Is within-host viral community assembly shaped by local adaptation?

Maija Jokinen[1], Hanna Susi[1,2] & Anna-Liisa Laine[1]

[1] Organismal and Evolutionary Biology Research Programme, Faculty of Biological and Environmental Sciences, PO BOX 65, 00014, University of Helsinki, Finland

[2] Department of Agricultural Sciences, Faculty of Agriculture and Forestry, PO BOX 27, 00014, University of Helsinki, Finland

Corresponding author: Maija Jokinen maija.jokinen@helsinki.fi

Running title: Viral local adaptation

local adaptation, coevolution, viruses, host-parasite coevolution

Abstract

Host-parasite coevolution describes the continuous reciprocal selection driving host defense and parasite infectivity, with direct consequences for disease dynamics. While abundant evidence exists for coevolution shaping host-parasite dynamics within the 'one host-one parasite' framework, hosts are typically infected by multiple parasites and the extent to which coevolutionary processes shape within-host parasite communities remains poorly understood. Investigating these interactions is essential for understanding how coevolution drives parasite diversity, competition, and coexistence within hosts. Here, we conducted a local adaptation experiment to investigate the effects of coevolution on within-host viral community assembly in *Plantago lanceolata*. Greenhouse-grown individuals were reciprocally transplanted into wild populations during natural viral epidemics. We combined small-RNA sequencing to identify the viral communities and joint species distribution modelling to quantify the effects of local adaptation, population and host characteristics on viral community assembly. Our results show that host populations vary in the extent to which local adaptation influences within-host viral diversity. Across all populations, host maternal line and origin population were the main determinants of viral community composition and infection status. The effects varied across virus families, suggesting virus-specific assembly processes and variation in the potential for coevolution to shape these interactions.

Introduction

Coevolutionary theory predicts reciprocal selection to drive key interaction traits in hosts and parasites – resistance and infectivity, respectively (1,2). Coevolution is fundamental for understanding host-parasite interactions and disease dynamics in nature, as the presence of parasites depends on the availability of susceptible hosts. Indeed, host-parasite interactions provide some of the most compelling evidence for the theory of coevolution, often demonstrated through local adaptation experiments (3–6). However, much of this work has focused on the one-host-one-parasite framework, although in nature hosts are rarely infected by a single parasite and often support complex parasite communities (7–10). Despite the growing interest in within-host parasite communities in natural environments, there remains a gap in our understanding of how coevolution can shape these complex communities (11,12).

Genetic variation and genotype-genotype specificity in the interaction are prerequisites for coevolution. Indeed, the ability to infect or resist infection can be genotype-dependent: some parasite genotypes can infect only certain host genotypes, while some host genotypes exhibit resistance to specific parasite genotypes (13–15). This variation is maintained by evolutionary mechanisms, such as parasite-imposed negative frequency-dependent selection and arms-race dynamics, which can favour different host genotypes in different populations, contributing to local adaptation (3,15,16). Notably, the outcome of host genotype × parasite genotype interactions may be altered under multiple parasite attack (17,18), with co-occurring parasites influencing community assembly either directly through parasite-parasite interactions (19,20) or indirectly through host-mediated responses (14,21). If host colonization ability – a trait expected to be shaped by coevolution – is sensitive to co-occurring parasites, then we may expect community assembly to be shaped by both ecological and evolutionary dynamics (22). The community monopolization hypothesis – evoked to explain evolutionary priority effects – predicts that locally adapted resident species can have a competitive advantage over later

arriving individuals, potentially influencing parasite community dynamics (23,24). It has been demonstrated that adaptation can reduce competitive dominance with direct consequences for community assembly (24), and that locally adapted parasites can influence the composition of the entire community (25,26).

Viruses, similar to other parasites, can form highly diverse communities (20,27–32). As obligate parasites, viral reproduction relies on the virus' ability to infect and hijack host cell machinery (33), making host-virus interactions a key factor in shaping viral communities (14,34,35). Here, to investigate how viral community assembly is influenced by coevolution, we conducted a reciprocal transplant experiment, by placing naïve *Plantago lanceolata* individuals as sentinels in sympatric and allopatric populations during naturally occurring viral epidemics. We sampled the plant individuals at the end of the growth season for small-RNA sequencing to characterize viral communities and used joint species distribution modelling (36) to tease apart the effects of local adaptation, population and host characteristics on viral community assembly. Specifically, we ask: i) Can we detect viral local adaptation? ii) What is the importance of local adaptation in determining viral community assembly? iii) What is the role of population and host characteristics in viral community assembly?

Materials and Methods

Study species

The host, *Plantago lanceolata,* is a perennial herb reproducing clonally with side rosettes or sexually with wind-dispersed pollen (37). *Plantago lanceolata* occurs worldwide, and in Finland, *P. lanceolata* can be found mainly in SW Finland. In the Åland Islands (an area spanning 50 × 70 km), *P. lanceolata* forms a large network consisting of over 4000 small fragmented populations (38).

The *P. lanceolata* host populations in the Åland Islands harbour complex viral communities (19,20). Five novel *P. lanceolata* infecting viruses have been characterised from this system, and PCR primers have been developed for their detection (14,39,40). Viral symptoms in wild hosts are challenging to identify but can include yellowing or redness of the leaf, curliness and necrotic lesions (40–42). *Plantago lanceolata latent virus* (PILV) infection has been linked to yellowing of the leaf (40,43).

Preparation of host plant material and field experiment

To investigate the role of local adaptation in viral community assembly, we conducted a reciprocal transplant experiment in three *P. lanceolata* populations (ID: s: 9205, 876, and 950) in the Åland Islands. In autumn 2020, seeds were collected from eight individuals per studied population and germinated in early April 2021 with the aim of obtaining up to 15 offspring per maternal line. The seeds from 24 maternal lines (Supplementary table 1), were sown in peat pots with a 3:1 mixture of potting soil and sand and then placed in a growth chamber with a 16:8 h light-dark cycle. After three weeks, the seedlings were transferred to the greenhouse and replanted into 10 cm ×10 cm pots filled with a 1:1 mixture of potting soil and sand. The plants were watered as needed and, when large enough, fertilized weekly with NPK fertilizer (7:2:2). During the growth period in the greenhouse, leaf samples were collected for PCR screening to confirm that each maternal line was virus-free of PlLV, *Plantago latent caulimovirus*, *Plantago betapartitivirus*, *Plantago enamovirus,* and *Plantago closterovirus,* all of which are among the most common viruses in the Åland Islands populations (39,40). Two weeks prior to the transplant experiment, the plants were treated with fungicide (Bordeaux mixture).

In early June 2021, the greenhouse-grown naïve plants were taken to the Åland Islands and placed in their transplant populations. For each maternal line, five offspring were placed

115 in their sympatric *P. lanceolata* population and five in each of the two allopatric populations
116 (Figure 1). For four maternal lines with fewer offspring, priority was given to sympatric
117 placement, and the remaining individuals were distributed among the two allopatric
118 populations (Supplementary table 1). Finally, the experiment consisted of 348 plants across the
119 three transplant populations (Supplementary table 1). The plants were randomly placed among
120 the natural vegetation and kept in pots placed inside plastic boxes (approximately 13 cm × 11
121 cm) to isolate them from the local soil. To minimize within-population spatial effects, we
122 shuffled the plants among the plastic boxes three times per week for the duration of the
123 experiment. The plants were watered as needed.

124 After six weeks of exposure, a 3 cm^2 piece of leaf tissue was collected for RNA
125 extraction and snap-frozen in liquid nitrogen. At this time, we also recorded host characteristics
126 that prior work suggests could affect viral infections on *P. lanceolata*. Plant size was measured
127 as $n \times A$, where n is the number of leaves and $A = \pi ab$, where a is the half axis of the width of
128 the largest leaf, and b is the half axis of the length of the largest leaf (14,19).

129

130 RNA extraction and RNA purification

131 Total RNA was extracted using a modified acid phenol-chloroform extraction protocol (44). A
132 3 cm^2 leaf tissue sample was ground in liquid nitrogen, after which 800 µl of warm extraction
133 buffer was added and mixed thoroughly. The extraction buffer consisted of 2%
134 hexadecyltrimethylammonium bromide (Sigma-Aldrich USA), 2% of polyvinylpyrrolidone K-
135 30 (MW 40 000, Sigma-Aldrich USA), 100 mM of Tris-HCl (pH 8.0, Thermo Fisher Scientific,
136 USA), 25 mM of Ethylenediaminetetraacetic acid (pH 8.9, Sigma-Aldrich, USA), 2.0 M of
137 NaCl (Sigma-Aldrich, USA) and 2% of β-mercaptoethanol (Sigma-Aldrich, USA). Next, 800
138 µl of acid phenol-chloroform-isoamyl alcohol (IAA; 25:24:1) was added, and the sample was

centrifuged at 13500 rpm for 15 minutes at RT. The supernatant was transferred to a clean tube, mixed with 1 ml phenol-chloroform-IAA and centrifuged under the same conditions. RNA was precipitated by adding 160 µl of 10 M LiCl and incubating overnight at +4 °C. The following day, samples were purified with chloroform-IAA (24:1) purification step and washed twice with ethanol. The RNA pellet was resuspended in 25 µl of nuclease-free water and treated with Ambion® DNA-*free*™ DNA removal Kit (Invitrogen, USA). RNA concentration was measured using Nanodrop 2000 (Thermo Fischer Scientific, USA) and Qubit (Thermo Fischer Scientific, USA), and RNA was stored at -80 °C.

Small-RNA sequencing and bioinformatic pipeline

To identify the viral communities present in the sentinel plants, we assigned the samples to small-RNA (sRNA) sequencing. From the 348 sampled experimental plants, we randomly selected samples from three individuals from each maternal line from each transplant population to be assigned for sRNA sequencing. From maternal line 876-4, we sequenced three samples from the sympatric transplant population but only one sample from one of the allopatric populations, resulting in 211 samples assigned for sRNA sequencing. The RNA extracted from the selected samples was diluted with nuclease-free water and sent to the sequencing facility according to the sequencing company's instructions (Fasteris SA, Switzerland).

The sRNA sequencing and library preparation were carried out at Fasteris SA (Switzerland). Small-RNA cDNA libraries were prepared using QIAseq miRNA Library Kit (Qiagen) according to Fasteris SA Small RNA-Seq Gel-free protocol with 100 ng of total RNA. Sequencing was performed using Illumina NovaSeq 6000 (Illumina Inc, San Diego, California, USA) and targeted insert sizes from 0 nt to 43 nt with an average library yield of 1779 Mb.

Inserts with sizes from 20 nt to 25 nt were selected for bioinformatic analyses. Sequencing adapter removal was done using Trimmomatics software (45), and the reads were de novo assembled to contigs using VirusDetect software (46). VirusDetect software conducts BLASTX and BLASTN searches against curated plant virus database (vrl_Plants_248_U100) of VirusDetect for each sample separately. We used default parameters BLASTX and BLASTN searches, default similarity 25 % and p-value 1e-5. We then assigned the obtained contigs to virus family level for the statistical analyses (Supplementary table 2).

Statistical analysis

All statistical analyses were conducted in R (version 4.2.2; (47). To test whether local adaptation influenced host infection status (infected by any studied virus= 1, not infected by any studied virus = 0), we fitted generalized linear mixed models (GLMM) using the "glmmTMB" R-package (48) with binomial distribution and logit link function. Specifically, we constructed GLMMs to test the two key metrics of local adaptation: i) local *vs.* foreign and ii) home *vs.* away (49,50). For the local *vs.* foreign model (LF), a categorical variable representing sympatry or allopatry, nested within transplant population, was included as a fixed effect. Seed origin population and plant size were included as additional fixed effects and maternal line nested within seed origin population was included as a random effect to account for genetic variation among hosts. For the home *vs.* away model (HA), the model structure was identical, except that the categorical variable of sympatry or allopatry was nested within seed origin population and included as a fixed effect. Model assumptions were assessed using R-package “DHARMa” (51). The significance of the main effects were evaluated using Wald X^2 tests (function “Anova” in R-package “car”; (52). For significant effects, pairwise comparisons of the estimated marginal means were performed using functions “contrasts” and “emmeans”

187 from the R-package “emmeans” (version 1.8.8; (53), applying Tukey’s method for multiple
188 comparisons.

189 To investigate the effects of local adaptation, population and host characteristics on
190 within-host viral diversity, while also accounting for viral (co-)occurrence patterns in the
191 transplant experiment, we implemented Joint Species Distribution Modelling (JSDM) using
192 the hierarchical modelling of species communities (HMSC) framework (54,55). HMSC is a
193 hierarchical generalized linear mixed model with Bayesian inference and allows the analysis
194 of multiple species’ responses to ecological variables while incorporating species- and
195 community-level parameters and accounting for covariation among species. Th response
196 variables in our HMSC model were the occurrences of the three most prevalent virus families:
197 *Caulimoviridae*, *Partitiviridae* and *Pospivirodae*. As fixed effect predictors, we included 1)
198 maternal line ID, 2) seed origin population, 3) sympatry/allopatry, 4) plant size, and 5) signs
199 of herbivory. Transplant population was included as a random effect. Including
200 sympatry/allopatry as a fixed effect allowed us to directly estimate the effect of local adaptation
201 in our model. We used four separate Markov chain Monte Carlo (MCMC) chains to sample the
202 posterior distribution. Each chain was run for 1 875 000 iterations, and the first 625 000 were
203 discarded as burn-in. Subsequently, the remaining iterations were thinned by 5000, resulting in
204 250 posterior samples per chain. Finally, we obtained a total of 1000 posterior samples across
205 all four chains. The model fit was evaluated by examining explanatory and predictive
206 performance via ten-fold cross-validation, using Tjur’s coefficient of determination (Tjur R^2)
207 and area under the curve (AUC), respectively. The HMSC analyses were ran using the R-
208 package “Hmsc” (version 3.0-14).

209

210

Results

Description of the sRNA sequencing data

From the 211 sequenced individuals, the sRNA sequencing yielded on average 23,799,485 reads per plant tissue sample (min 17,364,260; max 49,152,805; SD 7,738,962). The VirusDetect pipeline assembled 2374 contigs ranging from 41 to 2080 nt in length (mean length of 159 nt and SD 163 nt). Of these, 11% of contigs had virus-specific BLASTN hits with 80–100% identity (mean 93%), while 89% had BLASTX hits with 22–100% identity (mean 67%).

In total, we assembled 1151 plant virus-associated contigs across the 211 individuals, representing six plant virus families: *Tymoviridae, Botourmiaviridae, Closteroviridae, Partitiviridae, Caulimoviridae* and *Pospiviroidae* (Figure 2C, Supplementary table 2). From each family, we identified 1 to 3 virus genera and 3 to 842 contigs for each genus. At the species level, we acquired BLAST hits to 1 to 15 species, depending on the virus genus (Supplementary table 2). Overall, 26% of the host individuals were infected, and of those 86% were colonized by one virus family and 14% by two virus families. The most prevalent families were *Caulimoviridae* and *Pospiviroidae* (both in 43% of the infected individuals), whereas *Tymoviridae* and *Botourmiaviridae* were the rarest (both in 2% of the infected individuals; Figure 2C).

Analysis of viral local adaptation: local *vs.* foreign

Using the local *vs.* foreign criterion, we observed indications of viral local adaptation in transplant population 876, where local hosts had higher infection rates than foreign hosts. A similar trend was observed in population 950, where local hosts showed the second-highest infection rates (Figure 2A). Conversely, in population 9205, local hosts harboured fewer infections than foreign hosts – suggesting viral maladaptation. However, the GLMM (LF) did

not provide statistical support for these trends (Wald X^2 = 1.53, P = 0.673; Table 1). Seed origin populations differed significantly in infection rates (Wald X^2 = 7.37, P = 0.025; Table 1), with individuals originating from population 9205 having significantly fewer infections than those originating from population 876 (Figure 2A, Supplementary table 3A; estimate = 2.071, SE = 0.778, z-ratio = 2.661, P = 0.021).

Analysis of viral local adaptation: home *vs.* away

Applying the home *vs.* away criterion, we found no evidence of viral local adaptation (Figure 2B). Hosts from populations 876 and 950 had lower infection rates in their respective home populations than in their away populations, suggesting viral maladaptation (Figure 2B). Our statistical analysis (model HA) did not detect significant differences in infection rates between home and away habitats. However, model coefficients for the "sympatry" term nested within seed origin population were lower, suggesting higher infection rates in away habitats (Supplementary table 4). Additionally, seed origin population significantly influenced host infection status (Wald X^2 = 9.09, P=0.010; Table 2). Post hoc comparisons showed that individuals from population 876 had significantly higher infection rates than those from population 9205 (estimate = 1.818, SE = 0.649, z-ratio = 2.802, P = 0.014; Supplementary table 3B., Figure 2B and C).

Analysis of viral (co-)occurrence patterns

We applied the HMSC approach to investigate the factors influencing the (co-)occurrence of the detected virus families in a local adaptation experiment. The model predicted virus family occurrences well, although model performance varied among virus families (Supplementary table 5). Tjur R^2 and AUC were used to quantify the explanatory and predictive performance of the model, with a mean Tjur R^2 of 0.27 (range among the detected virus families 0.10-0.47) and a mean AUC of 0.89 (0.80-0.98). The predictive power of the model was based on ten-fold

cross-validations, where the mean Tjur R^2 was 0.19 (range 0.01 - 0.4) and the mean AUC was 0.74 (range 0.54-0.88; Supplementary table 5) varying among virus families.

In terms of contributions to the explained variation in our HMSC model, host maternal line was the strongest determinant of viral occurrences, explaining on average 62% of the variance. However, the effect varied among virus families and was most pronounced for *Pospiviroidae* (73%) and less important in explaining *Partitiviridae* (60%) and *Caulimoviridae* 54%) occurrences (Figure 3, Supplementary table 6). For example, maternal line 876-6, displayed the highest infection rates, with 89% of the individuals infected (Figure 2C). Seed origin population was the second most important predictor, explaining on average of 29% of the variance. The effect of host maternal line varied among virus families, with a more pronounced role for *Caulimoviridae* (35%) and *Partitiviridae* (33%), while being less important for explaining the occurrences of *Pospiviroidae* (18%; Figure 3, Supplementary table 6). Consistent with this, individuals from seed origin population 876 harboured 50% of all detected viral infections, whereas individuals originating from population 9205 harboured only 16% of all infections (Figure 2C).

Host plant size accounted for an average of 4% of the variation in viral occurrences, with the strongest effect observed for *Caulimoviridae* (7%). Local adaptation (sympatry/allopatry) had a smaller role in contributing to explained variation, accounting for 2% on average across virus families (Figure 3, Supplementary table 6). Herbivory had minimal effect, explaining only 0% to 0.1% of the viral occurrences. The random effect of transplant population explained on average 2% of the variation across virus families and was slightly more important in explaining *Pospiviroidae* occurrences (4%, Figure 3, Supplementary table 6). Residual correlations among virus families at the random level were not significant, suggesting that after accounting for the effects of the fixed explanatory variables, viral occurrences were not influenced by interactions between virus families.

Discussion

Here, we used a reciprocal transplant experiment combined with sRNA sequencing and JSDM modelling to investigate the role of local adaptation in shaping within-host viral (co-)occurrences. Although we observed trends suggesting viral local adaptation and maladaptation when applying the local *vs.* foreign and home *vs*. away criteria, the effects were not statistically significant. Instead, we found host maternal line and host seed origin population to be the most important determinants of host infection status and viral community structure. The strength of these effects varied across virus families, indicating virus-specific assembly processes and variation in the extent to which coevolution shapes these interactions. Jointly our results identify key drivers of viral community assembly and provide insight into how within-host dynamics could scale up to predict the ecological and evolutionary consequences of disease in natural systems.

Using sRNA sequencing, we detected viruses from six virus families, five of which have been previously identified from this system (20,43). Overall, 21% of the sampled sentinel plants were infected, exhibiting a lower infection rate than previously reported from hosts in this system (20,43). Despite the low overall infection prevalence, we found individuals originating from population 876 harbouring significantly higher infection rates than those from population 9205. Viral community composition also varied among seed origin populations and among maternal lines. Individuals from seed origin population 876 harboured viruses from five different virus families (*Caulimoviridae*, *Pospiviroidae*, *Partitiviridae*, *Botourmiaviridae* and *Closteroviridae*), whereas individuals from population 950 were infected by only three virus families (*Pospiviroidae*, *Caulimoviridae* and *Partitiviridae*). The overall lower infection prevalence may be due to differences in exposure time to viral epidemics and additionally, viral prevalence may vary annually due to several factors, such as temperature, humidity and vector

behaviour — components of natural systems that are difficult to control in a field experiment (56–58).

Using a reciprocal transplant experimental approach, we were able to apply the two key metrics of local adaptation: local *vs.* foreign and home *vs.* away. While we observed signs of viral local adaptation in transplant population 876 under the local *vs*. away criterion, the pattern was not statistically significant (GLMM LF). Similarly, analysis on the home *vs.* away metric showed no statistically significant effect of local adaptation on host infection status (GLMM HA). In line with these results, when investigating the effects of local adaptation on viral (co-)occurrence patterns with JSDM in the HMSC framework, we found local adaptation to explain on average only 2.3% of the viral occurrences. However, when using the home *vs.* away criterion (GLMM HA), individuals from seed origin populations 876 and 950 harboured the lowest infection rates in their home populations, suggesting viral maladaptation. Patterns of maladaptation are not unexpected given the dynamic, cyclic nature of coevolutionary interactions between the host and its parasite (59). In the Aland Islands *P. lanceolata* populations are highly fragmented, and the connectivity levels of the populations vary (60,61) and consequently too high or low gene flow between populations could facilitate parasite maladaptation (59,62–64). Previous studies have shown that well-connected host populations are less affected by disease (65,66), a phenomenon that is likely due to higher resistance diversity in these populations maintained by gene flow (61).

Seed origin population was a strong predictor of host infection status. Individuals originating from population 876 were more frequently infected and harboured the most diverse viral communities. In contrast, hosts from population 9205 exhibited high resistance to viral infection and consequently harboured less complex viral communities. Our HMSC analysis mirrored these findings, identifying maternal line and seed origin population as the strongest determinants of viral occurrence across virus families, explaining on average 62% and 29% of

the variation, respectively (Figure 3). The variation in infection rates among host origin populations, together with the strong maternal line effects for viral occurrences across virus families, highlights host genetic diversity as a key driver of viral community assembly and composition in this system. Although evidence for viral local adaptation was limited, the variation in infections prevalence among host maternal lines indicates strong potential for coevolution, as genetic variation is a main driver of coevolution (67–69). Moreover, high host genetic diversity in natural populations can mitigate disease risk, a phenomenon known as the monoculture effect (70,71).

Hosts encounter a myriad of parasites throughout their lives (43,72–74), and these interactions can have far-reaching consequences for host-parasite coevolution and population dynamics (75). Despite this, much of the research on local adaptation has focused on pairwise host-parasite interactions (76–78), with little focus on the role of parasite communities in coevolutionary processes. To our knowledge, our study is among the first to study viral local adaptation within a community ecology framework. After accounting for host attributes, we found no evidence of virus-virus interactions shaping within-host viral diversity. Instead, host characteristics, represented by maternal line and host seed origin population, emerged as the most important predictor of viral community structure and host infection status. Our findings highlight the importance of host genetic variation in shaping viral communities and contribute to the growing field of viral community ecology research. Understanding the drivers of complex host-parasite interactions and processes at the community level is essential for predicting how disease dynamics scale up from individuals to populations and understanding the ecological and evolutionary conditions from which novel viral diseases may emerge.

Acknowledgements

We thank Krista Raveala, Aura Palonen, Suvi Sallinen, Marijke Iso-Kokkila and Jere Lentonen for the help with the transplant experiment and RNA extractions. We thank Suvi Sallinen for the help with statistical analysis. The CSC – IT Center for Science, Finland, is acknowledged for computational resources. The work was funded by grants from the European Research Council (AdG 101097545 Co-EvoChange), and Academy of Finland (334276, 362242) to A.-L.L. and Academy of Finland (321441) to H.S.

Author contributions

M.J., H.S. and A.-L.L. designed the study. M.J. performed the field experiment, data collection, and statistical analysis. M.J., H.S. and A-L.L. prepared the manuscript.

Conflict of Interest

The authors declare no conflict of interests.

Data availability Statement

The data and R scripts used in this study have been submitted to GitHub (https://github.com/maijajoki/ViRAL21).

References

1. Buckingham LJ, Ashby B. Coevolutionary theory of hosts and parasites. J Evol Biol. 2022;35(2):205–24.
2. Thompson JN, Cunningham BM. Geographic structure and dynamics of coevolutionary selection. Nature. 2002;417(6890):735–8.

3. Decaestecker E, Gaba S, Raeymaekers JAM, Stoks R, Kerckhoven LV, Ebert D, et al. Host-parasite 'Red Queen' dynamics archived in pond sediment. Nature. 2007;450(7171):870–3.

4. Greischar MA, Koskella B. A synthesis of experimental work on parasite local adaptation. Ecol Lett. 2007;10(5):418–34.

5. Kaltz O, Shykoff JA. Local adaptation in host–parasite systems. Hered 1998 814. 1998 Oct 1;81(4):361–70.

6. Dybdahl MF, Storfer A. Parasite local adaptation: Red Queen versus Suicide King. Trends Ecol Evol. 2003 Oct 1;18(10):523–30.

7. Johnson PTJ, Hoverman JT. Parasite diversity and coinfection determine pathogen infection success and host fitness. Proc Natl Acad Sci U S A. 2012 June 5;109(23):9006–11.

8. Ben-Ami F, Mouton L, Ebert D. The effects of multiple infections on the expression and evolution of virulence in a Daphnia-endoparasite system. Evolution. 2008;62(7):1700–11.

9. Tollenaere C, Susi H, Nokso-Koivisto J, Koskinen P, Tack A, Auvinen P, et al. SNP Design from 454 Sequencing of Podosphaera plantaginis Transcriptome Reveals a Genetically Diverse Pathogen Metapopulation with High Levels of Mixed-Genotype Infection. PLoS ONE. 2012 Dec 27;7(12).

10. Griffiths EC, Pedersen AB, Fenton A, Petchey OL. The nature and consequences of coinfection in humans. J Infect. 2011 Sept;63(3):200–6.

11. Betts A, Gray C, Zelek M, MacLean RC, King KC. High parasite diversity accelerates host adaptation and diversification. Science. 2018;360(6391):907–11.

12. Hall AR, Ashby B, Bascompte J, King KC. Measuring Coevolutionary Dynamics in Species-Rich Communities. Trends Ecol Evol. 2020 June 1;35(6):539–50.

13. Hily JM, Poulicard N, Mora MÁ, Pagán I, García-Arenal F. Environment and host genotype determine the outcome of a plant–virus interaction: from antagonism to mutualism. New Phytol. 2016 Jan 1;209(2):812–22.

14. Sallinen S, Norberg A, Susi H, Laine AL. Intraspecific host variation plays a key role in virus community assembly. Nat Commun. 2020;11(1):1–11.

15. Gandon S. Local adaptation and the geometry of host-parasite coevolution. Ecol Lett. 2002;5(2):246–56.

16. Woolhouse MEJ, Webster JP, Domingo E, Charlesworth B, Levin BR. Biological and biomedical implications of the co-evolution of pathogens and their hosts. Nat Genet. 2002;32(4):569–77.

17. Tollenaere C, Susi H, Laine AL. Evolutionary and Epidemiological Implications of Multiple Infection in Plants. Trends Plant Sci. 2016 Jan 1;21(1):80–90.

18. Karvonen A, Fenton A, Sundberg LR. Sequential infection can decrease virulence in a fish–bacterium–fluke interaction: Implications for aquaculture disease management. Evol Appl. 2019 Dec 1;12(10):1900–11.

19. Jokinen M, Sallinen S, Jones MM, Sirén J, Guilbault E, Susi H, et al. The first arriving virus shapes within-host viral diversity during natural epidemics. Proc R Soc B [Internet]. 2023 Sept 13 [cited 2023 Nov 30];290(2006). Available from: https://royalsocietypublishing.org/doi/10.1098/rspb.2023.1486

20. Norberg A, Susi H, Sallinen S, Baran P, Clark NJ, Laine AL. Direct and indirect viral associations predict coexistence in wild plant virus communities. Curr Biol. 2023 May 8;33(9):1665-1676.e4.

21. Tollenaere C, Susi H, Laine AL. Evolutionary and Epidemiological Implications of Multiple Infection in Plants. Trends Plant Sci. 2016 Jan 1;21(1):80–90.

22. Gorter FA, Manhart M, Ackermann M. Understanding the evolution of interspecies interactions in microbial communities. Philos Trans R Soc B [Internet]. 2020 May 11 [cited 2024 Oct 14];375(1798). Available from: https://royalsocietypublishing.org/doi/10.1098/rstb.2019.0256

23. Urban MC, De Meester L. Community monopolization: local adaptation enhances priority effects in an evolving metacommunity. Proc R Soc B Biol Sci. 2009 Dec 7;276(1676):4129–38.

24. Nadeau CP, Farkas TE, Makkay AM, Papke RT, Urban MC. Adaptation reduces competitive dominance and alters community assembly. Proc R Soc B [Internet]. 2021 Feb 24 [cited 2023 Apr 14];288(1945). Available from: https://royalsocietypublishing.org/doi/10.1098/rspb.2020.3133

25. Pantel JH, Duvivier C, Meester LD. Rapid local adaptation mediates zooplankton community assembly in experimental mesocosms. Ecol Lett. 2015;18(10):992–1000.

26. Gómez P, Paterson S, De Meester L, Liu X, Lenzi L, Sharma MD, et al. Local adaptation of a bacterium is as important as its presence in structuring a natural microbial community. Nat Commun [Internet]. 2016 [cited 2024 Oct 11]; Available from: www.nature.com/naturecommunications

27. Breitbart M, Hewson I, Felts B, Mahaffy JM, Nulton J, Salamon P, et al. Metagenomic analyses of an uncultured viral community from human feces. J Bacteriol. 2003 Oct;185(20):6220–3.

28. Brum JR, Ignacio-espinoza JC, Roux S, Doulcier G, Acinas SG, Alberti A, et al. Ocean Viral Communities. Science. 2015;348(6237):1261411–98.

29. Durham DM, Sieradzki ET, Ter Horst AM, Santos-Medellín C, Winston C, Bess A, et al. Substantial differences in soil viral community composition within and among four Northern California habitats. ISME Commun 2022 21. 2022 Oct 13;2(1):1–5.

30. Pratama AA, van Elsas JD. The 'Neglected' Soil Virome – Potential Role and Impact. Trends Microbiol. 2018;26(8):649–62.

31. Rwahnih MA, Daubert S, Úrbez-Torres JR, Cordero F, Rowhani A. Deep sequencing evidence from single grapevine plants reveals a virome dominated by mycoviruses. Arch Virol. 2011;156(3):397–403.

32. Yang K, Wang X, Hou R, Lu C, Fan Z, Li J, et al. Rhizosphere phage communities drive soil suppressiveness to bacterial wilt disease. Microbiome. 2023;11(1):1–18.

33. Christiaansen A, Varga SM, Spencer JV. Viral manipulation of the host immune response. Curr Opin Immunol. 2015;36:54.

34. Coloma S, Gaedke U, Sivonen K, Hiltunen T. Frequency of virus-resistant hosts determines experimental community dynamics. Ecology. 2019;100(1):1–10.

35. Roossinck MJ, Bazán ER. Symbiosis: Viruses as Intimate Partners. Annu Rev Virol. 2017 Sept 29;4:123–39.

36. Norberg A, Abrego N, Blanchet FG, Adler FR, Anderson BJ, Anttila J, et al. A comprehensive evaluation of predictive performance of 33 species distribution models at species and community levels. Ecol Monogr. 2019 Aug 1;89(3):e01370.

37. Sagar GR, Harper JL. Plantago Major L., P. Media L. and P. Lanceolata L. J Ecol. 1964 Mar;52(1):189.

38. Ojanen SP, Nieminen M, Meyke E, Pöyry J, Hanski I. Long-term metapopulation study of the Glanville fritillary butterfly (Melitaea cinxia): Survey methods, data management, and long-term population trends. Ecol Evol. 2013 Oct 1;3(11):3713–37.

39. Susi H, Laine AL, Filloux D, Kraberger S, Farkas K, Bernardo P, et al. Genome sequences of a capulavirus infecting Plantago lanceolata in the Åland archipelago of Finland. Arch Virol. 2017 July 1;162(7):2041–5.

40. Susi H, Filloux D, Frilander MJ, Roumagnac P, Laine A liisa. Diverse and variable virus communities in wild plant populations revealed by metagenomic tools. 2019;

41. Adams IP, Skelton A, Macarthur R, Hodges T, Hinds H, Flint L, et al. Carrot yellow leaf virus is associated with carrot internal necrosis. PLoS ONE. 2014 Nov 3;9(11).

42. Biswas KK, Bhattacharyya UK, Palchoudhury S, Balram N, Kumar A, Arora R, et al. Dominance of recombinant cotton leaf curl Multan-Rajasthan virus associated with cotton leaf curl disease outbreak in northwest India. PLOS ONE. 2020 Apr 1;15(4):e0231886.

43. Susi H, Filloux D, Frilander MJ, Roumagnac P, Laine AL. Diverse and variable virus communities in wild plant populations revealed by metagenomic tools. PeerJ [Internet]. 2019 [cited 2020 Feb 19];2019(1). Available from: http://doi.org/10.7717/peerj.6140

44. Chang S, Puryear J, Cairney J. A simple and efficient method for isolating RNA from pine trees. Plant Mol Biol Report. 1993;11(2):113–6.

45. Bolger AM, Lohse M, Usadel B. Genome analysis Trimmomatic: a flexible trimmer for Illumina sequence data. 2014;30(15):2114–20.

46. Zheng Y, Gao S, Padmanabhan C, Li R, Galvez M, Gutierrez D, et al. VirusDetect: An automated pipeline for efficient virus discovery using deep sequencing of small RNAs. Virology. 2017 Jan 1;500:130–8.

47. R Foundation for Statistical, Computing, Vienna A. R: A language and environment for statistical computing. 2022; Available from: https://www.r-project.org/

48. Brooks ME, Kristensen K, Van Benthem KJ, Magnusson A, Berg CW, Nielsen A, et al. glmmTMB Balances Speed and Flexibility Among Packages for Zero-inflated Generalized Linear Mixed Modeling. R J. 2017;9:2.

49. Kawecki TJ, Ebert D. Conceptual issues in local adaptation. Ecol Lett. 2004 Dec 1;7(12):1225–41.

50. Blanquart F, Kaltz O, Nuismer SL, Gandon S. A practical guide to measuring local adaptation. Ecol Lett. 2013 Sept;16(9):1195–205.

51. Hartig F. DHARMa: Residual Diagnostics for Hierarchical (Multi-Level / Mixed) Regression Models. 2022; Available from: https://cran.r-project.org/package=DHARMa

52. Fox J, Weisberg S. An R Companion to Applied Regression. Sage Publ. 2019;

53. Russell V. Lenth RV, BB [ctb], PB [ctb], IGV [ctb], MH [ctb], MJ [ctb], JL [ctb], FM [ctb], JP [ctb], HR [ctb], SH. Package “Emmeans”. 2018;

54. Ovaskainen O, Tikhonov G, Norberg A, Guillaume Blanchet F, Duan L, Dunson D, et al. How to make more out of community data? A conceptual framework and its implementation as models and software. Ecol Lett. 2017 May 1;20(5):561–76.

55. Ovaskainen O, Abrego N. Joint Species Distribution Modelling [Internet]. Cambridge University Press; 2020 [cited 2022 Oct 17]. Available from: https://www.cambridge.org/core/product/identifier/9781108591720/type/book

56. Trebicki P. Climate change and plant virus epidemiology. Virus Res. 2020;286(June):198059.

57. Kendig AE, Borer ET, Mitchell CE, Power AG, Seabloom EW. Characteristics and drivers of plant virus community spatial patterns in US west coast grasslands. Oikos. 2017 Sept 1;126(9):1281–90.

58. Jeger MJ, Fereres A, Malmstrom CE, Mauck KE, Wintermantel WM. Epidemiology and Management of Plant Viruses Under a Changing Climate. Phytopathology. 2023 Nov 4;113(9):1620–1.

59. Kaltz O, Gandon S, Michalakis Y, Shykoff JA. Local maladaptation in the anther-smut fungus Microbotryum violaceum to its host plant Silene latifolia: Evidence from a cross-inoculation experiment. Evolution. 1999;53(2):395–407.

60. Jousimo J, Tack AJM, Ovaskainen O, Mononen T, Susi H, Tollenaere C, et al. Ecological and evolutionary effects of fragmentation on infectious disease dynamics. Science. 2014;344(6189):1289–93.

526 61. Höckerstedt L, Siren JP, Laine AL. Effect of spatial connectivity on host resistance in a
527 highly fragmented natural pathosystem. J Evol Biol. 2018;31(6):844–52.

528 62. Gandon S. Local adaptation and the geometry of host-parasite coevolution. Ecol Lett.
529 2002;5(2):246–56.

530 63. Hoeksema JD, Forde SE. A meta-analysis of factors affecting local adaptation between
531 interacting species. Am Nat. 2008 Mar;171(3):275–90.

532 64. Oppliger A, Vernet R, Baezà M. Parasite local maladaptation in the Canarian lizard
533 Gallotia galloti (Reptilia: Lacertidae) parasitized by haemogregarian blood parasite. J Evol
534 Biol. 1999;12(5):951–5.

535 65. Höckerstedt L, Numminen E, Ashby B, Boots M, Norberg A, Laine AL. Spatially
536 structured eco-evolutionary dynamics in a host-pathogen interaction render isolated
537 populations vulnerable to disease. Nat Commun. 2022;13(1):1–11.

538 66. Carlsson-Granér U, Thrall PH. Host resistance and pathogen infectivity in host populations
539 with varying connectivity. Evolution. 2015 Apr 1;69(4):926–38.

540 67. Paterson S, Vogwill T, Buckling A, Benmayor R, Spiers AJ, Thomson NR, et al.
541 Antagonistic coevolution accelerates molecular evolution. Nature. 2010 Mar
542 11;464(7286):275–8.

543 68. Buckingham LJ, Ashby B. Coevolutionary theory of hosts and parasites. J Evol Biol. 2022
544 Feb 1;35(2):205–24.

545 69. Laine AL, Burdon JJ, Dodds PN, Thrall PH. Spatial variation in disease resistance: From
546 molecules to metapopulations. J Ecol. 2011;99(1):96–112.

547 70. Laine AL, Burdon JJ, Dodds PN, Thrall PH. Spatial variation in disease resistance: From
548 molecules to metapopulations. J Ecol. 2011 Jan;99(1):96–112.

549 71. Ridenhour BJ, Nuismer SL. Polygenic traits and parasite local adaptation. Evolution.
550 2007;61(2):368–76.

551 72. Vayssier-Taussat M, Kazimirova M, Hubalek Z, Hornok S, Farkas R, Cosson JF, et al.
552 Emerging horizons for tick-borne pathogens: From the ‘one pathogen-one disease’ vision
553 to the pathobiome paradigm. Future Microbiol. 2015 Dec 1;10(12):2033–43.

554 73. Susi H, Barrès B, Vale PF, Laine AL. Co-infection alters population dynamics of infectious
555 disease. Nat Commun. 2015 Jan 8;6(1):1–8.

556 74. Boots M, White A, Best A, Bowers R. The importance of who infects whom: The evolution
557 of diversity in host resistance to infectious disease. Ecol Lett. 2012;15(10):1104–11.

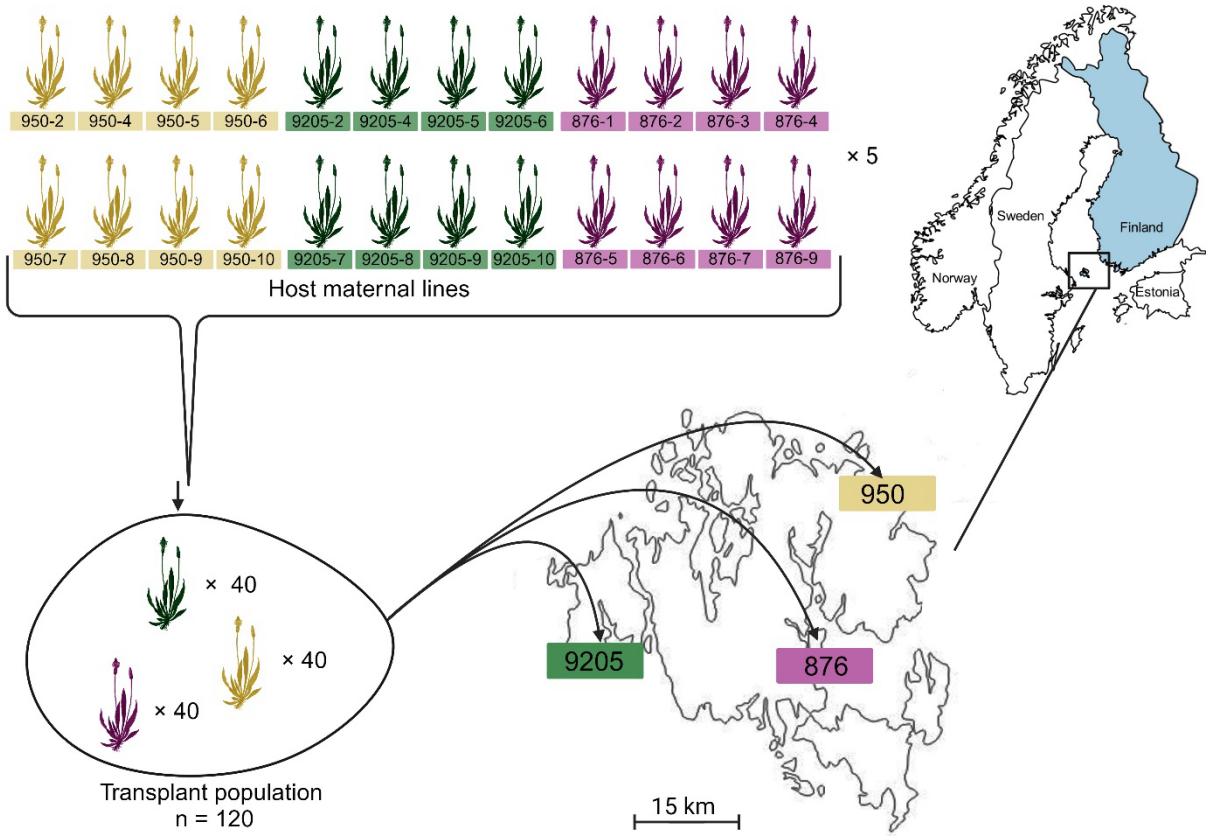
558 75. Hall AR, Ashby B, Bascompte J, King KC. Measuring Coevolutionary Dynamics in
559 Species-Rich Communities. Trends Ecol Evol. 2020;35(6):539–50.

560 76. Ridenhour BJ, Nuismer SL. Polygenic traits and parasite local adaptation. Evolution. 2007
561 Feb;61(2):368–76.

562 77. Laine AL. Detecting local adaptation in a natural plant-pathogen metapopulation: A
563 laboratory vs. field transplant approach. J Evol Biol. 2007 Sept;20(5):1665–73.

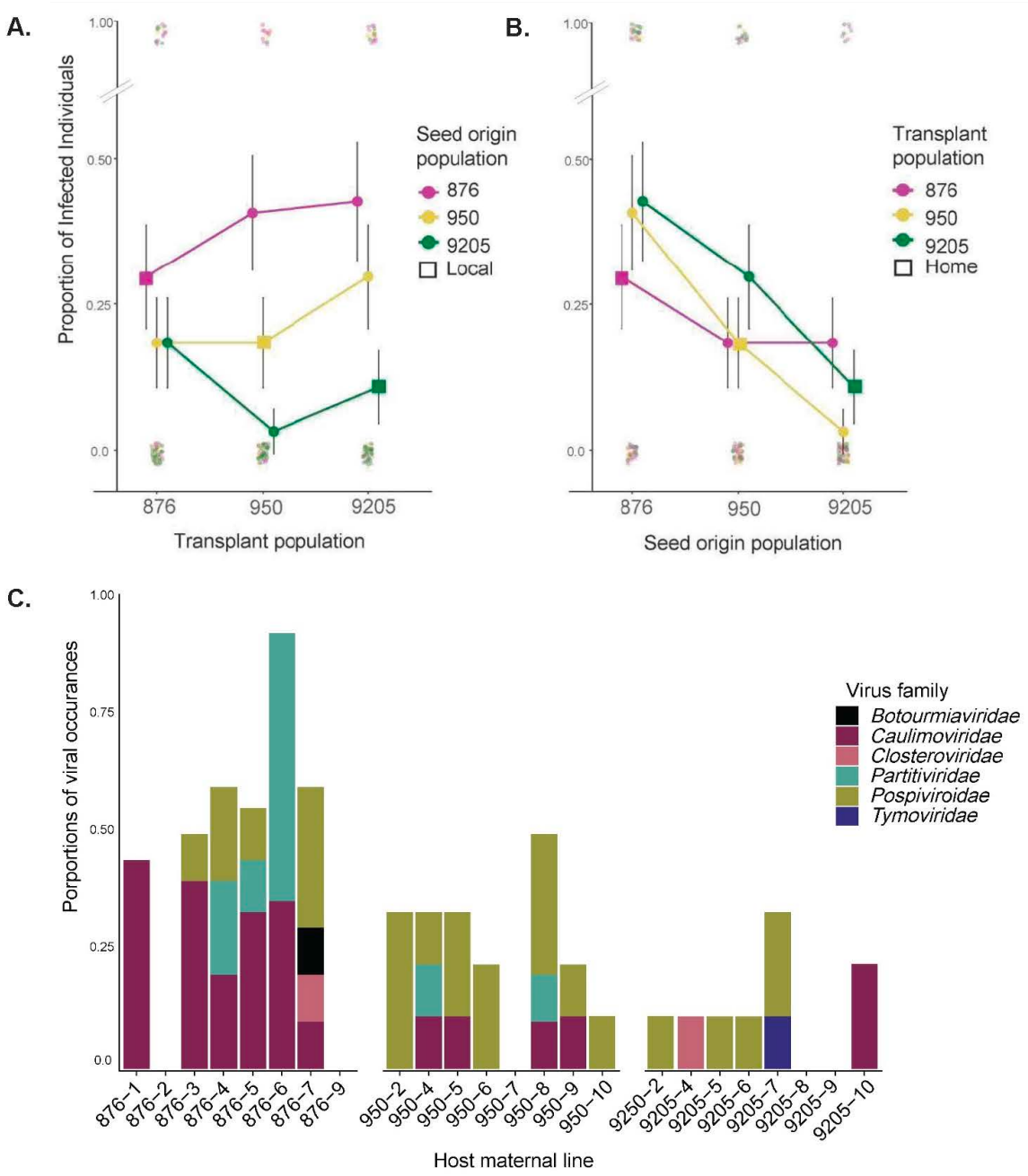
564 78. Mauck KE, De Moraes CM, Mescher MC. Evidence of Local Adaptation in Plant Virus
565 Effects on Host–Vector Interactions. Integr Comp Biol. 2014 July 1;54(2):193–209.

566



567

568 Figure 1. Reciprocal transplant experiment where *Plantago lanceolata* individuals from 24
569 maternal lines originating from three populations were transplanted into their sympatric and
570 two allopatric populations during natural viral epidemics. We placed 40 individuals into
571 sympatric population and ~80 individuals into two allopatric populations, with a total of 348
572 plant individuals across three populations in the Åland Islands SW Finland.



573

574 Figure 2. Proportions of virus infected *Plantago lanceolata* host individuals in a reciprocal
575 transplant experiment using (A) local vs. foreign, (B) home vs. away metrics of local
576 adaptation, and (C) infection pattern across host maternal (n =24) line grouped by seed origin
577 population. In panel A, colours indicate the seed origin populations and the squares mark the
578 local host. In panel B, the colours represent the transplant populations and the squares mark
579 the home habitat of the host (purple = seed origin/transplant population 876 yellow = seed
580 origin/transplant population 950, and green = seed origin/transplant population 9205). In panel
581 C colours represent the six virus families detected with sRNA sequencing.

582

583

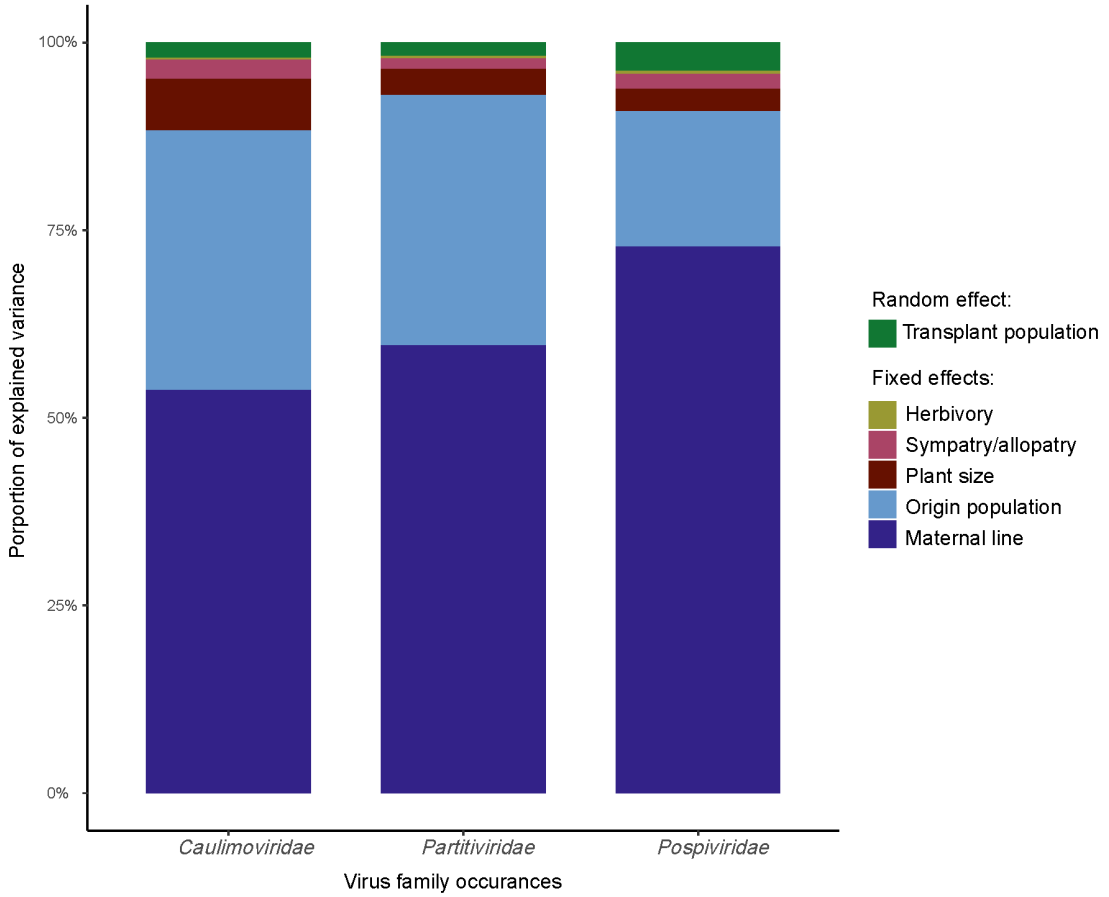
Table 1. Type II Wald X^2 test for Generalizer linear mixed model estimating the effects of plant size, seed origin population, transplant population and local *vs.* foreign metric of local adaptation on host infection status (1=infected, 0=uninfected) in a reciprocal transplant experiment in the Åland Islands (model LF).

Fixed effect	*Wald* X^2	*Df*	*p-value*
Plant size	1.71	1	0.190
Seed origin population	**7.37**	**2**	**0.025**
Transplant population	1.99	2	0.369
Transplant population : sympatry/allopatry	1.53	3	0.673

Table 2. Type II Wald X^2 test for Generalizer linear mixed model testing for the effects of plant size, seed origin population, transplant population and home *vs.* away metric of local adaptation on host infection status (1=infected, 0=uninfected) in a reciprocal transplant experiment in the Åland Islands (model HA).

Fixed effect	*Wald* X^2	*Df*	*p-value*
Plant size	1.71	1	0.190
Seed origin population	**9.09**	**2**	**0.010**
Transplant population	1.64	2	0.438
Seed origin population : sympatry/allopatry	1.53	3	0.673

Figure 3. Variance partitioning of the fixed and random effects in the Hierarchical Modelling of Species Communities model for the three most prevalent virus families (*Caulimoviridae*, *Partitiviridae*, *Pospiviridae*) in the reciprocal transplant experiment. The six variables explaining the occurrences the three virus families were: maternal line, seed origin population, plant size, sympatry/allopatry, herbivory and transplant population (random effect).



605
606
607
608
609
610
611
612
613
614
615
616
617
618
619
620

621 **Supplement**

622

623 **Supplementary table 1.** The host maternal lines included in a reciprocal transplant
624 experiment studying viral local adaptation in *Plantago lanceolata* host populations in the Åland
625 Islands during naturally occurring viral epidemics. In the table are included the ID of each
626 maternal line, the ID of the origin population of each maternal line, the ID of the transplant
627 population where the plants were placed during the experiment and the number of individuals,
628 and finally, the number of sequenced individuals.

Maternal line	Seed origin population	Transplant population	No individuals in the experiment	Number of individuals sequenced
876-1	876	876	5	3
876-1	876	950	5	3
876-1	876	9205	5	3
876-2	876	876	5	3
876-2	876	950	5	3
876-2	876	9205	5	3
876-3	876	876	5	3
876-3	876	950	5	3
876-3	876	9205	5	3
876-4	876	876	5	3
876-4	876	950	1	1
876-4	876	9205	0	0
876-5	876	876	5	3
876-5	876	950	4	3
876-5	876	9205	3	3
876-6	876	876	5	3
876-6	876	950	5	3
876-6	876	9205	5	3
876-7	876	876	5	3
876-7	876	950	5	3
876-7	876	9205	5	3
876-9	876	876	5	3
876-9	876	950	5	3
876-9	876	9205	5	3
950-10	950	876	5	3
950-10	950	950	5	3
950-10	950	9205	5	3
950-2	950	876	5	3
950-2	950	950	5	3
950-2	950	9205	5	3
950-4	950	876	5	3
950-4	950	950	5	3
950-4	950	9205	5	3
950-5	950	876	5	3
950-5	950	950	5	3
950-5	950	9205	5	3
950-6	950	876	5	3
950-6	950	950	5	3
950-6	950	9205	5	3
950-7	950	876	5	3
950-7	950	950	5	3
950-7	950	9205	5	3
950-8	950	876	5	3
950-8	950	950	5	3
950-8	950	9205	5	3
950-9	950	876	5	3
950-9	950	950	5	3
950-9	950	9205	5	3

9205-10	9205	876	5	3
9205-10	9205	950	5	3
9205-10	9205	9205	5	3
9205-4	9205	876	5	3
9205-4	9205	950	5	3
9205-4	9205	9205	5	3
9205-5	9205	876	5	3
9205-5	9205	950	5	3
9205-5	9205	9205	5	3
9205-6	9205	876	5	3
9205-6	9205	950	5	3
9205-6	9205	9205	5	3
9205-7	9205	876	5	3
9205-7	9205	950	5	3
9205-7	9205	9205	5	3
9205-8	9205	876	4	3
9205-8	9205	950	5	3
9205-8	9205	9205	5	3
9205-9	9205	876	5	3
9205-9	9205	950	5	3
9205-9	9205	9205	5	3
9250-2	9205	876	5	3
9250-2	9205	950	5	3
9250-2	9205	9205	5	3

635

636

637

638

639

640

641

642

643

644 **Supplementary table 2.** Virus families detected by small-RNA sequencing on
645 *Plantago lanceolata* individuals (n = 211) included in a transplant experiment in the Åland
646 Islands studying viral local adaptation. The genera belonging to each virus family are shown
647 as well as the number of contigs and virus species within each virus family. Reference to the
648 literature describing the detected family listed in the "reference" column [1–3].

Family	Genus	Contigs	Species	References
Botourmiaviridae		3	1	
	Gammascleroulivirus	3	1	
Caulimoviridae		896	21	[1,2,3]
	Badnavirus	1	1	
	Caulimovirus	842	15	
	Soymovirus	52	5	
Closteroviridae		13	5	[1,2,3]
	Ampelovirus	1	1	
	Closterovirus	11	3	
	unclassified	1	1	
Partitiviridae		212	11	[1,2,3]
	Betapartitivirus	113	8	
	unclassified	99	3	
Pospiviridae		24	1	[3]
	Pospiviroid	24	1	
Tymoviridae		3	1	[1,2,3]
	Maculavirus	3	1	

649

Supplementary table 3. Post hoc test comparing the infection status of the host *Plantago lanceolata* from the three seed origin populations in a local adaptation experiment in the Åland Islands during naturally occurring viral epidemics. Pairwise comparison of the estimated marginal means calculated from both generalized linear mixed effects models A) model LF and B) model HA (Table 1 and 2). P-values are Tukey adjusted.

A.

Contrast	*Estimate*	*SE*	*Df*	*Z ratio*	*p-value*
Seed origin population 876 – Seed origin population 950	1.283	0.703	Inf	1.825	0.161
Seed origin population 876- Seed origin population 9205	**2.071**	**0.778**	**Inf**	**2.661**	**0.021**
Seed origin population 950 – Seed origin population 9205	0.788	0.736	Inf	1.070	0.532

B.

Contrast	*Estimate*	*SE*	*Df*	*Z ratio*	*p-value*
Seed origin population 876 – Seed origin population 950	0.703	0.597	Inf	1.176	0.467
Seed origin population 876- Seed origin population 9205	**1.818**	**0.649**	**Inf**	**2.802**	**0.014**
Seed origin population 950 – Seed origin population 9205	1.115	0.664	Inf	1.679	0.213

Supplementary table 4. Model coefficients (model HA) testing for the effects of local adaptation on host infection status using the home *vs.* away metrics of local adaptation. For all variables one levels is a reference level included in the intercept.

Parameter	*Coefficient*	*Std. Error*	*z-ratio*	*p-value*
(Intercept)	0.46958	0.76237	0.61595	0.53792
Plant size	-0.00088	0.00067	-1.31035	0.19008
Seed origin population 950	-1.28293	0.70295	-1.82505	0.06799
Seed origin population 9205	**-2.07082**	**0.77828**	**-2.66078**	**0.00780**
Transplant population 950	-0.47045	0.67071	-0.70142	0.48304
Transplant population 9205	0.28314	0.59902	0.47267	0.63645
Seed origin population 876 × sympatric	-0.86730	0.80862	-1.07257	0.28346
Seed origin population 950 × sympatric	0.29316	0.83422	0.35141	0.72528
Seed origin population 9205 × sympatric	-0.36200	0.92003	-0.39346	0.69398

685

686

687 Supplementary table 5. Explanatory and predictive performance of the HMSC model
688 of viral occurrence in the experimental plant individuals in terms of Tjur R^2 and AUC. The
689 model predictive performance is based on 10-fold cross-validation.

690

	Model explanatory performance		Model predictive performance with 10-fold cross validation (cv)	
Response variable	**Tjur R^2**	**AUC**	**Tjur R^2 (cv)**	**AUC (cv)**
Caulimoviridae	0.24	0.91	0.16	0.81
Partitiviridae	0.47	0.98	0.4	0.88
Pospivirodae	0.1	0.8	0.01	0.54

691

692

693

Supplementary table 6. Exact values of the HMSC model variance partitioning for the three most prevalent virus families detected in a reciprocal transplant experiment studying local adaptation. Six variables explaining the virus family occurrence in a reciprocal transplant experiment were: maternal line ID, seed origin population ID, sympatry/allopatry, plant size, herbivory and transplant population ID (random).

Model parameter		**Response variable**		
Fixed effects:		*Caulimoviridae*	*Partitiviridae*	*Pospivirodae*
	Maternal line	0.54	0.60	0.73
	Seed origin population	0.35	0.33	0.18
	Sympatry/allopatry	0.03	0.02	0.02
	Plant area	0.07	0.03	0.03
	Herbivory	0	0	0
Random effect:				
	Transplant population	0.02	0.02	0.04

706 **References**

707

708 1. Hammond J. 1982 Plantago as a host of economically important viruses. *Adv. Virus Res.*
709 **27**, 103–140. (doi:10.1016/S0065-3527(08)60434-0)

710 2. Susi H, Filloux D, Frilander MJ, Roumagnac P, Laine AL. 2019 Diverse and variable
711 virus communities in wild plant populations revealed by metagenomic tools. *PeerJ*
712 **2019**. (doi:10.7717/peerj.6140)

713 3. Norberg A, Susi H, Sallinen S, Baran P, Clark NJ, Laine AL. 2023 Direct and indirect
714 viral associations predict coexistence in wild plant virus communities. *Curr. Biol.* **33**,
715 1665-1676.e4. (doi:10.1016/J.CUB.2023.03.022)

716

717