Trends in Parasitology

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Review

Managing bees in a pathogenic world: research priorities to protect biodiversity

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Pathogens associated with managed bees are frequently detected in wild arthropods, raising concerns about potential negative impacts on biodiversity and fueling scientific, societal, and political debate. However, major gaps remain in our understanding of the mechanisms driving pathogen spillovers, their impacts on recipient hosts, and mitigation strategies. We propose an integrative, holistic research framework beyond the current focus on pathogen detection that centers on the impacts of shared pathogens and on management strategies to mitigate risks. This reframing will help guide future research design and interpretation beyond precautionary principles, advance our understanding of epidemiological risks, and support evidence-based policymaking that balances food security with biodiversity conservation.

Highlights

Pathogens associated with managed bees may spill over to wild arthropods, but their impacts remain largely unknown.

The current emphasis on pathogen detection cannot provide insight into the mechanisms and impacts of spillover, thereby hindering the development of effective mitigation.

Bee management modulates epidemiological risk, providing opportunities for proactive mitigation by following the precautionary principle to reduce transmission and spillover even if impacts on biodiversity remain uncertain.

Assessing ecological risks and guiding mitigation strategies require a holistic research framework that links pathogen spillover to fitness consequences, providing the necessary evidence to better safeguard the health of both managed and wild arthropods.

Are managed bees amplifiers of epidemiological risks?

Insects are declining at alarming rates in many regions of the world, with important variations across geographic locations and taxa [1]. Simultaneously, the use of managed bees for pollination services and the number of bee species being bred and commercialized are increasing [2]. However, **bee management practices** (see Glossary) entail risks, as high densities may lead to competition for floral resources and may facilitate the spread of pathogens to other insects [3], which has led to increasing scientific, societal, and political debate about potential negative **impacts** on wildlife.

While substantial evidence demonstrates that bee management influences intraspecific pathogen **transmission** and pathogen loads and that pathogens are shared between managed bees and wild arthropods [4,5], key knowledge gaps remain regarding the impact on **recipient hosts** and the extent to which management practices shape **epidemiological risks**. Furthermore, the contexts under which pathogens associated with managed bees may affect wild arthropods and whether competition for food and other biodiversity stressors may exacerbate these effects remain poorly understood. In this review, we suggest strategies to address these knowledge gaps to stimulate innovative, evidence-based mitigation strategies to prevent epidemiological risks (Figure 1, **Key Figure**). We highlight the need to shift from predominantly descriptive research toward a holistic understanding of drivers, mechanisms, and consequences of pathogen transmission between managed bees and wild arthropods. Such an approach is essential, as pathogen detection alone does not demonstrate **infection**, measurable **fitness** effects, or population-level consequences in recipient hosts. Yet, this knowledge is necessary for moving beyond precautionary measures with uncertain outcomes toward evidence-based policymaking and sustainable pollinator management that balance food security objectives with conservation of biodiversity.

The effect of bee management on pathogen dynamics

Livestock systems face elevated epidemiological risks. High host densities, reduced genetic diversity due to selective breeding, and frequent animal movement increase pathogen

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transmission among stocks of cattle, pigs, poultry, and bees [6,7]. These risks increase pathogen prevalence and disease burden in managed populations and can extend beyond agricultural systems through **spillover** into wildlife. Among pollinators, this raises a central question: does bee management pose epidemiological risks to other arthropods?

The management of bees varies widely across taxa and regions in terms of intensity, density, disease management, and transportation. Honeybees represent the most intensively managed pollinators, with the western honeybee, *Apis mellifera*, and the eastern honeybee, *Apis cerana*, often kept at high colony densities [7]. Several species of stingless bees are managed in tropical regions for small-scale honey production, with colonies frequently transported regionally to meet pollination needs [8]. Multiple bumblebee species are also commercialized worldwide, particularly

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Key figure

Overview of the epidemiological risks associated with bee management

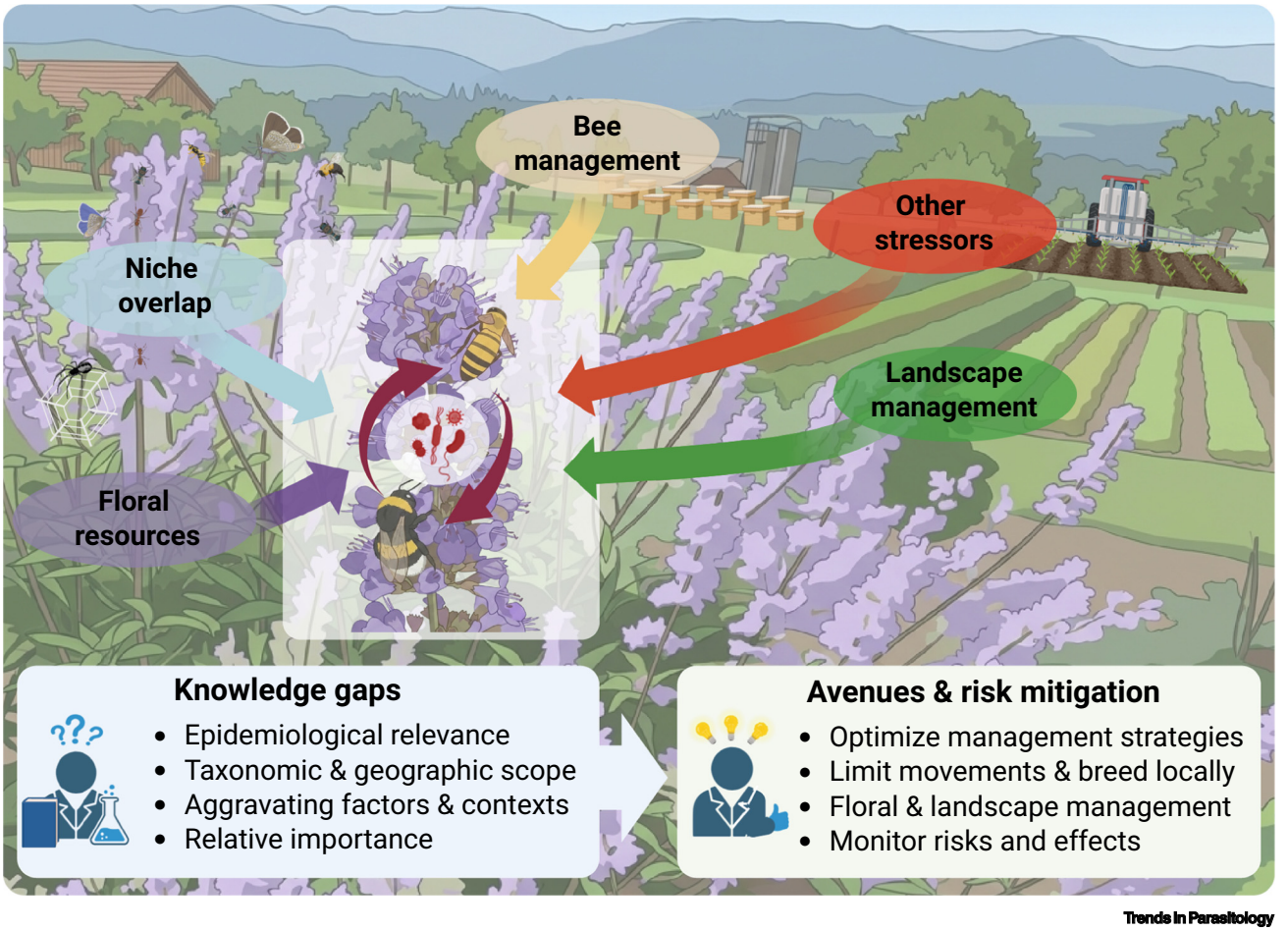


Figure 1. Upper part: factors known to influence epidemiological risks associated with bee management. Lower part: main knowledge gaps and research avenues to mitigate these risks. Figure created using BioRender (https://BioRender.com); original landscape and bee pictures by Alexis Beaurepaire.

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for crop pollination in greenhouses [8]. Finally, solitary bees, for example, *Osmia* spp., *Megachile* spp., and *Nomia* spp., are increasingly managed in artificial nesting systems, sometimes outside their native ranges to increase pollinator density [8].

Understanding epidemiological risks under bee management is complex because management practices vary across ecological contexts, landscape structures, nutrition, and the presence of disease vectors (Figure 2) [9]. Furthermore, the social lifestyle of several managed bee species, with populous colonies, overlapping generations, brood care, and high genetic relatedness, promotes pathogen multiplication and spread, as well as spread beyond the colony [10]. In these social bees, management can further weaken natural colony defenses through routine practices, such as disrupting the colony's physical barriers and homeostasis by opening hives or providing inadequate nutrition during periods of limited floral resources. Other practices directly affect disease dynamics, including parasite control, reuse of contaminated hive material and tools, and breeding practices that reduce genetic diversity and, thus, resilience to disease [9]. Unnaturally high colony densities, while logistically advantageous, further promote the movement of workers among colonies (via drifting) and robbing when food becomes scarce, both amplifying transmission risk [7,11]. High-density host systems can support large pathogen populations, resulting in greater pathogen genetic variation through mutation and recombination and thereby increasing the probability that variants with enhanced spillover potential or increased **virulence** will emerge [12].

Local and international trade, transport, and translocation of managed bees and bee products can introduce novel pathogen species or strains, create new transmission networks, and alter host-pathogen coevolutionary dynamics. Well-documented examples include the global spread of the ectoparasitic mite *Varroa destructor* (Box 1) or pathogen spillovers caused by the introduction of nonendemic managed bees such as *Bombus terrestris* [23] or *Osmia* species [24]. Migratory beekeeping, which can involve transport of colonies over thousands of kilometers annually, may also be associated with elevated parasite and pathogen loads [25]. Similarly, the trade in bees, queens, and hive products such as honey, wax, and pollen is a recognized pathway for pathogen spread, as is the case in many other veterinary diseases [26,27]. Finally, multispecies management in syntopy, such as co-occurring *A. mellifera* and *A. cerana* in Asia [28] or honeybees kept near stingless bees [29], creates additional opportunities for interspecific transmission.

From managed bees to recipient hosts—determinants of epidemiological risk

To understand the epidemiological risk to wild arthropods posed by pathogens associated with managed bees, it is essential to consider pathogen spillover as a series of events, the success of which depends on crossing sequential barriers shaped by host traits, pathogen traits, and environmental contexts [30,31]. Epidemiological impacts arise when these processes result in a measurable reduction in health, fitness, or genetic diversity of recipient hosts. Moreover, additional risks may emerge through pathogen **spillback**, which means that reducing transmission to wild hosts is also of considerable interest for managed bee health. In this section, we follow the journey of a pathogen from managed bees (**reservoir host**) to a **novel host** (Figure 3) and discuss the factors influencing transmission, spillover, and impacts.

From reservoir to recipient: wild arthropod exposure to pathogens associated with managed bees

Direct and indirect contacts between reservoir and recipient hosts drive pathogen transmission. Indirect contacts, such as shared floral resources, are particularly important for interspecific transmission of pathogens associated with managed bees [32,33]. Hence, high densities of

Glossary

Basic reproduction number (R_0): the expected number of secondary infections caused by a single infected individual in a susceptible population.
Bee management practices: interventions or strategies used by humans to maintain bees for pollination services and/or bee products.
Covert infection: an infection in which the infected host shows no clinical signs.
Dead-end host: a host species in which transmission is absent or insufficient to maintain the pathogen in the population.
Dilution effect: a reduction in pathogen transmission or epidemiological risk resulting from increased host diversity, typically through the presence of less-susceptible or less-competent hosts that disrupt transmission.
Epidemiological risk: the probability of adverse consequences for a recipient host population or community arising from pathogen transmission, spillover, and establishment.
Fitness: an organism's ability to survive, reproduce, and pass on its genes to the next generation.
Host competence: the ability of an infected host to transmit the pathogen onward. Competence is determined by a multitude of factors, including the ability of hosts to support pathogen replication, pathogen shedding rates, host contact rates, and behavioral traits.
Host shift: the establishment of sustained transmission of a pathogen in a host species in which it had not previously been maintained, typically resulting in expansion of its host range.
Host susceptibility: the probability of developing an infection in a given organism.
Impact: the realized effect of a pathogen on an individual host's condition, survival, and/or reproduction, as well as the resulting demographic consequences for the host population.
Infection: the invasion, establishment, and replication of a pathogen within a host's tissues.
Mechanical carrier: an organism that carries a pathogen from one host to another without itself being infected.
Novel host: a host species that is newly infected by a pathogen outside its historically recognized host range.
Overt infection: an infection characterized by the presence of clinical signs.
Pathogenicity: the ability of a microbe to cause disease in an infected host.

managed bees and increased interspecific mixing elevate the likelihood that pathogens encounter recipient hosts. Accordingly, the detection of honeybee-associated pathogens in wild pollinators increases with resource overlap with *A. mellifera* and with apiary density [33,34].

Infected managed bees may contaminate flowers when collecting nectar and/or pollen (Figure 3, step 1), with pathogen **shedding rates** usually varying depending on host species and the pathogens considered (Box 2). Once the pathogen has exited the reservoir host, the next challenge on its journey is to survive in the environment (Figure 3, step 2). Pathogen resilience depends on attributes such as resistance to UV radiation and tolerance to temperature and humidity [34,44], as well as floral traits that influence sun exposure and thermal stress [44]. The number of reproductive structures per inflorescence can influence pathogen resilience, potentially through effects on microclimates mediating pathogen viability. If a different host species visits a contaminated flower while the pathogens remain viable (Figure 3, step 3), pathogen particles may be transferred to and infect the recipient host, with floral traits such as corolla shape and nectar quantity, attractiveness, and accessibility further influencing host exposure to contaminated surfaces [44].

In addition to floral, pathogen, and host traits, the structures of the landscape and of plant–pollinator networks strongly affect contact rates and thereby the opportunities for pathogen transmission [45]. Landscapes with low floral diversity and abundance can concentrate pollinators, increasing resource overlap with reservoir hosts and consequently pathogen exposure, prevalence, and loads in wild recipient hosts [34,46]. Network structures modulate pathogen transmission across species and space. Indeed, highly connected networks usually facilitate the spread. In parallel, greater host diversity may lead to a disease **dilution effect** through transmission to hosts of lower competence, which may act as dead-ends, and greater floral diversity may reduce transmission through decreased niche overlap [34,40,47].

While transmission via shared floral resources predominates among pollinators, other pathways may be more important among other species (e.g., ants, wasps, and spiders), in which predation, scavenging, or direct contact is likely to drive exposure [16]. Additionally, invasive species can restructure interaction networks, introduce novel pathogens, act as novel reservoirs, alter contact patterns, or serve as vectors that create additional transmission pathways connecting otherwise weakly linked hosts [48]. Climate change, by modifying community networks and species distributions, may further exacerbate epidemiological risks, although the extent of these complex effects remains poorly studied [49].

Establishment of infection within the recipient host

Once a pathogen encounters a novel host species, a series of physical, physiological, and immunological barriers must be overcome to establish infection [30,50,51] (Figure 3, step 4). Because many of these barriers are dynamic, infection success depends strongly on transmission routes, exposure rates, and inoculum sizes. While the insect cuticle represents a largely nonpermissive barrier [52], some fungal pathogens can breach this barrier using specialized structures and enzymes [53]. More commonly, pathogens enter via wounds, vector-mediated injection, or natural openings (e.g., the mouth) [49], thereby bypassing some host barriers. Ingested pathogens face additional barriers in the gut, including the peritrophic matrix, digestive enzymes, pH gradients, and resident microbiota [51,54]. Nevertheless, certain bacterial and microsporidian pathogens can bypass these barriers, germinate, replicate in the midgut lumen, and invade host tissues [55,56]. Vector-mediated injection bypasses external and gut barriers, increasing infection efficiency and potentially leading to higher pathogen loads, prevalence, and altered virulence, as shown most prominently for *V. destructor*-transmitted viruses (Box 1) [15,16]. Amplified infections can, in turn, increase shedding and environmental contamination, thereby elevating spillover risk.

Recipient host: a host in contact with a pathogen originating from another host species (regardless of whether infection occurs).
Reservoir host: a host species in which a pathogen is maintained over time and from which transmission to other hosts can occur.
Shedding rates: the speed and quantity at which an infected host expels infectious agents into the environment.
Spillback: the successful transmission of a pathogen from a recipient host species back to the original reservoir host species.
Spillover: the successful transmission of a pathogen from a reservoir host species, leading to infection of a novel host of a different species, irrespective of whether the infection leads to sustained transmission and a subsequent host shift. Critically, we define spillover by the occurrence of infection in the novel host, rather than by pathogen detection or epidemiological similarity across species; this distinguishes our definition from broader uses of the term that include pathogen detection in multiple species, detection of shared strains or variants, or common spatial and temporal prevalence patterns.
Transmission: the process by which a pathogen passes from one host to another, regardless of whether an infection is established.
Virulence: the degree of harm a pathogen causes to its host, often measured by mortality or reductions in fitness.

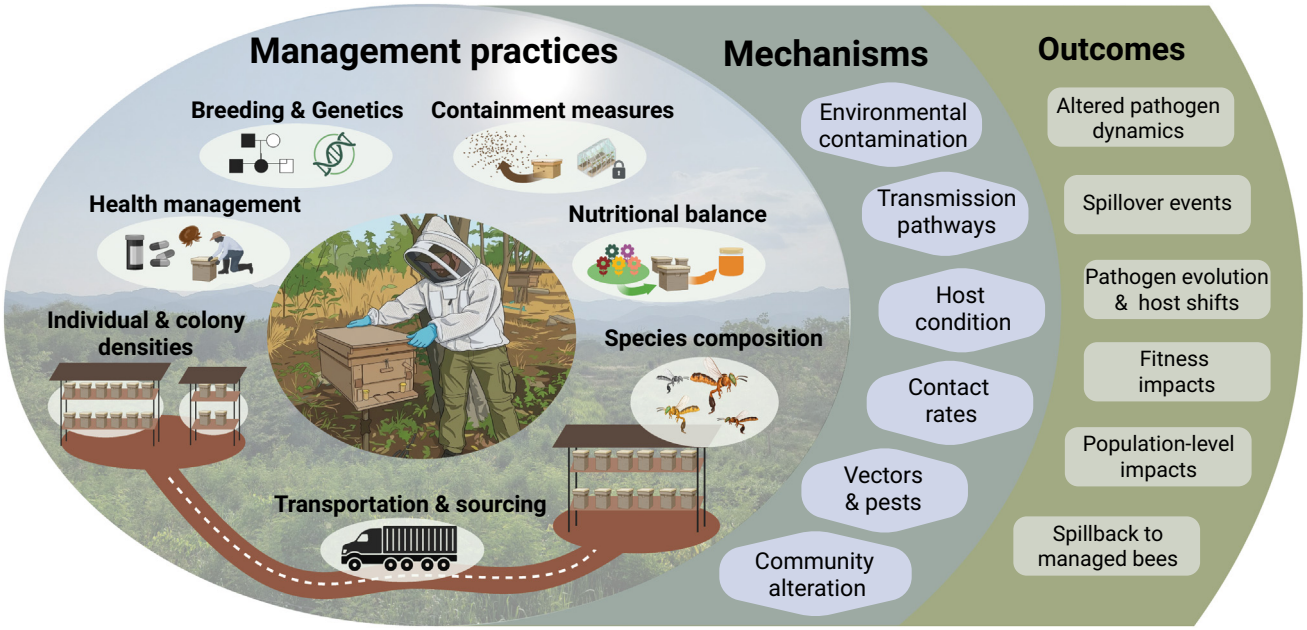


Figure 2. Factors underpinning epidemiological risks of bee management. Various factors of bee management practices (left) potentially alter biological and environmental mechanisms in pathogen transmission (center) that ultimately shape disease dynamics and epidemiological outcomes in host communities (right). Figure created using BioRender (https://BioRender.com); original landscape and beekeeping pictures by Alexis Beaurepaire.

Following host entry, the outcome of an infection depends on host compatibility. Tissue tropism and receptor compatibility constrain infections to hosts with suitable cellular entry pathways and intracellular environments, often limiting pathogen spillovers to phylogenetically related species [57]. More generally, pathogen features, such as replication strategy and the ability to evade or suppress host defenses, critically determine establishment success [58,59]. Hosts can counter infections via innate immune pathways that activate cellular responses (e.g., phagocytosis), humoral defenses (e.g., antimicrobial peptides and melanization) [60], and RNA interference and cyclic GMP-AMP synthase stimulator of interferon genes (cGAS–STING)-mediated antiviral

Box 1. Invasive mites change the bee pathogen landscape

Due to its negative effects on western honeybee health and on the pollination services they provide, the ectoparasitic mite *Varroa destructor* ranks fifth among the 100 socioeconomically worst alien species [13]. This mite originally infested exclusively colonies of the eastern honeybee, *Apis cerana*. In its original host, *V. destructor*'s reproduction is usually restricted to male (drone) brood, which, together with several host defense mechanisms, keeps the population of mites below damage thresholds [14]. When western honeybee (*Apis mellifera*) colonies were imported into Asia during the 20th century, the opportunities for contact between the mite and *A. mellifera* became frequent, leading to several host shifts as early as the 1950s (e.g., Japan in 1957 or Hong Kong in 1963) [15]. Through this novel susceptible host, *V. destructor* successfully expanded its range, greatly aided by international trade [2], and is now present in almost all regions where *A. mellifera* colonies are managed [15]. *Varroa destructor* feeds on the fat body and hemolymph of adult honeybees and honeybee brood [15], acting as a potent vector of viral pathogens [16]. As a consequence, the spread of *V. destructor* was followed by dramatic changes in the viral landscapes of honeybee colonies [13,15,17], and led to the DWV pandemic [18]. Prior to the *V. destructor* invasion, DWV relied on much less effective oral or sexual transmission, resulting in low viral loads that mostly caused **covert infections** [19]. Vector transmission enables viruses to evade the honeybees' innate immune defenses, such as the cuticle and epidermis, while simultaneously increasing inoculum size [16]. Consequently, the formerly benign DWV has become a highly prevalent virus triggering symptomatic infections that often lead to mortality in honeybee colonies [20]. The high prevalence and viral loads of DWV increase opportunities for transmission to other species, and, consistent with this, DWV is the honeybee-associated pathogen most frequently detected in a wide diversity of hosts [5,20–22].

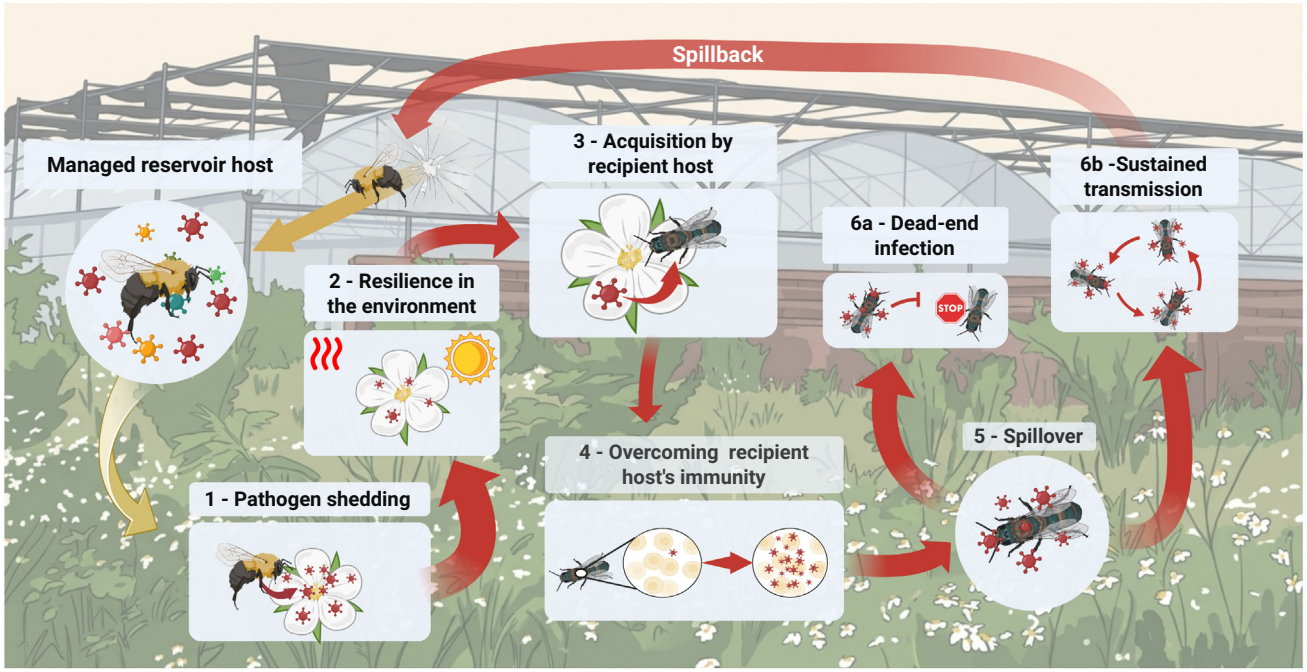


Figure 3. Path to pathogen spillover from managed bees to wild arthropods. Sequential barriers that a pathogen has to overcome to spill over from a managed reservoir bee host to a recipient host. The figure illustrates how a reservoir host, a managed bumblebee, escapes the containment of a greenhouse and releases pathogens onto flowers, which are visited by a recipient host, resulting in transmission. Once within the recipient host, the pathogen overcomes its immune response and establishes infection, resulting in successful transmission (i.e., spillover) and impacts. Additionally, the pathogen may spill back to its original host. Figure created using BioRender (https://BioRender.com); landscape picture provided by Lars Straub.

defenses [61]. In social insects, these defenses are complemented by social immunity, that is, collective behavioral mechanisms such as allogrooming and social interaction patterns that reduce infection and transmission [10,62,63]. The effectiveness of these defenses depends on host health and immune capacity, both of which are mediated by factors such as nutrition and

Box 2. Superspreaders and highly transmissible pathogens

Not all hosts play the same role in pathogen dynamics. While some act as dead-end hosts [35], others function as reservoir hosts that maintain pathogens and facilitate cross-species transmission. Transmission heterogeneity emerges from variation in contact rates, pathogen loads, immune competence, and behavior. This can give rise to highly connected transmission hubs and even superspreaders, defined as individuals responsible for a disproportionate number of secondary infections [36]. In multihost systems, superspreading is most likely when a host shows disproportionately high abundance, high infection prevalence, and high shedding rates [32], which can be fueled by pathogen tolerance. In pollinator communities, interspecific transmission most likely occurs at shared floral resources, and traits such as generalist foraging habits and large foraging ranges can therefore increase the probability of spillover [37]. Specific pollination behaviors (e.g., bumblebee buzzing [38] and flower constancy) or larger body sizes [39] have also been linked to higher pathogen deposition rates in bumblebees. Finally, eusociality, enhancing direct contact between individuals and overlap of generations, can also promote intraspecific transmission, resulting in higher epidemiological risks around colonies. Accordingly, honeybees (*Apis mellifera* and *Apis cerana*) and bumblebees (*Bombus* spp.) may function as superspreaders [40]. Other taxa, such as stingless bees, exhibit similar traits but remain relatively understudied. In parallel, some pathogens show disproportionately high interspecific transmission rates [41]. For instance, RNA viruses (e.g., DWV) and microsporidia (*Vairimorpha ceranae*, formerly known as *Nosema ceranae*) exhibit traits enhancing spread, such as vector-mediated amplification and transmission (e.g., by *Varroa destructor* for DWV [42]; Box 1) and high environmental resilience [43], respectively. Given the ubiquity, broad host range, and high adaptive potential of some pathogens infecting managed bees, these pathogens may pose substantial threats to wild arthropods in the future. Adapting bee management practices to limit transmission risks therefore appears increasingly urgent.

stressors [45,47]. Once a pathogen has overcome host defenses and established infection, spillover has occurred (Figure 3, step 5), potentially leading to sustained transmission and/or impacts in recipient host populations (i.e., a **host shift**).

Infection outcomes: host range and impacts

At the population level, spillover impacts are mediated by **host susceptibility** and **host competence**, which determine whether transmission results in isolated dead-end infections (Figure 3, step 6a) [35,64] or self-sustaining transmission chains (Figure 3, step 6b) [65]. Pathogens associated with managed bees show a range of host specificity, spanning from specialists to broad generalists. Host phylogeny is considered an important determinant of pathogen spillover, but ecological opportunities can weaken the effect of phylogenetic distance [66]. Many RNA viruses show a broad host range, linked to high mutation rates and quasispecies dynamics that facilitate compatibility with novel hosts [67,68]. Replication of honeybee-associated viruses has been reported in bumblebees, solitary bees, vespid wasps, ants [4,21,69–73], and even spiders [106]. However, fitness impacts in non-*Apis* hosts remain highly variable and often undetectable [45]. Microsporidia have an intermediate host range, in which spillover of *Vairimorpha bombi* (formerly *Nosema bombi*) from managed to wild bumblebees is well documented and linked to regional wild bumblebee declines [74]. Major bacterial pathogens in managed honeybees, however, almost exclusively infect *A. mellifera* despite their durable resistant forms and extreme environmental persistence [75], although exceptions have been documented for bacteria such as *Melissococcus plutonius*, the causative agent of European foulbrood [76–78]. Finally, fungi causing chalkbrood disease (*Ascosphaera* spp.) and co-introduced into the United States along with nonnative managed mason bees (*Osmia* spp.) have spilled over to native mason bee species and may be linked to population declines in these recipient hosts [24]. Ultimately, epidemiological risk is determined by infection outcomes and transmission dynamics, and thus quantifying fitness and population-level impacts in wild arthropods is key to understanding the extent of these threats.

Mitigation strategies

Although uncertainty remains about the ability of pathogens associated with managed bees to affect other species following transmission, a precautionary approach is advisable to reduce pathogen spillover risks to wild arthropods. Mitigating such transmission pathways would also be in the interest of managed bee health, especially considering the risk of spillback, that is, the successful transmission from managed bees to wild arthropods and back to managed bees [79].

Adapting bee management practices

While there is a paucity of information regarding best management practices for most managed bees, there is accumulating evidence that honeybee management strategies have downstream effects on pathogen transmission. Some solutions are available to reduce transmission and spillover risk, including:

i. Pathogen and vector control. Except for *V. destructor* and deformed wing virus (DWV) [42] (Box 1), the effect of pathogen control measures on reducing spillovers to synanthropic wild arthropods is unknown. While eradication is unrealistic for many pathogens, it is reasonable to expect that keeping pathogen loads low in managed bees minimizes potential impacts on wildlife [80]. For *V. destructor* and DWV, controlling mite infestations below the recommended treatment threshold reduces DWV prevalence in wild pollinators by 39–85% [105]. Additionally, ensuring that management operations coordinate treatments—as recommended for mite control in honeybees [81]—is essential to prevent cross-transmission.

ii. Reduce overcrowding. Individual or colony densities and spatial arrangement can impact pathogen build-up, with potential ramifications for spillover. For instance, small distances between colonies can elevate populations of *V. destructor* and associated viral pathogens in honeybees [7,11] and may result in higher pathogen transmission (Box 2). While empirical evidence remains limited, reducing managed bee densities may be an effective way to reduce spillover risk.
iii. Ensure adequate nutrition. Sufficient and continuous access to natural floral resources can improve immunity to pathogens and parasites [82]. In honeybees, where supplemental feeding with sugar syrup and pollen patties during periods of resource dearth is common practice, over-harvesting of honey should also be avoided to ensure good colony functioning and immunity.
iv. Choice of operation location. Although beekeepers can feed managed bees, providing sufficient natural resources to them is key to fostering their immunity and avoiding the spread of diseases. A good understanding of their dietary needs can help in choosing locations that provide access to key resources (e.g., propolis in honeybees). In parallel, given that the magnitude of pathogen deposition onto floral resources decreases with increasing distance from honeybee apiaries [46], the location of bee management operations could be adjusted to limit epidemiological risks, particularly near habitats supporting imperiled pollinators. However, field validation is needed to assess the magnitude of these potential benefits.
v. Supplements. Recent findings suggest that yeast supplements can be used to improve colony development in managed honeybees [83] and that algae can improve immunity [84]. Propolis is also a well-known natural bee-derived antimicrobial product, which can be collected from healthy colonies and used to control diseases [85]. Supplements may therefore be used to indirectly reduce epidemiological risks to wild arthropods, but these indirect effects remain to be tested under field conditions.
vi. Containment. Preventing managed bees from escaping containment can reduce pathogen transmission and other risks to wild arthropods, such as genetic pollution [80]. Further restrictions, such as limiting or banning the use of managed bees in a given region, should be considered and debated in light of the local context, including whether the species in question is native or introduced [3]. In honeybees, swarm control limits a potential dispersal pathway for pathogens, reducing the establishment of feral colonies that could act as reservoirs for spillover into wild pollinator communities. However, more work is necessary to move beyond this precautionary approach because it is currently unknown whether such free-living colonies pose an epidemiological risk to wild arthropods, and the current evidence on the health status of feral colonies compared with sympatric managed colonies is mixed (e.g., [86,87]). In bumblebees, individuals escaping from greenhouses have led to the spread of exotic pathogens suspected of causing declines in native bumblebees [88].
vii. Prevent interspecific transmission. Reducing unnatural interactions between managed bee species may reduce the risk of pathogen transmission. Honeybee pollen fed to commercial bumblebee and stingless bee colonies should be sterilized systematically to reduce the risk of pathogen contamination and transmission [89]. Additionally, multispecies beekeeping (e.g., combining stingless bees and honeybees within the same apiary) should be avoided, especially when closely related species affected by the same diseases and vectors are kept, for example, *A. cerana* and *A. mellifera* (Box 1).
viii. Hygienic use of equipment and products. The use of contaminated equipment or products (e.g., wax) poses a major transmission risk, and good practices should be strictly observed to avoid spreading diseases both within and between management operations, and beyond [16]. Fostering good practices in the use of bee management equipment is a promising avenue to limit epidemiological risks.

Limit the movement of managed bees

A common-sense strategy for limiting pathogen spillover from managed pollinators is to limit long-distance transportation of managed bees by using native bee species and local populations [90,91], though such utilization must be informed by wild population size assessments to avoid over-exploitation. If wild populations are insufficient to ethically source managed stocks, breeding captive populations of native managed species is an alternative, as is often done with honeybees and bumblebees.

To avoid the movement of pathogens and the spread of diseases, native bees should be preferred over nonnative bees. However, if the use of nonnative bees is unavoidable or already in place (e.g., the use of *A. mellifera* outside its native distribution range [2]), comprehensive health checks (e.g., pathogen screening, quarantine, and certification) must be required prior to any movement [90]. While some international regulations on the import and movement of managed bees exist, they are not always sufficient to protect wild arthropod communities [88].

Adapt floral resources and landscape management to decrease transmission

Effects of floral resource availability on pathogen prevalence appear to depend on landscape context, with high floral abundance amplifying pathogen incidence among bees in anthropogenic habitats with few other resources, likely due to the concentration of bees [92]. High floral resource abundance and diversity can also reduce the prevalence of pathogens, including DWV, *Andrena*-associated bee virus-1 [93], and black queen cell virus [94] in honeybees and other pollinator species.

Knowledge of pollinator diets can aid in developing strategies to reduce interspecies transmission. For instance, a recent study [95] found that honeybees and wild bumblebees had distinct preferences such that visitation patterns did not overlap, thus spatially preventing pathogen transmission. Moreover, when planting wildflower strips in croplands, the pathogen transmission capacity of floral species should be considered [44]. While the mechanisms are unclear, Adler *et al*. found that some plant species could reduce the parasite burden of *Crithidia bombi* compared with other species [82]. Further research may identify additional plants that reduce the risk of pathogen transmission and quantify the magnitude of these potential benefits under field conditions.

Knowledge gaps and research perspectives

Despite growing evidence of pathogen transmission between managed bees and wild arthropods, key knowledge gaps remain about the associated impacts on wild hosts, as well as the role of bee and landscape management in shaping these processes.

From detection to host ranges and epidemiological relevance

While several pathogens associated with *A. mellifera* have been detected across a wide range of arthropods [4,5,69], host ranges and roles (e.g., **mechanical carrier** vs. reservoir host), as well as impacts, remain unclear [80]. Additionally, publication bias may result in studies reporting nondetections being less likely to be published than those reporting positive detections, potentially obscuring host ranges.

A major challenge is moving beyond mere detection of pathogens associated with managed bees in wild hosts: molecular detection alone does not demonstrate replication, pathology, or onward transmission, and thus is insufficient to infer realized host range and epidemiological risks. For most host–pathogen combinations, host susceptibility, pathogen shedding capacity, and competence remain largely unknown. Addressing these gaps requires moving beyond

presence/absence data toward a hierarchical evidence framework, progressing through (i) detection, (ii) infection, (iii) secondary transmission, (iv) measurable fitness effects, and ultimately (v) population-level impacts.

Distinguishing true infections from carriage and linking replication to **pathogenicity** remain key challenges [65], and evidence for secondary transmission among novel hosts after spillover remains scarce. This limits our ability to determine whether pathogens persist in novel hosts or rely on repeated spillover from reservoirs. For example, DWV infects *A. mellifera* as well as *Bombus* spp. [69,94], and bumblebees can show **overt infections** and clinical signs [96]. Recent studies indicate that transmission occurs predominantly from honeybees to bumblebees, with bumblebees likely acting as **dead-end hosts** with limited onward transmission, in which DWV is unlikely to be maintained in the absence of honeybees [35,45]. Another study found that only half of detected insect virus taxa showed signs of replication in bumblebees and honeybees [73]; therefore, positive detections even in susceptible hosts may not indicate true infections. Similarly, assessing fitness and population-level effects can be challenging but are essential to inform impact assessments. Fitness proxies were quantified in the European orchard bees (*Osmia cornuta*), for which correlations with offspring fitness were only significant for resident viruses (Ganda bee virus, Scaldis River bee virus), but not for honeybee-associated viruses [97].

Future research should combine controlled inoculation experiments in diverse arthropod host species with ecologically relevant exposure scenarios, while accounting for pathogen–pathogen interactions and potentially aggravating environmental stressors [98]. In such studies, pathogen replication and dynamics over time should be quantified while assessing lethal and sublethal effects. Notably, biomarkers of stress and immune activation can be used as indicators of negative impacts, even in the absence of overt clinical signs [57,58]. In field studies, spillover can be demonstrated by pathogen emergence following managed bee introduction, differences in pathogen prevalence along gradients of managed bee abundance, or pathogen phylogeny [20]. Finally, confirming secondary transmission among and between host species can help in understanding the roles species play in pathogen networks (Box 2). Insights from genomic and modeling studies will be essential to track different pathogen strains, resolve transmission directionality, estimate key parameters such as the **basic reproduction number (R_0)**, distinguish reservoirs from recipient hosts, and inform mitigation strategies. Resolving population-level effects remains challenging due to limited long-term data and the difficulty of manipulating unmanaged pollinators; consequently, evidence remains largely correlative [24,80,99]. Progress will require integrating longitudinal monitoring, natural experiments, and modeling approaches that infer transmission pathways and demographic impacts while linking infection dynamics to host population trajectories [74,100] as well as using standardized methods to increase comparability among studies [101].

Expanding the taxonomic and geographic scope

Current understanding of pathogen spillover from managed bees is heavily biased toward *A. mellifera* and *Bombus* spp. (mainly *B. terrestris* and *Bombus impatiens*), despite more than 60 species being managed globally [8]. For most, basic data on pathogen diversity and management-specific effects on transmission risks are lacking. There is a need to establish which management practices known to minimize disease in bees can be transferred across species. Not all measures are universally effective; for example, disinfecting trap nests does not consistently reduce chalkbrood in *Megachile rotundata*, likely because transmission is dominated by environmental sources [102]. Research is similarly biased toward a narrow subset of wild hosts, leaving the susceptibility of most taxa uncharacterized.

Geographic biases further constrain inference, as most studies originate from Europe and North America. In contrast, biodiversity hotspots in which a high diversity of wild bee species co-occur alongside introduced and native managed bees [14] offer valuable opportunities to increase knowledge of the mechanisms underpinning transmission [65]. Addressing taxonomic and geographic biases will require coordinated global surveillance, standardized diagnostics, and inclusion of underrepresented taxa in experimental infection studies. International and interdisciplinary collaboration among wild and managed bee specialists, epidemiologists, ecologists, and evolutionary biologists is essential to tackle this multifaceted challenge.

Bee and landscape management

Bee and landscape management can shape host–pathogen dynamics by altering exposure and selection pressures [45]. Although key drivers have been identified, substantial knowledge gaps around management decisions on pathogen dynamics remain. Central questions include whether specific management practices select for increased virulence, whether migratory practices promote strain homogenization or diversification, and whether repeated transmission across multiple host species favors the evolution of broader host ranges and higher virulence. The role of floral traits, host behavior, and foraging plasticity in shaping transmission risk also remains poorly understood.

Optimizing bee and landscape management to minimize epidemiological risk, particularly the probability of rare but consequential host shifts, will require targeted approaches that link mechanisms to outcomes. Because bees adjust their foraging to support their health [34,103], managing landscape structure, floral communities, and supplementary feeding may offer opportunities to improve nutrition while reshaping contact networks to reduce transmission between reservoirs and wild hosts [104]. In addition, longitudinal genomic surveillance of pathogens can track spread, strain turnover, and transmission directionality. Experimental evolution in managed colonies can test whether high-density or multihost transmission selects for increased virulence or broader host ranges. Controlled infection experiments embedded within plant–pollinator networks can quantify how floral resources and contact structure influence transmission among species. Finally, multigenerational monitoring of managed and wild populations is needed to assess whether pathogen spillovers cross annual life cycle bottlenecks and translate into population-level effects.

Concluding remarks

The central challenge in determining the epidemiological risks that pathogens associated with managed bees pose to biodiversity is identifying when spillovers lead to sustained transmission and ecological impacts (see Outstanding questions). Although current evidence for population-level impacts remains limited, we advocate considering a precautionary approach grounded in current knowledge while working toward a more holistic understanding of epidemiological risks posed by pathogens associated with managed bees. The ubiquity of hosts and frequent interspecific interactions increase opportunities for consequential spillover and spillback events, risks that may further increase under climate change [49]. At the same time, pathogen spillover from managed bees should be viewed in the broader context of multiple stressors driving insect declines. Although spillovers can occur and may have consequences under certain circumstances, their contribution to arthropod declines remains uncertain and is likely context-dependent.

Managed bees provide essential ecosystem services, with elevated epidemiological risks arising primarily under specific management and environmental conditions. Improving practices that reduce pathogen loads, limiting unnecessary exposure, adapting floral resources and landscapes, and controlling managed bee movements more strictly may benefit both managed and

Outstanding questions

In what contexts and to what extent do managed bee pathogens pose a threat to wild arthropods?

Which hosts and pathogens are most prone to spillovers?

Which traits determine recipient host susceptibility to spillover?

Which management practices most effectively reduce epidemiological risks?

How can we anticipate and mitigate pathogen spillovers without compromising the services provided by managed bees?

How can we leverage landscape structure, floral resources, and plant–pollinator networks to minimize epidemiological risks?

How do epidemiological risks associated with bee management rank compared to other threats to wild arthropods?

wild pollinators, particularly given the potential for bidirectional transmission (i.e., spillover from managed to wild arthropods or vice versa). Accordingly, bee management and landscape design should follow an evidence-based approach that minimizes cross-species transmission while supporting resilient arthropod communities. By integrating molecular, evolutionary, and ecological perspectives, we can move beyond pathogen detection toward predictive disease ecology, helping to safeguard biodiversity while ensuring food security through managed bee pollination.

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The authors declare no competing interests.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used DeepL Write, Grammarly, and ChatGPT to assess the grammar. After using these tools, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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