



Healthy Plants • Healthy World

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To Whom It May Concern:

The American Phytopathological Society (APS), founded in 1909, is the premiere educational, professional and scientific society dedicated to the promotion of plant health and plant disease management for the common good. The Society, representing the interests of 5000 scientists whose pivotal research advances in the understanding of the science of plant pathology and its application to plant health, is pleased to have the opportunity to provide comment on the nature and descriptions of the current NIH Study Sections as they relate to microbial pathogenesis and host disease responses.

Considerable interest has emerged within the past decade on the remarkable similarity among virulence-related genes and host defense related genes in animal and plant disease systems. Several pathogens (for example, the bacteria *Pseudomonas aeruginosa*, *Burkholderia cepacia* and *Serratia marcescens*) bridge the kingdom gap, causing disease in both animals and plants. *B. cepacia* and *S. marcescens* have been isolated from clinical samples much more frequently in recent years, and are resistant to several common antibiotics. In some cases the same species occupy endophytic (but beneficial) or saprophytic environmental (soil, water supplies, etc) niches. Gene homologies among these pathogens, and even among pathogens whose host ranges are less overlapping, have shown that certain strategies used by microbes to recognize host cells and colonize their tissues, to maximize their stealth in host invasion, and to optimize the delivery of virulence-related or host defense-suppressive response products, are conserved among pathogens once believed to inhabit specific and limited ecological niches. Relevant genes are turning out to be those encoding environmental signal sensing molecules, gene regulators and delivery mechanisms. What accounts for these similarities? Did these pathogens evolve from common ancestors that possessed these or precursor genes? Did the mechanisms (and their genes) evolve more than once in diverse microbes faced with similar adaptation challenges? Or, have pathogenicity-related genes been much more freely exchanged among natural microbial populations than was once assumed?

For example, one of the genetic mechanisms underlying *B. cepacia*'s remarkable adaptability is insertional activation of gene expression by transposable elements, a strategy particularly effective with genes carried on conjugative plasmids. This regulatory process enables the bacterium to profit from foreign genetic material that otherwise would be expressed poorly or not at all, thereby contributing to the microbe's nutritional versatility and presumably also to its ability to colonize hosts as widely divergent as humans and onions. In addition, *B. cepacia* grows at temperatures up to 42 C, exceptionally warm for growth of a phytopathogen, but colonization of both humans and plants would require a broad range of growth-permissive temperatures.

The bacterium *S. marcescens* is important in many agricultural and human endeavors, in roles that, from the human perspective, are both positive and negative. This remarkable range of ecological roles for a single bacterial species, some of which are contradictory in their impact, prompt us to ask what genetic and molecular mechanisms account for them. What determines that these microbes will be plant pathogens in one instance and seemingly harmless endophytes in another, or whether bacteria in either of these ecological niches could be human pathogens? Can a single *Sm* strain assume any or all of these different roles, depending on circumstances, or are the bacteria in the different roles already so far apart evolutionarily that they are no longer able to function in similar roles?

Similarly, several fungi also appear to cross kingdom lines in infectivity. Species of *Aspergillus*, *Fusarium*, and others may also parasitize humans, though generally only those humans whose immune systems are compromised by another condition. Fungal mechanisms such as control of dimorphism and virulence are common in both plant (*Ustilago maydis*) and human (*Candida albicans*) pathogens. Infection mechanisms and gene silencing among human, animal and plant viruses share similarities as well.

Research to provide the answers to questions like those posed above will accelerate our understanding of both pathogenesis and host defense responses. It will help to clarify what, in the end, defines a pathogen. Model systems from the animal kingdom may be used to investigate pathogenicity of plant pathogens, and vice versa. Genome sequences of pathogens from different hosts can be mined to gain molecular tools for use with conserved genetic systems. Regulatory mechanisms common to widely divergent pathogen systems will prove important in both basic understanding and in the development of molecular tools for research, for rapid and sensitive diagnostics, and for the design of new tools for disease management.

The NIH has an opportunity to create a niche for a valuable new type of research partnership, which itself bridges kingdoms of study. Much can be learned by cooperative projects in which investigators from the plant and animal sciences form partnerships and work collaboratively to explore the dimensions of these common issues of co-evolution and adaptability. Developing a new research funding program, or expanding an existing one by the addition of specific language, will provide the financial backing to encourage and support such interactive activities. I urge you to consider this program strongly, and to provide this mechanism to encourage new ventures in research that will ultimately provide new understanding, new products for disease control, and new opportunities for synergism among the scientific community.

Sincerely yours,



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