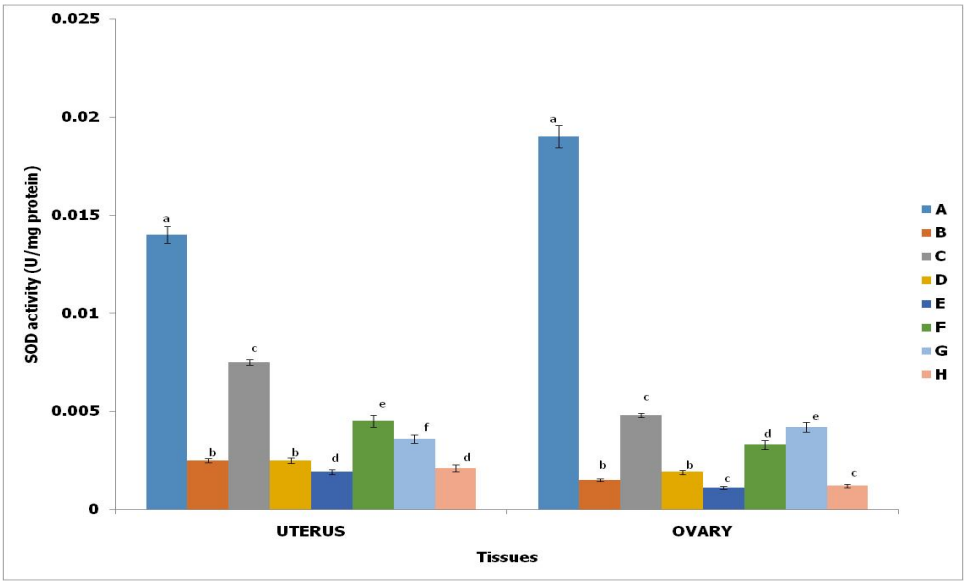


**Figure 5:** Specific activity of Superoxide dismutase of uterus and ovary of non-postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

Group A (Control), exposed only to sprayed water, demonstrates the highest SOD activity in both tissues, with significantly higher values ($p<0.05$) than all other groups. This high SOD activity in the control suggests optimal antioxidant protection in the absence of pesticide exposure.

Group B, exposed to dichlorvos, shows a marked reduction in SOD activity ($p<0.05$) in both uterus and ovary tissues compared to the control, though its activity is still higher than in some other exposure groups, indicating a partial compromise in antioxidant defenses due to dichlorvos exposure. Similarly, Group C, exposed to dimethoate, exhibits significantly reduced SOD activity ($p<0.05$) relative to the control in both tissues. Interestingly, SOD activity in the ovary appears slightly higher in Group C than in Group B, possibly suggesting a lesser impact on ovarian tissue from dimethoate alone.

In Group D, exposed to cypermethrin, the lowest SOD activity is observed in both uterus and ovary tissues. This activity is significantly lower ($p<0.05$) than in the control and most other groups, except for Group E in the uterus, indicating a severe reduction in SOD activity and suggesting strong oxidative stress effects from cypermethrin exposure. Group E, exposed to a combination of dichlorvos and dimethoate, also shows low SOD activity, with values significantly lower than the control ($p<0.05$), though the difference from Group D in the uterus is not statistically significant ($p>0.05$).

Group F, which was exposed to dichlorvos and cypermethrin, exhibits moderate SOD activity levels. These are significantly higher ($p<0.05$) than those in Group D but remain lower than the control group, indicating a partial preservation of SOD activity despite combined pesticide exposure. Group G, exposed to dimethoate and cypermethrin, follows a similar trend to Group F, with SOD activity levels that are significantly lower than the control ($p<0.05$) but still higher than the individual cypermethrin-exposed group, suggesting some relative preservation of antioxidant capacity when dimethoate is combined with cypermethrin.

Lastly, Group H, exposed to a mixture of dichlorvos, dimethoate, and cypermethrin, displays SOD activity significantly lower ($p<0.05$) than the control group. In the ovary, its activity is not significantly different ($p>0.05$) from Group G, indicating that this multi-pesticide combination does not further exacerbate the reduction in SOD activity beyond the effects observed in Groups F and G.

Overall, SOD activity is highest in the control group and decreases progressively with pesticide exposure, with the most substantial reductions observed in groups exposed to cypermethrin alone or in combination with other pesticides. These results suggest that pesticide exposure, especially to cypermethrin, significantly impairs antioxidant defense mechanisms in both uterine and ovarian tissues.

Figure 6 presents the SOD (Superoxide Dismutase) specific activity (U/mg protein) in the uterus and ovary tissues across various exposure groups. Group A, the control group exposed only to sprayed water, exhibits the highest SOD specific activity in both uterus and ovary tissues, with values significantly higher ($p<0.05$) than in all other groups. This elevated activity in the control group reflects optimal antioxidant defense in the absence of pesticide exposure.

Group B, exposed to dichlorvos, shows a substantial reduction in SOD specific activity compared to the control ($p<0.05$) in both tissues. However, it retains higher activity than groups exposed to dichlorvos in combination with other pesticides, indicating that dichlorvos alone, while impactful, may have a less severe effect on SOD activity than its combinations. Group C, exposed to dimethoate, also demonstrates significantly lower SOD specific activity ($p<0.05$) than the control in both uterus and ovary tissues, although the activity in the ovary remains higher ($p<0.05$) than in Group D, suggesting some preservation of antioxidant function in the ovary with dimethoate exposure.

Group D, exposed to cypermethrin, exhibits one of the lowest SOD specific activity levels among all groups, with significantly reduced activity ($p<0.05$) relative to the control, particularly in the uterus. This indicates a substantial oxidative stress effect from cypermethrin exposure, with a strong reduction in SOD capacity. Group E, a combination of dichlorvos and dimethoate, also shows low SOD specific activity, with levels significantly lower ($p<0.05$) than in the control group, suggesting an additive effect on reducing antioxidant function due to the combination of these pesticides.

In Group F, which was exposed to both dichlorvos and cypermethrin, SOD specific activity in the ovary is significantly higher ($p<0.05$) than in Group D, although it remains below control levels. This indicates that the combination of dichlorvos with cypermethrin does not further decrease activity as severely as cypermethrin alone in the ovary. Group G, exposed to dimethoate and cypermethrin, shows a significantly lower SOD specific activity ($p<0.05$) in both the uterus and ovary compared to the control, reflecting the detrimental effects of

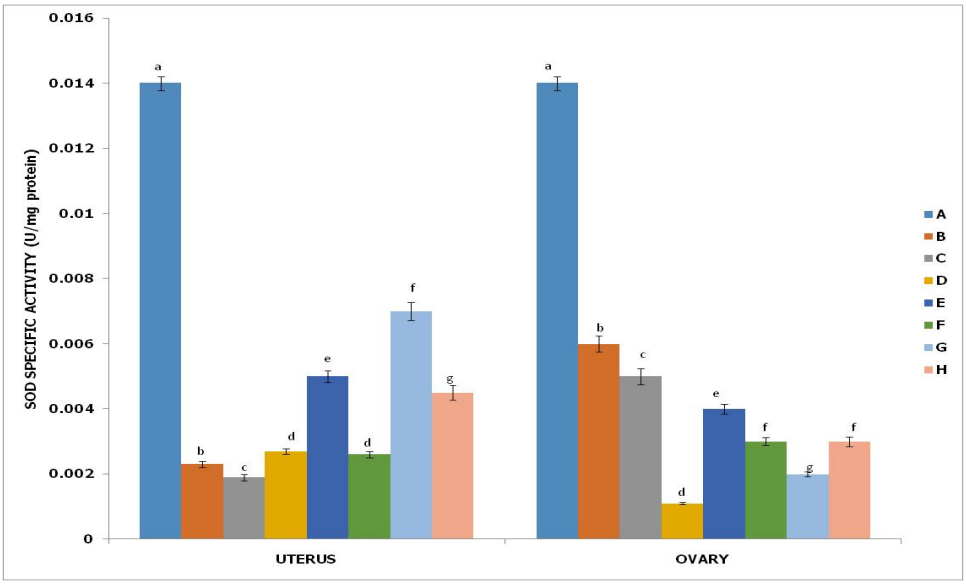


**Figure 6:** Specific activity of Superoxide dismutase of uterus and ovary of postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

this pesticide combination on antioxidant defenses.

Lastly, Group H, exposed to a mixture of dichlorvos, dimethoate, and cypermethrin, displays significantly lower SOD specific activity ($p<0.05$) than the control in both tissues. Its activity is not significantly different ($p>0.05$) from Group G, indicating that the addition of a third pesticide does not exacerbate the reduction in SOD activity beyond the effect observed with the dimethoate and cypermethrin combination alone.

In summary, the SOD specific activity is highest in the control group and decreases progressively with pesticide exposure, with the most substantial reductions observed in groups exposed to cypermethrin, alone or in combination. These findings suggest that pesticide exposure, particularly with cypermethrin, severely compromises antioxidant defenses in uterine and ovarian tissues, increasing the risk of oxidative stress.

Figure 7 illustrates the specific activity of catalase in the uterus and ovary tissues of non-postpartum female albino rats across various exposure groups.

**In the Uterus,** Group A (Control) demonstrates the highest catalase activity, significantly higher ($p < 0.05$) than Groups C, D, F, and G, but not significantly different ($p > 0.05$) from Groups B and H. This high catalase activity in the control group reflects an optimal antioxidant environment in the absence of pesticide exposure. Group B, while lower than the control, still shows catalase activity levels significantly higher ($p < 0.05$) than Groups C, D, F, and G, indicating a moderate resilience against oxidative stress induced by dichlorvos. In contrast, Groups C and D exhibit lower catalase activities, both significantly lower ($p < 0.05$) than the control, which suggests compromised antioxidant defenses in response to dimethoate and cypermethrin exposure. Groups E and H show moderate catalase activity, with no significant difference ($p > 0.05$) between them, placing them between the high control levels and the reduced levels seen in other pesticide-exposed groups like C and D.

**In the Ovary,** Group A (Control) again shows the highest catalase activity, significantly surpassing ($p < 0.05$) all other groups, reflecting strong antioxidant capacity in the absence of chemical exposure. Group B follows, with catalase activity significantly higher ($p < 0.05$) than in Groups C, D, E, F, and H, suggesting a lesser but still effective antioxidant response when exposed only to dichlorvos. Groups C and D, exposed to dimethoate and cypermethrin respectively, have catalase activities significantly higher than Group E but comparable to Group F, placing them in a moderate range of antioxidant response. However, Groups F and G exhibit significantly lower ($p < 0.05$) catalase activity than the control group and most other pesticide-exposed groups, with Group G showing the lowest activity, indicating a more substantial depletion of antioxidant defense in the ovary tissue with this exposure combination.

In summary, the catalase activity is highest in the control group for both uterus and ovary tissues, reflecting strong antioxidant defenses in the absence of pesticide exposure. Pesticide exposure, particularly in Groups C, D, F, and G, leads to significantly lower catalase activity, indicating compromised antioxidant function and increased vulnerability to oxidative stress. This decrease in catalase activity across pesticide-exposed groups, especially in the ovary, highlights the oxidative damage potential of these pesticides on reproductive tissues.

Figure 8 details the specific activity of catalase in the uterus and ovary tissues of postpartum female albino rats exposed to various pesticides.

**In the Uterus,** Group A (Control) shows the highest catalase activity, with levels significantly higher ($p <0.05$) than all other groups. Group B, while lower than the control, retains catalase activity that is significantly higher than in Groups C, D, E, F, G, and H, which show progressively declining levels. A stepwise decrease is noted in Groups C, D, and E, whereas Groups F, G, and H exhibit the lowest catalase activity, with no significant differences ($p > 0.05$) observed among them. This pattern indicates a cumulative impact of pesticide exposure on catalase activity, with combined exposures leading to greater reductions in enzymatic activity.

**In the Ovary,** Group A again has the highest catalase activity, significantly higher ($p < 0.05$) than in any of the other groups, underscoring strong antioxidant function in the absence of chemical exposure. Group B follows, with catalase activity that is significantly higher than in Groups C, D, E, and the others, showing a gradual decline across subsequent groups. Group H shows the lowest catalase activity, indicating an especially pronounced decline in antioxidant response with combined pesticide exposure. Groups E, F, G, and H exhibit progressively lower catalase activity levels, each significantly lower ($p < 0.05$) than Group B, highlighting the detrimental impact of cumulative pesticide combinations on ovarian catalase activity.

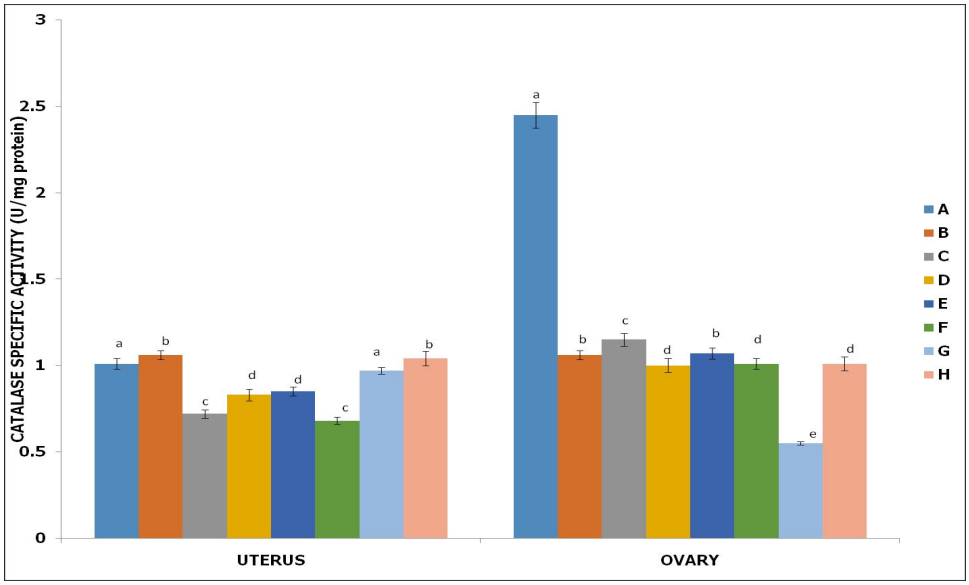


**Figure 7:** Specific activity of catalase of uterus and ovary of non-postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

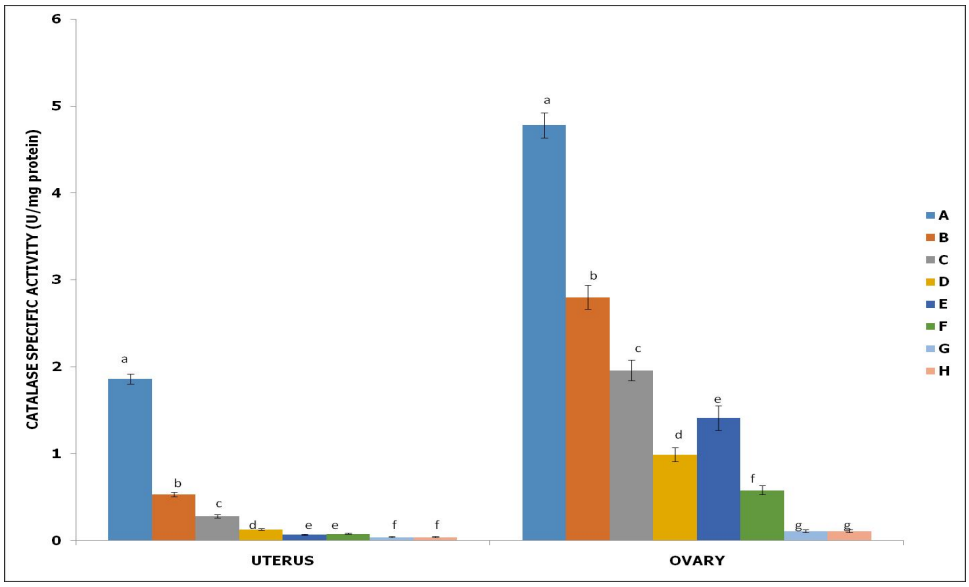


**Figure 8:** Specific activity of catalase of uterus and ovary of postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

Catalase activity is consistently highest in the control group (Group A) across both uterus and ovary tissues in postpartum rats, with a marked decline in groups exposed to pesticides. Significant differences in catalase activity exist between the groups ($p < 0.05$), with pesticides such as dichlorvos, dimethoate, and cypermethrin associated with reduced enzymatic activity, particularly in the postpartum condition. Combined exposures (Groups F, G, and H) result in even lower catalase activity than single pesticide exposures, emphasizing the cumulative oxidative stress effects of these chemical combinations on catalase function in reproductive tissues.

Figure 9 illustrates the Glutathione S-Transferase (GST) specific activity in the ovary and uterus of non-postpartum female rats across various pesticide exposure groups.

**Group A (Control - Exposed to Sprayed Water)** shows significantly higher ($p<0.05$) GST activity in both the ovary and uterus compared to all other groups, representing normal GST function without pesticide exposure. This high activity level reflects optimal antioxidant defense mechanisms in the absence of toxic agents.

**Group B (Exposed to Dichlorvos)** exhibits significantly lower ($p<0.05$) GST activity in both tissues relative to the control, although the levels remain higher than in Group H. The reduction in GST activity highlights the inhibitory effect of dichlorvos, though it is not the most toxic single pesticide treatment.

**Group C (Exposed to Dimethoate)** also demonstrates significantly lower ($p<0.05$) GST activity in both the ovary and uterus compared to the control, with activity levels higher than in Groups D and H. This indicates a negative impact on GST activity from dimethoate exposure, though it is less severe than the effects seen with cypermethrin and the three-pesticide mixture.

**Group D (Exposed to Cypermethrin)** shows a significant decrease ($p<0.05$) in GST activity in both tissues compared to the control and Groups B and C, although GST activity remains higher than in Group H. This suggests that cypermethrin is more toxic than either dichlorvos or dimethoate when administered individually.

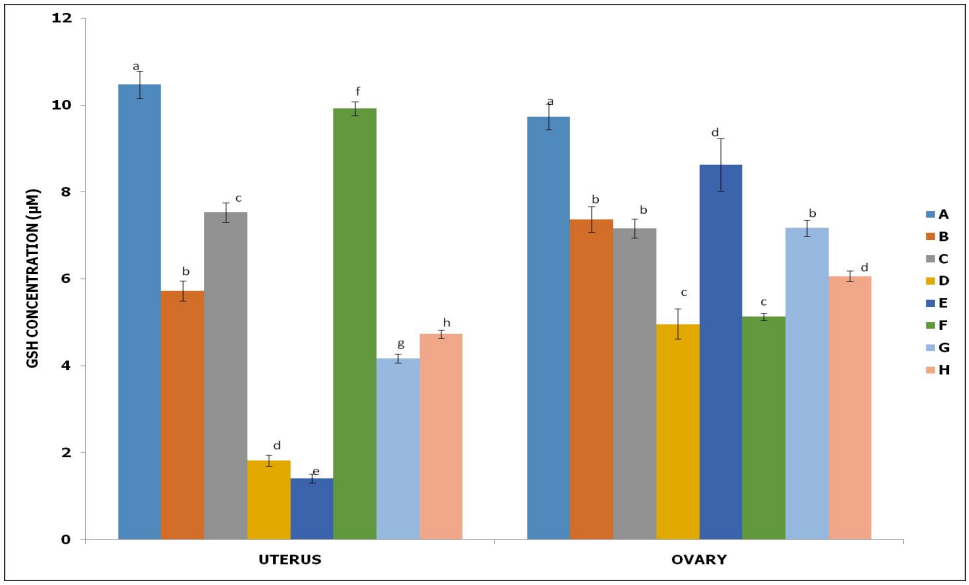


**Figure 9:** Specific activity of glutathione-S-transferase (GST) of uterus and ovary of non-postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$)

**Group E (Exposed to Dichlorvos and Dimethoate)** presents significantly lower ($p<0.05$) GST activity in both the ovary and uterus than the control, indicating a compounded toxic effect due to the combination of these two pesticides. The reduction in GST activity here is more pronounced than with single pesticide exposures.
**Group F (Exposed to Dichlorvos and Cypermethrin)** shows a marked reduction ($p<0.05$) in GST activity in both tissues compared to the control, with activity levels higher than in Groups E and H. This indicates that while the dichlorvos and cypermethrin combination significantly impacts GST function, it is not the most severe among combinations.
**Group G (Exposed to Dimethoate and Cypermethrin)** has GST activity significantly lower ($p<0.05$) than in Groups A, B, and C but higher than in Group H. The combined exposure of dimethoate and cypermethrin reduces GST activity substantially, though it is less toxic than the triple pesticide mixture.
**Group H (Exposed to Dichlorvos, Dimethoate, and Cypermethrin)** displays the lowest GST activity in both tissues, significantly lower ($p<0.05$) than all other groups. This triple pesticide exposure results in the strongest compounding toxic effect on GST activity in non-postpartum female rats, indicating a severely compromised antioxidant defense.

Figure 10 illustrates the Glutathione S-Transferase (GST) specific activity in the ovary and uterus of postpartum female rats across different pesticide exposure groups.
**Group A (Control - Exposed to Sprayed Water)** shows significantly higher ($p<0.05$) GST activity in both the ovary and uterus compared to all other groups, mirroring the control activity levels in non-postpartum rats seen in Figure 7. The postpartum state does not appear to affect the baseline GST activity in control animals, maintaining optimal antioxidant function without pesticide exposure.
**Group B (Exposed to Dichlorvos)** presents significantly lower ($p<0.05$) GST activity in both tissues relative to the control, although GST levels remain higher than in Groups D, E, and H. This indicates a toxic effect of dichlorvos, with postpartum rats exhibiting a similar pattern of GST reduction as non-postpartum rats, showing no substantial change due to postpartum physiology.
**Group C (Exposed to Dimethoate)** also demonstrates significantly lower ($p<0.05$) GST activity than the control, though GST activity levels are higher than in Groups D and H. Dimethoate's impact on postpartum rats aligns with its effects on non-postpartum rats, indicating a moderate reduction in GST activity but less severe toxicity compared to combined pesticide exposures.
**Group D (Exposed to Cypermethrin)** shows a marked reduction in GST activity, with significantly lower ($p<0.05$) levels than in Groups A, B, and C but higher than in Group H. This suggests that cypermethrin has a strong toxic effect on GST activity, more pronounced than either dichlorvos or dimethoate alone, consistent with findings in non-postpartum rats.
**Group E (Exposed to Dichlorvos and Dimethoate)** displays significantly lower ($p<0.05$) GST activity in both tissues compared to the control and single pesticide exposures, underscoring the heightened toxic effect of the dichlorvos and dimethoate combination in postpartum rats.
**Group F (Exposed to Dichlorvos and Cypermethrin)** exhibits a significant reduction in GST activity compared to the control, though the levels remain higher than in Groups E and H. This combination reduces GST activity but does not have as extreme an impact as the triple pesticide mixture.
**Group G (Exposed to Dimethoate and Cypermethrin)** shows significantly lower ($p<0.05$) GST activity in both tissues compared to Groups A, B, and C but maintains higher levels than Group H. This combined exposure significantly decreases GST, though it is slightly less toxic compared to the effects of the triple pesticide mixture.
**Group H (Exposed to Dichlorvos, Dimethoate, and Cypermethrin)** demonstrates the lowest GST activity across all groups, significantly reduced ($p<0.05$) compared to the other groups. This triple exposure exerts the strongest toxic effect on GST activity in postpartum rats, similar to the trend observed in non-postpartum animals, highlighting the severe impact of combined pesticide exposures.

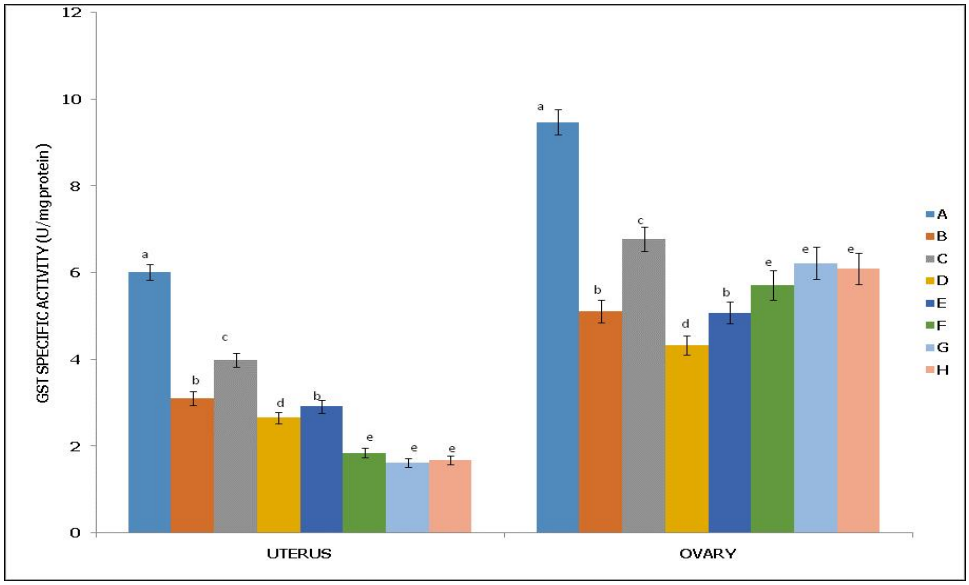


**Figure 10:** Specific activity of glutathione-S-transferase (GST) of uterus and ovary of postpartum female wistar albino rats exposed to mixture of dichlorvos, dimethoate and cypermethrin. Calculated values are means of four determinations ± SEM. Bars bearing different alphabets are significantly different ($p<0.05$))

## 4. Discussion

The investigation revealed significant reductions in antioxidant enzyme activities, including superoxide dismutase (SOD), catalase, glutathione S-transferase (GST), and glutathione (GSH), across groups exposed to Dichlorvos, Dimethoate, and Cypermethrin, both individually and in combinations. Malondialdehyde (MDA), a marker of lipid peroxidation, showed substantial increases, particularly in combined exposure groups. These results highlight the oxidative burden induced by pesticides, with the most pronounced effects observed in groups exposed to Dichlorvos-containing mixtures.

The oxidative stress induced by pesticide exposure is well-established in the literature. Manna et al. [20] documented elevated MDA levels and reduced SOD and catalase activities in rats exposed to Cypermethrin, linking these changes to increased reactive oxygen species (ROS) production and lipid peroxidation [20, 21] reported reductions in erythrocyte glutathione peroxidase and plasma membrane fluidity in Cypermethrin-treated rats, attributing these effects to oxidative damage [21].

The observed increases in MDA levels in pesticide-exposed groups corroborate findings from other studies, such as those by [22], who showed that Cypermethrin exposure significantly raised MDA levels while decreasing GSH, indicating lipid peroxidation and compromised antioxidant defenses [22]. Çelik et al. [23] also found that Dichlorvos exposure led to decreased antioxidant enzyme activities and increased oxidative stress biomarkers, supporting the present study's findings [23].

The exacerbated oxidative stress in groups exposed to combined pesticides aligns with studies demonstrating the additive or synergistic effects of multiple toxicants. Lengyel et al. [24] reported that combined exposure to Dimethoate and Cypermethrin significantly heightened oxidative stress, as evidenced by increased MDA and altered cortical responses [24]. The current findings further reinforce the potential for combined pesticides to overwhelm antioxidant defense systems, leading to heightened lipid peroxidation and cellular damage.

Postpartum rats showed relatively higher SOD, catalase, and GST activities compared to non-postpartum females, suggesting enhanced antioxidant capacity. This observation aligns with research indicating physiological adaptations in postpartum states that may confer resistance to oxidative insults. Further exploration is required to elucidate the underlying mechanisms, such as hormonal modulation or enhanced metabolic activity.

The data unequivocally indicate that pesticide exposure disrupts antioxidant defenses, elevates oxidative stress markers, and induces lipid peroxidation, particularly in the context of combined exposures. These findings underscore the importance of limiting exposure to pesticide mixtures and suggest the need for therapeutic interventions targeting oxidative stress pathways. Investigations into the protective role of postpartum physiology could inform strategies to mitigate oxidative damage.

## 5. Conclusion

This study highlights the substantial oxidative stress induced by pesticide exposure, particularly dichlorvos, dimethoate, and cypermethrin, in postpartum and non-postpartum female Wistar rats. The findings demonstrate that combined pesticide exposures significantly exacerbate oxidative stress, as evidenced by elevated malondialdehyde (MDA) levels and reduced antioxidant enzyme activities, including superoxide dismutase (SOD), catalase, and glutathione S-transferase (GST). The triple combination of pesticides had the most pronounced adverse effects, severely compromising antioxidant defenses in both uterine and ovarian tissues.

Notably, postpartum rats exhibited relatively enhanced antioxidant capacities compared to non-postpartum counterparts, suggesting potential physiological adaptations that confer some resilience against oxidative insults. This observation emphasizes the complex interplay between biological states and toxicant susceptibility.

The study underscores the compounded risks posed by pesticide mixtures and the necessity of stringent regulatory measures to limit such exposures. Furthermore, it advocates for the exploration of therapeutic strategies targeting oxidative stress pathways to mitigate pesticide toxicity. These findings contribute to the understanding of reproductive toxicology and provide a basis for developing interventions to protect female health during critical life stages. Future research should delve into the mechanisms underpinning the observed physiological

adaptations in postpartum states and explore the potential long-term impacts of pesticide exposure on reproductive health and offspring development.

## References


[1] M. Jokanoviŏ107. Neurotoxic effects of organophosphorus pesticides. *Environmental Toxicology and Pharmacology*, 58:37–41, 2018. doi: 10.1016/j.etap.2018.07.001.

[2] X. Zhang, L. Guo, and Y. Wang. Pyrethroid and organophosphate pesticide-induced oxidative stress: A comparative review. *Toxicology Reports*, 7:1135–1146, 2020. doi: 10.1016/j.toxrep.2020.08.019.

[3] V. I. Lushchak. Free radicals, reactive oxygen species, oxidative stress, and their physiological roles. *Physiological Reviews*, 94(3): 909–950, 2014. doi: 10.1152/physrev.00008.2014.

[4] H. Sies, C. Berndt, and D. P. Jones. Oxidative stress. *Annual Review of Biochemistry*, 86:715–748, 2017. doi: 10.1146/annurev-biochem-061516-045037.

[5] C. Gomez-Alonso, C. Alvarez-Builla, and M. San Andres. Endocrine disruptors and oxidative stress in female reproductive health. *Toxicology Letters*, 312:56–68, 2019. doi: 10.1016/j.toxlet.2019.07.020.

[6] N. Chellan, V. Choudhary, and S. K. Gupta. Hormonal fluctuations and metabolic challenges in postpartum and non-postpartum females: Implications for oxidative stress. *Reproductive Endocrinology and Biology*, 28(1):12–20, 2021. doi: 10.1007/s12020-021-01789-4.

[7] J. D. Hayes, J. U. Flanagan, and I. R. Jowsey. Glutathione transferases. *Annual Review of Pharmacology and Toxicology*, 45:51–88, 2005. doi: 10.1146/annurev.pharmtox.45.120403.095857.

[8] S. P. Singh and V. Sharma. Biomarkers of oxidative stress in pesticide toxicity. *Journal of Toxicological Sciences*, 44(6):409–417, 2019. doi: 10.2131/jts.44.409.

[9] S. Tripathi and A. K. Srivastava. Impacts of pesticide exposure on antioxidant enzyme activities in experimental models. *Environmental Research*, 204:112378, 2022. doi: 10.1016/j.envres.2022.112378.

[10] L. R. Borges, M. C. Oliveira, A. R. Silva, and A. C. Resende. Physiological adaptations during the postpartum period and their impact on female health. *Journal of Reproductive Biology*, 15(2):45–52, 2017. doi: 10.1016/j.jreprobiol.2017.02.004.

[11] R. E. Smith and K. M. Taylor. Hormonal stability in non-postpartum females and its role in oxidative stress modulation. *Reproductive Toxicology*, 80:57–63, 2018. doi: 10.1016/j.reprotox.2018.02.006.

[12] M. R. Miller, A. K. Smith, and D. P. Jones. Pesticide exposure and antioxidant defenses in female reproductive health. *Environmental Health Perspectives*, 128(6):061002, 2020. doi: 10.1289/EHP6760.

[13] C. D. Nwani and H. R. Singh. Effects of pesticide mixtures on oxidative stress biomarkers in experimental animal models. *Ecotoxicology and Environmental Safety*, 221:112452, 2021. doi: 10.1016/j.ecoenv.2021.112452.

[14] R. P. Bird, H. H. Drapper, and V. E. Valli. Toxicological evaluation of malondialdehyde: a 12-month study of mice. *J. Toxicol. Environ. Health*, 10:897–905, 1982.

[15] D. J. Jollow, J. R. Mitchell, and J. R. Gillette. Bromobenzene induced liver necrosis: Protective role of glutathione and evidence for 3,4-bromobenzene oxide as the hepatotoxic metabolite. *Pharmacol.*, 11:151–169, 1974.

[16] A. G. Gornal, J. C. Bardawill, and M. M. David. Determination of serum proteins by means of biuret reaction j. *Biol. Chem.*, 177: 751–760, 1949.

[17] H. P. Misra and I. Fridovich. The role of superoxide anion in the antioxidation of epinephrine and a simple assay of superoxide dismutase. *J.*, Bio1. Chem:2417–3170, 1972.

[18] K. A. Sinha. Colorimetric assay of catalase. *Anal. Biochem.*, 47:389–394, 1971.

[19] W. H. Habig, M. J. Pabst, and W. B. Jakoby. Glutathione-s-transferase: the first step in mercapturic acid formation. *J. Biochem.*, 249: 7130–7139, 1974.

[20] S. Manna, D. Bhattacharyya, T. K. Mandal, and S. Das. Repeated dose toxicity of cypermethrin in rats. *Journal of Veterinary Science*, 5(3):241–245, 2004. doi: 10.4142/jvs.2004.5.3.241.

[21] R. Gabbianelli, G. Falcioni, C. Nasuti, and F. Cantalamessa. Cypermethrin-induced plasma membrane perturbation in rat erythrocytes: Reduction of antioxidant capacity. *Toxicology*, 179(1-2):63–71, 2002. doi: 10.1016/S0300-483X(02)00304-4.

[22] F. Rashid, F. Ali, and T. Khan. Cypermethrin-induced oxidative stress and lipid peroxidation: Modulation by antioxidants in a rat model. *Environmental Toxicology and Pharmacology*, 94:103852, 2022. doi: 10.1016/j.etap.2022.103852.

[23] I. Çelik, Z. Yılmaz, and M. Büyüköztürk. Dichlorvos-induced oxidative stress and its effects on antioxidant enzyme activities in rats. *Ecotoxicology and Environmental Safety*, 72(3):905–911, 2009. doi: 10.1016/j.ecoenv.2008.11.007.

[24] G. Lengyel, T. MŎ0f3zes, K. Fekete, and E. Hermesz. Combined effects of dimethoate and cypermethrin on biochemical and electrophysiological parameters of the brain in rats. *Pesticide Biochemistry and Physiology*, 83(1):60–67, 2005. doi: 10.1016/j.pestbp.2005.03.009.