Letter to the Editor

Some Concepts of Physiological Plant Pathology

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The following statements are offered as concepts of physiological plant pathology by the above members of a class studying this area. Their formulation is based on the definition (3) that a concept is a general notion or idea. These concepts are based on information and ideas gleaned from different sources with heavy reliance on the text by Heitefuss and Williams (1) and Volume III of "Plant Disease—An Advanced Treatise," which was edited by Horsfall and Cowling (2).

We recognize that many areas in the field of physiology of plant disease are in a state of flux (eg, the role of phytoalexins, common antigens, and nonhost-specific toxins) and that for these more information must be gained before concepts can be formulated.

The authors feel that many of these concepts would be accepted as principles based on current information. Others are based on minimal information and must await the test of time and more information. We hope by presenting the statements in this manner to promote discussion and enough response from students and research workers to allow the synthesis of a set of principles applicable to physiological plant pathology.

1. Disease in plants is an adverse alteration in energy utilization.
2. Pathogenicity, susceptibility, resistance, and pathogenesis are processes or attributes governed by the interaction of the genetic characteristics of two or more organisms and the physical environment in which they operate.
3. Resistance is the inherent ability of an organism to prevent or restrict the establishment and/or subsequent activities of a potential pathogen.
4. Pathogenicity is the relative ability of the pathogen to cause disease in a susceptible host.
5. Passage of a fungal penetration peg through the epidermal wall is accomplished by mechanical and/or localized chemical alteration of the cuticle and the wall.
6. Hyphae from germinating spores of rust fungi are induced to form infection structures by the physical and chemical stimuli provided by the host surfaces.
7. Root exudates differentially affect the composition of the rhizosphere microflora which may affect the development of the disease.
8. The initiation of plant infection by viruses involves removing the protein coat of the viral genome.
9. Invading pathogenic bacteria must occupy a specific site in the suscept to initiate the expected disease syndrome.
10. The haustorium of an obligate parasitic fungus does not penetrate the plasmalemma.
11. The stability of noninjured and injured plant cell walls depends on proteins and phenolic compounds.
12. Degenerative effects of infection on host cytology are commonly found in both obligate and nonobligate fungal parasitism, but occur at much later stages of infection in the former.
13. Pectic enzymes involved in cell wall breakdown during disease development may be either of host or pathogen origin or both depending on the particular interaction.
14. Membrane damage to plant cells, hence cell death, in tissues macerated with pectic enzymes is a result of enzymatic degradation of the cell walls.
15. The toxic compounds produced by host-specific toxigenic pathogens account for all or some of the host selectivity.
16. Host-specific toxins affect the plasmalemma or other cellular membranes of susceptible plants.
17. Interactions between the pathogen and the carbohydrates of the host affect the production of the host cell wall-degrading enzymes.
18. Alteration of host cell functions by plasmid DNA from the pathogen causes normal plant cells to become tumor cells.
19. Infection is accompanied by a release of preformed enzyme from lysosomes as well as de novo enzyme synthesis in the invaded cell.
20. The transpiration rates of plants with vascular disease, before symptoms are visible, are equal to or only slightly less than those of healthy plants but these decrease markedly as vascular dysfunction develops.
21. Loss of cell osmotic function is associated with plant tissue necrosis.
22. Increased respiration rates in diseased plant tissue is not caused by an uncoupling of the rate limitation of ATP formation and oxygen uptake, except in the case of the separate action of *H. maydis* toxin in maize.
23. Host-mediated ethylene production in diseased plants affects disease development and resistance.
24. Hypoplasia and hyperplasia in diseased plants result from an imbalance of growth inhibitors and promoters.
25. Changes in plant metabolic activity during normal senescence parallel those associated with the later stages of diseases caused by obligate parasites.
26. The qualitative and quantitative changes in oxidative enzyme activity in infected plants are not solely the consequence of mechanical damage.
27. Parasitic specificity depends upon interactions of complementary host and pathogen molecules that facilitate multiplication of the potential pathogen.
28. Extracellular polysaccharides produced by virulent pseudomonads cover the binding sites on the host wall lectin which allows bacterial multiplication in the intercellular spaces.
29. Resistance to the pathogen is conditioned by one or several dominant or recessive genes or extrachromosomal factors. Likewise, avirulence is a product of a functional dominant or recessive gene or genes or extrachromosomal factors.

LITERATURE CITED

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