Predicting the emergence of host-adapted bacterial phytopathogens

Bacterial phytopathogens are hard to control and therefore pose a high risk to crop production and the wider natural environment. In order to mitigate that risk, we propose multidisciplinary research into the tempo at which and the mechanism by which bacteria adapt to hosts. Our findings will support new developments in disease management and control strategies.

The host range of a pathogen encompasses all the species it can successfully infect and colonise. It is hypothesised that plant species outside this range mount an effective non-host resistance response, preventing colonisation. Plant pathologists traditionally define some pathogens as having a wide host range (generalists) whilst others are limited to one or a few hosts (specialists), although a continuum between these two life strategies probably exists. Our work focuses on the ubiquitous bacterial species complex *Pseudomonas syringae* (Ps), lineages of which are pathogens of over 300 different plant species. At least eight lineages of Ps are known to cause bacterial canker of cherry trees including the recognised pathogens *Ps pv. morsprunorum* and *Ps pv. syringae*.

From our preliminary work, sampling from the leaf and shoot surface across cherry orchards around the UK, we have shown that there are large regional variations in Ps populations. We have found that, in addition to the known pathogens, many additional lineages of Pseudomonas (which have the potential to be pathogenic) are present on non-diseased cherry leaves and such strains are extremely widespread across orchards and regions. These epiphytic (surface) populations of pseudomonads may either be donors or repositories of bacterial genes predicted to have a key role in host adaptation to Prunus. We have shown that there is significant variation in resistance to canker within cherry cultivars and also between genotypes of wild cherry. What we do not know is whether or not the epiphytic populations of Ps are similar or variable between cultivated and wild cherry hosts, or if there is a flow of bacterial strains between wild and cultivated cherry.

In this multidisciplinary research proposal, we will extend our initial experiments to study the ecological niches occupied by Ps. Using repeated sampling and genome sequencing of isolates from cultivated crops and surrounding plant species, we will determine if epiphytic Ps populations, some of which contain known pathogens, are stable over time and space. In controlled field experiments we will also ask whether agronomic interventions, such as nitrogen rates and polytunnel covering of crops also play a role in shaping bacterial populations.

Our previous work has shown that key genes have been transferred between Pseudomonas lineages by phages and plasmids. In order to explore the molecular factors that may affect virulence and lead to new disease outbreaks, we will carry out tests to determine whether epiphytic lineages have 'pathogenic potential' and study the mechanisms of host range expansion through a range of directed evolution experiments. Our analysis will include controlled assessment of gene exchange between bacteria through phage infection.

Finally, we will explore whether machine learning approaches can predict the host range of a Pseudomonas isolate with any degree of certainty from its genome sequence alone- a feat that is currently impossible with our current knowledge base, without direct pathogenicity testing upon a host. These predictions will be tested and validated using existing datasets but also on new datasets gathered as part of this work.