Straw men don’t get Lyme disease: response to Wood and Lafferty

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Wood and Lafferty [1] (hereafter WL) attempt a synthesis of two views of the ecological factors underlying variable Lyme disease (LD) risk. LD emerged during the 1970s following the post-agricultural reforestation of the northeastern USA, which provided the habitat required by the blacklegged tick vector (Ixodes scapularis) and many of its hosts [2]. However, within the large and growing North American LD zone, risk and incidence vary enormously. To explain LD risk, WL contrast a ‘traditional’ perspective, in which forestation is associated with high risk, and a ‘dilution effect’ perspective, in which loss of vertebrate diversity is associated with high risk. Unfortunately, this dialectic confuses the objectives of each perspective and distorts relevant evidence.

WL conflate ‘forestation’ and ‘biodiversity’, epitomized by their repeated use of the term ‘forestation and/or biodiversity’ ([1] pp. 240 and 244). Although forest is required for blacklegged tick populations, host diversity within these forests and associated landscapes varies dramatically. Reforestation during the 20th century of agricultural land has been linked to LD emergence, but more recent forest fragmentation has been linked to increased LD risk (reviewed in [2]).

WL engage in fallacious reasoning, arguing that, because LD would disappear if all biodiversity were eliminated, increasing biodiversity amplifies LD. Indeed, their characterization of the ‘traditional’ approach leads them to the untenable position that the most effective means of reducing LD risk is to deforest the landscape, an option that they find ‘inadvisable’ ([1] p. 246). It also leads them to state ([1] p. 246) that, ‘most evidence currently available points to a monotonic increase in disease risk with increasing biodiversity’, a statement utterly devoid of support (and unrefenced). WL are critical of the ‘dilution effect’ perspective, contending that it ‘is part of a growing effort to market conservation actions based on the utilitarian services that biodiversity can provide for human society’ ([1] p. 246). We disagree that efforts to use scientific understanding to inform policy should be considered ‘marketing’.

WL’s discomfort with the dilution effect stems from a series of mischaracterizations. First, they contend that the dilution effect ‘is premised on the unreasonable belief that biodiversity must always benefit human society’ ([1] p. 243). On the contrary, the dilution effect literature clearly shows that biodiversity can either dilute or amplify disease risk, specifying the conditions under which each would be expected [3,4]. Current evidence that high diversity dilutes far more often than it amplifies, at scales from local to global, is strong [5–7], but we find no assertions that this pattern is universal. Second, they argue that LD risk is tightly coupled to abundance of deer and, therefore, is unrelated to the remaining host community. The basis for this argument is a study [8] in which deer were eradicated from a small island (Monhegan Island, ME), after which tick populations declined dramatically. Unfortunately, these results have little applicability to most of the northeastern USA. On Monhegan Island, no other hosts for adult ticks are present [8], so it is not surprising that the elimination of the only adult-tick host caused the demise of ticks. Outside of small islands, however, other mammals host adult ticks, and complete eradication of deer is not feasible; hence, the relation between deer abundance and LD risk is often weak or absent [2,9]. Third, WL equate biodiversity with species richness. In fact, the dilution effect literature has long argued that species composition of the host community (a measure of biodiversity) is a better predictor of LD risk than is species richness alone [10,11]. Importantly, whenever community assembly or disassembly is nonrandom, community composition will change predictably with changing richness. This is probably why species richness alone is often a significant predictor of LD risk [2]. Fourth, WL imply, incorrectly, that the dilution effect assumes a linear relation between biodiversity and LD risk. In fact, prior studies specify the conditions under which this relation is expected to be asymptotic or unimodal [2,12]. Fifth, WL claim that the dilution effect is a guaranteed outcome of the model used by LoGiudice et al. [10], but this too is false. Ostfeld and LoGiudice [11] showed that this same model produces an amplification effect (increased disease risk with increasing diversity) when species are added in random sequence. By contrast, a dilution effect occurs when white-footed mice (Peromyscus leucopus) are the first species to colonize and last to disappear, a phenomenon repeatedly confirmed by empirical study.

Attempts to integrate biodiversity with other factors as determinants of disease risk are to be encouraged, but they should combine a sophisticated understanding of theory, natural history, and quantitative methods. Recent approaches (e.g., [6,7]) might serve as models for future efforts.

References

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