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Evolutionary dynamics of pathogen population genetics - II

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Introduction

Plant diseases challenge global food security and societal stability. In modern agriculture, these diseases are managed through resistance genes in high-performing cultivars and agrochemicals. Agricultural ecosystems see faster plant pathogen evolution than natural ones due to high-density monocultures of genetically uniform crops, extensive agrochemical use, and international trade (Zhan *et al*., 2014, 2015). Limited genetic diversity in major crops makes them vulnerable to pathogens, leading to rapid spread of virulent strains. Even with new resistant cultivars, local pathogens evolve to overcome resistance, making resistant varieties and agrochemicals ineffective within a few years. This results in "boom-and-bust" cycles where a resistance gene becomes widespread ("boom"), followed by pathogen adaptation ("bust"), as seen in cereal rusts (Kolmer, 1996; McIntosh and Brown, 1997) and cereal powdery mildews (Wolfe and McDermott, 1994), involving gene-for-gene interactions (Flor, 1956).

Understanding plant pathogen population genetics is essential for grasping disease epidemiology, ecology, and evolution, aiding the effective use of resistant cultivars and agrochemicals for disease control (Palumbi et al., 2001). Genetic variation within pathogens allows them to adapt to changes, such as global warming, new resistance genes, and new agrochemicals. Studying pathogen population genetics also sheds light on natural evolutionary phenomena like virulence emergence and sexual reproduction.

In gene-for-gene interactions, pathogens release elicitors recognized by plant receptors, triggering defense responses that often kill infected cells and inhibit the pathogen. Mutations from avirulence to virulence alter or eliminate the elicitor, preventing recognition by the host receptor,

and as these mutations spread, resistance fails. The resistance gene loses effectiveness as virulent strains become prevalent. Major-gene resistance targets only pathogen strains producing specific elicitors and is prone to boom-and-bust cycles. Plant resistance can also involve phytoalexins, barriers, PR proteins, and enzymes, contributing to quantitative resistance with small, additive effects, known as minor-gene or partial resistance, which does not follow the boom-and-bust pattern.

Evolutionary forces in bacteria

Bacteria can undergo changes through bacterial symbiosis, a process facilitated by bacterial conjugation. Bacterial conjugation involves the exchange of genetic material, either directly between cells or indirectly through a bridge-like connection. Despite sharing some features with genetic exchange, bacterial conjugation is distinct from sexual reproduction or mating. For conjugation to occur, a host, typically a conjugative or mobilizable plasmid or transposon, must be present in one of the bacteria—the donor (Ryan and Ray, 2004). Conjugative plasmids often incorporate precautions to prevent the recipient cell from having a similar component already. Two types of transferable conjugative plasmids exist: self-transmissible, promoting cell-to-cell contact and DNA transfer, and mobilizable, transferring successfully when coexisting with self-transmissible plasmids. Large self-transmitting plasmids carry genetic instructions for bacterial mating and DNA transfer. Receiving cells gain benefits like xenobiotic tolerance, metabolite utilization, and antibiotic resistance. These plasmids are akin to endosymbionts. Conjugative elements act as genetic parasites, evolving through conjugation to spread to new hosts.

Transformation

Bacteria can take up naked DNA molecules and maintain them through a process called "transformation," a phenomenon discovered by Griffith in 1928. Bacteria employ intricate methods to attach and transfer DNA pieces into cells. Competence, the ability to absorb foreign DNA from the environment, exists in both natural and synthetic forms. About 1% of bacteria naturally possess the ability to absorb DNA in a lab setting, driven by gene sets describing DNA movement through cell membranes. Artificial competence is generated in a lab under unusual conditions, rendering cells passively DNA-permeable (Kunik *et al*., 2001).

Transduction

Transduction is the process through which bacteriophages transfer genes from one bacterial cell to another. This bacteriophage-mediated gene transfer occurs in two forms: generalized transduction and specialized transduction.

The lytic cycle results in generalized transduction, where certain bacteriophages randomly package bacterial chromosomal regions along with their DNA. This leads to two types of particles in the lysate, distinguished by the DNA they carry. While most particles contain viral DNA, a small percentage (possibly up to 1%) carries portions of the bacterial chromosome. When introduced into the cell, these particles, despite containing only bacterial genes, do not cause negative effects and are open to recombination with the chromosome. Small numbers of transducing bacteriophages can insert 100–200 kilobases of DNA, allowing almost every bacterial gene to be transduced due to the essentially random packaging (hence, "generalized" transduction). Phages can also transduce entire plasmids, especially those encoding antibiotic resistance in staphylococci, indicating their role in the spread of antibiotic resistance.

Specialized transduction relies on a temperate bacteriophage. In this process, a bacterial gene integrates into the bacteriophage genome through recombination. When a lysogenic temperate bacteriophage infects a new bacterial host, the associated bacterial gene is transmitted. Since it is a bacterial gene, the bacteriophage repressor that inhibits lytic activity has no effect on it. Specialized transduction can lead to three outcomes: the DNA can splice two copies of bacterial genes into the recipient cell's genome while still behaving as a virus, exchange information with homologous DNA of the recipient cell, or be taken in and reused as spare parts.

Evolutionary forces in viruses

The emergence of a plant virus can be affected by various genetic processes, including recombination, reassortment, migration to new agroecosystems, changes in vector population dynamics, and acquisition of novel virus-like entities. Often, a combination of these pathways contributes to the spread of a plant virus.

Mutation

The understanding of the genetic composition of populations over time is greatly shaped by the occurrence of

spontaneous mutations. Genetic diversity, driven by mutations, is crucial for the mechanisms of natural selection and genetic drift. RNA viruses exhibit mutation rates ranging from 0.03 to 2 per genome per replication round, which are orders of magnitude higher than those observed in their DNA-based hosts (Chao *et al*., 2002).

Reassortment and Recombination

Recombination, in addition to expanding the viral host range, increasing pathogenicity, and aiding host immune evasion, has been associated with the emergence of antiviral resistance. When a virus is co-infected by two distinct strains, one or more new viral strains may emerge, exhibiting differences in characteristics such as virulence and symptomatology compared to the original strains initially introduced into the host.

Sequence analyses of global tomato yellow leaf curl virus (TYLCV) isolates have indicated widespread recombination (Fauquet and Stanley, 2003). The highly lethal EACMV-UG2 strain, responsible for these disease outbreaks, resulted from the recombination of East and Africa cassava mosaic viruses (EACMV and ACMV). This recombination involved the flipping of homologous capsid protein (CP) gene sequences from ACMV and EACMV. Furthermore, the emergence of other highly virulent strains in various southeast African regions is attributed to reassortment between distinct recombinant EACMV components (Pita *et al*., 2001). These new viruses pose a severe threat to Africa's cassava production, exacerbated by an increase in the population of whiteflies that feed on cassava.

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