**Results**

**Experimental Design**

We wanted to directly measure the impact of tomato domestication and genetic variation on quantitative resistance. To measure quantitative resistance, we infected tomato leaflets with a collection of 91 diverse *B. cinerea* isolates. *B. cinerea* is an endemic necrotroph, and host resistance to this generalist pathogen is quantitative, with no evidence of qualitative defense loci (CITE). Previous studies have examined the contrast in *B. cinerea* resistance between wild and domesticated tomato using distantly related species such as *S. chilense* (Nicot, Moretti et al. , Ten Have, van Berloo et al. 2007), *S. chmielewskii* (Nicot, Moretti et al.), *S. habrochaites* (Finkers, van Heusden et al. 2007, Ten Have, van Berloo et al. 2007), *S. hirsutum* (Egashira, Kuwashima et al. 2000, Nicot, Moretti et al.), *S. lycopersicoides* (Guimaraes, Chetelat et al. 2004), *S. neorickii* (Ten Have, van Berloo et al. 2007, Finkers, Bai et al. 2008), *S. peruvianum* (Egashira, Kuwashima et al. 2000, Nicot, Moretti et al.), *S. pennellii* (Nicot, Moretti et al.) and *S. pimpinellifolium* (Egashira, Kuwashima et al. 2000, Nicot, Moretti et al.). These single-isolate studies found a wide range of pathogen susceptibility levels both within and between tomato species, though none of the studies directly compared wild versus domesticated genotypes. We selected *S. pimpinellifolium*, the closest wild relative of *S. lycopersicum*, to directly study the selection associated with the impact of domestication. We selected tomato genotypes including 6 domesticated *Solanum lycopersicum* cultivars and 6 wild *S. pimpinellifolium* genotypes. We infected all 91 *B. cinerea* isolates onto each plant genotype in 3-fold replication across 2 independent experiments in a randomized complete block design, giving 6 measurements per plant-pathogen combination, for a total of 3,276 lesions. We digitally measured the area of the developing lesion at 72 hours post infection (HPI) (Figure R1). At 72 hours, significant lesion growth was visible, but no lesions had grown to completely consume infected leaflets. Lesion area is a composite phenotype from the interaction of host and pathogen genetics that has been utilized in a number of studies on the molecular and quantitative genetic basis of plant-*Botrytis* interactions {Rowe 2008}.

We performed statistical analysis of lesion size with a generalized linear model (GLM). Within the model, we tested the fixed effects of isolate genotype, plant species (domesticated or wild), plant genotype (which is nested within species), and position of sampled leaflet (apical or basal). We also considered the random effects of experiment, block (nested within experiment), individual plant, and individual leaf (nested within sample plant). The terms for individual plant, leaf, and leaflet position did not significantly improve the model, so we omitted them from further analysis. Our final model also included the interaction terms of isolate by plant species, and isolate by plant genotype (nested within species). The final model shows that genetic variation within both the host plant and the pathogen affect lesion growth (Table R1). Domestication also impacted lesion formation, as shown by the significant effects of tomato genetic variation between domesticated and wild species. We did not find evidence for significant interaction effects between isolate and plant genotypes. This may have been due to the vast number of degreese of freedom, as the isolate-plant interactions contribute a large proportion of the variance in lesion size (Table R1).

**Domestication and lesion area**

Existing literature, largely studying qualitative resistance to biotrophic pathogens, has proposed that domestication increases susceptibility to pathogens and decreases plant genetic variation for disease resistance due to selection bottlenecks during domestication (CITE).

In agreement with domestication theory, lesion size is significantly greater on average (18% increase) on domesticated tomato compared to wild tomato (p = XXX, Table R1) (Figure R2).

In contrast to theory, the domesticated tomato genotypes had a wider range of average lesion formation than wild genotypes (5% to 95% interval: 2.03 on domesticated, 1.76 on wild).A domestication bottleneck would lead to reduced variation for lesion size across domesticated tomato genotypes; instead we observe an increased range of lesion sizes in domesticated compared to wild tomato. Additionally, the coefficient of variation (CV) of lesion size cannot be statistically differentiated between wild and domesticated tomato (Wilcoxon signed-rank test, V=2275, p=0.7163), indicating a lack of evidence for a domestication effect on lesion size variance (Figure R3). Overall, we see evidence for a slight domestication impact on *Botrytis cinerea* defense, but

**Host variation and lesion area**

Domestication does impact lesion size, but most variance is due to genetic variation in the isolate and in host plant genetics (Table R1).

**Pathogen**

Our 91 B. cinerea genotypes were isolated from various eudicot plant hosts, including tomato stem tissue (2 isolates; T3, KT) and tomato fruit (3 isolates; KGB1, KGB2, Supersteak)

Isolates collected from tomato stem or fruit tissue are not among the most virulent group (Figure R4F). For *B. cinerea* genotypes isolated from tomato tissue vs. other hosts, there is no significant difference in lesion size across all hosts on either domesticated (t-test; t=-1.10, 4.3 df, p=0.330) or wild (t-test; t=-1.09, 4.2 df, p=0.332) tomato. In fact, one isolate collected from tomato tissue (KGB1) is within the 10 least-virulent isolates (Figure R4F). This may suggest a generalist strategy for individual isolates, due to this apparent lack of host-specificity.

**Host-pathogen interactions**

**Domestication**

We also tested whether genetic differences between wild and domesticated hosts interact with pathogen genetics to determine lesion size. Lesion size varies across host for many of the isolates, suggesting an interaction between the genomes of *B. cinerea* and tomato (Figure R4). However, domestication did not have a significant interaction effect with isolate genotype (Table R1). This is likely due to the many degrees of freedom in calculating this interaction effect.

Second, isolate ranking by mean lesion size differs between domesticated and wild hosts (Wilcoxon signed-rank test, V=4322, p=2.586e-12) (Figure R3).

**Per genotype**

Tomato genotype within each species did not have a significant interaction effect with isolate genotype (Table R1). The F-test could identify significant effects of X,Y,Z but not the interaction between them, because F-tests with high numbers of degrees of freedom can be significantly underpowered, like in the case of the isolate x plant genotype interaction term (df: XXX). We took an additional approach to statistically test for an interaction between *B. cinerea* and host genotype. We split the dataset by isolate, and within each new dataset performed GLM ANOVA with the fixed effects of domestication and plant genotype nested within domestication, and the random effect of experiment. Through this single-isolate GLM analysis, a subset of isolates show a significant (p < 0.05) interaction with host genotype (Figure R4E). \*\*still need to use FDR correction\*\*.

**FIGURES**

Figure R1. Will be an image of the detached leaf assay and leaf/ lesion calls.

Figure R2. Violin plots of lesion size due to Botrytis cinerea growth on tomato host genotypes. Plots include all replicates of lesion size measurements across isolates.

Figure R3. Interaction plot for domestication. Each line traces the average lesion size for a single Botrytis isolate.

Figure R4. Interaction plot of lesion size due to individual Botrytis cinerea isolates on tomato host genotypes. Each line traces the average lesion size across plant genotypes for a single Botrytis isolate.

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