Pathways for Novel Epidemiology: Plant–Pollinator–Pathogen Networks and Global Change

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Review

Pathways for Novel Epidemiology: Plant–Pollinator–Pathogen Networks and Global Change

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Multiple global change pressures, and their interplay, cause plant–pollinator extinctions and modify species assemblages and interactions. This may alter the risks of pathogen host shifts, intra- or interspecific pathogen spread, and emergence of novel population or community epidemics. Flowers are hubs for pathogen transmission. Consequently, the structure of plant–pollinator interaction networks may be pivotal in pathogen host shifts and modulating disease dynamics. Traits of plants, pollinators, and pathogens may also govern the interspecific spread of pathogens. Pathogen spillover–spillback between managed and wild pollinators risks driving the evolution of virulence and community epidemics. Understanding this interplay between host–pathogen dynamics and global change will be crucial to predicting impacts on pollinators and pollination underpinning ecosystems and human wellbeing.

Highlights

Flower sharing amongst pollinator species represents a conduit for interspecific insect pathogen transmission.

Plant–pollinator network structure and species traits shape pathogen dynamics in the pollinator community.

Global change (climate change, invasive species, agricultural intensification, and urbanisation) can modulate species interactions, host susceptibility, and pathogen virulence, thereby creating novel epidemiological risks.

Multiple global change effects can interact in synergistic or antagonistic ways, creating additional risks and complicating predictions of pathogen dynamics and evolution.

Flower-mediated insect pathogen transmission provides a model framework to understand the interplay of interspecific pathogen dynamics under global change.

Pollinator Disease in the Anthropocene

Wild and managed pollinators provide crucial ecosystem services and diverse economic and noneconomic values that support ecosystem health, agricultural production, and human wellbeing [1]. Multiple, potentially interacting, anthropogenic pressures threaten and drive declines in managed and wild pollinators [1,2]. Intra- or interspecific spread of pathogens (viruses, microsporidian fungi, bacteria, protozoa, and parasitic invertebrates) in novel host communities assembled under global changes are a likely emerging threat [1,3]. Horizontal transmission of pathogens occurs when individual insects share flowers providing nectar and pollen resources [4,5] as well as during social interactions [6] and mating, the latter leading to vertical transmission between generations [7].

Emerging zoonotic diseases due to pathogen host shifts are a current and growing threat under anthropogenic global change [8]. Although interspecific pathogen transmission is more limited than intraspecific spread in Hymenoptera [9], phylogenetic studies indicate that pathogen host shifts are frequent [8]. Why particular pathogens jump to some host species, but not others, remains only partly understood [10]; but the lack of host–pathogen coevolution means these events can elevate mortality, reduce fitness, and alter population and community structure [11]. Multi-host systems can drive the evolution of pathogen virulence and infectivity [12], and so it is critical to understand how the mechanisms of interspecific host–pathogen dynamics impact on disease [8], including spillover between wild and managed bee species.

Given the importance of pollinators and pollination [1], it is vital to understand how anthropogenic pressures such as land use change [13], chemical stressors [14], altered habitat resources [15],

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climate change [16], and alien species [17] modulate pollinator–pathogen dynamics. Such pressures may raise pollinator disease risk by altering the dominance, distribution, and interaction of primary or secondary host species and asymptomatic pathogen vectors. Understanding this interplay between host–pathogen dynamics and global change will be crucial to predicting impacts on pollinator populations, communities, and pollination functions underpinning ecosystems and human wellbeing (Figure 1).

Disease and the Plant–Pollinator–Pathogen Network

The distribution, diversity, and abundance of floral resources in a landscape are key to concentrating or diluting interspecific pollinator interactions and potential pathogen transfer. Pollinator species are obligate flower visitors, foraging and feeding on pollen and/or nectar for at least part of their life cycle, and therefore both compete for and share floral resources in time and space [1]. This makes flowers hotspots of co-occurrence, direct contact, or indirect interaction (mediated by pollen or nectar) between pollinator species in a habitat or landscape [4,5,15,18].

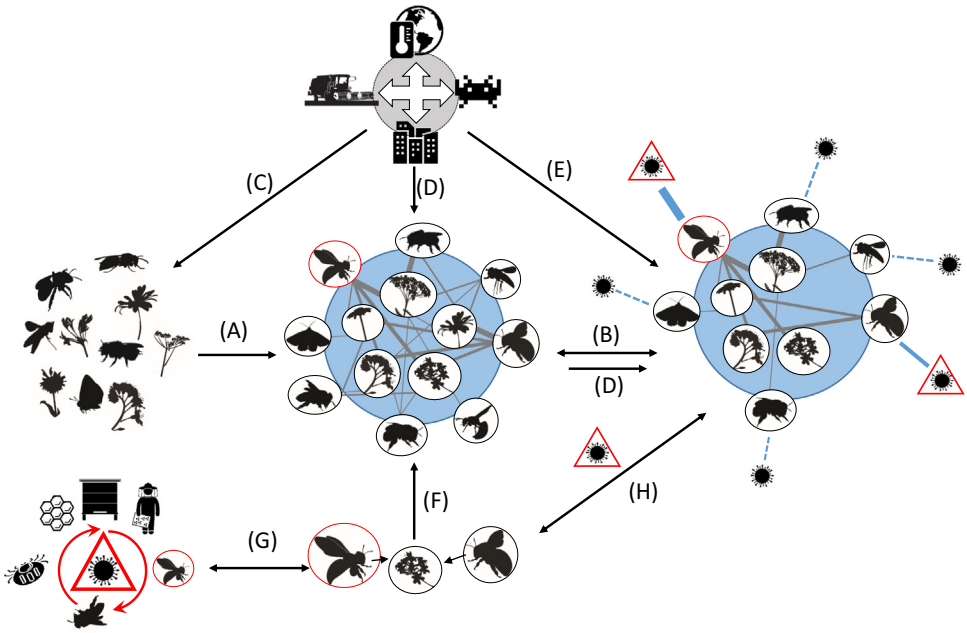

Figure 1. Potential Impacts of Anthropogenic Environmental Pressures and Bee Management on Flower-Mediated Pathogen Transmission between Pollinator Species. Plant–pollinator community interactions are determined by the species pool (A). Pathogen transmission through shared flowers is mediated by intrinsic species traits and plant–pollinator interaction network structure, with potential network rewiring following differential disease impacts on pollinator species (B). Environmental pressures (climate change, invasive species, agricultural intensification, and urbanisation, see Figure 2) affect pathogen dynamics directly by introducing new species differing in disease resistance or vectoring capacity (C). Environmental pressures may also rewire the network through changes in plant–pollinator species composition, abundance, behaviour, and phenology (D) or introduce novel pathogens and/or alter infectivity, virulence, and susceptibility in pollinator populations (E), unbroken blue lines show established pathogenicity, broken blue lines show hypothetical vectoring or pathogenicity. The Western honey bee *Apis mellifera* (red circle) is the most widespread managed pollinator species and plays a dominant role in plant–pollinator network structure (F). *A. mellifera* may act as a superspreader (H) exacerbated through intra-colony co-infection dynamics (G), as in the case of Deformed wing virus (DWV) which became more prevalent and virulent through association with *Varroa destructor* parasitism in managed hives (G). Honey bees can thus introduce pathogens into the wild pollinator community (spillover), or contract them from co-occurring wild pollinators (spillback) (H), which may drive further evolution of virulence (G, H). Icons sourced from: https://thenounproject.com/CC BY-NC-ND 2.0: (Iluisa Iborra, Yu Luck, Les vieux garçons, Gan Khoon Lay, Blair Adams, Nareerat Jaikaew, and Prosymbols) and https://www.shutterstock.com (a free trial).

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Accordingly, flowers can serve as hubs of pathogen transmission within and between species and pathogens may 'travel' through the network of plant–pollinator interactions, potentially crossing species boundaries and infecting new hosts [18]. Studying this tripartite plant–pollinator–pathogen network can therefore help to identify potential flower-mediated transmission routes and elucidate how anthropogenic disruptions to network structure may alter or create novel pathogen dynamics.

Plant–Pollinator Traits Governing Pathogen Transmission

Pollinator species traits determine their susceptibility to different environmental pressures and their potential to transmit pathogens [19]. Species' diet breadth and preference may modulate the probability of encountering pathogens in nectar/pollen, on infected hosts, or deposited on flowers. Relatively specialised pollinators that forage on few plant taxa may have a lower exposure risk than species with broader floral diets. Less specialised species function as generalist pollinators in interaction networks, being highly connected to multiple forage plant species and thus indirectly to other pollinators [13]. Consequently, generalists may have a greater probability of pathogen exposure or infection (novel or otherwise) and accordingly vector multiple pathogens across the network. Generalist species, however, may differ in foraging preferences and individual foragers of a generalist species may select for specific subsets of forage plant species, both of which may mitigate the risk of flower-mediated pathogen spread through a community [20]. Moreover, generalist pollinators may dilute the risk of transmission per plant species [13] and an emergent community epidemic by spreading their flower visits over multiple plant species.

Sociality is a key trait affecting disease dynamics. Eusocial pollinators (*Apis*, *Bombus*, Meliponini) play a disproportionately large role due to their life history, cooperative brood care, and overlapping generations in densely populated colonies exacerbating intra-colony disease spread [15]. Along with their generalised floral diets and long flying season bridging the phenology of other pollinator species, eusocial bees likely elevate the probability of interspecific contact and pathogen spillover. However, eusocial species may also mitigate the risk of increased pathogen burden through social immunity (e.g., behaviours to combat disease such as removing diseased larvae and dead workers from the colony [21]). Elucidating how gradients in foraging traits (e.g., pollen uptake and deposition rates, pollen transportation mode), colony sizes, and trait plasticity (e.g., facultative sociality in Halictidae) among taxa affect pathogen transmission, remains largely unexplored and is an important future direction for pollinator eco-epidemiology.

Plant traits may also modulate pollinator disease risk, with the potential for both facilitation and inhibition of pathogen transfer [5]. Floral architecture, by constraining access to pollen and nectar to pollinator species with the requisite morphological adaptations, filters insect visitor interactions and contacts [5]. Plants with structurally simple, large floral displays (e.g., Apiaceae, Rosaceae) attract numerous pollinator species and may serve as hubs facilitating interspecific pathogen transmission [22]. Additionally, plants with a more central position in a plant–pollinator network often contain a high pathogen load [23]. More specialised plants with complex flower structures requiring intimate and sustained contact with a specialised pollinator may limit transmission to a particular species or functional group [5]. Alternatively, other floral characteristics, such as organic volatiles produced by plants to attract mutualists, may be toxic to certain pathogens [24]. Nectar and pollen rewards may contain phytochemicals that inhibit pathogens when consumed by the host (e.g., reducing *Crithidia* sp. infection in bumble bees [25]), potentially reducing pathogen loads and community spread. Combined with limited pathogen resistance to environmental degradation (e.g., temperature, UV-radiation), such plant traits may limit pathogen survival on or within floral resources and subsequent pollinator infections [26,27].

Host–Pathogen Interactions and other Biota Affecting Disease

The interaction between the physiology of a pathogen and alternative hosts also has implications for epidemiological dynamics at population and community levels. Certain pollinator individuals or species may be immune to a particular pathogen or function as asymptomatic vectors, whereas others suffer from disease, leading to physiological stress, altered behaviour, and reduced fitness or death [28]. Additionally, pathogen infections may differ in their effects according to the host insect nutritional status, influence of other stressors, and environmental context [29].

While pathogen research in pollinators is heavily biased towards honey bees and bumble bees, recent studies point to broader pathogen host ranges, such as *Crithidia bombi* infections in multiple bee families [30]. Pathogen strains or taxa can behave as specialists or generalists in terms of host range, virulence, and infectivity, affecting their fitness and ability to cross species boundaries [28]. Single-stranded RNA viruses tend to pose the highest risk of co-infecting multiple species due to their high mutation rate, short generation time, and ability to cross phylogenetic boundaries [31–33]. Multiple co-infections of pathogens and parasites (RNA viruses, microsporidia, Acari) may further alter dynamics. For example, colonies of *Apis mellifera* infested with *Varroa destructor* mites can show a higher viral load and virulence of the RNA virus Deformed wing virus (DWV) [34]. Alternatively, pathogen co-infections may be asymmetric with intra-host competition inhibiting infection and disease manifestation [35].

Pathogens may also alter pollinator host behaviour to increase transmission potential. For example, bumble bees infected with trypanosomatid parasites defecate more frequently on flowers thus spreading the pathogen [27] and fungal pathogens may alter floral scent to lure pollinators [36]. Certain pathogens (e.g., *Nosema* spp.) increase their infectivity by forming cysts or spores that promote environmental persistence and probability of encountering susceptible hosts [37].

Beyond pathogens, other biota (e.g., gut microbiota) interacting with the host in mutualistic, commensal, or antagonistic ways, may modify pathogen dynamics. *Lactobacilli* inadvertently collected with pollen during flower visits may improve bee resistance to pathogens [38]. Arbuscular mycorrhizal fungi infecting plant roots can interact with soil fertility to alter nectar chemistry (e.g., alkaloids) in ways that influence pathogen prevalence [39]. Interactions between virus-vectoring and non-vector herbivores mediated by changes in plant secondary chemicals (e.g., reduction of flavonoids) can indirectly influence plant virus transmission [40]. It remains unknown whether similar complex trophic interactions might affect pathogens infecting pollinators.

Architecture of Plant–Pollinator Networks Governs Potential Pathogen Dynamics

Plant–pollinator networks describe the community of interacting species [41,42]. The pool of species traits in a habitat or landscape, pollinator behaviour, metabolic and dietary needs, and the phenological synchrony of potential mutualisms dictate the architecture of a network [43] (Figure 1A). Plant–pollinator networks are typically asymmetrical in form (Box 1). Specialist pollinators tend to forage on a limited number of plant taxa (e.g., within a single family) that represent a subset of the plants visited by generalist species leading to a nested structure of the community network [41,42]. Plant–pollinator networks can also consist of weakly interlinked subsets of species modules that comprise sets of strongly interacting species [44]. This modularity can arise from convergent trait sets and mutualist coevolution, caused for example by inter- and intraspecific competition among generalist pollinators, driving flexible foraging at the individual level and concentrating visitation on a subset of the available floral species [45].

Stochastic or environmentally driven variation in this fundamental process of community assembly (Figure 1A,C,D) results in different network structures (Box 1) that provide variable transmission

routes for pathogen propagation through the community (Figure 1B). Highly connected networks with a large diversity or number of interactions may elevate interspecific contact and potential pathogen transmission [46], or decrease pathogen prevalence due to a dilution effect [13], the latter of which may explain lower disease prevalence in diverse pollinator communities [47]. Highly nested networks may result in increased pathogen persistence [22], with generalist species serving as hubs of pathogen spread to specialist species. Increased modularity should buffer against risk of flower-mediated pathogen spillover and spread across entire communities, by compartmentalising pathogens and disease outbreaks within modules [13,48]. As a corollary, pollinator species within a module may be at increased risk of pathogen spillover from strongly interacting species (e.g., honey bees and bumble bees) with high pathogen loads [48].

Plant–pollinator–pathogen network structure will not be static, however, but dynamically shift with intra- and inter-annual temporal shifts in community composition and interactions [49] (Box 2). Foraging plasticity of many pollinator species can rewire interaction networks [50,51] that may affect host–pathogen exchange and dynamics [15,52]. Even in modular networks, generalist individuals may switch floral fidelity during their lifespan potentially enhancing pathogen spread between network modules [20]. Longer-term species turnover and species population dynamics can also rewire interaction networks through species extinctions, range extensions, and uptake of vacant niches [53,54]. While network rewiring lends stability to plant–pollinator networks [54], it offers a mechanism by which novel interactions may lead to pathogen shifts and microevolution. The relative contribution and interrelationships of different network properties [55], and the precise effect on host–pathogen dynamics, remain key questions in pollinator epidemiology (Box 2). Recent advances in predicting the structure and dynamics of plant–pollinator interactions by considering trait-matching of species [56–58] promise a route towards a more mechanistic understanding of pathogen transmission in plant–pollinator networks [22] (Box 2).

Anthropogenic Environmental Change

Aside from their separate impacts on pollinators and flora [1,2], anthropogenic environmental pressures are likely to alter plant–pollinator–pathogen dynamics [59], directly or via their synergistic or antagonistic effects (Figure 2). These global change pressures and their interlinked effects are ultimately caused by common drivers (e.g., human population growth, resource consumption) making it very difficult to disentangle and rank their relative importance [1]. Impacts of pressures may occur through apparent competitive and facilitative interactions [53,60,61] driven by altered abundances, diversity, or multispecies interactions in plant–pollinator–pathogen networks. We

Box 1. Key Network Metrics affecting Pathogen Transmission

Plant–pollinator interactions can be displayed as bipartite networks, in which species are nodes and interactions connecting lines. Connectance, nestedness, and modularity (Figure I) are arguably the most important metrics predicting pathogen transmission throughout a network [13]. While often used for unweighted (binary) networks, indices exist for these metrics that can characterise weighted networks (i.e., where the interactions are weighted by their frequency), which is the most common and most accurate way to display plant–pollinator networks.

Connectance is the proportion of realised ecological interactions to potential ecological interactions. Hence, a plant–pollinator network containing five pollinator species and ten plant species, with ten realised interactions, has a connectance of 0.20, since 50 interactions are theoretically possible.

Modularity refers to the division of a network in separate modules, which have a high within-module connectance, but few interactions between modules. As for nestedness, multiple indices exist to express the level of modularity. Newman's modularity (Q), for example, is calculated as the fraction of interactions within each module minus the fraction of expected interactions in a random network with the same connectance [115].

Nestedness in bipartite networks is described as the tendency for specialist species to interact with generalist species, resulting in specialist species' interactions being a subset of generalist species' interactions [116]. There are several nestedness indices, of which nestedness metric based on overlap and decreasing fill (NODF) is considered the most robust [117].

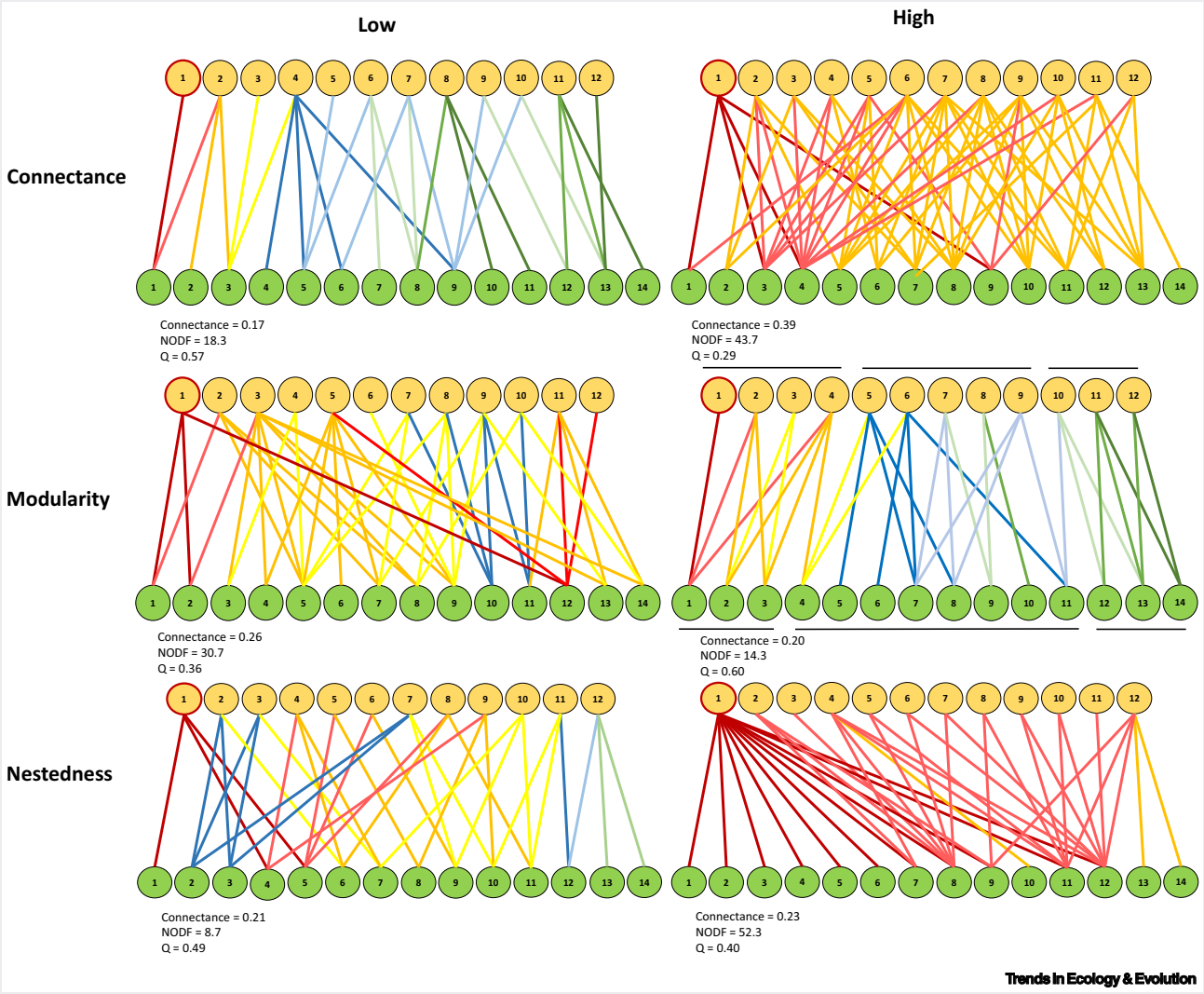

Figure I. Example Unweighted (for Simplicity) Networks Created by Randomly Compiling Plant–Pollinator Networks at a Predefined Connectance (C = 0.15 in Network with Low Connectance; C = 0.40 in Network with High Connectance; C ≈ 0.20 in Other Networks). Networks are composed of 12 pollinator species (yellow nodes) and 14 plant species (green nodes). Possible routes of disease spread, starting at pollinator species 1 are displayed by indicating path length between nodes (1, dark red; 2, light red; 3, orange; 4, yellow; 5, dark blue; 6, light blue; 7, light green; 8, green; 9, dark green). High connectance and nestedness may increase disease spread, while high modularity may keep disease spread limited to the affected module. In the network with high modularity, the three modules are indicated by horizontal lines. In the highly nested network, specialist pollinators visit a subset of plants visited by generalists. Abbreviations: NODF, nestedness metric based on overlap and decreasing fill; Q, Newman's modularity.

expect that pathogen spread within a pollinator population or interspecific spillover may be reduced or exacerbated by: (i) invasions by novel species or subspecies [17] (Figure 1C); (ii) the extirpation of pre-existing populations or species [62] (Figure 1C); (iii) modification of the relative abundance of species and dominant interactions [63] (Figure 1D); (iv) disruption of species' phenology [64,65] (Figure 1D); (v) shifting species distributions at landscape, regional, or global levels [62]; or (vi) changing species physiology, altering disease resistance, infectivity, or virulence [17,66] (Figure 1E). While scientific and policy awareness of pressures on wild pollinators has increased and led to regulations at various levels protecting pollinators and their habitats, less regulatory

attention has been devoted to wild pollinator diseases and their interplay with managed bees and environmental change (Table S1 in the supplemental information online).

Climate Change

Climate change has the potential to cause changes in pollinator disease risk. Elevated temperatures may increase pollinator resistance or susceptibility to pathogens according to physiological constraints (e.g., thermal stress) and changes in immune response [66–68]. Local climatic changes may increase niche suitability for pathogens, facilitating range extensions and enhancing virulence or conversely, lower virulence and abundance of pathogens or their vectors [69,70] (Figure 1E). Similarly, as with human diseases [71], pathogen vectoring by range expanding

Box 2. Mechanistic Process-Oriented Modelling of Wild Pollinator Disease

Empirically detecting disease events in wild pollinator–plant–pathogen systems is challenging because of the tangled network of direct and indirect interactions between pathogens, plants, and pollinators. Agent-based spatially explicit models may address this complexity [118]. Mechanistic models have previously been used to upscale from individual life cycle, resource use, and behaviours, to population level impacts of stressors and diseases in managed bee colonies [119,120].

Forecasting the course and impact of disease in multispecies pollinator systems is another order of complexity [13]. Recent use of the susceptible-infectious-susceptible (SIS) epidemiological cohort framework illustrated how disease persistence was promoted by network nestedness and specialisation, while pollinator foraging selectivity, specialised mutualist pairs, and generalist flowers increased bee contact and disease spread [22]. Whilst addressing the key assumption of disease modelling of individuals mixing uniformly and randomly, this study overlooked how contacts can emerge from individual behaviour and life cycle or other trait variation among species. Integrating interaction networks [22,116] with dynamic pathogen infestation states creates temporally variable contact structures [121]. This may elucidate whether pollinator traits (e.g., diet breadth, feeding rates) or environmental heterogeneity (e.g., resource accessibility, clustering) limit or facilitate pathogen persistence or transmission [20]. Equally, they may identify methodological caveats to empirical detection of pathogens (e.g., sampling effort or observer's collection window), which can improve monitoring protocols and target experiments.

Mechanistic process-oriented modelling of wild pollinator disease in support of planning experiments and data exploration requires knowledge of emergent trait-based life cycles and foraging activities. Sampling for construction of tripartite networks will provide individual (plant–pollinator–pathogen) data (occurrence, abundance, and contact), which can be translated to individual-oriented model characteristics for experimentation.

Potential components for modelling of pollinator disease tailored to experimentation/observations with literature examples:

Pollinator Population Dynamics [13,119,120]

- Static populations
- Population-level reproduction rate
- Explicit population turnover by individual nesting and reproduction

Landscape Heterogeneity, Dynamic Landscapes, Explicit Forage Environment [20,122–124]

- Homogeneous landscapes
- Spatial habitat distribution using, for example, species distribution modelling (SDM)
- Landscape maps by fitness indices using resource needs and reachability
- Standardised and systematic landscape generation algorithms

Foraging and Visitation Behaviour, Pollen Collection [20,125,126]

- Diffusive foraging and kernel models
- Explicit traits and behavioural trade-offs
- Temporally varying individual-level preferences of food or habitat
- Emergent foraging behavioural responses by exploring spatial explicit structures

Virtual Insect Sampling: Simulated Data and Models Mimicking Real Species in Landscapes

- Model sampling methods as empirically tested
- Resample transect path in real landscapes

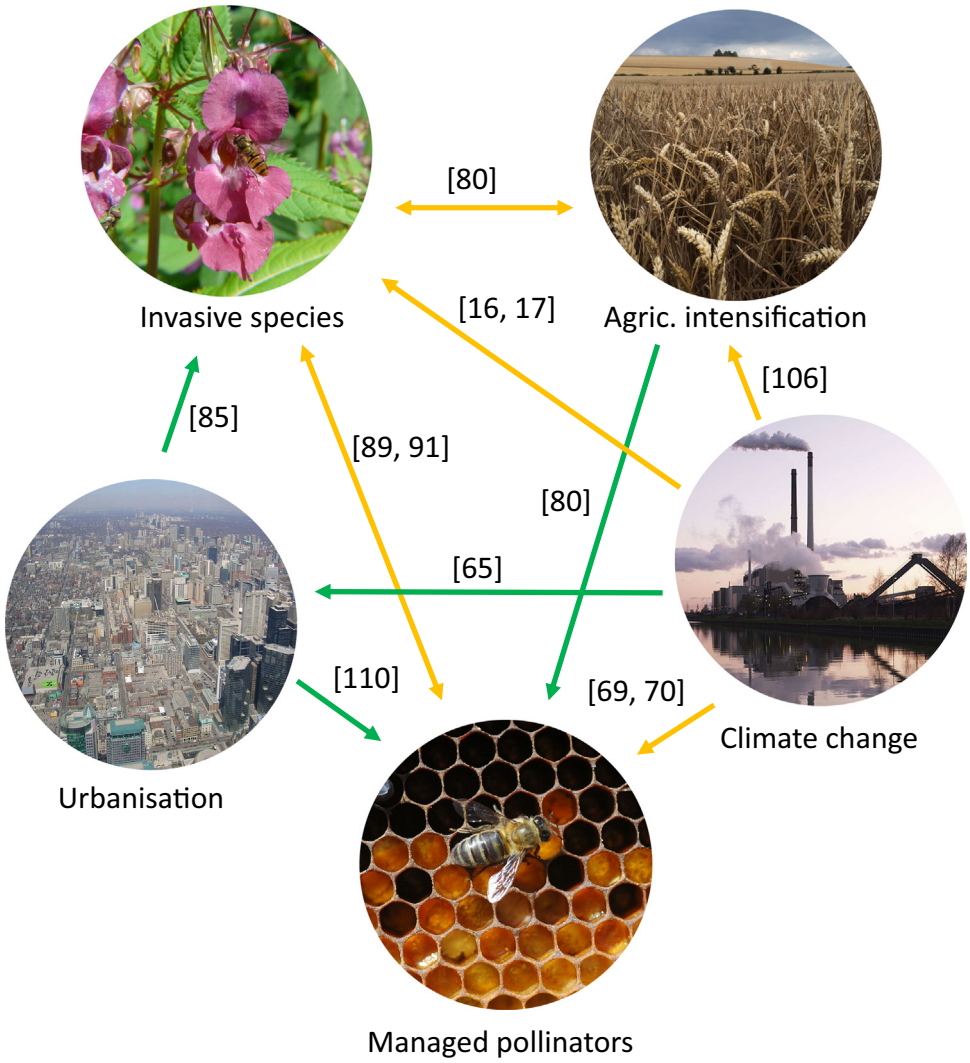

Figure 2. Interactive Effects between Environmental Pressures. Green arrows indicate synergistic effects, orange arrows complex, trait, and context-dependent effects. Arrow points in the direction of the effect. References mentioning the respective interactions are displayed in square brackets [16,17,65,69,70,80,85,89,91,106,110]. Concurring environmental drivers may reinforce each other, as is the case with climate change exacerbating the effects of urban heating, urbanisation leading to a higher pressure of invasive species, and higher density of managed pollinators, or intensive agriculture increasing transport and demand for managed pollinators. Conversely, most interactions between global change drivers have context-dependent effects on pathogen transmission, as is the case with climate change, which has far-reaching effects on agricultural practices and modulates potential for invasive species depending on species traits, or invasive plants, pollinators, and pathogens interacting in complex ways with managed pollinator behaviour, physiology, and epidemiology. Photographs by Dan Chapman, ©NERC, (WT-EN) Bulliver CC-BY-SA 1.0, Piscisgate CC BY-SA 4.0, and Arnold Paul CC BY-SA 3.0.

pollinators can introduce novel infections to native pollinator communities. Climate-induced plant extinctions may also affect flower-mediated pathogen transmission by shifting community structure to be dominated by plant species with different potential to act as hubs of pathogen transmission [72] (Figure 1D). Additionally, pollinator forage plants may be extirpated or produce less flowers or lower-quality floral rewards under changed climatic circumstances [73], causing nutritional stress and higher susceptibility of hosts [74].

Phenological or spatial mismatch of species interactions [64,75] due to life-history differences in tracking climate changes, or climate-induced species extirpations, or range shifts, may alter host activity and species relations [62,76]. Such mismatches, extinctions, and subsequent rewiring of networks [53,75], may change the prevalence of pathogens or parasites in pollinator communities and pathways to infection [77] (Figure 1D).

Non-random processes such as trait matching will modulate the impact of climate change on plant–pollinator–pathogen communities [75]. Overall, rarer species with narrow climate niches, high specialisation, low dispersal abilities, and restricted geographic distribution are likely to suffer most. Climate impacts may thus inflate network-level generality of interactions [53], possibly increasing pathogen spread, although this may be mitigated should specialist interactions be sufficiently robust to disturbance [57]. Climate change has been shown to increase nestedness and decrease modularity of plant–pollinator networks [78], indicating potential for increased pathogen prevalence [22] (Box 1).

Introduced Species

Invasive Alien Species

Anthropogenic translocations of species often lead to alien species invasions, which have had severe consequences for plant–pollinator populations and communities [2,11,17]. Species invasions and the novel contact opportunities they create present risks of pathogen host shifts, coinfections, and the evolution of virulence with detrimental effects on species lacking immunity or resistance [79]. Alien pollinators can directly compete with native species via interference and resource competition, or impact them indirectly by introducing novel pathogens [80] or enhancing pathogen transmission [81]. Potentially invasive pollinators (e.g., *Bombus terrestris*), often introduced deliberately or accidentally via the bee trade into regions outside of their native distribution range, are usually highly competitive generalist foragers with an extensive flight period that profoundly disrupt recipient communities [80,82]. Dominant invasive species may push native species to shift onto and compete for suboptimal and less abundant forage plants, potentially elevating nutritional stress and pathogen susceptibility [60].

Invasive plant species often provide copious floral resources dominating pollinator diets and rearranging interactions to disrupt the modular structure and increase the generalism and nestedness of the plant–pollinator network [83,84]. Consequently, invasive plant flowers can serve as potential hubs for pathogen transfer by concentrating pollinator activity and diversity [85] and reducing network modularity to facilitate pathogen spread throughout the community. Similarly, non-insect pollinated invasive plant species can concentrate pollinator activity and contact by suppressing native vegetation and limiting forage plant availability [86]. Invasive plants may also increase pollinator susceptibility to pathogens by supplying copious yet suboptimal pollen or nectar lacking essential nutrients or containing secondary compounds toxic to certain pollinator species [87]. In the longer term, invasive plants can lead to differential reproduction of native plant species due to facilitation or competition for insect pollination [88], potentially reconfiguring plant–pollinator interactions to produce a novel epidemiology (Figure 1D,E).

Invasions by other functional groups may also profoundly affect pollinator–pathogen dynamics. Invasive non-pollinating insects may affect pathogen dynamics by directly vectoring pathogens to shared plant species [32], exerting novel predation pressure upon pollinator populations [89] or reorganising the plant community through novel herbivores [90]. Such changes to food webs may affect pathogen circulation and interspecific processes (facilitation, competition) governing disease dynamics.

Managed Bee Species

Within their original and translocated ranges, honey bees and some bumble bees, stingless bees, and solitary bees, are managed for hive products and/or crop pollination services, with *A. mellifera* the most widespread and abundant managed species [1]. Apart from the risk of emerging disease in previously unaffected regions due to transport of managed bees (particularly *A. mellifera*, but also e.g., *Bombus terrestris* and *Bombus impatiens*) over large distances [1,11,80,91], bee management presents additional epidemiological risks. Decreases in managed *A. mellifera* genetic diversity, caused by selective breeding [92], may weaken *A. mellifera* immune responses [93] increasing population pathogen levels. Selective pollinator breeding could increase pathogen resistance or tolerance [94], the latter potentially exacerbating pathogen spillover. Managed *A. mellifera* are kept at higher colony densities than occur naturally, which together with their supergeneralism positions them as a central hub or a 'superspreader' reservoir host in plant–pollinator networks (Figure 1F–H) [95].

One consequence of the global translocation of *A. mellifera*, for pollination services and hive products was parasite and pathogen transfer (e.g., *V. destructor*, *Nosema ceranae*) from the original Asian host species, *Apis cerana* [11,96]. Parasitism of *A. mellifera* by *V. destructor* exacerbates transmission and increases prevalence of several RNA viruses including DWV infecting the honey bee [31,34] (Figure 1G), hence potentially spreading pathogens to these susceptible alternative hosts during foraging [74,97,98] (Figure 1H), while driving nutritional stress in wild pollinators by outcompeting them for scarce food. Weakening of the host's immune system by combined parasite and pathogen infections may induce rapid microevolutionary changes that increase virulence [31,97]. Moreover, this may be a dynamic process with multiple spillover and spillback events iteratively increasing the risk of pathogen exchanges and development of novel community epidemiology (Figure 1G,H). Such host shifts highlight the risks of species invasions for creating novel species interactions and epidemiology for populations and communities [11].

Conventional Agricultural Intensification

Conventional intensive agriculture is a globally important pressure on pollinators and pollination [1,2], that may modulate pathogen transmission or pollinator susceptibility. Generalist pollinators able to adapt to the cropped environment are usually less affected than more specialised species by landscape simplification and management intensification [99]. Use of herbicides, fertiliser, and destruction of seminatural elements reduce richness and abundance of floral resources providing nectar and pollen [1], which can lead to network generalism [100]. Consequently, plant–pollinator networks in intensively farmed areas tend to be generalised with low modularity and nestedness and high connectance [59,63]. Such a network architecture can either limit or propagate community-wide pathogen transmission (Box 1).

Intensively managed agricultural landscapes often hold transient or low pollinator population densities [13] that may reduce pathogen spread [101]. Conversely, the risk of pathogen exposure could increase if pollinator interactions become concentrated on the few remaining natural floral resource patches in intensive agricultural landscapes [1,86] (Figure 1D). Mass-flowering crops provide huge, transient pulses of pollen and nectar food resources that attract and provide nutrition supporting the fitness of large numbers of diverse pollinators [2,102]. Domination of pollinator interactions by mass-flowering crops could create infection hubs by concentrating the pollinator community, reducing pathogen transmission by spreading pollinator activity over a large continuous area of floral resource [15] (Figure 1D).

Mass flowering crops present additional potential risks of pesticide exposure that may also weaken pollinator immune responses, leading to increased pathogen load and virulence [14]. Recently, the fungicide chlorothalonil has been associated with high parasite loads in bumble bees and resulting

declines and range contractions [103]. Additionally, the paucity of floral resources in intensive agricultural landscapes may mean pollinators are unable to optimise their nutrient intake [104] and suffer nutrient deficits that compromise immune function and pathogen resistance [105]. Future changes in agricultural practices driven by climate change [106] may significantly reshape the agricultural landscape, possibly with large impacts on plant–pollinator–pathogen dynamics.

Urbanisation

Urban environments are increasing in extent worldwide. They have higher ambient temperature and levels of pollution (e.g., pesticides, heavy metals, and airborne particulates), large areas of impervious surfaces, and few semi-natural areas, but provide pollinator habitats in anthropic green spaces and gardens supporting a richness of native and alien plant species [107]. Multiple factors may affect pollinator–pathogen dynamics in urban areas where high pathogen loads have been detected in honey bees and bumble bees [93,108]. Similar to climatic influences at a continental or global scale, higher urban temperatures may drive the evolution of virulence in pathogens [17] or vector activity [71], creating local patterns of pathogen prevalence (Figure 1E). Urban heating may also drive phenological mismatch between plants and bees, due to differential adaptation to the novel microclimate [65]. Urban heat islands coupled to a proliferation of non-native horticultural and weed plant species, possessing intrinsic differences in flowering phenology, can extend the pollinator foraging season in urban areas [17]. This lengthened foraging season has potential impacts on pathogen transmission risk especially as levels of *V. destructor* and associated viruses in honey bees tend to peak later in season, exposing late-season active species to higher levels of pathogens [109]. Patchily distributed floral resources in urban landscapes increase pollinator interspecific contacts at shared flowers, which together with growing popularity of urban beekeeping (Figure 1G,H) may elevate pathogen transmission and co-infections [85,108,110].

Overall, urbanisation favours generalist species [111] and very high levels of urbanisation reduce pollinator diversity [112] (Figure 1E). Urban environments thus generally produce a network containing fewer, generalised pollinator species, with high connectance and low evenness and robustness [113,114]. This network architecture may promote pathogen transmission and infections in concert with other pressures of urban settings.

Concluding Remarks

Flower-mediated pathogen transmission is driven by a complex interplay between species traits, plant–pollinator network architecture, and anthropogenic drivers, often interacting in synergistic or antagonistic ways. While recent studies have shed light on several individual facets, many questions remain, especially on the complex interactions between the various aspects modulating disease transmission and on the exact mechanisms that drive host shifts (see Outstanding Questions). As anthropogenic environmental change continues and intensifies the risks of novel plant–pollinator–pathogen interactions and epidemics are likely to rise. Given the complex and highly context dependent outcomes, future research needs to cover a wide range of landscapes, climates, and local circumstances, to derive a robust overview on how pathogen dynamics will respond to anthropogenic pressures at different spatiotemporal scales. The interplay of global change pressures may reorganise pollinator assemblages and species interactions in ways that elevate the risks of emergent pathogens and population, or community epidemics, an outcome that can undermine policies (Table S1 in the supplemental information online) to safeguard pollinators and pollination services.

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Outstanding Questions

Epidemiology: Mechanisms that Govern Pathogen Host Shifts

- Can pathogen host range be predicted by host phylogeny or ecology? Which phenotypic (plant/pollinator/pathogen) traits facilitate intra- or interspecific pathogen transmission?
- How susceptible are different pollinator species to generalist pathogens and, if they are competent hosts, can they also transmit pathogens onward?
- What is the role of dominant species (e.g., honey bees) in driving pathogen community dynamics?
- How is pathogen fitness affected by novel host and abiotic environments? Does evolutionary change precede or follow from a host shift and is host switching accompanied by a loss of fitness in the original reservoir host?
- Do pollinator hosts evolve increased tolerance or resistance to pathogen infection?

Network Ecology: Transmission

- How strongly does pollinator network structure predict pathogen transmission within and between hosts?
- How dynamic is the flower–pollinator–pathogen network within and between years?
- Does network modularity provide protection to the pollinator community from generalist pathogens?
- To what extent is mass action (homogeneous and well-mixed host populations) sufficient to explain patterns of pathogen prevalence across communities of pollinators?

Landscape Ecology

- How does landscape context mediate pathogen spillover between managed and wild pollinators?
- Which landscape features, habitats, or species serve as hotspots or hubs facilitating pathogen spread, dilution, or concentration in pollinator populations or communities?

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Declaration of Interests

No interests are declared.

Supplemental Information

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- How does floral quantity and quality in contrasting landscapes or plant species affect pathogen fitness in different host pollinators?
- How do agri-environment measures (e.g., wildflower strips) affect pollinator health and disease?

Pollinators and Global Change

- How do beekeeping practices affect pathogen spillover between managed and wild pollinators, and how can it be reduced?
- How does the degree of urbanisation (town to city) affect the structure of plant–pollinator–pathogen networks?
- Can we experimentally determine how invasive plant species shift the community structure and pollinator behaviour to drive declines in co-flowering native species and changes in pathogen and parasite loads?
- How do synergistic and antagonistic effects of global change drivers affect pathogen dynamics in different pollinator hosts?
- What are the effects of policy actions (agricultural, climate, biodiversity conservation, and pollinator conservation) on pathogen dynamics?

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