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***Research Article***

# HISTOLOGICAL CHANGES IN LIVER OF ALBINO MICE DUE TO CHRONIC ADMINISTRATION OF ACRYLAMIDE

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## ABSTRACT
Adult Mice (B.Wt. 30 gms were treated at a dose of 10 mg \kg BWt. of acrylamide for 10 days in drinking water. Control animals were given equal dose of deionised water. Qualitative changes were studied in Liver. Histopathological changes in liver lobules, hepatocytes etc. were evaluated. Significant changes were noticed**.** These changes were characterized by disorganization of lobules. Besides this the necrotic changes were also noticed. There was a significant decrease in the number of hepatocytes also. Body weight of the animal was slightly altered after challenge with acrylamide.

***Key Words****: Acrylamide, Toxicity, Liver, Histopathology, Albino Mice*

## INTRODUCTION
Acrylamide is one of the most important contaminant in the environment, which was shown to be neurotoxicity, reproductive toxicant and carcinogenic in animals (Johnson, *et al.,* (1986). Acrylamide (AA) is an important industrial chemical that is neurotoxic, mutagenic to somatic and germ cells, and carcinogenic in chronic rodent bioassays. Recent findings of AA in many common starchy foods have sparked renewed interest in determining toxic mechanisms and in understanding the cancer, neurotoxicity, and reproductive risks from typical human exposures (Doerge *et al.,* 2005).
There was no visual observation of neurotoxicity in any study animal but sciatic nerve degeneration was observed in the male and female high-dose groups. Increased mortality related to acrylamide was observed in the high-dose male and females during the month of treatment (Friedman *et al.,* 1995). Males and females in the high-dose groups as well as females of the low-dose group had significantly ($p< 0.001$) increased thyroid follicular cell adenomas and adenocarcinomas. A variety of other tumor types observed with increased incidence in a previous acrylamide oncogenicity study (i.e., combined CNS glial neoplasms, papillomas of the oral cavity, adenomas of the clitoral gland, and uterine adenocarcinomas) were not observed to be present at increased incidence in this study. This study confirms previously described acrylamide induction of benign tumors of the thyroid and mammary glands as well as mesotheliomas of the testis. By using a larger number of animals with an unbalanced study design, this study showed that acrylamide did not induce glial tumors and demonstrated that the no-observable-effect level for scrotal mesotheliomas is 0.5 mg/kg. Based on neuropathic signs and symptoms and quantifiable indicators of peripheral nervous dysfunction, such as vibration thresholds and electroneuromyography measurements, a neurotoxicity index (NIn) specific for acrylamide-induced peripheral neuropathy was designed (Calleman *et al.,* 1994).
The fact that acrylamide, a proven rodent carcinogen, is present in significant quantities (up to several mg/kg of foodstuff) in a wide range of commonly consumed human foods is alarming. In a review of research on the known and hypothetical modes of action of acrylamide of relevance for carcinogenesis, both genotoxic and non-genotoxic modes of action of acrylamide are discussed with special emphasis on DNA-adduct targeted mutagenesis. Mechanistic data are presented from various experimental systems including in vitro experiments and in vivo rodent and human studies with special focus on mouse models. Updated human exposure data, estimates of daily intake of dietary acrylamide in different populations, and the corresponding cancer risk assessments are provided. The significant gaps in knowledge, which currently preclude a more definitive evaluation of human cancer risk due to exposure to dietary

acrylamide are highlighted. Future directions for research on acrylamide and cancer are outlined, and potential challenges are underscored. (Besaratinia *et al.,* 2007)

In 1925-2002 study period, (Marsh *et al.,* 2007) observed both deficit and excess overall mortality risks among the U.S. cohort for cancer sites implicated in experimental animal studies: brain and other central nervous system, thyroid gland, testis and other male genital organs and for sites selected in earlier exploratory analyses of this cohort: respiratory system cancer, oesophagus, rectum, pancreas, and kidney. (Marsh *et al.,* 2007) found that acrylamide exposure at the levels present in their study sites was not associated with elevated cancer mortality risks.

Acrylamide is also a common chemical which is used in both industrial and laboratory processes. It is formed in heated starchy foods especially potato products. The aim of the present study was to clarify the possible involvement of acrylamide induced hepatotoxicity. Histopathologcal investigations revealed necrotic and degenerative changes in the liver of acrylamide treated Albino mice. Acrylamide is an alpha beta unsaturated vinyl monomer of poly- acrylamide (conjugated) reacted molecule. The co-polymers and polymers of acrylamide have vast range of applications. It is used in paper manufacture, cosmetic industries, plastic, aesthetic surgeries and other industrial and laboratory processes. (Klaunig and Kamendulis, 2005; Asha *et al.,* 2008 and Schwend *et al.,* 2009). Nowadays appreciable amount of ACR are formed in western diets which extensively invade our markets, this prompted renewed interest in its potential toxicity.

## MATERIALS AND METHODS

Young Mice of B.Wt. 30 gms were used as a model in the present study they were treated at 10 mg \kg BWt. of Acrylamide for 10 days in drinking water. Control animals were given equal dose of deionised water. Total three groups of mice were set in the experiment. Each group had five mice. They were acclamitised to laboratory conditions for 15days prior to the commencement of the treatment. Rats were kept in open air cages at room temperature. Rats were fed standard rodent palate diet (Hindustan Lever Ltd). Experimental animals were given acrylamide orally through drinking water.

Animals of experimental and control group were sacrificed on tenth day of treatment by cervical dislocation. The liver of experimental and control group of mice were fixed in formalin for 4 hrs. They were dehydrated, in graded EtOH series, cleared in xylene, infilterated with and embedded in pure filtered paraffin wax (M.P.58 degree centigrade). Deparaffinised sections (5-7 microns) were stained by haematoxylin and eosin to monitor the extent of changes in the liver histoarchitechture. Every alternate section of the liver was microscopically examined and appropriate areas were microphotographed and enlarged. Lobular disintegration was also microphotographed to record the vulnarebility to acrylamide toxication. The behaviourable changes in mice were also observed.

The population of hepatocytes in each lobule was also quantitate approximately .The degenerating cells were identified on the basis of loss of shape and necrotic patches which were seen and microphotographed.

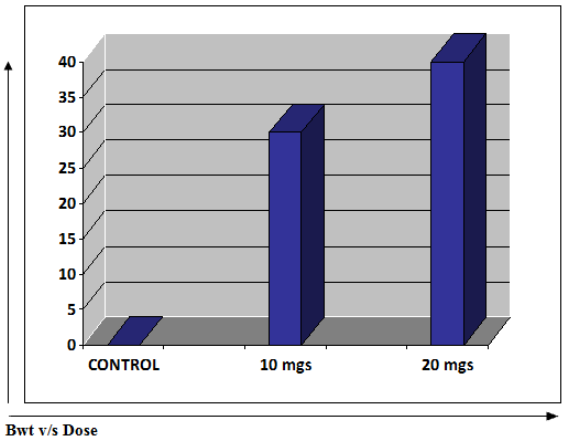
## RESULTS AND DISCUSSION

### *Histological Observations of Liver*

Histopathological changes of acrylamide treated rats of 30gms BWt with a dose of 10mgs/kg body wt showed little degenerative changes characterized by cloudy swelling or hydropic degeneration of some hepatic cells hypotrophied kupffer's cells together with dialated and congested blood vessels and hepatic sinusoids besides numerous bile ductules. Acrylamide treated rats with a body weight 30 gms, 20mgs\kgBWt showed comparatively more necrotic changes in the hepatic cells. The liver consisted of numerous lobules .Lobular cells degeneration occurred in experimental animals markedly but was not marked in control animals.

Most of the lobules reached the maturity in mice with a BWt of 30gms treated with 10mgs\KgBWt.

**Graph I: Histogram of Body Weight v/s Dosage Showing Hepatocyte Disintegration in Mice**



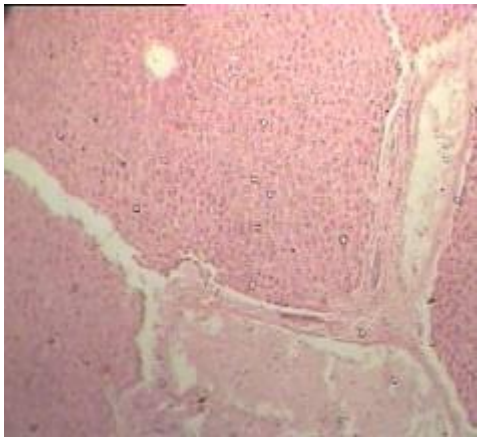



**Figure 1: Liver of albino mice (control)**

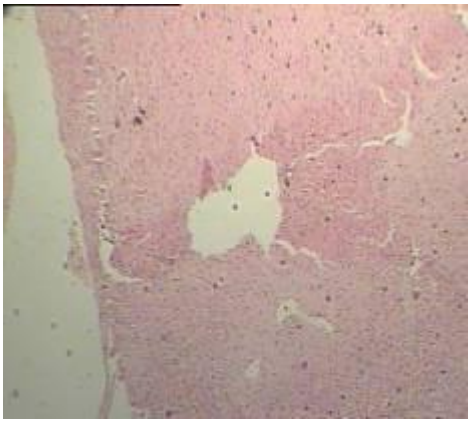


**Figure 2: Liver of 30gms mice with 10 mgs\KgBwt dose**

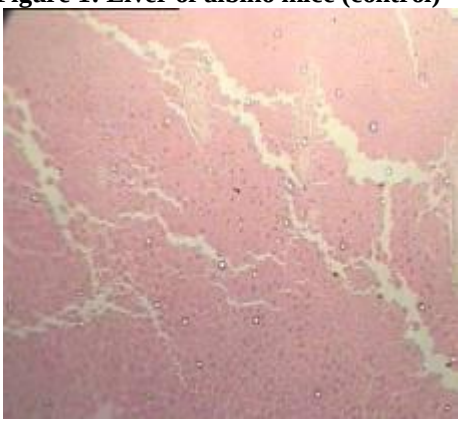


**Figure 3: Liver of 30gms mice with 20 mgs\KgBwt dose**

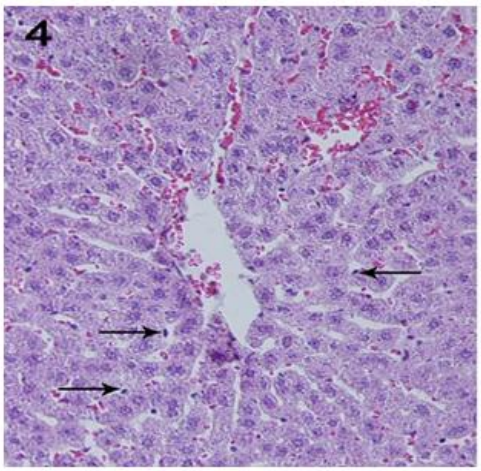



**Figure 4: Section in liver of albino mice orally administered acrylamide dose of 20mg/kg b.wt for 10 days showing degenerative changes characterized by cloudy swelling or hydropic degeneration of some hepatic cells, hypotrophied Kupffer cells (arrow).**

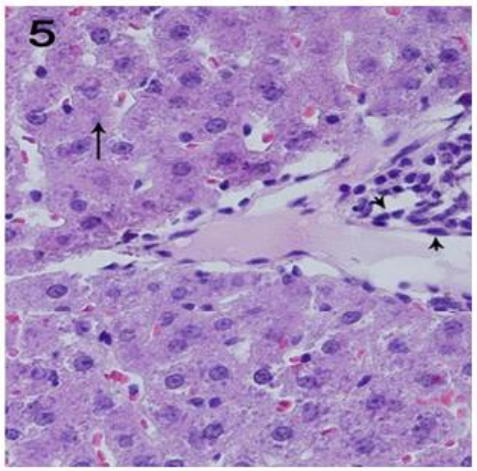



**Figure 5: Section in liver of albino mice orally administered acrylamide dose of 10 mg/kg b.wt for 10 days showing mild necrotic changes in the hepatic cells (arrow) with focal mononuclear cells infiltration (head of arrow) in portal.**

In mice with 30gms BWt treated with 20mgs\KgBwt and mice with 30gms treated with 10 mgs\kgBWt dose showed lesser and still lesser disintegration respectively. The overall shape of the liver was not

altered nor there do any significant change in organ weight as compare with control. The percentage of degenerating lobules was hence found to be still high (Graph-I) as compared with control in mice with a BWt of 30gms treated with 20mgs\KgBwt.

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