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Research



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Seasonality, land use and plant community diversity shape microbiome–pathogen interactions in wild populations of *Arabidopsis thaliana*

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The microbiome often protects plants against pathogens, but most findings are limited to controlled experiments in the lab. In the context of wild populations, one key challenge is to understand sources of variation that impact the commensal microbiome, which in turn shapes the degree of protection. Here, we surveyed both disease symptoms and microbiomes from wild populations of *Arabidopsis thaliana* over four consecutive seasons (Autumn/Spring) across three different land use types. Land use types varied in the extent of anthropogenic influences and included forest meadows, human-impacted fields adjacent to agriculture or municipal parks and highly disturbed habitats near railroad tracks. By building an integrative map of abiotic and biotic variables, we find that a key predictor of disease was biodiversity across ecological scales. Plant communities with higher diversity were associated with increased microbial diversity and reduced disease burden in *A. thaliana* populations, particularly in the Spring. However, the diversity–microbiome–disease relationships were all sensitive to season and further modulated by land use. Taken together, our work highlights the importance of anthropogenic change reshaping species interactions across ecological scales to impact disease risk in wild plant populations.

This article is part of the theme issue ‘Wild plant pathosystems’.

1. Introduction

Rapid anthropogenic change is reshaping ecological processes at multiple scales, each of which can impact infectious disease dynamics [1–3]. For example, warmer temperatures can exacerbate disease by increasing pathogen growth rates and at the same time shift plant physiological states that dysregulate the immune system and diminish defence responses [4]. Land use change associated with the intensification of anthropogenic effects also alters resource availability that shapes intra- and inter-specific interactions within a community [3]. Other anthropogenic effects, like the introduction of invasive species, can alter disease dynamics, but whether invasions increase or decrease disease will depend on frequency- and density-dependent

transmission properties of specific pathogens [5]. In sum, anthropogenic change alters many abiotic and biotic factors, and these disturbances are often implicated in reshaping the networks of ecological interactions [6]. In addition, many pathogens are generalists that can infect multiple host species [7,8], and the impact of anthropogenic change on the distribution of any of these hosts can have ripple effects on disease dynamics throughout the ecological networks in which these hosts participate.

Despite the importance of the interactions between biodiversity and disease, the links between the two remain poorly understood. The dilution effect proposes that higher species diversity in communities dilutes disease risk by changes in community composition, context-dependent variation in immune competence and life history traits that decrease pathogen spread through a community [1,9,10]. Indeed, farmers have long recognized that intercropping and increased biodiversity in their fields are associated with lower disease incidence [11]. Yet, higher biodiversity is also often associated with higher pathogen richness and may constitute hotspots for pathogen emergence and spillover [12,13]. An emerging synthesis suggests several key traits for understanding how land use change impacts disease dynamics. If land use changes increase the degree of habitat fragmentation, increase plant density and decrease plant community diversity, then disease risk can be amplified [3]. However, the risk of disease spread will depend on factors that impede or facilitate microbial transmission within and between plant species [3,14,15].

Microbiomes have emerged as an important factor in protecting plants against pathogens [16–18], offering potentially new approaches for combating emerging pathogens [19,20]. The protection is thought to result from microbe–microbe resource competition or intermicrobial warfare, both of which inhibit pathogen colonization and growth [16,21]. Microbes can also indirectly modulate the host immune system and thus alter the effectiveness of defence against pathogens [22,23]. However, these findings are limited to controlled experiments in simplified ecological contexts. In realistic ecological contexts, plant disease dynamics are very different. Disease is often less severe in wild plants than in agricultural settings [24,25], and when disease outbreaks do occur, the incidence of coinfection is higher in wild populations [26]. While microbiomes are generally protective, interactions between the commensal microbiome and pathogen can instead lead to overall increases in pathogen diversity and burden [27,28]. Thus, microbiome–pathogen interactions potentially shape disease dynamics, but the impact remains poorly understood in naturalistic ecological contexts.

Understanding these complex microbiome–disease dynamics requires characterizing wild populations for both disease dynamics and microbiome variation. *Arabidopsis thaliana* is an excellent model as it inhabits a broad range of environments, including highly disturbed habitats [29], and co-occurs with diverse assemblages of other plant species. In the wild, the *A. thaliana* phyllosphere harbours diverse microbiomes distinct from the rhizosphere [30], shaped by a combination of host genetics [31,32], abiotic conditions [33], growing season [34] and the surrounding plant community [33,35,36]. *Sphingomonas*, *Pseudomonas*, *Flavobacterium*, *Massilia* and *Rhizobium* species are frequently observed bacteria in the phyllosphere [31,33–35]. Commonly detected fungi in the phyllosphere include *Plectosphaerella* species, Helotiales genera and Mycosphaerellaceae genera [32,37]. The phyllosphere is often environmentally responsive, but the response of the microbiome can also depend on host genetic variation. For example, a survey of European *A. thaliana* populations revealed that a combination of drought and host genetic variation in host immunity shaped microbiome composition [33]. Also in the wild, members of the *A. thaliana* microbiome can promote plant growth and can protect its host against pathogens, such as *Pseudomonas* [35]. Beyond single pathogens, a humpbacked biodiversity–disease relationship between commensal microbes and pathogen co-infection was observed in a survey of natural *A. thaliana* populations in France [27]. These complex dynamics suggest that a range of outcomes is possible for the impact of commensal microbiomes on pathogen dynamics, ranging from no impact to suppression to facilitation.

Consideration of the microbiome can provide new insights into how changes in land use alter the ecological networks that spread pathogens as well as other commensal and beneficial microbes. Plant communities in disturbed habitats are thought to be hotspots for the emergence of novel diseases because of pathogen sharing between native and invasive species, which then can serve as corridors that facilitate the spread of disease to agricultural fields and other ecosystems [3,38]. The most common pathogens that infect *A. thaliana* are often generalists that infect many plant species, such as bacteria from the *Pseudomonas syringae* complex, *Xanthomonas campestris*, *Pantoea agglomerans*, as well as fungi and oomycetes [27,39,40]. For these generalist pathogens shared across host species, land use change that reshapes ecological dynamics may also impact pathogen transmission across different host species [3]; whether this applies to the transmission of non-pathogenic microbes is unclear. Thus, while it is known that land use change can shape disease dynamics, the impact of the microbiome on disease remains poorly understood in naturalistic ecological contexts.

Here, we sampled wild *A. thaliana* populations in western Michigan across Autumn and Spring for two years. *A. thaliana* was introduced to North America by European settlers in the 1600s [41] and, like in Eurasia, it is often found in disturbed habitats where it is likely to interact with both native and introduced species. Our sampling included *A. thaliana* populations from different land use types that ranged in anthropogenic influence, from forests to plots immediately adjacent to railroad tracks. To investigate how land use change alters the ecological context that shapes the frequency and type of disease in wild *A. thaliana*, we characterized the abiotic and biotic environments to identify drivers of microbiome variation and associations with disease in the *A. thaliana* phyllosphere. If anthropogenic influence is restructuring ecological interactions, then we expect to see differences in microbial sharing between the surrounding plant community and focal *A. thaliana* stands across land use types. We then relate how these microbial dynamics are associated with disease dynamics by tracking variation in disease incidence and symptomology across the land use types and seasons. Together, our results highlight how anthropogenic change shapes ecological interactions across biological scales, from within the microbiome to between plant species to overall disease dynamics.

2. Methods

Please refer to the electronic supplementary material for full methodological details.

(a) Site characterization and sample collection

Arabidopsis thaliana populations were sampled from 19 sites in southwest Michigan in the Autumn and Spring between Autumn 2021 and Spring 2023 (figure 1A, electronic supplementary material, table M1 for site details, dates and sample sizes); we note that site 17 was only sampled once and subsequently replaced with site 19. As *A. thaliana* at these sites are winter annuals, collections were timed to start shortly after germination in November and harvesting was completed in the Spring, after bolting reproductive stalks but before flowering and senescence.

Sites were chosen to encompass the range of environments in which *A. thaliana* is commonly found in southwest Michigan, and the sites included populations previously identified for long-term genetic studies [42–44], including the 1001 Genomes Project [45]. For each site, two transects were established to capture the range of micro-environmental variation, and each subsequent sampling returned to approximately the same transect locations within the site. Transects varied in size, from 1.5 to 17.5 m in length and with widths deviating as much as 1.5 m from the centre of the transect. The length of transects was adjusted so that we could always collect 8–12 *A. thaliana* individuals. As we describe below (§2a), all plants were scored for disease symptoms. Every other plant along the transect (e.g. individual #1, 3, 5, etc. from start to finish of transect) was chosen to be enriched for endophytic microbiomes, to ensure that we did not bias microbiome samples in some way across potentially unknown environmental gradients. As we describe below (§2a), we further collected soil to assay for soil chemistry properties and microbiome content. We also characterized the companion plant community and their microbiomes. As the two transects were chosen to span environmental variation within the site, we pooled data from both transects within each site and seasonal sampling (e.g. Site 1 Autumn Year 1, Site 1 Spring Year 1, etc.). We focus on site-level variation, as site explains much more variance than transects for both macro- and microbiome community diversity metrics (electronic supplementary material, table M2). Thus, our analyses focused on differences between sites.

We classified the land use type for sites into forest ($n = 7$), anthropogenic ($n = 5$) or railroad ($n = 7$). Railroad sites were directly adjacent to active railroad tracks, while anthropogenic sites were often near agricultural fields or municipal parks. We used imperviousness data [46] to quantitatively distinguish forest and anthropogenic sites. Imperviousness data were determined at different spatial scales (every 50 m between 50 and 500 m, then every 100 m up to 1 km). Forest sites were those with <5% imperviousness at all spatial scales, and the remaining non-railroad sites were classified as anthropogenic (electronic supplementary material, figure M1); railroad sites had the highest imperviousness percentages at all spatial scales.

At each site, we characterized the abiotic environment relevant to plant–microbe interactions [36] using mean temperature on the day of sampling, cumulative precipitation in the three weeks preceding sampling and the first two principal components of soil chemistry properties. Soil chemistry analysis was performed by A&L GreatLakes Laboratories (Fort Wayne, Indiana, USA).

For the biotic environment, we focused on the companion plant community that co-occurs with *A. thaliana* following established protocols [47]. To further characterize companion plant communities at each sampling, in areas with at least one *A. thaliana* individual within the quadrat, two 50 × 50 cm quadrats were placed in the approximate centre of each transect. Per cent coverage of the community was assessed visually using the quadrats. We then identified morphotypes as proxies for plant species, counted each morphotype, and saved a voucher for molecular identification using the chloroplast marker, *matK* [47,48]. Community diversity was summed from the two transects and analyses used plant counts with the genus-level *matK* assignment for each morphotype. Bray–Curtis dissimilarity was used to partition variance in compositional changes owing to land use type, and Shannon diversity was used as the primary alpha-diversity measure in subsequent analyses. We refer to this as the companion plant community (CPC). We also characterize the microbiome (see §2b), and for simplicity, we refer to this as the companion plant microbiome (CPM) throughout the manuscript.

We collected the above-ground rosettes of 16–24 *A. thaliana* individuals at each site per sampling period ($n = 1327$ plants total). The distribution of *A. thaliana* population size was approximated at a log₁₀ scale. All plants were visually scored for the following disease symptoms [49]: chlorosis, necrosis, hypersensitive response (HR)-like lesions, *Albugo* presence, *Hyaloperonospora* presence, general fungal damage and herbivory. A composite disease score (excluding herbivory) was built using the presence/absence of each symptom using principal components analysis. Other plant phenotypes measured were diameter, number of leaves and bolting.

The leaf microbiome is composed of both epiphytic microbes that live on the surface along with endophytic microbes that colonize inside the leaves [30]. Many pathogens exhibit endophytic lifestyles [21,30,50], but the endophytic compartment also contains many commensals and thus probably captures relevant pathogen–commensal interactions. Approximately half of the *A. thaliana* samples were randomly selected to be enriched for the endophytic portion of the microbiome in the field by depleting the loosely associated epiphytic microbes through a series of washes following established protocols [51,52]. Plants were briefly washed with 1× TE+0.01% Triton-X, 80% ethanol, 2% bleach and sterile water. Rosettes were blotted dry and placed immediately on dry ice until returned to the lab. We also collected a small portion of each non-*A. thaliana* morphotype to characterize the companion plant microbiome from pooled samples, as well as soil samples for microbiome characterization. These companion plant and soil microbiome samples were collected from the quadrats used to characterize the companion plant community and were roughly in the centre of each transect. As before, we summed together these microbial data to estimate the environmental reservoirs for the *A. thaliana* microbiome. Thus, microbiome analyses were focused on the three sample types: individual *A. thaliana* endophyte samples, companion plant pool and soil.

(b) Microbiome characterization

We characterized the *A. thaliana* microbiome using host-associated microbe PCR (hamPCR) [53] to simultaneously assess microbial load and microbial community diversity (see electronic supplementary material for full details). hamPCR co-amplifies a microbial marker gene with a single-copy host gene in a single PCR reaction and then incorporates Illumina adapters for high-throughput sequencing. We used hamPCR for both bacteria (16S rDNA V5V6V7) and fungi (ITS1-2) with *GIGANTEA* as the host gene; the same amplicons were used for soil and companion plant microbiomes without *GIGANTEA*. Sequencing was performed on the Illumina NovaSeq platform, 1× 300 bp at the NYU Genomics Core.

Microbial load is calculated by dividing the microbial reads by the *GIGANTEA* reads, and composition is determined using tools implemented in QIIME2 v.2023.2 [54] and phyloseq [55]. Amplicon sequence variants (ASVs) were called using DADA2 [56] and taxonomically assigned using classify-sklearn Bayes classifier [57]. 16S rDNA ASVs were classified using the GreenGenes database [58] and Internal Transcribed Spacer (ITS) sequences were classified using the UNITE database [59]. Fungal trophic modes were assigned using the FUNGuild package [60]. Microbial load and composition were treated as separate traits, and we did not scale endophytic microbial composition by load. By treating microbiome composition as a distinct trait, we could compare relative abundances of particular microbes in the endophyte samples and in the sample types without measured microbial load (companion plants and soil).

For the *A. thaliana* endophyte samples only, we assessed finer-scale phylogenetic resolution by developing primers that target strain diversity within *Sphingomonas* and other closely related genera (*mmdA* gene) and within the *Pseudomonas* genus (*HSD* gene). We chose to focus on these groups because *Sphingomonas* was the most abundant bacterial taxon and *Pseudomonas* contains both plant growth promoting strains [15] and important pathogens of *A. thaliana* as well as many other plants. *mmdA* and *HSD* amplicon libraries were prepared without co-amplifying *GIGANTEA*, and sequencing data were processed as stated previously for 16S rDNA and ITS, but taxonomy was assigned using a custom database. Sequencing was performed on the Illumina MiSeq platform, 1× 300 bp at the NYU Genomics Core.

(c) Statistical approach

Given the complexity of the following analyses, we briefly outline our general approach here, but provide more specific tests in §3 (please see electronic supplementary material for full details). Our approach was to first determine the common factors that explain significant variance within each dataset (e.g. disease, microbiome diversity metrics, etc.). Then, we applied these insights to build more integrative analyses to connect plant disease dynamics to the microbiome and environmental factors. Generally, we analysed our data using mixed linear models and PERMANOVAs to identify associations between *A. thaliana* phenotypes (i.e. disease symptoms, microbiome variation) and environmental factors (see electronic supplementary material for full details). For all mixed linear models, the term ‘site’ was nested within the sampling year as a random effect. Using this random effect allowed us to control for the many biological factors that were not directly measured as well as technical factors such as variation in germination time, number of plants sampled, transect length variation and other idiosyncrasies that occur over multi-year sampling of wild populations.

For PERMANOVAs assessing compositional differences (CPC and all microbiome beta-diversity estimates), population membership was used as blocks and year as a main term. For all microbiome beta-diversity analyses, we used a modified Aitchison distance [61]. While this distance metric is largely insensitive to read depth, we included read depth as a covariate because rarefaction was not performed before ordination. Alpha-diversity metrics for all amplicons were rarefied to 1000 reads for endophyte samples and 16 963 reads for companion plant and soil microbiomes (see electronic supplementary material, figure M2, supplemental methods for more detail). For models that focused only on *A. thaliana* endophytes (e.g. disease dynamics, microbial load and diversity), plant diameter was included as a fixed effect to control for differences in host size. As land use type and season often co-varied with many other factors, we primarily used these qualitative factors but delineated when we used quantitative, site-level factors (e.g. CPC diversity, soil chemistry PC1, etc.). We often tested for interactions between season and land use, but if interactions were not significant, the interaction terms were dropped. Mixed effects models were implemented in lme4 [62]. Residuals were assessed for dispersion, normality and outliers using DHARMA [63]. Beta-diversity analyses used the adonis2 implementation of PERMANOVA in vegan [64].

To link all the datasets and explore direct and indirect effects on disease dynamics, we used structural equation modelling to examine the impacts of land use, season, CPC and *A. thaliana* microbial variation (i.e. diversity and load). Path analysis enabled us to test specific hypotheses and was informed by results from mixed linear effects modelling, which suggested the importance of the *A. thaliana* bacterial microbiome in shaping disease PC1 and *A. thaliana* fungal microbiome in shaping disease PC2. Given the strong effects of season and to better visualize patterns, we further separated path analysis between seasons. We constructed models that tested for the direct effects of non-microbial variation (land use type, season, population, year, plant size, CPC Shannon diversity) and *A. thaliana* microbial variation (bacteria/fungal Shannon diversity, bacterial/fungal load) on disease. Both CPC and CPM are correlated with disease. To test the hypothesis that the companion plants impact *A. thaliana* microbiome and disease interactions, mitigating the potential for overfitting, we used CPC because the disease correlation explains marginally more variance (CPC $R^2 = 0.07$ versus CPM $R^2 = 0.04$). Then, we modelled the indirect effects on non-microbial variation on *A. thaliana* microbial variation. Structural equation modelling was performed using lavaan [65]. Variance in model parameters was inspected to ensure it was within an order of magnitude; no scaling was necessary. We fitted four models in our path analysis:

- (1) Disease PC-Land use + *A. thaliana* microbial Shannon diversity + *A. thaliana* microbial load + CPC Shannon diversity + Plant size + Population + Year

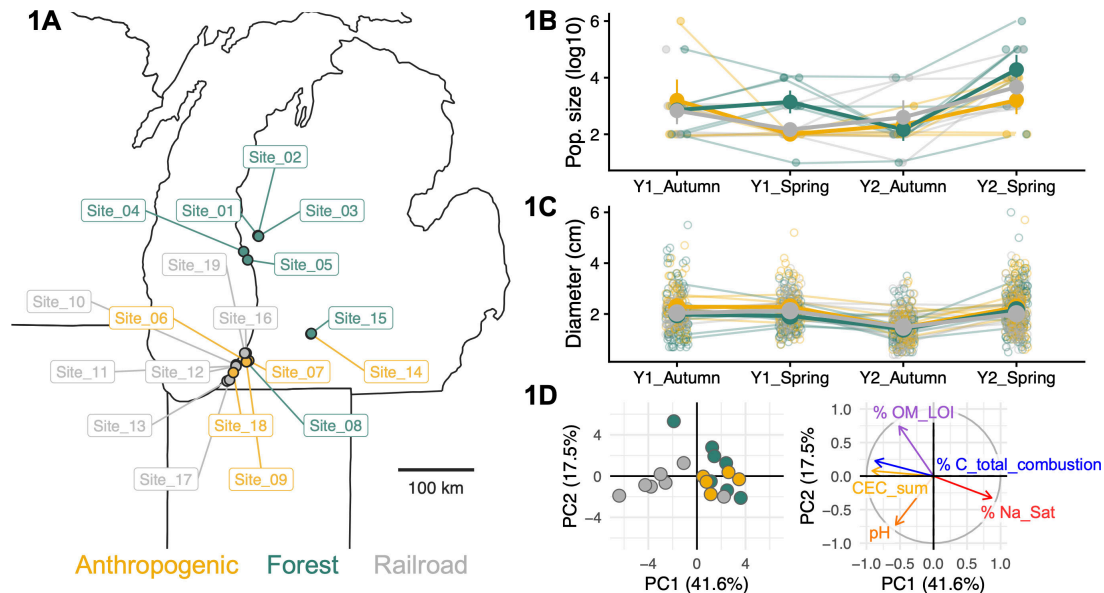


Figure 1. Survey of *A. thaliana* populations. A) Map of populations near Lake Michigan, USA, colour-coded by land use. (B) Population size across seasons, coloured by land-use type. (C) Size of individual plants across seasons. For B and C, smaller hollow circles and thin lines represent each population, while solid larger circles and thick lines represent the average from each land use category, with s.e. (D) PCA biplot showing soil chemistry results, with sites coloured by land use on the right, and the left panel showing the top five most important loadings (text colour matches arrow colour to aid in visualization; %OM_LOI: percent soil organic matter by loss-on-ignition, %C_total_combustion: total carbon content, CEC_sum = sum of cation exchange capacity, %Na_Sat: percent sodium saturation).

- (2) *A. thaliana* microbial Shannon diversity ~ Land use + CPC Shannon diversity + Plant size + Population + Year
- (3) *A. thaliana* microbial load ~ Land use + CPC Shannon diversity + Plant size + Population + Year
- (4) CPC Shannon diversity ~ Land use + Population + Year

Residual covariances were set between *A. thaliana* microbial Shannon diversity and load. Informed by our mixed linear models, we ran path analysis separately to account for the bacteria–disease PC1 and fungi–disease PC2 relationships (full details in electronic supplementary material); the ‘disease PC’ and ‘microbiome’ terms listed above were adjusted accordingly. Model diagnostics to assess fit included χ^2 goodness of fit, comparative fit index (CFI) and standardized root mean square residual (SRMR).

3. Results

(a) Land use shapes abiotic and biotic factors in *A. thaliana* populations

Across the 19 populations sampled, *A. thaliana* population sizes varied idiosyncratically across years and seasons, with no significant difference between land use types (figure 1B, electronic supplementary material, table R1). *Arabidopsis thaliana* plant size increased in the Spring, again with no significant differences between land use type (figure 1C, electronic supplementary material, table R2).

We then characterized how abiotic and biotic factors vary across land use types for *A. thaliana* populations. For abiotic factors, we focused on temperature, precipitation and soil chemistry. Neither temperature nor precipitation significantly differed between the land use types (electronic supplementary material, figure R1, table R3). Soil chemistry differed between land use types, where railroad soils were separated from anthropogenic and forest soils along PC1 (figure 1D; full details in electronic supplementary material, figure R2); railroad soils tended to have higher cation exchange capacity and organic matter levels.

For biotic factors, we characterized the co-occurring CPC, which potentially shares microbes with the focal *A. thaliana* individuals. The composition of the CPC was primarily structured by land use type ($R^2 = 0.09$, $F_{2,66} = 2.71$, $p = 0.002$), with soil chemistry, temperature and year explaining marginal, but significant amounts of variance (electronic supplementary material, figure R3, table R5). The most abundant plants were *Arenaria* sp., *Cerastium* sp. and *Veronica* sp., as well as genera that were unique to each land use type (electronic supplementary material, figure R3). Alpha-diversity of the CPC did not significantly differ across land use types or seasons (electronic supplementary material, figure R4, table R6). Taken together, the different land use categories are associated with different abiotic and biotic factors.

(b) Land use modulates seasonal patterns of disease dynamics

We scored approximately 1300 *A. thaliana* individuals for disease symptoms across the four sampling periods. The most frequently observed symptom was chlorosis, followed by HR-like lesions and necrosis (figure 2A). Many *A. thaliana* individuals

often featured multiple disease symptoms, with the most common co-occurrence being chlorosis, HR-like and necrosis. Notably, we detected a strong seasonal association for plants with multiple symptoms, with most plants in the Spring exhibiting at least two different symptoms (figure 2B). Further, the dominant symptoms exhibited different temporal fluctuations in the different land use types (electronic supplementary material, figure R5). For example, the presence of HR-like lesions decreased in the Spring for forest populations, but increased for railroad populations; anthropogenic sites did not display seasonal dynamics for this symptom.

To understand how land use influences disease symptoms, we first integrated the different disease symptoms into a composite disease score using PCA loadings. PC1 separated the single- and multiple-symptom plants, and PC2 separated plants by symptom types (figure 2C). With this composite score, we detected a significant three-way interaction between disease PC1 and land use, season and Shannon diversity of the CPC (interaction Wald $\chi^2 = 33.3$, $p < 0.001$, figure 2D,E, electronic supplementary material, table R7). Forest populations exhibited increases in symptoms in the Spring, and these swings were greater than in railroad populations (figure 2D). Populations at sites with strong anthropogenic influence, however, did not display a seasonal pattern. CPC Shannon diversity was negatively correlated with disease PC1 across all land use types in the Spring but only in the railroad populations during the Autumn (figure 2E). In the Spring, the relationship between disease PC1 and CPC diversity was steepest for anthropogenic sites. Overall, we find that the diversity of CPC impacted disease symptoms in wild *A. thaliana*, but this impact was sensitive to land use and season.

(c) Season strongly structures the endophytic microbiome of *A. thaliana*, but land use shapes microbial sharing with companion plants

We next assessed factors shaping microbial dynamics in *A. thaliana*. We explored three axes of variation in the bacterial and fungal microbiomes: (i) microbial load in *A. thaliana* endophyte samples (standardized ratio of microbial read number to host read number); (ii) the drivers of beta- and alpha-diversity differences between and within sample types; and (iii) microbial sharing between *A. thaliana*, companion plant microbiome (CPM) and soil microbiome.

Microbial load of *A. thaliana* endophytes was significantly higher in the Spring than in the Autumn for both bacteria and fungi (bacteria: $\beta = 0.89 \pm 0.04$ s.e., $p < 0.0001$, fungi: $\beta = 0.16 \pm 0.07$ s.e., $p = 0.02$, figure 3A,B, electronic supplementary material, table R8); more variance was explained for bacterial load (marginal $R^2 = 0.29$) than fungal load (marginal $R^2 = 0.01$). Bacterial and fungal loads were positively correlated ($\beta = 0.26 \pm 0.03$ s.e., $p < 0.001$), although the strength of this correlation differed between Autumn and Spring (interaction Wald $\chi^2 = 23.8971$, $p < 0.0001$, electronic supplementary material, figure R6, table R9). Land use did not significantly affect *A. thaliana* bacterial or fungal load.

Next, we focused on drivers of compositional differences between sample types. Most microbes were found across all sample types, but their relative abundance differed (see electronic supplementary material, figure R7 for bacteria, electronic supplementary material, figure R8 for fungi); these microbes are also commonly observed in wild *A. thaliana* populations [31,33–35]. Sphingomonadaceae were the most abundant bacteria across all sample types, while the most abundant fungal taxa varied (table 1). Sample type only explained 8.1% of variance in bacterial microbiomes using a modified Aitchison distance metric ($F_{2,879} = 50.7$, $p = 0.001$, electronic supplementary material, figure R9, table R10). Less variance was explained for fungal microbiomes, although sample type explained the most variance ($R^2 = 0.04$, $F_{2,760} = 14.8$, $p = 0.001$, electronic supplementary material, figure R9, table R10). We note that other beta-diversity metrics explained less variance, but show qualitatively similar results (electronic supplementary material, figure R10, table R11). Alpha-diversity was highest in soil and lowest in endophyte samples for both bacterial and fungal microbiomes (electronic supplementary material, figure R11, table R12).

While sample types generally shared the same microbes, different forces structured the microbiomes within each sample type. In these analyses, we included qualitative site descriptors (e.g. land use, season) as well as quantitative measures (e.g. CPC PC1, temperature, etc.). Season structured beta-diversity in the *A. thaliana* bacterial microbiomes ($R^2 = 0.28$, $F_{1,743} = 349.3$, $p = 0.001$) and CPM ($R^2 = 0.32$, $F_{1,67} = 36.5$, $p = 0.001$), while soil bacterial microbiomes were more strongly structured by land use ($R^2 = 0.44$, $F_{2,67} = 47.8$, $p = 0.001$, figure 3C,D, electronic supplementary material, table R13). For fungal endophytes, season explained most of the little variation that could be explained ($R^2 = 0.02$, $F_{1,624} = 13.3$, $p = 0.001$, figure 3E,F, electronic supplementary material, table R14). Like for bacteria, season structured the fungal microbiomes in CPM ($R^2 = 0.32$, $F_{1,67} = 26.7$, $p = 0.001$, figure 3E,F, electronic supplementary material, table R14). Other variables generally did not explain substantial variance structuring either bacterial or fungal microbiomes, with the exception that CPC PC1 explained significant variance in the soil bacterial community ($R^2 = 0.14$, $F_{1,67} = 30.7$, $p = 0.001$, figure 3D–F, electronic supplementary material, table R13).

For alpha-diversity, we focused on the *A. thaliana* endophytes and expanded our dataset to include two additional amplicons, *mmdA* and *HSD*, which capture fine-scale diversity within *Sphingomonas* and *Pseudomonas*, respectively, allowing us to compare the response of microbiomes to land use at different phylogenetic scales. To test this, we compared patterns in temporal change for Shannon diversity for the different amplicons and found that a three-way interaction between amplicon, land use and season shapes temporal dynamics (three-way interaction Wald $\chi^2 = 15.4$, $p = 0.02$, figure 3G, electronic supplementary material, table R15). At the community level (i.e. 16S rDNA and ITS), we saw few trends within land use types, except for consistent decreases in alpha diversity within forests in the Spring. At the strain level, we found that *Pseudomonas* alpha diversity tended to decrease over sampling periods for all land use types (figure 3G). The pattern was less clear for *Sphingomonas*, in which alpha diversity increased in the Spring across all land use types in the first, but not second, year of our study. There were no effects of land use or season on alpha diversity of the fungal or bacterial microbiomes in the soil or CPM (electronic supplementary material, figure R12, table R16); recall that we did not investigate *Sphingomonas* or *Pseudomonas* diversity in these sample types. Finally, we examined correlations between load and Shannon diversity to understand how land use affected

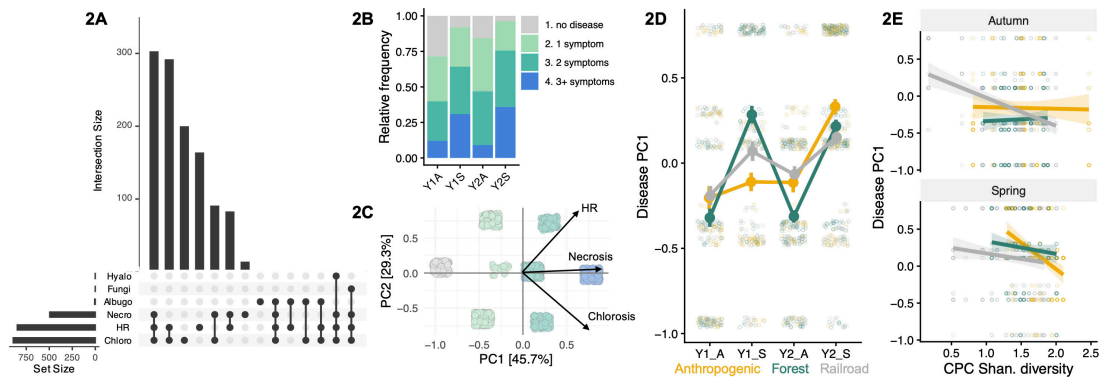


Figure 2. Interactions between land use and season shape disease dynamics. A) Upset plot showing the overlap between different disease symptoms and counts across all plants sampled. (B) Relative frequency of plants with the number of symptoms observed. (C) PCA showing composite disease score, where each point is an individual sample, coloured by the number of symptoms observed that separate PC1. The arrows indicate the driving symptoms that separate PC2. (D) Temporal dynamics in disease dynamics as indicated by the first principal component of the composite disease score. Smaller hollow circles represent individual plants, while the larger solid circles and thick lines represent the average from each land use category per season, with standard error. (E) Companion plant community (CPC) Shannon diversity is negatively correlated with disease PC1, but depends on land use and season. Small points represent individual plants, coloured by land use.

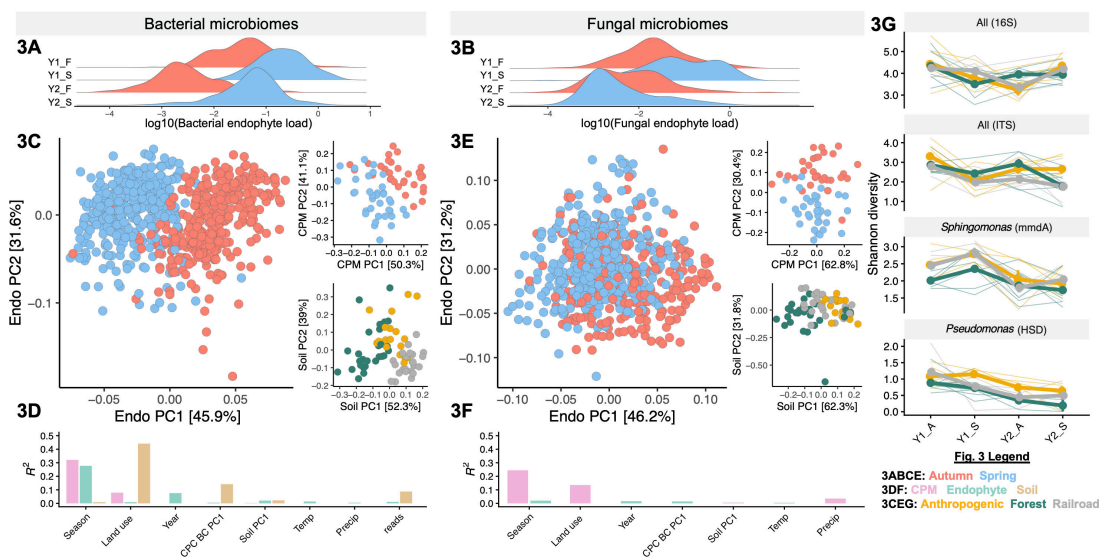


Figure 3. Season and land use impact microbiomes across sample types. A) Bacterial load in Spring (blue) and Autumn (red). (B) Fungal load in Spring and Autumn. (C) Ordinations for bacterial microbiomes in *A. thaliana* endophyte samples, companion plants (abbreviated as 'CPM') and soil based on modified Aitchison distance matrix. Points are coloured by the variable that explained the most variance. (D) Bar plots of the R^2 value of each factor in PERMANOVA analysis for bacterial microbiomes, with colour representing sample type (CPC BC PC1 = plant community beta-diversity using Bray–Curtis PCoA Axis 1). (E) Ordinations for fungal microbiomes, coloured by variables that explained the most variance. (F) Bar plots of the R^2 value of each factor in PERMANOVA analysis for fungal microbiomes, with colour representing sample type. No significant variance was explained by any factor for soil microbiomes. (G) Shannon diversity of each amplicon across the four sampling seasons, coloured by land use. Thin lines represent each population, while thick lines and solid circles show mean, with standard error.

Table 1. Top three most abundant microbes for each sample type.

Bacteria	
Endophytes	Sphingomonadaceae (33.7%), Oxalobacteraceae (15.9%), Comamonadaceae (8.5%)
Companions	Sphingomonadaceae (25.4%), Oxalobacteraceae (10.3%), Comamonadaceae (8.7%)
Soil	Sphingomonadaceae (13.4%), Actinobacteria (6.3%), Beijerinckiaceae (5.6%)
Fungi	
Endophytes	Mycosphaerellaceae (15.2%), Amphisphaeriaceae (13.5%), Helotiaceae (9.1%)
Companions	Didymellaceae (13.0%), Mycosphaerellaceae (10.5%), Cladosporiaceae (7.2%)
Soil	Nectriaceae (10.6%), Trichomeriaceae (5.3%), Helotiaceae

these two aspects of microbial variation. Shannon diversity and load were weakly correlated for bacterial microbiomes, with the direction of the correlation differing between land use types (interaction Wald $\chi^2 = 13.0$, $p = 0.001$, electronic supplementary

material, figure R13, table R17). For fungal microbiomes, a strong negative correlation was observed between Shannon diversity and load, but without an effect of land use ($\beta = -1.04 \pm 0.06$ s.e., $p < 0.0001$, electronic supplementary material, figure R13, table R17). These high-load, low-diversity fungal microbiomes are not dominated by necrotrophs, nor are the fungal taxa in these samples unique; the same fungal taxa are also observed across samples with higher diversity and lower load (electronic supplementary material, figure R14). Taken together, we conclude that season and land use structure the alpha-diversity of endophytic communities within *A. thaliana* endophyte microbiomes, with differing impacts at microbial species versus strain levels, but no obvious effect for the CPM and soil microbiomes.

The phyllosphere is assembled through microbial dispersal from the air, soil and other co-occurring plants [30]. If land use is impacting the potential for microbial spillover from other sources, then we would expect to see associations between microbes in *A. thaliana* and microbes in the CPM or soil. We first examined this at the macro-community level to understand how the CPC affected *A. thaliana* microbial diversity. Indeed, CPC Shannon diversity, and thus the potential diversity of microbial sources, and *A. thaliana* endophyte bacterial and fungal diversity were positively correlated, but the correlation differed between land use types (bacterial interaction Wald $\chi^2 = 8.55$, $p = 0.01$; fungal interaction Wald $\chi^2 = 6.49$, $p = 0.04$; electronic supplementary material, table R18); there was no interaction with season. For *A. thaliana* endophytic bacteria, a positive correlation was observed for all land use types, but was steeper for anthropogenic sites (figure 4A). For *A. thaliana* endophytic fungi, the positive correlation was only observed in forest populations (figure 4B). Furthermore, *A. thaliana* microbial Shannon diversity was correlated with both CPM and soil microbial Shannon diversity, but the effects of land use and season differed between bacteria and fungi (electronic supplementary material, figure R15, table R19).

Next, we assessed microbial sharing between sample types using correlations for the dominant bacterial taxa in endophytes, Sphingomonadaceae, across land use types and seasons. We compared *A. thaliana* microbes with those in companion plants because of the detected effect of CPC diversity on microbial diversity (figure 4A), and also with soil microbes because of the strong effect of land use (figure 3D). We did not include fungi because the dominant taxa varied across sample types more than in bacterial microbiomes, resulting in insufficient sample sizes to make correlations. Each sample type displayed unique patterns of seasonal change across the land use types (electronic supplementary material, figure R16), with seasonal fluctuations consistently observed only in endophytic Sphingomonadaceae. Sphingomonadaceae relative abundance in endophyte samples depended on its relative abundance in the CPM, but was shaped by a three-way interaction with season and land use (interaction Wald $\chi = 15.0$, $p = 0.0006$, figure 4C, electronic supplementary material, table R20). A negative correlation was observed in the Autumn for sites with strong anthropogenic influence, which changed to a positive correlation in the Spring; we observed the opposite pattern for forest and railroad populations. Endophyte–soil (electronic supplementary material, figure R17) did not display the three-way interaction for Sphingomonadaceae, but two-way interactions between land use and season explained significant variance (figure 4D, electronic supplementary material, table R20); no significant association was observed for soil–CPM correlations. Overall, we observe that the strength and direction of microbial sharing differed across seasons and land use types at different levels of organization, from the macro-plant community to microbial diversity to Sphingomonadaceae relative abundance.

(d) Microbial abundance and diversity are associated with disease dynamics, but vary across land use types

Variance in disease PC1, which represents the number of symptoms, was better explained by bacterial than by fungal variation. Associations between *A. thaliana* bacterial load and disease PC1 depended on land use type and season (figure 5A, electronic supplementary material, table R21). As mentioned previously, disease PC1 was higher in the Spring ($\beta = 0.27 \pm 0.06$ s.e., $p < 0.001$, figure 5A). According to Akaike information criterion, the best fit model included the interaction between disease PC1 and bacterial load (electronic supplementary material, table R21B), although this interaction was not significant (two-way interaction Wald $\chi^2 = 5.1264$, $p = 0.07$). For example, forest populations tended to have consistent bacterial loads across disease PC1, while anthropogenic and railroad populations were more variable. Additionally, disease PC1 was negatively correlated with *A. thaliana* bacterial Shannon diversity, with more symptoms observed in the plant associated with lower diversity (figure 5B). CPC Shannon diversity still explained significant variance when accounting for bacterial effects on disease PC1 (electronic supplementary material, table R21). By contrast, disease PC2, which represents the symptom types, was better explained by variation in *A. thaliana* fungal microbiomes. Primarily, interactions between fungal load and season explained significant variance for disease PC2 (interaction Wald $\chi^2 = 10.0$, $p = 0.002$, figure 5C, electronic supplementary material, table R22), with a stronger and negative correlation in the Autumn. We note that models that included fungal diversity and the CPC were outperformed by the best model with fungal load as the only microbiome characteristic (electronic supplementary material, table R22).

We next performed path analysis to explore direct and indirect drivers that might explain significant variance in disease PC scores, given the complexity of the linear mixed model regressions as described in the previous paragraph. These paths represent hypotheses, and to better separate the interactions with season, we separated the analysis by season and between disease PC1 and PC2. The data fit well the path analysis for bacteria–disease PC1 in the Autumn (χ^2 d.f. = 1, $p = 0.096$, CFI = 0.995, SRMR = 0.015), bacteria–disease PC1 in the Spring (χ^2 d.f. = 1, $p = 0.403$, CFI = 1.000, SRMR = 0.006) and fungi–disease PC2 in the Spring (χ^2 d.f. = 1, $p = 0.403$, CFI = 1.000, SRMR = 0.007). While the fit for fungi–disease PC2 in the Autumn was substantially worse (χ^2 d.f. = 1, $p = 0.03$, CFI = 0.980, SRMR = 0.021), this is consistent with the lack of relationship between fungal load and disease PC2 in the Autumn determined using mixed linear models (figure 5C). Overall, while a complex set of interactions linked land use and CPC Shannon diversity to microbial traits, only *A. thaliana* microbiomes were directly associated with disease PC1 (bacterial diversity) or PC2 (fungal load) in the Spring (figure 5D; see electronic supplementary material, tables R23–26 for a full summary of model estimates).

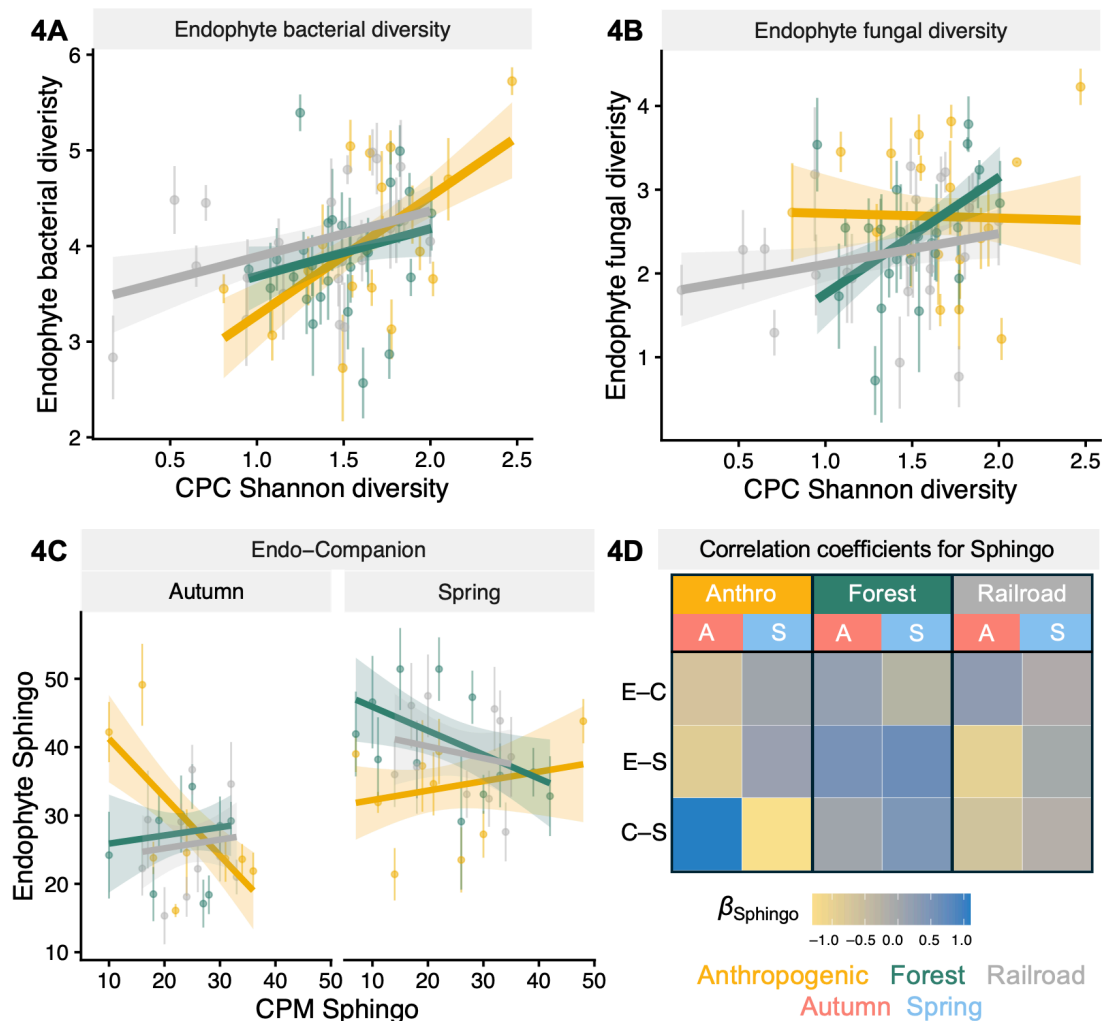


Figure 4. Land use impacts microbial diversity and sharing between sample types. For A,B, points represent the correlation between the CPC Shannon diversity and mean microbial Shannon diversity ($\pm$ s.e.) in the *A. thaliana* endophyte samples for each population, coloured by land use. (A) *A. thaliana* endophyte bacterial diversity is positively correlated with the companion plant community diversity, but the slope differs between land use types. (B) *Arabidopsis thaliana* fungal diversity is also positively correlated with CPC Shannon diversity, but not for anthropogenic sites. (C) Correlations for the relative abundance of the dominant bacterial family, Sphingomonadaceae, between the companion plant microbiome (CPM) and *A. thaliana* endophytic microbiomes. Other sample types are in electronic supplementary material, figure R17. Points represent the mean ($\pm$ s.e.) for the relative abundance of Sphingomonadaceae. (D) Boxes represent the Sphingomonadaceae correlation coefficients (β) for a given sample type pair. Y-axis titles are abbreviated comparisons for E–C: endophyte–CPM, E–S: endophyte–soil and C–S: CPM–soil. Sphingo, Sphingomonadaceae

Taken together, these analyses show the importance of considering multiple facets of microbial diversity (e.g. load and diversity from bacteria and fungi) to explain patterns in disease dynamics. Importantly, we find diversity at both the community and microbial levels to be associated with reduced disease, highlighting how processes across ecological scales potentially impact disease dynamics.

4. Discussion

Here, we surveyed wild populations of *A. thaliana* across Autumn and Spring seasons for disease symptoms and microbiome variation. While most studies focus on only microbiomes or disease in the field, our integrative study reveals how land use impacts disease by modulating different aspects of the bacterial and fungal microbiomes, including diversity and load. These impacts of land use on disease were primarily shaped by interactions between season and land use, suggesting a strong seasonal sensitivity of the disease dynamics across land use types. In the Spring, more individuals display multiple disease symptoms, with higher microbial load and changes to microbial community composition (figures 2 and 3). Increased CPC diversity was associated with reduced occurrence of these multiple disease symptoms, especially in the Spring. Notably, the negative relationship between CPC diversity was observed only in railroad populations for both Autumn and Spring. This relationship also impacted different characteristics of bacterial and fungal microbiomes as well as land use types (figure 4). Intriguingly, CPC diversity was positively associated with increased *A. thaliana* microbial diversity (figure 4), with the strongest positive relationship observed in anthropogenic sites. This higher bacterial diversity was also associated with reduced disease in *A. thaliana* in the Spring, but not Autumn (figure 5). Our results highlight how land use type can impact multiple modes of ecological interactions that in turn can alter disease risk in wild populations.

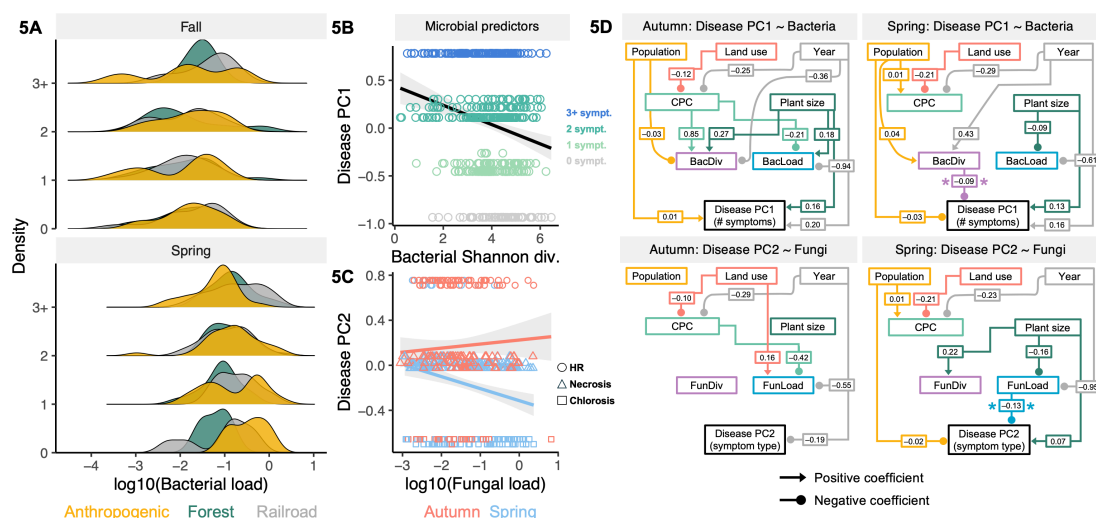


Figure 5. Associations between microbiome variation and disease symptoms vary by season, but differ between bacteria and fungi. (A) Associations between bacterial load and disease PC1 as a function of land use type and season. Density plots reflect the number of individuals for a given disease PC1 (i.e. multiple-symptom class), coloured by land use type. In B,C, we illustrate the negative association between disease PC1–bacterial diversity and disease PC2–fungal load. (B) Association of Shannon diversity bacterial microbiomes with disease PC1 scores, with points coloured by the number of symptoms observed. (C) Association of fungal load with disease PC2, with points coloured by season and shape reflecting symptom type associated with PC2 score. (D) Path analysis results to explore hypotheses for how environment and microbiome contribute to variation in disease PC1 and PC2. Data were analysed separately for each season to better visualize seasonal effects. Only in the Spring did bacterial diversity directly impact disease PC1 and fungal load directly impact disease PC2. Only paths with significant coefficients ($p < 0.05$) were visualized, with the text boxes showing their coefficients. Direct paths between microbial traits in the Spring are noted with asterisks.

We observed that higher biodiversity at two ecological scales—the plant community and within the *A. thaliana* microbiome—resulted in less disease. This result is suggestive of the dilution effect, where higher host diversity dilutes the disease risk in a multi-host pathogen system [1,9,66]. Biodiversity loss associated with urban and agricultural environments often results in communities enriched for species with life history traits associated with fast pace-of-life and reduced investment in immune defenses, increasing the risk of disease spillover [3,66]. While we did not measure these traits in the CPC, land use types differed in both plant community composition (electronic supplementary material, figure R3) and the correlation between plant diversity and disease symptoms (figure 2D), illustrating variation in the traits underlying the diversity–disease relationship. The impact of this variation in disease-relevant plant traits can be further modulated by the abiotic factors, such as soil chemical properties [49]. In the Spring, higher-diversity microbiomes often provide greater protection against pathogens in plants [16,18,67], as we observed here, but this protection depends on microbial load (figure 5). In particular, protection by diverse microbiomes occurs when commensals saturate the host niche space and consume resources that effectively outcompete invading pathogens [68,69]. Beyond mere competition over resources, microbiome members can also directly inhibit other microbes through the production of antimicrobial agents [21] or indirectly by priming the plant immune system [22,23]. Identifying potential synergies between the mechanisms underlying the dilution effect at the plant community scale and within the microbiome is an important research priority.

Our results are consistent with several scenarios, but we believe that the following is particularly attractive. First, CPC diversity protects against disease through plant traits that dilute the community-level disease burden. In turn, increases in CPC diversity promote diversity within the *A. thaliana* microbiome, resulting in increased protection and reduced disease. Our path analysis (figure 5D) supports a scenario whereby land use reshapes CPC diversity, which in turn reshapes microbial diversity and load. Although these effects are observed in both the Autumn and Spring, bacterial diversity and fungal load were directly associated with disease dynamics only in the Spring. Our results raise the possibility that there are seasonal differences in the relationship between CPC and *A. thaliana* microbiome dynamics that also impact disease dynamics, but this is largely hypothetical. Path analysis suggests that the CPC and other factors impact the bacterial and fungal microbiomes in *A. thaliana* in the Autumn, but do not impact disease. By contrast, CPC has no impact on either the *A. thaliana* bacterial or fungal microbiome in the Spring, but bacterial diversity and fungal load do directly impact disease. This is consistent with our mixed linear modelling approach that detected significant interactions with season. While it is difficult to know why effects of CPC diversity were not observed in the Autumn, the pattern is consistent with transitory dynamics dominating as the microbiome community becomes established [70]. One potential issue is that we hypothesized a specific directionality in our path analysis, and that feedbacks within these scenarios remain unaccounted for. Additional support is required to untangle these potential outcomes, preferably from targeted manipulation of microbiomes, pathogens and CPC. We note that the dilution effect often depends on the spatial scale over which these interactions occur [71,72]; it remains an open question how to apply the framework of the dilution effect to host-associated microbiomes [1,71].

The concomitant increase in diversity between the CPC with increased microbial diversity in *A. thaliana* exposes a tension in the diversity–disease relationship. This diversity–begets–diversity relationship has been observed in a range of host systems, including *A. thaliana* [27,66,73], but primarily for pathogens rather than commensal microbes. The positive relationship between the CPC and microbial diversity appeared to be beneficial in our setting, but it also potentially amplifies disease risk by exposure to novel microbes. Microbial sharing between host species is expected to accelerate microbial evolution, particularly

under environmental change [15,74], but whether cross-species transmission differentially impacts pathogens compared with commensal microbes is an open question. Intriguingly, the correlation of the relative abundance of Sphingomonadaceae in *A. thaliana* endophyte samples and the other sample types suggests that land use alters microbial sharing (figure 4C,D). Sphingomonadaceae abundance is often protective against bacterial pathogens [22,75], but as we did not assess disease in the CPC, we do not know if this occurred in the surveyed plants.

We note that our approach to characterizing both the CPC and CPM reflects only an estimate of the companion plant dynamics for a given site (electronic supplementary material, table M2); this approach has been used in other studies of wild *A. thaliana* [35,47]. However, CPC along with the CPM and soil microbiomes are likely to vary spatially within each site, although we did not systematically sample this spatial variation. It is possible that the distance between *A. thaliana*, companion plant species and soil samples may be more important drivers of microbial sharing. Because we only sampled the companion plants and soil at limited locations within each site, we cannot assess the role of physical distance in shaping microbial sharing. However, we note that transect length did not vary significantly with our major predictors of land use or season (electronic supplementary material, table D1), suggesting that our analyses on microbial sharing are robust to variation in transect length. A key research priority is to link how these different eco-evolutionary forces shape the microbial evolution in wild host-microbe systems.

While it is tempting to attribute the negative relationship between microbial diversity and disease to protection provided by the microbiome or the dilution effect operating at the microbial scale, we do not know microbiome diversity prior to infection and thus cannot attribute causality. Furthermore, we failed to detect strong effects of disease on structuring the beta-diversity of the endophytic microbiome (electronic supplementary material, figure D1), suggesting that microbial taxa did not consistently differ between healthy and sick individuals. One issue is that pathogens often share the same 16S rDNA sequence as non-pathogenic microbes in plant disease systems, such as in the *P. syringae* complex [52], and so we have limited insight from our amplicon sequencing to identify pathogens. We attempted to use the *HSD* amplicon to identify pathogens among *Pseudomonas* genera, but we did not find any associations with the presence of HR-like lesions or the total number of disease symptoms in diseased plants (electronic supplementary material, figure D2). While there may be technical limitations, Shannon diversity was the primary microbial trait associated with increased disease severity in *A. thaliana* during the Spring, leaving much of the variation in disease-microbiome interactions unexplained.

It is remarkable that so many *A. thaliana* individuals were observed to have multiple disease symptoms, and understanding this resilience of *A. thaliana* to disease pressure remains a key question. While multi-pathogen systems are common in the wild ecosystems, individuals seem rarely to experience strong negative effects of coinfection [24,25], which is aligned with our observations here. Additionally, infections did not appear to cost *A. thaliana* in growth or reproduction, as larger plants tended to have more disease symptoms and were just as likely to be bolting as healthy plants (electronic supplementary material, figure D3). Note, however, that we did not measure phenotypes more directly associated with fitness. We also do not know when plants had been infected or if they had cleared prior infections. It is possible that disease symptoms may not necessarily be independent, as the same pathogen can cause multiple symptom types [76]. We cannot confirm the association between the number of pathogens and the number and type of symptoms owing to the lack of resolution in amplicon sequencing.

This lack of resolution applies to both bacterial and fungal microbiomes. As previously mentioned, neither 16S rDNA nor *HSD* amplicons unequivocally identified microbes associated with diseased plants. Other amplicons, like *gyrase B*, can identify pathogenic bacteria within the plant microbiome [27] and their use would be an important next step in the type of work we describe here. Particularly for fungal microbiomes, the ITS1-2 region provides limited insight owing to high intra- and intergenomic variation in fungal genomes, although it remains the best option for fungal metabarcoding [77]. Annotated databases, like FUNGuild [60], can help link ITS variation to function, but their utility depends on the training data. We classified the trophic mode for the fungal genera observed here, and most were simultaneously classified as pathotroph, saprotroph and symbiotroph (electronic supplementary material, figure D4). The assignments of detected fungi also did not vary across disease states, providing limited insight into potential causative agents of the variation in symptoms observed. In addition, the symptoms observed here may not be owing solely to disease, but may also reflect exposure to environmental stress. Diagnosing the causative agents of symptoms combined with transcriptomic profiling would provide clarity into the relationship between land use and disease in *A. thaliana*.

Finally, it is important to consider the role of plant genetics and demography in shaping interactions between pathogens and the microbiome. Host genetics impacts microbiome composition in *A. thaliana* [31–33,36,78] as well disease resistance [32,79–81]. Land use in turn impacts host genetics, as different urban environments filter for different *A. thaliana* genotypes [82]. Further, the *A. thaliana* populations studied here were probably introduced during European colonization, with strong genetic bottlenecks and reduced genetic diversity in North America compared with the native range [41,45]. This reduction in genetic variation may increase disease incidence if plant defence genes do not sufficiently match the local disease pressures [83–85]. For example, pathogens that are shared across many host species may not cause severe disease in an introduced species potentially owing to the costs of generalism, as previously observed in interactions between maladapted *Pseudomonas syringae* and introduced *A. thaliana* from the midwestern USA [86]. Strain level analyses of pathogens and identifying the causative disease agents are necessary to investigate these processes. *A. thaliana* has also been shown to adapt locally to the companion plant community, particularly for alleles in immunity genes [47]. Railroads are implicated in increasing gene flow in a closely related species, *Arabidopsis arenosa* [87]. Whether this applies to the *A. thaliana* populations here remains unknown—especially whether the introduced alleles by railroad-assisted gene flow would be associated with host control over the microbiome or disease resistance in the populations sampled. Both studies of urban adaptation [82] and introgression facilitated by railroads [87] identified selection on alleles associated with life-history traits that shorten the life cycle. Changes to the demography of *A. thaliana* across different land use types may lead to multiple cohorts of germination during each growing season. If these

germination cohorts differ from the temporal dynamics of pathogen exposure then this may explain seasonal differences in both the individual symptoms (electronic supplementary material, figure R4) and composite disease scores (figure 2C) observed across land use types. Repeated sampling at smaller time intervals would help to elucidate how temporal variation in pathogen exposure intersects with *A. thaliana* genetic variation and demography.

In conclusion, our results highlight the multitude of ways in which land use affects ecological interactions, from the macro-community level down to within-host microbial variation to plant disease dynamics. For the many ecological communities impacted by rapid anthropogenic change, our work here highlights the importance of understanding the interaction between seasonality and ecological context that will shape disease dynamics in natural populations. Harnessing these interactions will be critical in developing the microbiome as a potential solution to provide protection against pathogens in wild and agricultural settings.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. Data are available on Zenodo [88]. Code is available on Zenodo here [89]. Sequencing data are uploaded to NCBI SRA, as Bioprojects PRJNA1279102 for 16S rDNA, PRJNA1282642 for ITS1-2, PRJNA1282654 for HSD, and PRJNA1282661 for mmdA.

Supplementary material is available online [90].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. L.H.: conceptualization, data curation, formal analysis, investigation, methodology, visualization, writing—original draft, writing—review and editing; A.R.: conceptualization, data curation, investigation, methodology, writing—review and editing; E.L.: data curation, formal analysis, investigation, methodology, writing—review and editing; R.L.: data curation, investigation, methodology; B.M.: data curation, investigation, methodology; T.P.: investigation, methodology; F.R.: conceptualization, funding acquisition, investigation, methodology, project administration, supervision, writing—review and editing; D.W.: conceptualization, funding acquisition, investigation, methodology, project administration, supervision, writing—review and editing; J.B.: conceptualization, funding acquisition, investigation, methodology, project administration, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. D.W. holds equity in Computomics, which advises plant breeders. D.W. has also consulted for KWS SE, a globally active plant breeder and seed producer. All other authors declare no competing interests.

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References

1. Keesing F *et al.* 2010 Impacts of biodiversity on the emergence and transmission of infectious diseases. *Nature* **468**, 647–652. (doi:10.1038/nature09575)
2. Baker RE *et al.* 2022 Infectious disease in an era of global change. *Nat. Rev. Microbiol.* **20**, 193–205. (doi:10.1038/s41579-021-00639-z)
3. Laine AL. 2023 Plant disease risk is modified by multiple global change drivers. *Curr. Biol.* **33**, R574–R583. (doi:10.1016/j.cub.2023.03.075)
4. Laine AL, Leino S. 2025 Plant microbiomes feel the heat. *Science* **388**, 1150–1152. (doi:10.1126/science.adw3659)
5. Young HS, Parker IM, Gilbert GS, Sofia Guerra A, Nunn CL. 2017 Introduced species, disease ecology, and biodiversity-disease relationships. *Trends Ecol. Evol.* **32**, 41–54. (doi:10.1016/j.tree.2016.09.008)
6. Bascompte J, Stouffer DB. 2009 The assembly and disassembly of ecological networks. *Phil. Trans. R. Soc. B* **364**, 1781–1787. (doi:10.1098/rstb.2008.0226)
7. Woolhouse ME, Taylor LH, Haydon DT. 2001 Population biology of multihost pathogens. *Science* **292**, 1109–1112. (doi:10.1126/science.1059026)
8. Morris CE, Moury B. 2019 Revisiting the concept of host range of plant pathogens. *Annu. Rev. Phytopathol.* **57**, 63–90. (doi:10.1146/annurev-phyto-082718-100034)
9. Keesing F, Holt RD, Ostfeld RS. 2006 Effects of species diversity on disease risk: effects of species diversity on disease risk. *Ecol. Lett.* **9**, 485–498. (doi:10.1111/j.1461-0248.2006.00885.x)
10. Johnson PTJ, Ostfeld RS, Keesing F. 2015 Frontiers in research on biodiversity and disease. *Ecol. Lett.* **18**, 1119–1133. (doi:10.1111/ele.12479)
11. Curl EA. 1963 Control of plant diseases by crop rotation. *Bot. Rev.* **29**, 413–479. (doi:10.1007/BF02860813)
12. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, Daszak P. 2008 Global trends in emerging infectious diseases. *Nature* **451**, 990–993. (doi:10.1038/nature06536)
13. Keesing F, Ostfeld RS. 2021 Impacts of biodiversity and biodiversity loss on zoonotic diseases. *Proc. Natl Acad. Sci. USA* **118**, e2023540118. (doi:10.1073/pnas.2023540118)
14. Parker IM, Saunders M, Bontrager M, Weitz AP, Hendricks R, Magarey R, Suiter K, Gilbert GS. 2015 Phylogenetic structure and host abundance drive disease pressure in communities. *Nature* **520**, 542–544. (doi:10.1038/nature14372)
15. McDonald BA, Stukenbrock EH. 2016 Rapid emergence of pathogens in agro-ecosystems: global threats to agricultural sustainability and food security. *Phil. Trans. R. Soc. B* **371**, 20160026. (doi:10.1098/rstb.2016.0026)
16. Berg M, Koskella B. 2018 Nutrient- and dose-dependent microbiome-mediated protection against a plant pathogen. *Curr. Biol.* **28**, 2487–2492. (doi:10.1016/j.cub.2018.05.085)
17. Fitzpatrick CR, Salas-González I, Conway JM, Finkel OM, Gilbert S, Russ D, Teixeira PJPL, Dangl JL. 2020 The plant microbiome: from ecology to reductionism and beyond. *Annu. Rev. Microbiol.* **74**, 81–100. (doi:10.1146/annurev-micro-022620-014327)
18. Wang NR, Wiesmann CL, Melnyk RA, Hossain SS, Chi MH, Martens K, Craven K, Haney CH. 2021 Commensal *Pseudomonas fluorescens* strains protect *Arabidopsis* from closely related *Pseudomonas* pathogens in a colonization-dependent manner. *MBio* **13**, e0289221. (doi:10.1128/mbio.02892-21)
19. Henry LP, Bruijning M, Forsberg SKG, Ayroles JF. 2021 The microbiome extends host evolutionary potential. *Nat. Commun.* **12**, 5141. (doi:10.1038/s41467-021-25315-x)
20. Henry LP, Bergelson J. 2023 Evolutionary implications of host genetic control for engineering beneficial microbiomes. *Curr. Opin. Syst. Biol.* **34**, 100455. (doi:10.1016/j.coisb.2023.100455)
21. Russ D, Fitzpatrick CR, Teixeira P, Dangl JL. 2023 Deep discovery informs difficult deployment in plant microbiome science. *Cell* **186**, 4496–4513. (doi:10.1016/j.cell.2023.08.035)

22. Vogel C, Bodenhausen N, Gruissem W, Vorholt JA. 2016 The *Arabidopsis* leaf transcriptome reveals distinct but also overlapping responses to colonization by phyllosphere commensals and pathogen infection with impact on plant health. *New Phytol.* **212**, 192–207. (doi:10.1111/nph.14036)
23. Maier BA *et al.* 2021 A general non-self response as part of plant immunity. *Nat. Plants* **7**, 696–705. (doi:10.1038/s41477-021-00913-1)
24. Stukenbrock EH, McDonald BA. 2008 The origins of plant pathogens in agro-ecosystems. *Annu. Rev. Phytopathol.* **46**, 75–100. (doi:10.1146/annurev.phyto.010708.154114)
25. Alexander HM. 2010 Disease in natural plant populations, communities, and ecosystems: insights into ecological and evolutionary processes. *Plant Dis.* **94**, 492–503. (doi:10.1094/PDIS-94-5-0492)
26. Tollenaere C, Susi H, Laine AL. 2016 Evolutionary and epidemiological implications of multiple infection in plants. *Trends Plant Sci.* **21**, 80–90. (doi:10.1016/j.tplants.2015.10.014)
27. Bartoli C *et al.* 2018 In situ relationships between microbiota and potential pathobiota in *Arabidopsis thaliana*. *ISME J.* **12**, 2024–2038. (doi:10.1038/s41396-018-0152-7)
28. Emmenegger B, Massoni J, Pestalozzi CM, Bortfeld-Miller M, Maier BA, Vorholt JA. 2023 Identifying microbiota community patterns important for plant protection using synthetic communities and machine learning. *Nat. Commun.* **14**, 7983. (doi:10.1038/s41467-023-43793-z)
29. Krämer U. 2015 Planting molecular functions in an ecological context with *Arabidopsis thaliana*. *eLife* **4**, e06100. (doi:10.7554/eLife.06100)
30. Vorholt JA. 2012 Microbial life in the phyllosphere. *Nat. Rev. Microbiol.* **10**, 828–840. (doi:10.1038/nrmicro2910)
31. Horton MW *et al.* 2014 Genome-wide association study of *Arabidopsis thaliana* leaf microbial community. *Nat. Commun.* **5**, 5320. (doi:10.1038/ncomms6320)
32. Brachi B *et al.* 2022 Plant genetic effects on microbial hubs impact host fitness in repeated field trials. *Proc. Natl Acad. Sci. USA* **119**, e2201285119. (doi:10.1073/pnas.2201285119)
33. Karasov TL *et al.* 2024 Continental-scale associations of *Arabidopsis thaliana* phyllosphere members with host genotype and drought. *Nat. Microbiol.* **9**, 2748–2758. (doi:10.1038/s41564-024-01773-z)
34. Almario J, Mahmoudi M, Kroll S, Agler M, Placzek A, Mari A, Kemen E. 2022 The Leaf Microbiome of *Arabidopsis* displays reproducible dynamics and patterns throughout the growing season. *MBio* **13**, e0282521. (doi:10.1128/mbio.02825-21)
35. Lundberg DS *et al.* 2022 Contrasting patterns of microbial dominance in the *Arabidopsis thaliana* phyllosphere. *Proc. Natl Acad. Sci. USA* **119**, e2211881119. (doi:10.1073/pnas.2211881119)
36. Roux F, Frachon L, Bartoli C. 2023 The genetic architecture of adaptation to leaf and root bacterial microbiota in *Arabidopsis thaliana*. *Mol. Biol. Evol.* **40**, msad093. (doi:10.1093/molbev/msad093)
37. Bergelson J, Mittelstrass J, Horton MW. 2019 Characterizing both bacteria and fungi improves understanding of the *Arabidopsis* root microbiome. *Sci. Rep.* **9**, 24. (doi:10.1038/s41598-018-37208-z)
38. Goss EM, Kendig AE, Adhikari A, Lane B, Kortessis N, Holt RD, Clay K, Harmon PF, Flory SL. 2020 Disease in invasive plant populations. *Annu. Rev. Phytopathol.* **58**, 97–117. (doi:10.1146/annurev-phyto-010820-012757)
39. Vinatzer BA, Monteil CL, Clarke CR. 2014 Harnessing population genomics to understand how bacterial pathogens emerge, adapt to crop hosts, and disseminate. *Annu. Rev. Phytopathol.* **52**, 19–43. (doi:10.1146/annurev-phyto-102313-045907)
40. Durán P, Thiergart T, Garrido-Oter R, Agler M, Kemen E, Schulze-Lefert P, Hacquard S. 2018 Microbial interkingdom interactions in roots promote *Arabidopsis* survival. *Cell* **175**, 973–983.e14. (doi:10.1101/354167)
41. Leventhal L, Ruffley M, Exposito-Alonso M. 2025 Planting genomes in the wild: *Arabidopsis* from genetics history to the ecology and evolutionary genomics era. *Annu. Rev. Plant Biol.* **76**, 605–635. (doi:10.1146/annurev-arplant-071123-095146)
42. Bergelson J, Stahl E, Dudek S, Kreitman M. 1998 Genetic variation within and among populations of *Arabidopsis thaliana*. *Genetics* **148**, 1311–1323. (doi:10.1093/genetics/148.3.1311)
43. Nordborg M *et al.* 2005 The pattern of polymorphism in *Arabidopsis thaliana*. *PLoS Biol.* **3**, e196. (doi:10.1371/journal.pbio.0030196)
44. Platt A *et al.* 2010 The scale of population structure in *Arabidopsis thaliana*. *PLoS Genet.* **6**, e1000843. (doi:10.1371/journal.pgen.1000843)
45. Alonso-Blanco C *et al.* 2016 1,135 Genomes reveal the global pattern of polymorphism in *Arabidopsis thaliana*. *Cell* **166**, 481–491. (doi:10.1016/j.cell.2016.05.063)
46. National land cover database class legend and description. See <https://www.mrlc.gov/data/legends/national-land-cover-database-class-legend-and-description>.
47. Frachon L, Mayjonade B, Bartoli C, Hautekèete NC, Roux F. 2019 Adaptation to plant communities across the genome of *Arabidopsis thaliana*. *Mol. Biol. Evol.* **36**, 1442–1456. (doi:10.1093/molbev/msz078)
48. Heckenhauer J, Barfuss MHJ, Samuel R. 2016 Universal multiplexable *matK* primers for DNA barcoding of angiosperms. *Appl. Plant Sci.* **4**, 1500137. (doi:10.3732/apps.1500137)
49. Roux F, Frachon L. 2022 A Genome-Wide Association study in *Arabidopsis thaliana* to decipher the adaptive genetics of quantitative disease resistance in a native heterogeneous environment. *PLoS One* **17**, e0274561. (doi:10.1371/journal.pone.0274561)
50. Wilson M, Hirano SS, Lindow SE. 1999 Location and survival of leaf-associated bacteria in relation to pathogenicity and potential for growth within the leaf. *Appl. Environ. Microbiol.* **65**, 1435–1443. (doi:10.1128/AEM.65.4.1435-1443.1999)
51. Agler MT, Ruhe J, Kroll S, Morhenn C, Kim ST, Weigel D, Kemen EM. 2016 Microbial hub taxa link host and abiotic factors to plant microbiome variation. *PLoS Biol.* **14**, e1002352. (doi:10.1371/journal.pbio.1002352)
52. Karasov TL *et al.* 2018 *Arabidopsis thaliana* and *Pseudomonas* pathogens exhibit stable associations over evolutionary timescales. *Cell Host Microbe* **24**, 168–179. (doi:10.1016/j.chom.2018.06.011)
53. Lundberg DS, Pramoj Na Ayutthaya P, Strauß A, Shirsekar G, Lo WS, Lahaye T, Weigel D. 2021 Host-associated microbe PCR (hamPCR) enables convenient measurement of both microbial load and community composition. *eLife* **10**, e66186. (doi:10.7554/eLife.66186)
54. Bolyen E *et al.* 2019 Reproducible, interactive, scalable and extensible microbiome data science using QIIME 2. *Nat. Biotechnol.* **37**, 852–857. (doi:10.1038/s41587-019-0209-9)
55. McMurdie PJ, Holmes S. 2013 phyloseq: an R package for reproducible interactive analysis and graphics of microbiome census data. *PLoS One* **8**, e61217. (doi:10.1371/journal.pone.0061217)
56. Callahan BJ, McMurdie PJ, Rosen MJ, Han AW, Johnson AJA, Holmes SP. 2016 DADA2: High-resolution sample inference from Illumina amplicon data. *Nat. Methods* **13**, 581–583. (doi:10.1038/nmeth.3869)
57. Bokulich NA, Kaehler BD, Rideout JR, Dillon M, Bolyen E, Knight R, Huttley GA, Gregory Caporaso J. 2018 Optimizing taxonomic classification of marker-gene amplicon sequences with QIIME 2's q2-feature-classifier plugin. *Microbiome* **6**, 90. (doi:10.1186/s40168-018-0470-z)
58. McDonald D, Price MN, Goodrich J, Nawrocki EP, DeSantis TZ, Probst A, Andersen GL, Knight R, Hugenholtz P. 2012 An improved Greengenes taxonomy with explicit ranks for ecological and evolutionary analyses of bacteria and archaea. *ISME J.* **6**, 610–618. (doi:10.1038/ismej.2011.139)
59. Abarenkov K *et al.* 2024 The UNITE database for molecular identification and taxonomic communication of fungi and other eukaryotes: sequences, taxa and classifications reconsidered. *Nucleic Acids Res.* **52**, D791–D797. (doi:10.1093/nar/gkad1039)

60. Nguyen NH, Song Z, Bates ST, Branco S, Tedersoo L, Menke J, Schilling JS, Kennedy PG. 2016 FUNGuild: An open annotation tool for parsing fungal community datasets by ecological guild. *Fungal Ecol.* **20**, 241–248. (doi:10.1016/j.funeco.2015.06.006)
61. Martino C, Morton JT, Marotz CA, Thompson LR, Tripathi A, Knight R, Zengler K. 2019 A novel sparse compositional technique reveals microbial perturbations. *mSystems* **4**, 00016–19. (doi:10.1128/msystems.00016-19)
62. Bates D, Sarkar D, Bates MD, Matrix L. 2007 The lme4 package. *R package version*.
63. Hartig F. 2024 DHARMA: Residual Diagnostics for Hierarchical (Multi-Level / Mixed) Regression Models
64. Oksanen J *et al.* 2015 Vegan community ecology package: ordination methods, diversity analysis and other functions for community and vegetation ecologists. R package v. 2–7.
65. 2025 lavaan: Latent Variable Analysis. Comprehensive R Archive Network (CRAN). (doi:10.32614/CRAN.package.lavaan). See <https://cran.r-project.org/web/packages/lavaan/index.html>.
66. Halliday FW, Rohr JR, Laine AL. 2020 Biodiversity loss underlies the dilution effect of biodiversity. *Ecol. Lett.* **23**, 1611–1622. (doi:10.1111/ele.13590)
67. Hu J, Wei Z, Kowalchuk GA, Xu Y, Shen Q, Jousset A. 2020 Rhizosphere microbiome functional diversity and pathogen invasion resistance build up during plant development. *Environ. Microbiol.* **22**, 5005–5018. (doi:10.1111/1462-2920.15097)
68. Spragge F, Bakkeren E, Jahn MT, Araujo EBN, Pearson CF, Wang X, Pankhurst L, Cunrath O, Foster KR. 2023 Microbiome diversity protects against pathogens by nutrient blocking. *Science* **382**, eadj3502. (doi:10.1126/science.adj3502)
69. Schäfer M, Pacheco AR, Künzler R, Bortfeld-Miller M, Field CM, Vayena E, Hatzimanikatis V, Vorholt JA. 2023 Metabolic interaction models recapitulate leaf microbiota ecology. *Science* **381**, eadf5121. (doi:10.1126/science.adf5121)
70. Beilsmith K, Perisin M, Bergelson J. 2021 Natural bacterial assemblages in *Arabidopsis thaliana* tissues become more distinguishable and diverse during host development. *MBio* **12**, e02723–20. (doi:10.1128/mBio.02723-20)
71. Rohr JR, Civitello DJ, Halliday FW, Hudson PJ, Lafferty KD, Wood CL, Mordecai EA. 2020 Towards common ground in the biodiversity–disease debate. *Nat. Ecol. Evol.* **4**, 24–33. (doi:10.1038/s41559-019-1060-6)
72. Keesing F, Ostfeld RS. 2021 Dilution effects in disease ecology. *Ecol. Lett.* **24**, 2490–2505. (doi:10.1111/ele.13875)
73. Johnson PTJ, Wood CL, Joseph MB, Preston DL, Haas SE, Springer YP. 2016 Habitat heterogeneity drives the host-diversity–begets–parasite-diversity relationship: evidence from experimental and field studies. *Ecol. Lett.* **19**, 752–761. (doi:10.1111/ele.12609)
74. Thines M. 2019 An evolutionary framework for host shifts – jumping ships for survival. *New Phytol.* **224**, 605–617. (doi:10.1111/nph.16092)
75. Vogel CM, Potthoff DB, Schäfer M, Barandun N, Vorholt JA. 2021 Protective role of the *Arabidopsis* leaf microbiota against a bacterial pathogen. *Nat. Microbiol.* **6**, 1537–1548. (doi:10.1038/s41564-021-00997-7)
76. Katagiri F, Thimony R, He SY. 2002 The *Arabidopsis thaliana*–*Pseudomonas syringae* interaction. *Arabidopsis Book* **1**, e0039. (doi:10.1199/tab.0039)
77. Kauserud H. 2023 ITS alchemy: On the use of ITS as a DNA marker in fungal ecology. *Fungal Ecol.* **65**, 101274. (doi:10.1016/j.funeco.2023.101274)
78. Bergelson J, Brachi B, Roux F, Vaillau F. 2021 Assessing the potential to harness the microbiome through plant genetics. *Curr. Opin. Biotechnol.* **70**, 167–173. (doi:10.1016/j.copbio.2021.05.007)
79. Barrett LG, Kniskern JM, Bodenhausen N, Zhang W, Bergelson J. 2009 Continua of specificity and virulence in plant host–pathogen interactions: causes and consequences. *New Phytol.* **183**, 513–529. (doi:10.1111/j.1469-8137.2009.02927.x)
80. Todesco M *et al.* 2010 Natural allelic variation underlying a major fitness trade-off in *Arabidopsis thaliana*. *Nature* **465**, 632–636. (doi:10.1038/nature09083)
81. Karasov TL *et al.* 2014 The long-term maintenance of a resistance polymorphism through diffuse interactions. *Nature* **512**, 436–440. (doi:10.1038/nature13439)
82. Schmitz G *et al.* 2024 Environmental filtering of life-history trait diversity in urban populations of *Arabidopsis thaliana*. *J. Ecol.* **112**, 14–27. (doi:10.1111/1365-2745.14211)
83. Parker IM, Gilbert GS. 2004 The evolutionary ecology of novel plant–pathogen interactions. *Annu. Rev. Ecol. Syst.* **35**, 675–700. (doi:10.1146/annurev.ecolsys.34.011802.132339)
84. Flory SL, Clay K. 2013 Pathogen accumulation and long-term dynamics of plant invasions. *J. Ecol.* **101**, 607–613. (doi:10.1111/1365-2745.12078)
85. Fahey C, Koyama A, Antunes PM. 2022 Vulnerability of non-native invasive plants to novel pathogen attack: do plant traits matter? *Biol. Invasions* **24**, 3349–3379. (doi:10.1007/s10530-022-02853-z)
86. Kniskern JM, Barrett LG, Bergelson J. 2011 Maladaptation in wild populations of the generalist plant pathogen *Pseudomonas syringae*. *Evolution* **65**, 818–830. (doi:10.1111/j.1558-5646.2010.01157.x)
87. Baduel P, Hunter B, Yeola S, Bomblies K. 2018 Genetic basis and evolution of rapid cycling in railway populations of tetraploid *Arabidopsis arenosa*. *PLoS Genet.* **14**, e1007510. (doi:10.1371/journal.pgen.1007510)
88. Henry L. 2025 Seasonality, land use, and diversity shape microbiome–pathogen interactions in wild populations of *Arabidopsis thaliana* [Data set]. Zenodo (doi:10.5281/zenodo.15769719)
89. Henry L. 2026 code for Seasonality, land use, and diversity shape microbiome–pathogen interactions in wild populations of *Arabidopsis thaliana*. Zenodo (doi:10.5281/zenodo.19572218)
90. Henry L, Rat A, Laderman E, Lion R, Mayjonade B, Roux F *et al.* 2026 Supplementary material from: Seasonality, land use, and plant community diversity shape microbiome–pathogen interactions in wild populations of *Arabidopsis thaliana*. Figshare. (doi:10.6084/m9.figshare.c.8476648)