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Effects of Chlorpyrifos Technical Grade on Glycogen Metabolism in Channa Punctatus

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Abstract

This study investigates the biochemical effects of chlorpyrifos technical grade, an organophosphate insecticide, on glycogen metabolism in the freshwater fish *Channa punctatus*. Glycogen content was assessed in the liver, brain, kidney, gill, and muscle tissues after exposure to lethal and sub-lethal concentrations of chlorpyrifos for 24 hours. The results demonstrated a significant reduction in glycogen levels across all tissues, with the liver exhibiting the most considerable depletion. The depletion of glycogen is attributed to increased energy demands under pesticide-induced stress, along with potential inhibition of enzymes involved in glycogen synthesis. This study highlights the adverse metabolic effects of chlorpyrifos exposure, emphasizing its potential risks to aquatic ecosystems.

Keywords: Chlorpyrifos, organophosphate insecticide, glycogen metabolism, Channa.

Introduction

Glycogen metabolism is a fundamental biochemical process responsible for energy storage and mobilization in organisms. Under normal physiological conditions, carbohydrates are stored as glycogen primarily in the liver and muscles, providing an essential energy reserve to support various metabolic activities (Lehninger, 2004). However, environmental stressors such as toxic compounds, including pesticides, can disrupt carbohydrate metabolism, leading to adverse effects on the survival and health of aquatic organisms.

Chlorpyrifos, a widely used organophosphate insecticide, has been identified as a potent environmental contaminant with harmful effects on non-target aquatic species (Latner, 1975; Harper, 1985). It interferes with acetylcholinesterase activity, causing neurotoxic effects, and may also disrupt metabolic pathways, including those related to carbohydrate metabolism (O'Conner et al., 1982; Bhatia et al., 1978).

Previous studies have reported significant changes in glycogen levels in various aquatic species exposed to pesticides. For instance, Israel Stalin et al. (2012) observed the highest decrease in glycogen levels in the liver of fish exposed to chlorpyrifos. Similarly, Neera Srivastava and Hemlata Verma (2009) noted significant glycogen depletion in the ovaries of fish due to increased energy demands for oocyte development.

In this study, we aim to investigate the effects of chlorpyrifos technical grade on glycogen metabolism in *Channa punctatus*, focusing on the biochemical changes in different tissues and the potential implications for aquatic health.

Materials and Methods

Chemicals and Reagents

Chlorpyrifos (technical grade) was procured from a certified supplier. Reagents for glycogen estimation included anthrone, concentrated sulfuric acid, and distilled water.

Experimental Design

Adult *Channa punctatus* specimens were obtained from a local fish farm and acclimatized in laboratory conditions for one week. Fish were divided into three groups:

- **Control Group:** No exposure to chlorpyrifos.
- **Sub-lethal Exposure Group:** Exposed to 0.1 mg/L chlorpyrifos for 24 hours.

- **Lethal Exposure Group:** Exposed to 0.5 mg/L chlorpyrifos for 24 hours.

Tissue Collection

After the exposure period, fish were sacrificed, and tissues (liver, brain, kidney, gill, and muscle) were dissected, weighed, and stored in cold conditions for biochemical analysis.

Glycogen Estimation

The glycogen content in each tissue was determined using the enthrone method (Florkin & Stout, 1959). Tissue homogenates were prepared, and glycogen levels were measured spectrophotometric ally at 620 nm. Results were expressed as mg of glycogen per gram of wet tissue weight.

Results

The glycogen content in different tissues of *Channa punctatus* significantly decreased after exposure to chlorpyrifos (Table 1). The liver showed the highest depletion, followed by muscle, kidney, gill, and brain.

Table 1: Glycogen Content (mg/g Wet Weight) and% Change over Control in Different Tissues of *Channa punctatus* Exposed to Chlorpyrifos Technical Grade for 24 Hours

S.No	Tissue	Control (mg/g)	Sub-lethal (mg/g)	% Change (Sub-lethal)	Lethal (mg/g)	% Change (Lethal)
1	Gill	44.24 ± 0.82	35.86 ± 0.93	18.94% ↓	28.64 ± 0.48	35.26% ↓
2	Brain	42.12 ± 0.97	35.69 ± 0.84	15.26% ↓	32.26 ± 2.52	23.04% ↓
3	Liver	68.62 ± 0.48	54.72 ± 1.04	20.25% ↓	39.84 ± 2.01	41.94% ↓
4	Kidney	18.48 ± 0.87	14.42 ± 0.82	21.96% ↓	11.76 ± 1.19	36.36% ↓
5	Muscle	52.69 ± 0.93	42.32 ± 0.93	19.68% ↓	32.84 ± 0.84	37.67% ↓

Values are mean ± SD of five observations. Significant at $p < 0.05$.

Discussion (Expanded)

The significant reduction in glycogen content across all tissues of *Channa punctatus* after exposure to chlorpyrifos technical grade highlights the extensive metabolic disruption caused by this organophosphate insecticide. This study aligns with previous research showing that pesticide exposure adversely affects carbohydrate metabolism in aquatic organisms (Holden, 1974; Sastry et al., 1984).

1. Tissue-Specific Glycogen Depletion

The liver showed the most considerable glycogen depletion (41.94% under lethal conditions), which is consistent with its primary role in glycogen storage and metabolism. The liver is the central organ for gluconeogenesis and glycogen synthesis, and any disruption in its function can lead to widespread metabolic disturbances. The high sensitivity of liver tissues may be due to their high metabolic rate and constant involvement in detoxification processes, which increase energy demands under stress conditions (Lehninger, 2004).

Similarly, the muscle tissue also exhibited significant glycogen depletion (37.67% under lethal exposure). Muscle tissues rely heavily on glycogen for anaerobic metabolism, especially under stress conditions. The depletion here suggests that chlorpyrifos may impair not only the synthesis of glycogen but also affect the mobilization of energy reserves required for basic cellular activities.

2. Role of Stress-Induced Energy Demands

Exposure to chlorpyrifos induces physiological stress in fish, triggering a cascade of metabolic responses. Stress leads to the activation of the hypothalamic-pituitary-interrenal (HPI) axis, resulting in the release of cortisol, a hormone known to promote glycogenolysis and gluconeogenesis (Radhaiah, 1988). This metabolic shift is aimed at providing immediate energy to cope with the stress, leading to the depletion of glycogen reserves.

Moreover, chlorpyrifos-induced oxidative stress can damage cellular structures, further increasing energy demands for repair and maintenance. This is supported by studies showing elevated levels of reactive oxygen species (ROS) in aquatic organisms exposed to pesticides (Rama Murthy, 1988).

3. Enzymatic Disruption and Metabolic Pathway Impairment

The depletion of glycogen in the liver, brain, and kidney suggests a potential inhibition of key enzymes involved in glycogen metabolism, such as glycogen synthase and glycogen phosphorylase. Chlorpyrifos has been shown to interfere with enzyme activities related to acetylcholine metabolism, which could indirectly affect enzymes responsible for carbohydrate metabolism (O'Brien, 1977).

Additionally, the reduction in glycogen content could result from an increase in glycogenolysis, where stored glycogen is broken down to glucose to meet the heightened energy demands. This process may be accelerated under chlorpyrifos-induced stress, leading to rapid depletion of glycogen reserves.

4. Comparative Analysis with Previous Studies

Our findings are consistent with previous studies on the impact of pesticides on glycogen metabolism. For instance, Sastry et al. (1984) reported reduced glycogen levels in *Channa punctatus* after exposure to Quinolphos, while Moorthy et al. (1985) observed similar effects in freshwater mussels exposed to methyl parathion. These studies underscore the universal metabolic stress imposed by organophosphates across different aquatic species.

Furthermore, the observed glycogen depletion in the brain and kidney suggests systemic metabolic disruption, as these tissues are vital for neurological and renal functions, respectively. The decline in glycogen here could impair not only energy metabolism but also critical physiological processes such as neurotransmission and osmoregulation

Similar studies have reported glycogen depletion in various aquatic species exposed to pesticides, including methyl parathion and endosulfan (Sastry et al., 1984; Moorthy et al., 1985).

The significant decrease in glycogen levels in the kidney, muscle, and brain suggests that the toxic effects of chlorpyrifos extend beyond the liver, affecting multiple tissues involved in energy metabolism.

Conclusion

This study provides compelling evidence that chlorpyrifos technical grade significantly disrupts glycogen metabolism in *Channa punctatus*, with the most pronounced effects observed in the liver and muscle tissues. The reduction in glycogen content under both sub-lethal and lethal exposures indicates that chlorpyrifos imposes substantial metabolic stress, likely due to increased energy demands and enzymatic disruption.

The findings have important ecological implications, as the depletion of glycogen reserves can compromise the survival, growth, and reproductive capacity of aquatic organisms. Prolonged exposure to such pesticides may lead to chronic metabolic imbalances, affecting population dynamics and biodiversity in aquatic ecosystems.

Key Takeaways

1. **Chlorpyrifos Exposure Causes Glycogen Depletion:** Both sub-lethal and lethal exposures resulted in significant glycogen loss across all tested tissues, with the liver being the most affected.
2. **Metabolic Stress Response:** The observed glycogen depletion is likely due to increased energy demands triggered by pesticide-induced stress, leading to enhanced glycogenolysis and impaired glycogen synthesis.
3. **Enzymatic Disruption:** Chlorpyrifos may inhibit key enzymes involved in carbohydrate metabolism, exacerbating glycogen depletion.
4. **Ecological Concerns:** The metabolic disruption observed in *Channa punctatus* raises concerns about the potential impacts of chlorpyrifos on aquatic biodiversity and ecosystem health.

Recommendations for Future Research

- **Long-Term Studies:** Investigate the chronic effects of chlorpyrifos exposure on glycogen metabolism over extended periods.
- **Enzymatic Analysis:** Assess the activity of key enzymes involved in glycogen metabolism to confirm the mechanistic pathways of disruption.
- **Multispecies Studies:** Conduct similar studies on different aquatic species to evaluate the broader ecological implications of chlorpyrifos contamination.

Final Thoughts

The findings underscore the urgent need for regulatory measures to limit chlorpyrifos use in agricultural practices near aquatic habitats. Monitoring pesticide residues and their biochemical effects on aquatic organisms should be a priority to ensure the sustainability of freshwater ecosystems.

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