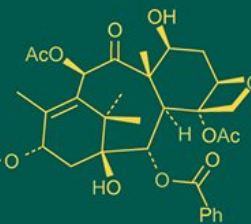
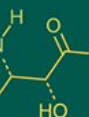
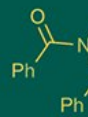
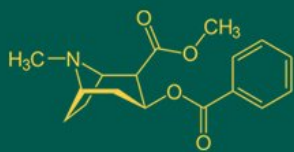


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Atrazine induced toxicity in mammals: A review

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Abstract

Atrazine is most commonly used triazine herbicides globally, mainly applied for the control of broadleaf and grassy weeds in agricultural fields. Due to its extensive use, atrazine toxicity has been frequently detected in soil, surface water and groundwater, raising concerns about its potential toxic effects on non-target organisms, including mammals. This review critically examines the current knowledge on atrazine-induced toxicity in different organ systems of mammals, with a focus on its mechanism of action, target organs and health outcomes. Experimental and epidemiological studies indicate that atrazine exposure is associated with endocrine disruption, particularly affecting the hypothalamic pituitary gonadal axis, leading to reproductive and developmental abnormalities. Additionally, atrazine has been shown to induce inflammation, oxidative stress and mitochondrial dysfunction, contributing to hepatic, renal, neurological and immunological toxicity. Alterations in hormone regulation, especially involving estrogen, testosterone and luteinizing hormone, have been widely reported in laboratory animals. Emerging evidence also suggests potential links between chronic atrazine exposure and metabolic disorders, neurobehavioral changes and increased cancer risk, although findings remain inconsistent across studies. The review further discusses dose-dependent effects, routes of exposure and species-specific responses, highlighting gaps in current risk assessment models. Overall, this review emphasizes the requisite for comprehensive chronic studies to better understand the implications of atrazine exposure on mammalian health and to inform regulatory policies. Improved monitoring and stricter control measures are essential to minimize environmental contamination and reduce potential health risks associated with atrazine.

Keywords: Atrazine, organ toxicity, inflammation, endocrine disruption

Introduction

Triazine herbicides are widely employed for their effectiveness in eradicating weeds in agricultural crops, fruits, and vegetables. Atrazine (ATZ) is a selective systemic triazine herbicide, among the most widely used triazine herbicides, with annual global consumption in the billions of tons ^[1, 2]. Atrazine (ATZ) is employed as a selective pre- and post-emergent herbicide in contradiction to broadleaf and grassy weeds for crops including coffee, corn, sorghum, wheat, sugarcane christmas tree farms and residential lawns ^[3]. It is among the most extensively utilized herbicides, with reports indicating its presence exceeding permissible limits in aquatic environments ^[4]. The principal target of ATZ in humans is the endocrine system, which may ultimately alter the body's regulatory and metabolic functions (USEPA). Pesticides and herbicides are chemical substances that are toxic and hazardous to humans and are frequently utilized in agriculture. Nonetheless, it is widely acknowledged that its utilization adversely affects human health ^[5]. The Environmental Protection Agency categorizes ATZ as class 3 toxicity on a scale of 1 to 4, with 1 being the most harmful and it is classified as an endocrine disruptor by the US Environmental Protection Agency ^[6]. Atrazine is a persistent environmental contaminant due to its widespread usage, limited biodegradability, and prolonged half-life in water (>200 days) and soil (14 to 109 days) ^[7]. Atrazine was firstly documented to affect reproduction, with possible disruptive concerns subsequently associated with the immune system, cancer, stress response, neurological disorders and cardiovascular diseases in animal models.

Atrazine is a water-soluble herbicide, enabling it to readily penetrate many aquatic environments. Moreover, careless repeated applications of atrazine led to extensive amounts of the herbicide being precipitated and entering aquatic ecosystems. A crucial aspect to evaluate while investigating the effects of atrazine on various animal and cell culture models is the challenge of determining an optimum dosage and concentration. The acute oral fatal

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dose of atrazine for laboratory rats, resulting in 50% mortality is 1900–3000 mg/kg⁸. ATZ has been shown to disrupt lipid metabolism, potentially resulting in metabolic disorders such as diabetes^{9, 10, 11}. It has been associated with the disturbance of various body systems and illness conditions including reproductive disorders, stress response, neurotoxicity, cardiovascular disease, immunotoxicity, cellular activation and cancer¹². Following absorption from the gastrointestinal tract, atrazine is predominantly metabolized in the liver by cytochrome P450 (CYP450) enzymes. Extra-hepatic metabolism is feasible due to the high expression of these enzymes in several tissues, including the kidney and placenta¹³. Atrazine's detrimental impact on reproductive parameters has consistently suggested an endocrine-disrupting mechanism. Atrazine has recently been recognized for its obesogenic effects in rodents, specifically by disrupting metabolic activity through interference with mitochondrial function.

A typical endocrine disruptor is a chemical that parodists, blocks, or interferes with the functions of the body's hormones, namely gonadal hormones like estrogens, progesterone and androgens¹⁴. Atrazine is an endocrine disruptive drug that impairs normal hormonal activities, including spermatogenesis, steroidogenesis and insulin resistance, as proven by numerous studies^[15, 10, 16]. This review summarizes the toxic effects of atrazine on major mammalian physiological systems. In addition, epidemiological investigations associating atrazine exposure

with human pathological outcomes are briefly addressed. Where available, current insights into the underlying mechanisms of action of atrazine are also discussed.

Environmental Occurrence and Mammalian Exposure

Atrazine is moderately persistent in the environment and exhibits high mobility in soil, enabling leaching into groundwater. Human exposure to atrazine predominantly occurs via drinking water, with amounts ranging from 0.01 to 5 mg/L, regardless of urban or rural residency. Mammals may be exposed to atrazine through oral, dermal and inhalation routes. Laboratory studies have commonly employed oral administration to evaluate systemic toxicity, while epidemiological studies suggest that chronic low-dose exposure is more relevant to human health risk assessment. The bioavailability and toxicity of atrazine are influenced by exposure duration, dose, metabolic capacity and species-specific physiological factors. Atrazine exhibits variable persistence in soils, with reported half-lives ranging from days to several months and undergoes degradation through microbial activity and abiotic reactions, yielding metabolites including desethylatrazine (DEA), desisopropylatrazine (DIA) and hydroxyatrazine. The United States Environmental Protection Agency (EPA) establishes the legal thresholds for water pollutants and the timetables for water testing, as stipulated in the Safe Drinking Water Act. The maximum contamination limit (MCL) for atrazine in potable water is 3 µg/L¹⁷.

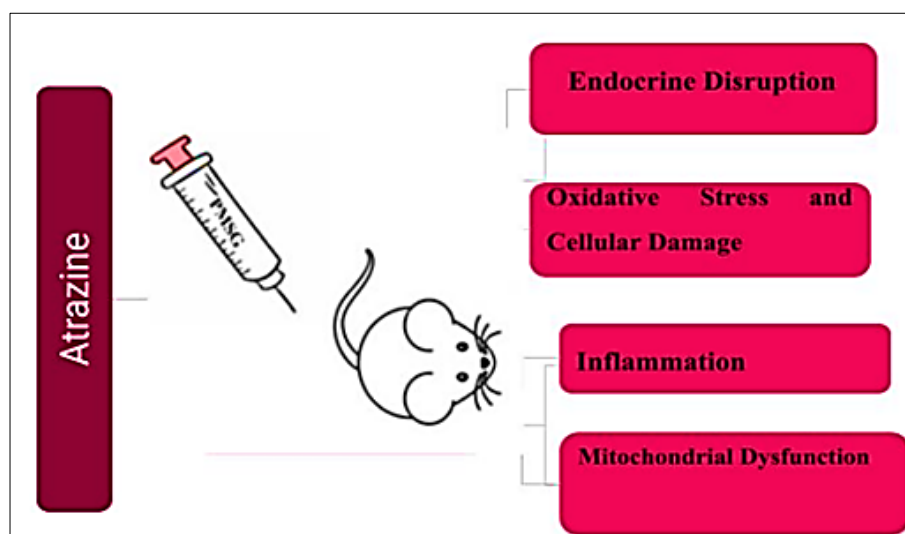


Fig 1: Mechanism of Atrazine Toxicity

Mechanisms of Atrazine Toxicity

Endocrine Disruption

One of the most extensively studied toxic effects of atrazine is its endocrine-disrupting potential. An endocrine disruptor is a drug that can imitate, obstruct or directly interfere with the functions of the body's hormones, especially gonadal hormones like estrogens, progesterone and androgens¹⁴. The hypothalamus functions as the central regulatory centre of the reproductive axis, initiating reproductive signalling through the secretion of gonadotropin-releasing hormone (GnRH). Gonadotropin-releasing hormone (GnRH) is conveyed through the hypophyseal portal circulation to the anterior pituitary, where it prompts the production and secretion of gonadotropins, primarily luteinizing hormone (LH) and follicle-stimulating hormone (FSH)¹⁸. Luteinizing hormone (LH) is essential for female ovarian

physiology, where it regulates follicular development and induces ovulation¹⁹. In males, LH stimulates testosterone synthesis and secretion by Leydig cells²⁰. In contrast, follicle-stimulating hormone (FSH) supports follicular maturation and estrogen production in females and promotes spermatogenesis in males through its actions on Sertoli cells.

Oxidative Stress and Cellular Damage

Atrazine exposure is associated with heightened generation of reactive oxygen species and diminished antioxidant defence systems. Highly reactive chemicals like reactive oxygen radicals (ROS) are produced in excess during oxidative stress, which damages living things^{21, 22}. Oxidative stress is pivotal in atrazine-induced cellular injury, leading to lipid peroxidation, protein oxidation, and

DNA damage²³. These effects are implicated in tissue injury and organ dysfunction. Pruett *et al* ²⁴ were the first researchers to document an increase in the stress hormone corticosterone following atrazine treatment. Recent studies have demonstrated that oxidative stress (OS) is associated with ATR toxicity, as evidenced by the assessment of particular biomarkers in organs including the liver, RBC's and testes in rats ^{25,26}.

Inflammation and Mitochondrial Dysfunction

Several studies report that atrazine elicits inflammatory responses by altering cytokine expression and immune signalling pathways. Atrazine is theorized to produce

immunological suppression through the before described elevation in corticosterone, but it may also have direct harmful effects on the immune system. Immunotoxicity refers to the detrimental effects of xenobiotics on the immune system, resulting in modifications to both nonspecific and acquired immunity. Immunotoxins exert both direct and indirect effects, such as thymic atrophy and splenomegaly, which influence immunocyte populations and alter cytokine concentrations and intracellular signaling pathway²⁷. In addition, atrazine-induced mitochondrial dysfunction has been observed, resulting in impaired energy metabolism and apoptosis in mammalian cells.

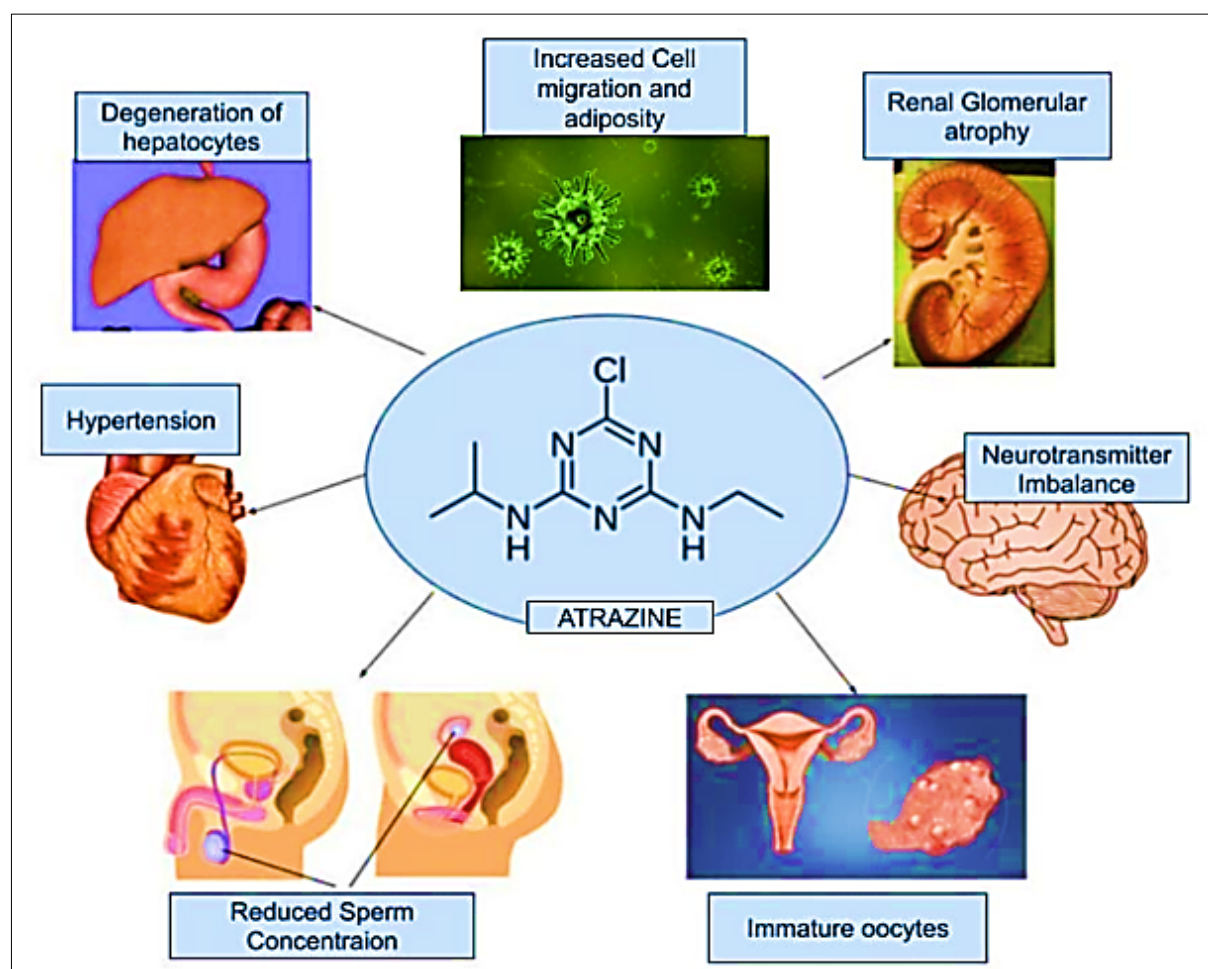


Fig 2: Effect of atrazine on different Organs

Organ-Specific Toxicity

Reproductive Toxicity

As the atrazine is present in water sources, concerns have arisen over its possible impacts on human health. Atrazine has been identified in human plasma, urine, semen, follicular fluid, cervical mucus and amniotic fluid²⁸. Its exposure has been linked to reduced fertility, altered spermatogenesis, reduced sperm concentration, delayed puberty and developmental abnormalities in laboratory animals.

Both male and female reproductive systems appear prone to atrazine toxicity, with effects often dependent on dose and exposure period. In male rats, atrazine exposure particularly at subchronic and chronic doses has been associated with decreased testicular weight, degeneration of seminiferous tubules, reduced spermatogenic cell layers, Leydig cell

dysfunction, and increased germ cell apoptosis, resulting in diminished sperm count, motility, viability, and increased morphological abnormalities²⁹. These structural and functional impairments are strongly linked to altered steroidogenesis, characterized by reduced circulating testosterone levels and disrupted expression of key steroidogenic enzymes such as StAR, 3 β -HSD, and 17 β -HSD. In female rats, atrazine exposure has been shown to disrupt estrous cyclicity, prolong diestrus, induce follicular atresia, reduce ovarian and uterine weights, and impair ovulation, effects that are often accompanied by altered estrogen and progesterone levels. Oxidative stress and increased lipid peroxidation in ovarian and testicular tissues further contribute to cellular damage, while enhanced inflammatory signaling and mitochondrial dysfunction exacerbate reproductive tissue injury. Developmental and

perinatal exposure appear particularly concerning, as early-life atrazine administration has been linked to delayed puberty onset, altered mammary gland development, and long-term reproductive dysfunction in adulthood.

Hepatic and Renal Toxicity

The liver and kidneys are primary targets of atrazine toxicity due to their role in metabolism and excretion. Histopathological alterations, elevated liver enzymes, and impaired renal function have been reported following atrazine exposure in experimental mammals. Atrazine administration led to increased levels of liver markers (AST, ALT, and ALP) and kidney markers (creatinine and urea) in both atrazine-treated and diabetic rats subjected to atrazine treatment. The markedly elevated AST and ALT activity in rats treated with atrazine (300 $\mu\text{g kg}^{-1}$ body weight) compared to control groups is attributed to the leakage of aminotransferase (AT) enzymes from damaged liver cells. In the liver, atrazine induces sinusoidal dilation, hepatocellular degeneration, cytoplasmic vacuolization, inflammatory cell infiltration and in severe cases, necrosis, as evidenced by histopathological evaluations. Structural modifications coincide with notable increases in liver indicators, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), signifying reduced membrane integrity and diminished liver function. Systematically, atrazine enhances the generation of free radicals and lipid peroxidation while depleting endogenous antioxidant defences such as superoxide dismutase (SOD), glutathione (GSH), and catalase (CAT), thereby promoting mitochondrial dysfunction and activation of pro-inflammatory signaling pathways²⁶. In kidney tissues, atrazine treatment leads to glomerular shrinkage or congestion, tubular epithelial degeneration, interstitial inflammation and thickening of the basement membrane, accompanied by elevated serum creatinine, urea, and blood urea nitrogen (BUN) levels, reflecting impaired glomerular filtration and tubular reabsorption capacity. The nephrotoxic effects are likewise linked to oxidative stress and apoptotic signaling within renal tubular cells. Additionally, chronic exposure may disrupt xenobiotic-metabolizing enzymes and interfere with detoxification pathways, exacerbating cumulative organ damage³⁰.

Neurotoxicity and Behavioural Effects

Evidence suggests that atrazine can affect the central nervous system, leading to neurochemical alterations and behavioral changes such as impaired learning, memory deficits, and altered motor activity. These effects may be linked to oxidative stress and neurotransmitter imbalance. In rats, atrazine easily crosses the blood-brain barrier, causing oxidative stress by increasing reactive oxygen species (ROS) generation, lipid peroxidation, and nitric oxide levels. Endogenous antioxidant defenses such as SOD, CAT, and GSH are all suppressed at the same time. The biochemical changes are followed by mitochondrial dysfunction, reduced ATP synthesis, and activation of pro-inflammatory pathways, including elevation of cytokines (e.g., $\text{TNF-}\alpha$, $\text{IL-1}\beta$) and microglial activation, indicating a state of chronic neuroinflammation³¹. Atrazine exposure has also been shown to interfere with neurotransmitter systems, particularly dopaminergic, serotonergic, and GABAergic signaling, leading to altered dopamine turnover in the

striatum and changes in acetylcholinesterase activity, thereby affecting synaptic transmission and neuronal communication.

Immunotoxicity

Atrazine has been shown to modulate immune responses, including changes in immune cell populations and suppressed antibody production, potentially increasing susceptibility to infections and immune-related disorders. Immunotoxicity refers to the detrimental effects of xenobiotics on the immune system, resulting in alterations to both nonspecific and acquired immunity. Immunotoxins exert both direct and indirect effects, including thymic atrophy and splenomegaly, which influence immunocyte populations and alter cytokine levels and intracellular signaling pathways²⁷. In adult male Balb/c mice treated with atrazine for 28 days, there was a diminished proliferative response to Con A, heightened apoptosis in lymphocytes, and a reduction in interferon- γ (IFN- γ) and interleukin-4 (IL-4) levels²⁹.

Carcinogenic and Metabolic Effects

Long-term exposure studies indicate a possible association between atrazine and increased risk of certain tumors in rodents, although results remain inconsistent. Additionally, emerging research suggests that atrazine may contribute to metabolic disturbances such as insulin resistance and obesity, warranting further investigation. Atrazine has been extensively investigated for its carcinogenic and metabolic effects in mammals, with evidence suggesting that its toxicity is largely mediated through endocrine disruption, hormonal imbalance, and metabolic dysregulation rather than direct genotoxicity. In rodent models, chronic atrazine exposure has been associated with an increased incidence of mammary gland tumors, particularly in female rats, a phenomenon attributed to disrupted hypothalamic-pituitary-gonadal (HPG) axis function and prolonged estrogen and prolactin stimulation resulting from altered luteinizing hormone (LH) regulation. Atrazine is known to interfere with steroidogenesis by modulating aromatase activity and altering the synthesis and metabolism of sex steroids, thereby creating a hormonal environment conducive to tumor development in hormonally sensitive tissues³². Although atrazine is generally considered non-mutagenic in standard genotoxicity assays, it may promote carcinogenesis indirectly through oxidative stress, chronic inflammation, epigenetic modifications, and dysregulated cell proliferation and apoptosis pathways. Beyond carcinogenic potential, atrazine has also been implicated in metabolic disturbances in mammals, including insulin resistance, altered glucose homeostasis, dyslipidemia, and increased adiposity following subchronic or chronic exposure. Mechanistic studies indicate that atrazine can impair mitochondrial function, reduce oxidative phosphorylation efficiency, and disrupt energy metabolism in hepatic and adipose tissues, thereby contributing to metabolic imbalance. Additionally, oxidative stress and inflammatory cytokine activation in metabolic organs such as the liver, pancreas, and adipose tissue further exacerbate metabolic dysfunction and may predispose exposed animals to features resembling metabolic syndrome.

Conclusion and Future Perspectives

Atrazine-induced toxicity in mammals involves complex mechanisms, including endocrine disruption, oxidative

stress, inflammation, and mitochondrial dysfunction, affecting multiple organ systems. Although substantial evidence demonstrates its potential health risks, gaps remain regarding chronic low-dose exposure and long-term effects. Future research should focus on comprehensive mechanistic studies, improved exposure assessment, and epidemiological investigations to better inform regulatory decisions. Enhanced environmental monitoring and stricter control measures are essential to reduce mammalian exposure and safeguard public health. In conclusion, accruing evidence from mammalian studies validates that atrazine exerts multi-organ toxicity characterized by neurotoxic, reproductive, hepatic, renal, carcinogenic and metabolic disturbances, largely mediated through endocrine disruption, oxidative stress, mitochondrial dysfunction, inflammation. Experimental findings consistently show that both acute and chronic exposures, including environmentally relevant doses, can impair hormonal homeostasis, compromise organ veracity and induce long-term functional deficits, with developmental exposure posing particular concern due to its potential to cause persistent physiological and metabolic reprogramming. Although atrazine is not widely classified as a direct genotoxic carcinogen, its capacity to alter steroidogenesis, disrupt the hypothalamic–pituitary axes, and promote oxidative and inflammatory microenvironments underscores its role as an indirect toxicant with significant systemic consequences.

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