Review

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Tansley insight

Rapid evolution in plant–microbe interactions – an evolutionary genomics perspective

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Contents

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Summary

Access to greater genomic resolution through new sequencing technologies is transforming the field of plant pathology. As scientists embrace these new methods, some overarching patterns and observations come into focus. Evolutionary genomic studies are used to determine not only the origins of pathogen lineages and geographic patterns of genetic diversity, but also to discern how natural selection structures genetic variation across the genome. With greater and greater resolution, we can now pinpoint the targets of selection on a large scale. At multiple levels, crypsis and convergent evolution are evident. Host jumps and shifts may be more pervasive than once believed, and hybridization and horizontal gene transfer (HGT) likely play important roles in the emergence of genetic novelty.

I. Introduction

Studies of genetic variation in plant pathogens have revealed exceptional levels of diversity between related species and, for some, even among individuals of the same species (Möller & Stukenbrock, 2017; Wang *et al.*, 2017). Comparative genomic studies within and between species have provided multiple examples of highly dynamic genome architectures of fungal pathogens, many of which may be linked to host specialization or rapid adaptation (Rouxel *et al.*, 2011; van Dam *et al.*, 2016; Yan *et al.*, 2018). For example, genes encoding effectors (Box 1) are among the most rapidly evolving genes in plant pathogen genomes (Hall *et al.*, 2009; de Vries *et al.*, 2017). Many of these rapidly evolving genes have been shown to affect pathogen virulence.

Studies thus far indicate that some plant pathogens show high intraspecific diversity at the scale of populations (Möller & Stukenbrock, 2017). This applies to individual genes as well as larger genomic regions. Some rapidly evolving genomic regions are enriched for transposable elements (TEs). These may coincide with clusters of virulence determinants (Box 1), including, but not limited to, effectors (Raffaele *et al.*, 2010; Stewart *et al.*, 2018). Such TE-rich genomic compartments (Box 1) have been associated with the ability of filamentous pathogens to rapidly adapt to new host resistance and environmental change (Whisson *et al.*, 2012; Kasuga & Gijzen, 2013; Frantzeskakis *et al.*, 2019). While the origin and maintenance of such genomic compartments is still poorly understood, TEs are typically concentrated in regions of reduced recombination. In the case of many filamentous

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Box 1 Glossary

Effector: An effector is a protein that is secreted by a pathogen to interfere with the host's biology. Effectors can act in the apoplast or cytoplasm of host plants. They can increase the virulence of a pathogen and often show signatures of rapid evolution.
Virulence: A pathogen species is virulent on a given host if its colonization causes severe reduction in the fitness of its host. Virulence-associated genes/virulence determinants encoded by pathogens are genes that enable the pathogen to successfully colonize its host, exploit its host and/or evade the host's immune system.
Genomic compartment: In this context, genomic compartment means a region in the genome associated with a certain characteristic, for example a region with high gene density or elevated evolutionary rates.
Host jump: A host jump occurs when an individual of a pathogen species is able to successfully infect and complete its life cycle on a new plant species that is different from the ancestral host species. Over time, a population may become established on this new host and expand the host range and/or split from the ancestral pathogen population.
Reticulate evolution: Reticulate evolution describes the evolutionary histories of genes or species that cannot be explained by vertical inheritance; rather, they originate from hybridization, introgression or HGT events.
Latent pathogen population: A latent pathogen population is a population that has been present in a specific environment causing no symptoms or disease on its host plant, and that after some time or under certain environmental conditions can act as a pathogen on its host.

pathogens, we consistently observe distinct genomic compartments with different rates and modes of evolution (Raffaele *et al.*, 2010; Möller & Stukenbrock, 2017). In-depth characterization of the origin and maintenance of these distinct genomic compartments in pathogens relies upon resolution at the population genomic level, including the in-depth sampling of nucleotide diversity from rapidly evolving genomic compartments.

In addition to elucidating genomic architecture associated with the ability of filamentous pathogens to rapidly adapt to new host varieties, population genomic analyses can also be applied to infer the demography and population history of plant pathogens. Such data provide a cornerstone for the forecasting of disease presence and spread and serve as a window into the evolutionary potential of disease-causing pathogens. Detailed analyses of genetic variation among geographically separated populations of pathogens have illuminated migration routes within and across continents and permitted inferences about the evolutionary history of the species. Moreover, collections from isolates infecting different host species allow for the inference of gene flow between diverged pathogen populations on distinct host species (Gladieux *et al.*, 2018).

As is evident from the above examples, intraspecific and interspecific genomic analyses provide essential information about plant pathogen evolution. With these datasets becoming available for more and more pathogen species, new possibilities are opened for understanding both universal and pathogen-specific evolutionary patterns. In this Tansley insight article, we highlight some of the main discoveries from the field of evolutionary genomics of plant pathogens over the last few years.

II. Signatures of selection

Applying population genetic analysis in the framework of genomic surveys of plant pathogens is an efficient way to identify genetic targets of selection (Grünwald *et al.*, 2016; Badouin *et al.*, 2017; Hartmann *et al.*, 2018; Mohd-Assaad *et al.*, 2018; Thilliez *et al.*, 2019; Fig. 1). For example, the distribution and frequency of single nucleotide polymorphisms (SNPs) across the genome can be used to identify genomic regions that have been targets of recent directional selection (Stephan, 2016). In some cases, putative targets of selection have been associated with pathogen virulence and/or host specificity (e.g. Hall *et al.*, 2009; Poppe *et al.*, 2015). Some virulence-associated factors show signs of balancing or diversifying selection in which alternative alleles are maintained within populations (Fig. 1b) as, for example, effector-encoding genes: *AvrStb6* from *Zymoseptoria tritici* (Brunner & McDonald, 2018) and *Avrblb2* in *Phytophthora infestans* (Oliva *et al.*, 2015). In other cases, targets of natural selection are not directly related to host adaptation per se, but are related to other biotic or abiotic interactions of the pathogen, for example adaptation to fungicides applied to field crops (Mohd-Assaad *et al.*, 2018).

It has been proposed that TEs are instrumental for rapid adaptation in fungal and oomycete pathogens (Whisson *et al.*, 2012; Kasuga & Gijzen, 2013; Frantzeskakis *et al.*, 2019). TE expansion may facilitate gene gain or loss and thereby introduce genetic novelty (Yoshida *et al.*, 2016, Hartmann & Croll, 2017, Tsuhima *et al.* 2019). However, TE-derived mutations can negatively impact fitness as well (e.g. Kidwell & Lisch 1997; Tenaillon *et al.*, 2010). Grandaubert *et al.* (2019) recently used a population genomic dataset combined with a statistical genetics approach to quantify rates of adaptation across the genome of *Z. tritici*. While the authors confirmed that high rates of adaptive substitutions occurred in effector-encoding genes, these high rates of adaptive evolution did not correlate with the presence or density of TEs. Instead, sexual recombination enhances adaptive evolution, allowing beneficial mutations to be rapidly fixed in this pathogen.

III. Convergent evolution

Consistent with strong selection, convergent evolution is pervasive. Convergent evolution can be defined as the independent origin of the same phenotype or trait in two distinct populations or species. Convergent evolution can be through the independent loss-of-function mutations or deletions in the same genetic region as a means to overcome a novel resistance specificity or shift to a new host (Biju *et al.*, 2017; Hartmann *et al.*, 2017). Moreover, since different pathogens can be exposed to the same selective agent, for example commonly used fungicides, resistance to such fungicides may be conferred by independent mutations at the same molecular target present across these pathogens. Such an example has been described for the evolution of resistance to sterol demethylation inhibitor fungicides, in which distinct mutations occurred

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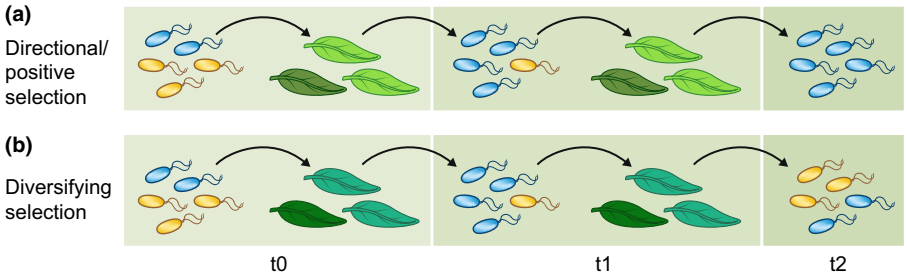

Fig. 1 Types of natural selection that operate on plant pathogens. Different forms of selection may operate on plant pathogens. These forms of natural selection leave distinct signatures in the genomes of pathogens. Virulence-associated genes are common targets of natural selection. (a) Directional/positive selection. Directional selection will lead to the fixation of a single allele through a selective sweep of the allele conferring higher fitness. Under this scenario, polymorphism is transient. Here the blue allele displaces the ancestral orange allele in the pathogen population. (b) Diversifying/balancing selection. Diversifying or balancing selection leads to the retention of sequence diversity within populations. In this example, two alleles of a virulence factor (orange and blue) are maintained in the pathogen population. The allele frequencies can vary over time depending upon the variation of resistance factors present in the host population, here visualized by two alleles (dark green and blue-green leaves).

independently in different pathogen lineages in the same pathogen target gene, *CYP51* (Wyand & Brown, 2005; Cools *et al.*, 2011).

Convergent evolution can also involve the independent origin of targeting of conserved host molecules by pathogens. Carella *et al.* (2018) describe examples in which distinct pathogen lineages convergently target the same signaling factors or hubs in host plants (Fig. 2a). Additionally, pathogens from different kingdoms have independently evolved similar mechanisms to protect their cell walls from lysis and detection by the host (Rovenich *et al.*, 2016).

IV. Cryptic genetic variation

Just as convergent evolution is a common theme among pathogen species, cryptic genetic variation is also a common theme within pathogen species. Pathogens that appear phenotypically identical may be genetically quite different. For example, a high degree of genetic differentiation was detected within the coffee leaf rust pathogen *Hemileia vastatrix* (Silva *et al.*, 2018). Population genetic analyses of this pathogen revealed that most of the genetic variation is explained by variation among individuals within sub-populations, combined with long-distance dispersal by the pathogen. Another form of crypsis encountered in host–parasite interactions is the emergence of a formerly undetected or latent pathogen population (Box 1), as was reported for the pathogen of oilseed rape, *Verticillium longisporum*, in the UK (Depotter *et al.*, 2017). Likewise, the sudden appearance of ash dieback in Europe was at first a mystery, because the pathogen is phenotypically indistinguishable from the harmless common saprotroph *Hymenoscyphus albidus* (Fig. 2b). However, population genetic analyses showed that this emergent pathogen was a distinct species, *H. fraxineus* (Queloz *et al.*, 2011; Bengtsson *et al.*, 2012; McKinney *et al.*, 2012), spawned by two haploid isolates from Japan (McMullan *et al.*, 2018).

V. Evidence of host jumps

Rapid evolution in pathogens is also visible at the phylogenetic level (Thines, 2019). Increasingly, studies challenge the paradigm of a strict pattern of co-speciation by pathogens on a group of related hosts, instead indicating that host jumps are common. This implies that pathogens are not gradually tracking the phylogenetic divergence of their hosts, but instead that host shifts and subsequent specialization may underlie the phylogenetic congruence between host and pathogen (Escudero, 2015). Within a phylogenetic context, these 'jumps' are not necessarily ancient, tracing back to deep phylogenetic lineages of the pathogen. Rather, some host jumps can be detected at the tips of the inferred pathogen phylogenetic tree (Choi & Thines, 2015).

Shifts to distantly related host species, as well as host range expansions (the ability to infect multiple hosts – as in the case of two divergent populations of the rust pathogen *Sporisorium reilianum* (Fig. 2d)), nicely illustrate the evolutionary potential of pathogen populations. Host jumps can be the result of a diversity of genetic events (for example discussed in Corredor-Moreno & Saunders, 2019). In some cases, host jumps may be enabled by cryptic intraspecific genetic variation at the level of gene expression (De Fine Licht, 2018). Minor changes in expression of few key transcription factors can result in large changes across multiple regulatory networks. If such minor expression differences prove to be beneficial on a new host, these novel expression profiles may ultimately be fixed by natural selection and enable a host shift or range expansion of this pathogen species.

In some cases, host range expansions are associated with greater longevity of pathogen lineages over evolutionary time (Navaud *et al.*, 2018). Whether a host shift or range expansion occurs, calibrated phylogenetic analyses allow us to gauge the speed at which these adaptive events take place and how they affect the diversification or extinction rate of pathogen lineages. Moreover, comparative genomics can be used to identify the results of host jumps. For example, in different *formae specialis* of *Blumeria graminis*, changes in the secretome content were associated with host shifts (Frantzeskakis *et al.*, 2018). Given the pervasiveness of convergent evolution and the action of strong selection in pathogen populations, recent host shifts or changes in host range are not unexpected.

VI. The role of reticulate evolution

New pathogens sometimes arise through hybridization of genetically distinct lineages (Fig. 2c). The degree to which hybridization between species or HGT among asexual lineages is elevated relative

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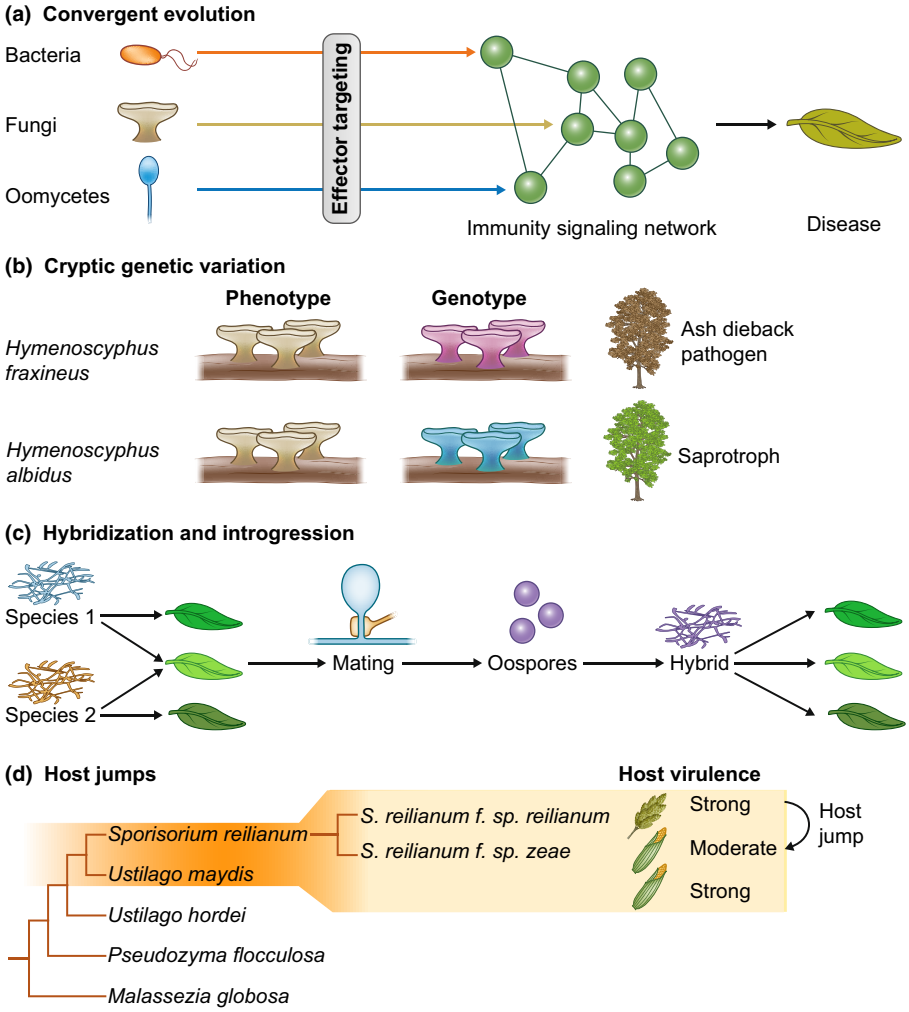

Fig. 2 Evolutionary mechanisms that occur in plant pathogens. Different mechanisms affect pathogens and impact their evolution. (a) Convergent evolution can be defined as the independent origin of the same phenotype or trait in distinct populations or species. In this hypothetical example, pathogens from different kingdoms have independently evolved proteins that converge on the same signaling network, which regulates plant immunity. Manipulation of this signaling network allows these pathogens to successfully colonize and cause disease on their host. (b) Cryptic genetic variation is widespread in pathogen species and may underlie some unexpected evolutionary transitions, such as the rapid emergence of virulence or origin of new pathogenic lineages. In this example, the invasive pathogen *Hymenoscyphus fraxineus,* which is morphologically indistinguishable from another European fungal species, *Hymenoscyphus albidus*, was recently discovered to be a distinct species. Both species produce ascocarps on ash rachises; however, *H. fraxineus* is pathogenic and causes ash dieback, while *H. albidus* is saprotrophic. (c) Hybridization and introgression can introduce novel genetic variation into a species. In this hypothetical example, two species of oomycetes are shown. Species 1 and Species 2 can colonize two host species each (indicated by different shades of green), and one host is shared between the two pathogens. Depending on the strength of sexual isolation of the two oomycete species, it may be possible for the two pathogens to mate on their shared host. If they mate and form viable offspring, these hybrid individuals may have a broader host spectrum than either of the parents. Therefore, through a rare hybridization event, a potentially new pathogen species with a broader host range may emerge. (d) Host jumps or shifts are common in pathogens. Here we depict the case of the smut fungus *Sporisorium reilianum*, which has evolved into two *forma specialis*. *S. reilianum* f. sp. *reilianum* is restricted to sorghum, which is believed to be the original host, while *S. reilianum* f. sp. *zeae* is restricted to maize, as is the closely related pathogen species *Ustilago maydis*. While both *U. maydis* and *S. reilianum* f. sp. *reilianum* are highly virulent on their respective hosts, *S. reilianum* f. sp. *zeae* causes only mild symptoms on maize.

to their non-pathogenic relatives is not known. Nevertheless, both hybridization and HGT seem to be important factors in the emergence of novel pathogen lineages (reviewed in Depotter *et al.*, 2016). Through the analysis of the distribution of effector genes across host specific lineages of *Albugo candida*, McMullan *et al.* (2015) found evidence of adaptive introgression. In this system, the frequent exchange of effector repertoires among different host-specific lineages may have facilitated host jumps. Moreover, comparative genomic analyses of three distantly related fungal pathogens of wheat, *Parastagonospora nodorum*, *Pyrenophora tritici-repentis*, and *Bipolaris sorkinia*, have revealed that these species share a toxin-encoding gene, *ToxA,* which has likely been exchanged between the species by HGT (Friesen *et al.*, 2006; McDonald *et al.*, 2017).

Population genomic analyses have demonstrated how pathogens with new host compatibilities can evolve from hybridization events, as demonstrated for the mildew pathogen *B. graminis* sp. *triticale* (Menardo *et al.*, 2016). The signature of hybridization may be a

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mosaic of genomic blocks that share sequence similarity with parental species (Stukenbrock *et al.*, 2012; Menardo *et al.*, 2016). Hybridization followed by extensive backcrossing to one of the parental species may leave shorter fragments of introgressed sequences, which exhibit a distinct phylogenetic signature (and possibly nucleotide composition) compared to the remaining genome (Feurtey *et al.*, 2019). Multiple examples from fungal pathogens show that hybridization has occurred by sexual recombination between distinct species. Other examples suggest that hybridization occurs by vegetative fusion resulting, for example, in the formation of an allopolyploid hybrid lineage (Depotter *et al.*, 2018; Fogelqvist *et al.*, 2018). Overall, detailed studies of multiple fungal pathogen genomes have identified a signature of past or recurrent hybridization, and these findings highlight the challenge of defining species in many groups of fungi.

Given the overwhelming evolutionary potential of pathogens, which can never be matched in crop species, it may seem remarkable that any pathogen threat can be controlled. Even in natural ecosystems, the pathogens typically have an evolutionary advantage, due to their much larger population sizes and shorter generation times. However, although the evolutionary potential of pathogens is expected to outstrip that of their hosts, the fact that these interactions stably persist indicates that mechanisms are operating to moderate the pace of adaptation between host and pathogen. These mechanisms that limit adaptation by pathogens can be revealed through detailed population sampling of host and pathogen. For example, limits to pathogen adaptation may be imposed by spatial structure and genetic diversity in the host populations, which may reduce dispersal or migration between susceptible hosts/patches (Thrall & Burdon, 2002; Laine *et al.*, 2014). Evolutionary potential is also tied to genetic variation. Many pathogen populations reproduce asexually (including some fungi and oomycetes) or are otherwise limited in their rate of outcrossing. For example, even in species which can sexually reproduce, some populations lack both mating types, and persist as asexual lineages (Martin *et al.*, 2019). Variation in ploidy within pathogen species also affects the outcrossing rate. Some widespread pathogen lineages are triploid, as has been observed for some isolates of the late blight pathogen of potato (Li *et al.*, 2017). Also, bottlenecks during a range expansion on a new host or geographic area can limit the genetic diversity and evolutionary potential of a pathogen, as appears to be the case for *H. fraxineus* in the UK (McMullan *et al.*, 2018) and *Z. tritici* in Australia (Hartmann *et al.*, 2017). Clearly, a wide array of complementary forces serves to throttle pathogen evolution in both agronomic and natural ecosystems. Were these not in place, the distribution of certain plant species and our ability to cultivate them would be severely affected.

VII. Concluding comments

Detailed analyses of genetic variation within plant pathogen genomes can inform us about the population biology and the demographic history of species. We argue here that comparative evolutionary genomic analyses at the population level of crop pathogens are crucial to understand patterns of dispersal and reproduction, as well as rates of evolution and population history. This knowledge of the evolutionary history of plant pathogens is essential when, for example, designing sustainable crop protection strategies in future agro-ecosystems. Along these lines, a central question is how modern agricultural systems based on monoculture crop production impacts plant pathogen evolution. Plant breeding and disease control strategies, including fungicide application, exert strong directional selection pressure on pathogen populations. Yet, evidence from population genomic studies suggests that plant pathogen populations can maintain high levels of genetic variation in the field. Hence, population genomic analyses that compare the population structure of the pathogen species or closely related species on wild and cultivated plants are needed to understand the impact of agriculture on the evolution of pathogens, foremost the effective population size. To date, unfortunately just a small number of wild plant pathogens are being studied, most of which are pathogens of Arabidopsis (Herlihy *et al.*, 2019). Thus, more genomic data from wild pathogen populations on non-model species are needed in the future. In addition, many population genetic and genomic analyses rely on underlying evolutionary models. Often these models are developed with model organisms such as *Drosophila melanogaster* in mind (Stephan, 2016). Unfortunately, most plant pathogens do not meet the underlying assumptions of such models: for example, many pathogens engage in both sexual and asexual reproduction. Hence, the accuracy of these analyses may be compromised, and new theoretical approaches, taking into account, for example, mixed mating systems, highly heterogeneous rates of mutation and recombination across genomes and recurrent introgression, are needed to improve our inference of demographic histories of plant pathogen populations. In summary, while the accumulation of more and more population genomic data is crucial to answer questions on pathogen evolution, new mathematical models and a shift to non-model organisms capable of infecting wild and cultivated crops is required to move the field forward.

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