

ObPV-inoculation resulted in the marked up-regulation of genes encoding PR-proteins, a patatin-like lipase (lipid acil hydrolase), a defensin, a 1-aminocyclopropane-1-carboxylic acid (ACC) oxidase and a dioxygenase participating in carotenoid degradation. In addition, ObPV-inoculation led to a rapid and massive up-regulation of several individual 9-lipoxygenase (*9-LOX*) genes. In contrast, *13-LOX* genes were only moderately induced by ObPV. The expression of several genes encoding WRKY transcription factors were also induced by ObPV. In contrast, the expression of defense genes increased in most cases to a lesser extent in PMMoV-inoculated, susceptible leaves or in mock-inoculated leaves. Plant hormones and an ethylene precursor (salicylic acid, methyl-jasmonate, and ACC) induced very differently the expression of individual *LOX* and *WRKY* genes.

In summary, our results showed that the rapid and massive up-regulation of defense genes encoding PR-proteins, LOXs and WRKY transcription factors in the incompatible pepper-ObPV interaction contributes to antiviral resistance. We suppose that by the rapid up-regulation of *9-LOX* genes pepper plants are able to alter the structure of intracellular membranes in order to inhibit the replication of invading tobamoviruses.

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## Investigation of the different mechanisms of the innate immune response of *Drosophila melanogaster*

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*Drosophila melanogaster* has been widely used model organism to study host response to microbial and parasitic infections. The chitin cuticle of the adult *Drosophila* is the first barrier against microbial invasion. Injury of the cuticle activates hemolymph clotting, which blocks the loss of body fluids and the spreading of the microorganisms into the hemocoel by immobilizing bacteria at the wound site. Pathogens entering the hemocoel activate both cell-mediated and humoral immune responses. The cell-mediated arm of the immune response is carried out by the hemocytes, the production of antimicrobial peptides are regulated by the Toll and the immune deficiency (*Imd*) pathways.

We developed and validated a new method to identify novel factors involved in the hemolymph coagulation and in the host-pathogen interactions after septic injury.

The method, based on inducing lesion by removing the tarsal segments of the first pair of legs of *Drosophila* adults and exposing them to different bacteria, imitates injury that often occurs in the natural habitat. The technique was validated by using mutant variations of different components of the immune response; blood clotting as well as the involvement of a number of genes known to be instrumental in the humoral and cell-mediated immune responses of *Drosophila* was confirmed. We used the slightly pathogenic *E. coli*, the semi-pathogenic *B. cereus* and the highly pathogenic *S. marcescens* and monitored the viability of the flies. First, we tested the survival of the control *w<sup>1118</sup>* and mutant flies after sterile injury and the survival of the non-injured *w<sup>1118</sup>* and mutant lines (*spz<sup>2</sup>/spz<sup>4</sup>*, *Dredd<sup>EP1412</sup>*, *Rel<sup>E20</sup>* and *Hml<sup>f03374</sup>*) treated with *E. coli*, *B. cereus* and *S. marcescens*. We found that the survival of non-injured mutant flies treated with *E. coli*, *B. cereus* and *S. marcescens* were similar. The injury itself do not affect the survival of the animals, except for the *Hml<sup>f03374</sup>* homozygotes, which lose more hemolymph after wounding and showed decreased survival rate following both sterile and septic injury compared to the control. We found that the *Imd* pathway mutants *Dredd<sup>EP1412</sup>* and *Rel<sup>E20</sup>* and the hemolymph clotting factor *Hemolectin* (*Hml<sup>f03374</sup>*) mutant flies showed reduced viability after either *B. cereus* or *E. coli* infection, while the *spätzle* (*spz<sup>2</sup>/spz<sup>4</sup>*), involved in the Toll pathway, was significantly sensitive to *B. cereus* infection. By using this novel method, we have found that the *raspberry* gene is involved in the survival of the fly after septic injury, since the mutants have decreased survival rate after *B. cereus* infection. This gene encodes the *Drosophila* inosine monophosphate dehydrogenase, and is a key enzyme of the *de novo* synthesis of guanine nucleotides. In mammals, *de novo* GMP synthesis is required for lymphocyte proliferation and in the immune response. We will study the function of the *raspberry* in the immune response of the *Drosophila*.

Our new method is suitable for high-scale screening of key factors involved in host-pathogen interactions following a septic injury. It also offers an alternative to previous experiments, where microinjection needle were used to administer microbes into the body cavity. A major advantage of this method is that the wound by itself is insignificant, the effect on survival can be attributed entirely to the infection and the defensive capabilities of the host organism.

Furthermore, we identified a new marker molecule 3A5 in the cytoplasm of a subset of plasmatocytes in all hematopoietic compartments, in the circulation, in the lymph gland and in the sessile tissue and in the hemolymph. We study the function of 3A5 molecule in the *Drosophila* immune response and in the coagulation reaction.

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