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Paradol Enhances Memory and Cognitive Function in an Amnesic Zebrafish Model

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INTRODUCTION & AIM

Cognitive impairment represents a hallmark of Alzheimer's disease, a progressive neurodegenerative condition characterized by significant deficits in memory and learning. Paradol, a bioactive compound derived from ginger, has emerged as a subject of interest due to its potential neuroprotective properties. This study aimed to evaluate the efficacy of Paradol in restoring cognitive function within a preclinical zebrafish model. Specifically, the research focused on whether Paradol administration could mitigate amnesia induced by okadaic acid, targeting both spatial and recognition memory processes.

METHOD

The experiment utilized sixty adult zebrafish, which were randomly assigned to six distinct experimental cohorts. To simulate Alzheimer-like pathology, amnesia was induced using 10 nM okadaic acid over a period of four days. Paradol was administered via immersion every three days for a total duration of one week. The treatment groups were organized as follows:

- **Control:** Dimethyl sulfoxide (DMSO)
- **Positive Control:** Galantamine (1 mg/L)
- **Amnesic Model:** Okadaic acid only
- **Treatment Groups:** Okadaic acid + Paradol at concentrations of 1, 3, and 6 µg/L

Cognitive performance was quantified through the Y-maze task to assess spatial memory and the Novel Object Recognition (NOR) test to evaluate recognition memory.

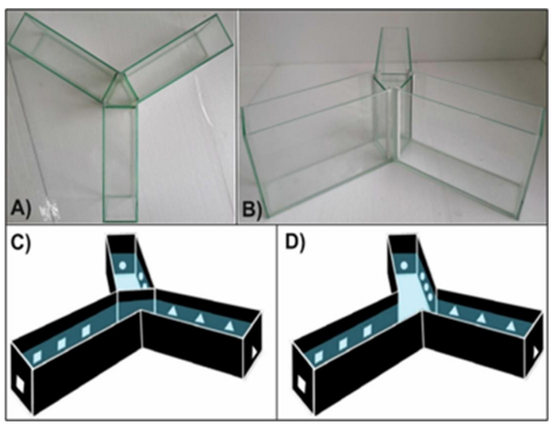

Figure 2. Y-maze experimental setup. Views include top (A) and side (B) perspectives. During the training phase, the novel arm was inaccessible via a sliding glass plate (C), while it remained open during the testing session (D). Spatial orientation was facilitated by visual cues positioned around each arm.

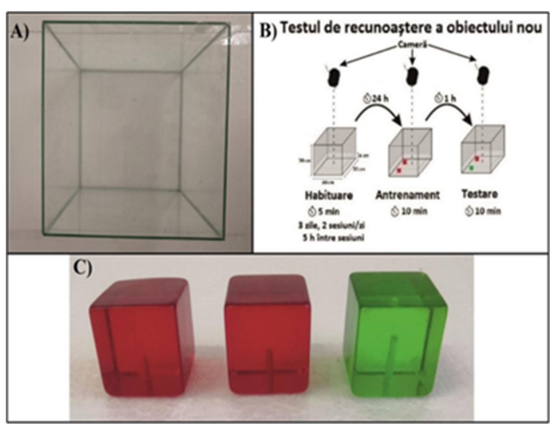

Figure 3. Novel Object Recognition (NOR). (A) Experimental aquarium utilized for the NOR procedure. (B) Procedural overview: zebrafish underwent a 3-day acclimation period (two sessions/day, 5-hour intervals). On the fourth day, a 10-minute training session was conducted using two familiar objects (F), followed by a 10-minute test session one hour later, where a familiar object was replaced by a novel stimulus (N). (C) Representative objects employed during the training and testing phases.

RESULTS & DISCUSSION

Figure 3. Adult *Danio rerio* used as the experimental model to study Alzheimer-like cognitive impairment.

Statistical analysis (One-way ANOVA, Tukey's post-hoc, $p < 0.05$) confirmed that okadaic acid induced significant cognitive impairment, evidenced by reduced exploration in the Y-maze and a lack of preference for novel objects in the NOR test. These deficits indicate a clear disruption in hippocampal-like processing and recognition memory. Conversely, treatment with Paradol at 3 and 6 µg/L significantly attenuated these impairments ($p < 0.0001$). Zebrafish demonstrated a robust recovery of cognitive function, with increased novel arm exploration and a higher discrimination index in the NOR test. These results suggest that Paradol effectively counteracts okadaic acid induced neurotoxicity, potentially by stabilizing synaptic plasticity and enhancing cholinergic neurotransmission, positioning it as a promising candidate for restorative therapy in Alzheimer-like models.

CONCLUSION

The findings demonstrate that Paradol serves as a potent cognitive enhancer in an amnesic zebrafish model. By significantly improving performance in memory-related tasks, Paradol presents a promising natural avenue for future therapeutic research regarding Alzheimer's disease and other neurodegenerative disorders.

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