Physiological and Behavioral Impacts of a Neonicotinoid Insecticide and Its Transformation Product on a Non-Target Keystone Species

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Imidacloprid (IMI) is the most utilized insecticide and an emerging contaminant. IMI acts as a nicotinic acetylcholine receptor (nAChR) agonist. 6-chloronicotinic acid (6-CNA) is a photodegradation product of IMI that is found in the environment. The microscale ecological consequences of environmental contamination by IMI and transformation product 6-CAN are not understood. Thus, these compounds are not subject to current regulation in the U.S. Objectives of this study were to determine the toxicity of IMI and 6-CNA in the non-target keystone species, Daphnia pulex, using novel optical physiological and behavioral tracking systems, and to elucidate the cholinergic mechanisms of the Daphnia central nervous system. Physiological assays were performed through quantification of key parameters heart rate (HR) and appendage beat rate (ABR). Behavior was assessed through quantification of mean accumulative distance and angular change. 256 microM IMI decreased ABR by over 50% ($P<0.05$) within 60 minutes. 6-CNA did not affect HR or ABR (60 minutes). Swimming behavior was significantly stimulated by IMI exposure, and elicited a significant, concentration-dependent increase in accumulated distance traveled ($P<0.05$) and decrease in mean angle. These effects were blocked by 50 microM MCA ($P<0.05$) indicating that IMI affects motor function via nAChRs. 6-CNA did not elicit significant effects on behavior, but in contrast to IMI, 100% lethality from 24-hour exposure to 256 microM occurred. Results indicate that short and longterm exposure to IMI and 6-CNA can negatively affect non-target species through the promotion of maladaptive physiological and behavioral responses. IMI and 6-CNA are significant stressors and may adversely impact aquatic ecosystems through foodweb disruption.

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