A neonicotinoid pesticide affects the firing properties of a looming-sensitive neuron in *Locusta migratoria*

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1. INTRODUCTION

Since their introduction in the 1990s, neonicotinoids have been commercialized as a miracle pesticide with low toxicity to mammals, low risk of bioaccumulation, high toxicity to target insects, and convenient function as systemic pesticides. Recently neonicotinoids have been receiving negative attention due to their effects on non-target organisms, especially birds and bees. Neonicotinoids are nicotine mimics, and act as agonists to nicotinic acetylcholine receptors (nAChRs) present on insect neurons.

The locust (*Locusta migratoria*) is one of the most devastating agricultural pests due to its ability to form high-density, mobile swarms. While the locust is not a typical target organism for neonicotinoid pesticides, it is a model organism in neuroethology. Two widely studied pairs of neurons, which code visual information from each of the locust’s eyes and synapse downstream with muscles involved with flight and jumping, are especially sensitive to looming stimuli. Each lobula giant movement detector (LGMD) receives visual information from the sensory cells of the ommatidia, and synapses directly with the descending contralateral movement detector (DCMD) at a one-to-one ratio. The present study aimed to determine if the neonicotinoid imidacloprid has an effect on the response of the DCMD to a looming stimulus. If imidacloprid binds to the DCMD or other upstream neurons, then the firing rate and other response parameters may be altered.

2. METHODS

![Diagram of experimental setup](image)

Rigid tether

Single hook electrode

A: Maximum firing rate per approach, normalized as a percent of the mean of T01, versus approach time. Bimodal responses occurred (means plotted). Half of animals experienced a sharp decrease in firing rate within 10 minutes of injection of imidacloprid, which then recovered to near or slightly lower than pre-injection rates (top). Means of each group in bold. Significance denoted by alternate letter (bottom).

C: Number of spikes (normalized) versus approach time for all animals, and mean (top). Similar bimodal response types observed, but with increased variability between treatment groups (bottom).

3. RAW TRACES & PERISTIMULUS TIME HISTOGRAMS

A: Raw trace and peristimulus time histogram (PSTH) for a control loom. Maximum firing rate, peak time, peak width at half height (Pw½h), and decay phase marked.

B: Raw traces and PSTH overlays for three phases of imidacloprid effect: pre-response (resembles control), hyperreception (sporadic firing), and recovery. Not pictured: no response phase (inactivity of DCMD).

C: PSTH overlays of one recovery phase loom from each animal (n=23) and all control looms (T01) (n=96). Recovery phase characterized by a long decay, lower peak, and slightly later peak time for the recovery looms.

4. FIRING PROPERTIES

A: Maximum firing rate per approach, normalized as a percent of the mean of T01, versus approach time. Bimodal responses occurred (means plotted). Half of animals experienced a sharp decrease in firing rate within 10 minutes of injection of imidacloprid, which then recovered to near or slightly lower than pre-injection rates (top). Means of each group in bold. Significance denoted by alternate letter (bottom).

B: Number of spikes (normalized) versus approach time for all animals, and mean (top). Similar bimodal response types observed, but with increased variability between treatment groups (bottom).

C: Peak time in relation to TOC as a function of approach time for all animals, plus mean. Throughout T04 peak time approaches TOC, with the median of T04 significantly later than all other groups (bottom).

5. HISTOGRAM SHAPE PROPERTIES

A: Pw½h for all animals, normalized as a percent of the mean of T01, versus approach time, and means (top). T04 responders display a decrease in Pw½h within 10 minutes of injection, followed by a partial or full recovery. Significance of denoted by alternate letter (bottom).

B: Decay phase for all animals versus approach time, and mean (top). Throughout T04 the decay phase lengths for all animals (bottom).

6. SUMMARY

- Imidacloprid alters the response of the DCMD to looming stimuli
- Agonizes nAChRs on the DCMD or upstream neurons (LGMD, sensory cells)
- Bimodal response types:
  - Responders: four phases of imidacloprid effect
  - Non-responders: pre-response and recovery phases only
- Phases of imidaclopid effect:
  - Pre-response: resembles control approaches, characterized by a short decay phase, and maximum firing rate ~ 0.03s before TOC
  - Hyperreception: high frequency, tonic firing of DCMD, not in response to stimulus
  - No response: DCMD ceases firing for a period
  - Recovery: DCMD responds to stimulus, but PSTH characterized by long decay phase, later peak time, and lower maximum rate.
- Long decay phase suggests imidacloprid alters inhibitory network.
- Future research:
  - Rising phase of PSTH (appears to shorten in recovery phase)
  - Longer-term effects of imidacloprid and metabolites

7. ACKNOWLEDGEMENTS & REFERENCES

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References