



Fig. 1 Termite mounds are conspicuous features of paddy fields in Laos and Cambodia. Their soil is used as amendment because it is enriched in organic matter and clay while its animal and plant diversity are used by the population as source of food or medicine (Miyagawa et al., 2011) (Photo: P. Jouquet, Cambodia, 2008).



Fig. 2 Termite mound made by *Odontotermes obesus* in Southern India (Photo: P. Jouquet, 2016). The offerings and the statuette show that termite mounds are used as a means to express one's devotion to Shiva.

Disclosure of interest The authors declare that they have no competing interest.

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<https://doi.org/10.1016/j.crv.2019.09.012>

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Using contact networks and next-generation sequencing for wildlife epidemiology

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Horizontal transmission of infectious diseases is strongly determined by the contact network between hosts [1]. Environmental and ecological variables that define the probability of contacts between individuals, such as food resource quality and quantity, have been largely neglected in models studying the dynamics and epidemiology of wildlife diseases.

In this work, we used pollinators and their pathogens to study the role of contact networks in a natural multi-host pathogen community. In agricultural landscapes, wild flowers are the most important food resources of insect pollinators such as bees and flies. To boost yields of agricultural crops and ensure pollinator conservation, the UK Environmental Stewardship scheme prompted farmers to grow pollinator-friendly wild flower margins along their fields (Fig. 1). Wild flower margins (Fig. 1) have proven to be efficient to increase both density and diversity of bees in agricultural areas, and to increase pollination success of surrounding crops.

As the rate of disease transmission should increase with host density, we hypothesize that the success of wild flower margins may generate hubs for pathogen exchange within the bee community. Several recent studies have illustrated the frequent transmission of infectious diseases between managed and wild bees, potentially via the shared use of flowers [2]. However, the environmental and ecological drivers of disease dynamics between pollinators remain uncharacterised [3]. To understand the role of flower density and diversity for bee disease transmission, we reconstructed high-resolution plant–insect visitor networks from flower visitation data collected in ten farms in Southern England (five farms participating in the scheme vs. five control farms), as well as a record of bee pollen collection to describe the resource bees were exploiting (pollen vs. nectar).

When analysing the plant–pollinator networks, we found that flower density and diversity strongly define pollinator density and foraging behaviour, and influence the structure of indi-





Fig. 1 Example of wild flower margin in one of our site. Photo: Vincent Doublet.

rect ‘contact networks’ among pollinators (via the use of flower species). Particularly, we found flower diversity to be positively correlated with the reduction of niche overlap between insect species. This response of insect pollinators suggest that wild flower margins with high plant species richness may reduce insect competition for resources, and potentially reduce the risk of inter-specific disease transmission by supporting diverse diet for insects exploiting different flowers. To test the effect of plant diversity on pathogen dynamics in bees, we sampled pollinators on these farms and characterized their virome by deep transcriptome sequencing. We are now combining these environmental data to virus discovery in order to reveal the impact of the agri-environmental scheme on viral dynamics. Ultimately, we aim to identify environmental (flower density, agricultural practices) and ecological factors (plant taxa, insect community assemblage) that significantly enhance the transmission of plant and pollinator viral diseases within our model to eventually improve agricultural practices and wildlife management.

Disclosure of interest The authors declare that they have no competing interest.

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<https://doi.org/10.1016/j.crv.2019.09.013>

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Microbial nutrient factories in insects on extreme diets

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Insects are renowned for their capacity to specialize on a wide diversity of diets, many of which are nutrient-poor or nutritionally unbalanced. For example, various insects feed through the life cycle on wood, vertebrate blood, plant sap and other extreme diets that are variously deficient in vitamins, sterols and essential amino acids. These insects circumvent the fundamental “rules” of animal nutrition because they



possess symbiotic microorganisms that overproduce the limiting dietary nutrients. Many associations between insects and microorganisms are evolutionarily ancient and involve the exquisite coevolution of metabolic function in the insect and microbial partners, including the restructuring of microbial metabolism as nutrient factories for the host (Fig. 1). These insects include major pests and vectors of animal, human and crop disease agents. Their dependence on specific microorganisms offers novel routes for the control of these globally important insect pests.

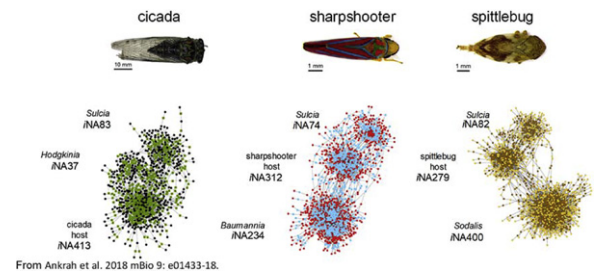


Fig. 1 Metabolic networks in xylem feeding insects.

Disclosure of interest The author declares that she has no competing interest.

Further reading

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<https://doi.org/10.1016/j.crv.2019.09.014>

Session IV. Interaction with other organisms

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The insect reservoir of diversity for viruses and antiviral mechanisms

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Insects originated more than 400 million years ago and have undergone since then an extraordinary diversification, associated with many spectacular innovations, such as flying or establishment of social societies. They have colonized all terrestrial ecosystems, and are exposed to a broad range of pathogens, including viruses, bacteria, fungi, and parasites. Like all animals, insects rely on innate immunity to control infections. Innate immunity is the first layer in host-defense in animals. It involves receptors sensing the presence of infectious microorganisms and triggering signaling that leads to the expression of genes coding effector molecules, which concur to counter the infection. In vertebrates, a subset of genes induced encode cytokines and coreceptors that activate a second layer

