

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

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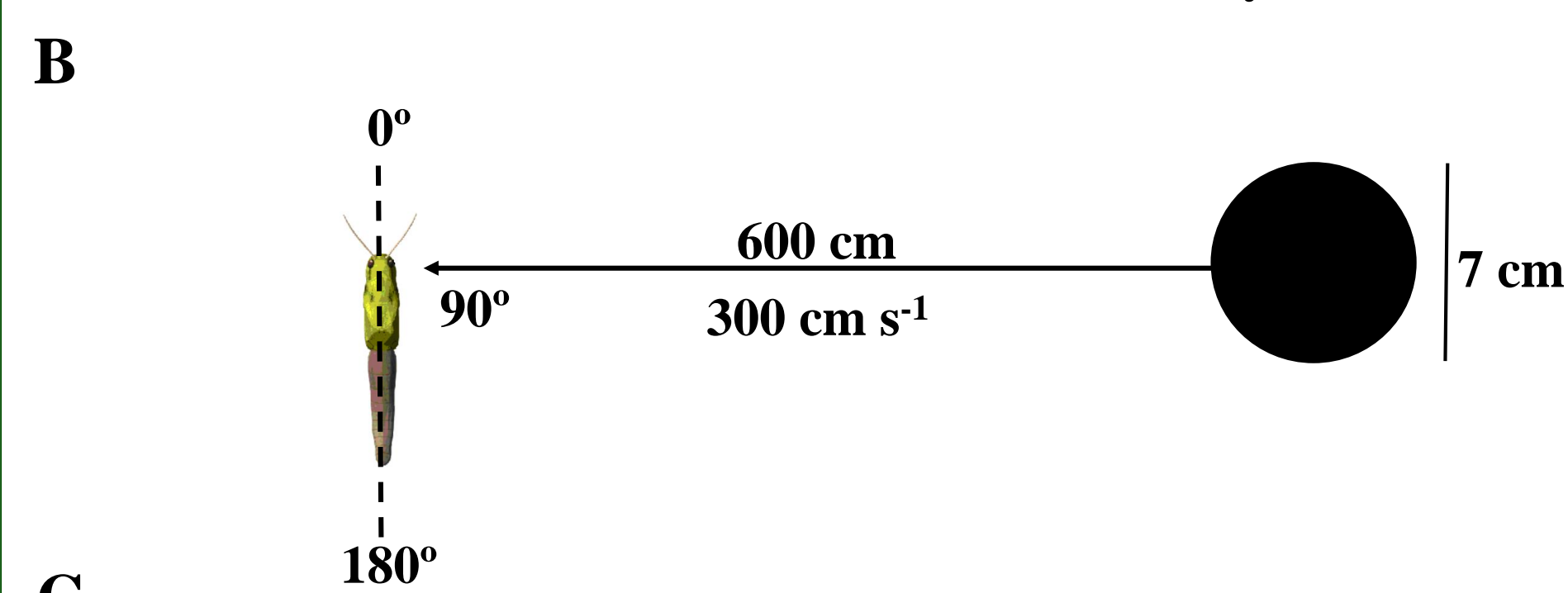
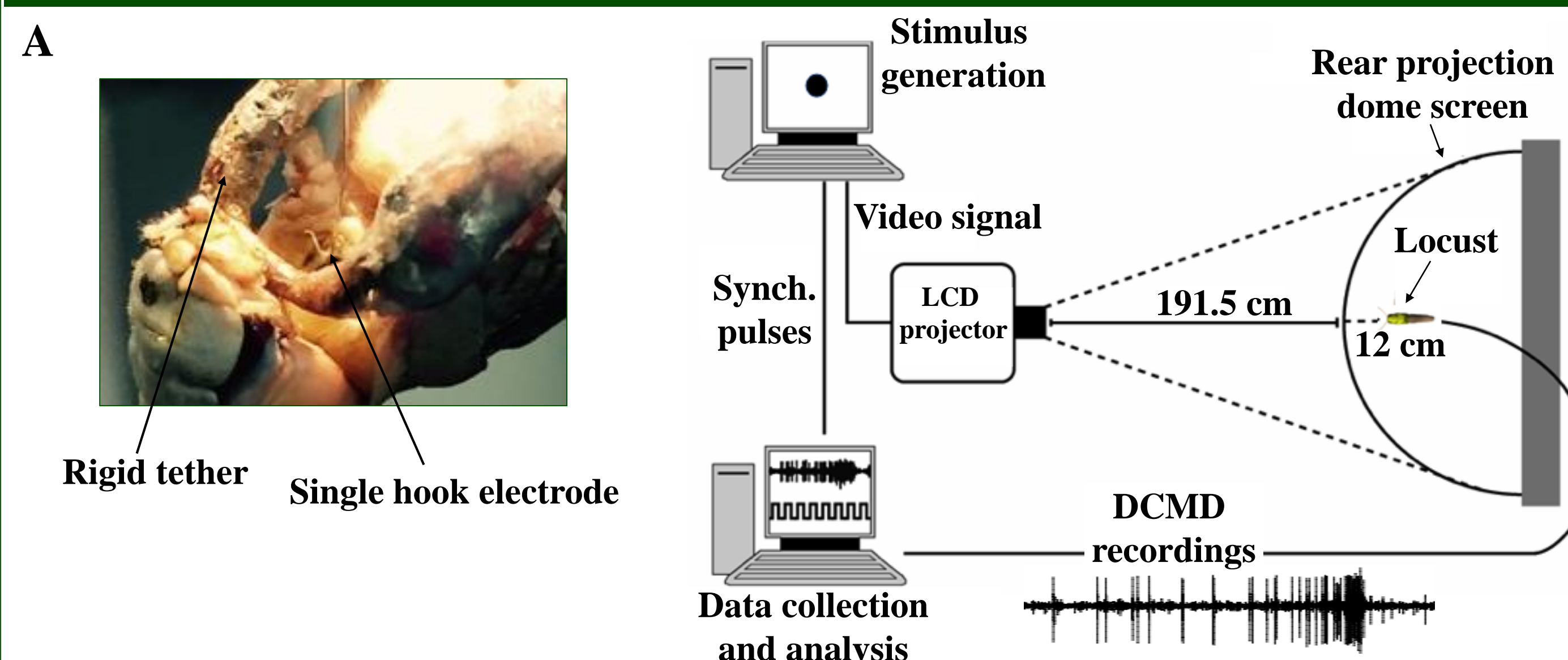
1. INTRODUCTION

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.¹ Recently neonicotinoids have been receiving negative attention due to their effects on non-target organisms, especially birds and bees.² Neonicotinoids are nicotine mimics, and act as agonists to nicotinic acetylcholine receptors (nAChRs) present on insect neurons.³

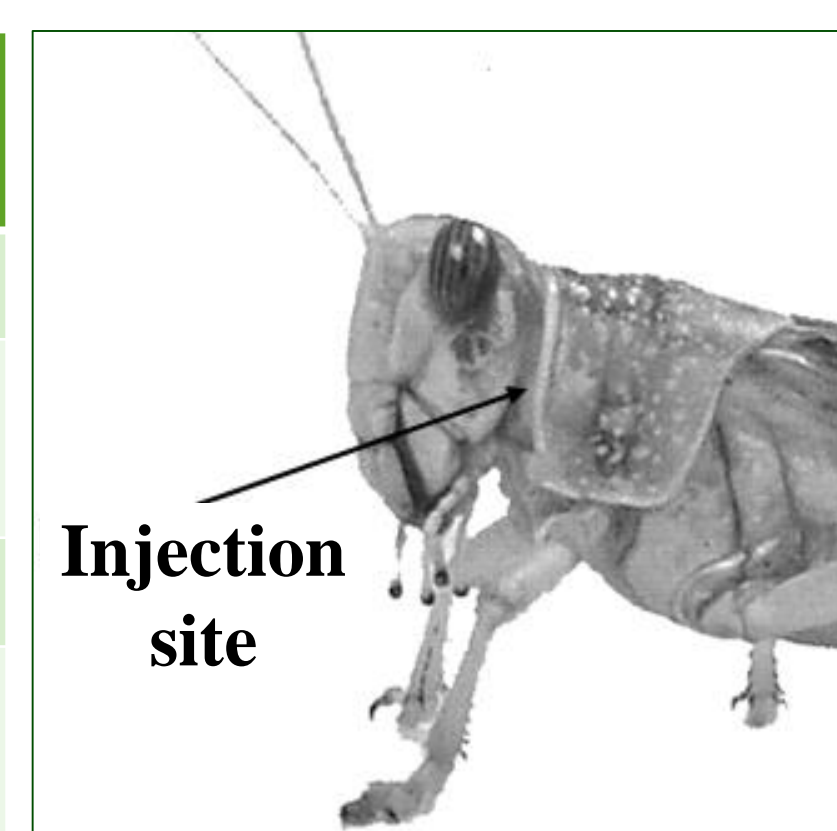
The locust (*Locusta migratoria*) is one of the most devastating agricultural pests due to its ability to form high-density, mobile swarms.⁴ While the locust is not a typical target organism for neonicotinoid pesticides, it is a model organism in neuroethology. Two widely studied pairs of neurons, which 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.⁵⁻⁷ 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.⁸

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.

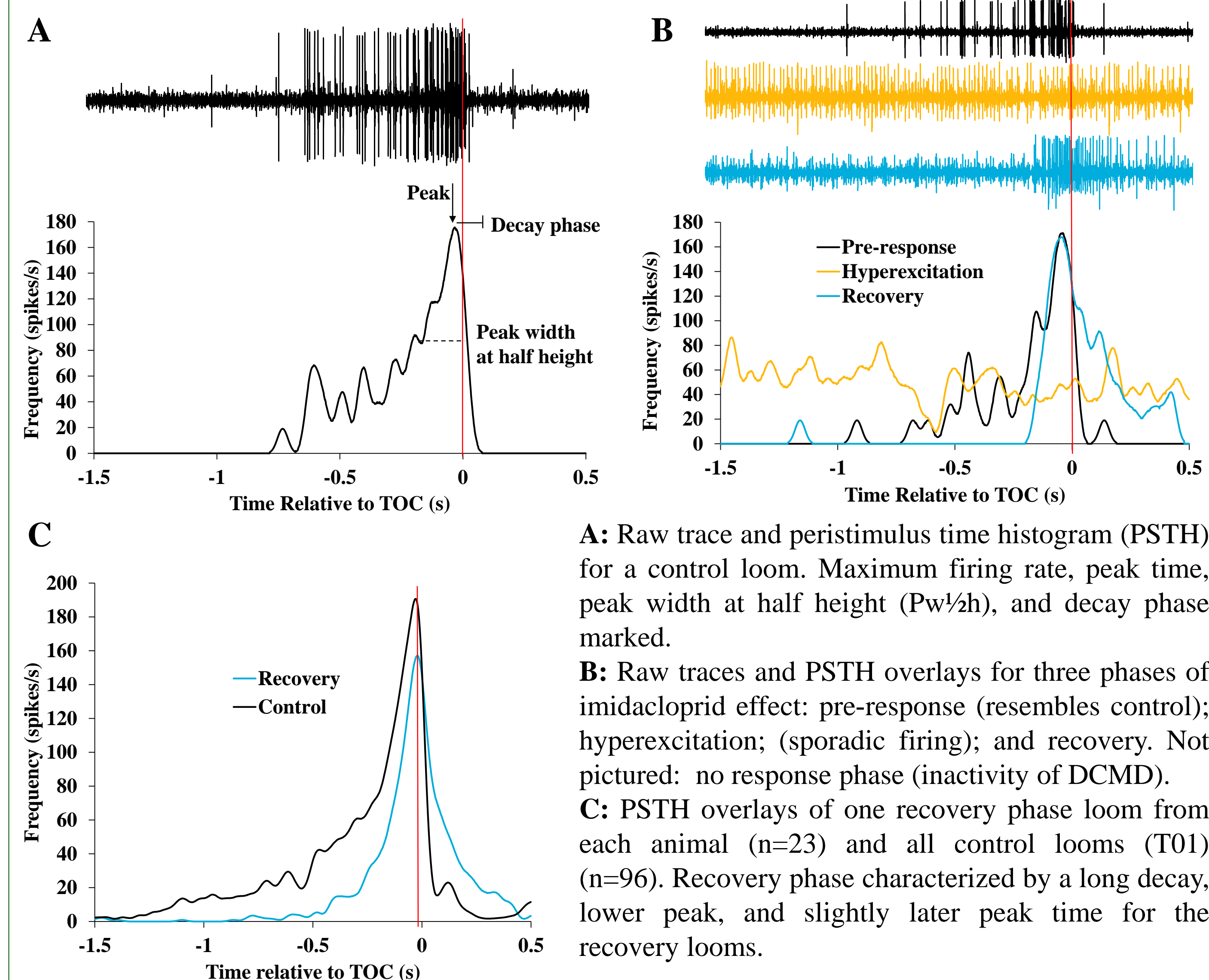
2. METHODS



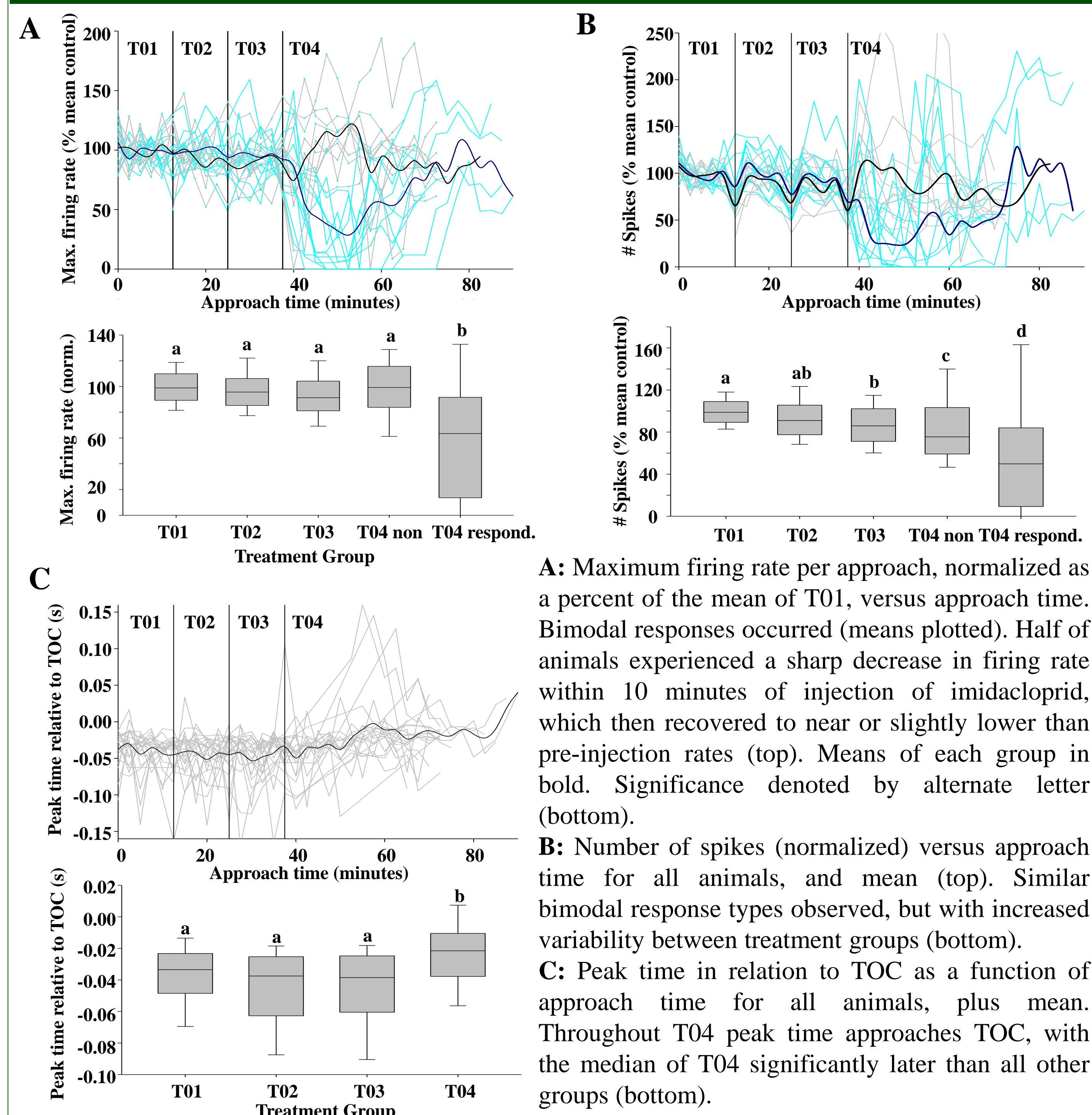
Treatment	Description	# approaches (2.5 min apart)
T01	After 20 minutes acclimation	5 (0-10 min)
T02	After piercing cuticle with microsyringe	5 (12.5-22.5 min)
T03	After injecting 200 µl saline	5 (25-35 min)
T04	After injecting 200 ng imidacloprid in 200 µl saline	15 to 20 (37.5 min onwards)



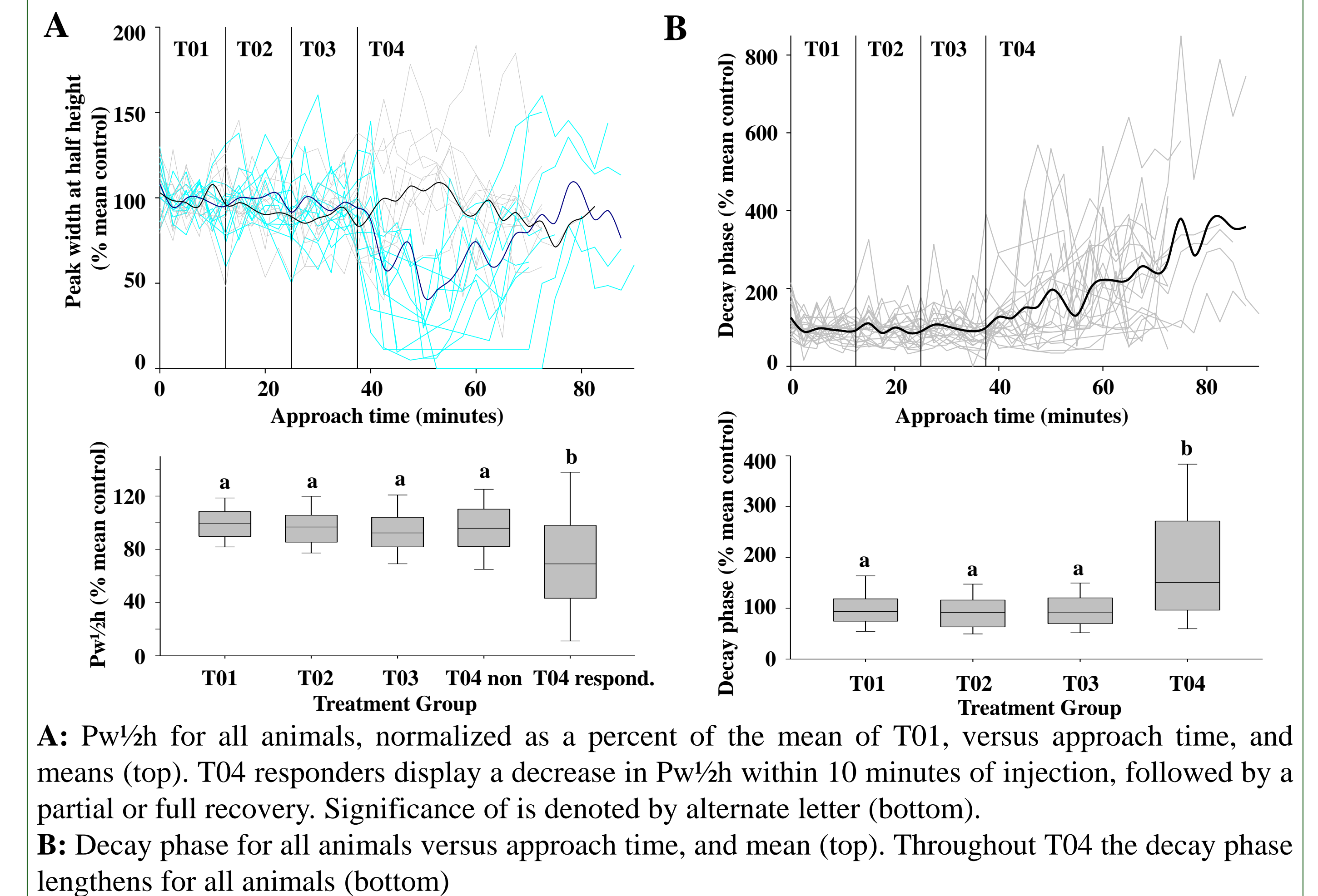
3. RAW TRACES & PERISTIMULUS TIME HISTOGRAMS



4. FIRING PROPERTIES



5. HISTOGRAM SHAPE PROPERTIES



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 imidacloprid effect:
 - **Pre-response:** resembles control approaches, characterized by a short decay phase, and maximum firing rate ~ 0.03s before TOC
 - **Hyperexcitation:** 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

Acknowledgements

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References

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