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Evolutionary dynamics of pathogen population genetics

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Introduction

Plant diseases pose a longstanding challenge to global food security and societal stability. In contemporary agriculture, the impact of these diseases is mitigated primarily through the incorporation of resistance genes into cultivars featuring superior agronomic traits and the application of agrochemicals. Agricultural ecosystems experience a swift evolution of plant pathogens compared to natural ecosystems. This accelerated evolution is attributed to factors such as the cultivation of high-density monocultures, characterized by genetically uniform crops across extensive areas; the widespread use of agrochemicals on a large scale; and the international trade of agricultural products (Zhan *et al*., 2014, 2015). Limited genetic diversity in major crops makes them susceptible to plant pathogens, resulting in the rapid spread of virulent pathotypes. Despite the introduction of new resistant cultivars, local pathogen populations often evolve to overcome resistance, rendering both resistant varieties and commercial agrochemicals ineffective within a few years. This breakdown is characterized by the widespread distribution of a single resistance gene (the "boom"), followed by the adaptation of the pathogen population to overcome the resistance (the "bust"). Boom-and-bust cycles are well-documented, especially in cereal rusts (Kolmer, 1996; McIntosh and Brown, 1997) and powdery mildews of cereals (Wolfe and McDermott, 1994), characterized by a gene-for-gene interaction with their hosts (Flor, 1956).

Understanding the population genetics of plant pathogens is crucial for comprehending disease epidemiology, ecology, and evolutionary trends. This knowledge is instrumental in the effective utilization of resistant cultivars and agrochemicals, leading to substantial control of plant diseases (Palumbi *et al*., 2001). Genetic variation within pathogen populations plays a key role in their ability to adapt to dynamic environments, with highly variable pathogens having an advantage in swiftly adjusting to changes like global warming, the adoption of new resistance genes, and the introduction of new agrochemicals. Furthermore, studying the population genetics of plant pathogens contributes to unraveling evolutionary mysteries in nature, such as the emergence of virulence and the origin and maintenance of sexual reproduction.

In the gene-for-gene interaction, pathogens release elicitors recognized by plant receptors. This recognition triggers a defense response, often leading to the death of the infected plant cell and inhibiting the pathogen. Pathogen mutations from avirulence to virulence alter or eliminate the elicitor, preventing recognition by the host receptor. Resistance breaks down as the frequency of pathogen strains with these

mutations increases. Host defenses failing to activate early allow virulent mutants to rise in frequency. As the virulence mutation becomes prevalent in the pathogen population, the resistance gene loses effectiveness, leading to resistance breakdown. Gene-for-gene resistance, or major-gene resistance, is potent but targets only the portion of the pathogen population producing the specific elicitor. Plant resistance can result from various genetic factors, such as phytoalexins, barriers, PR proteins, and enzymes. Unlike gene-for-gene interactions, these contribute to quantitative resistance with small, additive effects. This form, known as minor-gene or partial resistance, doesn't follow the typical boom-and-bust cycle.

Population Genetic Structure of Plant Pathogens

Genetic variation

Plant pathogen genetic diversity arises from mutation, gene flow, recombination, random genetic drift, and natural selection (Zhan and McDonald, 2004). Influenced by host-pathogen characteristics, environments, agricultural practices, and human activities, mutation, recombination, and gene flow increase genetic variation, while genetic drift decreases it. Selection can either boost or reduce genetic diversity, with directional selection reducing it rapidly, and balancing or frequency-dependent selection increasing it through rare allele preference or overdominance.

Genetic variation significantly influences the evolutionary potential of pathogen populations. Reduced genetic diversity lowers mean fitness, resilience, and long-term adaptability. According to Fisher's fundamental theorem of natural selection (Zhan and McDonald, 2005), a population's adaptability depends on its additive genetic variance in fitness-relevant traits. Pathogen populations with greater genetic variation evolve faster, making them more challenging to control using plant disease management methods like agrochemicals, resistant varieties, and biocontrol agents. In pathogen population studies, both gene and genotype variations are crucial. Gene-level variation can be assessed using measures like allelic richness and gene diversity, with allelic richness being particularly sensitive to sample size and indicative of evolutionary potential (Zhan and McDonald, 2005). Genotypic variation depends on factors like recombination rate and the pathogen's mating system. Populations with limited recombination or mostly asexual reproduction show low genotypic variation, characterized by a few clonal lineages, while sexual populations tend to have higher genotypic diversity.

Population differentiation

Population differentiation, driven by allele frequency variations, results from random genetic drift and selection. Limited gene flow can cause random fixation of neutral molecular variations, leading to nonadaptive genetic differentiation. Selection for ecological traits among isolated pathogen populations induces adaptive genetic divergence. Random genetic drift affects all loci uniformly, while selection-induced differentiation is locus-specific. Molecular markers like RFLP, RAPD, AFLP, or microsatellites often reflect population differentiation due to random genetic drift.

Population differentiation is assessed using contingency $\chi 2$ tables, genetic distances, and F-statistics (Fisher, 1930). Contingency tables test gene frequency homogeneity, with combined less frequent alleles for robust inference. Genetic distance measures nucleotide substitutions linearly related to divergence time.

F-statistics like FST, GST, θST, ϕST, and RST are widely used, with FST indicating heterozygosity deficiency due to nonrandom mating (for diploid pathogens), GST averaging FST over alleles (for any species with estimable allele frequencies), and θST emphasizing sample sizes and subpopulation numbers. Analysis of molecular variance (AMOVA) estimates evolutionary divergence based on mutational steps between haplotypes, yielding ϕST. These statistics are applicable to molecular data from nonrecombining regions. The use of FST and its analogues assumes a K-allele or infinite allele model, which may not be valid for many microsatellite markers. In such cases, RST, developed for markers with a stepwise mutation model, should be employed (Slatkin, 1995).

Evolutionary forces in Fungal plant pathogen variation

To survive, diverse infections employ various tactics, adapting when faced with new cultivars. Plant-pathogenic fungi, crucial for the food and agricultural industries, cause significant losses through different interactions. Gene flow is a vital source of genetic variation in pathogen populations, illustrated by the introduction of species like *Phytophthora infestans* to Europe and *Cryphonectria parasitica* to North America.

Recombination

Plant pathogens employ recombination for sexual reproduction, and it can also occur through cytoplasmic and nuclear material exchange in somatic hybridization. This process, known as nuclear fusion and recombination, enhances genotypic diversity in pathogen populations post the parasexual cycle. Sexual reproduction involves a change in ploidy, forming a diploid zygote through gamete fusion, driven by the meiotic process and genetic recombination (Schoustra et al., 2007). Goddard (1976) studied the impact of somatic hybridization on *Puccinia striiformis*, identifying 30 recombinant single-spore isolates with unique virulence responses compared to parental races.

Mutation

Mutation is the key source of genetic variation, causing changes in DNA sequences and generating new alleles. It plays a crucial role in creating virulent strains in plant pathogens that can break major gene resistance and enhance pathogenicity. In the gene-for-gene interaction, a mutation in the avirulence allele is necessary for the development of a virulent pathogen strain by altering the gene encoding the elicitor recognized by a resistance gene. Mutations from avirulence to virulence are uncommon and typically insufficient to break down resistance. However, when coupled with directional selection, such as deploying a resistance gene, virulent mutants can rapidly increase in frequency, rendering the resistance gene ineffective. While mutation rates are generally low, they can vary among loci and pathogens (Flor, 1958).

The concept of mutation as a mechanism for the emergence of new races in *P. striiformis* was initially proposed by Gassner and Straib, 1993. Mutation involves the alteration of an organism's genetic makeup, occurring either naturally or due to external stimuli. This modified genetic makeup is hereditarily passed down to descendants. Mutations manifest as changes in the DNA's base sequence, including additions, deletions, or substitutions of base pairs.

Heterokaryosis

Heterokaryosis occurs when fungal hyphal cells contain multiple genetically distinct nuclei. In

Basidiomycetes, the dikaryotic stage is distinct from the haploid mycelium. For example, *P. graminis* tritici's dikaryotic mycelium can grow on both wheat and barberry, unlike its haploid mycelium. Various spore types exhibit different infectivity patterns on wheat and barberry. The heterokaryotic state in heterothallic fungi arises through meiosis, mutation, anastomosis, and the inclusion of different nuclei in spores. Effective anastomosis enables genetic compatibility/incompatibility tests, revealing genetic exchange between diverse genotypes (Croll *et al.*, 2009). During a full parasexual cycle, mycelium growth occurs in heterokaryotes, involving fusion, equivalent nuclear fusion, and nuclei convergence. Mitotic crossing over may cut diploid nuclei in half. Heterokaryotic mycelium formation can happen through anastomosis, allowing foreign nuclei to spread. Ascomycetes demonstrate homokaryotic to heterokaryotic conversion by growing in one or more nuclei. Nuclear fusion during the heterokaryotic stage results in a mix of haploid and diploid nuclei. The mycelium may contain at least five types of nuclei. Mitotic crossing over, observed in organisms like *Penicillium chrysogenum* and *Aspergillus niger*, leads to novel pairings and connections. Diploid strains are divided to create uninucleate conidia, contributing to diploid mycelia. Certain fungi exist as isolated diploid strains with defective reproduction.

Haploidization

Diploid colonies typically manifest distinct sectors, generating haploid conidia that give rise to haploid colonies. The selection of specific diploid nuclei results in mycelium haploidization. Mitotic recombinations create novel linkage groups, producing genotypically distinct haploid strains. Genetic variance in the wheat karnal bunt pathogen arises from the recombination of genetic material through sexual and parasexual mechanisms (Gupta *et al.*, 2015). Chromosomal rearrangements contribute to morphological variability in *Tilletia indica* strains. The fungus *A. nidulans* exhibits a high mitotic recombination rate for rapid environmental adaptability during somatic growth. Nuclei with a different recombinant genotype in somatic tissue can produce asexual spores, suggesting the relevance of "parasexual recombination" or "somatic recombination" to evolution (Schoustra *et al.*, 2007).

Conclusion

In conclusion, understanding the disease races in a crop area is crucial for developing resistant crop varieties, especially when employing selective breeding for elite genotypes with stacked resistance genes. Pathogens rely on variation for survival, driven by selection pressure. Detecting and identifying variability promptly is essential for effective management. Molecular methods, being more efficient and precise than conventional ones, offer a preferable approach for pathogen characterization. Simple PCR-based detection methods can be developed for farming populations. Plant-pathogen identification, based on the complementarity between a plant's resistance gene (R) and the pathogen's avirulence gene (avr), facilitates the selection of breeding lines with specialized resistance. This knowledge allows the creation of cultivars with higher disease resistance levels (QTL). Additionally, understanding pathogen variability dynamics aids in developing strategies for stacking or deploying resistance genes, preventing the emergence of new virulence against existing resistance genes.

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