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Resumo	Introduction: Bee venom has therapeutics and pharmacological properties. Further toxicological studies on animal models are necessary due to the severe allergic reactions caused by this product. Method: Here, Caenorhabditis elegans was used as an in vivo toxicity model, while breast cancer cells were used to evaluate the pharmacological benefits. The bee venom utilized in this research was collected from Apis mellifera species found in Northeast Brazil. The cytotoxicity caused by bee venom was measured by MTT assay on MDA-MB-231 and J774 A.1 cells during 24 - 72 hours of exposure. C. elegans at the L4 larval stage were exposed for three hours to M9 buffer or bee venom. Survival, behavioral parameters, reproduction, DAF-16 transcription factor translocation, the expression of superoxide dismutase (SOD), and metabolomics were analyzed. Bee venom suppressed the growth of MDA-MB-231 cancer cells and exhibited cytotoxic effects on macrophages. Also, decreased C. elegans survival impacted its behaviors by decreasing C. elegans feeding behavior, movement, and reproduction. Results: Bee venom did not increase the expression of SOD-3, but it enhanced DAF-16 translocation from the cytoplasm to the nucleus. C. elegans metabolites differed after bee venom exposure, primarily related to aminoacyl- tRNA biosynthesis, glycine, serine and threonine metabolism, and sphingolipid and purine metabolic pathways. Our findings indicate that exposure to bee venom resulted in harmful effects on the cells and animal models examined.

	Conclusion: Thus, due to its potential toxic effect and induction of allergic reactions, using bee venom as a therapeutic approach has been limited. The development of controlled-release drug strategies to improve this natural product's efficacy and safety should be intensified.
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