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PP-099 [Bee Health]

Brain Fatty Acid Profile Alterations in Honey Bees Induced by Neonicotinoid Acetamiprid

Fanni Huber [1]*, Zsuzsanna Neogrády [1], Gábor Mátis [1,2], Evelin Kámán-Tóth[1], Hedvig Fébel [3], Máté Mackei [1,2]

[1] Division of Biochemistry, Department of Physiology and Biochemistry, University of Veterinary Medicine Budapest, István Street 2, H-1078 Budapest, Hungary
[2]National Laboratory of Infectious Animal Diseases, Antimicrobial Resistance, Veterinary Public Health and Food Chain Safety, University of Veterinary Medicine Budapest, István Street 2, H-1078, Budapest, Hungary
[3] Department of Obstetrics and Farm Animal Medicine Clinic, University of Veterinary Medicine Budapest, István Street 2, H-1078, Budapest, Hungary

INTRODUCTION

- Neonicotinoids pose a serious threat to pollinators such as the **Western honey bee** (*Apis mellifera*), which play a crucial role in ecosystem stability.
- Although lethal effects are infrequent, prolonged exposure to **sublethal doses** can adversely affect bee physiology and behavior.
- The biochemical pathways underlying such effects remain **poorly understood**
- Considering the **critical role of lipid metabolism in neural function**, this study explores how low-level acetamiprid exposure modifies the fatty acid composition in honey bee brains.
- **Acetamiprid**, widely applied in agriculture, may interfere not only with neural receptors but also **trigger oxidative stress and disrupt lipid metabolic processes**.

MATERIALS AND METHODS

- Field experiments were conducted at an apiary located in Ugod, Hungary.
- Honey bees were collected and maintained under strict laboratory conditions (30°C, 90% relative humidity) within specialized cages.
- The test groups were exposed to varying **sublethal concentrations of acetamiprid** (LD50/10, LD50/20, LD50/40 per bee per day) administered **orally** over a **48-hour** period.
- Post-exposure, brain tissues were dissected, homogenized, and subjected to **gas chromatography-mass spectrometry** (GC-MS) to quantify fatty acid profiles.

RESULTS

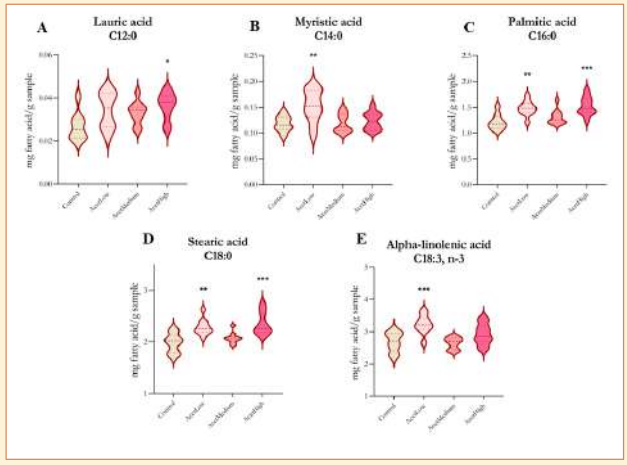
- Exposure to sublethal doses of acetamiprid induced marked alterations in the levels of specific fatty acids including **lauric** (C12:0), **myristic** (C14:0), **palmitic** (C16:0), stearic (C18:0), and **alpha-linolenic acid** (C18:3, n-3). **Total, saturated, and polyunsaturated fatty acid concentrations** were also significantly affected.

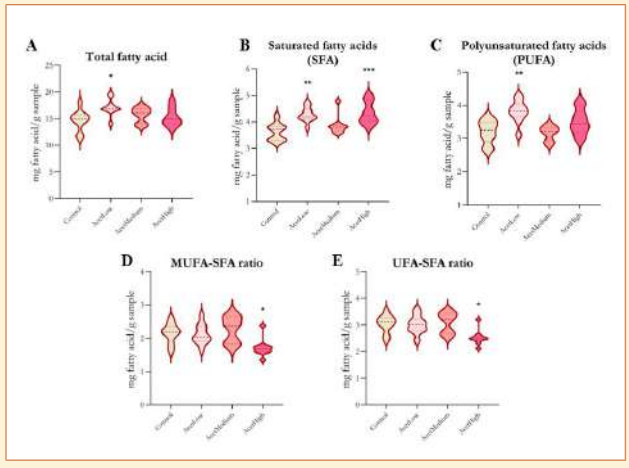
CONCLUSION

- These biochemical changes have **potential ramifications on neural integrity and colony health**, suggesting that even low-level pesticide exposure may impair bee populations.
- The findings underscore the importance for beekeepers to consider these subtle yet critical risks in agricultural landscapes. Further studies are warranted to elucidate **long-term ecological impacts and develop mitigation strategies** for pesticide use in apiculture.

AIM OF THE STUDY

INVESTIGATING THE EFFECTS OF ACETAMIPRID IN THE ALTERATIONS OF FATTY ACID STATUS OF HONEY BEE'S CENTRAL NERVOUS SYSTEM





The results were visualized using violin plots. AcetLow 9,09 µg/ml feeding solution, AcetMedium 18,17 µg/ml feeding solution, AcetHigh 36,33 µg/ml feeding solution, $p<0.05$

ACKNOWLEDGEMENTS
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[3]Department of Obstetrics and Farm Animal Medicine Clinic, University of Veterinary Medicine, 1078 Budapest, Hungary

Background/Purpose
Neonicotinoids pose a serious threat to pollinators like the Western honey bee (Apis mellifera), which plays a crucial role in ecosystem stability. While acute toxicity is less common, sublethal exposure may significantly impact bee physiology and behavior. Despite growing evidence of neonicotinoid-induced effects, the biochemical mechanisms remain unclear. Given the importance of lipid metabolism in neural function, we investigated how sublethal acetamiprid exposure alters the honey bee brain's fatty acid profile.

Problem Description
Global bee population declines are attributed to multiple stressors, including pesticides. Acetamiprid, a commonly used neonicotinoid, may not only interact with neural receptors but also induce oxidative stress and disrupt lipid metabolism. However, the extent of these effects on the honey bee brain remains insufficiently studied.

Methodology
Experiments were conducted at an apiary in Ugod, Hungary. Honey bees were collected and housed in controlled laboratory conditions (30°C, 90% humidity) in specialized cages. Experimental groups received varying acetamiprid doses (LD50/10, LD50/20, LD50/40 per bee per day) ad libitum for 48 hours. After treatment, bees were euthanized with dry ice and stored at -80°C. Their central nervous systems were dissected, homogenized, and analyzed via gas chromatography-mass spectrometry (GC-MS) to determine fatty acid composition.

Results and CONCLUSION: Acetamiprid exposure significantly altered brain fatty acid metabolism. Lauric acid (C12:0) and myristic acid (C14:0), markers of oxidative stress, increased. Additionally, linoleic acid (C18:2, n-9,12) and alpha-linolenic acid (C18:3, n-3; ALA), associated with neurodegeneration, were elevated. Stearic acid (C18:0) and palmitic acid (C16:0) also increased, along with shifts in total saturated and total fatty acid levels. Our findings indicate that acetamiprid disrupts lipid homeostasis in the bee brain, potentially impairing neural function.

Practical Implications for Beekeepers
Sublethal acetamiprid exposure, even at low doses, may contribute to colony health issues by affecting neural function. Beekeepers should consider these risks when managing hives in agricultural environments. Further research is needed to assess long-term ecological consequences and mitigate pesticide-related harm in apiculture.