A generalist pathogen view of diverse host evolutionary histories through polygenic virulence

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Keywords: plant-pathogen interactions, co-evolutionary model, Botrytis, fungus, host specificity, generalist pathogen, polygenic virulence, plant domestication, quantitative trait

Abstract:

Host-pathogen interactions display a continuum of host ranges from extreme specialists limited to single hosts to broad generalists with hundreds of hosts. However, the existing models for host-pathogen dynamics are dominated by observations derived from specialist pathogens with qualitative virulence and tight host-pathogen co-evolution. It is not clear how appropriate the co-evolutionary model is in generalist pathogens that present quantitative virulence and broad host specificity. We infected 98 strains of the generalist necrotroph fungus *Botrytis cinerea* on 90 genotypes representing eight plant species with wild and domestic lines. We show that plant-*Botrytis* interactions don't fit traditional co-evolution models as *Botrytis* interacts with the Eudicot species individually, with little link to the relatedness between plant species or plant domestication. Furthermore, *Botrytis* host specificity and virulence have distinct polygenic architectures suggesting that the evolution of the Eudicot/Botrytis interactions relies on genome-wide allelic diversity rather than few major virulence loci.

Introduction

Plant-pathogen interactions influence ecosystems by altering the diversity and structure of natural plant communities while also imposing significant yield losses in agricultural systems (Burdon and Laine, 2019). To respond to pathogen attacks, plants use resistance mechanisms composed of innate and inducible immune systems that recognize danger and mount physiological, physical and chemical responses (Jones and Dangl, 2006). To counter the plant defenses, pathogens use diverse mechanisms to attack and/or interfere with the host including virulence factors and toxins (Rodriguez-Moreno et al., 2018, Zhang et al., 2019). This dynamic co-evolution of plants and pathogens is shaped and altered by the interaction of the genetic diversity in both the host resistance and the pathogen virulence genes (Karasov et al., 2014, Gilbert and Parker, 2016, Stam and McDonald, 2018).

The dominant models in pathology describe host-specialist pathogen interactions that generally follow a co-evolutionary arms-race scenario. This co-evolutionary model also works with some plant and animal multi-host pathogens like *Fusarium oxysporum*, *Alternaria alternata*, and *Pseudomonas aeruginosa*; pathogen species comprised of individual strains that display host specialization linked to disposable chromosomes (van der Does and Rep, 2007, Bertazzoni et al., 2018, Mathee et al., 2008). In the arms-race model, defense innovation in the host rapidly selects a novel counter mechanism in the pathogen and virulence is driven by evolution in large effect genes linked to this interaction. This co-evolution model suggests strong directional pressure in pathogens to become specialists (Barrett et al., 2009, Leggett et al., 2013). This model also posits that over evolutionary distance, host resistance is expected to be highly similar between closely related species while slowly decaying with the evolutionary distance between species. Accordingly, if it was possible to measure pathogen virulence across diverse hosts, a pathogens general virulence is expected to track host evolutionary distances (Gilbert and Parker, 2016, Schulze-Lefert and Panstruga, 2011, Gilbert and Webb, 2007). Although, predictions from this model are successful when applied to host/specialist pathogens, they have not been formally tested through a coordinated measurement of how host/pathogen interactions are affected across host evolution. Testing this requires a pathogen where individual strains can infect broad ranges of host species.

Although they have a major influence on theoretical models, specialist pathogens are only a fraction of the pathogens found in nature (Barrett et al., 2009, Woolhouse et al., 2001). Pathogens have a large spectrum of host specificity and life styles, ranging from obligate biotrophs, specialist parasites that develop intricate long-term relationships within their hosts, to generalist necrotrophs that attack numerous host plants (Moller and Stukenbrock, 2017). It is not clear how applicable and extendable the specialist-driven co-evolutionary arms-race model is to generalist pathogens with

individual strains that can infect diverse host species (Karasov et al., 2014, Antonovics et al., 2011). First, the host diversity of generalist pathogens could weaken the central assumption of tight co-evolution between specific hosts and specific pathogens. Secondly, host resistance to generalist pathogens is quantitative rather than qualitative as described for most host-specialist interactions (Corwin et al., 2016, Corwin and Kliebenstein, 2017). Further, a lack of large effect genes means that generalist pathogens may have to interact with the host's multi-layered defense mechanisms that have diverse evolutionary histories. In plants, some host defense components such as resistance genes (Jacob et al., 2013) or specialized defense compounds (Chae et al., 2014) are specific to limited lineages or even individual plant species. In contrast other components such as cell wall (Sørensen et al., 2010), defense hormone signaling (Berens et al., 2017) or reactive oxygen species (Inupakutika et al., 2016) are widely shared across plant lineages. Crop domestication further complicates the ability to link the host-specialist model to generalist pathogens. Crop domestication can create a loss of genetic diversity in large-effect resistance genes and canonical reductions in plant defense compounds (Karasov et al., 2014, Chen et al., 2015). Thus, it is possible that host-generalist interactions follow a different co-evolutionary model than host-specialist pathogens.

In this study, we address how a pathogen on the generalist end of the host specificity continuum, *Botrytis cinerea* (grey mold; *Botrytis* hereafter) intersects with plant evolutionary histories by testing several assumptions formulated from the current co-evolutionary theory. Botrytis is a pan-global necrotrophic fungus whose individual strains can infect hundreds of plant species, from mosses to gymnosperms (Fillinger and Elad, 2015). Botrytis is an generalist pathogen noted for a lack of specificity in plant developmental stage, host organs or geographic limitations (Barrett and Heil, 2012). This enables *Botrytis* to cause billions/annum of crop damage both pre- and post-harvest to various crops from ornamentals to vegetables and fruits (Veloso and van Kan, 2018). The generalist ability of *Botrytis* strains to infect diverse species, families, orders and tissues has been postulated to arise from its high standing genetic variation (Atwell et al., 2015, Atwell et al., 2018), the production of diverse small RNAs released to silence host defenses (Weiberg et al., 2013) and an arsenal of enzymes and toxic metabolites used for host tissue penetration and necrosis (Nakajima and Akutsu, 2013, Gonzalez-Fernandez et al., 2015). Because *Botrytis* strains infect most plants, it is possible to use this system to empirically assess how host defenses have evolved across plant lineages and domestication by measuring virulence of a single collection of Botrytis strains across these diverse evolutionary distances. This allows us to explore the plant-pathogen co-evolution dynamics at the generalist end of the host-pathogen continuum (Burdon, 2019).

Results

Using a randomized replicated design (Fig. S1), we infected 98 *Botrytis cinerea* strains (Table S1) onto the leaves of 90 plant genotypes (Fig. S2, Table S2) representing 6-12 genotypes from each of eight different Eudicot plants (Fig.1, Fig.S1) for a total of 51,920 independent lesion measurements. As a common measure of the host-pathogen interaction, we utilized lesion area on detached adult leaves, as this is the most comparable tissue amongst these diverse species. The *Botrytis* strain collection contains extensive genetic diversity that has been highly shuffled by recombination creating an admixed pool of virulence mechanisms (Weiberg et al., 2013) (Table S1). Plant species are further defined as taxa covering various scenarios of seven distinct crop domestication events (see Material and Method). Within each plant species, six genotypes represented the high improvement germplasm (cultivar, inbred lines) and six represented the low improvement germplasm (wild, landraces). This allows us to compare the natural evolution between Eudicot lineages to the effect of short-term evolutionary modifications under human-driven artificial selection. As a reference comparison to the virulence on the crops, we also measured the virulence of the strain collection on five single gene mutants of *Arabidopsis* with compromised defense mechanisms *(Rowe et al., 2010, Zhang et al., 2017)*.

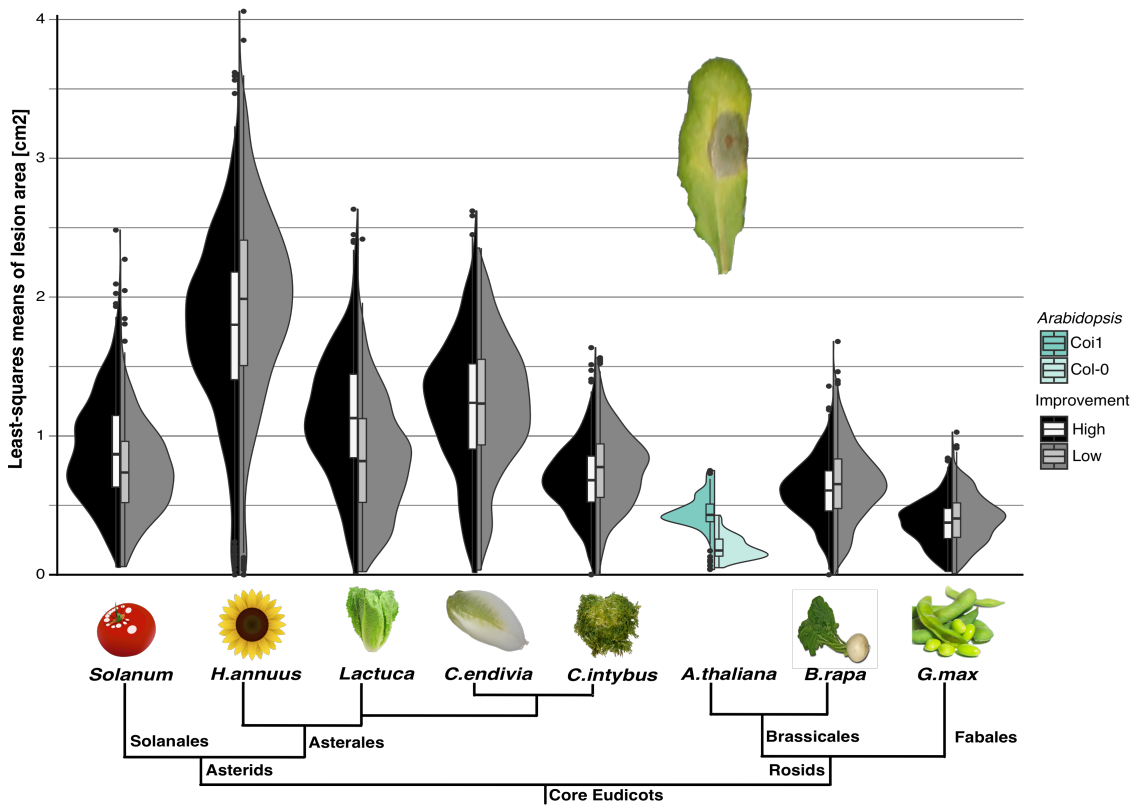

Fig. 1: Lesion areas at 72 hours post inoculation on seven crop species shows small and inconsistent effect of domestication on the *Botrytis* interaction. Half-violins

and boxplots (median and interquartile range) represent the mean lesion area distribution for genotypes with high (black, n=588) and low (grey, n=588) levels of improvement for all 98 Botrytis strains. *Lactuca* refers to *Lactuca sativa* for high improvement and *Lactuca serriola* for low improvement genotypes. *Solanum* refers to *Solanum lycopersicum* for high improvement and *Solanum pimpenellifolium for* low improvement genotypes. As reference, virulence on *A. thaliana* wild-type (Col-0, n=98) and jasmonic acid signaling mutant (*coi1,* n=98) are presented. The non-scaled tree represents the phylogenetic relationship between Eudicot species. A lesion example on an *A. thaliana* leaf is provided.

Host-Botrytis interactions

Lesion area is a heritable estimate of the total interaction of plant resistance with *Botrytis* virulence (Corwin et al., 2016, Zhang et al., 2017, Soltis et al., 2019). Lesion area represents an inclusive measurement of plant resistance as influenced by variation in genetically determined leaf physical characteristics, phytochemistry and induction of defense mechanisms. The eight Eudicot species present a wide distribution of susceptibility with mean lesion area ranging from no visible damage to over $4cm^2$ of necrotic leaf tissue after 72 hours of infection (Fig. 1). *A. thaliana* has the lowest susceptibility while *H. annuus* is the most susceptible species (Fig. 1). Within each species, the effect of genetic variation between the *Botrytis* strains (40-71% of the explained variance) and their interaction with the host genotype (15-35% of the explained variance) controls the vast majority of the variance in the model (Fig. 2B, Fig. S3B). Using the least-square mean lesions from each host genotype x *Botrytis* strain interaction, we extended this to a multi-hosts analysis across all seven crop species. This multi-hosts analysis showed that differences in *Botrytis* virulence between species are the main determinant of lesion area accounting for 52% of the total variance ($P < 2.2x10^{-16}$; Fig. 2A). Further, the interaction between the host species and strains (16% of total variance; $P < 2.2x10^{-16}$) matters more than the strains alone (12% of total variance; $P < 2.2x10^{-16}$). Finally, the host of origin for each strain (1.7% of total variance; $p< 2.2x10^{-16}$) and the geographical origin (0.2% of total variance; p=0.0015) have small effects on Botrytis virulence or specificity across the species tested (Fig. 3,4), confirming the low adaptation to specific hosts or population structure in Botrytis (Atwell et al., 2018, Soltis et al., 2019). As such, the host-Botrytis interaction is controlled by host genetic variation within and between plant species and its interaction with pathogen genotype.

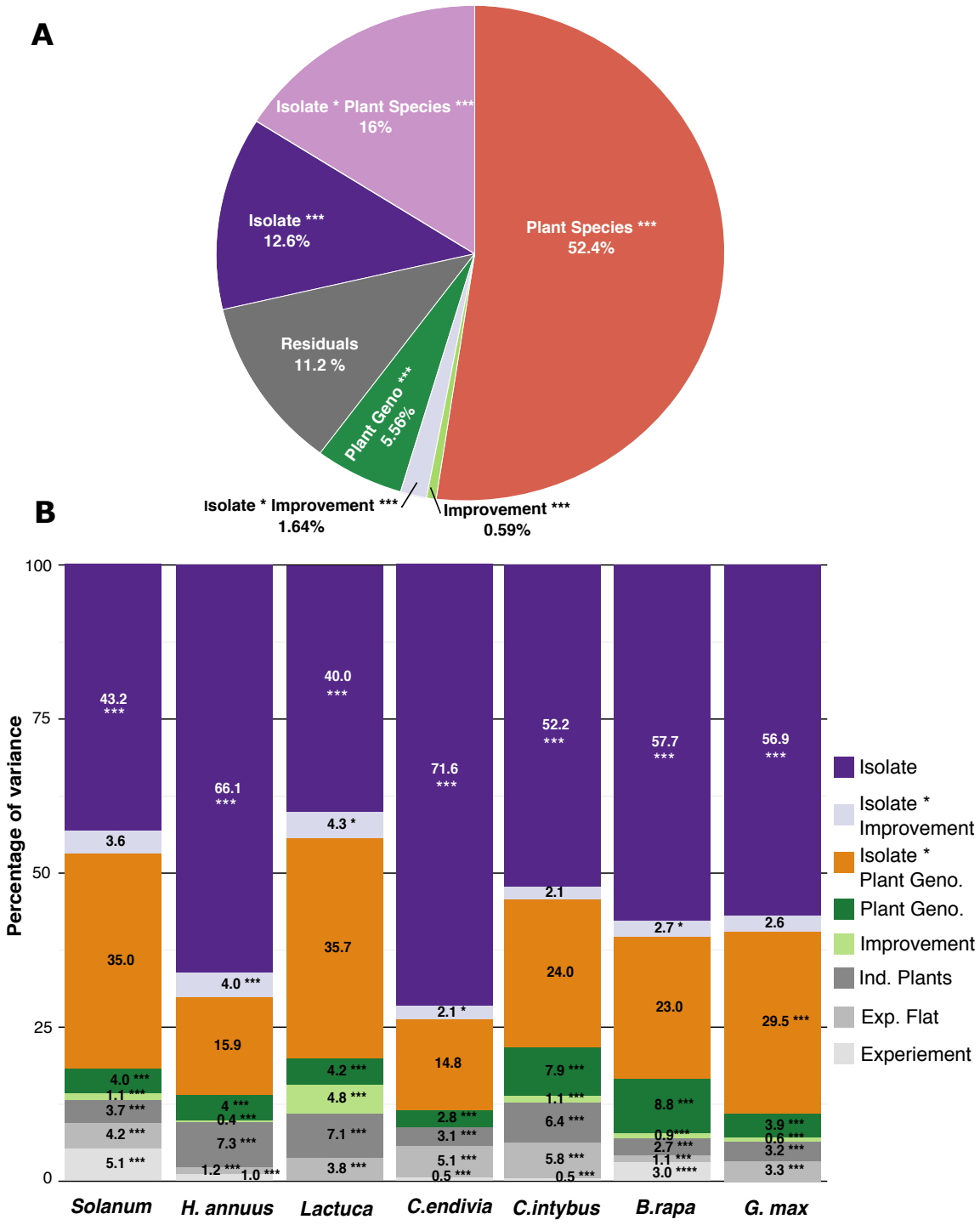

Fig. 2: Host variation predominates the outcomes of plant-*Botrytis* interactions across species while *Botrytis* variation predominates within plant species. A) Multi-host linear model estimating the contribution of plant species, plant genotypes, level of improvement, *Botrytis* strains and their interaction on the percentage of variance in lesion area. B) Species-specific linear mixed models that estimate the percentage of variance in lesion area. In grey are the experimental parameters classed as random factors. Two-tailed t-test: *p<0.05, **p<0.01, ***p<0.005.

Botrytis and crop domestication

For each crop species, high and low improvement genotypes were included to test how seven distinct domestication histories targeting seed, fruit or leaf (see Material & Method) may influence susceptibility to *Botrytis*. Domestication was shown to decrease resistance to insects herbivores (including tomato, lettuce, sunflower and chicory) at various magnitudes associated with the plant traits under artificial selection (Whitehead et al., 2017). Domestication was also shown to affect interaction with specialist pathogens (Karasov et al., 2014, Whitehead et al., 2017, Stukenbrock and McDonald, 2008, De Gracia et al., 2015). When infected with Botrytis, only tomato and lettuce showed the expected decreased resistance in high improvement genotypes (Fig. 1). The other five species (sunflower, endive, chicory, *Brassica* and soybean) showed the opposite trend with low improvement genotypes more susceptible to Botrytis than their cultivated relative (Fig.1). While the effect of crop improvement on lesion area is statistically significant ($P< 2.2x10^{-16}$), this effect is exceedingly small, 0.6% of the total variance across all plant species and 2.1-4.3% of variance within specific species models (Fig. 2). Further, the range of variation in resistance to the diversity of Botrytis strains was similar between wild and domesticated lines for all species. This shows that in a quantitative resistance system, domestication does not have a directional effect, highlighting the challenge of predicting resistance to generalist pathogen (Soltis et al., 2019).

Botrytis and the Eudicot phylogeny

Current plant pathology models posit that relatedness between host species correlates to relatedness in patterns of pathogen susceptibility (Gilbert and Parker, 2016, Schulze-Lefert and Panstruga, 2011). To test this hypothesis, we utilized the standardized least-square mean lesion area of every *Botrytis* strain on every plant genotype to test the relatedness between host species for this pathogen interaction. This showed that variation in host-*Botrytis* interactions identifies genotype groupings for all species, i.e. the virulence of the Botrytis strains across the host genotypes is most similar within a host species. However, beyond the individual species level, the relatedness between hosts did not correlate with the relatedness between the measured host-pathogen interactions (Fig. 3). For example, evolutionarily distant crops such as *B. rapa* and *Lactuca* have a similar susceptibility pattern to the 98 *Botrytis* strains. In contrast, the two sister species, *C. endivia* and *C. intybus*, have highly divergent susceptibility patterns. Thus, the interaction of *Botrytis* with host plants is predominantly defined by variation at the species-by-species level with minimal comparability between species of a particular family or genus.

To provide a mechanistic benchmark for how the host-pathogen relationships across the Eudicots compare to single large effect alterations in plant defense, we included

data for *Arabidopsis thaliana* Col-0 and five single gene knockout mutants (*coi1*, *anac055*, *npr1*, *tga3*, *pad3*) *(Rowe et al., 2010, Zhang et al., 2017)*. These mutants have a larger effect on plant susceptibility (39% of the variance in lesion area; Fig. S3) in comparison to variation between crop genotypes (4-8.8% of the variance within a species; Fig. 2B). While these mutants abolish major sectors of plant resistance, the *Botrytis*-host interactions still cluster these mutants to WT Arabidopsis (Fig. 3) with branch lengths comparable to variation between genotypes within crop species. Thus, *Botrytis* is interacting on a species level with the Eudicots and this is not inherently constrained by large effect variation in individual defense signaling pathways or defense metabolites.

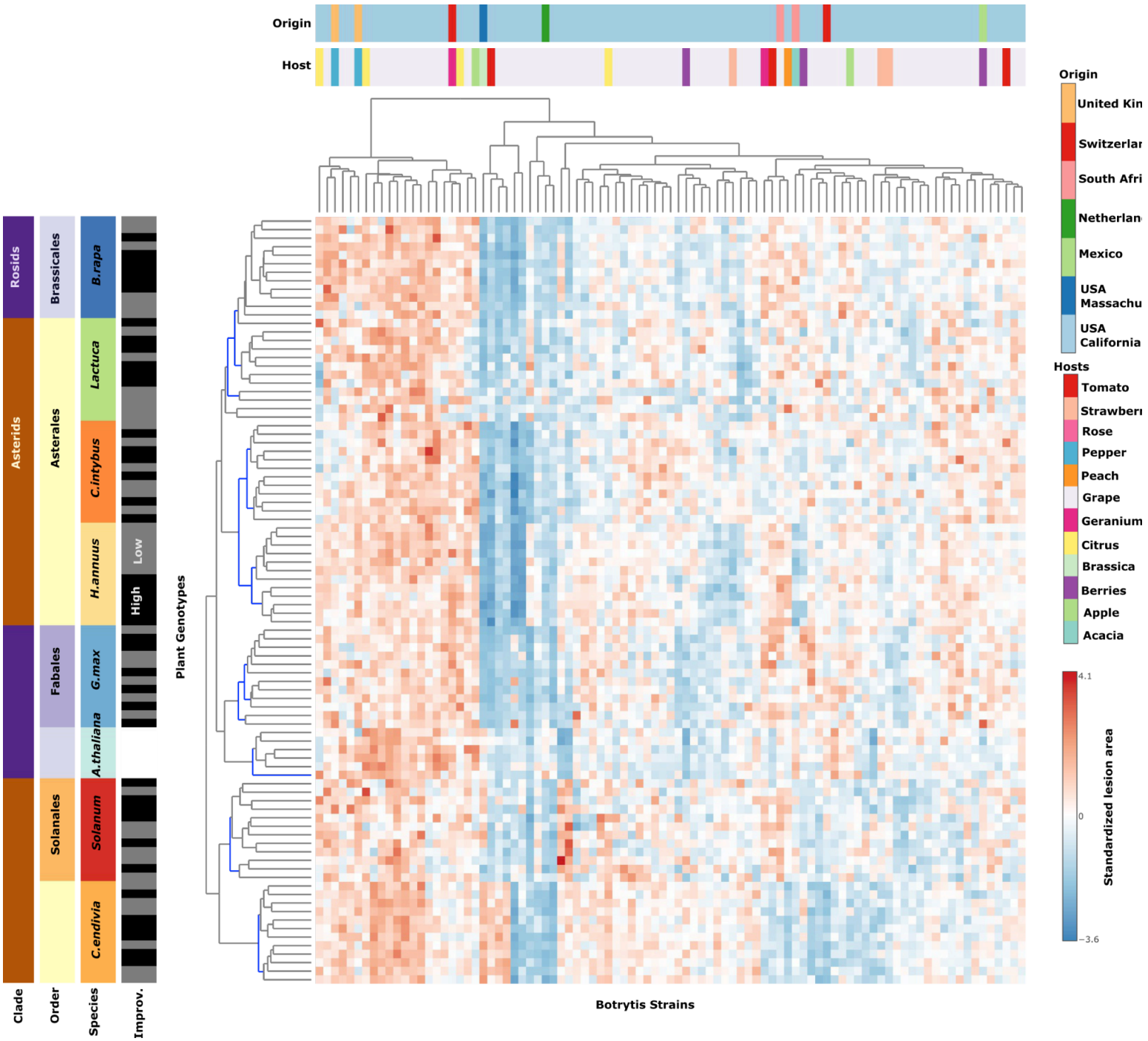

Fig.3: Variation of plant susceptibility in the *Botrytis* pathosystem does not track plant evolution. Heatmap of standardized (z-scored) least-squares means of lesion area (n=6) for *Botrytis* strains (x-axis) interacting with 90 plant genotypes (y-axis). The strains were isolated largely in California (light blue color in the origin bar) and on grape (light purple in the host bar). For *A. thaliana,* five single gene knockout mutants and the corresponding wild-type Col-0 are presented. For the seven crop species, six genotypes with low (grey) and six with high (black) level of improvement are presented. The seven crop species were chosen to represent a wide spectrum of phylogenetic distances across Rosids (Brassicales and Fabales) and Asterids (Asterales and Solanales). Branches in the dendrogram that are supported with 95% certainty after bootstrapping are indicated in blue. No branches in the Botrytis strain dendrogram were significant.

The balance between host specificity and virulence

In the generalism-specialism continuum, specialism is often described as a selected endpoint where high virulence is linked to specific host adaptation and resulting high host-specificity (Barrett et al., 2009, Leggett et al., 2013). Conversely, generalist behavior is assumed as sub-optimal virulence on any host. To test if these hypotheses fit *Botrytis* interactions, we estimated host specificity for each *Botrytis* strain as the coefficient of variation of mean lesion area across the Eudicot species (Fig. 4B). Using the same data we estimated the general virulence for each *Botrytis* strain as the mean of standardized lesion area across the Eudicot species (Fig.3). This creates a unique dataset to compare host specificity and overall virulence within a generalist pathogen. The *Botrytis* strains cover a range of host specificity and virulence (Fig.4). B05.10, the reference strain for the *Botrytis cinerea* genome and the strain used in >90% of all papers to assess host susceptibility to *Botrytis*, is the strain with the lowest host-specificity/highest generalist behaviour (Fig. 4). In contrast to theoretical expectations, strains with increased host specificity had on average lower virulence both across all Eudicots (Fig. 4). Further, strains with increased host-specificity were rare in the population. In combination, this suggests that *Botrytis* is under pressure to maintain broad host ranges and moderate virulence within individual strains.

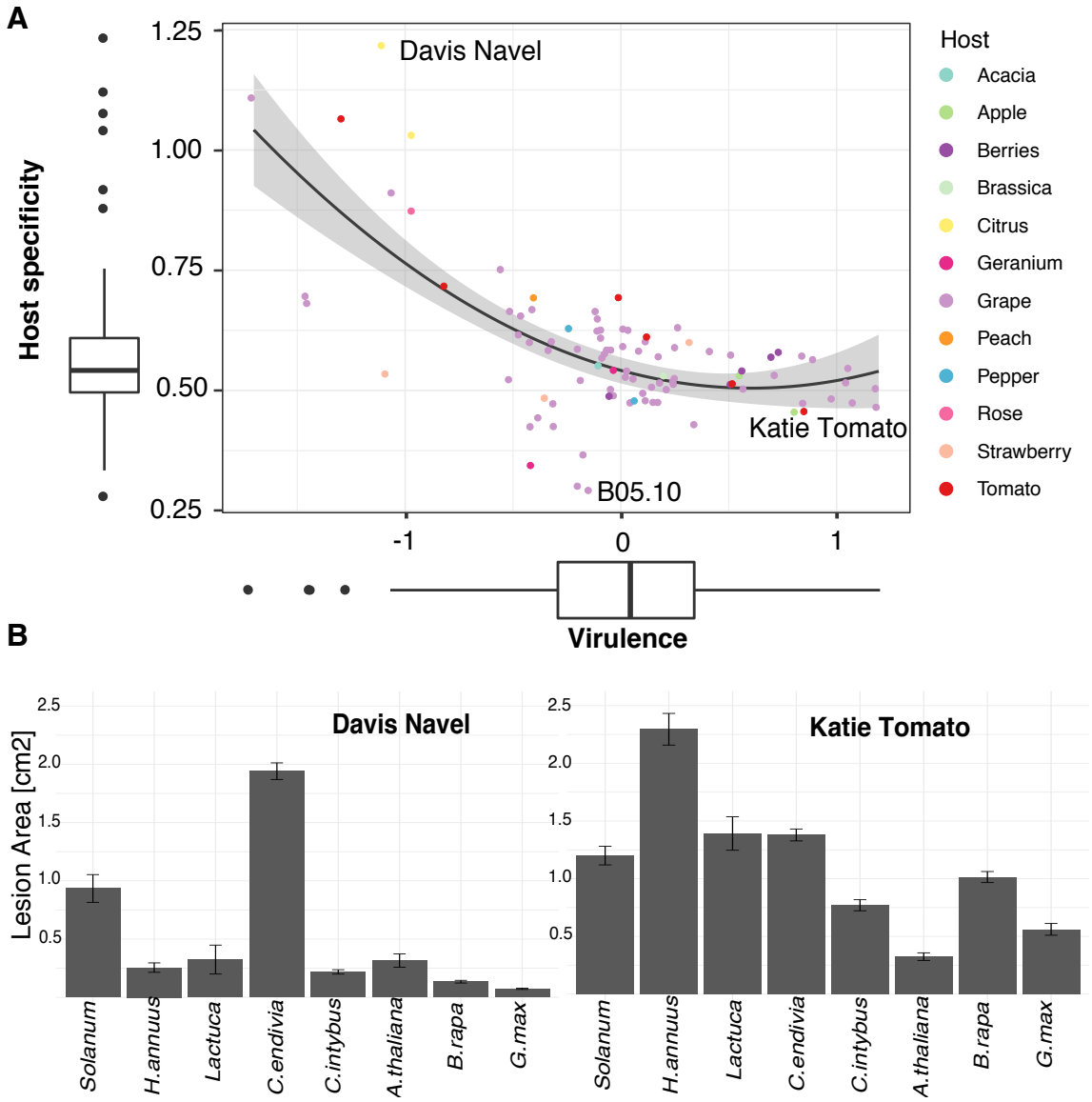

Fig. 4: In the *Botrytis* strain collection, low virulence is linked to high host specificity. A) Estimates of general virulence and host specificity across eight Eudicots for the *Botrytis* strains are shown. They strains are colored according to the plant host from which they were collected. A quadratic relationship was the optimal description of the relationships between specificity and virulence and is shown with a grey confidence interval (R^2=0.44, P=6.36x10^{-13}). B) Mean lesion area and standard error (n=12, except *A. thaliana* n=6) across the eight Eudicot species is provided for two strains at the extremes of the host specificity/virulence distribution.

The co-evolutionary arms-race model is often applied to explain why the genomic architecture of virulence and host specificity in specialist pathogens is linked and defined by few large effect loci. To test if a similar model fits for Botrytis, we conducted a genome-wide association study (GWAS) on virulence and host-specificity in this pathogen. GWAS revealed that both overall virulence across eight Eudicots and host specificity are highly polygenic traits with respectively 4351 and 5705 significant SNPs at a conservative 99.9% threshold (Fig. 5). The significant SNPs are spread across 16 of the 18 chromosomes and are of small effects. Using the effect sizes of SNPs significant at the 99.9% threshold to create a genomic prediction vector was able to capture a significant fraction of the variance between the Botrytis strains virulence (R^2=0.74, P <2.2x10^{-16}, Fig. S4A) and host specificity (R^2=0.63, P <2.2x10^{-16}; Fig. S4B). None of the SNPs on chromosome 17 and 18, hypothesized to be potential disposable chromosomes, were associated with virulence or host specificity in this dataset (Fig. 5) (Bertazzoni et al., 2018). This suggests that in contrast to pathogens with high host specificity strains such as *Alternaria* or *Fusarium*, *Botrytis* doesn't have specific genomic structures for host specificity. The significant SNPs are located in 1479 genes associated with virulence and 1094 genes associated with host specificity (Supplementary Dataset), which represent 12 % of the *Botrytis* genes. Agreeing with the idea that virulence and host specificity can be separated is the observation that the genetic architecture of virulence and host specificity is largely different with only 9% of SNPs and 18% of genes associating with both traits.

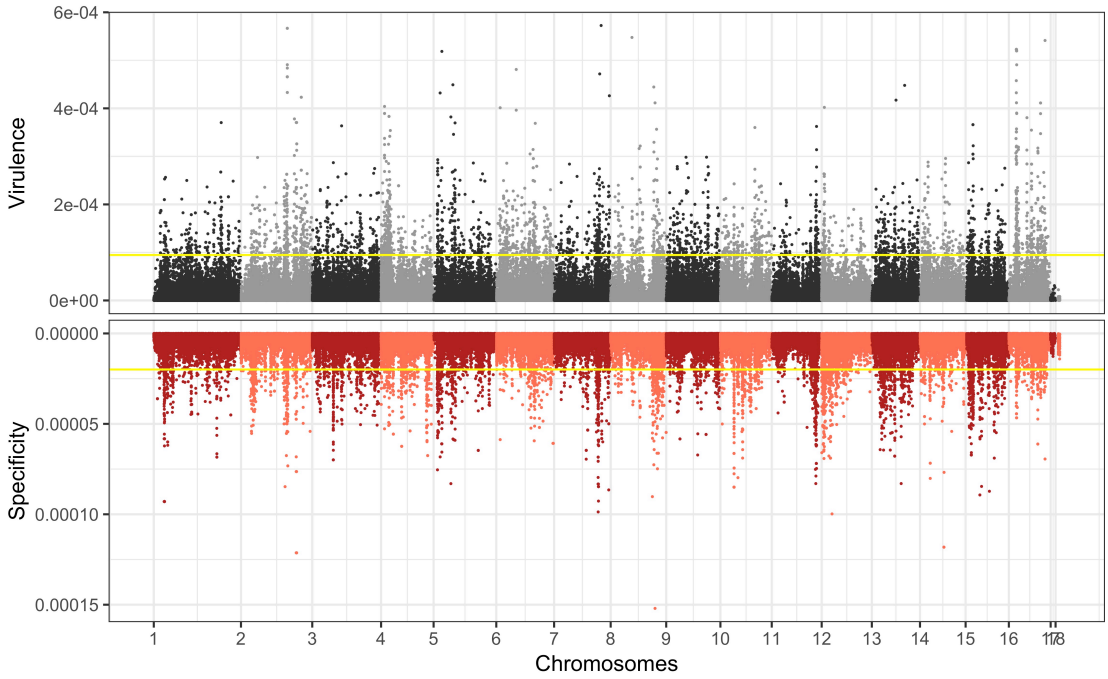

Fig. 5: *Botrytis* virulence and host specificity genetic architecture is polygenic. Effect size of 271,749 SNPs with reference to B05.10 genome estimated through ridge regression GWAS. In grey is plotted the effect on general virulence and in red is the effect on host specificity. The yellow lines represent the conservative significance threshold at 99.9% for each trait as determined by permutation.

Discussion

The co-evolutionary arms-race model has been successful at describing how large-effect virulence and resistance genes evolve and provided a framework for basic and applied research on resistance to specialist pathogens (Wu et al., 2018). However, tight host-specialist interactions are at one extreme end of the continuum of host-pathogen interactions (Barrett et al., 2009, Kirzinger and Stavrinides, 2012). In this study, we moved to the opposite end of the continuum and studied generalist pathogen-host interactions that are entirely quantitative in nature. This revealed the diversity of host-pathogen interactions and the limits of the current models at describing this diversity.

Botrytis interacts at a species level with the plants

We investigated how a generalist pathogen interacted with eight plant species across four plant orders from two clades in standardized conditions and how the standing variation in this pathogen may facilitate this process. First, we show that Botrytis virulence is largely defined by the diversity in resistance among the eight plant species (Fig. 2) with little relationship to evolutionary distances between the host species (Fig. 3). The absence of phylogenetic signal in the interaction with Botrytis is explained by the polygenic nature of both the host resistance and Botrytis virulence. It demonstrates that in quantitative systems, plant-pathogen interactions are governed by more than plant defense signaling and defense metabolisms but are constituted as multi-layer systems with components of small to moderate effects (Corwin and Kliebenstein, 2017). The additivity of these quantitative effects is enough to generate diverse resistance responses that change even among closely related species. Acknowledging the potential lack of phylogenetic signal in some host-pathogen interactions is important as the relation between species relatedness and susceptibility is a central assumption that determines risk assessment for ecological and agricultural decisions such as quarantines (Gilbert and Webb, 2007). This study shows that while this assumption may be true for host-pathogen interactions governed by qualitative traits, it should only be generalized with caution for the rest of the host-pathogen continuum.

Secondly, plant domestication has a significant but limited impact and no directional effect on the susceptibility to Botrytis. For a generalist pathogen with quantitative virulence like Botrytis, wild relatives of crop species may not consistently present increased resistance nor offer a potential pool of resistance to breed into cultivars. In addition, it should be considered that every single domestication events might have different effects on the susceptibility to a generalist pathogen.

All strains are not alike

By testing a collection of strains collected on various hosts from diverse geographical origins, we show that Botrytis contains the genetic diversity to display a wide range of virulence both across improvement status within species and between eudicot species (Fig. 1). While in specialist pathogens single genes to genomic islands or accessory chromosomes control host specificity (Kirzinger and Stavrinides, 2012), no such structures were detected in Botrytis (Fig.5). Instead, candidate genes associated to host specificity and virulence by the GWAS are spread across sixteen chromosomes (Supplementary Dataset).

The relationship between specialization and virulence is another aspect of the co-evolutionary model that *Botrytis* does not appear to follow. Specialization is often hypothesized as the most successful strategy for a pathogen, with generalism being selected against in natural environments due to the exponential cost of adaptation to counter innumerable host defenses (Barrett and Heil, 2012, Moller and Stukenbrock, 2017). This study shows that when quantifying host specificity, the vast majority of *Botrytis* strains are generalists with only a few individuals showing a propensity to prefer an individual host among the species tested. Critically, the strains that display host preference appear to have lost virulence on the non-preferred hosts rather than gained enhanced virulence on the preferred host. This could be favoured by host jumps at high frequency and low speciation rate (Navaud et al., 2018).

One potential model to explain these observations is that Botrytis holds extensive standing variation that allows Botrytis to overcome ecological challenges, from plant resistance to fungicides and environmental conditions (Walker et al., 2017, Atwell et al., 2015, Atwell et al., 2018). When coupled with recombination, the standing variation could generate quickly diverse virulence genes and alleles (McDonald and Linde, 2002, Woolhouse et al., 2001). In support to this hypothesis, Botrytis strains in the field showed extensive recombination occurring in a seasonal pattern (Fillinger and Elad, 2015, Walker et al., 2014). While more work is required to assess the evolution of virulence and host specificity in Botrytis, the relative lack of strains with any host-preference does suggest that the pathogen appears to be under pressure to maintain a generalist life style.

Specialist and generalist pathogens have different evolutionary trajectories

The general assumptions of the co-evolutionary arm-race model centered on host-specialist pathogens tested in this study did not display good fits with Botrytis virulence across the species tested. This suggests that pathogens on the specialist and generalist ends of the host-range continuum have very different evolutionary trajectories. However, it will require extensive work on other pathogens that occupy the remainder of the host-specificity continuum to test how these connect. Empirical

tests of other pathogens generalist at plant genera level to broad generalist such as *Sclerotinia*, *Fusarium*, *Verticilium* or *Pseudomonas* with and without strain specialization on large arrays of plant species in standardized conditions will allow a deeper development of how co-evolution changes across the host-specificity continuum.

While the gene-for-gene model works well for qualitative host-specialist pathogen interactions, a genome-for-genome model is required to apprehend the dynamic and resilience of generalist pathogens interactions. The Botrytis-Arabidopsis pathosystem recently highlighted such genome-for-genome interactions with metabolic battle between host and pathogen (Zhang et al., 2019, Zhang et al., 2017). Multi-species co-transcriptomic studies are now necessary to get an overview of the variability or conservation of such genome-for-genome interactions.

Methods

Plant material and growth condition

Eight species were chosen to represent a wide diversity of phylogenetic distance, geographical origin and histories across the core Eudicot (Fig. 1). For simplicity in the language, we refer to those as 'species' although referring to taxa would be more appropriate for tomato and lettuce for which we selected sister species for wild and domesticated genotypes. When referring to *Lactuca* (lettuce), *Lactuca sativa* was sampled for improved varieties and *Lactuca serriola* for wild accessions (Walley et al., 2017). When referring to *Solanum* (tomato), *Solanum lycopersicum* was sampled for improved varieties and *Solanum pimpenellifolium* for wild accessions native from Ecuador and Peru (Soltis et al., 2019, Lin et al., 2014).

This assay considers four Asterales species/taxa (*Helianthus annuus*, *Cichorium intybus*, *Cichorium endivia*, *Lactuca*), one Solanales (*Solanum*), one Fabales (*Glycine max*) and two Brassicales (*Brassica rapa, Arabidopsis thaliana*) (Fig. 1, Fig. 3, Table S2). *Arabidopsis thaliana* data was used as a reference for genotypes with altered plant susceptibility (Fig. S3). This reference dataset is composed of Col-0 and five knockout mutants altering plant immunity (Zhang et al., 2017, Atwell et al., 2018), through the jasmonic pathway (anac055, coi1), salicylic pathway (npr1, tga3) and camalexin pathway (pad3), an anti-fungal defense compound in *Arabidopsis*.

While the chosen species partially represent the Eudicot phylogeny (Fig. 1), they were also chosen to represent the diversity of plant domestication syndromes and geographical origins (Meyer et al., 2012) (Fig. S2). *H. annuus* and *G. max* were domesticated for seeds, *Solanum* for fruit (Lin et al., 2014) while *Lactuca*, *C. intybus* and *C. endivia* were domesticated for leaf and root (Dempewolf et al., 2008). All of these species underwent a single domestication events while *B. rapa* domestication is more complex (Bird et al., 2017). *B. rapa* was domesticated and re-selected multiple times for multiple traits including seed, leaf and root characteristics. For each species, twelve genotypes were selected, including six genotypes with low (wild or landrace) and six with high (cultivar, inbred lines) level of improvement (Table S2). The genotypes were chosen based on description of domestication status, and phylogeny for each species (Blackman et al., 2011, Walley et al., 2017, Lin et al., 2014, Dempewolf et al., 2008, Bird et al., 2017, Liang et al., 2014, Valliyodan et al., 2016). Landrace genotypes were selected for *C. endivia* for which no wild relative is known and two *B. rapa* genotypes (Fig. S2, Table S2). For soybean the comparison was within *G. max* as the growth behavior, vining, and growth conditions, tropical, for wild soybean, *G. soja*, was sufficiently different as to unnecessarily confound the comparison.

C. endivia, *C. intybus*, *B. rapa*, *G. max and A. thaliana* seeds were directly sowed in standard potting soil. *Solanum* and *Lactuca* seeds were bleach-sterilized and germinated on wet paper in the growth chamber using flats covered with humidity domes. After 7 days, the seedlings were transferred to soil. Seed surface sterilization and scarification was used for *H. annuus* to increase seed germination. Seeds were surface sterilized in 30% bleach for 12 minutes, followed by rinsing with sterilized distilled water, and then soaked in sterilized water for 3 hrs. ¼ of the seeds were cut off from the cotyledon end, then placed in 100 mg/L Gibberellic acid for 1 hour, followed by rinsing several times with sterilized distilled water. Treated seeds were then put in covered sterilized Petri dishes with wet sterilized germination disks at 4°C for 2 weeks and sowed.

All plants were grown in growth chambers in pots containing Sunshine Mix#1 (Sun gro Horticulture, Agawam, MA) horticulture soil at 20°C with 16h hours photoperiod at 100-120 mE light intensity. All plants were watered every two days with deionized water for the first two weeks and then with nutrient solution (0.5% N-P-K fertilizer in a 2-1-2 ratio; Grow More 4-18-38). All infection experiments were conducted on mature (non-juvenile) fully developed leaves collected on adult plants that grew in these conditions for four to eight weeks (Table S3) to account for the different developmental rates. As it is challenging to fully compare developmental stages across species (soybean stages are defined by nodes, sunflower by leaf size and so on), all leaves for the assays were collected on plants in the vegetative phase at least several weeks before bolting initiation to minimize ontogenetic effects.

Botrytis collection and growth condition

This study is based on a collection of 98 strains of *Botrytis cinerea.* The collection samples the *B. cinerea* strain diversity across fourteen plant hosts and in smaller degree across geographical origins. Ninety percent of the strains were isolated in California, largely in vineyards (70% of strains were collected on grape), while the remaining 10% of the collection are worldwide strains (Table S2). Although a large proportion of the strains were collected in California, the strain collection enfolds a large genetic diversity (Atwell et al., 2015, Atwell et al., 2018) and there is no significant effect on genetic diversity from either geographic or host species origin (Atwell et al., 2018). The spore collection is maintained for long-term preservation as conidial suspension in 60% glycerol at -80°C. The strains were grown for each experiment from spores on peach at 21°C for two weeks.

Detached leaf assay

To maximize the comparability across such a diverse collection of wild relatives and crop species domesticated for different trait, leaves were chosen as a common plant

organ. Detached leaf assays were conducted following (Corwin et al., 2016, Denby et al., 2004). The detached leaf assay methodology has been used for testing plant susceptibility to plant pathogens in more than 500 scientific publications and is considered a robust method when coupled with image analysis. In brief, leaves were cut and added to trays in which 1cm of 1% phyto-agar was poured. The phyto-agar provided water and nutrients to the leaf that maintained physiological functions during the course of the experiment. *Botrytis* spores were extracted in sterile water, counted with a hemacytometer and sequentially diluted with 50% grape juice to 10spores/ul. Drops of 4ul (40 spores of *Botrytis*) were used to inoculate the leaves. From spore collection to inoculation, *Botrytis* spores were maintained on ice to stop spore germination. Spores were maintained under agitation while inoculating leaves to keep the spore density homogeneous and decrease technical error. The inoculated leaves were maintained under humidity domes under constant light. To render a project of this size possible, a single time point was chosen to measure the plant-*Botrytis* interaction. Lesion area at 72 hours post infection (hpi) was chosen because the lesions are well defined on all targeted species but have not reached the edges of the leaves and thus are not tissue limited. Spore germination assays in 50% grape juice showed that strains germinate within the first 12 hours. On all species, the same pattern of growth was observed: after 48h most strains are visible within the leaf with the beginning of lesion formation. From 36h onward, Botrytis growth is largely linear and grows until the entire leaf is consumed (Rowe et al., 2010). The experiments included three replicates for each strain x plant genotype in a randomized complete block design and were repeated over two experiments, for a total of six replicates per strain x plant genotype.

Image analysis

The images analysis was performed with an R script as described in (Fordyce et al., 2018). Images were transformed into hue/saturation/value (hsv) color space and threshold accounting for leaves color and intensities were defined for each species. Masks marking the leaves and lesions were created by the script and further confirmed manually. The lesions were measured by counting the number of pixels of the original pictures within the area covered by the lesion mask. The numbers of pixels were converted into centimeter using a reference scale within each image.

Data quality control

A dataset of 51,920 lesions was generated in this project but not all leaves inoculated with *Botrytis* developed a visible lesion at 72hpi. These ‘failed lesions’ can be explained either by technical or biological failures. Technical failures, such as failed spore germination, stochastic noise or other non-virulence related issues can bias the true estimates of the mean. To partition biological from technical failures,

the lesion area distribution was analyzed for each species and empirical thresholds were fixed (Table S4). A lesion below that threshold was considered a technical error only if the median of lesion area for a plant genotype - strain pair was larger than the threshold. The rationale is the following: when most lesions are of small size, the likelihood of biological reasons for such small lesion areas is high, while when the majority of lesion areas are large, the likelihood of technical error is high. 6,395 lesions (13% of all lesions) were considered as technical failures and removed from the dataset. The statistical analyses and modeling were run on both original and filtered datasets. The removal of technical failures does not impact the effect size of the estimates but their significance and allowed for more variance to be attributed to biological terms in the model and less in the random error term. This is as expected if we are partitioning out predominantly technical failures.

Statistical analysis

All data handling and statistical analyses were conducted in R. Lesion area was modeled independently for each species using the linear mixed model with lme4 (Bates et al., 2014) :

Lesion.Area ~ Strain + Improv/PlantGeno + Strain*Improv + Strain*Improv/PlantGeno + (1|Exp.Replicate) + (1|Exp.Tray) + (1|Indiv.Plants)

Where plant genotypes are nested within improvement level. Experimental replicate and trays as well as the individuals plants on which were collected the leaves for the detached leaf assay are considered as random factors. Plant genotypes and *Botrytis* strains were coded as fixed factors, although they represent random sampling of the plant and fungal species. This simplification of the model was done because previous research (Corwin et al., 2016, Soltis et al., 2019, Fordyce et al., 2018) showed that this does not affect the effect size or significance of the estimates, while increasing dramatically the computational load. For each plant genotype, model corrected least-square means (LS-means) of lesion area were calculated for each strain from with Emmeans with the Satterwaite approximation (Lenth, 2018):

Lesion.Area ~ Strain + (1|Exp.Replicate) + (1|Exp.Tray) + (1|Indiv.Plants)

The meta-analysis model was run over all species LS-means with the linear model:

Lesion.Area ~ Strain + Species + Species/Improv + Species/PlantGeno + Strain*Species+ Strain*Species/Improv + Strain*Species/PlantGeno

Improvement levels and plant genotypes where nested within species to account for their common evolutionary history and possibly shared resistance traits of genotypes

with low and high levels of improvement. Variance estimates were converted into percentage of total variance to ease the comparison of the different models.

To visualize the relationship between strains and plant genotypes, a clustered heatmap was constructed on standardized LS-means with iheatmapr (Schep and Kummerfeld, 2017). The LS-means were standardized (z-score) over each plant genotypes by centering the mean to zero and fixing the standard deviation to one to overcome the large variation on lesion area across species and large variation in variance linked to the lesion area (Fig 1). Species with low lesion area had also small variance while species with large lesion area presented large variance. Seven strains that were not consistently infected on all 90 genotypes were dropped, as hierarchical clustering is sensitive to missing data. The unsupervised hierarchical clustering was run with the 'complete' agglomerative algorithm on Euclidean distances. The significance of the dendrogram was estimated with pvclust (Suzuki and Shimodaira, 2006) over 20000 bootstraps. The significance of branches was fixed at $\boldsymbol{\alpha}$ =0.95. For the plant genotypes dendrogram, branches were consistently assigned across hierarchical clustering methods (both 'complete' and 'average' algorithms were ran) and bootstrapping while in the *Botrytis* strains dendrogram, none of branches showed consistency.

The heatmap provides a global picture of how plant genotypes interact specifically with each *Botrytis* strains. To estimate the global virulence of each strain, we calculated the mean of the standardized LS-means of lesion area across the eight Eudicot species. The host specificity was calculated from the raw LS-means as the coefficient of variation (standard deviation corrected by the mean σ/μ) across the eight species (Poisot et al., 2012). Low host specificity indicates that strains grew consistently across the eight species, while high host specificity indicates large variation in lesion area across species (Fig. 4B), therefore preference for some species.

Genome-wide association

All strains were previously whole-genome sequenced at on average 164-fold coverage (Atwell et al., 2018). Host specificity and virulence were mapped using 271,749 SNPs at MAF 0.20 and less than 20% of missing calls with B05.10 genome as reference. The GWA was performed using a generalized ridge regression method for high-dimensional genomic data in bigRR (Shen et al., 2013). This method, which tests all polymorphisms within a single model, was shown to be adapted to estimate small effect polymorphism (Corwin et al., 2016, Fordyce et al., 2018). Due to the modeling of polymorphism as random factors, it imputes a heteroscedastic effect model as effect size estimates rather than p-values. Other GWA methods have been tested for mapping plant-*Botrytis* interactions *(Corwin et al., 2016, Atwell et al., 2018, Soltis et*

al., 2019) and hold comparable results to bigRR model. In particular, Gemma that accounts population structure does not perform significantly better due to the low population structure in the strain collection (Atwell et al., 2018). Furthermore, BigRR approach was chosen for its known validation rate (Corwin et al., 2016, Fordyce et al., 2018) in the pathosystem, the estimation of effect size, ability to perform permutation test on significance threshold and speed of calculation on GPU. Significance of the effect size was estimated based on 1000 permutations. The 99.9% threshold was used as a conservative threshold for SNP selection although 1000 permutations allow only an approximation of such a high threshold (Soltis et al., 2019). The genomic predictions were calculated by multiplying the effect size by their corresponding genotype and summing the resulting values over all SNPs for each strain. The genotypes were coded in reference to B05.10 genome where 0 was referring to B05.10 allele and 1 was different from B05.10. SNPs were annotated based on their location in ASM83294v1 assembly while gene annotation was extracted from the fungal genomic resources portal (fungidb.org).

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Acknowlegments: Seeds used in this study were provided by Laura Marek and Kathleen Reitsma at the US department of agriculture, Chris Pires, the UC Davis Tomato Genetics Resource Center, the Center for Genetic Resources Netherland (CGN) through Guy Barker and Graham Teakle. The experiments were performed with the help of Dihan Gao, Aysha Shafi, David Kelly, Matisse Madrone, Melissa Wang, Josue Vega, Aleshia Hopper and Ayesha Siddiqui. **Funding:** NSF IOS 1339125 and 1021861 to DJK.

Author contributions: Conceptualization and supervision: DJK; Data analysis: CC; Founding acquisition: DJK, Investigation: GS, CC, NS, RG, JC; Resources: SA; Writing: CC, DJK with final review and editing by all authors.

Author Information: The authors declare no competing interests. Correspondence and requests for materials should be addressed to Kliebenstein@ucdavis.edu. R codes and datasets are available on github.com/CaseysC/Eudicot_Rcode.

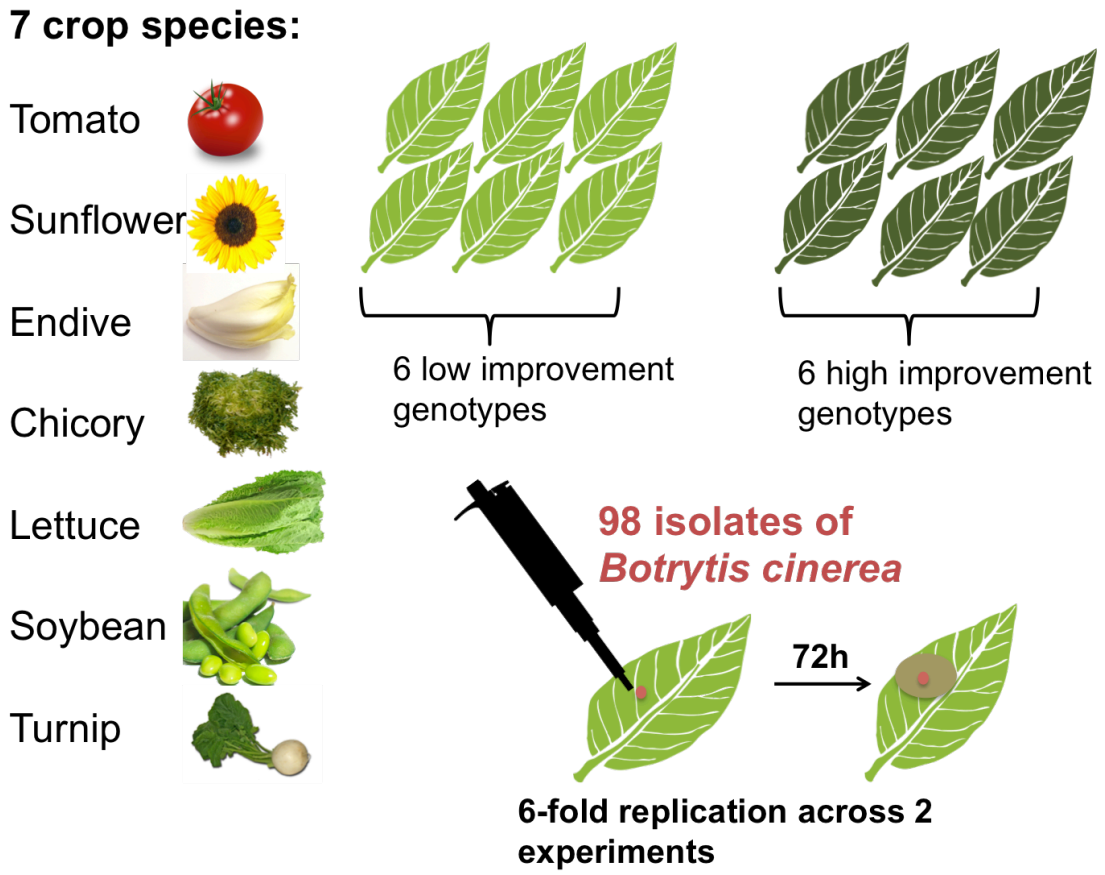

Fig. S1. Experimental Design. We tested the virulence of 98 *Botrytis cinerea* strains on seven Eudicot crop species using randomized complete block design detached leaf assays.

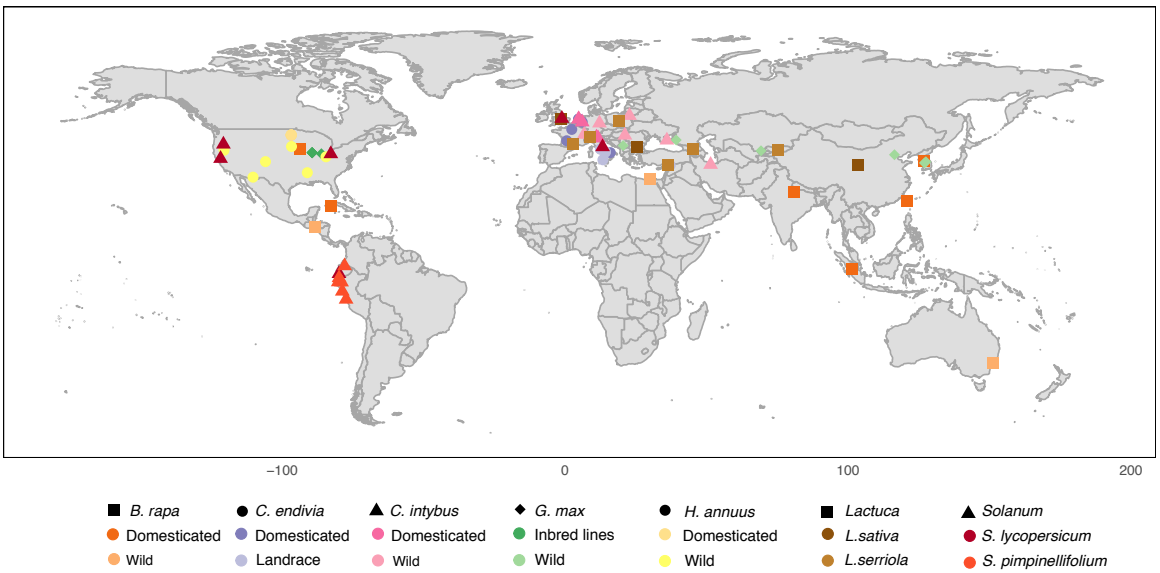

Fig. S2 | Geographical origin of the 84 Eudicot genotypes selected for this study. *B. rapa* samples come from all over the world, *C. endivia*, *C. intybus*, *Lactuca* and *G. max* come from Eurasia, *H. annuus* come from North America and *Solanum* from South America. Colored symbols represent the species and improvement status.

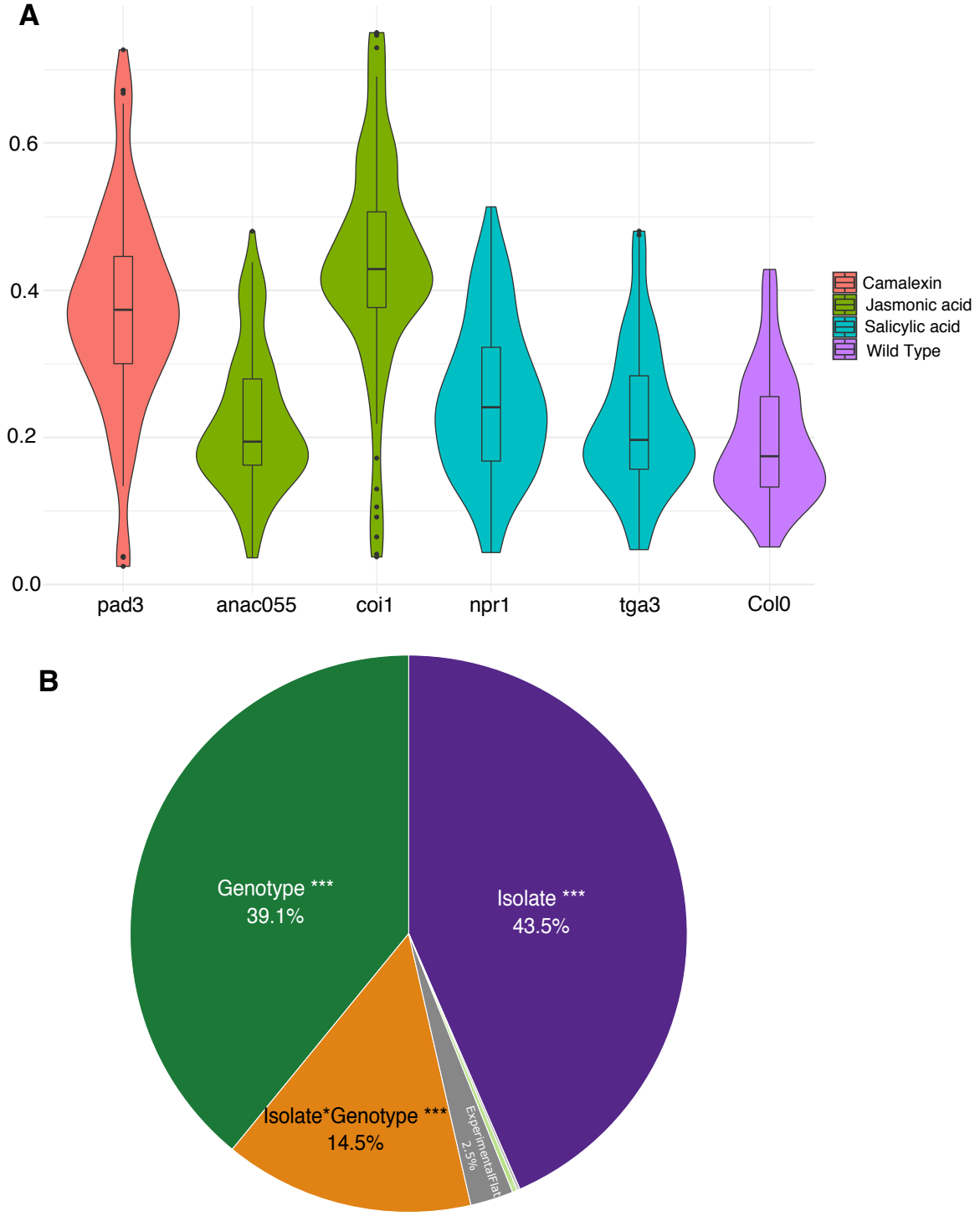
A
0.6
0.4
0.2
0.0
pad3
anac055
coi1
npr1
tga3
Col0
Camalexin
Jasmonic acid
Salicylic acid
Wild Type
B
Genotype ***
39.1%
Isolate ***
43.5%
Isolate*Genotype ***
14.5%
Experimental Fat
2.5%

Fig. S3. Variance in lesion area in *Arabidopsis thaliana.* A) Violin and boxplot represent the mean lesion area distribution for genotypes affecting camalexin synthesis (red), jasmonic acid signaling (green), salicylic acid signaling (blue) in comparison to the wild-type Col0 (purple) for all 98 Botrytis strains. B) Species-specific linear mixed model that estimate the percentage of variance in lesion area. In grey are the experimental parameters used as random factor. Two-tailed t-test: *$p<0.05$, **$p<0.01$, ***$p<0.005$.

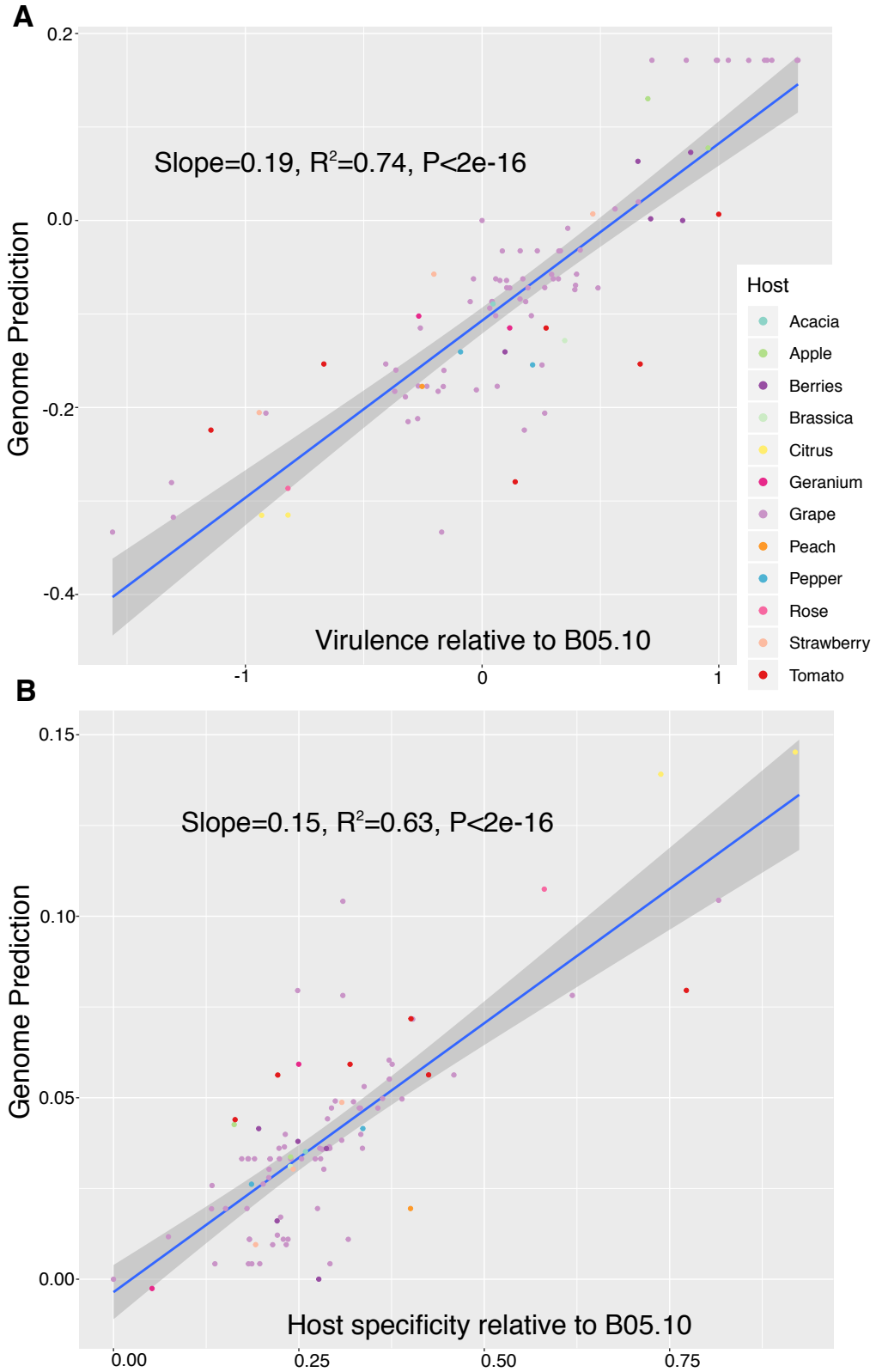
A
Slope=0.19, R²=0.74, P<2e-16
Genome Prediction
Virulence relative to B05.10
0.2
0.0
-0.2
-0.4
-1
0
1
Host
Acacia
Apple
Berries
Brassica
Citrus
Geranium
Grape
Peach
Pepper
Rose
Strawberry
Tomato
B
Slope=0.15, R²=0.63, P<2e-16
Genome Prediction
Host specificity relative to B05.10
0.15
0.10
0.05
0.00
0.00
0.25
0.50
0.75

Fig. S4. Linear model between genomic prediction based on significant SNPs at 99.9% threshold and phenotypes. Significance was calculated with a two-tailed t-test.

Table S1. Geographical origin and host at the time of collection for the 98 strains of *Botrytis cinerea*. A) Virulence relative to B05.10. B). Host specificity relative to B05.10. Dot colors represent the plant host Botrytis strains were collecting from

Botrytis Strains	Origin	Country	Host	Source
2004	California	USA	Grape	
01_01_01	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_01_02	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_01_03	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_01_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_01_06	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_01_15	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_01	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_02	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_03	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_05	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_06	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_13	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_15	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_16	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_17	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_18	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_02_20	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_02	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_12	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_16	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_18	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_19	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_20	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_22	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_03_23	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_01	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_02	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_03	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_05	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_12	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_15	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_17	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_19	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_20	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_21	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_04_25	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_11	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA

01_05_14	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_16	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_17	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_22	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
01_05_24	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_03	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_04	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_08	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_09	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_11	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_12	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_14	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_17	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_18	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_20	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
02_04_21	California	USA	Grape	Isolated 2011, J. Corwin, UC Davis, CA
94_1	California	USA	Lemon	D. Margosan, USDA, Parlier CA
94_4	California	USA	Orange	D. Margosan, USDA, Parlier CA
Acacia	SouthAfrica	SouthAfrica	Acacia	K. Denby, University of Warwick, UK
Apple404	California	USA	Apple	E. Mitcham, UC Davis, CA
Apple517	California	USA	Apple	E. Mitcham, UC Davis, CA
Ausubel	Massachusetts	USA	Brassica	J. Plotnikova, Massachusetts General Hospital, MA
B05_10	Netherlands	Netherlands	Grape	J. Van Kan, Wageningen University, NL
BMM	Switzerland	Switzerland	Geranium	O. Lamotte, University of Fribourg
BPA1	California	USA	Grape	Isolated 2005, D. J. Kliebenstein
DavisNavel	California	USA	Orange	Isolated 2004–05, H. C. Rowe
EsparatoFresa	California	USA	Strawberry	Isolated 2004–05, H. C. Rowe
Fd1	California	USA	Raspberry	Isolated 2004–05, H. C. Rowe
Fd2	California	USA	Raspberry	Isolated 2004–05, H. C. Rowe
Fresa525	California	USA	Strawberry	E. Mitcham, UC Davis, CA
FresaSD	California	USA	Strawberry	E. Mitcham, UC Davis, CA
Gallo1	California	USA	Grape	Isolated 2004–05, H. C. Rowe
Gallo2	California	USA	Grape	Isolated 2004–05, H. C. Rowe
Geranium	California	USA	Geranium	Isolated 2004–05, H. C. Rowe
Grape	SouthAfrica	SouthAfrica	Grape	M. Vivier, University of Stellenbosch
KatieTomato	California	USA	Tomato	Isolated 2004–05, H. C. Rowe
KernA2	California	USA	Grape	D. Gubler, UC Davis, CA
KernB1	California	USA	Grape	D. Gubler, UC Davis, CA
KernB2	California	USA	Grape	D. Gubler, UC Davis, CA
KGB1	California	USA	Tomato	Isolated 2004–05, H. C. Rowe
KGB2	California	USA	Tomato	Isolated 2004–05, H. C. Rowe
MEAP6G	Switzerland	Switzerland	Grape	M. Dewey, UC Davis, CA
Mex03	Mexico	Mexico	Blackberry	Isolated 2012, J. Corwin, UC Davis, CA
Molly	California	USA	Grape	M. Dewey, UC Davis, CA
Navel	California	USA	Orange	Isolated 2004–05, H. C. Rowe
NobleRot	California	USA	Grape	G. Allen, Far Niente Winery, CA
Peachy	California	USA	Peach	Isolated 2004–05, H. C. Rowe
Pepper	UK	UK	Pepper	G. Loake, University of Edinburgh
PepperSub	UK	UK	Pepper	K. Denby, University of Warwick, UK
PhiloMenlo	California	USA	Grape	D. Gubler, UC Davis, CA
Rasp	California	USA	Raspberry	Isolated 2004–05, H. C. Rowe
Rose	California	USA	Rose	D. Margosan, USDA, Parlier CA
Supersteak	California	USA	Tomato	Isolated 2006, H. C. Rowe
Triple3	California	USA	Tomato	Isolated 2004–05, H. C. Rowe
Triple7	California	USA	Tomato	Isolated 2004–05, H. C. Rowe
UKRazz	UK	UK	Raspberry	Isolated 2004–05, H. C. Rowe

Table S2. Genotype information for 84 accessions of seven crop species.

ID Number	Germplast Bank	Full species name	Order	Clade	Improvemen	Domestication
PI.347594	USDA Grin	Brassica rapa subsp. dichotoma	Brassicales	Rosids	high	Domesticated
PI.508409	USDA Grin	Brassica rapa subsp. Chinensis	Brassicales	Rosids	high	Domesticated
PI.633169	USDA Grin	Brassica rapa L. subsp. oleifera (DC.) Metzg.	Brassicales	Rosids	high	Domesticated
PI.649204	USDA Grin	Brassica rapa L. subsp. trilocularis (Roxb.) Hanelt	Brassicales	Rosids	high	Domesticated
PI.662686	USDA Grin	Brassica rapa L. subsp. rapa	Brassicales	Rosids	high	Domesticated
PI.662783	USDA Grin	Brassica rapa var. parachinensis	Brassicales	Rosids	high	Domesticated
PI.234600	USDA Grin	Brassica rapa subsp. pekinensis	Brassicales	Rosids	low	Landrace
PI.597831	USDA Grin	Brassica rapa subsp. oleifera	Brassicales	Rosids	low	Wild
PI.633181	USDA Grin	Brassica rapa L. subsp. rapa	Brassicales	Rosids	low	Wild
GC 1742-69	Chris Piers (U. Missouri)	B. rapa sylvestris	Brassicales	Rosids	low	Wild
GC 4623-77	Chris Piers (U. Missouri)	B. rapa sylvestris	Brassicales	Rosids	low	Wild
R500	Chris Piers (U. Missouri)	B. rapa	Brassicales	Rosids	Low	Landrace
PI.393808	USDA Grin	Cichorium endivia L.	Asterales	Asterids	high	Domesticated
PI.393811	USDA Grin	Cichorium endivia L.	Asterales	Asterids	high	Domesticated
PI.503585	USDA Grin	Cichorium endivia	Asterales	Asterids	high	Domesticated
PI.651905	USDA Grin	Cichorium endivia	Asterales	Asterids	high	Domesticated
PI.651906	USDA Grin	Cichorium endivia	Asterales	Asterids	high	Domesticated
PI.651915	USDA Grin	Cichorium endivia	Asterales	Asterids	high	Domesticated
PI.273578	USDA Grin	Cichorium endivia subsp. pumilum	Asterales	Asterids	low	Landrace
PI.652010	USDA Grin	Cichorium endivia L.	Asterales	Asterids	low	Landrace
PI.652011	USDA Grin	Cichorium endivia L.	Asterales	Asterids	low	Landrace
PI.652013	USDA Grin	Cichorium endivia L.	Asterales	Asterids	low	Landrace
PI.652014	USDA Grin	Cichorium endivia L.	Asterales	Asterids	low	Landrace
PI.652049	USDA Grin	Cichorium endivia	Asterales	Asterids	low	Landrace
PI.651886	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.651945	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.651971	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.652038	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.652041	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.652045	USDA Grin	Cichorium intybus L.	Asterales	Asterids	high	Domesticated
PI.652006	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
PI.652008	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
PI.652019	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
PI.652021	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
PI.652026	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
PI.652028	USDA Grin	Cichorium intybus L.	Asterales	Asterids	low	Wild
CL0J173-6-8		Glycine max (L.) Merr.	Fabales	Rosids	high	Domesticated
LD02-4485		Glycine max	Fabales	Rosids	high	Domesticated
LG04-4717		Glycine max	Fabales	Rosids	high	Domesticated
LG05-4832		Glycine max	Fabales	Rosids	high	Domesticated

LG98-1605		Glycine max	Fabales	Rosids	high	Domesticated
NE3001		Glycine max	Fabales	Rosids	high	Domesticated
PI.398881	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.404188A	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.427.136	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.437.169B	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.507681B	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.518751	USDA Grin	Glycine max (L.) Merr.	Fabales	Rosids	low	Wild
PI.599764	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.599782	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.633747	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.642777	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.664207	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.667183	USDA Grin	Helianthus annuus L.	Asterales	Asterids	high	Domesticated
PI.413153	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
PI.435584	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
PI.468627	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
PI.597899	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
PI.649853	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
PI.664809	USDA Grin	Helianthus annuus L.	Asterales	Asterids	low	Wild
LJ10318	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ10320	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ10326	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ10332	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ10335	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ14005	CGN	Lactuca sativa	Asterales	Asterids	high	Domesticated
LJ10400	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LJ10405	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LJ10409	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LJ14025	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LJ14033	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LJ14038	CGN	Lactuca serriola	Asterales	Asterids	low	Wild
LA2706	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA3008	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA3475	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA0410	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA4345	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA4355	UC Davis TGRC	Solanum lycopersicum	Solanales	Asterids	high	Domesticated
LA1547	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild
LA1589	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild
LA1684	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild
LA2093	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild
LA2176	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild
LA0480	UC Davis TGRC	Solanum pimpinellifolium L.	Solanales	Asterids	low	Wild

Species	Germination	Soil growth
B.rapa	Direct sowing	4 weeks
C.endivia	Direct sowing	4 weeks
C.intybus	Direct sowing	4 weeks
G.max	Direct sowing	4 weeks
H.annuus	Scarification +2 weeks	4 weeks
Lactuca	On wet paper 1 week	8 weeks
Solanum	On wet paper 1 week	6 weeks
Arabidopsis	Direct sowing	5 weeks

Table S3. Germination and growth conditions for the eight Eudicot species. All plants were grown in growth chambers at 20°C with 16h hours photoperiod at 100-120 mE light intensity for four to eight weeks to account for the different developmental rates.

Species	Lesion threshold [cm2]	Median threshold [cm2]	Data removed
B.rapa	0.15	0.2	6%
C.endivia	0.2	0.25	8%
C.intybus	0.2	0.25	10%
G.max	0.15	0.2	17%
H.annuus	0.3	0.35	5%
Lactuca	0.15	0.2	14%

Solanum	0.15	0.2	19%
Arabidopsis	0.15	0.2	26%
Average			13%

Table S4. Maximum lesion size and median thresholds. These thresholds were used for the partitioning of technical and biological failures in developing lesions as well as the percentage of data points removed in each of the eight Eudicot species.

Supplementary information: Information on the 1773 genes identified by GWAS for virulence and host specificity and their location in the B05.10 reference genome.