ABSTRACTS

Wpływy oddziaływań antropogenicznych na emisje lotnych związków organicznych z osadów dennych oraz strefy wymiany hydrosfera – osad denny czterech jezior Pomorza

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Przeprowadzono badania stężeń lotnych związków organicznych (LZO) w próbkach osadów dennych czterech jezior Pomorza (północna Polska). W trzech spośród badanych jezior: Jeleń, Jezioro Miejskie i jezioro Rychnowskie badano osady denne pobierane za pomocą czerpaka Eckmana z głębszych obszarów. Badane jeziora różnią się stopniem zantropogenizowania, a także wpływem zlewni leśnej. Czwartym jeziorem było jezioro Lubowidz, w obszarze którego znajdują się trzy plaże, w tym jedna intensywnie użytkowana w sezonie letnim. Do analiz w tym jeziorze wybrano próbki osadów dennych z obszaru plaż. Na wybranych stanowiskach badanych jezior pobrano próbki wody naddennej. W pozyskanych próbkach osadów dennych oraz próbek wody naddennej badano stężenie LZO za pomocą spektrometru masowego z reakcją przeniesienia protonu (PTR-MS). Technika ta umożliwia badanie emitowanych LZO z wysoką czułością i w szerokim zakresie m/z LZO. Przeprowadzone badania wskazują na wpływy antropogeniczne prawdopodobnie związane z zanieczyszczeniami pochodzącymi z graniczącym z Jeziorem Miejskim miastem Człuchów, na emisję części LZO z osadów dennych. W jeziorze Jeleń zaobserwowano wpływy zlewni leśnej. W jeziorze Lubowidz zaobserwowano prawdopodobne wpływy antropopresji na emitowane LZO w obszarze kąpieliska o intensywnym użytkowaniu przez plażowiczów.

Aflatoxin B1-induced disruption of nucleic acid homeostasis in fish tissues

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Mycotoxin contamination of feed and aquatic environments is a growing global problem in aquaculture. Aflatoxin B1, produced by fungi of the genus *Aspergillus*, is

considered one of the most hazardous mycotoxins due to its hepatotoxic, mutagenic, and carcinogenic properties. It can enter aquatic ecosystems through contaminated feed, leading to chronic exposure of fish species. Studies have shown that dietary aflatoxin B1 significantly reduces growth performance and disrupts metabolic processes in fish (Barany et al., 2021). Multi-mycotoxin contamination further increases toxicity and ecological risks in aquaculture systems (Gruber-Dorninger et al., 2025). At the cellular level, aflatoxin exposure is associated with oxidative stress and molecular damage, including nucleic acid degradation (El-Gendy et al., 2020; Guindon-Kezis & Mulder Massey, 2014). Therefore, understanding its impact on nucleic acid homeostasis is crucial for ecological safety assessment.

The study is based on analysis of experimental and literature data concerning the effects of aflatoxin B1 on aquatic organisms. The main biological models include fish species used in aquaculture research, where biochemical and molecular indicators of toxicity were assessed. Nucleic acid content (DNA and RNA) and their ratio were considered key indicators of cellular metabolic activity (Afanasieva & Chopei, 2024). Enzymatic activity of nucleases was used as an indicator of nucleic acid degradation processes. Molecular biology approaches were considered for interpreting cellular responses to toxic exposure (Afanasieva & Chopei, 2024). Data on oxidative stress, DNA damage, and metabolic disruption were analyzed based on previously published experimental findings (El-Gendy et al., 2020; Guindon-Kezis & Mulder Massey, 2014). Statistical evaluation of biological effects was interpreted using modern ecological and biomedical research approaches (Lukash et al., 2026).

The analysis demonstrated that aflatoxin B1 causes significant disruption of nucleic acid homeostasis in fish tissues. Exposure to this mycotoxin leads to a reduction in RNA levels and alterations in DNA/RNA ratios, indicating suppression of protein synthesis and metabolic activity. These changes reflect impaired cellular functioning and reduced physiological performance in fish organisms.

The toxic effects of aflatoxin B1 are strongly associated with oxidative stress, which triggers DNA damage and activation of repair mechanisms (Guindon-Kezis & Mulder Massey, 2014). In aquatic organisms, oxidative stress leads to lipid peroxidation and degradation of cellular components, including nucleic acids (El-Gendy et al., 2020). As a result, enzymatic degradation of DNA and RNA increases, contributing to cellular dysfunction.

Fish exposed to aflatoxin B1 demonstrate reduced growth performance and impaired metabolic regulation, as reported in aquaculture studies (Barany et al., 2021; Fornari et al., 2023). Liver tissue is the primary target organ due to its central role in detoxification and metabolic processes. Prolonged exposure results in structural and functional tissue damage.

Multi-mycotoxin contamination in aquaculture feed enhances toxic effects and increases ecological risks (Gruber-Dorninger et al., 2025). Combined exposure may intensify oxidative stress and accelerate nucleic acid degradation. Nucleic acid metabolism is therefore considered a sensitive indicator of early toxic effects in hydrobionts.

Modern molecular biology methods provide important tools for studying these processes at the cellular level (Afanasieva & Chopei, 2024). Disruption of nucleic

acid homeostasis reflects deep molecular damage and serves as an early biomarker of toxicity. Statistical approaches such as MANOVA improve reliability and interpretation of ecological and biomedical data (Lukash et al., 2026). Overall, aflatoxin B1 exerts a strong negative effect on fish physiology through oxidative and molecular mechanisms.

Aflatoxin B1 is a highly toxic mycotoxin affecting aquatic organisms at molecular and biochemical levels. It disrupts nucleic acid homeostasis in fish tissues by reducing RNA levels and altering DNA/RNA balance. Oxidative stress is the main mechanism of its toxic action. DNA damage and activation of repair systems are key cellular responses to exposure. Fish growth and metabolic activity are significantly impaired under aflatoxin B1 influence. Liver tissue is the primary target of toxic damage. Multi-mycotoxin contamination increases ecological risks in aquaculture systems. Nucleic acid metabolism is a sensitive biomarker of toxic exposure in hydrobionts. Molecular biology methods enhance understanding of toxicity mechanisms. Further research is needed to assess long-term and combined effects of mycotoxins in aquatic ecosystems.

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