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Histological alterations in arsenic induced various organs in rats

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Abstract
In the present study arsenic-induced histological alterations in rats studded. The Wistar rats were divided into 3 groups and treated as follows: Group 1: Control, 2: mild arsenic treated (sodium arsenite @ 5 mg/kg b. wt orally for 4 wks), 3: arsenic treated (sodium arsenite @ 10 mg/kg b. wt orally for 4 wks). Group 3 (arsenic treated) showed marked degenerative changes in heart, kidney, liver, testis, lung, intestine and stomach. Group 1 and Group 2 did not reveal any abnormalities on histopathology.

Keywords: Arsenic, histopathology, rat

Introduction
Environmental toxins (such as pesticides, solvents, and air pollutants) are associated with an increased prevalence of allergic diseases. Allergic diseases are the most common chronic inflammatory diseases in human and animals also represent a substantial morbidity and mortality burden in severe cases. The prevalence of allergic disorders has seen a worldwide increase over the last few decades. Most countries report population rates of allergic diseases between 15%-35%, with the worldwide average being 22% in animals.

Environmental toxicants contribute to the increasing prevalence of allergies and also affect various diseases. Arsenic is widely distributed in nature and principally occurs in the form of inorganic or organic compounds. An inorganic arsenical compound consists of arsenite, considered to be the most toxic form, arsenate the less toxic form, and organic forms the least toxic ones [1]. As a result of wide occurrence of arsenic in the environment, exposure to the metalloid becomes almost universal. The most common pathway for an elevated environmental exposure to inorganic arsenic worldwide is through drinking water. Chronic exposure to arsenic causes a wide range of toxic effects.

After ingestion, inorganic arsenic appears rapidly in the circulation, where it binds primarily to hemoglobin [2]. Skin, bone and muscle represent the major storage organs. Inorganic arsenic does not appear to cross the blood brain barrier, however transplacental transfer of arsenic in human and mice occurs. The metabolism of arsenic like other toxic metals is associated with the conversion of the most potent toxic form of this element to the less toxic form, followed by cellular accumulation or excretion. Biomethylation of arsenic is considered the primary detoxification mechanism, since the inorganic arsenics are more toxic to the living organisms [3]. The conversion of arsenic between oxidation states and organo-metalloid forms alter the binding affinities of arsenic for different proteins, thus altering the relative toxicities of the various arsenic species. To find out arsenic effect the present study was taken up for evaluation of arsenic-induced histological changes in various tissues.

Materials and Methods
Male Wistar rats weighing about 200-220 g were procured from department of veterinary pathology, Jaipur, India. The animals were housed in solid bottom polypropylene cages with 12 h-12 h light-dark cycle. Animals were placed on commercial standard pellet feed for rats and provided water *ad libitum.* Rats were divided into three groups of 6 rats in each with Group 1: Control, 2: mild arsenic treated (sodium arsenite @ 5 mg/kg b.wt orally for 4 weeks), 3: arsenic treated (sodium arsenite @ 10 mg/kg b. wt orally for 4 weeks). At the end of 28th day, animals were euthanized and heart, kidney, liver, testes, lungs, stomach and intestines

were collected in 10% buffered formalin for histopathology.

Results

The sections of heart showed haemorrhages in myocardium, and degeneration and separation of muscle bundles in group 3, when compared to group 2, and group 1 did not show any lesions of pathological significance. The sections of kidney in group 3 revealed moderate to severe fatty change, mononuclear cell infiltration in the tubules and reduced glomerular space. Sections from group 2 revealed very mild degenerative changes in tubules while group 1 did not show any significant lesions of pathological importance. The sections of liver in group 3 showed congestion of central vein with moderate degeneration and necrosis in hepatic parenchyma with mild to moderate fatty change. Groups 2 showed mild congestion of central vein and mild fatty change in hepatocytes, while group 1 did not reveal any lesions of pathological significance.

The sections of testis showed ruptured follicles and few follicles showing reduction in the number of spermatozoa and degenerated inter follicular septa in group 3. The sections of group 2 showed mild degenerative changes, while the rats belonging to group 1 did not show any lesions of pathological significance. The sections of lung showed congestion, emphysematous and ruptured alveoli in group 3. Groups 2 showed congestion and haemorrhage, few ruptured alveoli with infiltration of mononuclear cells, while group 1 did not show any lesions of pathological significance.

Discussion

In the present study enhanced oxidative stress in cardiac tissue due to arsenic exposure is the major factor for arsenic-induced cardiotoxicity [4]. The degenerative changes in the kidney tubular epithelium may be attributed to arsenic-induced lipid peroxidation as kidney is one organ that is rich in phospholipids and leads to the oxidative degradation of phospholipids; kidney is prone to oxidative damage leading to functional deterioration. In short-term studies (1-10 weeks), it was recently found that treatment of mice with high concentrations of arsenic administered as sodium arsenite in the diet induced hyperplasia in the bladder epithelium [5].

The mode of action of inorganic arsenic in rats and mice appears to involve urothelial cytotoxicity, increased cell proliferation and ultimately tumours. Cytotoxicity is likely due to the generation of reactive trivalent arsenicals excreted in the urine [6]. Chronic arsenic exposure and associated with methyl insufficiency and loss of DNA methylation in animals [7], may be reason for the histological changes.

The loss of germinal epithelium may be due to the oxidative stress. The results of present study are in agreement with Pant *et al.* [8]. Arsenic exposure in experimental rats has shown to produce steroidogenic dysfunction leading to impairment of spermatogenesis [9]. Arsenic causes genotoxicity in testicular tissue of mice [10] and recent study suggests that arsenic causes testicular toxicity probably by affecting the pituitary testicular axis [11]. Arsenic has a suppressive influence on spermatogenesis, and release of gonadotrophin and testosterone in rats [9]. More importantly, *in vivo* study with Syrian golden hamsters demonstrated that arsenic and cigarette smoke synergistically increased oxidative stress in lungs [12].

Conclusion

In the present study revealed that histological alterations in heart, kidney, liver, lung and testis are attributed to the induction of reactive oxygen species, depletion of antioxidant defences and eventual oxidative stress.

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