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# Effect of Sulphur Dioxide Inhalation on Oxidative and Histopathological Damage in Kidney and Spleen of *Rattus rattus*

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***Abstract*:** Effect of different concentrations of $SO_2$ (5, 10 and 15ppm) on antioxidant enzymes and histopathological changes were investigated in kidney and spleen of rats of both the sexes under natural and experimental conditions. Naturally exposed, house rat, *Rattusrattus*were collected from urban residential areas at Ludhiana, India. Laboratory rats were exposed to 5, 10 and 15 ppm of $SO_2$ for 5 hours /day for 28 days in 1 $m^3$exposure chamber. All the three concentrations of $SO_2$ showed significant increase in lipid peroxidation levels followed by significant decrease in the activities of superoxide dismutase, catalase and glutathione reductase and non-significant decrease in glutathione peroxidase and glutathione levels in kidney and spleen of rats of both the sexes. The results showed that $SO_2$ is an oxidative damage causing agent to kidney and spleen of rats of both the sexes.

***Keywords*:** Sulphur dioxide ($SO_2$), Rats, Lungs, Brain, Histopathology, Oxidative damage, Air pollutant

In most of the developing countries, air pollution is one of the major problems that arise due to industrial activities and fossil fuel consumption (Ghorani et al 2016). Sulphur dioxide ($SO_2$) is the air pollutant formed by the coal fired power plants, industrial processes and motor vehicle operations. It is present in high concentrations in urban and industrial locations and produces damaging effect on human health (Shen et al 2020). High concentrations of $SO_2$ can affect functioning of vital organs of animals and humans. In developed countries, $SO_2$ at 2 ppm concentration is the industrial maximal limit for 8 hour per day. According to facts, air pollution has contributed to premature death, diseases of cardiovascular system (Al-Kindi et al 2020), respiratory system, neurology system, cancer, diabetes mellitus (DM) and fertility disorders (Li et al 2019, Liu et al 2022). Above 3 ppm concentration, $SO_2$ gas has a strong, nauseating odour. In developed countries, $SO_2$ at 2 ppm concentration is the industrial maximal limit for 8 hour per day. In this study three doses of $SO_2$ i.e. 5, 10 and 15 ppm were taken and these were above the maximum industrial permissible limits of $SO_2$ present in the atmosphere. 5 ppm and 10 ppm concentration of $SO_2$ represents 10 and 20 fold greater than the typical urban concentration (0.5ppm) and is known to induce harmful effects to respiratory system of healthy individuals. Third concentration is 15 ppm which is beyond the natural exposure and is used to examine the effects of this higher concentration of $SO_2$ on the health of individuals.On the basis of this exposure limit three different concentrations of $SO_2$ were selected for the present study. In this study the kidney and spleen of rats were examined for oxidative damage and stress and histopathological damage instigated by $SO_2$ inhalation.

## MATERIAL AND METHODS

The male and female rats of 100-150 g were taken. Naturally exposed (Group I), house rat, *Rattusrattus*were collected from urban residential areas at Ludhiana, India and acclimatized for 1 month in the laboratory. Laboratory rats were divided into four groups. Each group was further subdivided into two subgroups having 6 male and 6 female rats. Group II (control rats) was exposed to filtered air in exposure chamber for 28 days, Group III, IV and V were treated with 5, 10 and 15 ppm of $SO_2$ for 5 hours /day for 28 days in 1 $m^3$ exposure chamber. $SO_2$ gas was provided to treated rats through a tube located at the top of each chamber and was distributed with the help of a fan in each chamber. Rats were kept in cages under standard conditions of humidity and temperature with light-dark cycle. Food (loose mixture of cracked wheat grains, powdered sugar and edible vegetable oil in ratio 96:2:2) and water were provided to control and treated rats *ad libitum*. The weight of individual rat was recorded weekly. After 28 days of exposure rats were dissected. The experiment was performed after the approval of Institutional Animal Ethical Committee, Guru Angad Dev Veterinary and Animal Sciences University Ludhiana, India under protocol No. (GADVASU/2022/IAEC/64/17).

**Assay of antioxidant enzymes:** After dissection kidney and spleen of male and female rats were removed and weighed. The tissues were sheared in 0.9% saline (chilled) and homogenization was done in 0.1 M phosphate buffer pH 7.4. The homogenates were centrifuged for 30 min at 1000 rpm at $4^0$C to obtain supernatants. The tissue supernatants were used for the assay of different antioxidant enzymes like superoxide dismutase (SOD), glutathione S-transferase (GST), catalase (CAT), glutathione (GSH), glutathione peroxidase (GPx), glutathione reductase (GR) and lipid peroxidation (LPO).

**SOD activity:** The kidney and spleensupernatant was used for estimating the activity of SOD by standard method of Marklund and Marklund (1974).

**Glutathione-S-transferase (GST) activity:** GST activity in tissue homogenate was estimated by the method of Habiget al (1974).

**GSH-Px activity:** The GSH-Px activityin tissue homogenate was determined by the method of Hafeman et al(1984).

**Glutathione reductase (GR) activity:** GR enzyme activity was estimated by the method given by Carlberg and Mannervik (1985) in tissue homogenate.

**CAT activity:** Catalase enzyme activity was estimated by the standard method of Aebi (1983).

**LPO activity:** Lipid peroxidation (LPO) activity was estimated by the method of Stocks and Dormandy (1971).

**GSH activity:** GSH activity was estimated by standard method of Jollow et al (1974).

**Protein assay:** In the estimations of all the antioxidant enzymes the total soluble protein content was estimated by Lowry et al (1991) taking BSA as standard.

**Histological studies:** Kidney and spleen of rats were cleared and fixed in 10% formaline for 24 hours. Then the tissues were dehydrated in different grades of ethanol, clearing was done in xylene and embedding was done in paraffin wax for the preparation of blocks. The 5-7µm thick sections were cut and stained in haematoxylin-eosin stain and mounted in DPX.

**Statistical analysis:** Statistical analysis software (SPSS) was used to analyse the data.

## RESULTS AND DISCUSSION

There were two important considerations for the planning of this experiment. Firstly, rats were exposed to $SO_2$ for a regular period (5 h/day for 7 days with 19 hours between exposures) with relief periods in between the exposure. Secondly, rats are nose breathers and most of inhaled gas is trapped in nasal chambers and only some amount is reaching to lungs that is why higher concentration of $SO_2$ (i.e.15 ppm) was used. Both the considerations may provide a repercussion to persons exposed to the gas in an occupational or industrial setting. $SO_2$ exposure caused decrease in SOD and CAT activities at all the three concentrations but the decrease was significant at 15 ppm as compared to control rats in both the sexes (Table 3 and 4). GSH level showed non-significant decrease at three

**Table 1.** Effect of $SO_2$ on net body weight of male and female rats

| Group | Treatment | Male rats | | Female rats | |
|---|---|---|---|---|---|
| | | Initial weight (g) | Final weight (g) | Initial weight (g) | Final weight (g) |
| I | Naturally exposed rats | 103.66± 1.85 | - | 107.66 ± 3.84 | - |
| II | Control | 118.33 ± 7.26 | 110.33 ± 6.38 | 115.00± 2.88 | 106.66 ±1.66 |
| III | 5 ppm $SO_2$ | 111.66± 9.27 | 106.66± 9.27 | 121.66 ± 1.66 | 116.00 ± 3.05 |
| IV | 10 ppm $SO_2$ | 105.66± 3.48 | 100.00 ± 3.78 | 114.00 ± 3.05 | 109.33 ± 1.76 |
| V | 15 ppm $SO_2$ | 105.33± 1.76 | 102.33 ± 1.45 | 125.66 ± 6.98 | 120.00 ± 5.77 |

Values are shown as mean±SE

**Table 2.** Effect of $SO_2$ on weights (g/100g b.w.) of kidney and spleen in male and female rats

| Group | Treatment | Male rats | | Female rats | |
|---|---|---|---|---|---|
| | | Initial weight (g) | Final weight (g) | Initial weight (g) | Final weight (g) |
| I | Naturally exposed rats | $0.40 \pm 0.01^{b}$ | $0.27 \pm .01^{a}$ | $0.42 \pm 0.005^{ab}$ | $0.30 \pm 0.006^{ab}$ |
| II | Control | $0.38 \pm 0.005^{ab}$ | $0.29 \pm 0.01^{a}$ | $0.43 \pm 0.01^{b}$ | $0.29 \pm 0.01^{ab}$ |
| III | 5 ppm $SO_2$ | $0.37 \pm 0.01^{ab}$ | $0.24 \pm 0.02^{a}$ | $0.43 \pm 0.02^{a}$ | 0.36 ± 0.02 |
| IV | 10 ppm $SO_2$ | $0.36 \pm 0.01^{a}$ | $0.24 \pm 0.02^{a}$ | 0.36 ± 0.01 | $0.24 \pm 0.02^{a}$ |
| V | 15 ppm $SO_2$ | $0.36 \pm 0.01^{a}$ | $0.24 \pm 0.02^{a}$ | $0.37 \pm 0.01^{ab}$ | $0.24 \pm 0.02^{a}$ |

Values are shown as mean±SE
$^{ab}$ represents significant difference between treatments at p≤0.05 as compared to control

concentrations of $SO_2$ and significant decrease at 15 ppm. GST, GPx and GR activities showed non-significant decrease at all the three concentrations when compared with control rats. LPO level was increased at all the three concentrations in the kidney and spleen of rats of both the sexes (Table 5, 6) compared with control rats. But the increase in LPO level was statistically significant at 15 ppm and it was more significant in spleen of male rats as compared to female rats.

Superoxide dismutase (SOD) helps in protecting the cells from molecular oxygen and also removes superoxide radicals and decreased SOD level in brain and lungs may lead to free radical damage at large scale because SOD is the first security against toxicity of molecular oxygen.The decrease in the level of GST, GSH and GPx at higher concentration of $SO_2$ was found to be dose dependent. A significant increase in Lipid peroxidation (LPO) activity was observed at 15 ppm concentration in brain and lungs from rats of both the sexes as compared to control rats. Higher $SO_2$ concentration decreased the activities of SOD, CAT, GST, GPx, GSH and increased lipid peroxidation indicating the increased oxidative damage and stress in cells and tissues in

**Table 3.** Effect of $SO_2$ on antioxidant parameters of kidney of male rats

| Group | Treatment | Antioxidant parameters | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Superoxide dismutase (SOD) | Catalase (CAT) | Glutathione-S-transferase (GST) | Glutathione Peroxidase (GPx) | Glutathione (GSH) | Glutathione reductase (GR) | Lipid peroxidation (LPO) |
| I | Naturally exposed rats | 12.66 ± 0.01$^{d}$ | 11.90 ± 0.14$^{c}$ | 0.40 ± 0.004$^{b}$ | 0.82 ± 0.03$^{b}$ | 46.60 ± 0.39$^{d}$ | 0.06 ± 0.003$^{c}$ | 0.47 ± 0.01$^{a}$ |
| II | Control | 13.72 ± 0.01$^{d}$ | 12.63 ± 0.06$^{d}$ | 0.41 ± 0.009$^{b}$ | 0.85 ± 0.009$^{b}$ | 48.75 ± 0.13$^{d}$ | 0.06 ± 0.001$^{c}$ | 0.46 ± 0.01$^{a}$ |
| III | 5ppm $SO_2$ | 9.45 ± 0.39$^{b}$ | 11.38 ± 0.19$^{c}$ | 0.34 ± 0.01$^{a}$ | 0.79 ± 0.007$^{b}$ | 41.49 ± 0.14$^{b}$ | 0.04 ± 0.002$^{b}$ | 0.76 ± 0.01$^{c}$ |
| IV | 10ppm $SO_2$ | 10.96 ± 0.02$^{c}$ | 10.16 ± 0.14$^{b}$ | 0.33 ± 0.01$^{a}$ | 0.81 ± 0.007$^{b}$ | 39.27 ± 0.22$^{a}$ | 0.05 ± 0.0007$^{b}$ | 0.52 ± 0.008$^{a}$ |
| V | 15ppm $SO_2$ | 9.66 ± 0.05$^{b}$ | 8.51 ± 0.19$^{a}$ | 0.33 ± 0.015$^{a}$ | 0.60 ± 0.04$^{a}$ | 40.79 ± 0.12$^{b}$ | 0.05 ± 0.001$^{b}$ | 0.61 ± 0.02$^{b}$ |

Values expressed as mean±SE
$^{abcd}$ represents significant difference between treatments at p≤0.05 as compared to control
Units: SOD (U/mg protein), CAT (µmole of $H_2O_2$decomposed/min/mg protein), GPx(U/mg protein), GST (µmoles of GSH-CDNB conjugate formed/min/mg protein) GR (µmoles of NADPH oxidized/min/mg protein), Lipid peroxidation (nmol MDA/100 mg tissue)

**Table 4.** Effect of $SO_2$ on antioxidant parameters of kidney of female rats

| Group | Treatment | Antioxidant parameters | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Superoxide dismutase (SOD) | Catalase (CAT) | Glutathione-S-transferase (GST) | Glutathione Peroxidase (GPx) | Glutathione (GSH) | Glutathione reductase (GR) | Lipid peroxidation (LPO) |
| I | Naturally exposed rats | 10.83 ± 0.07$^{d}$ | 11.57 ± 0.04$^{a}$ | 0.51 ± 0.04$^{a}$ | 0.71 ± 0.01$^{d}$ | 46.01 ± 0.12$^{c}$ | 0.06 ± 0.008$^{c}$ | 0.46 ± 0.01$^{a}$ |
| II | Control | 11.73 ± 0.07$^{d}$ | 12.86 ± 0.03$^{a}$ | 0.54 ± 0.06$^{a}$ | 0.74 ± 0.016 $^{d}$ | 48.47 ± 0.12$^{c}$ | 0.066± 0.0008$^{c}$ | 0.47 ± 0.011$^{a}$ |
| III | 5ppm $SO_2$ | 6.08 ± 0.15$^{a}$ | 10.95 ± 0.31$^{b}$ | 0.58 ± 0.04$^{a}$ | 0.66 ± 0.02$^{b}$ | 41.33 ± 0.40$^{b}$ | 0.061± 0.003$^{bc}$ | 0.93 ± 0.01$^{c}$ |
| IV | 10ppm $SO_2$ | 10.67 ± 0.04$^{c}$ | 11.40 ± 0.28$^{b}$ | 0.44 ± 0.001$^{ab}$ | 0.68 ± 0.02$^{bc}$ | 41.76 ± 0.30$^{b}$ | 0.05 ± 0.001$^{b}$ | 0.52 ± 0.008$^{a}$ |
| V | 15ppm $SO_2$ | 7.39 ± 0.23$^{b}$ | 10.68 ± 0.07$^{b}$ | 0.44 ± 0.14$^{a}$ | 0.76 ± 0.016$^{d}$ | 41.45 ± 0.92$^{b}$ | 0.05 ± 0.001$^{b}$ | 0.95 ± 0.01$^{c}$ |

See Table 3 for details

**Table 5.** Effect of $SO_2$ on antioxidant parameters of spleen of male rats

| Group | Treatment | Antioxidant parameters | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Superoxide dismutase (SOD) | Catalase (CAT) | Glutathione-S-transferase (GST) | Glutathione Peroxidase (GPx) | Glutathione (GSH) | Glutathione reductase (GR) | Lipid peroxidation (LPO) |
| I | Naturally exposed rats | 12.83 ± 0.27$^{c}$ | 9.57 ± 0.20$^{d}$ | 0.51 ± 0.03$^{b}$ | 0.85 ± 0.005$^{c}$ | 46.01 ± 0.17$^{c}$ | 0.05 ± 0.001$^{c}$ | 0.45 ± 0.01$^{a}$ |
| II | Control | 13.03 ± 0.19$^{c}$ | 9.99 ± 0.20$^{d}$ | 0.50 ± 0.001$^{b}$ | 0.87 ± 0.005$^{c}$ | 48.12 ± 0.18$^{c}$ | 0.06 ± 0.001$^{c}$ | 0.48 ± 0.019$^{a}$ |
| III | 5ppm $SO_2$ | 7.82 ± 1.30$^{a}$ | 8.48 ± 0.16$^{c}$ | 0.42 ± 0.001$^{b}$ | 0.82 ± 0.01$^{bc}$ | 42.33 ± 0.20$^{b}$ | 0.054 ± 0.001$^{b}$ | 0.73 ± 0.01$^{bc}$ |
| IV | 10ppm $SO_2$ | 9.71 ± 0.05$^{b}$ | 6.36 ± 0.12$^{a}$ | 0.43 ± 0.003$^{b}$ | 0.84 ± 0.007$^{bc}$ | 42.68 ± 0.38$^{b}$ | 0.052 ± 0.0006$^{b}$ | 0.52 ± 0.01$^{a}$ |
| V | 15ppm $SO_2$ | 7.82 ± 1.30$^{a}$ | 7.56 ± 0.12$^{b}$ | 0.45 ± 0.002$^{b}$ | 0.81 ± 0.007$^{b}$ | 42.00 ± 0.44$^{b}$ | 0.053 ± 0.0007$^{b}$ | 0.75 ± 0.01$^{c}$ |

See Table 3 for details

present case. Increased lipid peroxidation seems to be indicator of several disorders in cells, tissues or organs and it might be involved in different diseased state of cell such as aging, nervous disorders etc (Meng et al 2002a, Meng et al 2002b). Increased lipid peroxidation may have harmful effects on composition and function of biological membranes. $SO_2$ toxicity may involve oxidative stress to cells and tissues because of the production of free radicals during oxidation process (Meng 2003).

Histologically, kidney section of control and naturally exposed rats displayed normal bowman's capsule, proximal convoluted tubule and distal convoluted tubule while the 5 ppm $SO_2$ exposed group showed enlarged parietal layer of glomerulus and degenerated tubules and 10 ppm $SO_2$ exposed group showed blood congestion, degenerated tubules and degenerated glomerulus, 15 ppm $SO_2$ exposed group showed distorted bowman's capsule, degenerated tubules and collecting duct as compared to control rats (Fig. 1). Spleen tissue section of control rats and naturally exposed rats displayed normal morphologies, the boundary of the red pulp and white pulp is clear, the central artery is obvious, the trabeculae is clear, the white pulp lymphocytes and the macrophages are abundant. 5 ppm $SO_2$ exposed group showed white pulp hyperplasia and red pulp congestion and 10 ppm exposed group showed red pulp area having splenic cord hyperplasia and lymphatic nodules multiplied and 15 ppm $SO_2$ exposed group showed number of lymphocytes and macrophages decreased significantly compared with the control group (Fig. 2) (Gao et al 2018). Decreased antioxidant enzymes influence the organs (lungs and kidney) to increase free radical damage which in turn affects the defense system of body (Bakurt et al 2004). The main target organ for $SO_2$ intoxication are the lungs as it is

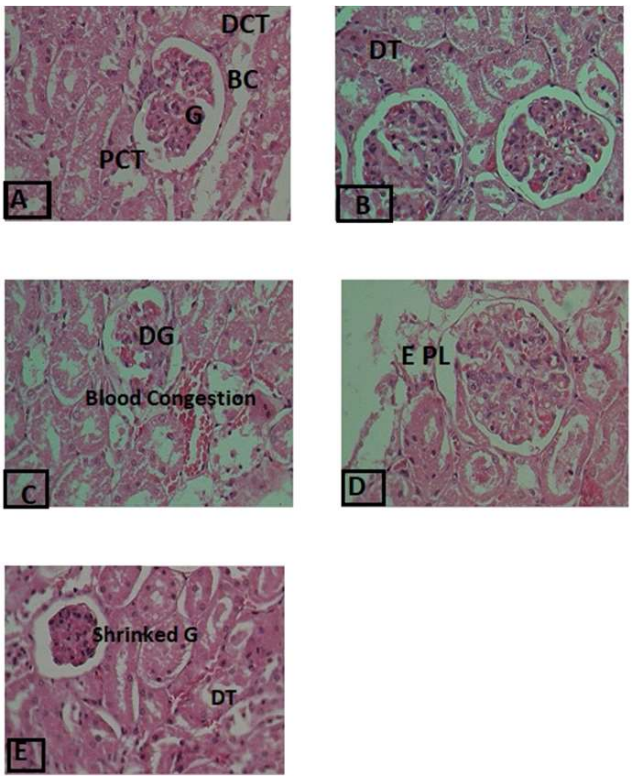


**Fig. 1.** Kidney section of control and naturally exposed rats (A&B) displayed normal Bowman's capsule (BC), Proximal Convoluted Tubule (PCT) and Distal Convoluted Tubule (DCT) (Fig 1(C) 5ppm $SO_2$ exposed group showed degenerated glomerulus (G), blood congestion and degenerated tubules(DT) Fig 1(D) exposed group showed Enlarged parietal layer(EPL) of glomerulus, blood congestion and degenerated tubules Fig 1(E) 15 ppm exposed group showed shrinked or distorted bowman's capsule, degenerated tubules and collecting duct as compared to control rats observed by light microscopy with X400 magnification

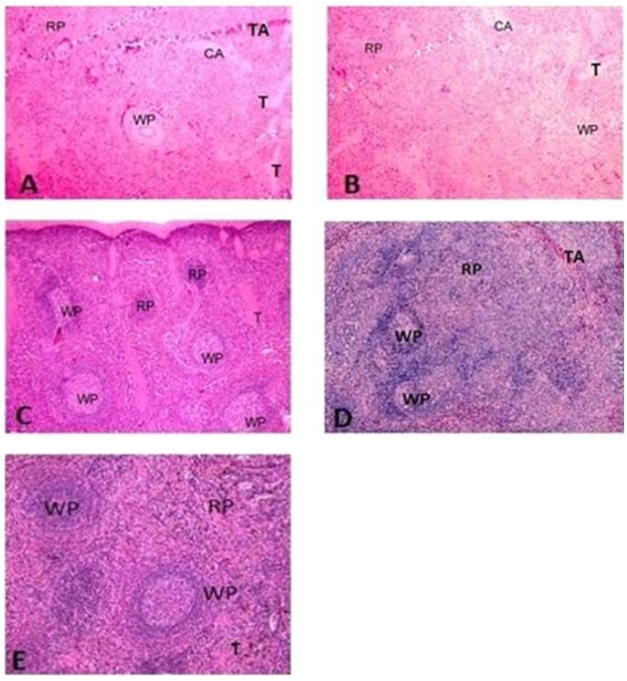


**Fig. 2.** Spleen tissue section of control group and naturally exposed rats (A&B ) displayed normal morphologies, the boundary of the red pulp (RP) and white pulp(WP) is clear, the central artery(CA) is obvious, the trabeculae (T) is clear, the white pulp lymphocytes are abundant and the macrophages are abundant. 5 ppm $SO_2$ exposed group (C) showed white pulp hyperplasia and red pulp congestion and 10 ppm exposed group (D) showed red pulp area having splenic cord hyperplasia and lymphatic nodules multiplied and 15 ppm $SO_2$ (E) exposed group showed number of lymphocytes and macrophages decreased significantly compared with the control group observed by light microscopy with X400 magnification

**Table 6.** Effect of $SO_2$ on antioxidant parameters of spleen of female rats

| Group | Treatment | Antioxidant parameters | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | Superoxide dismutase (SOD) | Catalase (CAT) | Glutathione-S-transferase (GST) | Glutathione Peroxidase (GPx) | Glutathione (GSH) | Glutathione reductase (GR) | Lipid peroxidation (LPO) |
| I | Naturally exposed rats | $13.80 \pm 0.26^{c}$ | $8.64 \pm 0.30^{c}$ | $0.51 \pm 0.003^{a}$ | $0.87 \pm 0.01^{a}$ | $47.09 \pm 0.19^{c}$ | $0.06 \pm 0.001^{c}$ | $0.49 \pm 0.02^{a}$ |
| II | Control | $14.73 \pm 0.13^{d}$ | $9.97 \pm 0.20^{d}$ | $0.50 \pm 0.001^{a}$ | $0.88 \pm 0.007^{b}$ | $48.73 \pm 0.13^{c}$ | $0.06 \pm 0.001^{c}$ | $0.48 \pm 0.03^{a}$ |
| III | 5ppm $SO_2$ | $8.85 \pm 0.67^{b}$ | $8.59 \pm 0.12^{c}$ | $0.69 \pm 0.08^{c}$ | $0.843 \pm 0.01^{a}$ | $40.86 \pm 0.14^{b}$ | $0.04 \pm 0.002^{b}$ | $0.92 \pm 0.004^{c}$ |
| IV | 10ppm $SO_2$ | $11.81 \pm 0.01^{c}$ | $7.90 \pm 0.23^{bc}$ | $0.48 \pm 0.008^{ab}$ | $0.841 \pm 0.006^{a}$ | $42.89 \pm 0.26^{c}$ | $0.05 \pm 0.001^{bc}$ | $0.56 \pm 0.02^{a}$ |
| V | 15ppm $SO_2$ | $8.85 \pm 0.67^{b}$ | $7.61 \pm 0.12^{b}$ | $0.45 \pm 0.01^{ab}$ | $0.83 \pm 0.01^{a}$ | $41.56 \pm 0.13^{b}$ | $0.05 \pm 0.001^{c}$ | $0.94 \pm 0.009^{c}$ |

See Table 3 for details

shown by oxidative damage and histopathological alterations in lungs., Besides lungs it also effects other organs like brain, kidneys, spleen and reproductive organs.

## CONCLUSIONS

The $SO_2$ inhalation have toxic effects in brain and lungs of rats of both the sexes. Brain and lungs were affected in terms of $SO_2$ induced oxidative stress by elevation of LPO and diminution of antioxidant enzymes markers in rats of both the sexes in dose dependent manner.

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