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Mechanisms and Consequences of Plant–Pollinator–Pathogen Interactions

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Abstract

Infectious disease is a major driver of biodiversity loss, but how disease threatens pollinator communities remains poorly understood. Here, we review the plant–pollinator–pathogen literature to identify mechanisms by which plant and pollinator traits and community composition influence pathogen transmission and assess consequences of transmission on plant and pollinator fitness. We find that plant and pollinator traits that increase floral contact can amplify transmission, but community-level factors such as plant and pollinator abundance are often correlated and can counteract one another. Although disease reduces pollinator fitness in some species, little research has assessed cascading effects on pollination, and taxonomic representation outside of honey bees and bumble bees remains poor. Major open challenges include (*a*) disentangling correlations between plant and pollinator abundance to understand how community composition impacts pathogen transmission and (*b*) distinguishing when pathogen transmission results in disease. Addressing these issues, as well as expanding taxonomic representation of pollinators, will deepen our understanding of how pathogens impact diverse pollinator communities.

1. INTRODUCTION

Our world is beautifully diverse, and we owe much of this beauty to plant–pollinator mutualisms. Pollination is implicated in the diversification of angiosperms (Dodd et al. 1999), and animal-pollinated crops constitute a major source of human nutrition (Eilers et al. 2011). Meanwhile, important pollinators such as hummingbirds, butterflies, and bees have evolved to depend on flowering plants as food resources, resulting in a fascinating diversity of behaviors and ecologies. However, in addition to providing essential food resources, flowers can be sites for the transmission of pollinator pathogens (Durrer & Schmid-Hempel 1997, Manley et al. 2019). Diseases associated with these pathogens have the potential to negatively impact diverse pollinator populations (Goulson et al. 2015), with cascading impacts on pollination of wild and cultivated plants (Ollerton et al. 2011).

Indeed, a growing body of literature suggests that infectious pathogens can have serious negative consequences for pollinator populations. For example, the microsporidian *Vairimorpha bombi* (formerly *Nosema bombi*) has been implicated in the decline of several North American bumble bee species (Cameron et al. 2011, Otterstatter & Thomson 2008, Szabo et al. 2012), and deformed wing virus is at epidemic levels in managed honey bee hives (Wilfert et al. 2016). Many unanswered questions remain, including whether insights from a relatively small number of well-studied bee species apply more broadly to the full breadth of pollinator taxa. However, it has become increasingly clear that plant-mediated transmission of pollinator pathogens, as well as the interaction of disease with related anthropogenic disturbances, can threaten diverse pollinator populations.

Here, we review the plant–pollinator–pathogen literature to investigate the mechanisms (**Figure 1*a***) and functional consequences (**Figure 1*b***) of plant–pollinator–pathogen interactions. Unfortunately, little is known regarding floral-mediated pathogen transmission among birds, mammals, and other non-insect pollinators, and our review focuses primarily on pathogens with ample literature. As such, our review is strongly biased toward bees, although we highlight a few non-bee examples to discuss these concepts more broadly. Section 2 provides background information on the biology of the major pathogen groups discussed in this paper. Section 3 addresses how plant and pollinator species traits affect floral-mediated transmission of pollinator pathogens—from pathogen deposition and pathogen acquisition on flowers to pathogen infection and associated changes in pollinator foraging behavior. Section 4 addresses how different measures of plant–pollinator community composition (e.g., plant and pollinator abundance and diversity) impact pathogen prevalence in diverse plant–pollinator communities. Section 5 examines when and where increasing pathogen prevalence in plant–pollinator communities will have an impact on the survival and fitness of different pollinator species, while Section 6 explores how altered foraging behavior of infected pollinators and disease-induced declines in pollinator populations might impact plant pollination. Finally, Section 7 discusses open questions and proposes novel directions for future research. As a whole, this review aims to improve our mechanistic understanding of pathogen transmission, including when and where these mechanistic processes are potentially generalizable across diverse species despite our current lack of taxonomically diverse data.

Pathogen: a bacterium, fungus, virus, or other microorganism that can potentially infect a host and cause disease

Disease: a disorder in an organism, especially one that has a known cause (e.g., pathogen infection) and distinctive symptoms

Pathogen transmission: the process by which pathogens move from one organism to another

Pathogen acquisition: when pathogens are acquired by new hosts; at this stage, pathogens may replicate or pass through hosts without consequence

Pathogen infection: when pathogens replicate in hosts, often followed by the onset of disease

Community composition: the number and types of species that make up a biological community

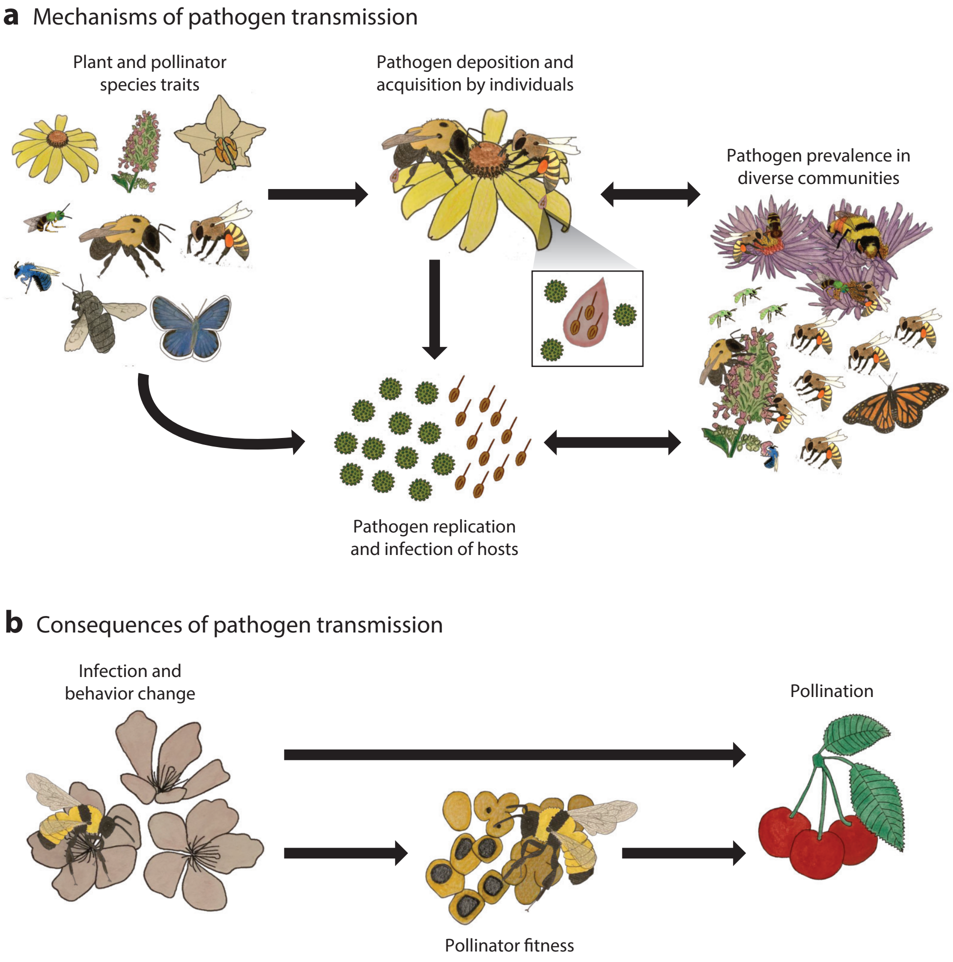

Figure 1

Mechanisms and consequences of plant–pollinator–pathogen interactions. (*a*) Plant and pollinator traits (e.g., plant chemistry, pollinator body size) can influence pathogen deposition and acquisition by altering the likelihood that individuals encounter pathogens while foraging on flowers. Processes impacting pathogen transmission among individuals can also scale up to affect pathogen transmission in plant–pollinator communities. At the community level, different measures of plant–pollinator community composition (e.g., plant and pollinator abundance and diversity) can increase or decrease community-level pathogen prevalence. Species traits and community composition can also impact infection (replication of pathogens in hosts) by altering host nutrition. (*b*) When pathogens cause disease, pathogen transmission may also impact pollinator fitness via altered survival and reproduction. Changes in foraging behavior and disease-induced declines in pollinator populations can also impact pollination and plant reproduction.

2. BIOLOGY OF POLLINATOR PATHOGENS

Pathogens of pollinators can be broadly categorized into a few major pathogen groups that vary in their biology, disease-causing symptoms, and mode of transmission (**Table 1**). In general, pathogens can be deposited on flowers mechanically (i.e., by passive transport on pollinator bodies or mouthparts) and fecal–orally (i.e., a pollinator regurgitates or defecates infectious particles).

Table 1 Commonly studied pollinator pathogens, including their mode of transmission, symptoms of associated disease, and a non-exhaustive list of pollinator taxa in which the pathogen has been detected

Pathogen group	Mode of transmission	Well-studied examples	Disease and/or associated symptoms	Pollinators in which the pathogen has been detected
Viruses	Injection by *Varroa* mites (honey bees) Mother-to-offspring Within-colony (social bees) Contaminated flowers	Deformed wing virus	Deformed wings, impaired foraging in adults, severely reduced lifespan	Honey bees, bumble bees, other bees, flies, ants, wasps, butterflies & moths, beetles, earwigs, etc.
		Black queen cell virus	Blackening and eventual death of queen pupae	Honey bees, bumble bees, other bees, wasps
Trypanosomes	Mother-to-offspring Within-colony (social bees) Contaminated flowers/ plants	*Crithidia bombi*	Increased defecation and reduced nutrition	Honey bees, bumble bees, other bees, flies
Neogregarines		*Ophryocystis elektroscirrha*	Impaired flight and reduced migration success	Monarch butterflies
Fungi	Mother-to-offspring Contaminated nest material and sites Contaminated flowers	*Ascosphaera* spp. (different species for different pollinators)	Chalkbrood: chalky-looking larvae that eventually die	Honey bees, bumble bees, other bees, wasps
		Vairimorpha spp. (formerly *Nosema*; different species for different pollinators)	Increased defecation and reduced nutrition	Honey bees, bumble bees, other bees, flies, wasps, butterflies & moths

Note that evidence of pathogen detection is not synonymous with evidence for disease and associated symptoms. For pathogens in the genus *Vairimorpha* and *Ascosphaera*, most pathogen species specialize on a restricted set of pollinator hosts. However, there are examples of honey bee–associated *Vairimorpha* species infecting bumble bees (Graystock et al. 2013) and mason bees (Müller et al. 2019) and studies showing that honey bee–associated *Ascosphaera* species (e.g., *Ascosphaera apis*) can cause infections in bumble bee and carpenter bee nests (Maxfield-Taylor et al. 2015). Similarly, there is considerable debate regarding the true host range of honey bee–associated viruses like deformed wing virus, given that this virus has been detected in over 65 arthropod species (Martin and Brettell 2019).

For pollinators to acquire these pathogens, the pathogen must survive in or on the flower before being picked up by a visiting pollinator. Once ingested, pathogens must also make it past host defense systems before they can cause disease and associated symptoms (Lovett & St. Leger 2017). Although this review focuses on floral-mediated transmission, pathogens can also be acquired from contaminated nest sites and from contact with infected nest mates and other organisms [e.g., *Varroa* mites (Wilfert et al. 2016)]. We focus on floral-mediated transmission because we know that pollinators visit diverse floral species (which should put pollinators in contact with various pathogens), yet we have relatively little evidence assessing which pollinators can acquire and be negatively impacted by different pathogens. This knowledge gap is both a key future direction and a guiding motivation for the topics discussed in this review.

3. PLANT AND POLLINATOR TRAITS INFLUENCING PATHOGEN DEPOSITION, ACQUISITION, AND INFECTION

Although it is important to extend the geographic and taxonomic breadth of the plant–pollinator–pathogen literature, a mechanistic and trait-based approach to understanding patterns of pathogen transmission may improve the generalizability of the current literature and guide more targeted future studies. Here, we review evidence assessing how plant and pollinator traits influence the deposition of infectious pathogens on flowers and pathogen acquisition as pollinators visit these contaminated flowers (**Figure 1*a***). We also assess how plant and pollinator traits impact whether acquired pathogens successfully infect (i.e., replicate in) different pollinator species.

3.1. Plant Traits Impacting Pathogen Deposition and Survival on Flowers

Floral morphology dictates the duration of contact between pollinators and potential pathogens as well as the likelihood and location of pathogen deposition, thereby shaping pathogen transmission. For widespread intestinal parasites of bees, such as *Crithidia bombi*, pathogen deposition occurs when bees deposit infected feces onto flowers while foraging (Graystock et al. 2015). Deposition of contaminated feces is strongly influenced by floral morphology (Figueroa et al. 2019, Pinilla-Gallego et al. 2022). For example, fecal deposition is higher in flowers with larger disk flower areas (Bodden et al. 2019) and in flowers that have wide displays (Pinilla-Gallego et al. 2022). Although it can be hard to distinguish between the direct role of trait variance and the underlying phylogenetic variance, experiments in which floral traits are manipulated confirm that reducing floral area reduces fecal deposition on flowers (Van Wyk et al. 2023). Floral morphology can also impact pathogen survival on flowers, which may influence the likelihood that floral visitors acquire pathogens while foraging. For example, exposure to ultraviolet (UV) light and desiccation can reduce the survival of *C. bombi* on flowers (Figueroa et al. 2019), and both UV exposure and desiccation can vary considerably with floral morphology. There is less evidence for the impacts of floral traits on the deposition and acquisition of viruses (McArt et al. 2014), but observational studies suggest that traits associated with increased *C. bombi* transmission (e.g., large and open bowl-shaped flowers) may have the opposite effect for viruses like deformed wing virus and black queen cell virus (Maurer et al. 2024). These contrasting results could be due to differential effects of increased UV light exposure on pathogen survival (Figueroa et al. 2019). However, Maurer et al. (2024) used an observational approach, and correlated factors (e.g., different patterns of pollinator visitation for different floral forms) may mask effects of floral traits on pathogen transmission. Manipulative studies could help untangle these correlated factors, and understanding the importance of floral traits in predicting virus deposition and acquisition is a rich area for future study.

3.2. Pollinator Traits Impacting Pathogen Acquisition

Plant traits influence patterns of pathogen deposition, but pollinator traits and behaviors ultimately determine whether pollinators come into contact and become infected by pathogens. Pollinators use a combination of visual, olfactory, and electrostatic signals and cues to make foraging decisions, and these cues may help pollinators detect and avoid pathogens on flowers. For example, bees are less likely to visit flowers contaminated with *C. bombi* (Fouks & Lattorff 2011) and *Ascosphera apis* (Yousefi & Fouks 2019). However, because pollinators navigate complex and constantly changing floral communities to acquire essential resources, they face constraints that may prevent them from accurately assessing flowers (Lichtenberg et al. 2020) and avoiding pathogens. Consequently, floral transmission of pathogens is common, and foraging behaviors shape pathogen contact patterns. For example, there is considerable variation in pollinator specialization and floral constancy (i.e., the tendency for individuals to specialize on a subset of flowers during a foraging bout), which determines the contact network in plant–pollinator–pathogen communities and how this network changes over time (Figueroa et al. 2020).

Increasing pollinator specialization and floral constancy are posited to influence pathogen acquisition in pollinators through a few mechanisms. First, in a community in which all floral visitors are highly specialized, pathogens may be contained within subnetworks of the larger plant–pollinator–pathogen contact network (Ellner et al. 2020). This is a double-edged sword; because some subnetworks contain species with traits more conducive to transmission, this partitioning into subnetworks can increase the persistence of pathogens that would have otherwise died out. On the other hand, partitioning also limits how widely the pathogens can spread across the full community. Additionally, many specialist bees and butterflies utilize host plants with chemical properties that reduce exposure to predation, parasitism, and/or pathogens [i.e., the enemy-free space hypothesis (Jeffries & Lawton 1984)]. For example, specializing on *Ranunculus* pollen significantly reduces the prevalence of chalkbrood in *Chelostoma florisomne* (Wynns 2012). Although we have good theoretical support for the potential advantages and disadvantages of specialization on pathogen spread, it is harder to pinpoint the impact of specialization in explaining results of observational studies. Certainly, the pollinators that tend to have the highest pathogen prevalence are highly generalist bumble bees and honey bees (e.g., Graystock et al. 2020). However, it is difficult to disentangle whether this association is due to social behavior, pollinator specialization, and/or host specialization by the pathogens themselves.

Other pollinator traits, such as body size, timing of emergence, and nesting habits, may also influence pathogen deposition and acquisition, but the available evidence is often contradictory. Studies that investigate within-species variation in body size find that larger bees have higher transmission potential due to more foraging trips, larger fecal volumes, and lower mortality rates (Van Wyk et al. 2021); however, evidence from studies investigating among-species variation is mixed (Figueroa et al. 2021). Similarly, monarch butterflies parasitized with *Ophryocystis elektroscirrha* often develop smaller wings as a result of larval infection, and these smaller butterflies may decrease parasite transmission through migratory culling, in which smaller, infected butterflies do not successfully migrate (Kendzel et al. 2023). As such, it is likely that increasing body size increases pathogen transmission in diverse pollinator species, particularly when body size is associated with increased dispersal and survival and larger gut volumes. Although variance in nesting habits could lead to differences in pathogen transmission among pollinator species (and bee species in particular), current work suggests these traits may be less relevant compared to other factors (Figueroa et al. 2021, Harmon-Threatt 2020). Likewise, the impacts of pollinator phenology are mixed and unlikely to be generalizable across diverse pollinators and ecosystems. For example, bee species emerging in the late summer are purported to be more vulnerable to pathogens (Hofmann et al. 2019), but some studies find that pathogen loads are higher in early-summer bees (Figueroa et al. 2021), and prevalence of honey bee-associated pathogens peaks in both early- and late-season wild bee communities (Graystock et al. 2020, Tiritelli et al. 2024).

Social behavior also has important consequences for pathogen transmission. Although most pollinator species are not highly social, social species are often the most common pollinators in plant–pollinator communities, and social behavior may thus disproportionately impact transmission dynamics (Figueroa et al. 2019). Eusocial pollinators live in dense social groups of closely related individuals, which increases the likelihood of transmission. However, social insects also possess behaviors to avoid, control, or eliminate infection, collectively referred to as social immunity behaviors (Cremer et al. 2007). For example, many species use hygienic behaviors such as allogrooming, corpse removal, and waste removal to prevent the accumulation of potentially harmful materials in the nest.

3.3. Plant and Pollinator Traits Impacting Infection

Although plant and pollinator traits influence pathogen deposition and acquisition, pathogen acquisition does not guarantee infection: Pathogen dose and immune defenses also play a role (Lovett & St. Leger 2017). As such, it is also important to separately consider how plant and pollinator traits impact the duration and severity of infections via changes in host nutrition, immunocompetence, and foraging behavior.

Well-nourished bees often experience fewer adverse effects of infection (Brown et al. 2003, Conroy et al. 2016), and proteins, lipids, and micronutrients in pollen can improve immunocompetence in bees (Brunner et al. 2014). However, well-nourished bees can sometimes harbor higher infection loads (Conroy et al. 2016), which may make them more likely to deposit pathogens on flowers. Furthermore, it is often unclear whether host nutrition improves tolerance to a pathogen (i.e., the pathogen is tolerated with minimal symptoms of disease) or resistance to a pathogen (i.e., the pathogen load is reduced and/or the infection is cleared) (Tuerlings et al. 2022). When resistance to a pathogen occurs, nutrition may help infected individuals clear infections quickly, reducing opportunities for transmission. In contrast, when tolerance to a pathogen occurs, good nutrition may help infected pollinators survive longer. This is good for the infected individual but may also increase opportunities for transmission to other individuals.

Pollinator dominance: a disproportionately high abundance of one pollinator species

The chemical and physical properties of nectar, pollen, and resin can also provide direct defenses against different pollinator pathogens. For example, the spiny structure of Asteraceae pollen grains helps bumble bees reduce *C. bombi* infections (Figueroa et al. 2023), although the specific mechanism remains unclear. Infected bumble bees spend more time collecting nectar from flowers with higher concentrations of iridoid glycosides (Richardson et al. 2016), which can reduce pathogen loads (Richardson et al. 2015). Similarly, monarch butterflies infected with *O. elektroscirrha* live longer when reared on *Asclepias curassavica* even though uninfected individuals live longer on *Asclepias incarnata* (Sternberg et al. 2012), presumably due to variable cardenolide composition in different milkweed species (Hoogshagen et al. 2024).

After pollinators become infected, behavioral changes in response to infection can further alter pathogen transmission. Host behavior may be altered (*a*) through adaptive defensive behaviors of the host, (*b*) through pathogen manipulation of the host, or (*c*) as an unintended byproduct of infection or immune activation. Changes in foraging behavior in response to infection have been documented in numerous pollinator species (Gegear et al. 2005, 2006; Wolf et al. 2014). However, although some behavioral changes benefit hosts, others may benefit pathogens and increase community-level transmission. For example, bumble bees infected with *C. bombi* tend to defecate more frequently (Giacomini et al. 2021), which could increase opportunities for pathogen deposition on flowers. Ultimately, given that almost all studies focus on bumble bees and honey bees, we need more studies with more diverse species to parse out the impacts of foraging behavior and social behavior in determining how infection status impacts pathogen transmission at the community level.

4. COMMUNITY-LEVEL FACTORS INFLUENCING PATHOGEN TRANSMISSION

Although it is important to understand the details of how pathogens move among individuals, it is also worth considering broader patterns of how pathogen transmission occurs in plant–pollinator–pathogen communities (**Figure 1*a***). For example, land-use change, beekeeping, and other anthropogenic activities can modify plant and pollinator communities, which may in turn affect pathogen transmission. Several empirical studies have been conducted to assess how community metrics such as the abundance and diversity of flowers and pollinators affect pathogen persistence and prevalence, and various mechanisms have been proposed to explain observed patterns. Here, we review these studies and mechanisms, with a focus on the effects of floral abundance, pollinator diversity, and pollinator dominance (i.e., a disproportionately high abundance of one pollinator species). One major challenge in interpreting and reconciling the results from different studies is the issue of context dependence, which we define here as variation in the sign and/or magnitude of ecological relationships when observed under different conditions or analyzed using different

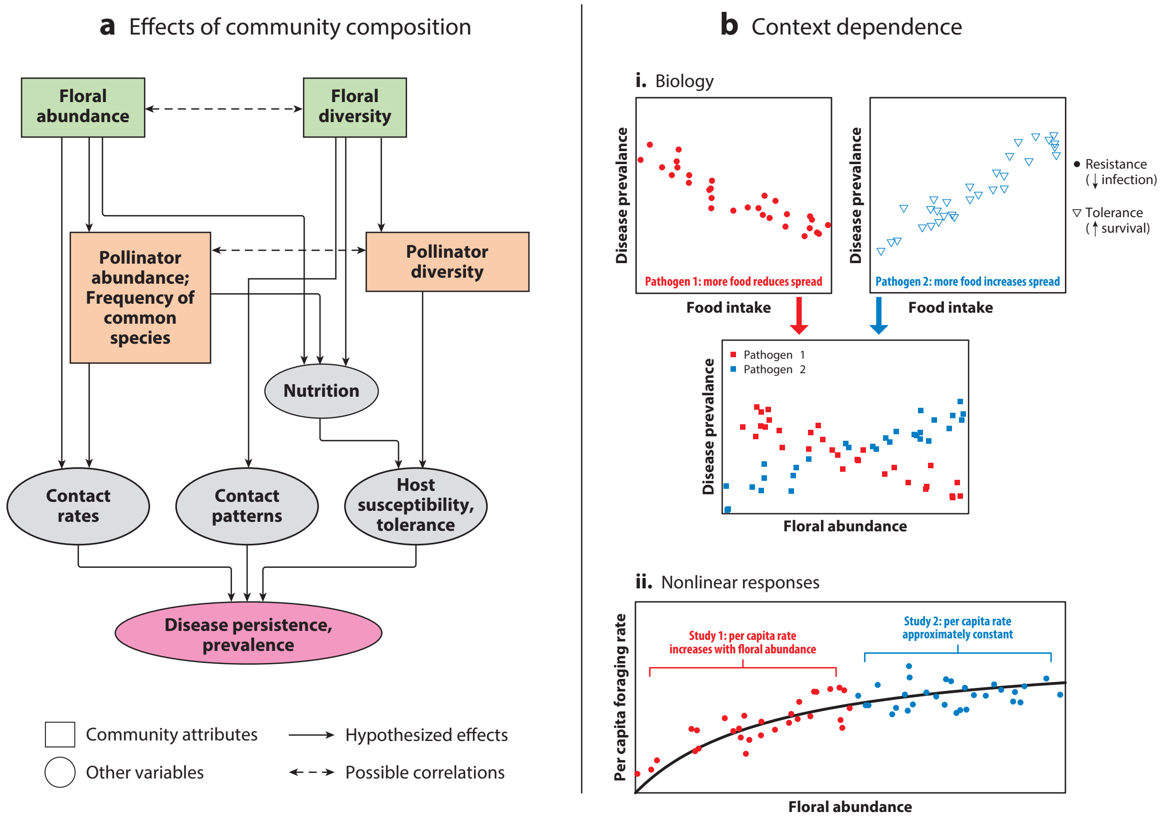

Figure 2

Ecological complexities present challenges in assessing the effects of community composition on pathogen transmission. Note that disease transmission is typically inferred in community-level studies, while pathogen prevalence is often what is measured. (*a*) Path diagram showing a non-exhaustive set of hypotheses (*solid unidirectional arrows*) for how plant and pollinator community composition may impact disease persistence or prevalence. Assessing the effects of a community attribute (*rectangles*) is complicated by the presence of multiple paths by which it can affect the outcome. For example, floral abundance can directly affect the rates at which pollinators encounter pathogens (dilution by numbers) and pollinator nutrition (which affects immune function and survival), but it can also indirectly affect these variables by modifying pollinator abundance. Additionally, depending on study design, floral or pollinator abundance may be correlated with other community attributes (*dashed bidirectional arrows*) that can also affect the disease outcome. (*b*) Context dependence can also lead to different outcomes between studies. (*i*) This may arise because of differences in biology between pathogens. For example, when food intake increases host resistance (i.e., pollinators reduce or eliminate infections), higher resource availability would be expected to decrease pathogen transmission by improving immunocompetence. In contrast, when food intake increases host tolerance to the studied pathogen (i.e., high infection loads with minimal symptoms), increasing floral abundance may increase host survival and amplify transmission. (*ii*) Context dependence may also arise because of nonlinear responses if studies differ in the range of values they span along a gradient. For example, if per capita foraging rate saturates with floral abundance, then a study across sites of low to medium floral abundance may find a strong relationship between foraging rate and floral abundance, whereas a study comparing sites with medium to high floral abundance may observe a much weaker relationship.

approaches (Catford et al. 2022). Context dependence may arise in multiple ways, as described below.

First, the community metric of interest may be correlated with or influence other community metrics that also impact disease transmission (**Figure 2*a***). For example, pollinator abundance is often correlated with floral abundance (Cohen et al. 2021, Piot et al. 2019), and these two factors may have opposite and counteracting effects on pathogen transmission. Second, pathogen biology influences the relationships between community metrics and transmission, so studies that focus on different pathogens may draw different conclusions. For example, increased food intake can increase host resistance to some pathogens (**Figure 2*b*, subpanel *i***), decreasing transmission by reducing pathogen deposition on flowers. However, increased food intake can also increase host tolerance, amplifying transmission by enhancing the survival of infected hosts, such that they spread pathogens longer. These competing mechanisms make it difficult to understand how increasing floral resource availability (which affects food intake) impacts pathogen transmission. Third, ecological relationships are often nonlinear, so two studies that span different ranges of values along a gradient may come to different conclusions (Catford et al. 2022). For example, if foraging rate saturates with increasing floral abundance (**Figure 2*b*, subpanel *ii***), then a study across sites of low to medium floral abundance may find a strong positive relationship between floral abundance and foraging rate (which in turn affects the rate of pathogen contacts), whereas another study across sites of medium to high abundance may observe a much weaker relationship. In the next section, we assess evidence for the impacts of floral abundance, pollinator diversity, and pollinator dominance on pathogen transmission. For each of these metrics of community composition, we also highlight the experimental contexts of different studies to shed light on why the results of empirical studies have been variable.

Pathogen dilution: when a more diverse community reduces transmission of a pathogen, or when a particularly abundant species reduces transmission

Pathogen amplification: when a more diverse community increases transmission of a pathogen or when a particularly abundant species increases transmission

4.1. Hypothesized Impacts of Floral Abundance on Pathogen Transmission

Floral abundance may either increase or decrease pathogen prevalence and infection outcomes through effects on host nutrition and immunity. Although adequate floral resources may improve pollinator nutrition and enhance immune response, thereby reducing prevalence (McNeil et al. 2020, Tuerlings et al. 2022), studies may fail to observe a negative relationship because the within-host biology is more complex than assumed or because floral abundance and floral diversity are correlated with other community metrics that also affect nutrition (McNeil et al. 2020, Tuerlings et al. 2022). First, some pathogens thrive in well-nourished hosts (Conroy et al. 2016), meaning that higher floral abundance could promote pathogen proliferation in communities. Similarly, improved nutrition may increase resistance (i.e., the pathogen load is reduced and/or the infection is cleared) against certain pathogens but increase tolerance (i.e., the pathogen is tolerated with minimal symptoms of disease) against other pathogens (Tuerlings et al. 2022) (**Figure 2*b*, subpanel *i***). Improved nutrition may counterintuitively amplify transmission in the latter case via increased host survival.

An alternative route by which floral abundance is hypothesized to influence pathogen prevalence is through the per capita contact rate (pathogen dilution by numbers). According to this hypothesis, when floral abundance is high, a susceptible pollinator is less likely to encounter contaminated flowers because there is a higher proportion of uncontaminated flowers (Graystock et al. 2020, Piot et al. 2019). However, this mechanism implies that the number of flowers visited by a pollinator during a foraging bout depends only weakly on floral abundance, because otherwise a strong increase in floral visits would compensate for the decreased probability of pathogen encounter per visit. Whether this assumption is valid or not depends on the relationship between visitation rates and floral abundance (**Figure 2*b*, subpanel *ii***). Alternatively, higher floral abundance may amplify pathogen prevalence by attracting large numbers of pollinators, increasing host density and hence the total pathogen contact rate (Cohen et al. 2021, Piot et al. 2019). If pollinator abundance increases at the same rate or faster than floral abundance, this may nullify any nutritional impacts, because pollinators will have either the same or lower access to food on a per capita basis.

Observational studies that relate floral abundance to pathogen prevalence demonstrate evidence of both pathogen dilution and pathogen amplification, although an important caveat is that most observational studies focus exclusively on bees. Fearon et al. (2023) found that high floral density was negatively associated with black queen cell virus prevalence across four bee genera, suggesting floral resources dilute pathogen prevalence. Similarly, McNeil et al. (2020) found a negative relationship between increasing quality of spring floral resources and pathogen loads in bumble bees. These observed correlations are partially corroborated by one manipulative study that found that floral supplementation on farms (using sown wildflower strips) reduced the prevalence of two out of the three viruses studied (Manley et al. 2023). However, these studies either do not test for or do not find correlations between floral abundance and pollinator:flower ratios, making it challenging to confidently conclude that floral abundance per se leads to a dilution effect via reduced pollinator:flower ratios (Fearon et al. 2023, Manley et al. 2023, McNeil et al. 2020). Additionally, dilution effects are often pathogen specific (Fearon et al. 2023, Ponisio et al. 2024), vary seasonally (Manley et al. 2023), and can depend on the surrounding landscape quality (Cohen et al. 2021, Piot et al. 2019).

Because floral abundance may simultaneously influence pollinator density, floral contact rate, and nutrition, it is challenging to disentangle competing mechanisms through which floral abundance impacts pathogen prevalence. For example, Cohen et al. (2021) found that when surrounding non-crop floral abundance was low, mass-blooming sunflower crops concentrate a large density of bees in sunflower fields, leading to high prevalence of a suite of pathogens (e.g., *Ascosphaera* spp., *Crithidia* spp., *Vairimorpha* spp.). Similarly, Piot et al. (2019) found that supplemental floral habitat amplifies pathogen prevalence in *Bombus pascuorum* foraging in low-quality habitat but not in high-quality habitat. To date, we are aware of only one study that has manipulated pollinator abundance (while keeping floral area constant), demonstrating that greater pollinator abundance increases the prevalence of one of the two pathogens studied (Bailes et al. 2020). This study is consistent with the hypothesis that higher pollinator:flower ratios amplify pathogen prevalence. However, the underlying mechanism remains elusive because increased flower sharing could increase pathogen prevalence via increased contact rates, but increased competition for flowers could also decrease nutrition (Page et al. 2024) and lead to similar results.

Taken together, these studies suggest that the impact of increasing floral abundance on pathogen transmission is likely nonlinear, with positive effects generally observed only when floral abundance and diversity are sufficiently high to counteract any correlated impacts of increased bee densities. However, these studies measure pathogen prevalence, not disease prevalence, and although contacting pathogens is a prerequisite to becoming infected, host nutrition, pathogen load, and other factors ultimately determine whether individuals are negatively affected via altered health and fitness. For example, increasing coverage of sunflower on farms was associated with reduced *C. bombi* infection and increased colony reproduction in *Bombus impatiens* (Malfi et al. 2023), consistent with results showing that sunflower pollen dramatically reduces infection (Figueroa et al. 2023). As such, floral abundance could alter community-level pathogen prevalence via changes in plant–pollinator–pathogen contact networks while having an entirely different impact on pathogen infection and host fitness outcomes via altered host diet.

4.2. Hypothesized Impacts of Pollinator Diversity on Pathogen Transmission

Pollinator diversity is hypothesized to dilute community-level pathogen prevalence by reducing the proportion of competent hosts (Fearon & Tibbetts 2021), and indeed some studies have observed a negative association between pollinator diversity and pathogen prevalence (Fearon & Tibbetts 2021, Graystock et al. 2020). However, as noted throughout the biodiversity-dilution literature (Johnson et al. 2015), the dilution hypothesis hinges on assumptions about community assembly and the transmission process. Community assembly refers to a conceptual framework in which a community can be regarded as being assembled from a regional species pool through a sequence of establishment and extinction events; the sequence of species additions and losses is known as the species assembly order. Community assembly can be additive when members of a new species simply add to community abundance. Alternatively, it can be substitutive when members of a new species replace existing individuals without affecting community abundance. Whether community assembly is additive or substitutive may affect the likelihood of observing a dilution effect, as explained below.

Pathogen spillover: when a reservoir species or population transmits a pathogen to a new species or population; this is not synonymous with disease spillover, because pathogens can spill over without causing disease

If diverse communities contain fewer competent hosts, biodiversity would be expected to decrease pathogen prevalence. This can occur if species assembly order is not random but correlated with host competence; for example, if more competent hosts tend to be earlier-establishing species and if less competent hosts are more likely to replace these species when invading a novel ecosystem. However, we may instead observe a positive relationship between diversity and pathogen transmission when invading species are more competent hosts and when these new hosts add to diversity and abundance. In these cases, pathogens may benefit from the increased density and frequency of competent hosts (Cohen et al. 2021). As such, the correlation between bee diversity and pathogen prevalence is often pathogen specific and context dependent (Ivers et al. 2022, Ponisio et al. 2024).

One reason why it may be difficult to assess the role of pollinator diversity on pathogen prevalence using observational data is that well-studied pathogens tend to affect common and well-studied species (e.g., honey bees and bumble bees), and the abundance of these species in a community can impact pathogen prevalence independently of diversity effects. Additionally, the abundance of common bee species can be both positively and negatively correlated with diversity. For example, honey bees are dominant visitors in biodiversity hotspots (Herrera 2020, Hung et al. 2019), but this pollinator dominance (i.e., disproportionately high abundance) is also associated with declines in the abundance and diversity of other bee species (Garibaldi et al. 2021). For these and other reasons, it is difficult to isolate the role of pollinator diversity on pathogen transmission.

4.3. Hypothesized Impacts of Pollinator Dominance on Pathogen Transmission

Increased species dominance can increase community-level pathogen transmission through two mechanisms. First, pollinator communities dominated by common species may show higher pathogen persistence if dominant species are known competent hosts. Second, these common species may act as reservoirs of both novel and endemic pathogens, increasing the risk of pathogen spillover to other species. Indeed, several studies have found a positive association between honey bee abundance and the prevalence of honey bee–associated pathogens (deformed wing virus and *Vairimorpha ceranae*) in bumble bees and mason bees (Alger et al. 2019, Fürst et al. 2014, Müller et al. 2019). Likewise, Graystock et al. (2020) found that *C. bombi* prevalence pooled across all bee species increased as the season progressed and honey bees and bumble bees became more dominant. On the other hand, Piot et al. (2019) did not find any association between honey bee abundance and the combined prevalence of six honey bee viruses in honey bees and in bumble bees. Fearon & Tibbetts (2021) and Fearon et al. (2023) also did not find any association between the abundance of honey bees and/or bumble bees and the prevalence of honey bee viruses (e.g., black queen cell virus, deformed wing virus).

Some of these discrepancies may be attributable to differences in study designs. For example, Piot et al. (2019) used the presence or absence of sown wildflowers as their treatment factor, making it hard to separate the effects of floral augmentation and honey bee abundance. It is also important to note that most of these studies used the abundance (i.e., total number of bees) rather than frequency (i.e., proportional abundance) of the dominant species as the predictor, therefore conflating the effects of total pollinator abundance and the frequency of common species. Some studies also suggest that resource overlap between honey bees and wild bees is a better predictor of pathogen prevalence than honey bee abundance (Maurer et al. 2024), because floral diversity and abundance also influence the likelihood that honey bees and other pollinators visit the same flowers. As discussed in the next section, it is important to reiterate that pathogen prevalence is not synonymous with disease prevalence or associated fitness outcomes, but the potential for dominant species to impact rare and declining species via disease spillover is certainly a growing concern for the conservation of healthy and diverse pollinator communities.

5. WHEN AND WHERE DOES PATHOGEN TRANSMISSION IMPACT POLLINATOR FITNESS?

Here, we delve into the spillover dynamics of common pollinator pathogens (**Table 1**) with a broader goal of understanding when and where pathogen transmission impacts pollinator fitness in diverse plant–pollinator communities (**Figure 1*b***). In particular, there is increasing concern that diseases plaguing managed bees may spillover to wild bee communities. For example, wild bumble bees near apiaries have higher incidences of honey bee–associated viruses (Manley et al. 2019, Singh et al. 2010), and bumble bee–associated pathogens like *C. bombi* and *V. bombi* are higher in wild bumble bees near managed bumble bee colonies (Colla et al. 2006). Honey bees may also be an important source population for early-season pathogen exposure because honey bees maintain high pathogen loads during winter months compared to overwintering queen bumble bees (Wham et al. 2024).

Although there is ample evidence that beekeeping activities can increase pathogen prevalence in wild bee populations, detecting viruses in wild bees does not necessarily mean that wild bees are competent hosts or that virus levels are high enough to cause disease. Additionally, many pathogens vary in their virulence, or harm-causing severity, depending on their host and the stressors those hosts encounter. For example, although negative consequences of honey bee–associated pathogens like deformed wing virus are well-documented in honey bees (Benaets et al. 2017), evidence for impacts on non-*Apis* species are mixed and context dependent. Some studies show that deformed wing virus in bumble bees can reduce colony fitness via rapid worker mortality (Fürst et al. 2014, Graystock et al. 2016). However, Tehel et al. (2020) found no impact of deformed wing virus inoculation on bumble bee survival, except under food starvation in lab settings. Similarly, Streicher et al. (2023) found that bumble bees inoculated with high levels of deformed wing virus via injection showed reduced survival under field conditions, but there were no impacts for orally inoculated bees, half of which were virus free by the end of the experiment. Taken together, these results suggest that high deformed wing virus levels can reduce bumble bee fitness, particularly under resource-stressed conditions, whereas low-level infections may have minimal impacts. An important caveat is that all published studies on this topic look at common and abundant host species such as *Bombus terrestris*. Other insect species may be more or less susceptible. However, deformed wing virus can cause wing deformities in both wasps and ants (Forzan et al. 2017, Miles et al. 2023), suggesting impacts may be prevalent across Hymenoptera.

Managed bumble bees, solitary bees, and other insects can also spread pathogens to other pollinator species. For example, the introduction of *B. terrestris* to Patagonia has been linked to the decline of *Bombus dahlbomii*, with pathogen spillover hypothesized to be a potential cause (Schmid-Hempel et al. 2014). Additionally, spillover of *V. bombi* from commercial to wild colonies is thought to be a major cause of the alarming declines in *Bombus occidentalis* and the potential extinction of *Bombus franklini* (Szabo et al. 2012). Most studies of spillover dynamics focus on social bees, but pathogens can also be spread by solitary bees and other insect pollinators. For example, non-native *Ascosphaera* species can spill over from non-native mason bees (*Osmia* spp.) to native species, with a high prevalence of non-native *Ascosphaera* species in nests of declining *Osmia georgica* bees (LeCroy et al. 2023). Additionally, spillback, a phenomenon in which pathogens spill into and are amplified in other species, then are reintroduced to the original host population, is an understudied risk with potential impacts for both wild and managed pollinator populations (Kelly et al. 2009). Although few studies unequivocally demonstrate spillback, there are several examples showing potential pathogen amplification in unconventional hosts. For example, deformed wing virus can spill over from honey bees to ants, amplifying virus loads in ant-infested hives (Dobelmann et al. 2023) and hoverflies can act as mechanical vectors of *C. bombi* (i.e., they transmit infectious pathogens passively, without being infected), potentially increasing spread among susceptible individuals (Davis et al. 2021). It is critical to understand these population and community level disease dynamics to draw a comprehensive picture of how disease impacts diverse pollinator populations.

6. HOW DOES PATHOGEN TRANSMISSION IMPACT POLLINATION?

A wide range of studies have estimated the value of global pollination services (e.g., Potts et al. 2016), with approaches that compare reproductive success between flowers with open pollination, hand pollination, and/or pollinator exclusion (Siopa et al. 2024). At the same time, studies of pollinator–pathogen dynamics often motivate their research by citing the value of pollination services (e.g., Adler et al. 2020) but do not assess the impacts of pathogens on such services. Here, we review the plant–pollinator–pathogen literature to assess whether pollinator pathogens impact pollination (**Figure 1*b***).

In general, there is a surprising lack of studies that explicitly connect pollinator–pathogen interactions to pollination (reviewed in Koch et al. 2017, Recart et al. 2023). Two observational studies have linked site-level pathogen prevalence to pollination, with contrasting results. Sites in the northeastern United States whose bumble bees (*Bombus* spp.) had high levels of *V. bombi* prevalence also had lower pollination services (conspecific pollen deposition and seed set) to two plant species (Gillespie & Adler 2013). In contrast, *V. bombi* and *C. bombi* prevalence was not correlated with pollination across a range of sites in Germany (Theodorou et al. 2016). Connections between pathogen prevalence and pollination could be driven by numerous mechanisms. Here we discuss four potential mechanisms (which are not mutually exclusive) by which pollinator pathogens could affect pollination.

First, and perhaps most obviously, pathogens could influence pollination by reducing the abundance of specific pollinator species. For example, in a U.S. survey, *V. bombi* prevalence was higher in *Bombus* species with sharply declining populations than in species with stable populations (Cameron et al. 2011). Deformed wing virus, along with the parasitic *Varroa destructor* mite, are together associated with heavy losses of honey bees worldwide (Martin et al. 2012). Reduced pollinator abundance could directly translate to reduced floral visitation by that species, and/or change dynamics that alter visitation by other pollinators (Taggar et al. 2021). Providing evidence for this mechanism relies on demonstrating that the reductions in pollinator abundance would reduce pollen delivery enough to impact plant reproduction (i.e., the quality and/or quantity of seeds sired or produced). Short-term experimental removal of a single pollinator species has been shown to reduce plant reproduction in one study (Brosi & Briggs 2013), but no studies to our knowledge unambiguously link pathogen-mediated pollinator decline to a loss in pollination and plant reproduction.

Second, even if pathogens do not reduce pollinator abundance, they can reduce visit frequencies and pollen collection. For example, honey bees infected with *Vairimorpha apis* foraged less, collected less pollen, and carried less pollen on their bodies compared to uninfected individuals (Lach et al. 2015). Similarly, free-foraging workers of several bumble bee species parasitized with *C. bombi* were less likely to collect pollen when compared to uninfected workers (Shykoff & Schmid-Hempel 1991), and *B. impatiens* experimentally infected with *C. bombi* made fewer foraging trips in tents than uninfected bees (Adler et al. 2020). Infection with deformed wing virus reduced the likelihood that honey bee workers survived to foraging age and reduced worker life span for those who did forage, reducing the number of foraging trips over a worker's lifetime (Banaets et al. 2017). In contrast, honey bees infected with *V. ceranae* took longer foraging trips and spent less time in the hive between successive foraging trips (Naug 2014). As such, different pathogens likely have different impacts on visit frequencies and pollen collection. However, the bulk of evidence suggests that pathogen infection reduces foraging, with likely negative effects on pollination.

Third, pathogens could alter other aspects of pollinator foraging behavior, including time spent per flower, the number of flowers visited per plant, or pollinator flight distances, which could affect both the quantity and genetic diversity of deposited pollen. For example, in a laboratory study, bumble bees (*B. impatiens*) infected with *C. bombi* visited fewer flowers per minute relative to uninfected bees (Otterstatter et al. 2005), and *C. bombi* infection increased flower handling times (Gegear et al. 2005). In a field study, free-foraging bees with higher levels of *C. bombi* infection spent less time foraging on damaged compared to control tomato plants (*Solanum lycopersicum*) (Aguirre & Adler 2022), suggesting that infection alters how bees respond to plant damage in ways that could affect pollen transfer. Honey bees infected with deformed wing virus had reduced flight distances and duration, which could affect plant outcrossing distances (Wells et al. 2016). As such, pathogens may negatively impact pollination by reducing both the quantity and quality of pollen transferred among plants. However, there remains a dearth of literature linking infection-mediated changes in foraging to pollination and plant reproduction.

Fourth, pathogens could affect pollinator preferences (Richardson et al. 2016, Schmid-Hempel & Schmid-Hempel 1990, but see Adler et al. 2020), as well as learning and memory (Gegear et al. 2006) in ways that might change conspecific and heterospecific pollen transfer. For example, free-foraging bumble bees infected with *C. bombi* foraged longer at *Chelone glabra* flowers with experimentally increased nectar iridoid glycosides (which reduce *C. bombi* infection) compared to flowers with low iridoid glycosides (Richardson et al. 2016). Uninfected bees had no preference, resulting in high-iridoid glycoside flowers donating more pollen to conspecific stigmas. Floral constancy can be altered by infection in species-specific ways; infection with tracheal mites increased floral constancy to a single flower color (Otterstatter et al. 2005), while *V. ceranae* infection in squash bees (*Eucera fervens*) was correlated with more pollen transported but not more pumpkin pollen, suggesting more generalist floral visits (Fernandez de Landa et al. 2022). Impacts on constancy may be related to cognition and learning. *B. impatiens* infected with *C. bombi* took longer to learn to associate cues with rewarding flowers in a laboratory study (Gegear et al. 2006), and honey bees infected with *V. ceranae* had modest impairment of olfactory learning (learning to associate an odor cue with sugar reward) compared to uninfected bees (Piiroinen & Goulson 2016). If impaired learning results in more generalized foraging patterns, this could increase heterospecific pollen deposition and reduce plant reproduction (as in Brosi and Briggs 2013).

Taken together, pathogen infection can reduce pollination through several mechanisms. Disease-induced declines in pollinator populations may reduce floral visits, while altered foraging may reduce the quantity and quality of conspecific pollen deposition. However, the overall impact of altered visitation and pollen deposition patterns depends on other factors, including the prevalence of infection and how much pollen delivery limits plant reproduction.

7. CONCLUSIONS AND FUTURE DIRECTIONS

Here, we summarize the concepts discussed in this review and suggest future research directions. Several conclusions are worth highlighting. First, plant and pollinator species traits influence pathogen transmission patterns, but these effects depend strongly on the biology of pathogens and their hosts. Second, although correlated factors impede our ability to assess the impact of plant and pollinator community composition on pathogen transmission, available evidence suggests that greater floral abundance and pollinator diversity reduce pathogen transmission, while greater pollinator dominance generally increases transmission. Third, there are several examples of pathogen spillover from managed social and solitary bees to wild bees, with associated reductions in bee fitness observed in some cases. This means spillover is a risk that should be taken seriously. Finally, infections can alter foraging behavior in ways that likely reduce pollen deposition and plant reproduction, but we need empirical studies that explicitly examine the link between pathogen transmission and pollination.

Taxonomic and geographic limitations are a consistent and consequential impediment to research progress, and we urge future studies to consider expanding the scope of their work to include more diverse organisms and study regions. However, beyond the obvious need to expand the taxonomic and geographic breadth of the current literature, we wish to highlight a few additional knowledge gaps. First, we need a better understanding of how abiotic factors influence pathogen survival in flowers. For example, although we know that desiccation and UV exposure can lead to degradation of *C. bombi* (Figueroa et al. 2019), we know less about how long different pollinator viruses remain viable in nectar, pollen, leaves and petals under different abiotic conditions. Understanding the impacts of abiotic factors like air temperature and humidity is important in evaluating the risk of pathogen transmission in different landscapes and under different climate change scenarios.

Second, context dependency makes it difficult to understand how community composition influences pathogen transmission, despite a wealth of excellent work on this topic. For example, higher pollinator abundance is hypothesized to amplify transmission, but it is often correlated with higher pollinator diversity, which is thought to dilute transmission. Likewise, increased floral abundance may reduce pathogen contact rates, but it may be hard to separate the effect of floral abundance from the effect of pollinator abundance, particularly given that social bees are often dominant visitors of more abundant flowers. To adequately assess how these factors impact pathogen transmission, future work should experimentally test the individual links between floral abundance, pollinator abundance, pollinator:flower ratios, quality of nutritional resources, and pathogen prevalence. In the absence of manipulative experiments, one way to disentangle the effects of floral abundance, pollinator diversity, pollinator abundance, and pollinator dominance would be to evaluate both flowers and bees for pathogens. Additionally, local versus landscape-scale effects may interact and should be considered in tandem. Local floral abundance, for example, may amplify pathogen prevalence when landscape-wide resources are low but dilute prevalence when landscape-wide resources are high (e.g., Cohen et al. 2021, Piot et al. 2019). Similarly, increasing pollinator abundance and dominance may increase pathogen transmission when bees are concentrated in low diversity floral patches but have less of an effect when spread throughout diverse resources across the larger landscape.

Third, pathogens associated with managed honey bee, bumble bee, and mason bee species have been detected in a wide diversity of other organisms (Colla et al. 2006, Deutsch et al. 2023, LeCroy et al. 2023), but we know surprisingly little about when and where pathogen acquisition leads to infection and disease. We have been excited to see many recent studies addressing this question (e.g., Streicher et al. 2023). However, there is still considerable uncertainty regarding who can be infected and how infection impacts mortality, behavior, and pollinator fitness outside of a few well-studied honey bee and bumble bee species. Relatedly, although disease-induced changes in pollinator populations and foraging behavior have the potential to impact pollination, no studies unambiguously link the transmission of pollinator pathogens to impacts on pollination, making this a particularly rich area for future study.

The manipulative experiments and observational studies required to address these questions are understandably difficult but will considerably deepen our understanding of how pollinator pathogens impact diverse plant–pollinator communities. We hope an improved understanding of the mechanisms and consequences of plant–pollinator–pathogen interactions can inform more effective conservation of diverse ecological communities while also unraveling the intricacies of these fascinating multitrophic interactions.

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