The Irish Potato Famine pathogen, *Phytophthora infestans* (Mont.) de Bary, continues to emerge causing epidemics globally. However, mechanisms that produce clonal lineages that are highly virulent remain uncharacterized. We used high throughput sequencing, from previously published sources as well as our own sequencing, to infer copy number variation, based on 200 kbp sliding windows of heterozygous sites, for *P. infestans* genomes from a global sample of isolates. Instead of observing individuals that were predominantly diploid or triploid we observed individuals that represented a full spectrum of intermediate states from diploid to triploid. Many of the clonal lineages that have led to epidemics exhibit copy number increases, including US-1, US-8, US-11, US-23, and US-24. However, the lineage that is thought to have caused the Great Famine appeared diploid. Isolates collected from Mexico, the center of genetic diversity for *P. infestans* and a region where it is characterized as exhibiting a sexual mode of reproduction, were also predominantly diploid. These results indicate that no simple assumption about the role of copy number variation in *P. infestans* biology can be made and highlights the importance of inference of copy number prior to genetic analysis. Our results present a new perspective of *P. infestans* epidemics as frequently being accompanied by changes in copy number that may contribute to the pathogenicity of the lineage.

Deciphering the mechanism of *E. coli* resistance to a membrane-targeting antimicrobial peptide through genomic and transcriptomic approaches

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Antimicrobial peptides (AMPs) are essential components of host innate immunity, representing the first line of defense in bacterial clearance. However, bacteria can develop resistance to AMPs. Using *Escherichia coli* strain BL21-Gold (DE3) as a model, we investigated the mechanism of bacterial resistance to AMPs. The strain was allowed to evolve resistance against an amphipathic 11 residue helical peptide (or P11). The minimal inhibitory concentration (MIC) of the resistant strain is 13-fold higher than that of the wildtype (or susceptible) strain. Genome sequencing of the resistant *E. coli* derivative revealed insertions and deletions in several genes. Through comparative genome analysis, we detected transposase insertions in genes involved in outer membrane (*asmA*) and lipopolysaccharide (*waaP*) biosynthesis. The *asmA* gene encodes assembly protein asmA, which is involved outer membrane assembly; whereas *waaP* encodes lipopolysaccharide core heptose (I) kinase, required for the heptose phosphorylation in lipopolysaccharide (LPS) core. We also detected a transposase insertion in the *Dihydrouridine synthase C* (*dusC*) gene, which encodes tRNA-dihydrouridine synthase. Knocking out these three genes resulted in 3-fold increase of the MIC compared to the control. Several mutations in genes that encode proteins are involved in interactions with phospholipids and membrane permeabilization of P11. Overall, our data suggest the collective action of genic and intergenic mutations contribute to resistance. Based on these observations, we designed a next-generation, 26 residue AMP (P26) that overcomes the bacterial resistance mechanism. Transcriptome profiling analysis is underway and should also be highly informative.

Exploring the genome of *Metschnikowia fructicola*, a biocontrol yeast effective against postharvest diseases

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The yeast *Metschnikowia fructicola* has been reported as an efficient biocontrol agent of postharvest diseases of fruit. The mechanisms of action by which *M. fructicola* inhibits postharvest pathogensinclude iron-binding compounds, induction of defence signalling genes, such as PRP and MAPK cascade genes, production of fungal cell wall degrading enzymes and relatively high amounts of superoxide anions. *M. fructicola* also exhibits chitinase activity and the chitinase gene, *MfChi,* was highly induced in response to fungal pathogen cell walls. Several studies have examined differential gene expression during the interaction of the yeast, *M. fructicola*, with a host fruit or a postharvest pathogen. In the current work, we report the assembly of the whole genome sequence of two strains of *M. fructicola* using PacBio and Illumina shotgun sequencing technologies. Using the PacBio, a high-quality draft genome consisting of 93 scaffolds, with an estimated genome size of approximately 26 Mb, was obtained. Comparative analysis of *M. fructicola* proteins with three available closely-related genomes revealed a shared core of homologous proteins. Comparing the genomes of the two *M. fructicola* strains using a SNP calling approach resulted in the identification of 564,302 SNPs/indels with a total of 2,004 predicted high impact mutations. Based on the assembled genome, sequences were annotated with gene description and gene ontology and clustered in functional groups. Analysis of CAZyme family genes revealed 1,145 putative genes. Transcriptomic analysis of CAZymes in *M. fructicola* during its interaction with either grapefruit peel tissue or *Penicillium digitatum* revealed a high level of CAZyme gene expression when the yeast was placed in wounded fruit tissue. The significance of the findings in biocontrol capabilities of *M. fructicola* will be discussed.

Investigating effector diversity as a source of cultivar-specific pathogenicity across global isolates of the lettuce bacterial leaf spot pathogen

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Bacterial Leaf Spot of lettuce has a 100-year history in the US, and in the 1990's it reemerged as a significant threat that breeding programs struggled to counter. Multilocus sequence analysis of global *Xanthomonas campestris* pv. *vitians* (*Xcv*) isolates using four housekeeping genes and evaluation of plant-pathogen interactions has revealed six groups with cultivar-specific resistance. We hypothesize that differences in effector repertoires cause this variation in cultivar-specificity across the genotypic groups. To test this, the genomes of 21 representative isolates of *Xcv* and relevant types and pathotypes were sequenced, assembled, and annotated. Barcoded libraries were created for each isolate using 2 µg of DNA extract and the Illumina TruSeq DNA PCR-Free kit. An equimolar pool of the libraries for each isolate was sequenced using Illumina MiSeq, generating 250 nt, paired-end reads. Yield was 185 Mb per sample; with the *Xcv* genome size of 5 MB, that computed to 37x coverage. The sequence data was aligned to a reference genome for assembly and annotation. Bioinformatics tools are being employed to identify sequences belonging to any of the more than 30 known effectors of *Xanthomonas* plant pathogens. Once these effector repertoires are determined, we will evaluate correspondence between effector repertoire composition and host reaction. This knowledge can be applied in development and deployment of lettuce with resistance across all genotypes of *Xcv*.

Evolution of necrotrophic phytopathogenic bacteria in the Enterobacteriaceae

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The emergence of plant pathogenesis by bacteria is the product of convergent evolution. Within the Enterobacteriaceae alone, two groups of bacteria have independently evolved the ability to cause plant disease. Soft rot Enterobacteria (SRE) represent one of these groups and include three genera: *Pectobacterium*, *Dickeya*, and *Brennaria*. While much research has focused on biotrophic bacterial plant pathogens, SRE pathogenicity is characterized by necrotrophy and the production of cell wall-degrading enzymes. Here we report a comparative genomic analysis of the Enterobacteriaceae to elucidate the evolution of necrotrophy in SRE. In total, 307 Enterobacteriaceae genomes were analyzed including 20 genomes from *Pectobacterium*,