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Toxicological Effects of Organ phosphorus Pesticide Chlorpyrifos Residues

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Introduction

Numerous environmental pollution issues have resulted from the misuse of chlorpyrifos (CHP), a common organ phosphorus pesticide, particularly its toxicological effects on non-target organisms. To start with, CHP advanced on the outer layer of plants enters biological system course along the well-established pecking order. Second, direct inflow of CHP into the water climate under the activity of water spillover unavoidably makes harmfulness non-target organic entities. To create a CHP exposure toxicity model and investigate the effects of CHP on rats, we used rats as a model. Additionally, we utilized chitosan oligosaccharide (COS)'s high water solubility and natural biological activity to investigate the alleviation effect of COS on CHP toxicity in rats in order to alleviate and remove the injuries caused by residual chlorpyrifos in vivo.

Toxicological Effects of Organ phosphorus

The findings demonstrated that COS has excellent removal and mitigation effects on the toxic damage caused by residual CHP in the environment, and that CHP can induce the toxicological effects of intestinal antioxidant changes, inflammation, apoptosis, intestinal barrier damage, and metabolic dysfunction in rats. In conclusion, COS demonstrated significant biological effects in removing and reducing changes in blood biochemistry, antioxidants, inflammation, apoptosis, the structure of the gut barrier, and metabolic function caused by environmental residual CHP [1].

After ingestion, the gut serves as the initial physiological barrier to exposure to the environmental pollutant CHP [2]. Human and animal digestion and nutrient absorption may be affected by intestinal barrier breakdown. CHP is detoxified into 3,5,6-trichloro-2-pyridinol and the active substance CHP-oxon by cytochrome P450 in vivo after being absorbed in the intestines [3]. By irreversibly inhibiting the activity of acetyl cholinesterase (Ache), the latter blocks nerve cells' cholinergic pathway [4]. During both sub chronic and chronic exposure to CHP, oxidative stress is another important toxicity mechanism. Through increased production of reactive oxygen species (ROS), DNA damage, and lipid peroxidation, CHP can cause neuronal damage [5]. This can lead to deficits in motor activity, cognitive performance, and coordination skills. Likewise, harmfulness of CHP to different frameworks has additionally been accounted for, including diminished male regenerative limit [6] and hepatotoxicity. Long haul openness to CHP can likewise prompt impeded ripeness, hatchability, and undeveloped organism distortions [7]. Exposure to CHP has the potential to affect the nervous system of the fetus, resulting in neurological damage in the newborn [8]. However, there is no evidence of the toxicological effects of CHP on gut health, and intestinal absorption is the primary route by which environmental residual CHP enters the body after oral administration. As a result, the impact of CHP on gut health is particularly troubling.

As a result, in order to examine the toxicological effects of CHP residues in the environment on the gut, we established a poisoning model employing rats as the research subject [9]. The harmfulness of CHP to organic entities and its atomic instruments were assessed from the parts of small digestive system cancer prevention agent limit, aggravation, apoptosis, gastrointestinal respectability, gastrointestinal metabolic capability, and microbial overflow. Furthermore, chitosan oligosaccharide (COS) is an oligomer with high water dissolvability and organic action that keeps up with huge normal natural action . It may have the ability to regulate toxins' metabolism and boost immunity .In addition, COS exhibits biological activity that enhances intestinal structure, enhances antioxidant capacity, and improves immunity , as well as growth , intestinal morphology and barrier function [10]. The scavenging of intracellular free radicals, significant inhibition of NF-kB activation, effective enhancement of systemic immune responses, and regulation of the function of immune reactive cells are all mediated by COS [11]. We then hypothesized whether COS could effectively remove and reduce the toxicological effects of the exogenous pollutant CHP on the intestines based on these positive effects. Our findings lend credence to the hypothesis that COS significantly reduces and eliminates the environmental toxicity brought on by residual CHP. In this experiment, we first looked into the related mechanisms and used COS to reduce the toxic effects of CHP. In addition, 16S rRNA sequencing and metabolomics were used to investigate the CHP-induced changes in rats' intestinal metabolites and microbes as well as the COS's protective effects. Finally, we added Spearman correlation analysis in comparison to previous experiments to predict the potential role of gut microbes in intestinal metabolic pathways [12]. The treatment and prevention of CHP stand to gain from these findings.

Discussion

O-diethyl-O-(3,5,6-trichloro-2-pyridyl) phosphorothioate of chlorpyrifos; After monocrotophos, acephate, and endosulfan, CHP] is the fourth most widely used pesticide worldwide. It is also the organ phosphorus insecticide that is most frequently detected. CHP is categorized as a moderately toxic class II pesticide by the Organ phosphorus Pesticides (OPPs) classification established by the World Health Organization (WHO); As a result, it is widely used and abused in agricultural production due to its relative safety in comparison to other OPPs. CHP is currently used extensively as a nematicide and acaricide to control various crops, lawns, and ornamental plants in agriculture, industrial sites, household pesticides, horticulture, and

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government. In 2014, China alone used more than 2000 tons of CHP, and Brazil used 570 million liters of it over the past decade. China used 28,600 tons of CHP in total by 2018 [2]. The sum following up on the expected objective after CHP application is under 0.1%, and the rest of in the climate. Rainwater runoff introduces residual and non-target CHP into the ecosystem's circulation, accumulates in river water, soil, and the atmosphere, and is then absorbed by non-target organisms. During the plant-growing season, CHP has been frequently detected in agricultural water environments [9]. Common crops like tomato, organic spinach, apple, and soybean have also been found to contain CHP residues What's more, as a result of major areas of strength for the of CHP, its half-life in the climate can surpass 2 years. All surface water samples showed a 54% detection rate as a result. Food containing residual CHP is also stable when stored and can even be detected in processed goods. For example, the residue in wheat reached 28% after 120 days of storage. CHP buildups in flour, chapattis, and bread in the wake of handling came to 65-83%, 29-51%, and 25-75%, separately. Subsequent to plying, maturation, and steaming, CHP deposits in Chinese steamed buns must be diminished by 36%, 5%, and 4-38%, separately. Through direct skin contact, food, and water intake, these residues eventually enrich CHP in animals and humans. For instance, pregnant women's urine has been found to contain CHP and its metabolites. Between 1974 and 2005, there were approximately 278 documented cases of human CHP poisoning. CHP use peaked in the 1960s. As a result, the US Environmental Protection Agency made a proposal in 2000 to stop using CHP, which was restricted and banned in many countries to protect the health of animals and the environment.

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