Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology
Volume 130, Issue 1, September 2001, Pages 107–117

Eicosanoids act in nodulation reactions to bacterial infections in newly emerged adult honey bees, *Apis mellifera*, but not in older foragers

Jon C. Bedick, Hasan Tunaz, A.R. Nor Aliza, Sean M. Putnam, Marion D. Ellis, & David W. Stanley

Abstract

Nodulation is the first, and qualitatively predominant, cellular defense reaction to bacterial infections in insects. We tested the hypothesis that eicosanoids also mediate nodulation reactions to bacterial challenge in adults of a social insect, the honey bee, *Apis mellifera*. Treating newly-emerged experimental bees with the eicosanoid biosynthesis inhibitor, dexamethasone, impaired nodulation reactions to bacterial infections, and the influence of dexamethasone was reversed by treating infected insects with arachidonic acid, an eicosanoid precursor. Several other eicosanoid biosynthesis inhibitors, including the cyclooxygenase inhibitor, indomethacin, and the dual cyclooxygenase/lipoxygenase inhibitor, phenidone, also impaired the ability of experimental honeybees to form nodules in reaction to bacterial challenge. The influence of phenidone on nodulation was expressed in a dose-dependent manner. However, in experiments with older honey bees foragers, similar bacterial challenge did not evoke nodulation reactions. We infer from our results that while eicosanoids mediate cellular immune responses to bacterial infections in newly emerged honey bees, and more broadly, in most insect species, nodulation reactions to bacterial challenge probably do not occur in all phases of insect life cycles.

Keywords: Eicosanoids; Bacterial infection; Insect immunity; Honey bees