Eicosanoids Rescue *Spodoptera exigua* Infected with *Xenorhabdus nematophilus*, the Symbiotic Bacteria to Entomopathogenic Nematode, *Steinernema carpocapsae*

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Xenorhabdus nematophilus is a pathogenic bacterium causing insect haemolymph septicemia, which leads to host insect death. To address the fundamental mechanisms underlying this haemolymph septicemia, or the immunodepressive response of the host insects following the bacterial infection, we tested a hypothesis that the insect immune-mediating eicosanoid pathway is blocked by inhibitory action of the bacterium. Haemocoelic injection of the bacteria into the fifth instar larvae of *Spodoptera exigua* reduced the total numbers of living haemocytes with postinjection time and resulted in host death in 16h at 25C. The lethal efficacy, described by the median lethal bacterial dose (LD50), was estimated as 33 colony-forming units per 5th instar larva of *S. exigua*. The lethal effect of the bacteria on the infected larvae decreased significantly with the addition of exogenous arachidonic acid (10g), a precursor of eicosanoids. In comparison, the injections of dexamethasone (10g), a specific inhibitor of phospholipase A2, and other eicosanoid biosynthesis inhibitors elevated significantly the bacterial pathogenicity. Live *X. nematophilus* induced the infected larvae to form much less nodule formation than did the heat-killed bacteria. But, the addition of arachidonic acid increased the number of nodule formations significantly in response to live bacterial injection. The treatment of dexamethasone and other inhibitors, however, decreased nodule formation after injection of heat-killed bacteria. These results indicate that eicosanoids play a role in the immune response of *S. exigua*, and suggest strongly that *X. nematophilus* inhibit its eicosanoid pathway, which then results in immunodepressive haemolymph septicemia.