Ecology Letters

WILEY

ECOLOGY LETTERS

LETTER OPEN ACCESS

Drivers of Viral Prevalence in Landscape-Scale Pollinator Networks Across Europe: Honey Bee Viral Density, Niche Overlap With This Reservoir Host and Network Architecture

Willem Proesmans[1,2] | Cedric Alaux[3] | Matthias Albrecht[4] | Karima Bassit[5] | Nathan Cyrille[1,6] | Anne Dalmon[3] | Virginie Diévart[3] | Christophe Dominik[7,8] | Emeline Felten[1] | Anna Gajda[9] | Jonna M. Heuschele[7,8] | Emilien Laurent[1] | Corina Maurer[4] | Peter Neumann[10] | Maryline Pioz[3] | Alexandria Schauer[10] | Oliver Schweiger[7,8] | Josef Settele[8,11] | Eckart Stolle[5] | Hajnalka Szentgyörgyi[12] | Orlando Yañez[11] | Yicong Liu[5,7] | Aleksandra Żmuda[12,13] | Robert J. Paxton[5,8] | Adam J. Vanbergen[1]

[1]Institut Agro Dijon, INRAE, UMR Agroécologie, Université de Bourgogne Europe, Dijon, France | [2]Laboratory of Zoology, Research Institute for Biosciences, University of Mons, Mons, Belgium | [3]INRAE, Abeilles et Environnement, Avignon, France | [4]Agroscope, Agroecology and Environment, Zürich, Switzerland | [5]General Zoology, Institute of Biology, Martin Luther University Halle-Wittenberg, Halle (Saale), Germany | [6]Biogéosciences, UMR 6282 CNRS, Université Bourgogne Europe, Dijon, France | [7]Department of Community Ecology, Helmholtz Centre for Environmental Research—UFZ, Halle (Saale), Germany | [8]German Centre for Integrative Biodiversity Research (iDiv) Halle-Jena-Leipzig, Leipzig, Germany | [9]Institute of Veterinary Medicine, Warsaw University of Life Sciences, Warsaw, Poland | [10]Institute of Bee Health, Vetsuisse Faculty, University of Bern, Bern, Switzerland | [11]Department of Conservation Biology and Social-Ecological Systems, Helmholtz Centre for Environmental Research—UFZ, Halle (Saale), Germany | [12]Institute of Botany, Faculty of Biology, Jagiellonian University, Cracow, Poland | [13]Doctoral School of Exact and Natural Sciences, Jagiellonian University, Cracow, Poland

Correspondence: Willem Proesmans (willem.proesmans@umons.ac.be)

Received: 17 March 2025 | **Revised:** 2 December 2025 | **Accepted:** 8 December 2025

Keywords: BQCV | directed acyclic graphs | DWV | floral resources | honey bee | modularity | niche overlap | RNA virus | urbanisation | wild bee

ABSTRACT

Viral transfer from managed pollinators potentially threatens wild pollinators and may be exacerbated by land-use changes. Our causal models and plant-pollinator network data from 48 European urban and agricultural landscapes revealed the ecological mechanisms underpinning viral transmission. Host identity, network architecture and land-use modulated viral dynamics (black queen cell virus, BQCV; deformed wing virus, DWV-A and DWV-B). Viral prevalence in wild pollinators was driven by viral density in the reservoir host: honey bees, and secondarily by trophic niche overlap with these managed pollinators. Modular networks limited BQCV prevalence, which was driven by reduced honey bee niche overlap, suggesting minimal onward transmission among wild pollinators. Landscapes supporting greater wild pollinator abundance diluted DWV-B transmission; in urban landscapes managed honey bees and wild pollinators experienced higher and lower BQCV prevalence, respectively. Disease in managed bee colonies and land-use changes that concentrate pollinator foraging interactions present potential viral risks to wild pollinator health.

1 | Introduction

Anthropogenic disruption of ecological communities creates novel species interactions that provide potential pathways of pathogen transfer to novel hosts, risking emerging infectious diseases (Mahon et al. 2024). RNA viruses are major disease-causing pathogens in insect pollinators, especially those of the managed western honey bee, *Apis mellifera* (IPBES 2016).

Managed honey bee colonies and their translocation locally and globally, together with the ability of RNA viruses to rapidly adapt to novel environments and hosts (Grozinger and Flenniken 2019), make honey bees a major source of pathogen transmission to sympatric wild pollinator populations (Vanbergen et al. 2018). Host-pathogen coevolution means that the identity and phylogenetic relatedness among host species often limit pathogen transmission from primary to alternative hosts (Grozinger and Flenniken 2019). Nonetheless, to date, viruses typically associated with managed honey bees have been associated with a range of wild social and solitary bee taxa (Fearon and Tibbetts 2021; Fürst et al. 2014; Manley et al. 2019). Such incidents may reflect the process of pathogen 'spill-over,' which may result in disease-causing infection in secondary competent hosts, but which starts with interspecific contact events that affect the transmission and prevalence of pathogens in the community (Becker et al. 2019).

Pathogen prevalence may also be affected by land-use changes that restructure plant-pollinator networks at landscape scales (Figueroa et al. 2020; Maurer et al. 2024; Redhead et al. 2018), potentially creating novel viral transmission pathways at whole community levels (Proesmans et al. 2021). However, the extent and species- or network-level mechanisms of interspecific pathogen transmission from managed honey bees foraging in phylogenetically diverse wild pollinator networks and how they are modulated by land use remain an active research question (Fearon et al. 2023; Fearon and Tibbetts 2021; Manley et al. 2023; Maurer et al. 2024).

Plant-pollinator network structure provides one paradigm for exploring the transmission dynamics of pollinator-borne pathogens. As highly abundant generalists, honey bees often occupy a central place in plant-pollinator networks, potentially acting as hubs for pathogen transmission (Proesmans et al. 2021). Faecal-oral transmission during shared flower use is one potential horizontal exposure pathway between pollinator species overlapping in trophic niche (Bodden et al. 2019; Burnham et al. 2021; Figueroa et al. 2019). Highly modular networks might, however, constrain pathogen spread to within subsets of pollinator species foraging on specific shared flowers, thereby limiting community-wide pathogen exchange (Evans et al. 2021).

Land-use changes such as urbanisation and agricultural intensification markedly influence the diversity, abundance and availability of floral resources and accordingly the diversity and foraging patterns of pollinators (Proesmans et al. 2024). Heterogeneity in floral communities also modulates pollinator species competition over pollen and nectar sources (Sponsler et al. 2023), altering the chance of contact within a network. Moreover, floral resource availability also governs honey bee densities through their foraging behaviour and beekeepers' decisions to migrate their hives (Lázaro et al. 2021). Land-use and the distribution of floral communities in landscapes are therefore key to influencing managed honey bee densities and the structure of pollinator networks, and thus the potential for pathogen exchange. Intensive agricultural landscapes are often deficient in floral resources and may concentrate pollinator foraging and densities on few mass-flowering crops (MFC), forming a hub for pathogen transmission (Cohen et al. 2021). In contrast, where MFC resource pulses cover a large area or where agri-environmental wildflower plantings occur, the chance of interspecific pollinator contacts and pathogen exchange may be diluted (Manley et al. 2023; Piot et al. 2021). Similarly, urban landscapes can produce a habitat mosaic harbouring diverse floral resources and pollinating insects (Baldock et al. 2015; Proesmans et al. 2024), potentially diminishing floral resource overlap or diluting the contact frequency of potential susceptible hosts. Few studies have considered how plant-pollinator-pathogen transmission networks respond to gradients in agricultural to urban land-use (Maurer et al. 2024) and none across a continent.

Here we identify the direct and indirect community mechanisms influencing viral pathogen transmission from managed honey bees to wild pollinators along landscape-scale gradients of agricultural and urban land use, and hence floral resources across temperate Europe (48 landscapes). We used a directed acyclic graph (DAG) to identify a causal framework of ecological predictors of the prevalence and load in wild insect pollinators of three highly abundant and virulent RNA viral targets (black queen cell virus-BQCV, deformed wing virus-DWV-A and DWV-B) (Nanetti et al. 2021) known to infect managed honey bees. Our causal Bayesian framework allowed us to go beyond correlative approaches to test hypothesized mechanisms (Franks et al. 2025).

We hypothesized that managed honey bees would act as a reservoir host for the viruses and be the principal driver of viral prevalence in recipient wild pollinator species via niche overlap in flower use. Furthermore, we hypothesized that urbanisation and agricultural intensification would affect viral transmission through changes in plant-pollinator community composition (e.g., occurrence of primary and alternative potential host insects) and network architecture.

2 | Material and Methods

2.1 | Study Landscapes

The study was conducted in 48 landscapes across Europe clustered in four regions: Burgundy (France), Northern Switzerland, Saxony-Anhalt (Germany) and Lesser Poland Voivodeship (Poland) (Figure S1). Within each region, 12 landscapes were selected: four dominated by intensive agricultural land use (≥ 75% of arable cropland and intensively managed grassland); four in mixed rural areas (~50%–50% mix of intensive agricultural land and semi-natural habitats); and four in urbanised areas (≥ 80% urban land use including buildings, roads, parks and gardens). To prevent spatial autocorrelation and ensure sites were independent according to pollinator mobility, landscape centroids were set at least 3.5 km apart. Within a 1000 m radius of the landscape centroids, land use was classified following the EUNIS (level 3) Habitat Classification (Davies et al. 2004).

2.2 | Pollinator Sampling

Each landscape was sampled for bees and hoverflies using standardised transects (between 09:00 and 18:00, low wind

speed (Beaufort < 3), dry day ≥ 14°C) at three time periods (early season: mid-April–mid-May; mid-season: late May–late June; late season: mid-July–mid-August) during 2021 (2020 in Switzerland). During each visit, all land use types within 500 m of the landscape centroid were classified into 'pollinator habitat' and 'non habitat' by assessing floral resources in situ over the sampling season (e.g., a field only was counted as pollinator habitat during a mass-flowering event, a forest with rich understory only counted during spring flowering). Sub-transects were situated in these different pollinator habitats, with their length proportional to total cover of each habitat (e.g., if meadows equaled 30% of the flowering pollinator habitat area, a 300 m sub-transect in meadows was sampled), which collectively summed to a total transect length of 1000 m per landscape. Each landscape was sampled for bee and hoverfly flower visitors within 1 m from the observer for 2 h total per landscape (i.e., 6 min per 50 m) and time period. Stopwatches were paused for handling and storing the insects and for noting the visited plant taxon. For an independent measure of honey bee density, the total length of 1000 m transect was re-walked for 20 min, during which all honey bees visiting flowers were counted.

Collected pollinators were immediately placed on dry ice and then stored at −80°C in the laboratory. Pollinators were identified to species level, or morphospecies when species identification was not possible (e.g., *Lasioglossum* cf. *simplex* or the *Bombus terrestris* complex).

2.3 | Floral Resources

Flowering plants were surveyed in 0.5 × 2 m quadrats along the pollinator transects, with 1 quadrat per sub-transect for homogeneous vegetation (e.g., crop or grass monoculture) and 3 quadrats per sub-transect for heterogeneous vegetation. Within each quadrat, the number of floral units per plant species (e.g., single flowers, umbels, capitules) was counted.

Total nectar sugar availability per m^2 was calculated by taking species (or closest relative if unavailable) nectar sugar content from existing datasets (Baude et al. 2015; Filipiak et al. 2022) and multiplying with the number of floral units. Mean potential nectar sugar content (mg m^{-2}) per sub-transect was calculated by averaging the values per quadrat, while the total potential nectar sugar content for the transect per landscape was calculated as the mean of the sub-transects, weighted for their respective length.

For each plot, floral Shannon diversity was calculated per quadrat, weighted by nectar production per plant species. This was then averaged over all floral quadrats of the same sub-transect type, and mean sub-transect nectar diversities were averaged and weighted by sub-transect length to derive a landscape-scale nectar diversity metric.

2.4 | Network Metrics

Plant-pollinator networks were constructed for each individual landscape visit (12 sites × 4 regions × 3 site visits = 144 networks) and modularity and connectance were calculated using the 'bipartite' package v2.19 (Dormann et al. 2009; Newman 2006) in R 4.4.0 (R Core Team 2024). Modularity (Q) was calculated by applying Newman's modularity measure in a bipartite weighted version, using the computeModules function (Newman 2006). If < 3 species of plants or pollinators occurred in the network, no modularity or connectance scores were calculated, as the small network size precludes a reasonable biological interpretation. Additionally, raw species numbers and pollinator abundance were calculated for each network. At the species level, for each network, the niche overlap of wild pollinators with honey bees was calculated as the respective Horn-Morisita distance to honey bees (Horn 1966), with 0 indicating no niche overlap and 1 indicating complete niche overlap.

2.5 | Viral Analysis

Among the most abundant species (defined as species with ≥ 7 specimens in any single network—that is, site visit—and occurring in three networks), up to 10 female honey bees, bumble bees, solitary bees or hoverflies per species and per network were screened for virus. In Poland and Germany, hoverflies were not analysed because they did not reach the threshold abundance. Samples were screened for three common honey bee viruses that have been frequently detected in other insects (Gisder and Genersch 2017): DWV-A, DWV-B and BQCV. Samples were randomised before RNA extraction to avoid bias towards species, season or collection site.

RNA extraction, cDNA synthesis and qPCR for absolute viral quantifications (viral loads per insect) were performed in every country following a standard protocol (de Miranda et al. 2021) (See Table S1; Appendix S2: Supporting Information: methods).

Technical deviations (e.g., instrumentation, reagents) among participating laboratories (INRAE-Avignon; Institute of Bee Health-Bern; Institute of Veterinary Medicine-Warsaw; Martin Luther University-Halle-Wittenberg) are described in Table S2. A ring test between these labs of a common set of 6 positive samples demonstrated uniformity in results, though to account for subtle differences in sensitivity we provide data as genome equivalents (GE) of virus per µg RNA. Sample sizes and number of positives per pollinator species are given in Table S3.

2.6 | Statistical Modelling

A Directed Acyclic Graph (DAG) was composed for models on wild pollinator BQCV and DWV-A/B prevalence (Figure S2) and load (Figure S3). Analysed variables are displayed in Table S4. Our model incorporated the causal hypotheses from our study (Table S5) and allowed us to compose models and account for confounding variables, yielding unbiased effect sizes (Arif and MacNeil 2023). All statistical analyses were carried out in R 4.4.0 (R Core Team 2024) and Stan 2.32.2 (Carpenter et al. 2016). Statistical models were composed based on the DAG. Continuous variables were scaled by subtracting the mean and dividing by the standard deviation. Models were fitted within a Bayesian framework, using a

Hamiltonian MCMC algorithm. For all model coefficient parameters, weakly informative priors were used, consisting of a normal distribution with mean 0 and standard deviation 1, while priors for standard deviations followed an exponential distribution with $\lambda = 1$. To account for potential spatial autocorrelation, in each model, we created four Gaussian Processes (McElreath 2020), with one single kernel for all regions, as the spatial scale for the autocorrelation was not assumed to differ between the regions. We used a squared exponential kernel defined as:

$$K_{ij} = \eta^2 \exp\left(-\rho^2 {D_{ij}}^2 \right) \quad (1)$$

with K_{ij} the covariance between two points and D_{ij} as the distance between the two points. For the η^2 and ρ^2 parameters, a prior with an exponential distribution with $\lambda = 1$ was used.

Models were fitted in Stan using the ulam function from the rethinking package v2.40 (McElreath 2020). We ran four chains with a warmup of 2000 iterations and 2000 sampling iterations. Convergence was determined by checking traceplots and trankplots, as well as confirming that the improved Gelman-Rubin convergence diagnostic (Rhat) was <1.03 for all parameters (Vehtari et al. 2021). Diagnostic plots were used to verify model assumptions.

As honey bee abundance, viral load and viral prevalence interactively increase exposure risk to all three viruses, a composite variable was created, named 'honey bee viral density', using the following equation:

$$\begin{aligned} &\text{HB viral density} = \text{HB Viral prevalence} \times \\ &\left[\log(\text{HB Abundance} + 1) + \log(\text{HB Viral load})\right] \end{aligned} \quad (2)$$

For 18 out of 144 plant-pollinator networks, honey bees were unobserved, making it impossible to assess their mean viral load and prevalence. Additionally, as the number of analysed honey bees differed between landscapes, this often led to different degrees of uncertainty of our measurements. For all viruses, we ran models based on the DAG to impute viral load where it was missing and to explicitly account for sample size and variance at the landscape level (M2a, b, c), with the model yielding a larger Bayesian standard deviation for sites with lower sample size. We similarly modelled viral prevalence, which allowed us to extract the modelled viral prevalence in honey bees per site (M1a, b, c). We then used the modelled distribution of the honeybee viral density (example in Figure S4) as an explanatory variable in our models.

In networks without honey bee observations, honey bee niche overlap (Horn-Morisita) was imputed using a regression model based on the DAG (Figure S2). In one network, nectar abundance and diversity data were missing, while in nine networks the network size was too small to calculate robust network metrics. These were similarly imputed using regression models based on the DAG.

A sensitivity analysis was carried out to calculate the relative contribution of the three variables of Equation 2 to honey bee viral density. Taking the partial derivatives for each variable on each data point, we then calculated each variable's contribution by multiplying the squared partial derivative per variable with its variance. We then calculated relative contribution as the relative contribution of each variable divided by the sum of the contributions of all variables.

Models were run addressing our hypotheses (Table S5) using the DAG to assess fixed effects to be included, to close backdoor paths and obtain unbiased effect sizes (Figures S2 and S3). For continuous response variables, a general linear mixed effects model was used, with landscape nested in country as a random effect. Viral prevalence was measured at the level of individual pollinators as a Bernoulli distributed variable, while the log-transformed viral load in positive individuals was modelled as a normally distributed variable. When viral prevalence was the response variable, a logistic regression was fitted, with landscape nested in country as a random effect. Viral load was modelled as a general linear mixed effects model, with landscape nested in country as a random effect.

Raw model outputs (M) are given in Tables S6–S49. We calculated 95% credible intervals by extracting the posterior samples and calculating the 2.5th and 97.5th percentiles.

3 | Results

In total, we observed 14,809 plant-pollinator interactions and analysed viral presence in 3734 pollinators. The models that were based on the DAGs (Figures S2 and S3) to obtain unbiased effect sizes for all variables are given in Tables S6–S49. We refer to the respective models (M) for each result.

3.1 | Viral Prevalence and Load Among Pollinators

Both BQCV (model M7a, Figure 1a) and DWV-B (M7c, Figure 1c) were widespread in pollinator communities, being most prevalent in honey bees ($N = 1297$; BQCV = 91.5, 95% CI [89.9–93.0]; DWV-B = 73.3 [70.4–76.0]%), then bumblebees ($N = 1156$; BQCV = 56.6 [52.0–61.0]; DWV-B = 42.0 [37.9–46.2]%), while in solitary bees ($N = 1125$; BQCV = 7.6 [6.0–9.4]; DWV-B = 36.8 [33.3–40.5]%) and hoverflies ($N = 156$; BQCV = 10.3 [6.7–15.1]; DWV-B = 18.9 [13.2–25.6]%) viral prevalence was low. DWV-A (M7b, Figure 1b) was most prevalent in honeybees (8.9 [7.0–11.0]%) compared to wild pollinators (bumblebees: 4.8 [3.4–6.5], solitary bees: 5.1 [3.8–6.6] and hoverflies: 5.7 [2.3–11.1]%). Viral load followed the same pattern (BQCV: M8a, DWV-A: M8b, DWV-B: M6c, Figure 1d–f). While prevalence and load of DWV-B mirrored phylogenetic and functional distance to the primary host, the honey bee, BQCV prevalence and load was slightly higher in hoverflies than in solitary bees (Figure 1a,d).

3.2 | Honey Bees as Primary Vectors

Honey bee viral density (Equation 2) was the predominant predictor of viral prevalence in wild pollinators for all viruses (BQCV: model M3a, $\beta = 0.89 \pm$ SD 0.20, 95% CI [0.51–1.28]; DWV-A: M3b, $\beta = 1.94 \pm 0.34$, [1.30–2.62]; DWV-B: M3c, $\beta = 1.03 \pm 0.23$ [0.59–1.48], Figure 2a–c). Our sensitivity analysis showed that

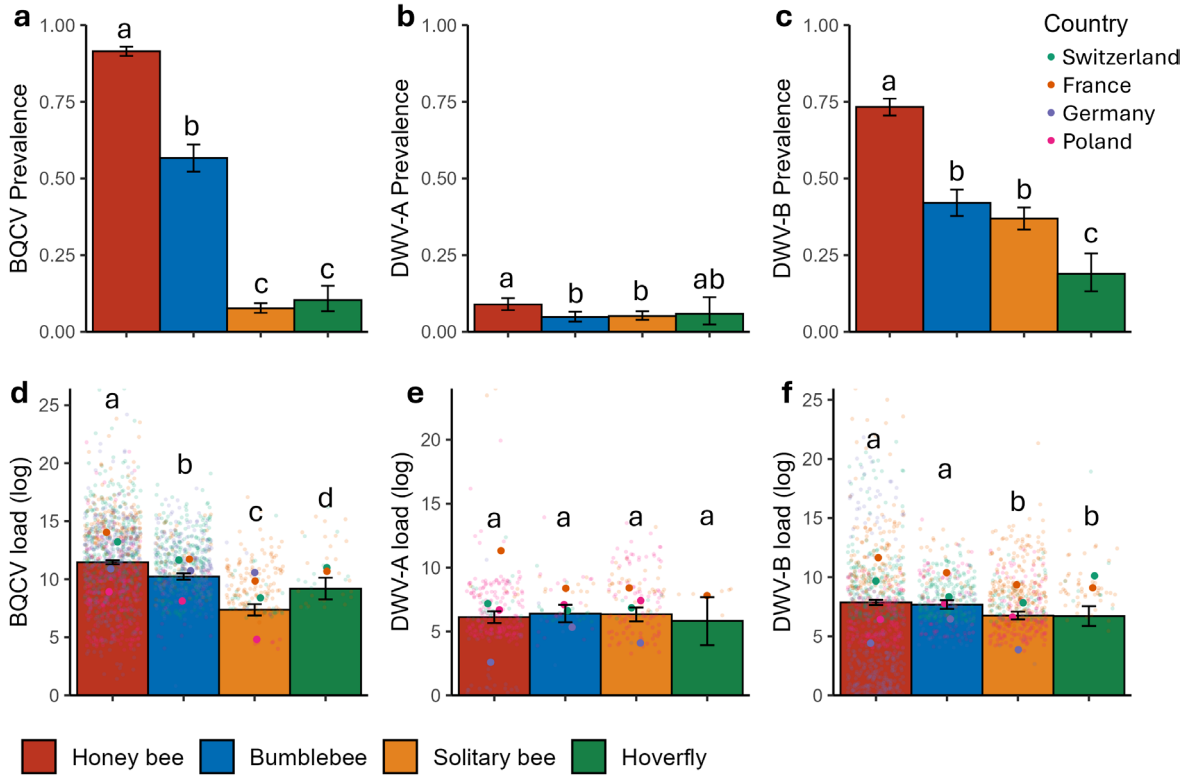

FIGURE 1 | Posterior estimates of BQCV, DWV-A and DWV-B prevalence and load per pollinator group. BQCV (a), DWV-A (b) and DWV-B (c) prevalence are highest in honey bees, followed by bumblebees. Solitary bees and hoverflies have the lowest prevalence, except for DWV-A, in which wild pollinators do not significantly differ in viral prevalence among each other. Viral load for BQCV (d), DWV-A (e) and DWV-B (f) is expressed as the log number of viral copies per μg of RNA. Values were calculated based on models 4 (prevalence) and 5 (load) under average values for urbanisation and agricultural intensification, and averaged over the effects of sampling round, country and site. The bars display the modelled posterior mean viral prevalence and load. Error bars indicate 95% credible intervals, while different letters indicate at 95% probability that groups differ. Raw data points are given for viral load, but not for viral prevalence, which is Bernoulli distributed (0 or 1). Diamonds indicate the mean observed viral load per country. Hoverflies were only analysed in Switzerland and France, which had a higher mean viral load, leading to higher observed viral loads compared to the overall modelled posterior.

variance in honey bee viral density was mainly due to viral load of the widespread BQCV and DWV-B (BQCV: 51.1%; DWV-A: 10.8%; DWV-B: 56.7%), while prevalence was the main factor explaining variance in viral density of the rarer DWV-A (BQCV: 35.8%; DWV-A: 87.0%; DWV-B: 31.0%), with minimal variance driven by differences in honey bee abundance (BQCV: 13.1%; DWV-A: 2.2%; DWV-B: 12.3%).

BQCV prevalence in wild pollinators increased with floral niche overlap with honey bees (M3a, 0.58 ± 0.09, 95% CI [0.41–0.75]). For DWV-A and DWV-B, the effect of niche overlap was much smaller (DWV-A: M3b, 0.09 ± 0.16, [−0.22 to 0.40]; DWV-B: M3c, 0.11 ± 0.10, [−0.08 to 0.30]), but it showed a positive interaction with honeybee viral density (DWV-A: M3b, 0.28 ± 0.12, [0.06–0.52]; DWV-B: M3c, 0.22 ± 0.08, [0.07–0.37]), indicating that niche overlap only plays a role when honeybee viral density is high (Figure 2d–f).

3.3 | Species Composition and Network Structure

For DWV-B, a strong dilution effect was detected in networks with a high wild pollinator abundance, which had considerably lower viral prevalence (M3c, −0.58 ± 0.24, [−1.05 to −0.13], Figure 3), while species richness showed an inverse effect (M15c, 0.48 ± 0.17 [0.14–0.81]).

Plant-pollinator network architecture modulated interspecific viral transmission. BQCV prevalence in wild pollinators was reduced in more modular networks (M6a, −0.40 ± 0.12, [−0.64 to −0.15]), DWV-B prevalence, contrarily, was increased by network modularity (M6c, 0.52 ± 0.14, [0.24–0.80]) and connectance (M6c, 1.05 ± 0.25, [0.56–1.56]). Honey bee niche overlap was lower in modular networks (M10, −0.62 ± 0.04, [−0.70 to −0.54]) and, after conditioning for this, modularity lowered BQCV prevalence (M3a, −0.49 ± 0.20, [0.11–0.89]) and connectance showed a positive direct effect on BQCV prevalence (M3a, 0.62 ± 0.23, [0.17–1.08]). Similarly, after conditioning on honey bee niche overlap, modularity and connectance increased DWV-B (M3c; modularity: 0.50 ± 0.21, [0.09–0.91]; connectance: 0.67 ± 0.27, [0.16–1.21]). DWV-A was not directly or indirectly affected by network structure (M3b, M6b).

3.4 | Landscape Context

Urbanisation (M12a, $\beta = -0.20 \pm 0.12$, [−0.44 to 0.05]) and agricultural intensification (M12a, $\beta = -0.22 \pm 0.12$, [−0.46 to 0.03])

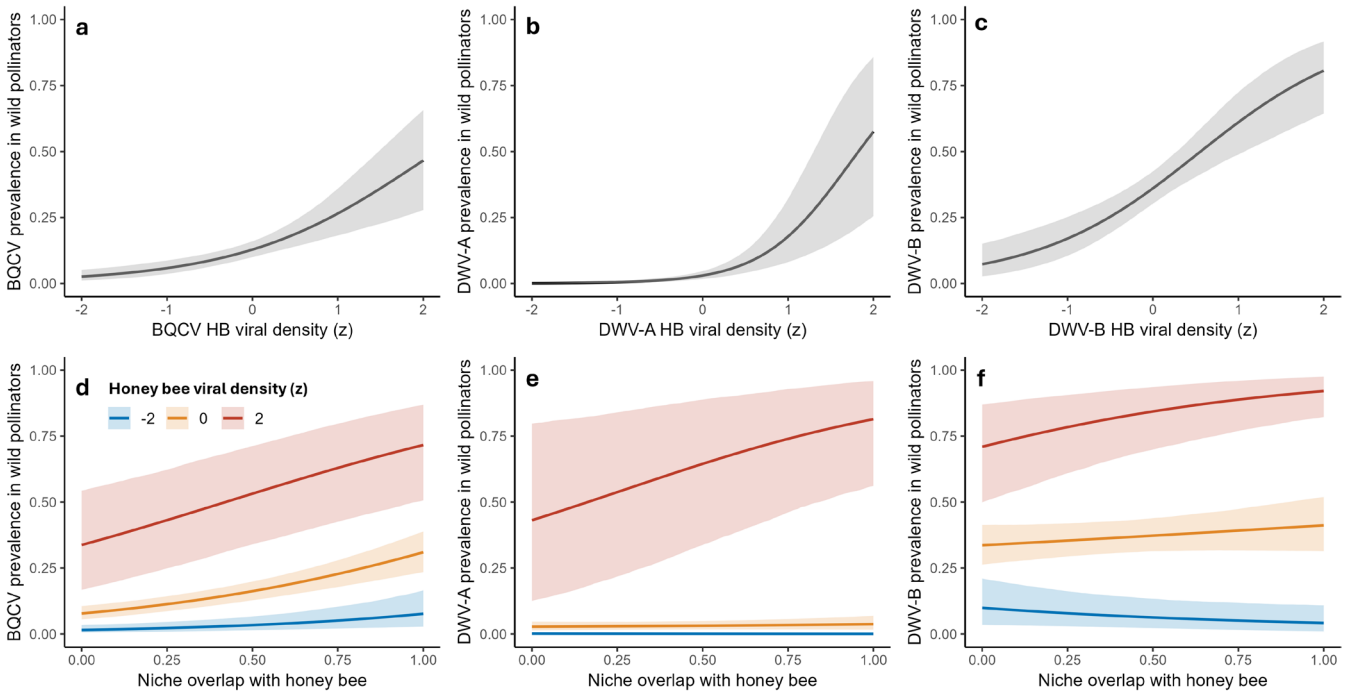

FIGURE 2 | The marginal effects of honey bee viral density and honey bee niche overlap on viral prevalences in wild pollinators. Graphs a-c show the relationship between viral prevalence in wild pollinators and viral density of the respective virus (in honeybees) averaged over taxonomic, seasonal, country and site effects and under average niche overlap. Shading represents the 95% credible intervals. BQCV (a), DWV-A (b) and DWV-B (c) prevalences in wild pollinators are strongly driven by their respective viral densities in honey bees. Graphs d-f show how the effect of niche overlap interactively changes under low ($z=-2$; blue), mean ($z=0$; orange) and high ($z=2$; red) honeybee viral densities. BQCV prevalence (d) strongly increased with niche overlap with honey bees, regardless of honeybee viral density, while niche overlap only had a strong effect on DWV-A (e) and DWV-B (f) prevalence under high honeybee viral density, with niche overlap playing a limited role under low honey bee viral densities.

produced less modular landscape networks after accounting for pollinator species number (network size). Additionally, niche overlap with honey bees increased with agricultural intensification (M9, $\beta=0.36\pm0.14$, [0.08–0.63]).

While viral load was uncorrelated with land use for all viruses (M2, M5), urbanisation modulated BQCV prevalence in honey bees and wild pollinators. Across Europe, greater urban cover was consistently associated with higher BQCV prevalence in the primary honey bee host (M1a, $\beta=0.49\pm0.24$, [0.01–0.96], Figure 4a). Conversely, BQCV prevalence tended overall to decrease in wild pollinators along the gradient of urbanisation (M4a, $\beta=-0.54\pm0.28$, [−1.11–0.01], Figure 4b–d), albeit with some idiosyncrasy among geographic regions for bumble bees and solitary bees (Figure 4b,c). The direct effect of urbanisation, after conditioning on all other factors, remained similar (M3a, $\beta=-0.71\pm0.26$, [−1.24 to −0.21]). Land use did not influence DWV-A or DWV-B prevalence for honey bees (M1b,c) or wild pollinators (M5b,c).

3.5 | Floral Resources and Viral Loads in Wild Pollinators

Higher nectar abundance was linked with higher viral prevalence for BQCV (M3a, 0.32 ± 0.13 [0.08–0.58]) and DWV-A (M3b, 0.77 ± 0.26 [0.27–1.30], Figure 5a), while DWV-B prevalence increased with increasing nectar diversity (M3c, 0.40 ± 0.15 [0.11–0.69], Figure 5b). Nectar abundance had opposite effects on viral loads, increasing it for BQCV (M13a, 0.36 ± 0.07 [0.22–0.49]), while decreasing it for both DWV genotypes (DWV-A: M13b, -0.34 ± 0.15 [−0.64 to −0.05]; DWV-B: M13c, -0.16 ± 0.06 [−0.27 to −0.04], Figure 5c). Nectar diversity increased viral loads for BQCV (M13a, 0.33 ± 0.07 [0.19–0.47]) and DWV-B (M13c, 0.43 ± 0.05 [0.33–0.54], Figure 5d). Conditioning on pollinator abundance to account for increased pollinator densities in nutrient-rich landscapes did not change these results (M14a-c).

3.6 | Co-Occurrence of Viruses

The presence of both DWV genotypes in a host positively affected the prevalence of BQCV in wild pollinators (M16a, $\beta_{\text{DWV-A}}=0.59\pm0.25$, [0.10–1.08]; $\beta_{\text{DWV-B}}=0.90\pm0.15$, [0.62–1.19]) and *vice versa* (M16b, BQCV on DWV-A: 0.52 ± 0.26, [0.01–1.04]; M16c, BQCV on DWV-B: 0.88 ± 0.15, [0.59–1.17]). Contrastingly, DWV-A presence was negatively related to DWV-B presence (-0.73 ± 0.24, [−1.19 to −0.25]) and *vice versa* (M16c: -1.23 ± 0.22, [−1.67 to −0.81]). All other coefficients in the direct effects model (M3a-c) remained similar when co-occurrence was included as an explanatory variable (M16a-c).

4 | Discussion

4.1 | Viral Prevalence in Flower-Pollinator Networks

DWV-A, DWV-B and BQCV are abundant viruses that we detected over a large taxonomic range of pollinators. We showed honey bee viral density was the predominant predictor of

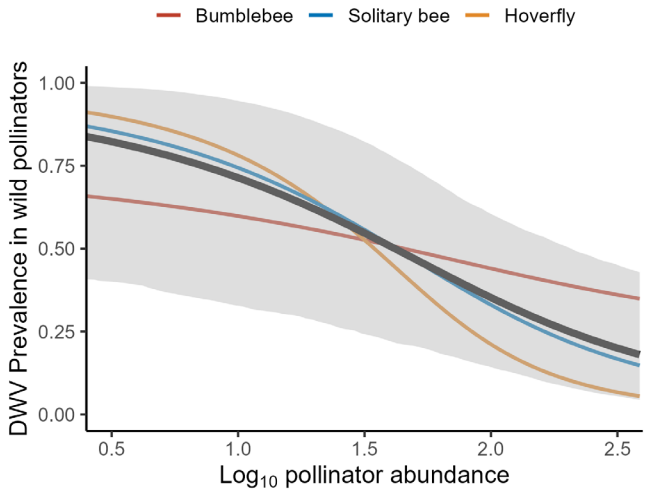

FIGURE 3 | High pollinator abundance reduces DWV-B prevalence in wild pollinators. All wild pollinator groups showed a strong relative response, which suggests that secondary hosts dilute virus and impede viral transmission, although hoverflies and solitary bees showed the strongest response. Coloured lines show the trends per pollinator group, while the thick black line shows the overall effect. Grey shading indicates 95% credible intervals.

BQCV and DWV prevalence in wild pollinators, corroborating earlier findings that honey bees are the main drivers of viral prevalence of these viruses in wild pollinators (Brettell et al. 2020; Fürst et al. 2014; McMahon et al. 2015; Pfeiffer et al. 2024; Piot et al. 2022). Our sensitivity analysis showed, however, that honey bee viral loads of BQCV and DWV-B (and viral prevalence for DWV-A), not honey bee densities, underpinned the pressure on wild pollinators from honey bee viral density. This indicates that hive management improvements to control pathogens, rather than managing population densities of honey bees per se, provide the best route to managing risks of transmitting honeybee-associated viruses to wild insects.

While honey bee viral density mainly drives viral prevalence, greater honey bee densities may drive floral resource competition and niche overlap (Mallinger et al. 2017). This represents an ecological mechanism that increases the chance of interspecific contacts and overall viral pressure from the reservoir host on the wild pollinator community. We found that overlap in flower use is an important route for BQCV transmission from managed honey bees to wild pollinators across Europe, generalising a recent finding from a single region (Maurer et al. 2024). We further reveal, in contrast to Maurer et al. (2024), that shared floral resource use produces a lesser risk for transmission of both DWV genotypes, compared to BQCV, which only becomes important under high honeybee viral densities. This might be explained by the overall higher BQCV prevalence than DWV in honey bees, making floral niche overlap a consistent viral exchange mechanism, regardless of viral densities. Honey bees are considered the reservoir host for DWV-B and transmission among secondary hosts or spillback to honey bees is rare under experimental conditions (Tehel et al. 2022), although there is other evidence that wild pollinators–particularly bumblebees–may also spread the virus (Burnham et al. 2021; Streicher et al. 2024). However, unlike other viruses, the increased virulence of DWV-B in honey bees driven by varroa mite parasitism (Vanbergen et al. 2018) may elevate the pressure of honey bee DWV-B exchange to wild pollinators (Manley et al. 2020). Additionally, the difference in viral prevalence and load between honey bees and wild pollinators was smaller for DWV-A and DWV-B than for BQCV. This might suggest a greater role of secondary hosts in onward DWV transmission and reduced role of floral resource overlap with honey bees, than for BQCV. However, because DWV-B transmission was also diluted by greater wild pollinator abundance, it seems that onward transmission among non-honey bee hosts is much less likely than from honey bees to wild pollinators. Dilution effects are common in nature (Keesing and Ostfeld 2021) including in species-rich pollinator assemblages (Cohen et al. 2022; Fearon and Tibbetts 2021). Here, however, wild pollinator abundance and not species richness drove down DWV-B prevalence, suggesting that most wild pollinators are suboptimal DWV hosts. Furthermore, detection of viral presence does not necessarily imply viral replication (Doublet et al. 2025), supporting the observed dilution effect.

While modularity does not automatically imply reduced pathogen transmission, fragmented networks with cohesive modules can lower the risk (Sah et al. 2017). BQCV was less prevalent in modular networks due to reduced niche overlap with honey bees, but after conditioning on niche overlap, BQCV and DWV-B prevalence both increased in modular, more connected networks. This contrasts with Figueroa et al. (2020), who showed in well-connected eukaryotic pathogen networks a stronger dilution effect than seen here with RNA viruses, probably due to the more restricted host range of those eukaryote pathogens.

4.2 | Viral Transmission in Urban and Agricultural Areas

Viral prevalence may be dictated by land use effects on the composition of habitat, floral resources, and the pool of foraging potential hosts (Proesmans et al. 2021). Here we found a consistent effect of urban land use increasing and decreasing BQCV prevalence in honey bees and wild pollinators, respectively.

We found that urbanisation limited BQCV prevalence in wild pollinators, possibly due to reduced network modularity (which indirectly has the potential to increase niche overlap), despite the near ubiquitous BQCV presence in honey bees, making transmission probable in all landscapes (BQCV presence in honey bees was 90.2%–98.5% in landscapes with 0% and 100% urban land use cover, respectively). Conditioning on measured environmental variables did not change the effect size of urbanisation on BQCV prevalence. It is probable that the reduced viral prevalence in wild pollinators in urban areas is driven by spatial features of urban landscapes that govern pollinator foraging on spatially heterogeneous floral resources (Baldock 2020) or urban microclimate effects (Piot et al. 2022).

Agricultural land cover reduced network modularity and produced higher niche overlap with honey bees, yet we did not

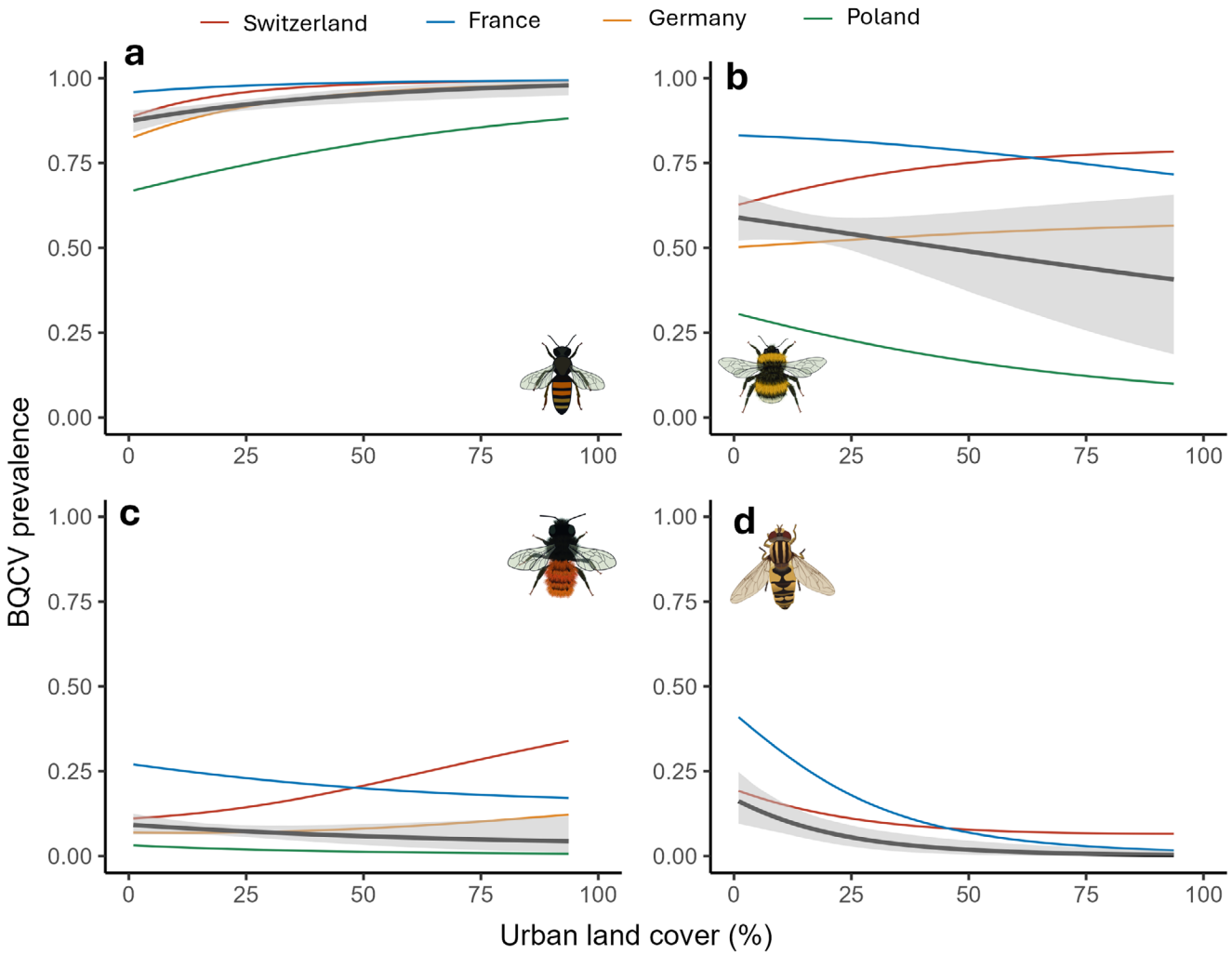

FIGURE 4 | The overall effect of urban land cover on BQCV prevalence in wild and managed pollinators: Honey bees (a) show an increase in viral prevalence as a response to urbanisation (mean: 0.54, 95% CI: [0.21–0.88]), while bumblebees (−0.21 [−0.56–0.13]) (b), solitary bees (−0.26 [−0.67–0.15]) (c) and hoverflies (−1.27 [−2.02 to −0.59]) (d) show a decrease. Coloured lines show the trends per country, while the thick black line shows the overall effect. Grey shading indicates 95% credible intervals. Icon credits: CC-BY-NC Jose Luis Ordóñez & Ignasi Bartomeus: bee images. kgena1: hoverfly image.

find a clear effect on prevalence of any virus. The quality of floral resources in agricultural landscapes can be variable and particularly depends on the presence, area and identity of mass-flowering crops, which can either concentrate or dilute pollinator visitations and hence affect pathogen transfer (Tuerlings et al. 2022). Further analysis on how pollinator-pathogen dynamics are affected by the nutritional or medicinal quality of mass-flowering crops and wild plants in different landscape configurations is needed before drawing general conclusions on the effect of agricultural intensification on viral transmission.

Different apiary densities or apicultural practices between urban and rural areas may affect honey bee viral prevalence, as BQCV is typically associated with high apiary density, while DWV is more linked with varroa mite prevalence (Pfeiffer and Crowder 2022). The diverse responses of wild pollinators and honey bees to viruses corroborate the idea that viral prevalence in honey bees mainly depends on apicultural management (Bartlett et al. 2021), while wild pollinator viral prevalence is driven largely by viral load in honey bees and environmental factors.

Land-use driven heterogeneity in floral resources is expected to affect pollinator nutrition and—after transmission—viral load in wild pollinators (Parreño et al. 2022; Vanbergen and the Insect Pollinators Initiative 2013). Viral prevalence was consistently higher in landscapes with abundant and diverse nectar sources, while the effect of nectar abundance on viral load was positive for BQCV and negative for DWV. Landscapes providing abundant and diverse floral resources allow dynamic foraging of pollinators to optimise individual physiology (Vaudo et al. 2024). This may include adjustment in nutrient intake that supports immune responses to viral infections or secondary metabolites with antiviral properties (Palmer-Young et al. 2017; Parreño et al. 2022). An increased nectar supply may have beneficial effects on pollinators, either allowing them to restrain viral replication (DWV) or alternatively to tolerate virus and survive despite higher viral loads (BQCV). The outcome is likely highly context dependent,

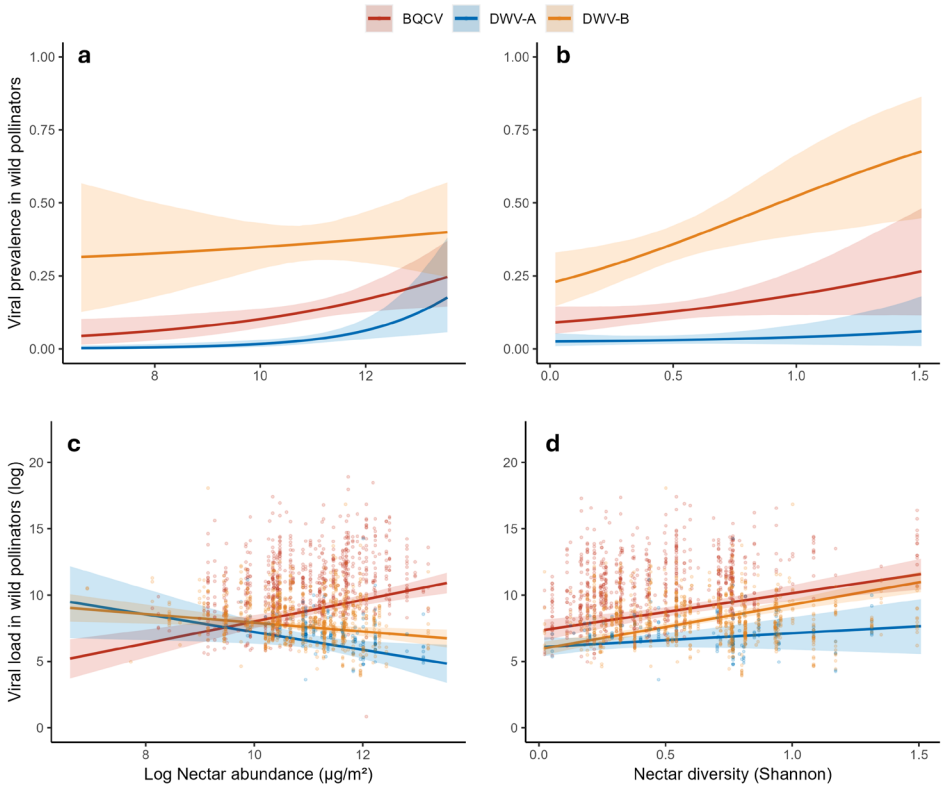

FIGURE 5 | Marginal effect of nectar abundance and diversity on viral prevalence and load: Nectar abundance, measured as the average amount of sugar in the landscape (in µg/m^2) is positively correlated with BQCV and DWV-A prevalence (a), while BQCV and DWV-B prevalence increased with nectar diversity (b). Viral load increased with nectar abundance for BQCV, while both DWV genotypes showed an opposite effect (c). BQCV and DWV-B load responded positively to nectar diversity (d). Points represent the partial residuals after subtracting the modelled effect of all other explanatory variables. The lines indicate the posterior mean marginal effect of nectar abundance/diversity, while shading represents the 95% credible intervals.

relating to host physiology, viral variant, as well as the chemistry of available pollen and nectar resources.

4.3 | Co-Occurrence of Viruses

Bees and hoverflies simultaneously harbour a variety of pathogens (Doublet et al. 2015, 2025; Schoonvaere et al. 2018; Tiritelli et al. 2024), and patterns of co-occurrence can be driven by various mechanisms (Figure S6). We found a strong co-occurrence of BQCV and DWV in individual pollinator hosts. One hypothesis is that this robust viral species co-occurrence may indicate a viral infection facilitating a coinfection via suppressive effects on host physiology, or both viruses acting synergistically—however, we have no data of within-host viral replication to test such a hypothesis. Alternatively, viral co-occurrence may be driven by a common environmental driver (e.g., local apicultural practices driving honey bee colony health and viral loads). Within a viral species, both DWV genotypes were strongly negatively correlated, which points to competitive exclusion. While DWV-A was historically the more abundant variant, DWV-B, which is more virulent in honeybees through mite-vectored transmission (McMahon et al. 2016), has in recent years become dominant, implying displacement of DWV-A (Paxton et al. 2022). The physiological effects of DWV on wild pollinators, and the consequences of the emergence of DWV-B for health and fitness of wild pollinators remain to be studied.

5 | Conclusion

We found that managed honey bees are the main reservoir host and driver of viral (BQCV, DWV) prevalence in wild pollinators. However, the risk of interspecific transmission is modulated by effects of land use, network architecture (niche overlap and modularity), and the taxonomy of the host. Differences in prevalence between secondary wild pollinator hosts were observable, with BQCV being predominantly confined to bumblebees and honey bees, while DWV was generally present across pollinator communities, despite greater secondary host abundances diluting DWV-B transmission. While the effects of honey bee-associated viruses on the health and fitness of wild pollinators remain a knowledge gap, reducing viral load in honey bees through maintaining colony health practices currently seems the best means of reducing viral prevalence in wild pollinators.

Author Contributions

Adam J. Vanbergen, Matthias Albrecht, Robert J. Paxton, Oliver Schweiger, Josef Settele, Hajnalka Szentgyörgyi, Peter Neumann, Anna Gajda conceived and designed the research project. Data collection in the field was carried out by Willem Proesmans, Emeline Felten, Emilien Laurent, Nathan Cyrille, Adam J. Vanbergen, Jonna M. Heuschele, Corina Maurer, Hajnalka Szentgyörgyi, Aleksandra Żmuda, Christophe Dominik, Yicong Liu. Insect identification was done by Willem Proesmans, Aleksandra Żmuda, commercial experts or

barcoding services. Molecular protocols and work were done by Anne Dalmon, Aleksandra Żmuda, Virginie Diévart, Anna Gajda, Karima Bassit, Peter Neumann, Robert J. Paxton, Alexandria Schauer, Eckart Stolle, Orlando Yañez. The modelling analyses and the writing effort were led by Willem Proesmans. All authors provided critical input in writing and analyses. All authors approved the final version.

Acknowledgements

We are grateful to field assistants Cassandre Vidal, Audrey Labonté, Augustin Chenut, Nicolas Henon and Chantal Ducourtieux for their help with the fieldwork in France, and Lea Bona and Stefanie Bossart in Switzerland. We thank the landowners for granting permission to carry out the research on their properties. We thank Frank Creutzburg for the identification of the bees collected in Germany as well as Anja Manigk and Henriette Kühn for assistance with their viral analysis. This work was carried out by project VOODOO (Viral eco-evolutionary dynamics of wild and domestic pollinators under global change www.voodoo-project.eu), funded under the joint call of BiodivERsA 2018-2019 'BIODIVERSITY AND ITS INFLUENCE ON ANIMAL, HUMAN, AND PLANT HEALTH' [Swiss National Science Foundation-SNSF 31BD30186532/1 (Peter Neumann and Matthias Albrecht); France-ANR-19-EBI3-0006 (Adam J. Vanbergen); German Research Foundation-DFG PA 623/10-1, PA 623/12-1 (Robert J. Paxton); Federal Ministry of Education and Research-BMBF and German Aerospace Research and Technology Centre no. 01LC1905A (Oliver Schweiger); and Poland-NCN UMO-2019/32/Z/NZ8/00006 (Hajnalka Szentgyörgyi)]. Willem Proesmans, Adam J. Vanbergen, Oliver Schweiger, Matthias Albrecht also received co-funding support from the European Union Horizon 2020 project Safeguard grant no. GA 101003476. Willem Proesmans also received funding from the European Union Horizon 2020 research and innovation programme under the C2W Marie Skłodowska Curie grant agreement No 101034383.

Funding

This work was supported by H2020 Marie Skłodowska-Curie Actions, grant agreement No 101034383. Horizon 2020 Framework Programme, GA 101003476. Biodiversa+, ANR-19-EBI3-0006, NCN UMO-2019/32/Z/NZ8/00006.

Data Availability Statement

Raw data and code from this study is openly available on Dryad at https://doi.org/10.5061/dryad.3bk3j9kww.

Peer Review

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/ele.70309.

References

Arif, S., and M. A. MacNeil. 2023. "Applying the Structural Causal Model Framework for Observational Causal Inference in Ecology." *Ecological Monographs* 93: e1554.

Baldock, K. C. R., M. A. Goddard, D. M. Hicks, et al. 2015. "Where Is the UK'S Pollinator Biodiversity? The Importance of Urban Areas for Flower-Visiting Insects." *Proceedings of the Royal Society B* 282: 20142849.

Baldock, K. C. R. 2020. "Opportunities and Threats for Pollinator Conservation in Global Towns and Cities." *Current Opinion in Insect Science* 38: 63–71.

Bartlett, L. J., M. Boots, B. J. Brosi, et al. 2021. "Persistent Effects of Management History on Honeybee Colony Virus Abundances." *Journal of Invertebrate Pathology* 179: 107520.

Baude, M., W. E. Kunin, and J. Memmott. 2015. "Nectar Sugar Values of Common British Plant Species [AgriLand]."

Becker, D. J., A. D. Washburne, C. L. Faust, et al. 2019. "Dynamic and Integrative Approaches to Understanding Pathogen Spillover." *Philosophical Transactions of the Royal Society, B: Biological Sciences* 374: 20190014.

Bodden, J. M., J. A. Hazlehurst, and E. E. W. Rankin. 2019. "Floral Traits Predict Frequency of Defecation on Flowers by Foraging Bumble Bees." *Journal of Insect Science* 19: 1–3.

Brettell, L. E., M. Riegler, C. O'Brien, and J. M. Cook. 2020. "Occurrence of Honey Bee-Associated Pathogens in Varroa-Free Pollinator Communities." *Journal of Invertebrate Pathology* 171: 107344.

Burnham, P. A., S. A. Alger, B. Case, H. Boncristiani, L. Hébert-Dufresne, and A. K. Brody. 2021. "Flowers as Dirty Doorknobs: Deformed Wing Virus Transmitted Between Apis Mellifera and *Bombus Impatiens* Through Shared Flowers." *Journal of Applied Ecology* 58: 2065–2074.

Carpenter, B., A. Gelman, M. Hoffman, et al. 2016. "Stan: A Probabilistic Programming Language." *Journal of Statistical Software* 76: 1–32.

Cohen, H., L. C. Ponisio, K. A. Russell, S. M. Philpott, and Q. S. McFrederick. 2022. "Floral Resources Shape Parasite and Pathogen Dynamics in Bees Facing Urbanization." *Molecular Ecology* 31: 2157–2171.

Cohen, H., G. P. Smith, H. Sardiñas, et al. 2021. "Mass-Flowering Monoculture Attracts Bees, Amplifying Parasite Prevalence." *Proceedings of the Royal Society B: Biological Sciences* 288: 20211369.

Davies, C. E., D. Moss, and M. O. Hill. 2004. "EUNIS Habitat Classification Revised 2004." European Environment Agency European Topic Centre on Nature Protection and Biodiversity.

de Miranda, J. R., I. Meeus, O. Yañez, et al. 2021. "Protocols for Assessing the Distribution of Pathogens in Individual Hymenopteran Pollinators." *Nature Protocol Exchange*. https://doi.org/10.21203/rs.3.pex-1453/v1.

Dormann, C. F., J. Fründ, N. Blüthgen, and B. Gruber. 2009. "Indices, Graphs and Null Models: Analyzing Bipartite Ecological Networks." *Open Ecology Journal* 2: 7–24.

Doublet, V., T. D. Doyle, C. Carvell, M. J. F. Brown, and L. Wilfert. 2025. "Host Ecology and Phylogeny Shape the Temporal Dynamics of Social Bee Viromes." *Nature Communications* 16: 2207.

Doublet, V., M. E. Natsopoulou, L. Zschiesche, and R. J. Paxton. 2015. "Within-Host Competition Among the Honey Bees Pathogens *Nosema Ceranae* and Deformed Wing Virus Is Asymmetric and to the Disadvantage of the Virus." *Journal of Invertebrate Pathology* 124: 31–34.

Evans, J. C., D. J. Hodgson, N. J. Boogert, and M. J. Silk. 2021. "Group Size and Modularity Interact to Shape the Spread of Infection and Information Through Animal Societies." *Behavioral Ecology and Sociobiology* 75: 163.

Fearon, M. L., and E. A. Tibbetts. 2021. "Pollinator Community Species Richness Dilutes Prevalence of Multiple Viruses Within Multiple Host Species." *Ecology* 102: e03305.

Fearon, M. L., C. L. Wood, and E. A. Tibbetts. 2023. "Habitat Quality Influences Pollinator Pathogen Prevalence Through Both Habitat–Disease and Biodiversity–Disease Pathways." *Ecology* 104: e3933.

Figueroa, L. L., M. Blinder, C. Grincavitch, et al. 2019. "Bee Pathogen Transmission Dynamics: Deposition, Persistence and Acquisition on Flowers." *Proceedings of the Royal Society B: Biological Sciences* 286: 20190603.

Figueroa, L. L., H. Grab, W. H. Ng, et al. 2020. "Landscape Simplification Shapes Pathogen Prevalence in Plant-Pollinator Networks." *Ecology Letters* 23: 1212–1222.

Filipiak, M., A. Walczynska, B. Denisow, T. Petanidou, and E. Ziołkowska. 2022. "Phenology and Production of Pollen, Nectar, and Sugar in 1612 Plant Species From Various Environments." *Ecology* 103: e3705.

Franks, D. W., G. D. Ruxton, and T. Sherratt. 2025. "Ecology Needs a Causal Overhaul." *Biological Reviews* 100: 1950–1969.

Fürst, M. A., D. P. Mcmahon, J. L. Osborne, R. J. Paxton, and M. J. F. Brown. 2014. "Disease Associations Between Honeybees and Bumblebees as a Threat to Wild Pollinators." *Nature* 506: 364–366.

Gisder, S., and E. Genersch. 2017. "Viruses of Commercialized Insect Pollinators." *Journal of Invertebrate Pathology* 147: 51–59.

Grozinger, C. M., and M. L. Flenniken. 2019. "Bee Viruses: Ecology, Pathogenicity, and Impacts." *Annual Review of Entomology* 64: 205–226.

Horn, H. S. 1966. "Measurement of "Overlap" in Comparative Ecological Studies." *American Naturalist* 100: 419–424.

IPBES. 2016. "The Assessment Report of the Intergovernmental Science-Policy Platform on Biodiversity and Ecosystem Services on Pollinators, Pollination and Food Production." *Secretariat of the Intergovernmental Science-Policy Platform on Biodiversity and Ecosystem Services*, edited by S. G. Potts, V. L. Imperatriz-Fonseca, and H. T. Ngo, 556.

Keesing, F., and R. S. Ostfeld. 2021. "Dilution Effects in Disease Ecology." *Ecology Letters* 24: 2490–2505.

Lázaro, A., A. Müller, A. W. Ebmer, et al. 2021. "Impacts of Beekeeping on Wild Bee Diversity and Pollination Networks in the Aegean Archipelago." *Ecography* 44: 1353–1365.

Mahon, M. B., A. Sack, O. A. Aleuy, et al. 2024. "A Meta-Analysis on Global Change Drivers and the Risk of Infectious Disease." *Nature* 629: 830–836.

Mallinger, R. E., H. R. Gaines-Day, and C. Gratton. 2017. "Do Managed Bees Have Negative Effects on Wild Bees?: A Systematic Review of the Literature." *PLoS One* 12: e0189268.

Manley, R., V. Doublet, O. N. Wright, et al. 2023. "Conservation Measures or Hotspots of Disease Transmission? Agri-Environment Schemes Can Reduce Disease Prevalence in Pollinator Communities." *Philosophical Transactions of the Royal Society, B: Biological Sciences* 378: 20220004.

Manley, R., B. Temperton, M. Boots, and L. Wilfert. 2020. "Contrasting Impacts of a Novel Specialist Vector on Multihost Viral Pathogen Epidemiology in Wild and Managed Bees." *Molecular Ecology* 29: 380–393.

Manley, R., B. Temperton, T. Doyle, et al. 2019. "Knock-On Community Impacts of a Novel Vector: Spillover of Emerging DWV-B From *Varroa*-Infested Honeybees to Wild Bumblebees." *Ecology Letters* 22: 1306–1315.

Maurer, C., A. Schauer, O. Yañez, et al. 2024. "Species Traits, Landscape Quality and Floral Resource Overlap With Honeybees Determine Virus Transmission in Plant–Pollinator Networks." *Nature Ecology & Evolution* 8: 2239–2251.

McElreath, R. 2020. *Statistical Rethinking: A Bayesian Course With Examples in R and Stan*. 2nd ed. Chapman and Hall/CRC.

McMahon, D. P., M. A. Fürst, J. Caspar, P. Theodorou, M. J. F. Brown, and R. J. Paxton. 2015. "A Sting in the Spit: Widespread Cross-Infection of Multiple RNA Viruses Across Wild and Managed Bees." *Journal of Animal Ecology* 84: 615–624.

McMahon, D. P., M. E. Natsopoulou, V. Doublet, et al. 2016. "Elevated Virulence of an Emerging Viral Genotype as a Driver of Honeybee Loss." *Proceedings of the Royal Society B* 283: 20160811.

Nanetti, A., L. Bortolotti, and G. Cilia. 2021. "Pathogens Spillover From Honey Bees to Other Arthropods." *Pathogens* 10: 1044.

Newman, M. E. J. 2006. "Modularity and Community Structure in Networks." *Proceedings of the National Academy of Sciences of the United States of America* 103: 8577–8582.

Palmer-Young, E. C., B. M. Sadd, R. E. Irwin, and L. S. Adler. 2017. "Synergistic Effects of Floral Phytochemicals Against a Bumble Bee Parasite." *Ecology and Evolution* 7: 1836–1849.

Parreño, M. A., C. Alaux, J. L. Brunet, et al. 2022. "Critical Links Between Biodiversity and Health in Wild Bee Conservation." *Trends in Ecology & Evolution* 37: 309–321.

Paxton, R. J., M. O. Schäfer, F. Nazzi, et al. 2022. "Epidemiology of a Major Honey Bee Pathogen, Deformed Wing Virus: Potential Worldwide Replacement of Genotype A by Genotype B." *International Journal of Parasitology: Parasites and Wildlife* 18: 157–171.

Pfeiffer, V. W., S. Basu, and D. W. Crowder. 2024. "Patterns of Virus Coincidence Between Honey Bees and Bumble Bees in the Pacific Northwest, USA." *Apidologie* 55: 30.

Pfeiffer, V. W., and D. W. Crowder. 2022. "Factors Affecting Virus Prevalence in Honey Bees in the Pacific-Northwest, USA." *Journal of Invertebrate Pathology* 187: 107703.

Piot, N., M. Eeraerts, M. Pisman, G. Claus, I. Meeus, and G. Smagghe. 2021. "More Is Less: Mass-Flowering Fruit Tree Crops Dilute Parasite Transmission Between Bees." *International Journal for Parasitology* 51: 777–785.

Piot, N., O. Schweiger, I. Meeus, et al. 2022. "Honey Bees and Climate Explain Viral Prevalence in Wild Bee Communities on a Continental Scale." *Scientific Reports* 12: 1904.

Proesmans, W., M. Albrecht, A. Gajda, et al. 2021. "Pathways for Novel Epidemiology: Plant-Pollinator-Pathogen Networks and Global Change." *Trends in Ecology & Evolution* 36: 623–636.

Proesmans, W., E. Felten, E. Laurent, et al. 2024. "Urbanisation and Agricultural Intensification Modulate Plant–Pollinator Network Structure and Robustness." *Functional Ecology* 38: 628–641.

R Core Team. 2024. "R: A Language and Environment for Statistical Computing." R Foundation for Statistical Computing, Vienna, Austria.

Redhead, J. W., B. A. Woodcock, M. J. O. Pocock, R. F. Pywell, A. J. Vanbergen, and T. H. Oliver. 2018. "Potential Landscape-Scale Pollinator Networks Across Great Britain: Structure, Stability and Influence of Agricultural Land Cover." *Ecology Letters* 21: 1821–1832.

Sah, P., S. T. Leu, P. C. Cross, P. J. Hudson, and S. Bansal. 2017. "Unraveling the Disease Consequences and Mechanisms of Modular Structure in Animal Social Networks." *Proceedings of the National Academy of Sciences* 114: 4165–4170.

Schoonvaere, K., G. Smagghe, F. Francis, and D. C. de Graaf. 2018. "Study of the Metatranscriptome of Eight Social and Solitary Wild Bee Species Reveals Novel Viruses and Bee Parasites." *Frontiers in Microbiology* 9: 177.

Sponsler, D., A. Iverson, and I. Steffan-Dewenter. 2023. "Pollinator Competition and the Structure of Floral Resources." *Ecography* 2023: e06651.

Streicher, T., P. Brinker, S. Tragust, and R. J. Paxton. 2024. "Host Barriers Limit Viral Spread in a Spillover Host: A Study of Deformed Wing Virus in the Bumblebee *Bombus terrestris*." *Viruses* 16: 607.

Tehel, A., T. Streicher, S. Tragust, and R. J. Paxton. 2022. "Experimental Cross Species Transmission of a Major Viral Pathogen in Bees Is Predominantly From Honeybees to Bumblebees." *Proceedings of the Royal Society B: Biological Sciences* 289: 20212255.

Tiritelli, R., S. Flaminio, L. Zavatta, et al. 2024. "Ecological and Social Factors Influence Interspecific Pathogens Occurrence Among Bees." *Scientific Reports* 14: 5136.

Tuerlings, T., L. Buydens, G. Smagghe, and N. Piot. 2022. "The Impact of Mass-Flowering Crops on Bee Pathogen Dynamics." *International Journal of Parasitology: Parasites and Wildlife* 18: 135–147.

Vanbergen, A. J., A. Espíndola, and M. A. Aizen. 2018. "Risks to Pollinators and Pollination From Invasive Alien Species." *Nature Ecology & Evolution* 2: 16–25.

Vanbergen, A. J., and the Insect Pollinators Initiative. 2013. "Threats to an Ecosystem Service: Pressures on Pollinators." *Frontiers in Ecology and the Environment* 11: 251–259.

Vaudo, A. D., L. A. Dyer, and A. S. Leonard. 2024. "Pollen Nutrition Structures Bee and Plant Community Interactions." *Proceedings of the National Academy of Sciences of the United States of America* 121: e2317228120.

Vehtari, A., A. Gelman, D. Simpson, B. Carpenter, and P. C. Bürkner. 2021. "Rank-Normalization, Folding, and Localization: An Improved R for Assessing Convergence of MCMC (With Discussion)." *Bayesian Analysis* 16: 667–718.

Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Appendix S1:** ele70309-sup-0001-AppendixS1.docx. **Figure S1:** Overview of the geographical composition of the study. The study was replicated in four regions in France, Germany, Switzerland and Poland (a), indicated by a grey diamond. Within each region, 12 landscapes were analysed within a 1000 m radius around the centroid, as exemplified by the French landscapes (b). Within each region, four landscapes consisted of a rural mosaic of farmland and semi-natural habitat (c), four consisted of intensively managed agricultural fields (d) and four consisted of an urbanised habitat (e). Yellow: cropland, dark green: semi-natural habitat, light green: parks and gardens, red: built up areas, blue: open water, purple: other. European base map sourced from www.freevectormaps.com, base map for the French region sourced from OpenStreetMap (www.openstreetmap.org). **Figure S2:** DAG on causal drivers behind the prevalence of DWV and BQCV in wild pollinators. We assumed similar drivers for both viruses. Season and landscape are overarching variables directly affecting honey bees, the wild pollinator community and the plant community. A direct effect on network metrics was also retained as the spatial distribution of resources, which was not explicitly modelled, may be affected by phenology and landscape. As honey bee abundance, honey bee viral prevalence and honey bee viral load are strongly interacting variables (see methods section), we created a composite variable 'Viral density A. mel.', which in most models was used instead of the three original honey bee variables. This variable is expected to directly affect viral prevalence through a (not shown) interaction with honey bee niche overlap. The plant and pollinator community (species diversity, nectar abundance and diversity) were expected to affect the viral prevalence indirectly through changes in the plant-pollinator network, in particular the network modularity and connectance, and the niche overlap with honey bees, which serve as the primary host of the studied viruses. In addition, both nectar abundance and diversity show a direct effect on viral prevalence through (not explicitly observed) health effects driven by food quality and quantity. Pollinator abundance is also expected to directly affect viral prevalence as viruses can be spread faster under high population density, or conversely, virus prevalence can be reduced if incompatible or suboptimal hosts are dominant in the pollinator community. *The variables 'urbanisation' and 'agricultural intensification' are grouped here as 'Landscape' to improve readability, as they are both hypothesized to affect the same variables. **Figure S3:** DAG on the viral load of BQCV and DWV in wild pollinators. While viral prevalence focused on variables modulating infection risk (e.g., network metrics, niche overlap with honey bees), viral load is affected by the intrinsic susceptibility to the virus (Taxonomy) and by the quantity and quality of available nutrients (nectar diversity, nectar abundance). Pollinator abundance is also added in the model as a measure of resource competition, which is expected to negatively impact pollinator health. *The variables 'urbanisation' and 'agricultural intensification' are grouped here as 'Landscape' to improve readability, as they are both hypothesized to affect the same variables. **Figure S4:** Modelled honeybee viral density per site and period. Grey lines divide early, mid and late season. Black dots give the observed honeybee viral density based on number of honeybees and observed viral prevalence and load, which is only available for sites where positive honeybees were caught. Red dots show the modelled honeybee viral density (posterior mean), based on models 1 (for prevalence) and 2 (for load), and the formula for honeybee viral density (HBVD = prevalence × [log(honeybee abundance + 1) + log(viral load)]). Grey bars give the 95% credible interval. In some sites only one or two honeybees were caught, which can strongly skew the measured viral density (e.g., when only one honeybee is caught, prevalence is always either 0 or 1, which does not reflect the reality), as well as potentially skewing the viral load, which shows considerable within-site variance. This explains why measured honeybee density strongly differs from modelled honeybee viral density. The modelled values were used as explanatory variables for honeybee viral density in further models, as these provide more reliable estimates than the observed honeybee viral density for reasons explained above. **Figure S5:** Overall phenological effects on viral prevalence and load: Posterior estimates of viral prevalence and load of BQCV, DWV-A and DWV-B in wild pollinators (honeybees excluded). BQCV (a) prevalence peaked in mid-season (late May to late June), while DWV-A (b) prevalence declined in late season (mid July to mid August) and DWV-B (c) prevalence was lowest in mid-season. Viral loads (d–f) showed a similar trend as the prevalence of the respective viruses except for DWV-A (e), which was lowest during early season (mid April to mid May). Values were calculated based on models 4 (prevalence) and 5 (load) under average values for urbanisation and agricultural intensification, and averaged over the effects of wild pollinator group, country and site. The bars display the modelled posterior mean viral prevalence and load. Error bars indicate 95% credible intervals, while different letters indicate a 95% probability that groups differ. Raw data points are given for viral load, but not for viral prevalence, which is Bernoulli distributed (0 or 1). Diamonds indicate the mean observed viral load per country. **Figure S6:** Potential causal links between co-occurrence of BQCV and DWV: Prevalence of BQCV is very strongly positively correlated with DWV prevalence in individual bees. While current knowledge cannot assume one single reason for this, it may be possible that BQCV is positively affected by the presence of DWV (a) or conversely that BQCV drives presence of DWV (b), that both viruses are affected by similar environmental drivers (e.g., practices that affect colony health of the primary host), which leads to a noncausal correlation between the two (c), or that both synergistically affect each other, even though this synergy should not necessarily be symmetrical (d). **Table S1:** qPCR primers and synthetic target sequences. **Table S2:** Details on protocols, reagents and machines used for viral analysis over the involved labs. **Table S3:** List with analysed species including positives for BQCV, DWV-A and DWV-B and total number of specimens tested. **Table S4:** Variables used in the statistical analysis, including the abbreviation used in the following model output tables. Variables were measured at the level of the individual insect (Individual), at the level of a single species within the plant-pollinator network (Species in network), at landscape-level during one specific visit (Network) or at the overall site level (Landscape). **Table S5:** List of models. We provide the research questions that are answered and describe the models used for them. Variables are explained in Table S4. Model outputs are displayed in Tables S6–S49. Ab = pollinator abundance, AI = agricultural intensification, Con = connectance, HBAb = honeybee abundance, HBN = honeybee niche overlap, HBVD = honeybee viral density, Mod = modularity, NecAb = nectar abundance, NecDiv = nectar diversity, Npol = pollinator species richness, Ph = phenology, Tax = taxonomy, U = urbanisation. **Table S6:** Effect of land use and phenology on the BQCV prevalence in honey bees (Model 1a). **Table S7:** Effect of land use and phenology on the DWV-A prevalence in honey bees (Model 1b). **Table S8:** Effect of land use and phenology on the DWV-B prevalence in honey bees (Model 1c). **Table S9:** Effect of land use and phenology on the BQCV load in honey bees (Model 2a). **Table S10:** Effect of land use and phenology on the DWV-A load in honey bees (Model 2b). **Table S11:** Effect of land use and phenology on the DWV-B load in honey bees (model 2c). **Table S12:** Direct effects of environmental variables on BQCV prevalence in wild pollinators (Model 3a). **Table S13:** Direct effects of environmental variables on DWV-A prevalence in wild pollinators (Model 3b). **Table S14:** Direct effects of environmental variables on DWV-B prevalence in wild pollinators (Model 3c). **Table S15:** Overall effects of land use and phenology on BQCV prevalence in wild pollinators, conditioned on taxonomy (Model 4a). **Table S16:** Overall effects of land use and phenology on DWV-A prevalence in wild pollinators, conditioned on taxonomy (Model 4b). **Table S17:** Overall effects of land use and phenology on DWV-B prevalence in wild pollinators, conditioned on taxonomy

(Model 4c). **Table S18:** Overall effects of land use and phenology on BQCV load in wild pollinators, conditioned on taxonomy (model 5a). **Table S19:** Overall effects of land use and phenology on DWV-A load in pollinators, conditioned on taxonomy (Model 5b). **Table S20:** Overall effects of land use and phenology on DWV-B load in pollinators, conditioned on taxonomy (Model 5c). **Table S21:** Overall effect of network metrics (modularity and connectance) on BQCV prevalence in wild pollinators (Model 6a). **Table S22:** Overall effect of network metrics (modularity and connectance) on DWV-A prevalence in wild pollinators (Model 6b). **Table S23:** Overall effect of network metrics (modularity and connectance) on DWV-B prevalence in wild pollinators (Model 6c). **Table S24:** Overall effect of taxonomy on BQCV prevalence (Model 7a). **Table S25:** Overall effect of taxonomy on DWV-A prevalence (Model 7b). **Table S26:** Overall effect of taxonomy on DWV-B prevalence (Model 7c). **Table S27:** Overall effect of taxonomy on BQCV load (Model 8a). **Table S28:** Overall effect of taxonomy on DWV-A load (Model 8b). **Table S29:** Overall effect of taxonomy on DWV-B load (Model 8c). **Table S30:** Overall effects of land use, phenology and taxonomy on niche overlap with honey bees (Model 9). **Table S31:** Effects of network metrics (modularity and connectance) on niche overlap with honey bees (Model 10). **Table S32:** Effect of honey bee abundance on niche overlap with honey bees (Model 11). **Table S33:** Effect of land use and phenology on network modularity, conditioned on pollinator species number (Model 12a). **Table S34:** Effect of land use and phenology on network connectance, conditioned on pollinator species number (Model 12b). **Table S35:** Overall effect of nectar abundance and diversity on BQCV load in wild pollinators (Model 13a). **Table S36:** Overall effect of nectar abundance and diversity on DWV-A load in wild pollinators (Model 13b). **Table S37:** Overall effect of nectar abundance and diversity on DWV-B load in wild pollinators (Model 13c). **Table S38:** Direct effect of nectar abundance and diversity on BQCV load in wild pollinators, conditioned on pollinator abundance (Model 14a). **Table S39:** Direct effect of nectar abundance and diversity on DWV-A load in wild pollinators, conditioned on pollinator abundance (Model 14b). **Table S40:** Direct effect of nectar abundance and diversity on DWV-B load in wild pollinators, conditioned on pollinator abundance (Model 14c). **Table S41:** Effect of pollinator species richness on BQCV prevalence (model 15a). **Table S42:** Effect of pollinator species richness on DWV-A prevalence (model 15b). **Table S43:** Effect of pollinator species richness on DWV-B prevalence (model 15c). **Table S44:** Effect of DWV-A and DWV-B prevalence on BQCV prevalence (model 16a). **Table S45:** Effect of BQCV and DWV-B prevalence on DWV-A prevalence (model 16b). **Table S46:** Effect of BQCV and DWV-A prevalence on DWV-B prevalence (model 16c). **Table S47:** Effect of season and land use on nectar abundance (m17a). **Table S48:** Effect of season and land use on nectar diversity (m17b). **Table S49:** Effect of urbanisation on each group of pollinators (m18). The graphs in Figure 4 are based on this model. **Appendix S2:** ele70309-sup-0002-AppendixS2.docx.