Functional Ecology

Traits of species predict the size of disease epidemics in multi-host communities

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One of the most compelling ideas in contemporary disease ecology is the dilution effect paradigm. A dilution effect is a pattern linking higher species diversity with lower disease risk. In other words, richer biodiversity might shield focal species from severe epidemics. Dilution effects have arisen in numerous human, wildlife, livestock, and plant diseases, and now seem quite general. From a conservation standpoint, this generality suggests a tantalizing win-win: Could the conservation of biodiversity also mitigate dangerous outbreaks of disease? Despite this promising outlook, the dilution effect paradigm is frequently criticized for being overly simplistic, idealistic, and unpredictable. These criticisms abound because few experiments grapple with the mechanisms that underlie dilution effects. Even fewer evaluate predictive frameworks along gradients of host traits. Thus, the dilution effect needs stronger mechanistic foundations.

Here, we evaluate a trait-centric framework for disease dilution with an experiment featuring planktonic hosts. The focal host— the water flea *Daphnia dentifera*—is a dominant grazer in many Midwestern lakes (pictured right). It frequently suffers autumnal epidemics caused by the virulent fungus, *Metchnikowia bicuspidata*. However, epidemics are smaller in lakes with competing zooplankton, especially *Ceriodaphnia sp.* (pictured left). Thus, in nature, these key competitor/diluters reduce disease and drive dilution effects. In our experiment, we selected different genotypes of the focal host that varied in both their susceptibility to infection and ability to compete against the competitor/diluters. We grew each genotype with and without competitor/diluters, and added fungal parasites to start epidemics. Then we tracked the size of epidemics over several host generations. Finally, we asked how traits of the focal host and impacts of the competitor/diluters shaped disease and dilution effects.

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We found that higher susceptibility of the focal host fueled larger epidemics. Simultaneously, weaker competitive ability indirectly suppressed epidemics, because it enabled higher densities of competitor/diluters. These higher densities of diluters reduced disease by competitively lowering the density of focal hosts, and hence inhibited density-dependent transmission of disease. This experiment strengthens the dilution effect paradigm with a predictable, traits-oriented framework. These traits—susceptibility and competitive ability—could help predict epidemic severity in a variety of other multi-host communities.