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In Male Mice, Fucoidan Guards Against Oxidative Stress and Hematological/Biochemical Changes Brought on by Acute Sulfoxaflor

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Introduction

In recent years, oxidative stress has emerged as a pivotal factor in the pathogenesis of various diseases and conditions, including cardiovascular diseases, neurodegenerative disorders, and cancer. It results from an imbalance between the production of Reactive Oxygen Species (ROS) and the body's ability to counteract or detoxify their harmful effects through antioxidants. Environmental and chemical stressors, including pesticides, have been shown to induce oxidative stress, leading to cellular and systemic damage. Among these chemicals, sulfoxaflor, a novel sulfoximine-based insecticide, has gained significant attention due to its widespread use and potential toxicity [1].

Sulfoxaflor is primarily used to control sap-feeding insects, such as aphids and whiteflies, which are major agricultural pests. Its mode of action involves targeting the nicotinic acetylcholine receptors in the nervous system of insects, leading to paralysis and death. While effective against pests, sulfoxaflor's safety profile for non-target organisms, including mammals, has been a subject of ongoing research and debate. Acute exposure to sulfoxaflor has been associated with various toxicological effects, including oxidative stress and alterations in hematological and biochemical parameters, which are indicative of systemic toxicity [2].

Description

Fucoidan, a complex polysaccharide found in brown seaweeds such as Fucus vesiculosus and Laminaria japonica, has gained recognition for its wide range of biological activities, including antioxidant, anti-inflammatory, and anticancer properties. The potential of fucoidan to mitigate the adverse effects of various toxicants, including those that induce oxidative stress, has been the focus of numerous studies. Fucoidan's antioxidant properties are attributed to its ability to scavenge free radicals, chelate metal ions, and enhance the activity of endogenous antioxidant enzymes. Oxidative stress is a condition characterized by an imbalance between the production of Reactive Oxygen Species (ROS) and the body's antioxidant defenses. ROS, including superoxide anion, hydrogen peroxide, and hydroxyl radicals, are highly reactive molecules that can damage cellular components such as lipids, proteins, and DNA [3].

Exposure to environmental and chemical stressors, including pesticides, can overwhelm the body's antioxidant defenses, leading to oxidative stress. Sulfoxaflor, a sulfoximine-based insecticide, is one such chemical that has been shown to induce oxidative stress in various organisms. The mechanism by which sulfoxaflor induces oxidative stress is thought to involve the generation of ROS and the depletion of endogenous antioxidants. This oxidative stress can lead to lipid peroxidation, protein oxidation, and DNA damage, ultimately resulting in cellular and systemic toxicity. In addition to oxidative stress, acute exposure to sulfoxaflor can cause significant alterations in hematological and biochemical parameters. Hematological parameters, including Red Blood Cell (RBC) count, hemoglobin (Hb) concentration, hematocrit (Hct), white blood cell (WBC) count, and platelet count, are critical indicators of the body's overall health and its ability to transport oxygen, fight infections, and clot blood. Biochemical parameters, including liver enzymes (such as Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST)), kidney function markers (such as Blood Urea Nitrogen (BUN) and creatinine), and lipid profiles (such as cholesterol and triglycerides), provide insights into the functional status of various organs and systems [4].

Studies have shown that acute exposure to sulfoxaflor can result in significant changes in these hematological and biochemical parameters, reflecting systemic toxicity. These changes may include decreases in RBC count, Hb concentration, and Hct, indicative of anemia; increases in WBC count, suggestive of an inflammatory response; and alterations in liver and kidney function markers, indicative of organ damage.

Fucoidan, a sulfated polysaccharide found in brown seaweeds, has been extensively studied for its diverse biological activities. Its antioxidant properties are of particular interest in the context of oxidative stress induced by chemical toxicants. Fucoidan's antioxidant effects are attributed to its ability to scavenge free radicals, chelate metal ions, and enhance the activity of endogenous antioxidant enzymes. These properties suggest that fucoidan could potentially mitigate the oxidative stress and associated toxicity induced by sulfoxaflor [5].

Conclusion

In conclusion, oxidative stress and hematological/biochemical changes induced by acute sulfoxaflor exposure pose significant health risks to non-target organisms, including mammals. The protective effects of fucoidan, a bioactive polysaccharide derived from brown seaweeds, against these adverse effects highlight its potential as a therapeutic agent for mitigating pesticide-induced toxicity. Fucoidan's ability to scavenge free radicals, chelate metal ions, and enhance the activity of endogenous antioxidant enzymes plays a crucial role in reducing oxidative stress and preventing cellular damage. Additionally, its anti-inflammatory properties and organ-protective effects further contribute to its overall protective actions. Studies using animal models, such as male mice, provide valuable insights into the mechanisms through which fucoidan exerts its protective effects. By normalizing oxidative stress markers, antioxidant enzyme activities, and hematological/biochemical parameters, fucoidan demonstrates its potential to counteract the toxic effects of sulfoxaflor.

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Conflict of Interest

None.

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