ISRAEL JOURNAL OF ECOLOGY & EVOLUTION, Vol. 56, 2010, pp. 239–250
DOI: 10.1560/IJEE.56.3-4.239

IJEE Soapbox: A WORLD FREE OF PARASITES AND VECTORS: WOULD IT BE HEAVEN, OR WOULD IT BE HELL?

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In a celebrated passage, Stephen Jay Gould mused about what would happen, were one to go to some point in the past, and start life again (Gould, 1989):

> Wind back the tape of life to the early days of the Burgess Shale; let it play again from an identical starting point, and the chance becomes vanishingly small that anything like human intelligence would grace the replay.

This is of course impossible, but Gould's thought experiment is a useful conceptual exercise which brings out key issues of determinism versus randomness in history. More broadly, it can be illuminating to ponder all kinds of implausible and radical scenarios, in effect bracketing the real world with visions of possible worlds. What would our world now be like if, at some time in the past, a mad chemist had come up with a concoction which, after being liberally sprinkled across the globe, eliminated all parasites and their vectors, once and forever? And were such a massive perturbation of the global ecosystem possible, *should* we carry out this mass elimination?

Thinking through such implausible scenarios is an example in ecology and evolutionary biology of a genre of historical analysis called "counterfactual history" (Bunzl, 2004). Winston Churchill, for instance, once mused about how history would have unfolded if Robert E. Lee had won at the Battle of Gettysburg (Churchill, 1931). As another, somewhat extreme example, what would have happened in world history if Pontius Pilate had taken pity on his captive, and released him into exile, rather than sending him on to a brutal execution? Think about it ... no Crusades, no Reformation, no Sistine Chapel, no Lourdes ... and on, and on, and on, one can spin out the ramifying causal implications of alteration in a single human decision.

In such counterfactual exercises, one tweaks the past in some small but key way, and tries to gauge how that change might have reverberated through history. What I

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Robert D. Holt is our first invited ***IJEE Soapbox*** essayist. Bob is Professor of Biology and Arthur R. Marshall, Jr., Chair in Ecology at the University of Florida, and is one of the foremost theoreticians in ecology and evolutionary biology. His research focuses on theoretical and conceptual issues at the population and community levels of ecological organization and on linking ecology with evolutionary biology. Bob is best known for his pioneering work on apparent competition, multispecies interactions in food webs (community modules) in time and space, and the evolution of niche conservatism.

would like to attempt in this essay is something a bit more grandiose, involving alteration in a pervasive feature of the world around us—namely, the permanent removal of all parasites, defined as organisms which live in or on and inflict damage on their hosts (see Lafferty and Kuris, 2002, for a more refined definition of parasitism in the context of general resource-consumption theory). At first glance, one might be tempted to say —go for it, eliminate all the damn parasites and their fellow travelers, such as the vectors that transmit them. Infectious diseases are, after all, a recurrent scourge of human history. Historians have argued that the spread of disease among human populations was a key driving force in watershed events ranging from the fall of the Roman Empire to the success of European conquest throughout the New World (McNeill, 1976). Antibiotics provided a respite for the middle of the 20th century from the fear of epidemics, but the conjunction of evolved antibiotic resistance with the increasing mobility of humans across the world now provides the opportunity for a "perfect storm" of future devastating epidemics. And chronic and acute diseases from parasites not yet tamed by antibiotics are in any case the source of an almost bottomless well of human misery, particularly in underdeveloped countries. The World Health Organization estimated in 2009 that malaria alone accounts for about 250 million cases of illness, and roughly one million deaths, per year (WHO, 2009). There is an enormous economic toll from pathogens of domestic livestock and agricultural crops. For instance, an outbreak of foot-and-mouth disease in Taiwan in 1997 led to the destruction of the Taiwanese pork industry, at a cost of billions of dollars of lost capital and revenue (USDA, 1998).

Many species in natural communities that we care about are also gravely threatened by parasites and infectious diseases, leading to increasing attention being give nby conservation biologists to infectious disease and parasitism (Haydon et al., 2002; Lafferty and Geber, 2002; Smith et al., 2009; Nichols and Gómez, 2011). Riordan et al. (2007) provide a particularly thoughtful overview of the conservation roles of parasites (and indeed, much of what I say here uses their review as a springboard). Examples of conservation risks for hosts due to parasites are widespread across taxa, habitat types, and biogeographical regions. For instance, the canine distemper virus has devastated populations of mammalian carnivores ranging from lions in Africa (Munson et al., 2008) to seals in the Caspian Sea (Kennedy et al., 2000). Chestnut blight in the first half of the 20th century eliminated this noble hardwood tree from large swathes of the eastern USA (Freinkel, 2007), and sudden oak death is currently threatening to do much the same number on tanoak (*Notholithocarpus densiflorus*), an abundant broadleaf tree in redwood forests (Ramage et al., 2011). There seems to be a relentless barrage of depressing news about novel pathogens emerging to devastating effect, such as the white nose syndrome which has recently killed over a million bats in the northeastern United States (USGS, 2011).

Given these facts about the human, economic, and conservation toll of pathogens, there would seem to be an overwhelmingly compelling argument that were one able to wave a technological wand and eliminate all pathogens and parasites, one should by all means do so. There would not likely be a groundswell of outraged public opinion, clamoring to "Save the Parasites!" McMahan (2010), in the blog The Stone, has recently

sketched a comparable ethical argument for why it would be justifiable to eliminate vertebrate carnivores, on the grounds that they inflict a continual barrage of suffering on their prey.

But not so fast. There are other considerations that should make one pause before hasty action. Some cautionary thoughts focus on intellectual issues, some on pragmatic considerations, and others on ethical concerns. Consider smallpox (which, for the sake of argument in this essay, I consider to be "alive"). The last known stocks of this virus are now held by the US and Russian governments. There is an ongoing controversy as to whether or not these stocks should be destroyed. The basic argument for doing so is that since we at long last have this perennial enemy of our species down on the mat, we should deliver the final blow, before it can escape to strike again.

Recently, Weinstein (2011) has made an intriguing counter-argument, in favor of retaining these stocks. He points out that our immune system has evolved in response to threats by smallpox (as well as many other parasites). Smallpox produces complex changes in the immune responses of human hosts. To understand the immune systems, one must understand it in the context of the specific selective factors which have shaped it. Destroying the last variola stocks will thus irrevocably shut the door on potentially fruitful lines of research in immunology. Another angle is the fascinating suggestion that smallpox (and vaccination against smallpox) in some fashion may have made the emergence of HIV more difficult (references in Weinstein, 2011). Understanding what is going on here could help unravel broader aspects of disease emergence in a multi-pathogen context.

Beyond these intellectual arguments, there are also pragmatic reasons for preserving the smallpox stocks. If there are actually other, hidden stocks out there, retained say by rogue nations or a cabal of mad scientists, we may need these governmental stocks in the future to ramp up vaccination programs once again. Weinstein (2011) also points out that diseases comparable to smallpox may well arise from other, related poxviruses, leading to another pragmatic justification for not destroying captive stock of the virus. Having stocks of smallpox on hand may be essential for comparative studies on treatment and vaccination procedures needed for dealing with potential future threats from related viruses.

What about ethics? That seems an odd issue to raise here. What value could a virus solely inflicting humans have, beyond the anthropogenically-centered pragmatic and intellectual benefits just noted? Some environmental ethicists have argued that one justification for conservation is that *all* species have an intrinsic value, and so have an inherent right to exist, or at least not to be deliberately or inadvertently exterminated by another species, that is, us. Ehrenfeld (1972), for example, states:

> The non-humanistic value of communities and species is the simplest of all to state: they should be conserved because they exist and because this existence is itself but the present expression of a continuing historical process of immense antiquity and majesty. Long standing existence in Nature is deemed to carry with it the unimpeachable right to continued existence.

Does the smallpox virus (or any parasite) really have an intrinsic right to exist? I am not sure that it does, but I do think this is a question that needs to be asked and pondered. In some ways, the question is comparable to that of capital punishment. The absolutist moral argument against capital punishment is that every human being has an intrinsic worth (an unconditional right to life, Jacquette, 2009)—no matter how heinous their acts—and that other humans, individually and collectively via our governments, do not have the right to expunge any given human life. Instead, as deterrence and punishment, one puts serial murderers into prison for life. [As an aside: of course, capital punishment has to do with the ethical standing of individuals who are rational, feeling beings, not entire species (i.e., a different level in the organization of life). A closer analogy to the question of the potential intrinsic value of a species might be the preservation of cultural diversity (e.g., ethnic groups, or languages). An ethnic or language group might be eliminated not because its members are killed, but because all infants are forcibly taken from their mothers and raised in another culture (akin to conservation threats posed by hybridization). After a single generation, that cultural entity will have been exterminated.]

These ruminations about possible reasons to eliminate smallpox are comparable to lines of argument in the biodiversity literature about how to evaluate the "worth" of ecosystems and their components (Maclaurin and Sterelny, 2008). It is difficult to defend the notion of an intrinsic right to exist, without making reference to rational agents with particular values, and—more critical—to articulate how to make this notion of intrinsic worth tractable in applied contexts. Moreover, note that in our discussion of smallpox up to now, we have implicitly assumed that it is retained only in laboratory stocks, not freely circulating in human hosts. This is akin to keeping fierce predators in a zoo, where they can do no harm. This raises an important general issue in conservation: is the goal to preserve lineages, or lineages with their traits and interactions as they currently are, in a concrete selective environment, in effect capturing in perpetuity a freeze-frame slice of life? Are there arguments for retaining smallpox in human populations, where they have evolved, and do damage? The harm they do to their hosts—us—would seem to vastly outweigh any possible intrinsic value they have to persist, *qua* themselves. I certainly would not argue for unleashing smallpox back upon humankind! But what about other, less dramatically harmful, parasites, including not just those afflicting humans, domestic animals, and crops, or species of conservation concern, but all the other host taxa out there? Almost every non-parasitic species, upon close examination, proves to harbor anywhere from one to many parasites. By some counts, parasites comprise up to half or more of the diversity of all living things (Price, 1978; Riordan et al., 2007). A great deal of the story of life is thus bound up in their lifestyles and phylogenetic histories. Losing them, we lose that dimension of the richness of life. This would certainly be an intellectual loss, most likely an aesthetic loss as well (there is a kind of beauty in the recondite host-switching habits of many parasites, for instance), and at least arguably an ethical loss (if one follows Ehrenfeld in seeing intrinsic value in each species, regardless of its habitus).

Beyond this, there would be serious pragmatic ramifications of such a mass para-cide. For starters, I think from Weinstein's reflections one can generalize about how smallpox

may have influenced the evolution of the immune system. The traits of all extant organisms are for the most part adaptations to their environment, including the biotic environment. Thus, any large, abrupt change in the environment is likely to lead to maladaptation, possibly severe. We are who are we are because of the challenging environments in which our ancestors lived. In the developed world, over the last several decades, there has been a highly disturbing surge in the incidence of diseases such as type I diabetes and multiple sclerosis, ailments which involve autoimmune pathology. Zaccone et al. (2006) argue that decreasing rates of infection, particularly in infants and juveniles, may have set the stage for this increasing prevalence of autoimmune diseases. Exposure to relatively (but not completely) harmless microbes early in life may be required to prime the immune system so as to deal with more serious threats later. More broadly, the immune system is intimately involved in nearly all aspects of human health, and eliminating all parasites from the environment could lead to a wide spectrum of degraded health conditions. If eliminating all parasites (many of which have minor fitness costs for their hosts) produces an environment to which hosts are maladapted, this would seem to be a bad thing, at least initially.

The elimination of parasites may not only make species maladapted in the short run; it could even precipitate their extinction. This is likely true for both the horizontal (within trophic level) and vertical (among trophic level) dimensions of community organization. Many lines of evidence and thought suggest that for a variety of reasons parasitism, just like predation (Chesson and Kuang, 2008), can permit the persistence of multiple host species competing for the same resource base (Mordecai, 2011). Janzen (1970) and Connell (1971) argued that specialist natural enemies (including pathogens) reduced the abundance of their host plants, freeing up resources which can sustain other species. Bagchi et al. (2010), for instance, demonstrated that fungal attack could nearly eliminate seedlings when dense, as expected beneath a maternal tropical tree species. The buildup of specialized soil pathogens may help maintain diversity in grasslands (Petermann et al., 2008; Fitzsimmons and Miller, 2010). Experimental elimination of foliar pathogens in grassland led to a decline in species richness (E. Allan et al., 2010). Parasites in effect create novel niche dimensions along which host species can differentiate, becoming differentially vulnerable to different parasite species. By differentially attacking competitively superior species, parasites can also help equalize fitness among host species. Both niche differences and fitness equalization are essential ingredients for competing species to coexist (Chesson, 2000). The elimination of parasites could permit species that can then reach large abundance to more effectively eliminate inferior competitors from systems. Kevin Lafferty (pers. comm.) has suggested that this might describe a world in which the combination of pesticides and genetic engineering has allowed humans to cover the world with monotonously monospecific stands of crops, reaching even higher levels of abundance, and thereby wreaking much damage on the rest of the biosphere. Yoel Margalith's seminal work with *Bacillus thuringensis* as a control agent of medically important insects exemplifies how humans can exploit the regulatory potential of pathogens to foster human well-being (see Tsurim et al., this issue).

There is growing recognition that parasite links are ubiquitous in food webs, and

that the amount of energy and nutrients flowing through parasites can match what moves through more familiar trophic links (Lafferty et al., 2008; Amundsen et al. 2009; Johnson et al. 2010). Sato et al. (2011) recently reported that crickets and grasshoppers parasitized by a nematomorph were much more likely to enter streams. There, these parasitized hosts are fed on by endangered Japanese species of trout, comprising 80% of their diet and 60% of the trout's annual energy budget. As in this example, parasitism often increases the susceptibility of prey to predation (Hudson et al.. 1992; Hatcher et al., 2006). For instance, the mortality of wood frog (*Rana sylvatica*) tadpoles from predation by salamanders increased from about 50% to 95%, when the tadpoles were exposed to trematode parasites (Belden and Wojdak, 2011). This effect is to be expected whenever host capture is required for a parasite to complete its life cycle, leading to host manipulation by the parasite (Lafferty and Morris, 1996; Lefevre et al., 2009), but it can also arise simply because parasitism impairs host defenses.

The immediate effect of a wholesale elimination of parasites across a community would likely be a reduction throughout the food web of the per capita rate of consumption by predators, per available prey (viz., reduced attack rates). The Japanese trout studied by Sato et al. (2011) would, for instance, lose over half the caloric content of its diet, and so could well face extinction. Some first-order rough expectations would be as follows: Specialist predators which largely depend in the first place on weakened or ill prey will become more vulnerable to extinction. Generalist predators that opportunistically include similar prey in their diet would lose these trophic links, simplifying the overall flow structure of the web. Food web theory suggests that weak links can stabilize trophic dynamics (McCann et al., 1998), so culling weak links that exist due to parasitism might unleash destabilizing strong interactions. Because more prey escape predation, their numbers can surge, leading to intensified exploitation of lower trophic levels (i.e., a trophic cascade). As an example, vaccination programs led to the control of rinderpest in livestock populations around the Serengeti ecosystem, which freed ungulate populations from spillover infection. Wildebeest surged in numbers. This led to an increase in grass consumption, widely reducing fuel loads and thus the extent of fire. This in turn altered the competitive balance between trees and grass in the Serengeti, and led to an increase in tree coverage (Holdo et al., 2009).

All these first-order expectations can, of course, be modified by the multitude of nonlinear feedbacks that are present in even modestly complex ecological systems. Parasites collectively drain energy and resources which otherwise might be available for herbivores and predators, so despite reduction in attack rates there might be a surge of extra production coursing through the remainder of the food web (K. Lafferty, personal observation). The papers collected in this special issue of *Israel Journal of Ecology & Evolution* on vector community ecology highlight some of the rich complexities and potential feedbacks that are present in many host–pathogen systems, which can include multiple species of hosts, parasites, and vectors engaged in a wide range of direct and indirect ecological interactions (Bonsall and Holt, 2010, this issue; Bonsall et al., 2010, this issue; Civitello et al., 2010, this issue; Duquesne and Liess, 2010, this issue; Juliano, 2010, this issue). West Nile virus, for instance, is transmitted by many species of

mosquitoes and can infect a wide range of vertebrate hosts, so prevalence in any given host in a sense reflects processes in a much wider community (Lord, 2010, this issue). Detailed behavioral responses such as mosquito biting rates (Roitberg and Mangel, 2010, this issue), oviposition (Vonesh and Blaustein, 2010, this issue), and habitat avoidance by hosts to either parasites or predators, can all modulate transmission dynamics (Allan et al., 2010, this issue; Kershenbaum et al., 2010, this issue). Relatively little is known about the fitness consequences of the transmitted parasite for the vector itself, either directly during transmission, or more indirectly via impacts on the abundance and availability of the hosts from which the vector draws a meal. Without having a detailed model in hand, it is difficult to make any more precise predictions about what would happen, were all parasites in a community suddenly to disappear. But it is a reasonable guess, given both the horizontal and vertical processes sketched above, that there would be a wholescale shift in host species' abundances, altered community and ecosystem stability, and likely many species' extinctions.

Beyond such effects over ecological time, there likely would be profound consequences at evolutionary time-scales. Within a single host species, the absence of parasites wiould alter microevolutionary processes, for instance, on life history traits (Hochberg et al., 1992). As an example, Fredensborg and Poulin (2006) argue that castrating parasitism on a gastropod has strongly selected for earlier maturation.

Among species, as I noted above, the struggle between parasites and their hosts provides avenues for ecological differentiation permitting species coexistence. Over evolutionary time, such axes of biotic differentiation in effect provide venues for adaptive differentiation. Ricklefs (2011) has recently argued that the coevolutionary struggle between hosts and pathogens may have been a major driver of, and constraint on, adaptive diversification and the filling of available niche space within evolving phylogenetic lineages (an idea that may stem back to Haldane, 1949). The basic idea is that when different populations of a species become reproductively isolated, they may not be able to become sympatric, even if they utilize somewhat different resources, if pathogens can readily spill over from one to the other. The unfolding of adaptive radiations thus might play out very differently in the absence of pathogens, with many fewer species (e.g., of tropical forest trees) stably coexisting at single locations because of the absence of strong intraspecific density dependence arising from specialized parasitism, and because there is no room for tradeoffs between ability to escape parasites, and resource use and other modalities of niche differentiation.

The term "adaptive radiation" describes the evolution of divergent species from a common ancestor, where descendents occupy different ecological niches. In sexual species, reproductive isolation—such as like mating with like—is an important demarcation of species identity, and so is a key driver of adaptive radiation. In asexual species, what counts as a "species" is harder to define. Much of the diversity of life—the glory of a male pheasant's plumage, the roar of a male elk in rut, the elaborately distinct genitalia of insects—revolves around sexuality.

But why is there sex in the first place? An individual well-adapted to its local environment would seem to have nothing to gain by combining its genes with less-adapted

individuals. If there is a "best" genotype in any given environment, this genotype can most effectively propagate itself by asexual cloning. So there is an inherent cost to sexual reproduction (not counting the costs of maintaining the sexual apparatus itself, seeking out partners, etc.). Understanding how sex persists in face of its manifest costs has been a major conceptual conundrum that has challenged evolutionary biologists for some years. John Jaenike (1978) and W.D. Hamilton (1980) ingeniously argued that for sex to be maintained, the environment in some sense had to be constantly becoming more hostile, and that parasites fit the bill for providing the directional degradation in the environment needed to maintain sex. A growing body of theory and evidence now suggests that an essential part of the explanation for the origination and maintenance of sex indeed comes from the coevolutionary dynamics between parasites and their hosts. Fitness in hosts corrodes as parasites adapt to them. Sex scrambles the genetic variation found in hosts, permitting some host individuals to have offspring that potentially escape the pathogens that harmed their parents. In species that have both sexual and asexual varieties, the former are more prevalent where parasites are more common (Jokela et al., 2009). A recent laboratory study using nematode worms attacked by a bacterial pathogen showed that populations of asexual worms basically could not keep up with the relentlessly accelerating attack of the pathogen, and so went extinct, whereas populations of sexual worms persisted (Morran et al., 2011). In the words of a journalist reporting on the Morran et al. study (Science Daily, 2011): "So we may well have to thank parasites—in spite of their nasty reputation—for the joy of sex."

Were we to eliminate all parasites, then over the long haul, we should expect sexuality to gradually become replaced with asexuality. It is a reasonable guess that much of the color and drama of the biotic world would then disappear. Once sex—with its genetic corollary, recombination—is in place, it does not merely facilitate hosts in their ongoing coevolutionary arms race with parasites. Constantly reshuffling of the genome as a happy byproduct can foster the appearance of evolutionary novelties, permit species to adaptively track changes in the abiotic environment, and provide mechanisms for reproductive isolation in many ways and hence foster speciation. Host–parasite interactions may thus indirectly foster adaptive diversification (an idea that goes back to Haldane, 1949), and so eliminating parasites would in the long run likely degrade the efflorescence of diversity across the tree of life. Going back to our initial impulse to eliminate all parasites at a stroke: it might be understandable and tempting, but it almost assuredly would be a very bad idea, at least if we wish to maintain a reasonable semblance of the rich diversity of life on the planet we now enjoy, and are at risk of losing for so many other reasons.

ACKNOWLEDGMENTS

I thank Kevin Lafferty very much for helpful comments and key references. I would also like to thank W.D. Hamilton, with whom I had a warmly wandering conversation over a leisurely meal, many years ago, about the pervasive influence of parasites in ecology and evolution, and Ernst Mayr, who when I was a graduate student long ago in the Museum

of Comparative Ecology shared with me his personal copy of Haldane (1949), as well as talked with me about a wide range of evolutionary themes related to disease. Some of the thoughts in this essay may well stem back to seeds planted in those long-ago ruminations. I also thank the University of Florida Foundation for support.

REFERENCES

Allan, B.F., Varns, T.S., Chase, J.M. 2010. Fear of parasites: lone star ticks increase giving-up densities in white-tailed deer. Isr. J. Ecol. Evol. 56: 313–324, this volume

Allan, E., van Ruijven, J., Crawley, M.J. 2010. Foliar fungal pathogens and grassland biodiversity. Ecology 9: 2572–2582.

Amundsen, P.A., Lafferty, K.D., Knudsen, R., Primicerio, R., Klemtsen, A., Kuris, A.M. 2009. Food web topology and parasites in the pelagic zone of a subarctic lake. J. Anim. Ecol. 78: 563–572.

Bagchi, R., Swinfield, T., Gallery, R.E., Lewis, O.T., Gripenberg, S., Narayan, L., Freckleton, R.P. 2010. Testing the Janzen–Connell mechanism: pathogens cause overcompensating density dependence in a tropical tree. Ecol. Lett. 13: 1262–1269.

Belden, L.K., Wojdak, J.M. 2011. The combined influence of trematode parasites and predatory salamanders on wood frog (*Rana sylvatica*) tadpoles. Oecologia 166: 1077–1086.

Bonsall, M.B., Holt, R.D. 2010. Apparent competition and vector-host interactions. Isr. J. Ecol. Evol. 56: 393–416, this issue.

Bonsall, M.B., Alphey, L., Alphey, N., Yakob, L. 2010. Transgenic control of vectors: the effects of interspecific interactions. Isr. J. Ecol. Evol. 56: 353–370, this issue.

Bunzl, M. 2004. Counterfactual history: a user's guide. The American Historical Review, June 2004. <http://www.historycooperative.org/journals/ahr/109.3/bunzl.html> (19 Aug. 2011).

Chesson, P. 2000. Mechanisms of maintenance of species diversity. Annu. Rev. Ecol. Syst. 31: 343–366.

Chesson, P., Kuang, J.J. 2008. The interaction between predation and competition. Nature 456: 235–238.

Connell, J.H. 1971. On the role of natural enemies in preventing competitive exclusion in some marine animals and in rain forest trees. In: den Boer, P.J., Gradwell, G., eds. Dynamics of numbers in populations. Proceedings of the Advanced Study Institute. Centre for Agricultural Publication and Documentation, Wageningen, The Netherlands, pp. 298–312.

Churchill, W. 1931. If Lee had not won the battle of Gettysburg. In: Squire, J.C., ed. If it had happened otherwise. Longmans, Green, London, UK. Reprinted in Wisconsin Magazine of History 44: 4, summer, 1961.

Civitello, D.J., Rynkiewicz, E., Clay, K. 2010. Meta-analysis of co-infections in ticks. Isr. J. Ecol. Evol. 56: 417–431, this issue.

Duquesne, S., Liess, M. 2010. Indirect effects of pesticides on mosquito larvae via alterations of community structure. Isr. J. Ecol. Evol. 56: 433–447, this issue.

Ehrenfeld, D. 1972. Conserving life on earth. Oxford University Press, New York.

Fitzsimmons, M.S., Miller, R.M. 2010. The importance of soil microorganisms for maintaining diverse plant communities in tallgrass prairie. Am. J. Bot. 97: 1937–1943.

Fredensborg, B.L., Poulin, R. 2006. Parasitism shaping host life-history evolution: adaptive responses in a marine gastropod to infection by trematodes. J. Anim. Ecol. 75: 44–53.

Freinkel, S. 2007. American chestnut: the life, death, and rebirth of a perfect tree. University of

California Pres, Berkeley, CA.
Gould, S.J. 1989. Wonderful life: the Burgess Shale and the nature of history. W.W. Norton, New York.
Haldane, J.B.S. 1949. Disease and evolution. Ric. Sci. Suppl. A 19: 68–76
Hamilton, W.D. 1980. Sex versus non-sex versus parasite. Oikos 35: 282–290.
Hatcher, M.J., Dick, J.T.A., Dunn, A.M. 2006. How parasites affect interactions between competitors and predators. Ecol. Lett. 9: 1253–1271.
Haydon, D.T., Laurenson, M.K., Sillero-Zubiri, C. 2002. Integrating epidemiology into population viability analysis: managing the risk posed by rabies and canine distemper to the Ethiopian wolf. Conserv. Biol. 16: 1372–1385.
Hochberg, M.E., Michalakis, Y., de Meeus, T. 1992. Parasitism as a constraint on the rate of life-history evolution. J. Evol. Biol. 5: 491–504.
Holdo, R.M., Sinclair, A.R.E., Dobson, A.P., Metzger, K.L., Bolker, B.M., Ritchie, M.E., Holt, R.D. 2009. A disease-mediated trophic cascade in the Serengeti and its implications for ecosystem C. PLoS Biol 7:e1000210. doi:10.1371/journal.pbio.1000210.
Hudson, P.J., Dobson, A.P., Newborn, D. 1992. Do parasites make prey vulnerable to predation? Red grouse and parasites. J. Anim. Ecol. 61: 681–692.
Jacquette, D. 2009. Dialogues on the ethics of capital punishment. Roman and Littlefield Publishers, Lanham, Maryland.
Jaenike, J. 1978. An hypothesis to account for the maintenance of sex within populations. Evolutionary Theory 3: 191–194.
Janzen, D.H. 1970. Herbivores and the number of tree species in tropical forests. Am. Nat. 104: 501–528.
Johnson, P.T.J., Dobson, A., Lafferty, K.D., Marcogliese, D.J., Memmott, J., Orlofske, S.A., Poulin, R., Theiltges, D.W. 2010. When parasites become prey: ecological and epidemiological significance of eating parasites. Trends Ecol. Evol. 25: 326–371.
Jokela, J., Dybdahl, M.F., Lively, C.M. 2009. The maintenance of sex, clonal dynamics, and host-parasite coevolution in a mixed population of sexual and asexual snails. Am. Nat. 174: S43–S53.
Juliano, S.A. 2010. Coexistence, exclusion, or neutrality? A meta-analysis of competition between *Aedes albopictus* and resident mosquitoes. Isr. J. Ecol. Evol. 56: 325–351, this issue.
Kennedy, S., Kuiken, T., Jepson, P.D., Deaville, R., Forsyth, M., Barrett, T., van de Bildt, M.W.G., Osterhaus, A.D.M.E., Eybatov, T., Duck, C., Kydrymanov, A., Mitrofanov, I., Wilson, S. 2000. Mass die-off of Caspian seals caused by canine distemper virus. Emerging Infect. Dis. 6: 637–639.
Kershenbaum, A., Stone, L., Kotler, B.P., Blaustein, L. 2010. Predation risk can drive cycles in zoonotic disease prevalence. Isr. J. Ecol. Evol. 56: 281–295, this issue.
Lafferty, K.D. Gerber, L.R. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. Conserv. Biol. 16: 593–604.
Lafferty, K. D., Kuris, A.M. 2002. Trophic strategies, animal diversity and body size. Trends Ecol. Evol. 17: 507–513.
Lafferty, K.D., Morris, A.K. 1996. Altered behavior of parasitized killifish increases susceptibility to predation by bird final hosts. Ecology 77: 1390–1397.
Lafferty, K.D., Allesina, S., Arim, M., Briggs, C.J., De Leo, G., Dobson, A.P., Dunne, J.A., Johnson, P.T.J., Kuris, A.M., Marcogliese, D.J., Martinez, N.D., Memmott, J., Marquet, P.A., McLaughlin, J.P., Mordecai, E.A., Pascual, M., Poulin, R., Thieltges, D.W. 2008. Parasites in food webs: the ultimate missing links. Ecol. Lett. 11: 533–546.

Lefevre, T., Lebarbenchon, C., Gauthier-Clerc, M., Misse, D., Poulin, R., Thomas, F. 2009. The ecological significance of manipulative parasites. Trends Ecol. Evol. 24: 41–48.

Lord, C.C. 2010. The effect of multiple vectors on arbovirus transmission. Isr. J. Ecol. Evol. 56: 371–392, this issue.

Maclaurin, J., Sterelny, K. 2008. What is biodiversity? University of Chicago Press, Chicago.

McCann, K., Hastings, A., Huxel, G.R. 1998. Weak trophic interactions and the balance of nature. Nature 395: 794–798.

McMahan, J. 2010. The meat eaters. September 19, 2010 in The New York Times (http://opinionator.blogs.nytimes.com/2010/09/19/the-meat-eaters/).

McNeill, W.H. 1976. Plagues and peoples. Anchor Press, Garden City, NY.

Mordecai, E.A. 2011. Pathogen impacts on plant communities: unifying theory, concepts, and empirical work. Ecol. Monogr. 81: 429–441.

Morran, L.T., O.D. Schmidt, Gelarden, I.A., Parrish, R.C., Lively, C.M. 2011. Running with the Red Queen: Host–parasite coevolution selects for biparental sex. Science 333: 216–218.

Munson, L., Terio, K.A., Kock, R., Mlengeya, T., Roelke, M.E., Dubovi, E., Summers, B.,Sinclair, A.R.E., Packer, C. 2008. Climate extremes promote fatal co-infections during canine distemper epidemics in African lions. PLoS One 3 (6) e2545, 1–6.

Nichols, E.,Gómez, A. 2011. Conservation education needs more parasites. Biol. Conserv. 144: 937–941.

Petermann, J.S., Fergus, A.J.F., Turnbull, L.A., Schmid, B. 2008. Janzen–Connell effects are widespread and strong enough to maintain diversity in grasslands. Ecology 89: 2399–2406.

Price, P. 1978. The evolutionary biology of parasites. Princeton University Press, Princeton, NJ.

Ramage, B.S., O'Hara, K.L., Forrestel, A.B. 2011. Forest transformation resulting from an exotic pathogen: regeneration and tanoak mortality in coast redwood stands affected by sudden oak death. Can. J. For. Res. 41: 763–772.

Ricklefs, R.E. 2011. Evolutionary diversification, coevolution between population and their antagonists, and the filling of niche space. Proc. Natl. Acad. Sci. USA 107: 1265–1272.

Riordan, P., Hudson, P.J., Albon, S. 2007. Do parasites matter? Infectious diseases and the conservation of host populations. In: Macdonald, D.W., Service, K., eds. Key topics in conservation biology. Blackwell Publishing, Oxford, UK, pp. 156–172.

Roitberg, B.D., Mangel, M. 2010. Mosquito biting and movement rates as an emergent community property and the implications for malarial interventions. Isr. J. Ecol. Evol. 56: 297–312, this issue.

Sato, T., Watanabe, K., Kanaiwa, M., Niizuma, Y., Harada, Y., Lafferty, K.D. 2011. Nematomorph parasites drive energy flow through a riparian ecosystem. Ecology 92: 201–207.

Science Daily. 2011. August 20, 2011, from http://www.sciencedaily.com/releases/2009/07/090706171542.htm

Smith, K.F., Acevedo-Whitehouse, K., Pedersen, A.B. 2009. The role of infectious diseases in biological conservation. Anim. Conserv. 12: 1–12.

Tsurim, I., Silberbush, A., Blaustein, L. In memory of Yoel Margalith. Isr. J. Ecol. Evol. 56: 449–450, this issue.

US Department of Agriculture—Foreign Animal Report 1998, p. 41 http://www.aphis.usda.gov/lpa/pubs/fadrep.pdf

USGS National Wildlife Health Center. 2011. http://www.nwhc.usgs.gov/disease_information/white-nose_syndrome/8/20/2011, p. 1–4.

Vonesh, J.R., Blaustein, L. 2010. Predator-induced shifts in mosquito oviposition site selection: A meta-analysis and implications for vector control. Isr. J. Ecol. Evol. 263–279, this issue.

Weinstein, R.S. 2011. Should remaining stockpiles of smallpox virus (variola) be destroyed? Emerging Infect. Dis. 17: 681–683.

World Health Organization. 2009. Malaria fact sheet. http://www.who.int/features/factfiles/malaria/en/index.html.

Zaccone, P., Fehervari, Z., Phillips, J.M., Dunne, D.W.D., Cooke, A. 2006. Parasitic worms and inflammatory diseases. Parasite Immunol. 28: 515–523.