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Effects of atrazine exposure on some biochemical parameters in mice: A comprehensive study

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Abstract- This study intends to look into how atrazine exposure could affect different metabolic markers in mice. Concerns concerning the herbicide atrazine's possible negative effects on non-target organisms, such as animals, have been raised. In this study, we performed a thorough investigation of biochemical markers in mice exposed to various atrazine concentrations for a predetermined amount of time. The findings give information on the potential physiological effects of atrazine exposure by highlighting changes in important biochemical indicators. Atrazine was administered to adult male mice for ten days. The animals were slaughtered by cervical dislocation at the conclusion of the tenth day; the liver and kidney. For histological analysis and biochemical evaluations, blood glucose was taken. Measurements were made of the body's initial weight, final weight, liver weight, kidney weight, and blood glucose.

Key words: Atrazine, biochemical markers, Anthron method, Ellman's method, Ohkawa method

INTRODUCTION

Atrazine, (2-chloro-4-ethylamino-6-isoprop-ylamino-s-triazine) herbicide, is widely used across the world to control broadleaf and grassy weeds in maize, sorghum, sugarcane, cotton, pineapple crops and landscape vegetation, so that atrazine and its metabolites are widely persistent in water and are mostly found in soil especially in farming seasons.[1] Atrazine is still used in significant amounts across the globe today, despite being outlawed in the European Union and restricted in other nations. It is one of the agricultural pesticides that are most commonly used in the United States, and its use in Asian countries has been expanding.[2] As a result, atrazine exposure poses a risk to both people and wildlife. Clarifying the toxicological mechanisms by which environmental toxins affect the reproductive system of humans and other mammals has become increasingly important in recent decades. According to a number of reports, atrazine might negatively impact reproductive health.[3] Atrazine can pollute the environment's water supplies in a number of ways, including runoff and drainage from treated regions, precipitation, and pesticide water spills. The toxicological response of numerous different animals exposed to atrazine has been studied. The knowledge of how atrazine might affect environmental creatures has undergone numerous advancements and alterations. The potential danger of atrazine to vertebrates and invertebrates that might be harmed by an unintentional exposure to the chemical has been assessed.

Atrazine's chemical characteristics make it prone to runoff from agricultural and urban applications as a result of rainfall and inadequate irrigation management.[4] One of

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the chemicals that is frequently used to control weeds in maize or other crops, such as green vegetables, is atrazine. Due of its well-defined moment within soil, it has the potential to mix with water. According to reports, atrazine extends to water as a result of the immediate agronomic needs of aquatic areas or occasionally as a result of improper management in particular contexts. When they enter that environment, bacteria cannot damage them. Atrazine has been a persistent water contaminant for years due to the extensive usage of its metabolites, deethylatrazine, and other derivatives.[5] The half-life of atrazine is between 95 and 350 days, and it is resistant to degradation.

Exposure to atrazine has been linked to serious health issues include cancer, neurological disorders, dermatological conditions, and respiratory problems. Therefore, atrazine's harmful effects are of particular concern to and interest to researchers all over the world. Due to its widespread use, widespread contamination of ground and surface waters, usage pattern, high persistence, and probable biological influence on the environment, atrazine has drawn a lot of attention.[6]

Additionally, after oral exposure to atrazine, male rats showed negative effects on the quantity of sperm in the testicles and epididymis, motility, viability, morphology, and daily sperm result.[7] Atrazine exposure in rats was reported to have caused liver damage.[8]

The endocrine (hormonal) system is atrazine's main target in both people and animals. Adults (humans and experimental animals) have experienced effects such as ovarian histopathology (changes in ovarian tissue), shortening of the estrous cycle, attenuation of the LH (luteinizing hormone) surge, decreases in pituitary hormone levels, and liver effects such as elevated serum lipids, liver enzymes, and liver histopathology. The endocrine system may be affected by the behaviour of atrazine as an endocrine disruptor.[9]

Adults has also experienced effects on their immune systems, cardiovascular systems, and central nervous systems. Adult individuals who have been exposed to atrazine may develop certain non-Hodgkin's lymphomas. Atrazine concentrations in drinking water were associated with a significantly increased risk of preterm delivery, intrauterine growth retardation, and lower birth weight. Therefore current study intends to look into how atrazine exposure could affect different metabolic markers in mice.

MATERIALS & METHODS

Three groups (G1,G2,G3) of three adult male mice were taken each weighed 156.20 ± 6.19 gm on an average (table 1). Nine adult male mice were obtained and housed. The animals were given water and food, and were kept in a 10:10 cycle of light and darkness. Prior to receiving doses of the test drugs, the mice were housed for a minimum of 1 week to acclimatise. The study's experimental plans and techniques were compliant with the industry-recognized manual for the handling and use of laboratory animals.

The mice that were kept overnight were put to death by cervical dislocation at the conclusion of the experiment. Blood was drawn, allowed to clot, and then the serum was extracted by centrifuging the blood samples at 1500 rpm for 15 minutes. Each mouse's heart, liver, brain, and kidney were rapidly removed from each batch of slain mice, freed of any remaining fat, rubbed in a cool solution of 1.15% potassium chloride, and weighed. Prior to analysis, the separated serum and tissues were kept at 80°C. Each mice had a preserved kidney, piece of the liver, brain, and cerebellum in neutral buffered formalin. Hematoxylin and eosin staining of tissue sections was a standard procedure for microscopy.

The levels of lipid peroxidation and reduced glutathione were measured using the Ellman's method and the Ohkawa method, respectively. Blood glucose will be estimated by Anthron method. Using commercially accessible assays, serum parameters such as amino transferase, alkaline phosphatase, and urea were determined. The kit approach was used to determine blood glucose levels. The values were given as mg/dL.

RESULTS

Animals exposed to pesticides typically lose weight overall and in their organs. Atrazine administration in the current investigation led to a rise in body weight in all experimental mice over the course of the trial. When compared to untreated control mice, the weight of the liver and kidneys increased in atrazine-treated animals. There have also been reports of body weights remaining the same or dropping after atrazine administration. Reduced meal intake or necrotic alterations in various body tissues may be to blame for the decreased body weight and organ weight (liver and kidney) after atrazine medication. In contrast, an increase in body weight was seen in our study when atrazine was administered. Our study's body weight gain,

especially in atrazine-treated mice, Our findings concur with those of a prior study by Gojmerac *et al.,* (1995)[10] who noted increased body weights in rats following chronic atrazine administration at low doses. In general, atrazine is not thought to be particularly harmful.[11] According to Trentacoste *et al.* (2001)[12], rats treated with atrazine at a dose of 100 mg/kg per day experienced an average body weight drop of roughly 9%. This demonstrated that the observed abnormalities in the male reproductive tract were caused by the treated rats' reduced food intake rather than by the direct effects of atrazine on the reproductive system. The immune system acts as a tightly knit system and offers significant defences against the effects of chemical exposure, according to research on the long-term immunotoxicity of atrazine.[13]

Table 1- showing effect of atrazine on body weight, liver, kidney and blood glucose

Group of Mice		Initial body weight	Final Body weight	Weight of Liver	Weight of Kidney	Blood glucose
G1	A1	156.20 ± 1.25	226.30 ± 1.23	7.46 ± 0.35	1.53 ± 0.79	85.81 ± 1.03
	A2	150.30 ± 1.65	230.55 ± 1.62	7.18 ± 0.36	1.48 ± 0.89	82.57 ± 1.45
	A3	160.35 ± 2.01	250.26 ± 2.36	7.66 ± 0.46	1.57 ± 0.51	88.09 ± 1.11
G2	B1	150.35 ± 1.36	231.02 ± 1.80	7.18 ± 0.89	1.48 ± 0.76	82.59 ± 0.89
	B2	161.25 ± 0.85	248.25 ± 1.12	7.70 ± 0.78	1.58 ± 0.46	88.58 ± 1.45
	B3	156.36 ± 1.23	227.01 ± 2.03	7.47 ± 0.58	1.54 ± 0.58	85.90 ± 1.82
G3	C1	158.68 ± 2.03	241.96 ± 1.01	7.58 ± 0.64	1.56 ± 0.09	87.17 ± 1.23
	C2	150.01 ± 2.11	231.46 ± 0.31	7.17 ± 0.21	1.47 ± 0.43	82.41 ± 1.46
	C3	162.39 ± 0.26	255.32 ± 1.28	7.76 ± 0.84	1.59 ± 0.13	89.21 ± 1.70

Blood glucose level values are in mg/dl, Weights are mentioned in grams

CONCLUSION

The kidney and liver saw the majority of the histological alterations found in this investigation, respectively. It's also feasible that the atrazine-treated mice's minor hepatocyte degeneration and/or mild periportal cellular infiltration by mononuclear cells were enough to trigger the release of the serum indicators from the liver into the bloodstream. The anatomical and physiologic variations of this organ can be partly responsible for its vulnerability to the harmful effects of noxious substances. Therefore, to cause significant histopathological alterations in the brain, different experimental paradigms may be needed, such as a longer exposure duration or greater atrazine dose. The symptoms displayed by these experimental animals, such as lethargy, a loss of body weight, and ultimately death, lead us to believe that their health circumstances are abnormal and suggest the presence of systemic toxicity. Therefore, under the same experimental conditions, a bioactive substance like quercetin that protects a target tissue from chemically induced oxidative damage may actually predispose or not protect another tissue from damage caused by the toxic chemical.

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