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SHORT COMMUNICATION

Cadmium (Cd) and Chlorpyrifos (CPF) Induced Pulmonary Toxicity in *Wistar* Rats

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ABSTRACT

The present study was aimed to know the pulmonary toxicity by individual toxicities of cadmium, chlorpyrifos and their combination in *albino wistar* rats. The experiment was carried out for 28 days. Group 1 - Control. Group 2 - Cadmium chloride (Cd) @ 22.5 mg/ kg b.wt /per oral / day. Group 3 - Chlorpyrifos (CPF) @ 25 mg/ kg b.wt /per oral / day. Group 4 - Cadmium chloride (Cd) @22.5 mg + Chlorpyrifos (CPF) @ 25 mg/ kg b.wt /per oral / day. Lungs showed mild to moderate congestion in groups 2 and 3 and moderate to severe in group 4 on 15th and 29th day of the experiment. Lung sections of control rats showed normal architecture. Lung sections of group 2 rats on 15th day showed hemorrhages in the interstitium spaces with infiltration of lymphocytes, On 29th day, mild hyperplasia and desquamated bronchial epithelial cells, peri bronchial and peri vascular lymphoid aggregates were noticed. The sections of lung on 15th day of group 3 rats showed exudate and desquamated epithelial cells in the lumen of secondary bronchiole , on 29th day, emphysematous alveoli with loss of architecture of alveolar epithelium, interstitial edema with infiltration of lymphocytes, mild hyperplasia of bronchial epithelial cells were also noticed. In group 4 rats, similar lesions as described in groups 2 and 3 were observed with severe intensity on 15th and on 29th day of the experiment. In combined toxicity group, the severity of lesions were more thus suggesting synergistic effects of these components.

Keywords: Pulmonary toxicity, cadmium, chlorpyrifos, *Wistar* rats

Cadmium (Cd) and Chlorpyrifos (CPF) are the most common toxicants among all toxic compounds in the environment. The Cd common sources of environmental contamination are industrial, mining activities, plastic stabilizers and batteries which may result in widespread into environment and agricultural fields (Cheng *et al.,* 2011). The OP insecticides are extensively used for control of insects in home and agricultural practices. Chlorpyrifos (CPF) is one of the most commonly used organophosphate pesticides in domestic and agricultural applications throughout the world (Asperlin, 1994). Cd and CPF intoxication may occur directly through drinking water, indirectly through irrigation water source and through feed ingredients of plant origin and also through inhalation of polluted air. Since the population tend to receive combination of multiple intoxicants through environment contamination, there is need for conducting induced toxicopathological studies to assess the impact of individual and combined environmental pollutants (Yuan *et al.*, 2014 and Yadala *et al.,* 2019). Cd induces oxidative stress and apoptosis (Henson *et al.,* 2004), CPF causes deleterious effects through acetylcholinesterase inhibition at synapse of central and peripheral nervous system (Gordon *et al.,* 1997), thereby causing damage to various vital organs. Along with the other organs, Lungs are susceptible for environmental pollutants (Abeer *et al.,* 2010 and Curcic *et al.,* 2012).

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The present study was focussed on pulmonary toxicity induced by Cd, CPF and their combination in rats.

Male *Wistar* albino rats (48) were procured from Sanzyme Laboratories Ltd., Hyderabad, animals were divided into 4 groups, 12 animals in each group. Rats were randomly divided into 4 groups consisting of 12 in each group. Group 1 serves as control. Group 2 rats were administered Cadmium chloride ($CdCl_2$) @ 22.5mg/ kg b.wt /per oral / day. Group 3 rats were administered Chlorpyrifos (CPF) @ 25 mg/ kg b.wt /per oral / day. Group 4 rats were administered Cadmium chloride (Cd) @22.5 mg + Chlorpyrifos (CPF) @ 25 mg/ kg b.wt /per oral / day for 28 days of experiment.

Detailed necropsy was conducted on 15th and 29th day of the experiment and gross changes were noticed, if any. Pieces of lung were collected in 10 % neutral buffer formalin (NBF). Samples were processed, sectioned (5µm), stained with Hematoxylin and Eosin (H&E) as per the standard protocol given (Luna, 1968).

Lungs showed mild to moderate congestion in groups 2 and 3 and moderate to severe in group 4 on 15th and 29th day of the experiment. Lung sections of control (Group 1) rats showed normal architecture. Lung sections of group 2 rats on 15th day showed hemorrhages in the interstitium spaces with infiltration of lymphocytes (Fig.1A), On 29th day, mild hyperplasia and desquamated bronchial epithelial cells, peri bronchial and peri vascular lymphoid aggregates were noticed (Fig. 1B). The present changes in the lungs might be due to Cd induced lipid peroxidation and oxidative stress. Similar lesions were reported by Newairy *et al.* (2007) and Verónica Souza Arroyo *et al.* (2012) in $CdCl_2$ toxicity. The sections of lung on 15th day of group 3 rats showed exudate and desquamated epithelial cells in the lumen of secondary bronchiole (Fig. 1C). On 29th day, emphysematous alveoli with loss of architecture of alveolar epithelium, interstitial edema with infiltration of lymphocytes, mild hyperplasia of bronchial epithelial cells (Fig. 1D) were also noticed. Pulmonary toxic effects of CPF are mainly attributed to induction of oxidative stress and suppression of antioxidant enzyme activities. Similar changes are reported by Khan and Kour (2007) and Abbas Jafari *et al.* (2014). In group 4 rats, similar lesions as described in groups 2 and 3 were observed with severe intensity (Fig. 1E and 1F) on 15th and on 29th day of the experiment. In combined toxicity group, the severity of lesions were more thus suggesting synergistic effects of these components.

In conclusion, the adverse effects of combined $CdCl_2$ and CPF group (Group 4) were more severe than the individual groups (Group 2 & 3) due to synergistic action of the combined pollutants.

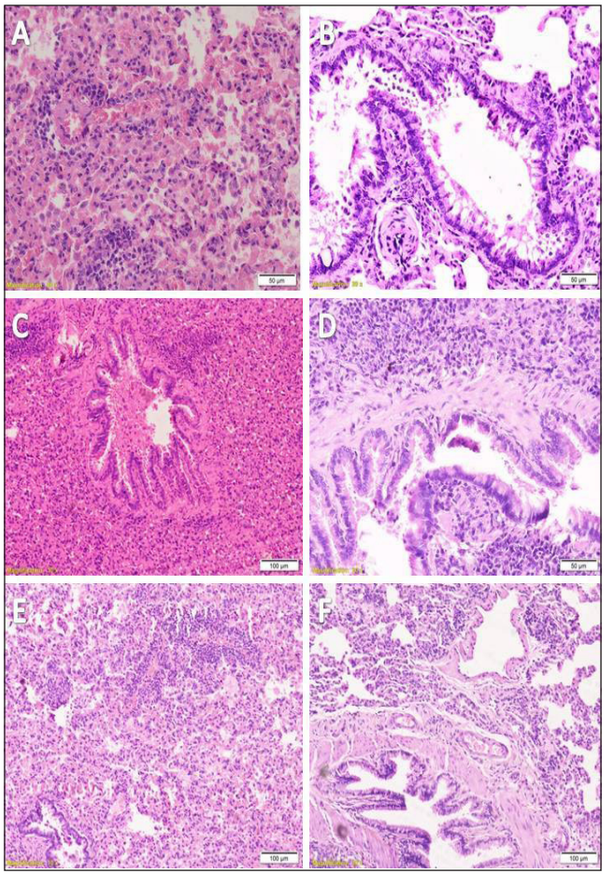

Fig. 1: Photomicrograph of lung showing **(A)** Hemorrhages in the interstitial spaces with infiltration of lymphocytes (Group 2, Day 15): H&E×200; **(B)** Mild hyperplasia of bronchiolar epithelial cells, peri bronchial emphysema, destruction bronchiolar epithelium with infiltration of lymphocytes in peribronchiol area and interstitium (Group 2, Day 29): H&E×200; **(C)** Exudate in bronchiole (Group 3, Day 15): H&E×100; **(D)** Hyperplasia of bronchial epithelial cells, mild to moderate peri-bronchial fibrosis with infiltration of lymphocytes (Group 3, Day 29): H&E×200; **(E)** Congestion of blood vessel, peri vascular infiltration of lymphocytes (Group 4, Day 15): H&E×100; **(F)** peri bronchial fibrosis with mild hyperplasia of bronchial epithelium, peri bronchial emphysema and congestion of peri bronchial blood vessels (Group 4, Day 29): H&E×200.

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