Jiang *et al.* report on the observation of interface-induced skyrmionic bubbles at room temperature in a magnetic layer grown epitaxially on a material with large spin-orbit coupling. The role of the substrate is twofold: First, it provides the interface at which the DM interaction is induced; and second, by driving a lateral current through the substrate, the charge is converted to a vertical spin current due to the spin Hall effect (*12*). The resulting spin-orbit torques act on the magnetic layer and induce a movement of the micrometer-sized skyrmions. All of them move in the same direction, which means they all have the same twist, imposed by the DM interaction.

The generation of individual skyrmions is realized by inhomogeneous spin-orbit torques occurring behind a geometric constriction (see the figure, panel C). Jiang *et al.* propose an intriguing model of how the bubbles are pinched off from a magnetic stripe domain driven through the constriction, and draw an analogy to the formation of soap bubbles. Thus, for the writing of a skyrmion into a track, a perpendicular geometry could be used, and with a lateral movement in the same direction, it should be possible to delete single magnetic objects by moving them out of the track (see the figure, panel D), unifying all relevant operations in one device.

With the goal of achieving a skyrmion racetrack in sight, there are several issues that still need to be resolved. The precision of the generation and the degree of control over each magnetic skyrmion need improvement. Jiang *et al.* demonstrate the generation of many skyrmionic bubbles at a time, and in their sample the movement is strongly influenced by local pinning due to the inhomogeneity of the film. Furthermore, the size of the skyrmions needs to be reduced to be competitive with present storage densities. Future research will show whether nanometer-sized skyrmions are possible in materials relevant for applications. However, such a small size of a skyrmion will not only limit the methods to investigate them but also challenge the mechanism and design of an appropriate readout device (see the figure, panel E). ■

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ECOLOGY

Is biodiversity good for your health?

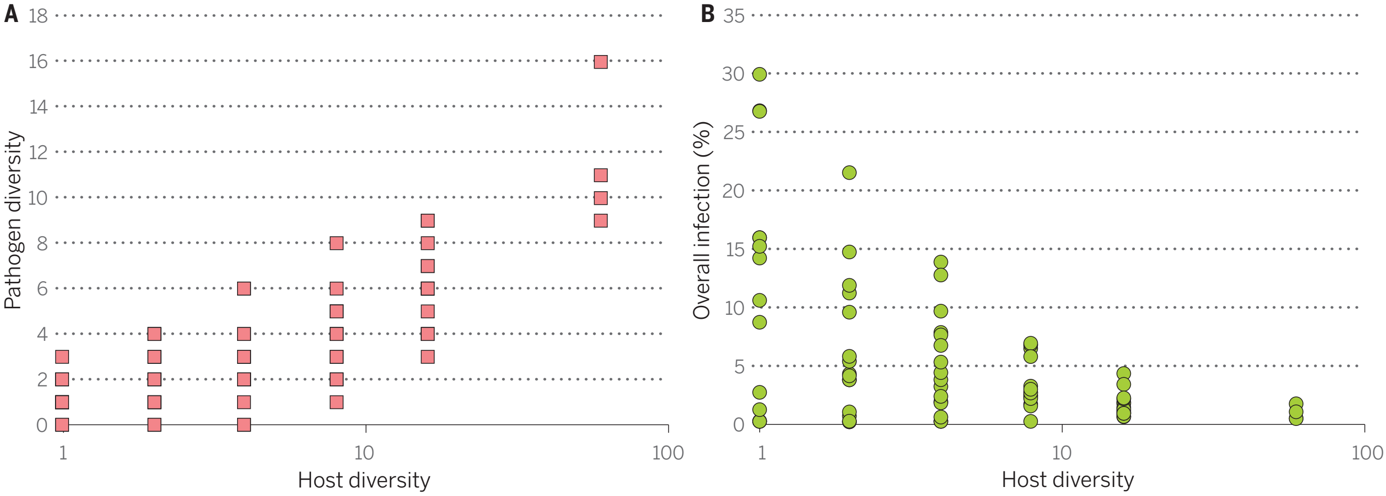
Disease incidence is often lower in more diverse communities of plants and animals

By **Felicia Keesing** *and* **Richard S. Ostfeld**

On a floodplain of the River Saale near Jena, Germany, grassland plants are naturally bombarded by spores of pathogenic fungi. But whether or not those fungi cause infection turns out to be largely about the neighborhood: Plants on highly diverse experimental plots have much lower levels of infection than plants grown in monoculture (*1*) (see the photos). The pathogens, it appears, are less likely to encounter their optimal host on the more diverse plots, which reduces disease prevalence and incidence. This protective effect of diversity has been found in many studies, not just for plants but also for diseases afflicting humans and wildlife. It has remained unclear, however, whether this observation holds generally (*2*, *3*). In a recent paper, Civitello *et al.* addressed this question in a rigorous meta-analysis of diversity-disease relationships (*4*).

Civitello *et al.* examined 202 results from studies of 61 parasites infecting both plants and animals. They found a clear and consistent reduction in disease prevalence when diversity was high. The effect was as strong for parasites that infect humans as for those that infect only wildlife and was seen both for microparasites (pathogens such as bacteria and viruses) and for macroparasites (such as trematodes and nematodes). The protective effect of diversity for human diseases is particularly important because it refutes the results of a previous meta-analysis

The benefits of diversity. Civitello *et al.* have performed a meta-analysis of studies of 61 parasites infecting both animals and plants. The results show that disease prevalence is often higher in less diverse systems (top, barley monoculture) than in more diverse systems (bottom).



Effects of plant diversity on fungal pathogens in Jena, Germany. Plots with higher host diversity generally had a higher diversity of pathogens (**A**) but also lower levels of overall infection (**B**). Drawn from data provided in (*1*). Note that the *x*-axis is on a log scale.

of only six parasites, which found no consistent effect of diversity (*5*). Civitello *et al.* also found that the effect of diversity was equally strong for both experimental and comparative studies. This is a key result, because although it has been known for some time that diversity can reduce disease in carefully controlled and constructed experimental communities, it has been less clear whether the effect translates into natural systems.

The widespread negative effect of high diversity on disease transmission documented by Civitello *et al.* suggests a consistent underlying mechanism that applies to diseases of humans, wildlife, and plants. One part of that mechanism has been hinted at in previous work: The best hosts for multihost pathogens are often abundant, widespread, and resilient species (*6*–*8*). A recent study by Han *et al.* (*9*) provides strong evidence for this pattern. The authors set out to determine what characteristics make some species good reservoir hosts of diseases that infect humans. Working with a database of 2277 rodent species and 66 pathogens, they used machine-learning algorithms to explore what aspects of host life history, physiology, behavior, ecology, and biogeography are associated with pathogens that spill over into human populations. Several patterns emerged. Good reservoir hosts have broad geographic ranges that contain comparatively few other species. They also reach maturity at a young age and have a short gestation period and a large litter size, all characteristics of what is often called a fast life history.

Why might species with fast life histories be good hosts for pathogens? Two hypotheses have garnered some support. First, parasites and pathogens that infect multiple species might evolve to exploit those hosts that they are most likely to encounter in nature—in other words, the most widespread, abundant hosts. Species that are widespread and abundant often have fast life histories (*10*). The second possibility is that fast-living hosts tend to be less resistant to, or more tolerant of, infection because of how they allocate their immune defenses (*11*).

Whatever the causes of these patterns, the consequences are intriguing. Species with fast life histories are often resilient to disturbance and are thus likely to persist in the face of environmental stressors that cause other species to decline or disappear (*12*). Together, these two patterns—the resilience and ubiquity of species with fast life histories and their odds of being high-quality hosts for parasites and pathogens—may interact to produce the diversity-disease relationship. As biodiversity is lost from ecological systems, the species most likely to persist may tend to be those most likely to harbor and transmit pathogens at high rates.

But won't ecological communities with a higher diversity of hosts also have a higher diversity of parasites and pathogens? And might a higher diversity of parasites and pathogens counteract the disease-suppressing effects of high host diversity? These questions have been at the heart of critiques of the diversity-disease relationship (*3*, *13*). An answer may be emerging. Back on the German floodplain, Rottstock *et al.* (*1*) found that the diversity of fungal pathogens was indeed higher in the plots with higher plant diversity, but the level of infection was lower (see the chart). Even the prevalence of coinfection—the simultaneous infection of a host with more than one pathogen—was lower where host and pathogen diversity were high. If this pattern of reduced disease with increased parasite diversity occurs in other systems, it would help to resolve what some have seen as a paradoxical relationship between diversity and disease.

Many fascinating questions remain about the relationship between diversity and disease, but Civitello *et al.*'s meta-analysis demonstrates that diversity frequently reduces disease. At the same time, Han *et al.* (*9*) have identified traits in rodents that strengthen the connection between life history and the probability that a species will be a reservoir for human pathogens. Case studies to evaluate diversity effects in specific disease systems will remain important, as will the pursuit of mechanisms that underlie the general pattern. Perhaps the most important question that remains is about the application of this knowledge to public policy. Should health protection be added to the long list of ecosystem services provided by biodiversity (*2*)? The meta-analysis by Civitello *et al.* suggests that it should. ■

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ILLUSTRATION: ADAPTED FROM DATA IN (*1*)

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