

'Vicious circles' and disease spread: elements of discussion

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In a recent article, Beldomenico and Begon (BB, [1]) propose that a synergy between host susceptibility and infection might potentially occur in host-pathogen interactions. They argue that hosts in poor condition are more susceptible to higher pathogen occurrence and infection intensity, which further weakens the host's condition [1]. The authors refer to the term 'vicious circles' to illustrate this escalating process between host condition and infection level. They conclude that vicious circles are a neglected process that could influence both pathogen and host dynamics. Although we fully agree with the important ideas developed by BB, we would like to raise two additional points that we believe important to consider for further examination of vicious circles.

First, BB [1] argue that vicious circles generate 'superspreaders' of disease, contributing disproportionately to the spread of pathogens in wild host populations. Although we do not question the existence of superspreaders, we believe that, in some situations, their importance for pathogen spread might be less important than proposed by BB [1]. Indeed, predation often disproportionately affects the most heavily parasitized hosts, that is, the superspreaders [2,3]. Considering the evolution of virulence, it has recently been demonstrated that selection against these superspreaders might be strong enough to reduce the level of virulence in host populations [4,5]. Similarly, we argue that in natural populations where predators are abundant, the impact of superspreaders on disease dynamics might be greatly diminished as a result of the 'purging effect' of predators. This idea would imply that ecosystems depleted of predators as a result of anthropogenic actions (e.g. harvesting, overexploitation) might facilitate the persistence of superspreaders, leading to an extra cost to ecosystems through vicious circles. Therefore, we argue that vicious circles should not be considered in isolation, but rather within a comprehensive framework, including the ecosystem as a whole and possible 'interactions between interactions' [5].

Second, vicious circles are a special case of reciprocal effects between host and pathogen [6]. Reciprocal effects occur when a phenotypic trait of the host controls infection rate and is then affected by the pathogen [6]. Reciprocal effects might not only be synergetic but also antagonistic, and we argue that antagonistic effects could themselves

generate superspreaders. For instance, hosts with a high growth rate before infection (i.e. in good condition) are more prone to infection by trophically transmitted parasites (because of a higher ingestion rate of infected prey). These parasites in turn reduce the growth rate and hence the condition of these hosts as well as the susceptibility to other pathogens [e.g. 6,7–9]. In this example, individuals in initially good condition become the source of parasite aggregation and hence become superspreaders. Furthermore, antagonistic effects have their own properties, and considering them could provide insights that go beyond the field of disease dynamics [6]. Even though synergetic effects are probably more common than antagonistic effects in natural populations, we argue that future studies should evaluate the part of reciprocal effects that is attributable to either synergetic or antagonistic effects.

To conclude, recognizing that vicious circles are undoubtedly an important process will improve our understanding of disease spread in the dynamics of natural populations [1]. However, we believe that by considering vicious circles as reciprocal effects operating in ecosystems of interacting interactions [5,6], further insights into the ecology and evolution of host-pathogen interactions in general could be gained.

References

- Beldomenico, P.M. and Begon, M. (2010) Disease spread, susceptibility and infection intensity: vicious circles? *Trends Ecol Evol.* 25, 21–27
- Temple, S.A. (1986) Do predators always capture substandard individuals disproportionately from prey populations? *Ecology* 68, 669–674
- Moller, A.P. and Erritzoe, J. (2000) Predation against birds with low immunocompetence. *Oecologia* 122, 500–504
- Williams, P.D. and Day, T. (2001) Interactions between sources of mortality and the evolution of parasite virulence. *Proc. R. Soc. B-Biol. Sci.* 268, 2331–2337
- Moller, A.P. (2008) Interactions between interactions: predator–prey, parasite–host and mutualistic interactions. *Ann. N. Y. Acad. Sci.* 1133, 180–186
- Blanchet, S. *et al.* (2009) Reciprocal effects between host phenotype and pathogens: new insights from an old problem. *Trends Parasitol.* 25, 364–369
- Blanchet, S. *et al.* (2009) Why parasitized hosts look different? Resolving the "chicken–egg" dilemma. *Oecologia* 160, 37–47
- Bourque, J.F. *et al.* (2006) Cestode parasitism as a regulator of early life-history survival in an estuarine population of rainbow smelt *Osmerus mordax*. *Mar. Ecol. -Prog. Series* 314, 295–307
- Thomas, F. *et al.* (1997) Hitch-hiker parasites or how to benefit from the strategy of another parasite. *Evolution* 51, 1316–1318