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| Title: | Eco-Immunology of laboratory-adapted Drosophila melanogaster populations |
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| Keywords: | Laboratory<br>Population<br>Drosophila |
| Issue Date: | May-2023 |
| Publisher: | IISER Mohali |
| Abstract: | At the heart of eco-immunology lies the observation that host populations exhibit a high degree of variability in terms of resistance to pathogens and parasites. A significant portion of this variability can be attributed to the genetic variation within the host and the pathogen populations, but even after accounting for such genetic variation, not all the variability in resistance can be explained away. Therefore, it has been argued that variability in resistance to pathogens and parasites can stem from environmental and other sources, such as previous experience with pathogens and reproduction-immunity trade-offs. To explore such non-genetic factors that determine host resistance to infections, I studied the effects of bacterial infections on the fitness of fruit flies (Drosophila melanogaster) in terms of survival and reproductive output, and what factors – either intrinsic (age and mating status) or extrinsic (resource availability and parental exposure to pathogens) – modify these effects. First, I demonstrated that resource limitation (via concurrent starvation and/or sexual activity induced systemic resource allocation) increases host susceptibility to bacterial infections in a pathogen-specific manner. This increase in susceptibility to infection was accompanied by increased within-host pathogen proliferation, suggesting that hosts with limited resources were incapable of restricting systemic pathogen growth.Second, I explored the effects of bacterial infection on host reproductive effort. I demonstrated that the post-infection change in host reproductive output of an individual host could not be predicted on the basis of the identity of the infecting pathogen, the outcome of infection, and how long the host survived following infection. Furthermore, the effect of infection on host reproductive effort is malleable according to host age and access to resources. Third, I investigated the trans-generational effects of bacterial infections. I demonstrated that offspring of parents infected with a bacterial pathogen survive better (compared to offspring of uninfected parents) when they are infected with the same pathogen. Increased post-infection survival was also observed in case of heterologous challenges, i.e., when parents are infected with one pathogen and the offspring is challenged with a different pathogen. Additionally, the increase in post-infection survival of the offspring could be explained by increased capacity to restrict systemic pathogen growth. In addition to the above three phenomena, I also explored the evolution of alternate defense strategies – resistance vs tolerance – when hosts are subjected to directional selection for improved post-infection survival. Resistance is the ability of the host to restrict within-host pathogen proliferation, and tolerance is the capacity of the host to ameliorate the negative effects of infection. My predecessor had experimentally evolved replicate fly populations to better survive following infection with a Gram-positive bacterium, Enterococcus faecalis. Using these populations, I demonstrated that hosts become more resistant when subjected to selection for increased post-infection survival, without any change in tolerance of infection. To conclude, during my PhD, I have demonstrated that (a) resource limitation compromises host resistance to bacterial infections in a pathogen specific manner, (b) the effect of bacterial infection on reproductive effort of individual hosts is independent of both pathogen identity and infection outcome, (c) the effect of bacterial infection on host reproductive effort isdependent on host age and access to resources, (d) parental exposure to bacterial infections makes offspring better defended against both homologous and heterologous infections, and (e) hosts selected to better survive after being infected evolve increased resistance, but not tolerance, to infections. |
| URI: | http://hdl.handle.net/123456789/5345 |
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