**IN BRIEF**

**97 Shades of Grey: Genetic interactions of the grey mold, *Botrytis cinerea*, with wild and domesticated tomato**

Although a vineyard full of decaying grapes infected with noble rot is a blessing for sweet wine producers, the causal agent, *Botrytis cinerea* (grey mold), is responsible for huge crop losses globally both pre- and post-harvest. Individual isolates of the necrotrophic fungus can infect an extremely broad host range, which includes cereals, fruits, vegetables and ornamentals (Elad et al., 2015). Such host promiscuity is due, in part, to the high level of genetic variation observed among *B. cinerea* isolates in multiple infection mechanisms which, when coupled with polygenic resistance in the host plant, results in quantitative variation in virulence (Rowe and Kliebenstein, 2008).

A new study by Soltis and colleagues utilises the tomato-*B. cinerea* pathosystem to examine in detail how genetic diversity in both the host plant and a generalist pathogen interact to impact resistance/susceptibility. A collection of 6 domesticated (*Solanum lycopersicum*) and 6 wild (*S. pimpinellifolium*) tomato accessions were infected with 97 *B. cinerea* isolates and the resulting lesion areas were measured.

General linear modelling of this dataset showed that lesion size was significantly and equally affected by tomato genotype and pathogen isolate. More surprising, however, was the finding that tomato domestication status was associated with only a small, albeit significant, increase in susceptibility to the pathogen. Generally, crop domestication not only decreases resistance to pathogens but also reduces genetic diversity in comparison to wild relatives, which is exemplified by lower phenotypic variation. In this study, however, such phenotypic bottlenecks were not observed, leading the authors to conclude that in tomato at least, domestication makes only a minor contribution to the virulence of *B. cinerea*. In testimony to the unfathomable host range of *B. cinerea*, the authors also found little evidence of isolate specificity to tomato.

So, what then is the genetic basis for the observed variation in *B. cinerea* virulence on different tomato accessions? Genome-wide association (GWA) mapping across the 97 pathogen isolates was employed to identify *B. cinerea* loci contributing to altered virulence, with each of the 12 tomato genotypes treated as independent traits for the purposes of the analysis. Specialist pathogens that infect a very narrow host range (often limited to a single species) frequently have only a handful of large-effect qualitative loci controlling virulence. In contrast, this GWA analysis revealed that the underlying genetic basis for the observed quantitative virulence of this generalist pathogen was highly polygenic, with numerous small-effect single nucleotide polymorphisms (SNPs) associated with lesion area. This is directly akin to the polygenic genetic architecture of resistance in the host plant, as previously described by this group (Zhang et al., 2017). Crucially, few of these SNPs were found to be significantly linked to *B. cinerea* virulence on all of the tomato accessions supporting the existence of host x pathogen genotypic interactions.

From the GWA mapping the authors were able to identify several hundred *B. cinerea* genes linked to differential resistance on tomato, including several novel virulence candidates. Interestingly, detailed positional examination of the multiple SNPs within one of these genes (encoding a pectinesterase), revealed that most were located in upstream regulatory regions suggesting that different *B. cinerea* isolates may deploy differential transcriptional regulation of downstream virulence-related mechanisms.

Two of the *B. cinerea* isolates exhibited significantly increased virulence on the domesticated tomato cultivars versus their wild relatives. Further modelling of these specific isolates identified a subset of pathogen loci, mainly involved in enzymatic and transport functions, that are highly responsive to tomato domestication, and potentially to domestication of other plant host species too.

Taken together, the findings of Soltis and coworkers truly highlight the tenacity of *B. cinerea* as a plant pathogen! Different isolates of the generalist pathogen are capable of intermating meaning that the fungus has at its disposal a huge repertoire of polymorphic virulence loci allowing it to not only customise its virulence according to host genotype but also enabling it to rapidly adapt to any newly evolved plant defense mechanisms. And if this weren’t enough, *B. cinerea* additionally possesses the ability to shift to a new host niche. Thus, breeding durable resistance to *B. cinerea* in the field is extremely challenging and will likely require a genome-wide appreciation of virulence/resistance at the level of both the pathogen and the host.

**Emily Breeze**

**School of Life Sciences,**

**University of Warwick, Coventry, UK**

[**emily.breeze@warwick.ac.uk**](mailto:emily.breeze@warwick.ac.uk)

**ORCID ID: 0000-0001-5383-5448**

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**Figure legend:**

***Botrytis cinerea* x tomato diversity.** Individual leaflets from 6 domesticated (*Solanum lycopersicum*) and 6 wild (*S. pimpinellifolium*) tomato accessions were inoculated with 97 *B. cinerea* isolates and lesion areas quantified. Domesticated tomato accessions exhibited decreased resistance over their wild relatives with 80% of the pathogen isolates being more virulent on domesticated tomato leaves.

*[Adapted from Soltis et al., (2019), Figures 1 and 3.]*

