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Protective Role of Mansonone G Against Alzheimer-Like Memory Impairment in *Danio rerio*

Isac Maria Crina [1]

[1] Alexandru Ioan Cuza University of Iași, Faculty of Biology, Department of Biology, 20A Carol I Boulevard, 700505 Iași, Romania

crina.isac@student.uaic.ro

INTRODUCTION & AIM

Research in applied biosciences emphasizes the identification of natural molecules and biological technologies with therapeutic potential for neurodegenerative disorders. Alzheimer's disease represents one of the most significant current challenges in biomolecular sciences due to its complexity and the limited efficacy of available treatments. Mansonone G, a bioactive compound isolated from *Mansonia gagei*, exhibits antioxidant and anti-inflammatory properties relevant to the development of neuroprotective strategies within an applied context. Evaluating this compound in a simple and efficient animal model, such as *Danio rerio*, aligns with the goals of the conference by exploring biological solutions applied to complex medical problems.

This study aims to determine the neuroprotective potential of Mansonone G in preventing or reducing cognitive deficits induced by okadaic acid in a zebrafish model frequently used in applied neuroscience research.

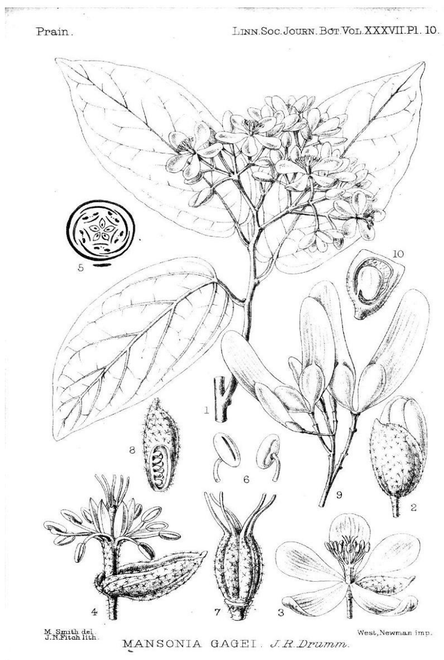

Figure 1. Source of Mansonone G: botanical illustration of *Mansonia gagei*, showing leaves, flowers, fruit, and seed. Original drawing by M. Smith, The Journal of the Linnean Society, Botany, vol. 37, 1904. Used under CC BY-NC-SA 3.0 via Useful Tropical Plants Database.

METHOD

The experimental model involved inducing an Alzheimer-like phenotype through exposure to okadaic acid, a compound known for its ability to disrupt essential neuronal processes. The zebrafish were divided into experimental groups treated with galantamine, OKA, or OKA combined with Mansonone G at three different concentrations. The behavioral tests used Y-maze and Novel Object Recognition assessed spatial memory, object recognition, and exploratory activity. Data analysis was conducted using statistical methods specific to applied biosciences, including multiple comparisons to determine significant differences between groups.

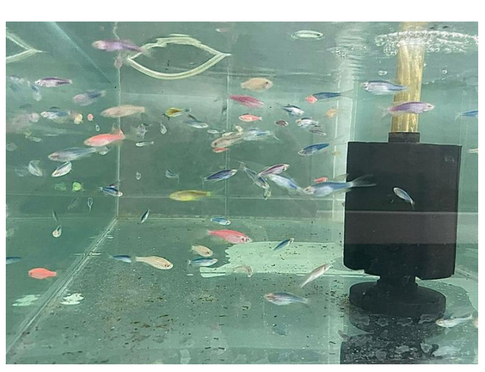

Figure 2. Adult *Danio rerio* used as the experimental model to study Alzheimer-like cognitive impairment.

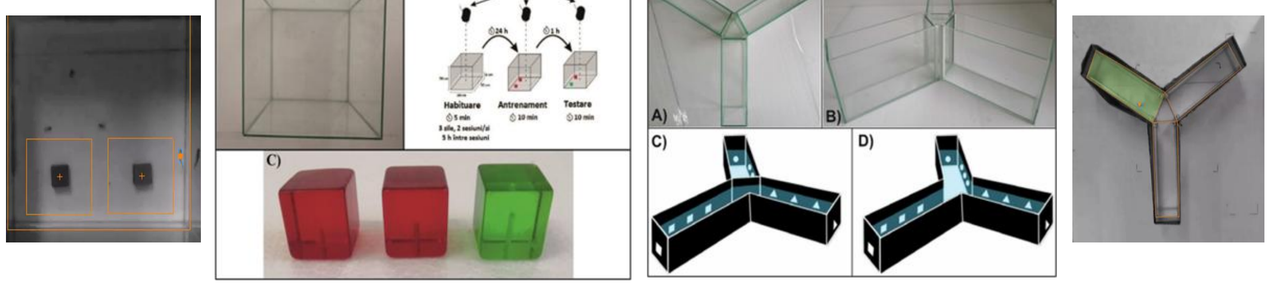

Figure 3. (A) Aquarium used for the Novel Object Recognition (NOR) test. (B) Experimental timeline: adult zebrafish were placed in the test aquarium for three consecutive days (two sessions per day, with a 5-hour interval between sessions). On the fourth day, a 10-minute training session was conducted with two familiar objects (F), followed one hour later by a 10-minute test session in which one familiar object was replaced by a novel object (N). (C) Objects employed during the training and test sessions.

Figure 4. Schematic illustration of the Y-maze used for behavioral testing. Top view (A) and side view (B) are shown. During the training session, the novel arm was blocked with a sliding glass plate (C), while in the testing session the novel arm was open (D). Visual cues were positioned on the sides and behind each arm to guide spatial navigation.

RESULTS & DISCUSSION

Exposure to OKA resulted in a clear deterioration of cognitive performance, confirming the functionality of the model. Galantamine administration restored cognitive parameters, providing a reference point for evaluating treatment response. Mansonone G at concentrations of 3 and 6 µg/L produced a notable improvement in spatial memory and recognition memory, as well as an increase in locomotor activity. The 1 µg/L concentration did not produce relevant changes. These results support the hypothesis that this natural compound has neuroprotective potential applicable to the development of new therapeutic approaches.

OKA exposure impaired memory and locomotor activity in *Danio rerio*, validating the AD-like model. Mansonone G at 3 and 6 µg/L improved cognitive performance and activity, indicating a dose-dependent neuroprotective effect. These results align with its known antioxidant and anti-inflammatory properties. The lowest dose (1 µg/L) had no significant effect, highlighting the importance of dosage. Overall, Mansonone G shows promise as a natural neuroprotective agent and warrants further preclinical investigation.

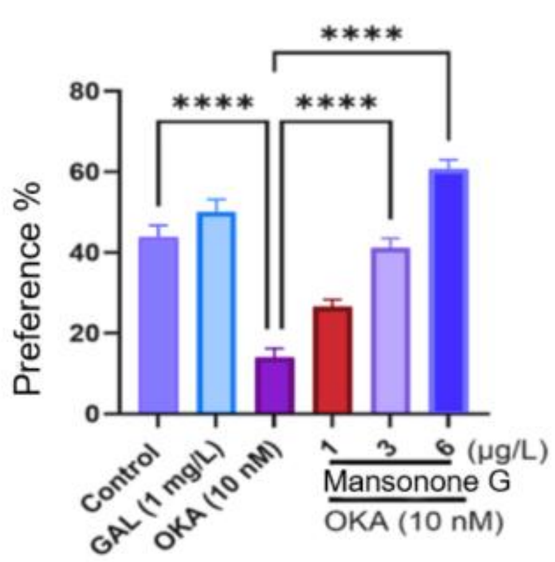

Figure 5. Effects of Mansonone G (1, 3, and 6 µg/L) on the percentage preference in the Novel Object Recognition (NOR) test in zebrafish treated with OKA (10 nM). Data are presented as mean ± SEM, with n = 10 animals per group. ****p < 0.00001 versus the OKA group.

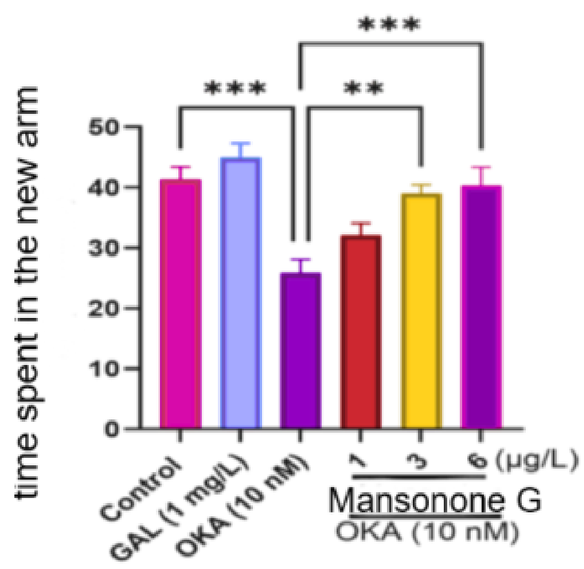

Figure 6. Effects of Mansonone G (1, 3, and 6 µg/L) on the time spent in the novel arm during the Y-maze test in zebrafish treated with OKA (10 nM). Data are presented as mean ± SEM, with n = 10 animals per group. **p < 0.001; ***p < 0.0001 compared to the OKA group.

CONCLUSION

Mansonone G demonstrates the ability to attenuate OKA-induced cognitive deficits in *Danio rerio*, supporting its exploration in applied research related to Alzheimer's disease therapy. Its positive impact on cognitive function highlights the relevance of natural compounds in applied biosciences and justifies further studies to clarify the mechanisms involved and extend research to higher-level models.

FUTURE WORK / REFERENCES

Liu Y. (2023). Zebrafish as a Model Organism for Studying Pathologic Mechanisms of Neurodegenerative Diseases and other Neural Disorders. Cellular and molecular neurobiology, 43(6), 2603–2620.

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