INVESTIGATING REPRODUCTIVE TOXICITY OF CARBOFURAN IN MALE ALBINO RAT WITH MECHANISTIC INSIGHTS

Project Reference No.: *48S_MSC_0059*

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Keywords: Carbofuran, Pesticide, Reproductive Toxicity, hormonal disruption, apoptotic pathways.

Introduction:

Carbofuran is a highly toxic carbamate pesticide extensively used in modern agriculture to manage a broad range of insect pests affecting key crops such as corn, rice, potatoes, and vegetables. Although effective in pest control, its persistent and non-selective nature has raised global concerns about its environmental safety and adverse effects on non-target species, including humans and wildlife. Residues of Carbofuran have been detected in soil, water, and agricultural produce, pointing toward a significant risk of bioaccumulation and chronic exposure.

Among the various health hazards posed by Carbofuran, its potential to induce reproductive toxicity is particularly alarming. The male reproductive system is highly susceptible to xenobiotic interference, especially chemicals that disrupt endocrine regulation, oxidative homeostasis, or testicular architecture. Several studies have reported that Carbofuran exposure may impair spermatogenesis, alter testosterone levels, reduce sperm motility and viability, and induce histological damage in reproductive organs. However, the precise mechanisms by which Carbofuran exerts its reproductive toxicity remain incompletely understood.

This study aims to systematically investigate the reproductive toxicity of Carbofuran in male albino rats, with a particular focus on identifying mechanistic pathways involved in testicular damage and hormonal disruption. A 40-day sub-chronic exposure model was employed, incorporating multiple dose groups, including a recovery group to assess potential reversibility of the toxic effects. Comprehensive evaluation includes analysis of sperm parameters, serum reproductive hormone levels, oxidative stress biomarkers, antioxidant enzyme activities, and histopathological alterations in testicular tissue.

Understanding the mechanistic basis of Carbofuran-induced reproductive toxicity is essential not only for toxicological profiling but also for formulating appropriate regulatory policies and public health interventions. This research contributes to the growing body of evidence on pesticide-induced reproductive risks and underscores the urgent need for safer pest management alternatives.

Objectives:

1. To elucidate mechanistic insights into Carbofuran's reproductive toxicity, with a focus on oxidative stress-mediated damage and hormonal disruption.

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Methodology:

Animal Model:

- **Species:** Wistar Male Rat
- **Age:** 10–11 weeks.
- **Weight:** 200–250 g.
- **Group Size:** 6 mice each group.

Chemical Properties:

- Carbofuran (2,3-dihydro-2,2-dimethyl-7-benzofuranol N-methylcarbamate; CAS No. 1563-66-2; 98% pure, was purchased from Sigma-Aldrich, Bangalore.
- **Chemical formula:** $C_{12}H_{15}NO_3$

- **Molecular weight:** 221.25 g/mol
- **Color**: White, Crystalline Solid.
- **Melting point:** 150-152°C
- **Solubility:** Water 700 ppm at 25°C.

We used Vehicle as Corn oil

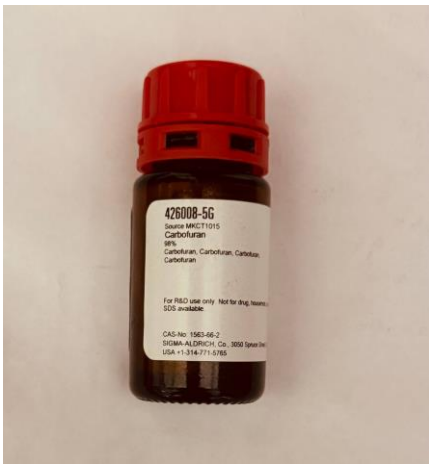
Experimental Groups:

1. **Normal Control group:** Vehicle only (Corn oil) kg body weight/day.
2. **Low-dose group:** Carbofuran at 0.5 mg/kg body weight/day.
3. **Medium-dose group:** Carbofuran at 0.7 mg/kg body weight/day.
4. **High-dose group:** Carbofuran at 1 mg/kg body weight/day.
5. **Recovery group:** Carbofuran at 1 mg/kg body weight/day for 14 days, followed by 14 days without exposure.

Ethical Approval and Precursor

All animal experiments received approval from the Institutional Animal Ethics Committee (IAEC) as identified by approval number (SUK/ZOL/IAEC/05/2024-25) through Sharnbasva University Department of Postgraduate Studies and Research in Zoology in Kalaburagi, Karnataka, India (CCSEA Regd. no.: 2236/PO/ReBiBt/S/23/CCSEA).

Result:

Oral Gavage Administration Protocol, Compound Preparation, and Animal Maintenance During Carbofuran Exposure in Male Albino Rats"		
		
		
		

Conclusion:

The study is currently evaluating the reproductive toxicity of Carbofuran in male albino rats through a 40-day exposure model. Preliminary observations suggest potential dose-dependent effects on body weight, hormonal balance, and testicular histology. Mechanistic insights into oxidative stress and apoptotic pathways are being analysed. Final results will clarify the extent of Carbofuran-induced damage and its reversibility, contributing to toxicological risk assessment and the need for safer agricultural alternatives. Further data will validate these findings and guide future research directions.

Future Scope:

The present study provides foundational insights into the reproductive toxicity of Carbofuran; however, several avenues remain open for further exploration. Future research should focus on elucidating the precise molecular mechanisms underlying Carbofuran-induced testicular damage, including the roles of gene expression alterations in steroidogenesis, apoptosis, and oxidative stress pathways. Long-term and transgenerational studies are essential to determine whether the toxic effects of Carbofuran persist beyond the exposure period or are heritable. Additionally, evaluating the efficacy of natural antioxidants, herbal formulations, or pharmacological agents in preventing or reversing reproductive toxicity could offer promising therapeutic strategies. Comparative toxicological studies across different species and age groups may help establish broader toxicokinetic profiles and identify vulnerable populations. Translating animal model findings to human risk assessment scenarios, particularly for individuals with occupational or environmental exposure, is crucial for regulatory policymaking. Lastly, the development of sensitive and specific biomarkers for early detection of reproductive toxicity would significantly enhance monitoring and intervention strategies in environmental health and toxicology.