

ASSESSMENT OF VENOM TOXICITY USING *DROSOPHILA MELANOGASTER* AS A MODEL ORGANISM: A REVIEW

Akanksha V. Joshi*, Dr. Mrunal Ghag Sawant, Sanika N. Parkar

Department of Zoonosis and Toxicology, Haffkine Institute for Training, Research & Testing, Parel, Mumbai,
Maharashtra 400012, India.



***Corresponding Author: Akanksha V. Joshi**

Department of Zoonosis and Toxicology, Haffkine Institute for Training, Research & Testing, Parel, Mumbai,
Maharashtra 400012, India.

DOI: <https://doi.org/10.5281/zenodo.19883650>

How to cite this Article: Akanksha V. Joshi*, Dr. Mrunal Ghag Sawant, Sanika N. Parkar (2026). Assessment Of Venom Toxicity Using *Drosophila Melanogaster* As A Model Organism: A Review. *European Journal of Pharmaceutical and Medical Research*, 13(5), 59–64.

This work is licensed under Creative Commons Attribution 4.0 International license.



Article Received on 26/03/2026

Article Revised on 15/04/2026

Article Published on 01/05/2026

ABSTRACT

Drosophila has been a very easy to culture model organism with advantages like its short life cycle and abundant progeny. Various studies have evaluated venom's toxicological effects on *Drosophila* as a model organism. *Drosophila* is used as a powerful genetic tool and to study different components of the signalling pathways in humans. Venom consists of different biologically active compounds like proteins, enzymes and peptides. Venom shows wide range of neurotoxic effects which may include symptoms like weakness, nausea, headache, confusion, and drowsiness, along with muscle-related problems such as pain, cramps with muscle twitching (fasciculations), and difficulty in breathing. As well as hemotoxic effects such severe disruptions in blood coagulation, tissue damage, and cardiovascular failure in most of the vertebrates. This review aims to understand toxicological effects in *Drosophila* by using various venoms of different organisms such as Snake, Scorpion, Bee, Spider, Wasps and other marine organisms.

KEYWORDS: toxicological effects, venoms, *Drosophila*.

INTRODUCTION

Drosophila melanogaster is widely used as a model organism in biological research because of its short life cycle, high reproductive capacity, and ease of maintenance in laboratory conditions. Many fundamental cellular and metabolic pathways are conserved between the two organisms, and a large proportion of genes related to human neurological functions have orthologs in *Drosophila*. Approximately 60% of the *Drosophila* genome shows homology with the human genome, and nearly 75% of genes associated with human diseases have functional counterparts in the fly. Although *Drosophila melanogaster*, commonly known as the fruit fly, and humans have followed different evolutionary paths, they share a considerable degree of genetic and cellular similarity. Because of these similarities, *Drosophila melanogaster* serves as a valuable model for studying various neurological and brain-related disorders. Its observable behavioural patterns and well-characterized genetics make it an effective system for investigating the mechanisms underlying neurological diseases in mammals, particularly humans.^[1]

The Venom studies have shown changes in movement, heat avoidance and changes in Circadian Rhythm in *Drosophila Melanogaster*. The neurotoxicity in this model system was observed by symptoms like paralysis or aberrant behaviour. When administered orally some marine venoms such as *P. physalis* venom show mortality in dose-dependent manner. Flies also show uncoordinated movement and disorientation at sub-lethal doses of *P. physalis* venom.^[2] Other symptoms like increased food intake were also observed in flies when administered with the venom of a marine predatory snail. The antinociceptive properties of venoms help in investigating the importance of ion channels.^[3] Different venoms show a wide range of effects in *Drosophila melanogaster*. In this study we investigate and review toxicological effects of wide variety of venoms in detail.

Toxicological impacts associated with various venoms Snake venom (*Naja Naja*) venom

Venom of the *Naja Naja* is a complex mixture of toxins mainly made up of non-enzymatic proteins (about 71–75%) and enzymes (about 25–29%). These components

produce strong neurotoxic, cytotoxic, and cardiotoxic effects, which can quickly lead to neuromuscular blockage.^[4]

The study shows that (*Naja naja*) venom improves cognitive behaviour in *Drosophila Melanogaster* as a model organism.^[5] Another study reported that treatment with snake venom helped restore normal behaviour in mutant flies. The treatment also reduced reactive oxygen species (ROS) levels and showed potential in inhibiting carcinogenesis. In contrast, when NDEA was administered to mutant flies, it induced significant oxidative stress, as this compound is known for its carcinogenic properties. The fly carcinogenesis may have been affected the cognitive behaviour too. Snake venom appears to counteract the damaging effects of NDEA, helping to restore the flies' behaviour to a state closer to normal. Snake venom contains components that can slow down the growth of cells and trigger cell death. Because of these properties, it has been studied for its potential role in controlling tumour growth in cancer cells. Venom can act in several ways, such as directly damaging cancer cells and producing free radicals that increase cellular stress. It can also block nucleic acid synthesis, which prevents cells from multiplying. In addition, snake venom may reduce the activity of matrix metalloproteinases, enzymes that help cancer cells spread, and inhibit integrins, which further limits cancer cell migration and invasion. Some venom components also show anti-angiogenic effects, meaning they can prevent the formation of new blood vessels that tumours need to grow and spread. The study looked at how the fruit fly *Drosophila melanogaster* responds to different environmental stimuli such as light, smell, taste, humidity, temperature, and gravity. The results showed that these behaviours can be observed and measured within a short time, about one minute. The chemicals tested in the study (NNV, CA, and PCA) may cause oxidative stress in flies, but they may also provide protection by neutralizing harmful free radicals and helping restore the normal balance inside cells.^[6] Since many biological processes in fruit flies are similar to those in humans, these findings may help scientists better understand cellular stress and biological rhythms in humans.

Overall *Naja Naja* Venom shows positive effects on *Drosophila Melanogaster* as a model organism like improving behavioural parameters and improving cognition. Counteracting the oxidative stress and neurotoxic effects caused by the carcinogens.^[7]

Honey bee venom

The venom of the honeybee (*Apis mellifera*) is produced in specialized venom glands and stored in a venom sac until it is used during a sting. When a bee stings, its barbed stinger becomes embedded in the skin, and venom is injected into the tissue through rhythmic muscle contractions that are controlled by a nerve ganglion located at the end of the abdomen. The venom itself is a

clear, acidic liquid with a pH of about 4.5–5.5 and has no noticeable colour or smell. It contains a variety of biologically active components, including peptides such as melittin and apamin, enzymes like phospholipases and hyaluronidase, and other substances such as biogenic amines, amino acids, sugars, minerals, and volatile compounds.^[8]

In the study, honeybee *A. mellifera* venom was administered in medium in which *Drosophila* thoracic muscle or CNS tissues are incubated and found out that there were changes in Physiological, Structural and biochemical parameters in the organs. Treatment with honeybee (*Apis mellifera*) venom was found to reduce adipokinetic hormone levels in the central nervous system (CNS) of *Drosophila*. The venom also caused several metabolic changes, including an increase in overall metabolic activity. This was indicated by higher cell viability in the examined tissues and elevated citrate synthase activity in thoracic cells. In addition, envenomation influenced the oxidative stress response in the flies. The activity of important antioxidant enzymes, superoxide dismutase (SOD) and catalase (CAT), was reduced in both the thoracic muscle and CNS tissues. Studies have shown that honeybee venom can damage the ultrastructure of muscle cells in *Drosophila*, particularly disrupting the normal organization of myofibrils. It also caused an increase in the levels of ARK released into the incubation medium. However, when Drome-AKH was administered together with the venom, many of these effects were reduced or modified.^[8]

In another study, researchers expressed the honeybee venom peptide apamin in *Drosophila* and discovered that it showed unexpected antimicrobial activity. Further investigation revealed that this activity was mainly concentrated in the gut. The effect was associated with increased proliferation of intestinal stem cells, acidification of the midgut environment, and activation of calcium signalling in enteroendocrine cells. When the influence of apamin on the nervous system was examined, neuronal expression of membrane-bound apamin was found to slightly affect locomotion and sleep behaviour. Additional studies involving *Drosophila* larvae examined different fractions of honeybee venom and identified two polypeptides, Minimine and melittin, that produced distinct physiological effects. Third-instar larvae exposed to the LD50 dose of Minimine (0.095 µg) survived but stopped feeding and growing and appeared lethargic, although they were not paralyzed. These larvae eventually developed into individuals that were about one-fourth the normal body size. Interestingly, their offspring developed normally and showed typical body size, feeding behaviour, and mobility during both larval and adult stages.^[9]

Research on the venom of *Apis mellifera* using *Drosophila melanogaster* has shown that the venom can influence several physiological processes in flies.

Exposure to the venom altered metabolism, reduced antioxidant enzyme activity, and caused structural changes in muscle tissues. The venom peptide Apamin was also found to have antimicrobial activity in the fly gut and slightly affected locomotion and sleep when expressed in neurons. In addition, other venom components such as Melittin and Minimine influenced larval growth and feeding behaviour, resulting in smaller flies that remained viable and produced normal offspring.

Marine Venom

The behavioural assays have been performed by using *Drosophila Melanogaster* as a model organism and its bioactivity is being assessed by using terebrid venom-peptide in the study. Bioactivities of *Physalia physalis* Venom are studied on *Drosophila Melanogaster*. The toxins of the *Physalia physalis* have been known for a long time, but they are still not studied in detail. This study suggests that the venom may affect important biological functions such as movement, circadian rhythm (daily biological clock), and heat-avoidance behaviour. These results show that more research is needed to understand exactly how these toxins work and which biological targets they affect. Future studies using approaches such as transcriptomics and proteomics would help identify the specific venom components responsible for these observed effects. This study also demonstrates that *Drosophila melanogaster* can serve as a valuable model organism for investigating the biological activity of *apis* venom. Overall, the findings contribute to a better understanding of the effects of crude *P. physalis* venom and suggest the potential for discovering novel bioactive molecules, particularly those with neuroactive properties. In addition, the study underscores the importance of marine organisms as promising sources of future therapeutic compounds.^[2]

Two venom peptides from marine snails showed different effects in fruit flies: one reduced the flies' response to heat-induced pain, while the other increased their food intake. These findings help researchers better understand how venom peptides can influence physiological processes such as pain perception and feeding behaviour. They tested two peptides from marine snails: *Terebra variegata* and *Terebra subulate* on *Drosophila melanogaster* as a model organism. One showed that it reduced the fly's response to heat pain (antinociceptive effect) and the other increased food intake. Overall, the study focuses on using behavioural assays in *Drosophila* to study and understand the biological activity of terebrid venom peptides.^[3]

In summary, marine venoms tested on *Drosophila Melanogaster* show effects like antinociceptive effects and changes in circadian rhythm. Some studies also showed that marine venoms will show increased food intake in fruit fly. The treatment with marine venoms also shows changes in heat avoidance behaviour and movement of the fly.

Wasp venom

Parasitic wasp venom blocks the calcium signalling in fruit fly immune cells, preventing the fly from mounting its normal defence. Studying this interaction helps scientists understand important and conserved immune mechanisms across animals.^[10] The venom of *H. hebetor* can affect several insect groups, including fruit flies, and stress-related hormones like AKH can modify how the venom works.^[11] In other studies, scientists have also studied how the parasitic wasp *Asobara japonica* affects the development of fruit fly larvae *Drosophila melanogaster*. When *A. japonica* infects fruit fly larvae, it damages the imaginal discs (future adult tissues), which slows down development and delays pupation, while other important organs such as brain and ventral nerve cord, and the prothoracic gland remain mostly unaffected.^[12] The study looked at how venom particles called venosomes from the parasitic wasp *Leptopilina boulardi* enter immune cells of the fruit fly *Drosophila melanogaster*. Wasp venosomes enter fruit fly immune cells through a special membrane pathway and deliver venom proteins that disrupt immune cell function, helping the parasite survive inside the host. These venom proteins may interfere with the normal function of immune cells, possibly damaging them or preventing them from forming the capsule that normally surrounds and kills the wasp egg. A venom protein called LbGAP moves to different parts of the cell such as recycling endosomes, endolysosomes, and the endoplasmic reticulum and interferes with normal function of immune cells and prevents them from forming an envelope that will help in surrounding and killing the wasp eggs.^[13]

The venom of *P. vindemiae* kills fruit fly immune cells mainly by triggering programmed cell death, weakening the host's immune system and helping the wasp larva survive inside the host. When these immune cells were treated with the venom, the immune cells changed shape, lost mitochondria, formed large vacuoles, and eventually died with features such as cell shrinkage, chromatin condensation, and nucleus fragmentation.^[14] The study found many venom proteins in *L. heterotoma* that likely help the wasp manipulate the fruit fly's immune system, growth, and metabolism. Understanding how these venom components work can improve our knowledge of host-parasite interactions and may also be useful for biological pest control and future scientific research.^[15] Venosomes from *L. boulardi* carry harmful proteins into fruit fly immune cells, weakening the fly's defense system and helping the wasp successfully parasitize the host.^[16]

In summary, various wasps such as *L. boulardi*, *L. heterotoma*, *P. vindemiae*, *Asobara japonica*, *H. hebetor* have venom proteins which have toxic effects on *Drosophila* Model system and the wasps use *Drosophila* as a host to lay their eggs and infect the fruit fly larvae. The wasps attack the immune system of *Drosophila* by attacking the immune cells, hence making it difficult for them to generate an immune response against the

wasp, it also affects their growth and metabolism. Wasp venoms cause cell death and destroy the imaginal discs in *Drosophila* larvae causing delayed growth and development in fruit fly larvae to adult.

Scorpion Venom

The study investigates how the venom of *Hottentotta judaicus* affects insects, especially their nervous system, behaviour, and survival. The venom of *Hottentotta judaicus* is not toxic to human cells but is harmful to insects and can also change insect behaviour. This suggests it may help the scorpion capture prey and could potentially be useful for pest control. The venom shows that at lower (non-lethal) doses, the venom changed fruit fly behaviour by: reducing their movement and increasing their sleep time but mosquitoes were more sensitive than *Drosophila* to this venom.^[17] A new microinjection technique using fruit flies allows scientists to test venom toxicity using much smaller sample amounts, making it useful for studying rare or limited venom samples. They tested several scorpion venoms, including those from *Androctonus australis*, *Leiurus quinquestriatus*, and *Tityus serrulatus* but fruit flies were sometimes less sensitive to some scorpion venoms.^[18] Researchers investigated C56 toxin, a component of the venom of the Indian red scorpion *Mesobuthus tamulus*, to understand its effects on nerve–muscle communication. The toxin was tested on the neuromuscular junction of larvae of the fruit fly *Drosophila melanogaster*, where nerve cells transmit signals to muscles. The results showed that the toxin increases nerve–muscle activity by promoting the entry of calcium ions through NMDA-type receptors, which enhances nerve signalling and leads to stronger muscle contractions.^[19]

In summary of the studies *Drosophila Melanogaster* are less sensitive to scorpion venoms compared to other insect models but experiments using the fruit fly *Drosophila melanogaster* demonstrated that scorpion venoms and toxins can reduce movement, change sleep patterns, or increase nerve activity. Overall, fruit flies serve as an effective model for studying the toxic and neurophysiological effects of scorpion venoms. These studies show that scorpion venoms can affect insects in different ways, including killing them, altering their behaviour, and disrupting nerve–muscle communication.

Spider Venom

In this study, researchers evaluated the effects of two spider venom peptides, J-atracotoxin-Hv1c and μ -theraphotoxin-Hhn2b, on the pest fruit fly *Drosophila suzukii*. They investigated how these peptides influence the flies' survival and examined changes in genes associated with detoxification and stress responses. The findings showed that exposure to these peptides activated stress and detoxification pathways in the flies, and interestingly, one of the peptides even increased their lifespan, suggesting that the insects may trigger protective defence mechanisms in response to the

treatment.^[20]

Scientists have developed a novel pest control strategy known as the toxic male technique (TMT). In this approach, genetically engineered male insects are designed to produce venom proteins in their reproductive tissues and transfer them to females during mating. As a result, females that mate with these males experience a significant reduction in lifespan, which can help lower pest populations and reduce disease transmission. This method was demonstrated in the fruit fly *Drosophila melanogaster*, where females that mated with engineered males lived 37–64% shorter than those mated with normal males. Modelling studies also suggest that this strategy could be effective in controlling disease-carrying mosquitoes such as *Aedes aegypti*.^[21] The spider toxin stops nerve–muscle communication in fruit flies by blocking presynaptic calcium channels, preventing normal nerve signalling. Researchers identified a presynaptic neurotoxin in the venom of the spider *Hololena curta* and investigated its effects using the neuromuscular junction of the fruit fly *Drosophila melanogaster*. Their findings showed that the toxin interferes with calcium channels located at the nerve terminals, which are essential for the release of neurotransmitters. By blocking these channels, the toxin prevents normal transmission of nerve signals to the muscles. As a result, communication between nerves and muscles is disrupted, leading to a strong inhibition of synaptic activity.^[22] α -Latrotoxin from black widow spider venom increases calcium in nerve cells and stimulates strong neurotransmitter release in fruit flies, making *Drosophila* useful for studying how this toxin works. Researchers examined the effects of α -latrotoxin, a toxin found in the venom of the black widow spider *Latrodectus mactans*, to understand how it influences nerve activity. Using the fruit fly *Drosophila melanogaster* as a model organism, researchers observed that the toxin increases calcium levels within nerve endings. This rise in calcium triggers a strong release of neurotransmitters at the neuromuscular junction. The results indicate that α -latrotoxin enhances communication between nerves and muscles by promoting greater calcium entry into nerve cells, which stimulates neurotransmitter release.^[23] Researchers isolated neurotoxic peptides from the venom of the spider *Plectreurys tristis* and examined how they affect nerve–muscle communication in the fruit fly *Drosophila melanogaster*. The study showed that these small peptide toxins act on the presynaptic region of nerve cells and inhibit the release of neurotransmitters, likely by blocking calcium entry into the nerve terminals. Because these toxins are active even at very low concentrations, they provide useful tools for studying the mechanisms that regulate synaptic transmission.^[24] Researchers investigated how different components of the spider venom of *Cupiennius salei* interact with each other. Using the fruit fly *Drosophila melanogaster* as a model organism, they observed that several venom compounds work together to enhance the overall insecticidal effect.

Certain components were found to increase the activity of neurotoxins, making the venom more effective. These results suggest that spider venom does not act through a single molecule alone, but rather through cooperative interactions among multiple venom components.^[25]

In summary, studies using fruit flies such as *Drosophila melanogaster* and *Drosophila suzukii* have shown that spider venoms can significantly affect insect physiology. Many of these effects occur through the disruption of calcium channels, changes in neurotransmitter release, and interference with communication between nerves and muscles. These findings indicate that venom-derived peptides may have practical applications, particularly in the development of new strategies for pest control. Approaches such as the Toxic Male Technique further highlight the potential of combining venom research with innovative pest management methods. Overall, fruit flies serve as a useful and accessible model system for understanding venom action and for exploring environmentally friendly ways to control insect pests.

CONCLUSION

Different venoms can produce a wide range of toxic effects when studied using *Drosophila* as a model system. Research using *Drosophila melanogaster* has shown that venoms from various organisms can influence several biological processes in flies. For example, venoms from species such as *Naja naja* and *Apis mellifera* have been reported to affect behaviour, metabolism, oxidative stress, and tissue structure. Marine venoms have also been shown to alter pain perception, circadian rhythm, feeding behaviour, and heat-avoidance responses. Parasitic wasps like *Leptopilina boulardi* use venom to suppress the immune system of the fly, allowing their larvae to develop successfully inside the host. In addition, venoms from scorpions and spiders can interfere with locomotion, sleep patterns, and neuromuscular communication in flies. Taken together, these findings demonstrate that *Drosophila melanogaster* is a useful and reliable model for studying the biological activity of venoms. Such studies can provide valuable insights into venom mechanisms and may also support future applications in biology, medicine, and pest management.

Authors contribution

Akanksha Joshi carried out the literature search and drafted the manuscript. Dr. Mrunal Ghag Sawant reviewed the manuscript, provided critical intellectual input, and supervised the overall preparation of the manuscript. Sanika N. Parkar supported the supervisory process during manuscript development.

ACKNOWLEDGEMENT

I would like to thank Department of Zoonosis and Toxicology, Haffkine Institute for Training, Research & Testing, Parel, Mumbai for providing the necessary facilities and resources for completing this work. The authors sincerely acknowledge the guidance and support

provided by Dr. Mrunal Ghag Sawant during the preparation of this manuscript. I would further like to express my gratitude to my colleagues Sahin Ansari, Akshata Deshmukh, and Shivangi Koundal for their valuable guidance and suggestions during the writing of the manuscript. I would also like to thank Sanika N. Parkar for supporting the supervisory process during manuscript development.

REFERENCES

- Giansanti MG, Frappaolo A, Piergentili R. *Drosophila melanogaster*: How and Why It Became a Model Organism. *Int J Mol Sci*, 2025 Aug 2; 26(15): 7485. doi:10.3390/ijms26157485. PMID: 40806617; PMCID: PMC12347407.
- Tomkielska Z, Frias J, Simões N, de Bastos BP, Fidalgo J, Casas A, Almeida H, Toubarro D. Revealing the Bioactivities of *Physalia physalis* Venom Using *Drosophila* as a Model. *Toxins (Basel)*, 2024 Nov 15; 16(11): 491. doi:10.3390/toxins16110491. PMID: 39591246; PMCID: PMC11598359.
- Eriksson A, Anand P, Gorson J, Grijuc C, Hadelia E, Stewart JC, Holford M, Claridge-Chang A. Using *Drosophila* behavioral assays to characterize terebrid venom-peptide bioactivity. *Sci Rep*, 2018 Oct 15; 8(1): 15276. doi:10.1038/s41598-018-33215-2. PMID: 30323294; PMCID: PMC6189199.
- K, N., Bakkannavar, S.M., Bhat, V.R. *et al.* Unveiling the nanotoxicology of snake venoms through functional and biochemical characterization of extracellular vesicles from *Naja naja* and *Daboia russelii*. *Sci Rep.*, 2025; 15: 42860. <https://doi.org/10.1038/s41598-025-27041-6>.
- S Rajan, Sheeja & Subramanian, Perumal. (2022). Indian Cobra (*Naja naja*) venom improves cognitive behaviour in *Drosophila melanogaster*, 7: 11-19.
- Leela Sudarsanan Amulya1, Perumal Subramanian1*, Jaime Jacqueline Jayapalan2, Puteri Shafinaz Abdul Rahman3 Effect of Indian snake (*Naja naja*) venom on cognition and biochemical indices in yellow mutant of *Drosophila melanogaster* treated with N-Nitrosodiethylamine *International Journal of Entomology Research* www.entomologyjournals.com ISSN: 2455-4758 Received: 12-01-2023, Accepted: 28-01-2023, Published: 14-02-2023 Volume 8, Issue 2, 2023, Page No. 76-84 <https://www.entomologyjournals.com/assets/archives/2023/vol8issue2/8029-503.pdf>.
- Oparin PB, Nikodimov SS, Vassilevski AA. Venoms with oral toxicity towards insects. *Toxicon*, 2023 Nov; 235: 107308. doi:10.1016/j.toxicon.2023.107308. Epub 2023 Oct 4. PMID: 37797725.
- Černý, J., Krishnan, N., Prokůpková, N. *et al.* Elimination of certain honeybee venom activities by adipokinetic hormone. *Sci Rep.*, 2025; 15: 18638. <https://doi.org/10.1038/s41598-025-02285-4>
- Lowy PH, Sarmiento L, Mitchell HK. Polypeptides

- minimine and melittin from bee venom: effects on *Drosophila*. Arch Biochem Biophys. 1971 Jul; 145(1): 338-43. doi:10.1016/0003-9861(71)90044-0. PMID: 5001226.
10. Mortimer NT, Goecks J, Kacsoh BZ, Mobley JA, Bowersock GJ, Taylor J, Schlenke TA. Parasitoid wasp venom SERCA regulates *Drosophila* calcium levels and inhibits cellular immunity. Proc Natl Acad Sci U S A, 2013 Jun 4; 110(23): 9427-32. doi:10.1073/pnas.1222351110. Epub 2013 May 20. PMID: 23690612; PMCID: PMC3677475.
 11. Černý J, Krishnan N, Hejníková M, Štěrbová H, Kodrík D. Modulation of response to braconid wasp venom by adipokinetic hormone in *Drosophila melanogaster*. Comp Biochem Physiol C Toxicol Pharmacol, 2024 Nov; 285: 110005. doi:10.1016/j.cbpc.2024.110005. Epub 2024 Aug 21. PMID: 39154974.
 12. Kamiyama T, Shimada-Niwa Y, Mori H, Tani N, Takemata-Kawabata H, Fujii M, Takasu A, Katayama M, Kuwabara T, Seike K, Matsuda-Imai N, Senda T, Katsuma S, Nakamura A, Niwa R. Parasitoid wasp venoms degrade *Drosophila* imaginal discs for successful parasitism. Sci Adv, 2025 Jan 31; 11(5): eadq8771. doi:10.1126/sciadv.adq8771. Epub 2025 Jan 29. PMID: 39879297; PMCID: PMC11777187.
 13. Wan B, Poirié M, Gatti JL. Parasitoid wasp venom vesicles (venosomes) enter *Drosophila melanogaster* lamellocytes through a flotillin/lipid raft-dependent endocytic pathway. Virulence, 2020 Dec; 11(1): 1512-1521. doi:10.1080/21505594.2020.1838116. PMID: 33135553; PMCID: PMC7605353.
 14. Wan B, Yang L, Zhang J, Qiu L, Fang Q, Yao H, Poirié M, Gatti JL, Ye G. The Venom of the Ectoparasitoid Wasp *Pachycrepoideus vindemiae* (Hymenoptera: Pteromalidae) Induces Apoptosis of *Drosophila melanogaster* Hemocytes. Insects, 2020 Jun 11; 11(6): 363. doi:10.3390/insects11060363. PMID: 32545289; PMCID: PMC7349765.
 15. Heavner ME, Gueguen G, Rajwani R, Pagan PE, Small C, Govind S. Partial venom gland transcriptome of a *Drosophila* parasitoid wasp, *Leptopilina heterotoma*, reveals novel and shared bioactive profiles with stinging Hymenoptera. Gene, 2013 Sep 10; 526(2): 195-204. doi:10.1016/j.gene.2013.04.080. Epub 2013 May 17. PMID: 23688557; PMCID: PMC3905606.
 16. Wan B, Gouquet E, Ravallec M, Pierre O, Lemauf S, Volkoff AN, Gatti JL, Poirié M. Venom Atypical Extracellular Vesicles as Interspecies Vehicles of Virulence Factors Involved in Host Specificity: The Case of a *Drosophila* Parasitoid Wasp. Front Immunol, 2019 Jul 17; 10: 1688. doi:10.3389/fimmu.2019.01688. PMID: 31379874; PMCID: PMC6653201.
 17. Wehbe R, Karaki A, Dassouki Z, Rima M, Borges A, Roufayel R, Legros C, Fajloun Z, Kambris Z. Characterization of *Hottentotta judaicus* Scorpion Venom: Toxic Effects and Neurobehavioral Modulation in Insect Models. Toxins (Basel), 2025 Nov 3; 17(11): 546. doi:10.3390/toxins17110546. PMID: 41295861; PMCID: PMC12656134.
 18. Escoubas P, Palma MF, Nakajima T. A microinjection technique using *Drosophila melanogaster* for bioassay-guided isolation of neurotoxins in arthropod venoms. Toxicon. 1995 Dec; 33(12): 1549-55. doi:10.1016/0041-0101(95)00107-7. PMID: 8866612.
 19. Gawade, S. P. (2003). Excitatory effects of *Buthus C56* toxin on *Drosophila* larval neuromuscular junction. Journal of Venomous Animals and Toxins Including Tropical Diseases, 9(1): 65–75. https://doi.org/10.1590/S1678-91992003000100004
 20. Regalado L, Sario S, Mendes RJ, Valle J, Harvey PJ, Teixeira C, Gomes P, Andreu D, Santos C. Towards a Sustainable Management of the Spotted-Wing *Drosophila*: Disclosing the Effects of Two Spider Venom Peptides on *Drosophila suzukii*. Insects, 2023 Jun 7; 14(6): 533. doi:10.3390/insects14060533. PMID: 37367349; PMCID: PMC10299698.
 21. Beach SJ, Maselko M. Recombinant venom proteins in insect seminal fluid reduce female lifespan. Nat Commun, 2025 Jan 7; 16(1): 219. doi:10.1038/s41467-024-54863-1. PMID: 39774598; PMCID: PMC11707029.
 22. Bowers CW, Phillips HS, Lee P, Jan YN, Jan LY. Identification and purification of an irreversible presynaptic neurotoxin from the venom of the spider *Hololena curta*. Proc Natl Acad Sci U S A. 1987 May; 84(10): 3506-10. doi:10.1073/pnas.84.10.3506. PMID: 3033650; PMCID: PMC304900.
 23. Umbach JA, Grasso A, Zurcher SD, Kornblum HI, Mastrogiacomo A, Gundersen CB. Electrical and optical monitoring of alpha-latrotoxin action at *Drosophila* neuromuscular junctions. Neuroscience. 1998 Dec; 87(4): 913-24. doi:10.1016/s0306-4522(98)00664-2. PMID: 9759979.
 24. Branton WD, Kolton L, Jan YN, Jan LY. Neurotoxins from *Plectreurys* spider venom are potent presynaptic blockers in *Drosophila*. J Neurosci. 1987 Dec; 7(12): 4195-200. doi:10.1523/JNEUROSCI.07-12-04195.1987. PMID: 2826721; PMCID: PMC6569098.
 25. Wullschlegel B, Nentwig W, Kuhn-Nentwig L. Spider venom: enhancement of venom efficacy mediated by different synergistic strategies in *Cupiennius salei*. J Exp Biol, 2005 Jun; 208(Pt 11): 2115-21. doi:10.1242/jeb.01594. PMID: 15914655.