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(REVIEW ARTICLE)

The impact of triclosan on renal and pulmonary systems: A comprehensive review

Shruti S. Rajak, Mangala T. Thakare *, Varsha T. Dhurvey and Vandana V. Patil

Department of Zoology, Rashtrasant Tukadoji Maharaj, Nagpur University, Nagpur-440033, M.S., India.

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Abstract

Triclosan (TCS) is a widely used antimicrobial agent found in personal care products, household items, and industrial applications. While its effectiveness as a bacteriostat is well documented, emerging research highlights its potential toxicity as it is an endocrine-disrupting chemical (EDC) that affects hormonal balance and is absorbed through skin contact, ingestion, and inhalation. This review underscores the potential health hazards of long-term TCS exposure, particularly in disrupting kidney and lung function. Experimental research in rodents and aquatic species has demonstrated that TCS exposure leads to weight loss, renal fibrosis, structural alterations, oxidative stress, and apoptosis. Histological and biochemical analyses reveal increased blood urea nitrogen and creatinine levels, indicating impaired kidney function. Pulmonary toxicity studies link TCS exposure to asthma, allergic reactions, and lung inflammation. Given its extensive use beyond regulated applications, future research should focus on understanding its long-term effects and developing safer formulations to mitigate public health risks.

Keywords: Triclosan Toxicity; Renal Dysfunction; Pulmonary Toxicity; Endocrine Disruptor; Oxidative Stress; Environmental Health

1. Introduction

Triclosan is a nonionic compound that appears as an off-white, odourless, and tasteless powder. Its chemical name is 2,4,4′-trichloro-2′-hydroxydiphenyl ether, and its molecular formula is $C_{12}H_7Cl_3O_2$ [1]. The substance has a long history as a potent bacteriostat and antiseptic. Its effectiveness and safety have made it a widely used component in personal care products designed for skin application, including soaps, deodorants, and skin cleansers [2].

Triclosan (TCS) was initially introduced into the healthcare industry in 1972 and later became toothpaste formulations in Europe by 1985 [1]. Products like soaps, cosmetics, and shampoos are regulated by the FDA, but this oversight only extends to antiseptic washes intended for non-medical use, such as antibacterial hand soaps for household settings. As a result, TCS continues to be used extensively in numerous commercial products that fall outside FDA regulation, particularly in items like furniture, clothing, and kitchenware, including knives and cutting boards [3].

TCS exerts its effect on the phospholipid membrane by acting like a detergent, disrupting the stability of lipid structures [4]. However, some bacterial species have evolved intricate mechanisms to counter the toxic effects of TCS. A key strategy involves non-specific multidrug resistance (MDR) efflux pumps [5].

1.1. General study of triclosan

Triclosan is classified as an endocrine disruptor (EDC) due to its potential to affect the estrogen, androgen, and thyroid systems, leading to hormonal imbalances [6,7,8]. Humans can come in contact with triclosan via contact on skin or

* Corresponding author: Mangala T. Thakare.

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even through oral consumption [9,10]. TCS in the environment is predominantly encountered through contaminated water, food, or animal sources [11].

Multiple studies have indicated that TCS exposure may promote liver tumor development in mice by increasing liver cell proliferation, inducing fibrogenesis, and elevating oxidative stress [12,13].

TCS exhibits toxic effects, such as reduced cell viability accompanied by morphological alterations in L2 rat epithelial lung cells, Additionally, a single intratracheal instillation at a dose of 1 mg/kg induced acute inflammation and increased lung permeability [14].

The kidney plays a crucial role in detoxification; however, studies indicate that exposure to environmental pollutants can contribute to early kidney damage, increase the risk of chronic kidney disease (CKD), and potentially progress to end-stage renal disease (ESRD) [15,16]. Moreover, higher levels of TCS exposure may increase the likelihood of developing asthma, allergies, and food sensitization [17]. It has also been suggested that frequent use of antimicrobial products is associated with an increased risk of wheezing and allergic rhinitis [18].

2. Source

This review presents a thorough assessment of triclosan's toxicity and associated risks, with a particular emphasis on its effects on the kidneys and lungs. The literature includes both experimental and non-experimental using databases such as PubMed, Scopus, Web of Science, Science Direct, EMBASE and Google Scholar, covering studies published between 2010 and 2024.

2.1. TCS on renal function

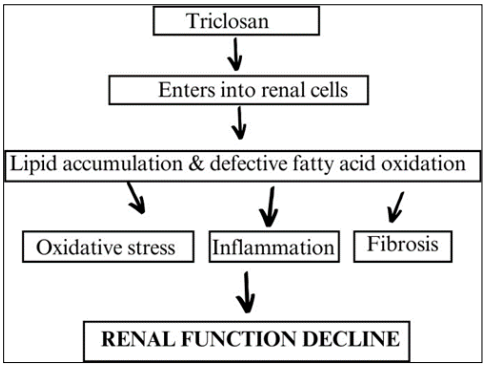

Figure 1 TCS mode of entry and its renal toxicity [19]

2.2. Survey Based Studies

The potential for early kidney damage due to environmental endocrine-disrupting chemicals (EDCs) and heavy metals was explored in a study using data from the Second Korean National Environmental Health Survey (2012–2014), they reported a positive association between higher levels of TCS and elevated urinary β2-microglobulin (β2M), a marker of kidney injury [16]. Similarly, the correlation between personal care product usage and urinary TCS concentrations was studied in 5962 participants aged 3 to 80 years, based on data from the Korean National Environmental Health Survey. The study found that men use fewer personal care products than women, resulting in higher TCS concentrations in females. Among urinary chemicals, the concentrations decreased in the order of propyl paraben > TCS > methyl paraben [20].

2.3. Body weights and Anatomical changes

A study examined the dermal toxicity of TCS where the mice received 0, 5.8, 12.5, 27, 58, and 125 mg/kg/bw for 13 weeks where substantial reductions in average body weights was seen. Additionally, Dermal fibrosis, inflammation, epidermal hyperplasia, necrosis, ulceration, and parakeratosis were observed. Low kidney weight in males and high in females indicated kidney damage [21]. Channa punctatus fish was exposed to TCS concentrations of 0.37 mg/L and 1.11 mg/L for 96 hours and depletion in the kidney was 36.31% in sub-lethal and 55.21% in lethal depletion of carbohydrate levels. This suggested that TCS exposure enhances glycogenolysis and glycolytic activity to meet increased energy demands under stress conditions. [22]. 30 male Sprague-Dawley and 15 bighead carp rats were exposed to TCS doses

of 0.25, 25, 250, or 750 mg/kg for 60 days and 15 days, respectively. The results reported a reduction in body weight, kidney weight necrosis of tubules, and increased urinary spaces. [15, 23]. The studies collectively demonstrate that exposure to TCS can result in notable physiological and anatomical alterations, such as reductions in body weight, organ-specific damage, and disruptions in metabolic processes across different species

2.4. Histological and Biochemical Alterations

According to [24] TCS exposure can lead to structural and functional alterations in the renal cortex, 200 mg/kg dose of TCS for 6 weeks was given to 30 adult male albino rats which resulted in significant histological abnormalities, including increased cellularity in the glomeruli and disorganization of the tubules. Moreover, blood urea nitrogen and serum creatinine levels were elevated, indicating impaired renal function.

Elevated levels of blood urea nitrogen and creatinine, alterations in Bowman's space, tubular occlusion, and epithelial cell degeneration were seen in rats and in mice renal hypertrophy showed a higher prevalence of apoptotic cells after 8 months of exposure to 0.008% TCS [15, 12]. Similarly, another study investigated the toxic effects of four samples of reclaimed water i.e. control, WTE, WTI, and MBRP (Membrane bioreactor permeate) on human embryonic kidney cells for 24 hours. The reclaimed water samples disrupted the levels of proteins involved in cell division and death, suggesting their harmful impact on kidney cells [25].

These studies collectively point to significant health risks from TCS exposure, with observed kidney damage, which includes fibrosis, cellular apoptosis, and impaired organ function, demonstrating the widespread toxic impact of triclosan.

2.5. Oxidative stress and Cell apoptosis

An in vitro study investigated TCS effects on human renal glomerular endothelial cells where cells were exposed to 5 - 100 μmol/L for 24 hours. TCS induced oxidative stress in endothelial cells, potentially leading to cell death by disrupting the PI3K/Akt signaling pathway. Apoptosis rates were 81.6%, 67.7%, and 40.5% at TCS concentrations of 15, 20, and 30 μmol/L, respectively, indicating that TCS significantly triggers cell death in a dose-dependent manner [26]. In the same way, increased high mobility group box 1 (HMGB1) protein expression confirmed tubular necrosis in rats [15].

TCS was orally given to 30 male C57BL/6 mice at doses of 10 and 100 mg/kg/day) for 10- 13 weeks which led to renal injury, increased oxidative stress markers like malondialdehyde (MDA) levels, along with reduced expression of superoxide dismutase (SOD) and total cholesterol (TCHO)pro-inflammatory cytokines, and fibrotic markers in a dose-dependent manner, Additionally, lipid accumulation and disrupted fatty acid metabolismand alterations in gut microbiota in mouse kidneys were seen which further can contribute to renal dysfunction [19,27] Taken together, these studies demonstrate that TCS exposure induces oxidative stress

apoptosis, and renal injury in a dose-dependent manner, with significant impacts on inflammation, fibrosis, and metabolic disruptions, further highlighting its potential to cause renal dysfunction

2.6. TCS on pulmonary function

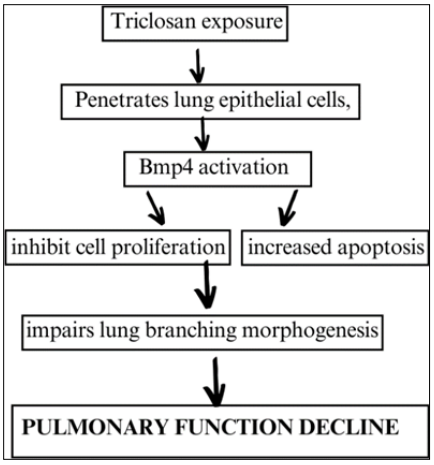

Figure 2 TCS mode of entry and pulmonary toxicity [28]

2.7. Survey-based and Case Studies

Panel studies highlighted a strong connection between TCS exposure and allergic disease occurrence in preschool-aged children [17,29]. Relationship between prenatal exposure to environmental phenol and phthalate biomarkers and respiratory or allergic conditions in children was examined studies found that prenatal exposure to TCS was linked to a higher likelihood of an asthma diagnosis due to activation of Bmp4 inducing lung apoptosis and cyst like formation in lungs [30,31]. Urinary TCS levels were significantly associated with an increased prevalence of asthma exacerbations, as analyzed using data from 600 samples in the National Health and Nutrition Examination Survey (NHANES) conducted between 2005 and 2010 [31]. A case study focused on a 26-year-old female with occupational asthma, who developed immediate asthmatic reactions triggered by exposure to Antibac cleaner containing triclosan. Serial peak flow

readings and specific inhalational challenges confirmed that TCS exposure contributed to allergic sensitivities to cleaning products and personal care items [32,33].

2.8. Anatomical and Histological Changes

Aerosol inhalation of TCS in rats over 28 days caused salivation, weight loss, nasal ulcers, severe inflammation, and histopathological changes in the nasal septum and larynx, with males and females equally affected at high concentrations [34]. In aquatic environments, TCS has also been shown to negatively impact respiratory systems. In one study, exposure of freshwater fish to sublethal concentrations of TCS caused significant gill damage, including thickened lamellae, swelling, and fusion, which impaired gas exchange [35].

Some studies found similar results in different species of fish where the authors observed an increase in lipid peroxidation (LPO) levels in the gills, suggesting oxidative stress. Higher concentrations caused 100% mortality and behavioural changes such as air gulping, mucous deposition, and hemorrhagic eyes, along with histopathological damage to the gills, including hyperplasia and lamellar disorganization, hypertrophy, lamellar fusion, ruptures, twisted, uplifted secondary lamellae, disorganization, and necrosis of epithelial cells [23,36,37,38].

2.9. Oxidative stress and Cell Death

In a study, dermal exposure to TCS in BALB/c mice co-sensitized with ovalbumin (OVA) led to heightened allergic responses and airway hyperreactivity [39]. Some studies evaluated the harmful effects of TCS in mitochondrial cells and lung epithelial cell where it caused mitochondrial depolarization, necrotic cell death and oxidative stress through interference with ATP synthesis, respiratory control and induced reactive oxygen species (ROS) production, further highlighting its capacity to induce pulmonary toxicity [40,41,42]. In an experiment with Spargue Dawley rats, single intratracheal instillation of TCS caused inflammation, acute lung injury, and elevated pulmonary toxicity markers, indicating its potential impact on lung function [14]. Similarly, TCS was found to promote epithelial-to-mesenchymal (ETM) transition in lung cancer cells, and mitochondrial depolarization by reducing E-cadherin levels and activating adhesion kinase, interfering with ATP synthesis, thereby enhancing the migration and growth of anoikis-resistant cancer cells [42, 43]. Another study demonstrated that the topical application of TCS (1–3%) on BALB/cAnTac mice and human skin tissue led to an enhanced immune

response, with upregulation of TSLP, IL-1β, and TNF-α, and an increase in draining lymph node cellularity, indicating the potential for acute allergic reactions in the lungs [44].

The combined effects of TCS (185 mg/kg) and sodium fluoride (50 mg/kg) were investigated in rats, revealing oxidative stress, reduced antioxidant enzyme levels (SOD, CAT, and GSH), and upregulation of apoptotic genes in lung tissue. Interestingly, co-exposure to TCS and NaF resulted in less oxidative stress compared to individual exposures, suggesting a potential interaction that mitigates certain adverse effects [45].

The available literature suggests that TCS causes serious environmental problems, affecting the aquatic ecosystem and also severe health hazards to humans.

3. Conclusion

Exposure to triclosan, often found in personal care and household items, can have significant health implications. Elevated levels of TCS can damage the vital organs of the human body, including the liver, kidneys, and thyroid, hence damaging the organs and also causing hormonal imbalances. In the kidneys, it may result in the induction of oxidative stress and inflammation and interfere with the process of filtration as well as fluid regulation. Long-term exposure of TCS inhalation will also irritate the lung tissue, leading to risks for respiratory problems like inflammation and

breathing difficulty, particularly among asthma or other respiratory-sensitive people. While these findings highlight serious concerns, further research is crucial to fully understand triclosan's long-term effects on human health and its impact on different populations.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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