# Are tolerant hosts more likely to become super-shedders of infection?

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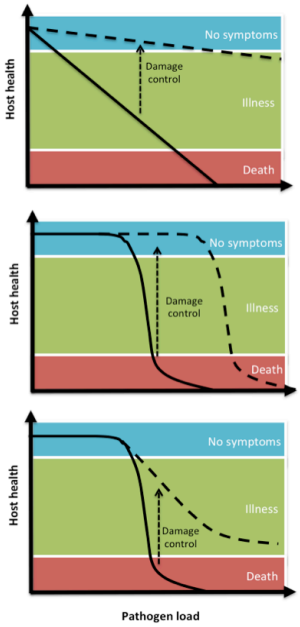
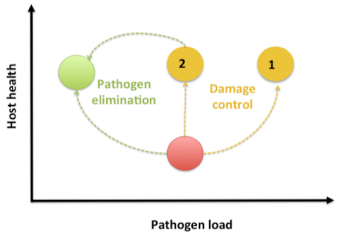
## DAMAGE CONTROL DURING INFECTION

Pathogen growth during infection causes host tissue damage and reduces host health.

Health may be regained through mechanisms that eliminate pathogens (resistance), but also by mechanisms that reduce the damage caused by pathogens, without eliminating them (leading to disease tolerance).

Hosts with more efficient damage control mechanisms, or that receive damage-control therapies, are therefore able to maintain a higher level of health during infection without reducing pathogen burdens.



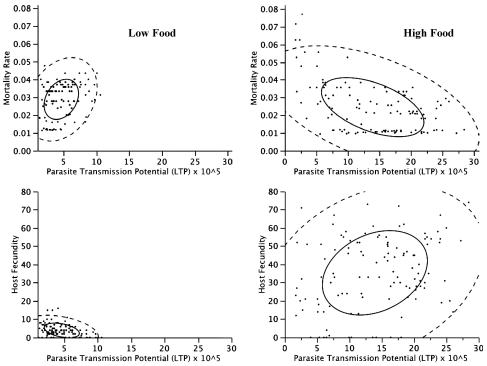

## WHY DOES THIS MATTER?

The epidemiological and evolutionary consequences of mechanisms that control damage during infection are still unclear.

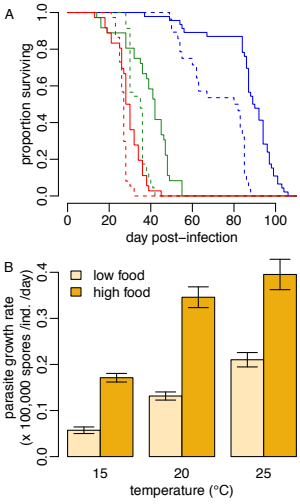
This knowledge may be especially useful to inform on the potential long-term consequences of disease control strategies such as anti-virulence drugs that, by reducing disease without eliminating pathogens, are essentially damage control therapies.

## DO CONDITIONS THAT PROMOTE TOLERANCE ALSO INCREASE TRANSMISSION?

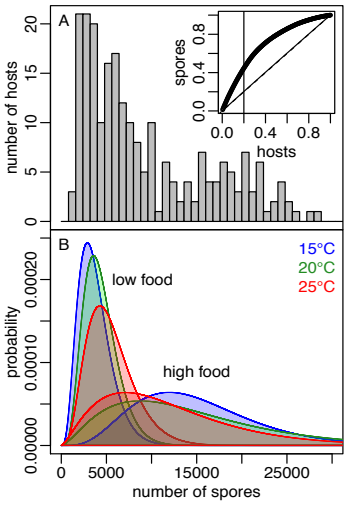
Host condition is one factor likely to affect tolerance to infection. For example, Daphnia infected with *Pasteuria ramosa*, a the sterilizing bacterial pathogen, are able to live longer and maintain reproduction when they are well fed.




This is not due to pathogen clearance (resistance): well-fed hosts harbour more and faster growing pathogens, yet still live longer. They tolerate infection.




As a result, high food conditions yield individual Daphnia contributing disproportionately more to the total pool of transmission: the most infectious 20% of hosts contributed nearly 50% of the total transmission spores.




## ONGOING WORK

Conditions that promote tolerance to infection allow hosts to enjoy higher health despite the presence of infection.

Tolerating infection is therefore beneficial for the individual host but may result in increased disease spread to the rest of the population.

I am currently using the fruit fly Drosophila as a model host to understand the genetic basis of tolerance to viral infection, and to test how tolerance may influence disease spread and evolution.

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