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Evaluation effects of zinc oxide nanoparticles on antioxidant markers including superoxide dismutase (SOD) and glutathione peroxidase (GPX), oxidative stress marker malondialdehyde (MDA) in male mice

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ABSTRACT

Background and aim: Humans are frequently exposed to zinc oxide nanoparticles (ZnO NPs) as they are widely used in several fields. Some studies have reported their toxic effects on various cells and proteins. Therefore, we aimed to study their effects on antioxidant markers including superoxide dismutase (SOD) and glutathione peroxidase (GPX), as well as the oxidative stress marker malondialdehyde (MDA) on male mice.

Materials and methods: A total of 30 adult NMRI male mice, two months of age and weighing 28-32 g, were used in this study. The mice were divided into five groups, each consisting of six mice. One group served as the control, one as the sham group and received only distilled water, and the remaining three were treatment groups that received intraperitoneal ZnO NPs at doses of 250, 500, and 700 mg/kg, respectively. After a 7-day observation period following ZnO NP administration, blood samples were collected, and serum SOD and GPX activities and MDA concentration were measured using commercially available assay kits according to the manufacturers' instructions. One-way ANOVA was used for statistical analysis.

Results: In male mice exposed to ZnO NPs, SOD activity increased progressively with increasing ZnO NP dose, with statistically significant increases at 500 and 700 mg/kg ($P<0.05$). GPX activity increased significantly following treatment with 250 and 700 mg/kg ZnO NPs, whereas a decrease was observed at 500 mg/kg. MDA concentration showed a slight but non-significant decrease following treatment with 250 and 500 mg/kg ZnO NPs, whereas a significant increase was observed following treatment with 700 mg/kg ZnO NPs.

Conclusion: The increased SOD and GPX activities at some ZnO NP doses may reflect activation of the antioxidant defense system, while the significant increase in MDA at 700 mg/kg indicates greater oxidative stress at the highest dose.

Keywords: Zinc oxide nanoparticles (ZnO NPs), oxidative stress, antioxidant activity, superoxide dismutase (SOD), glutathione peroxidase (GPX), malondialdehyde (MDA), male mice.

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Introduction

Today, zinc oxide nanoparticles (ZnO NPs) are widely used in biomedical fields, such as drug delivery, anticancer drugs, antimicrobials, and diagnosis, as well as in the food industry (as food additives), cosmetics (mainly in sunscreens and creams), environmental cleanup, paints, and electronics for their unique properties (1-3). These particles are in fact highly conductive, chemically stable, catalytic, and possess UV filtering features. Notably, they have an increased surface-to-volume ratio and a diameter of 1-100 nm, thus highly capable of penetrating cells (4).

The reduced size and increased surface area of NPs lead to greater contact with either organic or non-organic molecules, thereby leading to various chemical reactions. Thus, these NPs can greatly interact with biological systems leading to unforeseen results. Many studies have indicated the cytotoxic or genotoxic effects of ZnO NPs which include membrane disruption, cell apoptosis, DNA damage, and lipid peroxidation usually by elevating reactive oxygen species (ROS) levels and inducing oxidative stress in cells (5-7).

Oxidative stress occurs following an imbalance between ROS production and antioxidant defense mechanisms in the human body as indicated by the overproduction of ROS

induced by ZnO NPs (8). Following ROS production, free oxygen radicals increase which can lead to peroxidative breakdown of phospholipids causing malondialdehyde (MDA) accumulation. MDA is important in the modification of low density lipoprotein (LDL), which is involved in the patho-physiological alterations due to auto-oxidative glycosylation (9). Typically, cells employ several defense mechanisms against ROS and free radicals such as the production of enzymes that can be used for the elimination of ROS (10). These enzymes include various peroxidases such as glutathione peroxidase (GPX) which removes hydrogen peroxide, and superoxide dismutase (SOD) which removes superoxide radicals. In fact, these enzymes are particularly important in cell protection and maintenance of mitochondrial integrity following exposure to elevated levels of ROS (11). Although oxidative stress induced by ZnO NPs has been indicated, some studies have also suggested the antioxidant properties of these particles (12, 13). In spite of recent attempts, the underlying mechanisms of these nanoparticles in human body have not been thoroughly determined.

Recently, there has been much interest regarding the toxicity of ZnO nanoparticles as they can easily enter the body and reach the blood or several body organs through food or other routes due to their wide applications. This study aimed to investigate the in vivo effect of ZnO NPs on the status of oxidative stress in adult male mice by measuring the activities of antioxidative enzymes and the concentration of oxidative stress biomarker MDA.

Materials and Methods

ZnO nanoparticles

In this study, ZnO nanoparticles were purchased from Pars Lima Company (Tehran, Iran) in the form of white nanopowder with spherical shape. According to the manufacturer's specifications, these NPs were 20 nm in diameter, had a calculated surface area of >90 m^2/g, and were highly pure with a purity of >99% trace metal basis.

Preparation of ZnO NP solution

The required amount of ZnO NPs was weighed according to the body weight and assigned dose of each mouse and suspended in 1 mL of double-distilled water. The suspension was vortexed for 1–2 min at room temperature to ensure adequate mixing and was immediately administered intraperitoneally without storage. No additional surfactant, buffer, or dispersant was used.

Study design

In the current study, a total of 30 2-month-old male NMRI mice, weighing 28-32 g were purchased from Pasteur Institute of Iran. These animals were kept under standard housing conditions of 23 ºC and relative humidity of 45-50% and 12 h light/dark cycles. All animals had access to tap water and food pellets.

These animals were randomly assigned to five groups of six mice each as follows: Groups 1, 2, and 3 received a single intraperitoneal (IP) injection of 250, 500, and 700 mg/kg of ZnO NPs each, respectively. Group 4 was the sham group which received 1 mL of distilled water IP as a single injection, and Group 5 was the control group which received neither distilled water nor ZnO NP.

The mice were observed for 7 days after the injection, after which they were anesthetized and sacrificed. Anesthesia was induced by placing cotton pads soaked in diethyl ether inside a closed chamber containing the mice. The mice became anesthetized within several minutes, after which they were removed from the chamber and sacrificed. About 1-1.5 mL of blood samples were collected from their left ventricle. The blood samples were allowed to clot at room temperature for 30 min and were then centrifuged at 2500 rpm for 5 min. Serum was then separated and stored at -20 ºC for further analysis.

The doses of 250, 500, and 700 mg/kg ZnO NPs were selected based on our previous experimental work in adult male NMRI mice using intraperitoneal administration (14). The selected dose range was used to assess dose-related changes in oxidative stress and antioxidant responses, as reported in previous studies of ZnO NP exposure (15).

The serum SOD and GPX activities and MDA concentration were determined using commercially available assay kits (Nasdox™ Superoxide Dismutase Activity Assay Kit, Nagpix™ Glutathione Peroxidase Activity Assay Kit, and Nalondi™ Lipid Peroxidation (MDA) Assay Kit; Navand Salamat, Urmia, Iran) according to the manufacturers' instructions. SOD activity was determined based on the inhibition of pyrogallol autoxidation and measured colorimetrically at 405 nm. GPX activity was determined using a

colorimetric assay based on the enzymatic activity of glutathione peroxidase. MDA concentration was determined using the thiobarbituric acid reactive substances (TBARS) method. SOD activity was expressed as U/mL, GPX activity as U/L, and MDA concentration as μmol/L.

Statistical analysis

Data are presented as mean ± standard deviation (SD). One-way analysis of variance (ANOVA) followed by Tukey's multiple comparisons test was used to compare the groups using SPSS (Version 22.0). A p-value <0.05 was considered statistically significant.

Ethical approval

All experimental procedures involving animals were conducted in accordance with institutional and national guidelines for the care and use of laboratory animals and were reviewed and approved by the Ethics Committee of Islamic Azad University, Tehran Science and Research Branch, in accordance with institutional ethical requirements.

Results

In the current study, blood samples were collected from NMRI mice on day 7 after treatment and were used to determine the activities of oxidant and antioxidant parameters. SOD is an antioxidant enzyme that catalyzes the dismutation of superoxide, thereby converting these radicals to oxygen molecules and hydrogen peroxide.

As shown in Table 1, SOD activity was significantly increased in the 500 and 700 mg/kg ZnO NP groups compared with the control group (P<0.05). Although SOD activity also increased at 250 mg/kg, this difference was not statistically significant. Overall, SOD activity showed a progressive increase with increasing ZnO NP dose (Table 1; Figure 1).

GPX is an antioxidant enzyme involved in peroxide detoxification. Based on the data presented in Table 1, GPX activity increased significantly at 250 and 700 mg/kg of ZnO NPs compared with the control group, whereas a decrease was observed at 500 mg/kg. The increase at 700 mg/kg was statistically more significant than that at 250 mg/kg (P<0.01 vs. P<0.05) (Table 1; Figure 2).

MDA is produced following the peroxidation of polyunsaturated fatty acids. This final product is increased during oxidative stress. In the case of MDA levels, there was a slight and insignificant decrease following treatment with 250 and 500 mg/kg of ZnO NPs. However, treatment with 700 mg/kg of ZnO NP led to a significantly elevated level of this compound.

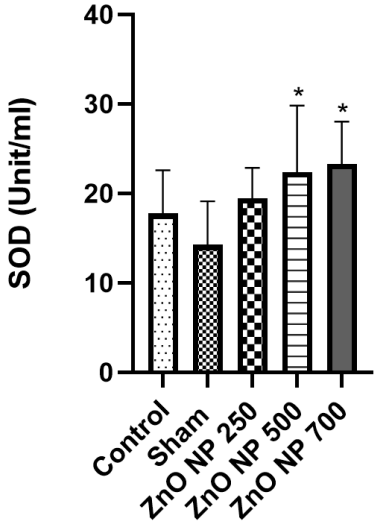


Figure 1. Effect of different concentrations of ZnO NPs on serum activities of SOD in NMRI mice. Error bars indicate mean ± SD. Asterisks indicate statistically significant differences compared with the control group. *P<0.05.

Table 1. The effects of ZnO NPs treatment on oxidant and antioxidant parameters in adult male NMRI mice.

group / parameter	Control	Sham	ZnO NP 250	ZnO NP 500	ZnO NP 700
SOD (unit/mL)	17.7733 ± 4.84026	14.2850 ± 4.86242	19.4667 ± 3.40803	22.4217 ± 7.43142*	23.3133 ± 4.72123*
GPX (unit/L)	1.9467 ± 1.06924	1.5833 ± 0.58688	3.1433 ± 2.89114*	1.5983 ± 0.89309	3.5550 ± 2.15878**
MDA (μmol/L)	1.4050 ± 0.71941	1.1550 ± 0.62298	1.2900 ± 0.61057	1.2433 ± 0.60228	2.1383 ± 0.64698*

All values are presented as mean ± SD. *P<0.05 and **P<0.01 versus the control group, based on one-way ANOVA followed by Tukey's multiple comparisons test.

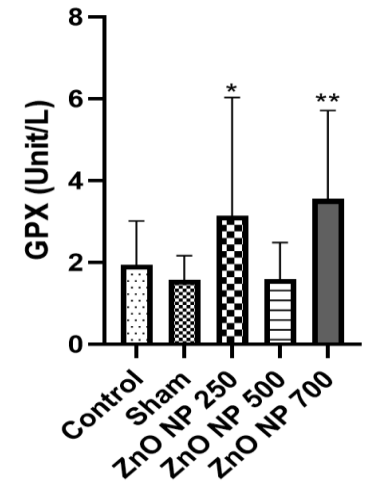


Figure 2. Effect of different concentrations of ZnO NPs on serum activities of GPX in NMRI mice Error bars indicate mean ± SD. Asterisks indicate statistically significant differences compared with the control group. *P<0.05; **P<0.01.

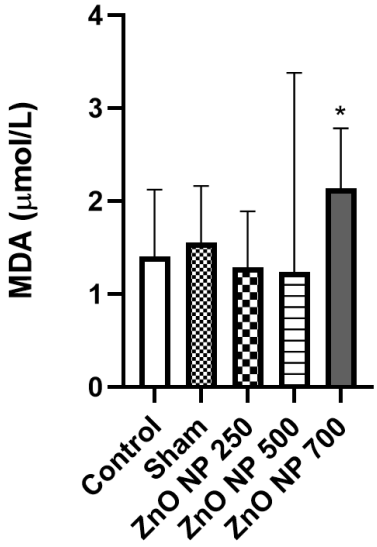


Figure 3. Effect of different concentrations of ZnO NPs on serum levels of MDA in NMRI mice. Error bars indicate mean ± SD. Asterisks indicate statistically significant differences compared with the control group.*P<0.05.

Discussion

At normal concentrations, ZnO NPs possess antioxidant ability at normal concentrations, however, at high levels, toxicity might be observed. ROS production and subsequent oxidative stress may account for this toxicity. The antioxidant defense systems comprise of several low-molecular non-enzymatic antioxidants and antioxidative enzymes. These antioxidant systems protect the cells from stress at molecular levels (16). The present study, therefore, investigated the activities of the main antioxidative enzymes such as SOD and GPX following a 7-day observation period after ZnO NP administration at 250, 500 and 700 mg/kg. Significant increases in SOD (at 500 and 700 mg/kg ZnO NPs) and GPX (at 250 and 700 mg/kg ZnO NPs) activities were observed. These changes in antioxidant enzyme activities may represent an adaptive response to ZnO NP exposure rather than a direct antioxidant action. Also, after assessing ROS production by the oxidative stress biomarker MDA levels, mice treated with high concentrations of ZnO NP (700 mg/kg) showed significantly elevated MDA levels, while at lower concentrations (250 and 500 mg/kg) a slight reduction in MDA levels was observed.

As superoxide is the main ROS generated from several sources, its dismutation by SOD is critical in cells. SOD is present in three forms, CuZnSOD, Mn-SOD, and EC-SOD, which are broadly expressed in the lungs are generally considered as bulk scavengers of superoxide radicals (17). Therefore, in our study, we assessed SOD activities as a marker for antioxidant activity of ZnO NPs. The increase in SOD activity with increasing ZnO NP dose may reflect activation of the endogenous antioxidant defense system in response to oxidative challenge. Therefore, the observed increase in SOD activity should not necessarily be interpreted as evidence of a direct antioxidant effect of ZnO NPs. Similarly, Afifi et al. (2015) revealed a significant upregulation of SOD in the testicular tissue of diabetic rats exposed to ZnO NPs (18). Also, another study revealed the protective effects of ZnO NPs against oxidative injury in mice intestinal epithelial cells (19). In contrast, a study reported that ZnO NPs inhibited the activity of liver SOD in fish after exposure to high concentrations of ZnO NPs (20). Similarly, it was revealed that ZnO NPs had an inhibitory effect on the activity and expression of SOD in gills and liver of *O. nilotica* exposed to high levels of ZnO NPs (21). These differences in results may be attributed to differences in the size of the used NPs. There is a hypothesis that NPs with smaller sizes may lead to more potent oxidative stress and damage, as some studies suggested that smaller NP sizes are associated with higher toxicity levels (22). Moreover, several *in vivo* and *in vitro* studies have shown the time-dependent toxicity of ZnO NPs and have reported that the duration of exposure to these NPs affects the induction of oxidative stress and the antioxidant defense

systems (23). In our study, the duration of exposure of mice to ZnO NPs was 7 days and apparently, antioxidative responses are gradually induced during the time of exposure to help animals adapt to possible oxidative damage induced by NPs (24).

Another antioxidant activity marker, GPX, is the general name of an enzyme family with peroxidase activity. This enzyme acts to protect cells from oxidative damage. The biochemical role of GPX is the reduction of lipid hydroperoxides to their corresponding alcohols and the reduction of free hydrogen peroxide to water (25). The significant increases in GPX activity at 250 and 700 mg/kg may reflect an adaptive or compensatory response of the endogenous antioxidant system to ZnO NP exposure rather than a direct antioxidant effect of the nanoparticles. In a similar study, Negahdary et al. (2015) studied the activity of GPX following exposure to ZnO NPs, Ag NPs, and Au NPs in three classes of male albino Wistar mice. According to their results, mice exposed to Ag NPs showed significantly increased activity of GPX, while ZnO and Au NPs significantly decreased the activities of this antioxidant enzyme (26). This difference from our findings suggests that the GPX response to ZnO NPs may vary according to experimental conditions, and the increase observed in the present study should therefore be interpreted as a possible adaptive response rather than a consistent antioxidant effect. This contrast could be explained by physicochemical properties of the NPs such as agglomeration property, surface shape, size, as well as liberation and solubility. Moreover, exposure duration, administration route, and animal species may affect oxidative stress parameters.

Generally, ROS can lead to lipid peroxidation and membrane lipid disruption, causing the inactivation of enzymes and membrane-bound receptors as well as elevation in tissue permeability. Unsaturated aldehydes and MDA are the products of lipid peroxidation which are capable of forming protein cross-linkages, thereby inactivating many proteins. Therefore, MDA is considered an important biomarker for monitoring lipid peroxidation and the condition of membranes rich in polyunsaturated fatty acids (27, 28). In the present study, although a slight decrease in MDA levels was observed, mice exposed to the highest concentration (700 mg/kg) exhibited a significantly elevated MDA levels. The slight reductions in MDA levels observed at 250 and 500 mg/kg were not statistically significant and therefore do not provide sufficient evidence for a direct antioxidant effect of ZnO NPs at these doses. The numerical reduction in MDA at these doses may reflect changes in oxidative status; however, the absence of statistical significance limits the strength of this interpretation.

The concurrent increase in MDA and antioxidant enzyme activities at the highest ZnO NP dose may indicate activation of antioxidant defenses in response to increased oxidative stress. However, the present study cannot establish a causal relationship between these changes. These results were supported by the study of Afifi et al. who reported greater MDA levels in zebrafish after exposure to higher doses of ZnO NPs (29). Similarly, elevated MDA levels following oral administration of 500 mg/kg ZnO NPs for 10 days have been reported (30). Also, the effect of ZnO NPs exposure on oxidative stress, toxicity, and DNA damage in embryo-larval zebrafish has been reported (31).

The dose-response patterns observed for GPX and MDA were not strictly monotonic. In particular, GPX activity increased at 250 mg/kg, decreased at 500 mg/kg, and increased again at 700 mg/kg, whereas MDA showed slight decreases at 250 and 500 mg/kg followed by a significant increase at 700 mg/kg. Dose response association of ZnO NPs must be assessed in more detail. In some studies, inverse dose dependency effects were suggested. Thus, size-response and concentration-response of ZnO NPs should be further investigated. Moreover, physicochemical properties of the NPs such as agglomeration property, shape, size should be taken into consideration. Finally, exposure duration, administration route, and animal species may affect oxidative stress parameters. Therefore, more studies should be conducted with different treatment groups considering all these influential parameters.

An important limitation of the present study is that body weight, general behavior, clinical signs of toxicity, and mortality were not systematically monitored or recorded during the 7-day observation period. Therefore, the contribution of

overt systemic toxicity to the observed changes in oxidative stress and antioxidant markers could not be assessed.

Conclusion

Here, we showed that SOD activity increased progressively with increasing ZnO NP dose in adult male NMRI mice. Also, a significant increase in GPX activity was observed after exposure to 250 and 700 mg/kg ZnO NPs, while at 500 mg/kg ZnO NPs, a non-significant reduction was observed. Finally, MDA levels were significantly increased following treatment with 700 mg/kg ZnO NPs, whereas slight reductions at 250 and 500 mg/kg were not statistically significant. The increases in SOD and GPX activities may reflect an adaptive or compensatory response to ZnO NP exposure rather than a direct antioxidant effect. In general, high doses of ZnO NPs should be used with caution, and further studies are required to evaluate the exact mechanism and effect of ZnO NPs on the oxidative status of cells and human health.

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