

# Agricultural land use disrupts biodiversity mediation of virus infections in wild plant populations

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## Summary

- Human alteration of natural habitats may change the processes governing species interactions in wild communities. Wild populations are increasingly impacted by agricultural intensification, yet it is unknown whether this alters biodiversity mediation of disease dynamics.
- We investigated the association between plant diversity (species richness, diversity) and infection risk (virus richness, prevalence) in populations of *Plantago lanceolata* in natural landscapes as well as those occurring at the edges of cultivated fields. Altogether, 27 *P. lanceolata* populations were surveyed for population characteristics and sampled for PCR detection of five recently characterized viruses.
- We find that plant species richness and diversity correlated negatively with virus infection prevalence. Virus species richness declined with increasing plant diversity and richness in natural populations while in agricultural edge populations species richness was moderately higher, and not associated with plant richness. This difference was not explained by changes in host richness between these two habitats, suggesting potential pathogen spill-over and increased transmission of viruses across the agro-ecological interface. Host population connectivity significantly decreased virus infection prevalence.
- We conclude that human use of landscapes may change the ecological laws by which natural communities are formed with far reaching implications for ecosystem functioning and disease.

## Introduction

Agriculture has replaced natural habitats across the world, and those natural habitats remaining are increasingly adjacent to lands subjected to agricultural practices. In addition to land use change, agriculture is responsible for increased global carbon emissions, freshwater withdrawals and fertilizer use (Foley *et al.*, 2005). Hence, it is not surprising that biodiversity has been heavily impacted by agriculture. Severe declines in plant, vertebrate and invertebrate species richness have resulted from agricultural intensification (Newbold *et al.*, 2015). Critical – although less well understood – are the effects of agriculture on the interactions (e.g. parasitism, pollination, disease vectoring) that link species to one another, and on the mechanisms that maintain biodiversity in natural populations (Tylianakis *et al.*, 2008). To date, agriculture-mediated changes in species interactions have been detected for example in collapses in native pollinator populations and an excess of honeybees spilling over to adjacent natural habitats. Together, these processes have changed plant–pollinator networks, and have jeopardized the reproduction of some wild plants (Potts *et al.*, 2010; Magrath *et al.*, 2017; Grab *et al.*, 2019). Host–pathogen interactions are often regulated by a network of interacting species – hosts, vectors and pathogens – and

little is known about how agriculture shapes the relationship among these trophic groups.

There is growing concern for how disease risks are changing in the environments surrounding agricultural areas, which are considered highly conducive for the emergence and dissemination of pathogens (McDonald & Stukenbrock, 2016). The wild and cultivated areas differ dramatically in many ways crucial for disease epidemics, with their interface being characterized by an abrupt change in plant density, species richness and nutrient status (Rogalski *et al.*, 2017). There is also evidence of nutrient-driven changes in host susceptibility to fungal and viral pathogens (Laine, 2007; Lacroix *et al.*, 2017), decrease in grassland species diversity (Harpole *et al.*, 2016), as well as changes in competitive interactions between plant species, thereby altering the composition of natural communities (Liu *et al.*, 2018). For pathogens that move across the agro-ecological interface, infection risk may increase in habitats in the proximity of cultivated areas due to the high transmission rates supported by high-density cropping systems (Papaix *et al.*, 2015; Bell & Tylianakis, 2016; Paap *et al.*, 2018). Moreover, wild plants may serve as reservoir hosts for pathogens infecting crops (Power & Mitchell, 2004).

Recently, there has been growing interest in the relationship between biodiversity and spread of infectious diseases (Liu *et al.*,

2020; Rohr *et al.*, 2020). While biodiversity is declining, epidemics of several infectious diseases have escalated worldwide threatening the health of humans, wild life and domesticated species (Keesing *et al.*, 2010; Civitello *et al.*, 2015). There are two opposing scenarios predicting the relationship between biodiversity and disease risk; biodiversity can either increase (amplification hypothesis) or decrease (dilution hypothesis) infection risk measured as pathogen infection prevalence or pathogen species diversity in a local community (Elton, 1958; Keesing *et al.*, 2006; Halliday *et al.*, 2017; Rohr *et al.*, 2020). The majority of studies have reported a negative correlation between biodiversity and disease risk of one focal host species, hence supporting the dilution hypothesis (Mitchell *et al.*, 2003; Pagán *et al.*, 2012; Civitello *et al.*, 2015; Fraile *et al.*, 2017; Liu *et al.*, 2020; Magnusson *et al.*, 2020; Rohr *et al.*, 2020). However, in some communities increasing biodiversity has led to more efficient spread of infection, supporting the amplification hypothesis (Hechinger & Lafferty, 2005; Halliday *et al.*, 2017), while in others host-related traits such as wide susceptibility (Pautasso *et al.*, 2005; Carnegie *et al.*, 2016) leads to rapid spread of the infection even in highly diverse host communities. The relationship between host and parasite species richness is generally considered positive (Hechinger & Lafferty, 2005; Lafferty, 2012; Kamiya *et al.*, 2014; Johnson *et al.*, 2016; Liu *et al.*, 2016), yet how sensitive this relationship is to environmental variation in natural communities remains unknown because most studies to date have focused on a limited number of populations (Liu *et al.*, 2020; Rohr *et al.*, 2020).

Given that human alteration of habitats has the potential to disrupt the ecological laws of competition, mutualism and antagonism by which species interact in wild communities (Tylianakis *et al.*, 2008; Wood *et al.*, 2018; Guo *et al.*, 2019), understanding how the biodiversity–disease relationship changes in plant populations adjacent to agricultural fields is of vital importance for predicting and preventing disease epidemics. Understanding virus dynamics at the agro-ecological interface may be particularly important, as many viruses are known to have wide host ranges that encompass both wild plants and crops (Power & Mitchell, 2004; Power *et al.*, 2011; Bernardo *et al.*, 2017; Fraile *et al.*, 2017). Moreover, viruses are typically transmitted by vectors, which may increase virus movements across landscapes (Hogenhout *et al.*, 2008). Here, we study whether the relationship between native host plant richness and infection risk varies between populations in natural and agricultural landscapes. First, we predict that biodiversity decreases infection prevalence, and increases pathogen richness (Lafferty, 2012; Rottstock *et al.*, 2014; Civitello *et al.*, 2015). The diverse communities are expected to harbour more diverse niches for specialist pathogens as well as alternative hosts for generalist pathogens, but to hinder the spread of the infections as the prevalence of highly competent hosts decreases in more diverse host communities as predicted by the dilution effect hypothesis (Rottstock *et al.*, 2014; Civitello *et al.*, 2015). Second, we predict virus species richness and infection prevalence to be higher in the proximity of agricultural land use (Papaix *et al.*, 2015). In the lands adjacent to agricultural practices, the plant susceptibility phenotype may be altered due to changes in temperature and water conditions, as well as leached fertilizers (Marshall, 2005). Agricultural practices may also alter pathogen

virulence and lead to transmission of novel pathogen isolates to wild populations without a history of co-evolution (Burdon & Thrall, 2009; Papaix *et al.*, 2015).

To test these predictions, we investigated the impact of agricultural land use on five recently described virus species (Susi *et al.*, 2017, 2019) in 27 *Plantago lanceolata* populations across the Åland Islands, south-west of Finland. In our study, we account for host population connectivity to understand how spatial distances separating host populations affect the distribution of virus infections (Parratt *et al.*, 2016). We expect population connectivity to increase virus transmission and thus to increase infection prevalence and virus richness in well-connected populations. Specifically, we ask: Does the structure of pathogen communities differ between natural and agricultural edge settings? Do nutrient levels vary between the two habitat types in a manner that explains differences in pathogen richness and infection risk? We expect both nitrogen (Mitchell *et al.*, 2003) and phosphorus (Borer *et al.*, 2014) to increase infection prevalence. Do the characteristics of host populations (population connectivity) and plant communities (plant richness and diversity) explain differences among virus infection prevalence and richness between these two settings? Our study allows for a joint analysis of the spatial, biotic and abiotic drivers of infection risk and diversity across a human-modified landscape.

## Materials and Methods

### Study system

*Plantago lanceolata* L. is a wind-pollinated monoecious rosette-forming herb with a worldwide distribution (Sagar & Harper, 1964). In the Åland Islands south-west of Finland, recent studies have identified five new viruses infecting *P. lanceolata*: *Plantago lanceolata* latent virus (PILV; (Susi *et al.*, 2017), *Plantago* latent caulimovirus, *Plantago* betapartitivirus, *Plantago* enamovirus and *Plantago* closterovirus (Susi *et al.*, 2019)). In the Åland Islands, *P. lanceolata* occurs as a network of *c.* 4000 highly fragmented populations that are annually surveyed for the presence of a fungal pathogen, *Podosphaera plantaginis*, and a butterfly, *Melitaea cinxia* (Ojanen *et al.*, 2013). During these surveys, data on the spatial characteristics including size and location of the populations are collected (Ojanen *et al.*, 2013). Due to the fragmented landscape structure in the Åland Islands, *P. lanceolata* populations are discrete patches, one population consisting usually of a single meadow or comparable site surrounded by unsuitable matrix (i.e. habitat in which *P. lanceolata* cannot grow) of rocky outcrops, cultivated field, waterbodies or dense forest (Ojanen *et al.*, 2013). The area of each population has been GPS-delineated, and the average population area is 0.5 ha (Ojanen *et al.*, 2013). In areas where populations occur densely, two patches must be separated by at least 20 m distance of unsuitable habitat or 50 m of suitable but non-*P. lanceolata* growing area (Ojanen *et al.*, 2013). Due to its long-term seed bank and vegetative spread, *P. lanceolata* populations are spatially stable and rarely go extinct (Ojanen *et al.*, 2013).

The study populations are located in the main islands of Åland where the main crops are silage crops (e.g. mixtures of grasses

and clovers) and cereals (e.g. oats, barley, wheat) that consist of 52% and 24% (respectively) of agricultural land in the study area (LUKE, 2020). In the Åland Islands, farms are typically small (average size 42 ha), practising annual rotation of crops, but without having regional specialization among cultivated crops (LUKE, 2020). At least three of the viruses used in this study belong to virus families – Caulimoviridae, Luteoviridae and Closteroviridae – that are generalists being capable of infecting of the crops commonly grown in the Åland Islands. Caulimoviridae infect cabbage (Raybould *et al.*, 1999) and oilseed rape, reducing yield (Walsh & Tomlinson, 1985). Luteoviridae viruses infect peas, clovers, cereals and Brassicae, typically causing yellowing symptoms and yield reduction (Cockbain & Gibbs, 1973; Raybould *et al.*, 1999). Closteroviridae infect cereals, clovers, cucumbers, beets, and lettuce (German-Retana *et al.*, 1999).

### Field survey and sampling

To evaluate the impact of agricultural land use on viral communities in *P. lanceolata* populations in the Åland Islands, we chose 15 populations (*agricultural edge*) that were located in the immediate proximity of crops or pastures (within a distance of 20 m), and 12 populations (*natural*) that were separated from agricultural land use by at least a distance of 200 m, and surrounded by a matrix of forests and rocky outcrops (Fig. 1). Agricultural practices are expected to impact on soil and plant species community dynamics within the 20 m field margins that we used here (Kivinen *et al.*, 2004; Marshall, 2005). We used a 200 m distance from agricultural fields as it is beyond relevant virus transmission distances by aphids (Loxdale *et al.*, 1993) as most aphid dispersal occurs over distances of only a few metres (Parry, 2013). The populations of both habitat types were selected to vary in their connectivity and to represent different areas of the Åland *P. lanceolata* population network. In July 2015, we collected DNA and RNA samples from up to five *P. lanceolata* plants expressing symptoms typical for virus infection (yellowing, redness, curliness, necrotic spots; Susi *et al.*, 2019), and five *P. lanceolata* plants without typical virus symptoms in each population. The symptomatic and asymptomatic samples were collected to test whether infection or co-infection prevalence is more frequent in plants that have virus symptoms. Altogether, 267 plants were sampled. From each plant, we collected 1 cm<sup>2</sup> sample of a young leaf and placed it into a microtube and stored it at –20°C until DNA extraction. For RNA extraction, three young leaves from each plant were collected and stored immediately in liquid nitrogen. In the laboratory, the RNA samples were stored at –80°C until RNA extraction.

To evaluate the association between viral infections and plant species, we counted the number of plant species in 0.5 m<sup>2</sup> vegetation plots around each sampled plant. Photographs on each of these 0.5 m<sup>2</sup> plots were taken for identification in the laboratory. Thus, the plant community here is the plant species community identified within the vegetation plots of one *P. lanceolata* population (0 = species not found in the given vegetation plot; 1 = species found in the given vegetation plot; for more information on plant communities in the populations, see Supporting

Information Table S1). In each *P. lanceolata* population, the plant species in each vegetation plot were identified and plant species prevalence was calculated as the sum of its occurrences within the up to 10 vegetation plots in the population. Shannon's index of diversity (Shannon & Weaver, 1949) was calculated based on the plant species and their prevalence in the population as:

$$H = -\text{SUM}[(pi) \times \log_e(pi)].$$

where *SUM* is summation, and *pi* is prevalence as the summed occurrence (0 = species not found in the given vegetation plot; 1 = species found in the given vegetation plot) of species/total number of vegetation plots within the patch.

To understand how spatial connectivity of the host populations impacts on the distribution of virus infection, *P. lanceolata* population connectivity was calculated as.

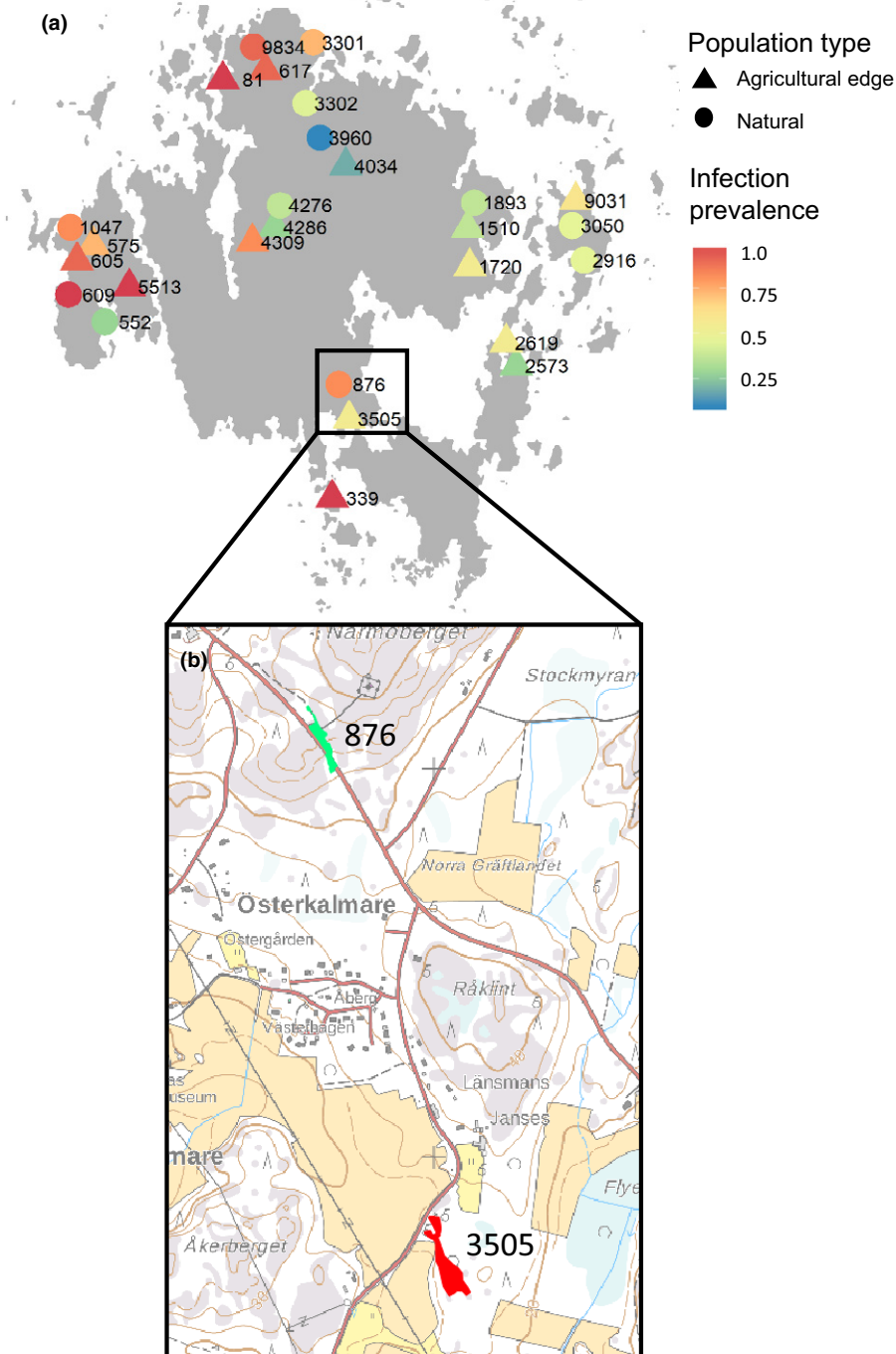
$$S_i^L = \sum \exp(-\alpha d_{ij}) \sqrt{A_j}$$

where  $d_{ij}$  is the Euclidian distance between patches *j* and *i* and  $\alpha$  is the parameter of the negative exponential dispersal kernel, which was set to 1 km<sup>-1</sup> (for more details see Jousimo *et al.*, 2014).  $A_j$  is the area of habitat patch *j*, and the square root transformation was used because this roughly corresponds to the scaling of host population size with patch area (Laine & Hanski, 2006). The connectivity parameter allows us to estimate how the spatial distance separating populations affects virus infection risk, and also captures potential unmeasured variation that declines with increasing distance separating the populations.

To assess the impact of soil nutrients on the viral infections, we collected a 0.5-l soil sample next to each sampled plant. The soil samples were stored in plastic bags at 5°C and all samples from a given population were pooled into a 0.5-litre sample in the laboratory. The samples were analysed for their nitrogen and phosphorus content at Hortilab (Närpiö, Finland). Soil nitrogen was measured as NO<sub>3</sub>-N from soil-water extraction with an ion-selective electrode (Thermo Scientific 9300) using analysis with a Thermo Scientific Orion SA 720. Soil phosphorus was measured from acetate extract using AQ2 Discrete Analyzer (Seal Analytical GmbH, Norderstedt, Germany).

### Nucleic acid extractions and virus detections

To detect the two DNA viruses (PILV and Plantago latent caulimovirus), DNA was extracted using an E.Z.N.A. Plant kit (Omega Biotek, USA) following the manufacturer's instructions with the final elution performed in 100 µl. To detect three RNA viruses (Plantago betapartitivirus, Plantago enamovirus and Plantago closterovirus), RNA was extracted using phenol/chloroform as in Chang *et al.* (1993). The RNA was reverse transcribed using iScript cDNA Synthesis Kit (Bio-Rad). The presence of the five viruses in the 267 *P. lanceolata* samples were detected using reverse transcription (RT)-PCR detection using specific primers (Table S2; Susi *et al.*, 2019). In the PCR, we used GoTaq (Promega) polymerase and the following thermal cycling



**Fig. 1** (a) Spatial variation in virus infection prevalence of 15 agricultural edge (triangles) and 12 natural (circles) *Plantago lanceolata* populations in the Åland Islands. The labels indicate population IDs. Ten plants from each population were sampled and (RT-)PCR was used to detect *Plantago* latent caulimovirus, *Plantago lanceolata* latent virus, *Plantago* betapartitivirus, *Plantago* enamovirus and *Plantago* closterovirus. (b) An example of the location of natural (green) and agricultural edge (red) populations located in the landscape.

conditions: 95°C for 2 min, 25 cycles of 95°C (40 s), 50–57°C (40 s) and 72°C (1 min), and a final extension of 72°C for 5 min. The amplicons were resolved on a 1.2% agarose gel and visualized using Gel Doc XR System (Bio-Rad).

### Statistical analyses

To test whether symptomatic plants are more commonly infected than the nonsymptomatic plants, a generalized linear mixed model was set up in SAS 9.1 PROC GLIMMIX (SAS Institute Inc.,

Cary, NC, USA) using the symptom appearance of each plant (0 = no symptoms, 1 = symptoms) as a binomial response variable. The infection status of the plant (0 = not infected, 1 = infected) was used as the class explanatory variable and population was used as the random variable. To understand whether symptomatic plants are more commonly co-infected than the non-symptomatic plants, a model with a similar structure was set up using co-infection prevalence (0 = singly infected, 1 = co-infected) of the plant as the class explanatory variable. In this analysis, only the 152 infected plants were used.

To understand whether the virus and plant species communities differed between agricultural edge and natural habitat types, we ran analysis of similarities (ANOSIM) in R software (R Development Core Team, 2014) using the VEGAN package (Oksanen *et al.*, 2018). In virus analysis, the prevalence of the five virus species in each population was used. Similarly, in plant analysis, the occurrences of the 151 plant taxa (Table S1) identified in the vegetation plots in each population were used.

To understand if the measured population characteristics (soil nutrient contents, host population connectivity, plant species richness and diversity) differed between agricultural edge and natural populations, we performed a set of five analyses as generalized linear models (GLMs) in SAS 9.1 PROC GLIMMIX (SAS Institute). Each variable was analysed separately and population type (agricultural edge or natural) was used as a class explanatory variable. Soil nitrogen and phosphorus values were log-transformed and a gamma distribution of error was assumed. In the models analysing host population connectivity, and plant diversity, a Gaussian distribution of errors was assumed. In the model analysing plant species richness, a Poisson distribution of error was assumed.

We analysed the drivers of virus infection prevalence and richness in the *P. lanceolata* populations using generalized linear models in SAS 9.1 PROC GLIMMIX (SAS Institute). First, we constructed a model with virus infection prevalence in the populations (proportion of *P. lanceolata* plants infected by one or more viruses) as a continuous response variable. A Beta distribution of error was assumed. Host population connectivity, mean number of plants species in the 0.5 m<sup>2</sup> plots, soil phosphorus and nitrogen were used as continuous explanatory variables. Agricultural land use (1 = agricultural land use, 0 = no agricultural land use) was used as a class explanatory variable. Second, to understand the drivers for virus richness in the population, the same model using number of virus species present in the population was fitted. In both models, interactions between agriculture and other explanatory variables were tested, as well as the interaction between soil nitrogen and phosphorus, and only statistically significant interactions with best fit based on Akaike's information criteria was used to select the final model. Next, to understand how plant diversity impacts on virus infection prevalence and

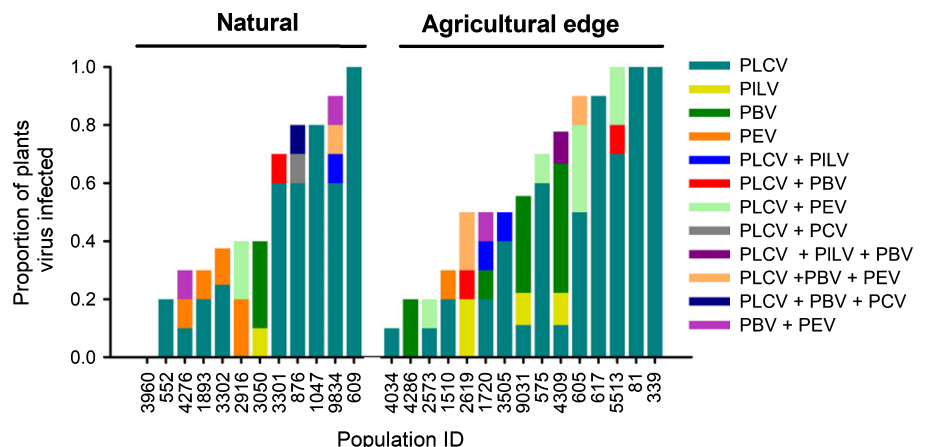
species richness, we fitted two models with similar structure. In these models, we used plant diversity Shannon's index value as a continuous explanatory value instead of plant species diversity. We obtained the slopes for the coefficients as well as the slopes for the agricultural edge and natural populations on soil nitrogen, plant richness and plant diversity from the models.

To test whether the studied parameters (virus infection and richness, plant diversity and richness, agricultural land use, connectivity, host plant coverage, as well as soil phosphorus and nitrogen) vary in space, we implemented regression analyses in SAS 9.1 PROC REG (SAS Institute) using each variable separately as a response variable and latitude and longitude as explanatory variables. Soil nitrogen and phosphorus values were log-transformed for the analysis.

## Results

Using the five virus-specific PCR primers (Susi *et al.*, 2019), we found high levels of virus infections across the 27 *P. lanceolata* populations with 57% of the 267 studied plants infected by one or more viruses (Fig. 2). Populations differed markedly in their virus infection prevalence (0–100% plants per population infected; Figs 1, 2) and in the richness of virus species (ranging between zero to four viruses; Fig. 2). The most common virus was Plantago latent caulimovirus, which was detected in 46.8% of the plants. The other four viruses were found in varying frequencies infecting 0.8–9.7% of the plants (Fig. 2). Co-infections consisting of two or more viruses were common as they were found in 17.1% of all infected plants (Fig. 2). We did not find any difference in the occurrence of virus infection ( $df = 1,116$ ;  $F = 0.79$ ;  $P = 0.375$ ) or co-infection ( $df = 1,63$ ;  $F = 0.07$ ;  $P = 0.788$ ) between symptomatic and asymptomatic plants. Using ANOSIM, we tested whether virus communities differ between agricultural edge and natural sites, and found that there was a significant difference between the two habitat types ( $R^2 = 0.149$ ;  $P = 0.012$ ).

We obtained the plant diversity measures by identifying and recording the presence–absence of all plant species in 0.5 m<sup>2</sup> vegetation plots surrounding each sampled plant. We identified in total 151 plant species (Table S1) across the vegetation plots in



**Fig. 2** Richness of the virus communities found in 27 *Plantago lanceolata* populations in the Åland Islands based on Plantago latent caulimovirus (PLCV), Plantago lanceolata latent virus (PILV), Plantago betapartitivirus (PBV), Plantago enamovirus (PEV) and Plantago closterovirus (PCV) PCR detections. The plants where more than one virus species was detected are presented as their specific species combinations as shown in the legend.

all populations. To test whether the plant community composition differs between agricultural edge and natural populations, we compared the plant species communities in the two habitat types using ANOSIM. The plant community composition did not differ significantly ( $R^2 = 0.022$ ;  $P = 0.325$ ) between the two habitat types.

Using the GLM framework, we tested if the two habitat types differed in plant species diversity or richness, nutrients, or connectivity. We found that neither plant species richness (as number of plant species) nor diversity (Shannon's diversity) differed between agricultural and natural populations (Table 1c; Fig. 3a, b). Phosphorus and nitrogen levels were markedly higher in agricultural populations than in natural populations (Table 1c; Fig. 3e,f). *Plantago lanceolata* population connectivity did not differ between population types (Table 1c; Fig. 3g).

**Table 1** Differences of virus infection prevalence and species abundance in *Plantago lanceolata* populations in models with (a) plant species diversity (Shannon) and (b) richness analysed with generalized linear models; (c) the variation between agricultural and natural population types in soil nutrient status, host population size and connectivity as well as plant species richness analysed with generalized linear models.

(a)	Virus infection prevalence			Virus species richness		
	df	F	P	df	F	P
Soil phosphorus	<b>1, 15</b>	<b>17.27</b>	<b>&lt; 0.0001</b>	1, 20	0.44	0.5154
Soil nitrogen	1, 15	7.76	0.1187	1, 20	0.95	0.3423
Host connectivity	<b>1, 15</b>	<b>41.44</b>	<b>&lt; 0.0001</b>	1, 20	1.03	0.3233
Agriculture	1, 15	0.00	0.9614	<b>1, 20</b>	<b>5.46</b>	<b>0.0299</b>
Plant diversity	<b>1, 15</b>	<b>25.43</b>	<b>0.0001</b>	1, 20	2.00	0.1722
Plant diversity × Agriculture				<b>1, 20</b>	<b>5.40</b>	<b>0.0308</b>
Soil nitrogen × Agriculture	<b>1, 15</b>	<b>16.2</b>	<b>0.0011</b>			

(b)	Virus infection prevalence			Virus species richness		
	df	F	P	df	F	P
Soil phosphorus	<b>1, 15</b>	<b>13.71</b>	<b>0.0021</b>	1, 20	0.12	0.7314
Soil nitrogen	1, 15	1.31	0.2695	1, 20	0.17	0.6878
Host connectivity	<b>1, 15</b>	<b>15.48</b>	<b>0.0013</b>	1, 20	2.90	0.1039
Agriculture	1, 15	0.69	0.4179	1, 20	<b>15.06</b>	<b>0.0009</b>
Plant species richness	<b>1, 15</b>	<b>5.78</b>	<b>0.0296</b>	1, 20	1.18	0.2901
Plant species richness × Agriculture				<b>1, 20</b>	<b>16.39</b>	<b>0.0006</b>
Soil nitrogen × Agriculture	<b>1, 15</b>	<b>10.51</b>	<b>0.0055</b>			

(c)	Virus infection prevalence		
	df	F	P
Soil phosphorus	<b>1, 25</b>	<b>4.36</b>	<b>0.0472</b>
Soil nitrogen	<b>1, 25</b>	<b>30.44</b>	<b>0.0027</b>
Host connectivity	1, 25	0.56	0.4594
Plant species richness	1, 25	0.00	0.9816
Plant diversity	1, 25	1.45	0.2401

Statistically significant results ( $P < 0.05$ ) are shown in bold.

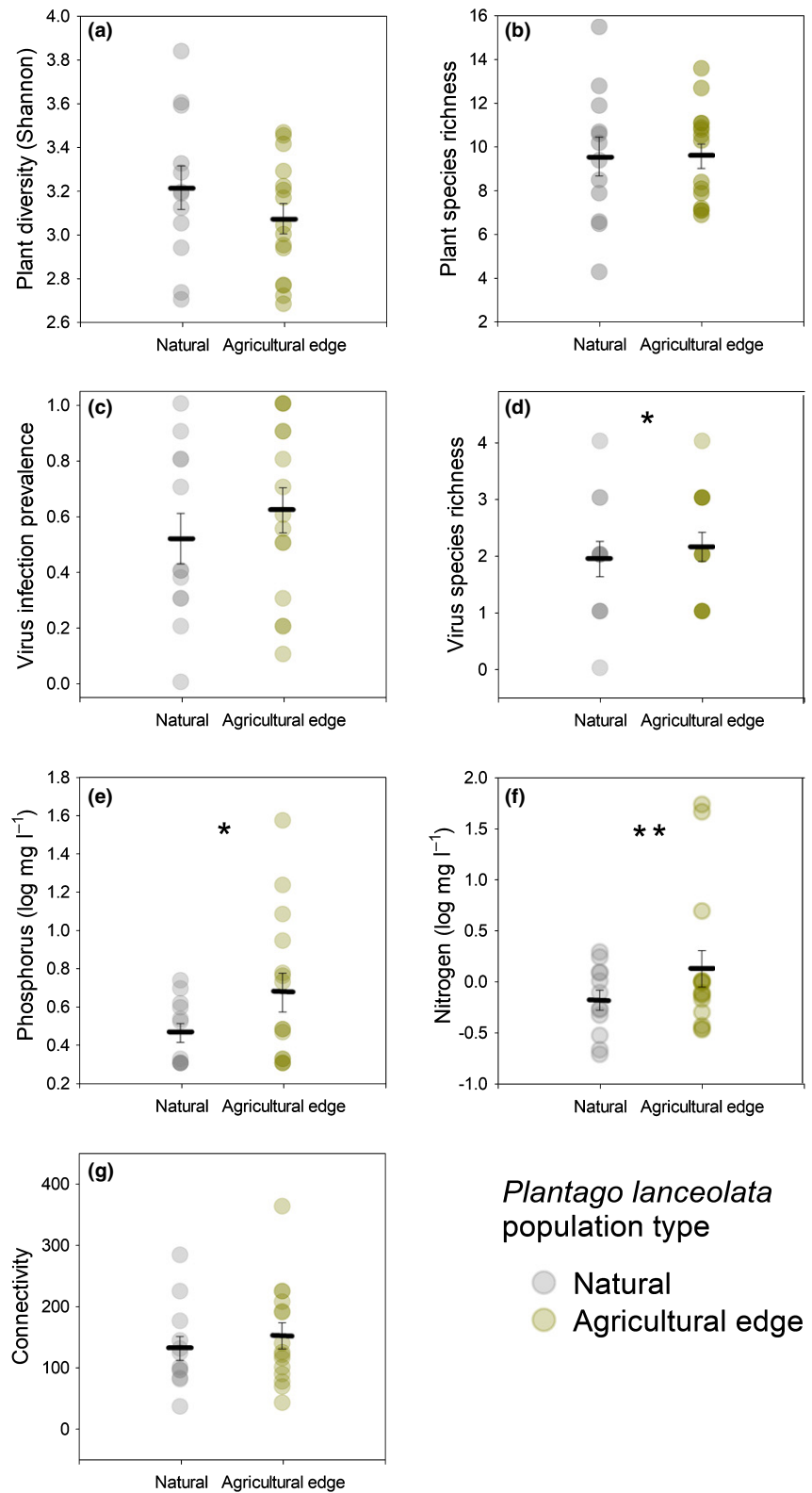
Infection prevalence was negatively correlated with plant diversity (Table 1a; Fig. 4a). Contrary to expectations, there was no difference in infection prevalence between agricultural edge and natural habitats (Table 1a; Fig. 3c). Population connectivity was the most significant driver of virus infection prevalence; well-connected *P. lanceolata* populations were less infected than isolated populations (Table 1a; Fig. S1). We found that positive correlation between soil nitrogen and virus infection prevalence was only observed in the agricultural edge populations (Tables 1, S2; Fig. S1), but that did not result in differences in virus prevalence among agricultural edge and natural populations. Similarly, in the model using plant species richness as a diversity measure, there was a negative correlation between infection prevalence and plant richness, and no effect of proximity to agricultural habitat (Table 1b; Fig. 4c). Phosphorus correlated positively and connectivity negatively with infection prevalence, but in this analysis no significant effect of nitrogen was detected (Tables 1, S2). The observed correlation between soil phosphorus and infection prevalence did not lead to a significant difference in virus infection prevalence between the two habitat types.

Consistent with predictions that agricultural practices may alter disease transmission and plant susceptibility, we found that virus richness was significantly higher in populations with proximity to agriculture than in the natural populations (Table 1a,b; Fig. 3d). We did not find a direct effect of plant diversity on the richness of virus species (Table 1a,b). Instead, we found that this relationship was moderated by proximity to agricultural land use (Tables 1a,b, S2; Fig. 4b). The negative correlation between plant species diversity and virus species richness observed in natural populations became nonsignificant in agricultural populations (significant interaction between agricultural land use and plant diversity; Tables 1b, S2; Fig. 4b). When plant species richness was used as a diversity measure, a negative correlation between plant richness was seen in natural populations but, again, was absent in agricultural populations (significant interaction between agricultural land use and plant richness; Tables 1b, S2; Fig. 4d). We found that neither host population connectivity nor soil nutrients had a significant effect on the virus species richness (Table 1a,b; Fig. S1).

Finally, we tested whether the variation in the studied variables was explained by latitudinal and longitudinal location of the populations by regressing each variable with the population latitudinal and longitudinal coordinates. We found that none of the studied variables showed significant variation along longitudinal or latitudinal axes (Table S3).

## Discussion

Species interactions are the building blocks of biodiversity, yet natural levels of biodiversity are changing rapidly (Sala *et al.*, 2000). There is considerable theoretical and experimental evidence showing that changes in host richness may have direct impacts on the richness of the associated pathogen communities, as well as on disease risk (Schmidt & Ostfeld, 2001; Keesing *et al.*, 2006; Johnson *et al.*, 2013). However, little is known about how human modification of natural landscapes changes these



**Fig. 3** Comparison of agricultural and natural *Plantago lanceolata* population characteristics in the Åland Islands. Mean (a) plant diversity (*Shannon*), (b) plant species richness, (c) virus infection prevalence, (d) virus species richness, (e) soil phosphorus ( $\log_e \text{mg l}^{-1}$ ), (f) soil nitrogen ( $\log_e \text{mg l}^{-1}$ ) and (g) connectivity in agricultural edge (green circles,  $n = 15$ ) and natural populations (black circles,  $n = 12$ ). Statistically significant effects from GLM analyses are indicated as: \*,  $P < 0.05$ ; \*\*,  $P < 0.01$ . Standard error of the mean is shown.

associations. Here, we analysed jointly the effects of host richness and diversity, spatial structure and soil nutrient conditions to understand how proximity to agricultural land use changes pathogen richness and infection prevalence across the landscape. Consistent with our predictions, we find more diverse virus

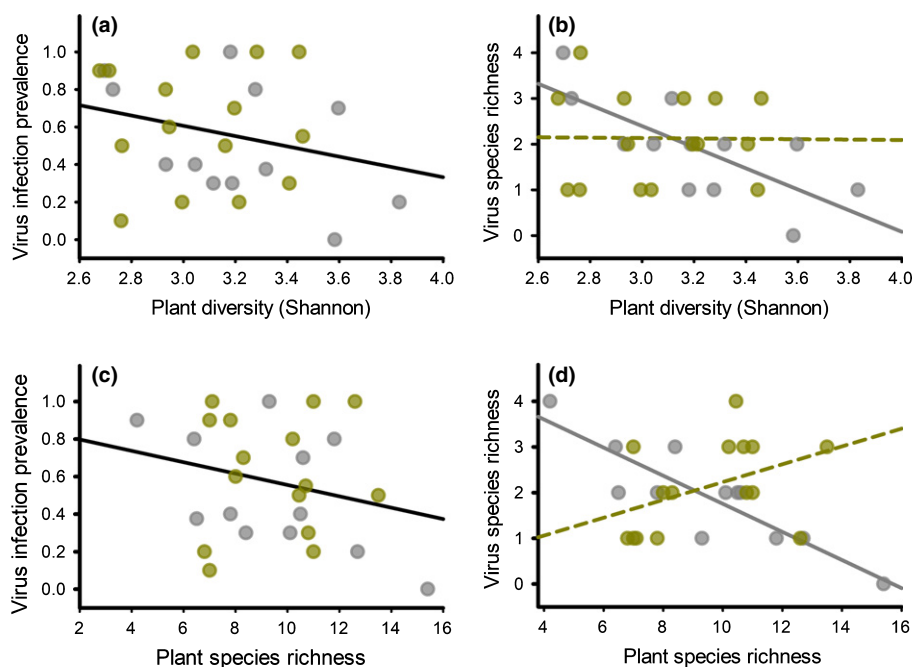
communities in host populations close to cultivated fields. Moreover, we find that agricultural land use can alter the mechanisms by which host species richness regulates disease pressure and richness in wild plant populations. High plant species richness and diversity were associated with low virus species richness in natural

plant populations while in agricultural edge populations virus species richness was moderately higher and not associated with plant richness.

We discovered that *P. lanceolata* populations located in the immediate proximity of cultivated fields and those surrounded by natural habitats differed significantly in soil nitrogen and phosphorus contents, with the agricultural populations having higher nutrient levels. Previously it has been shown that the competitive dynamics between viruses may be altered by the N : P ratio (Lacroix *et al.*, 2014), which could have a major impact on the resulting viral communities. Phosphorus has been shown to increase virus infection risk in grasses while no effect of nitrogen has been observed (Borer *et al.*, 2010). However, nitrogen addition increases levels of free amino acids in plant tissues which may attract more potential vectors, thereby increasing disease risk (Strengbom *et al.*, 2002). Plants use nitrogen and phosphorus in many functions supporting their growth (Mitchell *et al.*, 2003; Whitaker *et al.*, 2015; Lacroix *et al.*, 2017). Hence, the association between nitrogen and disease prevalence may be explained either by changes in plant growth and/or altered vector behaviour. Overall, the impact of agricultural fertilizers on disease risk in the surrounding wild populations may vary substantially, and our results suggest that spatial complexity needs to be taken into account when examining the effect of agricultural fertilizers on infection risk.

The spatial distribution of host populations is expected to be a key determinant of disease dynamics, with ecological theory predicting infection risk to increase with increasing host population size and connectivity to other populations (Parratt *et al.*, 2016).

Disease dynamics have rarely been examined in larger networks of populations varying in host population connectivity, and to date the evidence remains mixed (Carlsson-Granér & Thrall, 2002; Johnson & Haddad, 2011; Jousimo *et al.*, 2014). In the case of viral pathogens that are mostly vector-transmitted, dense networks of populations should favour infection spread (Sullivan *et al.*, 2011). However, the pattern we observed suggests that the well-connected populations are more resistant to pathogen attack, as predicted by spatial co-evolutionary theory where higher rates of gene flow are predicted to provide the upper hand in the arms race between hosts and their pathogens (Gandon & Michalakakis, 2002; Gandon & Nuismer, 2009). Indeed, previous studies in this same network of *P. lanceolata* populations confirmed disease resistance against the specialist fungal pathogen *P. plantaginis* to increase with increasing population connectivity (Jousimo *et al.*, 2014; Höckerstedt *et al.*, 2018). To date, nothing is known about resistance against viruses in *P. lanceolata* populations in the Åland Islands. Of the studied viruses, Plantago latent caulimovirus, Plantago enamovirus and Plantago closterovirus belong to virus genera (Fauquet *et al.*, 2012b) that have wide host ranges and are also frequently detected from crops (Seabloom *et al.*, 2009; Fauquet *et al.*, 2012a,c). Hence, they are not expected to be as sensitive to the spatial configuration of *P. lanceolata* as a specialist pathogen would be. The significant effect of host population connectivity on virus infection distribution may also indicate the effect of some other, unmeasured, variable on virus infections that could be biotic or abiotic. One possible explanation for higher infection rates in isolated populations is that in isolated populations plants may be under higher herbivore pressure, which in turn may impact virus



**Fig. 4** The effect of agricultural land use on the biodiversity–disease relationship. The effect of plant species diversity on (a) virus infection prevalence, (b) virus species richness, and of plant species richness on (c) virus infection prevalence and (d) virus species richness in agricultural edge (green circles and line,  $n = 15$ ) and in natural populations (black circles and grey line,  $n = 12$ ) *Plantago lanceolata* populations in the Åland Islands. The black line indicates the correlation measured in the whole dataset (both population types,  $n = 27$ ). Statistically significant correlations in GLM are indicated by a solid line ( $P < 0.05$ ); dashed line, nonsignificant.



infections (Burdon, 1996; Sallinen *et al.*, 2020). When we tested whether latitudinal or longitudinal location of the population explains the variation in disease risk or other variables studied, we found no significant effect in any of the variables. This is not surprising, as the landscape in the Åland Islands is highly patchy throughout rather than structured by region. Jointly, these results show how the processes governing infection dynamics at different spatial scales can be altered at the agro-ecological interface. Detailed studies assessing the extent of herbivory in populations representing varying spatial conditions would be needed to unravel the potential role of vector activity mediating virus infection prevalence in this system.

While infection risk did not differ between agricultural edge and natural populations, we find that the composition of virus communities differs between natural and agricultural edge populations, with the latter supporting higher virus richness. This finding is in agreement with a recent study on bacterial, fungal and oomycete pathogens, where higher diversity was reported in agricultural landscapes than natural habitats (Makiola *et al.*, 2019). Importantly, we show that high plant species richness was significantly associated with low virus species richness in natural populations, while in agricultural edge populations virus species richness was higher and not dependent on plant richness. While most studies have found a positive relationship between host and parasite species diversity (Hechinger & Lafferty, 2005; Johnson *et al.*, 2016), to date there is remarkably little data to evaluate how this relationship is played out in nature with varying degrees of human interference. The effect of host richness on infection prevalence was negative and not altered by agricultural land use. This is in contrast to the finding of increased infection risk of *Capsicum annuum* in populations with higher levels of human management (Pagán *et al.*, 2012; Fraile *et al.*, 2017), and with the finding of a decrease in diversity of viral pathogens in bats under anthropogenic influence (Bergner *et al.*, 2020). This suggests that host community history and type of anthropogenic disturbance may be critical for shaping host diversity – parasite diversity and infection risk relationships.

In our study, neither plant species richness nor diversity differed between the natural populations and those situated in the proximity of cultivated fields. In our analyses we control for the effects of distances separating populations and soil nutrients and, hence, this suggests that another unmeasured feature of the cultivated fields is interfering with the infection mediation mechanisms observed in the natural populations. We propose that the lack of dilution effect in lands adjacent to agricultural fields may be due to a combination of infection spillover – of potentially different virus isolates (Zhan *et al.*, 2015) – from crops to the natural environments (Power & Mitchell, 2004; Thrall *et al.*, 2011; Alexander *et al.*, 2014), and altered vulnerability in plant populations subjected to leached agrochemicals (Zhan *et al.*, 2015). This would be in line with other studies reporting altered virus dynamics due to agricultural practices (Pagán *et al.*, 2012; Bernardo *et al.*, 2017; Fraile *et al.*, 2017). Although we do not have detailed knowledge on vector communities, vector prevalence may be higher in agricultural landscapes (Clafin *et al.*, 2017), and vector efficiency may differ between wild hosts and

cultivated hosts (Hall *et al.*, 2010). Jointly, our results suggest that the frequency of competent hosts supporting higher viral diversity declines with increasing host diversity, and this host diversity mediated dilution of viral diversity is lacking in agricultural edge populations. Our finding is in contrast to some experimental studies on the host richness – disease relationship that suggest diverse host communities to increase parasite richness (Hechinger & Lafferty, 2005; Johnson *et al.*, 2016). One potential reason for this discrepancy may also be in the scale at which studies are done, as the biodiversity – disease relationship is suggested to follow a hump-shaped curve, and to depend on the spatial scale of the interactions (Halliday & Rohr, 2018).

In conclusion, our results suggest that nutrient spillover as well as potentially increased transmission from crops changes infection dynamics in the wild. Importantly, we discovered that, as a result, biodiversity mediation of pathogen richness disappears in the agricultural edge populations. Jointly, our results highlight the multiple ways in which human use of landscapes changes the ecological laws by which natural communities are formed with far-reaching implications for ecosystem functioning and disease.



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## Author contributions

HS and A-LL jointly conceived the study. HS oversaw data collection and analyses, and prepared the first draft of the manuscript. Both authors contributed significantly to the writing of the manuscript.

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## Data availability

The data that support the findings of this study are openly available in the Dryad Digital Repository: <https://doi.org/10.5061/dryad.3r2280gf1>.

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## Supporting Information

Additional Supporting Information may be found online in the Supporting Information section at the end of the article.

**Fig. S1** The impact of *Plantago lanceolata* population connectivity, soil nitrogen and phosphorus levels on virus infection prevalence, virus species richness, plant diversity and species richness in 27 *P. lanceolata* populations in the Åland Islands.

**Table S1** Plant communities in the habitat patches of the 27 *Plantago lanceolata* populations used in the virus detection in the Åland Islands.

**Table S2** The effects of soil nutrients (nitrogen and phosphorus), population connectivity, proximity to agricultural land use, plant species richness and diversity on virus infection prevalence and

virus species richness in *Plantago lanceolata* populations in the Åland Islands.

**Table S3** The results of regression analysis of the variation in virus infection prevalence and diversity, plant species richness and diversity, agricultural land use, agricultural land use, population size and connectivity, and soil phosphorus and nitrogen of 27 *Plantago lanceolata* populations with latitude and longitude in the Åland Islands.

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