

Letter to the Editor

Some Principles of Plant Pathology. II.

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A major essential of scientific writing is to put as much information into as few words, or as little space, as possible. To accomplish this, there are the perpetual problems of data vs. conclusions, details vs. principles, information vs. interpretation, etc. Although it is true that facts (data) remain constant while interpretations change, it is also true that many facts can be fitted into fewer interpretations. Some have argued that data should be deposited in a few available centers while only abstracts are published for wide dissemination (2); others have suggested that the data are not worth owning (3). This conflict cannot be readily resolved in plant pathology or any field of knowledge. But while there is an abundance of data (facts?) about plant pathology, there is a shortage of principles. The most recent compilation is by Kenaga (1). This is my second effort to condense much of the information of plant pathology into a few principles (positive statements of wide application). In the first attempt (4), 45 principles were itemized. The expansion to 93 here represents largely a recognition of new generalizations.

A. Etiology

1. Disease results from the continued irritation of plant by a living or nonliving pathogen.
 2. Most plant diseases are caused by microorganisms.
 3. Microorganisms associated with higher plants are usually noninjurious, commonly injurious, and rarely beneficial, but these effects are modified by environment.
 4. Fungi are the major microorganisms responsible for disease.
 5. The diagnosis of disease is based primarily on signs and symptoms.
 6. Koch's rules of proof are basic for diagnosis, but limited in application.
 7. Most diseases of economic plants have already been correctly diagnosed.
 8. Genetic constitution may cause disease.
 9. Unfavorable environments may cause disease.
 10. Pathogens may have a wide or narrow host range.
 11. Species of pathogens commonly exist in many physiologic forms.
 12. Pathogens adapt themselves physiologically and genetically to resistant varieties and unfavorable environments.
 13. The closer the genetic relation between hosts, the closer the genetic relation of their pathogens.
 14. Viruses can be regarded as microorganisms, as genes or as chemicals.
 15. Pathogens may lose virulence by growth on artificial substrata.
 16. Pathogens may change their virulence as a result of host passage.
- #### B. Importance
17. Several pathogens have the potential to destroy any crop but rarely do so.

18. Diseases reduce yield and quality of crop.
 19. Diseases increase the variability of crop production.
 20. Diseases increase the cost of crop production.
 21. Knowledge of disease has contributed to other basic knowledge.
 22. For the same average loss, epidemic diseases are more important than chronic diseases.
- #### C. Symptomatology
23. Symptoms of disease are correlated with the taxonomy of the causal agent.
 24. Symptoms of disease are usually a response to metabolites of the pathogen.
 25. Some infections cause no recognized symptoms.
 26. Parasites may increase the local or general growth of their hosts.
 27. Infections and injuries increase host respiration.
 28. Infections and injuries increase the permeability of their hosts.
 29. Fungi, bacteria, and nematodes usually cause localized infections.
 30. Viruses usually cause systemic infections.
 31. Symptoms may be systemic, even if the pathogen is localized.
- #### D. Life History
32. Most parasitic fungi have two or more morphologic stages which normally appear in a cyclic sequence.
 33. Most pathogens can be cultured on nonliving substrata.
 34. The asexual stage of fungi is most likely to be parasitic.
 35. The dormant stage of pathogens is more resistant to environment than the active stage.
 36. Most fungus and nematode pathogens enter their hosts actively, whereas bacteria and viruses enter passively.
 37. Some pathogens have alternate hosts, and these may be obligate or facultative.
 38. Pathogens and disease increase at a logarithmic rate.
 39. Pathogens are inactivated at a logarithmic rate.
 40. Dispersal of pathogens is usually passive.
 41. Pathogens are more likely to be transmitted by vegetative plant parts than by seed.
 42. Disseminated pathogens decrease as the logarithm of the distance from the source.
 43. Systemic infections increase at a slower rate than local infections.
 44. The principal vectors of pathogens are wind, water, arthropods, and man.
 45. Specific pathogens enter through specific sites and colonize specific tissues.
 46. Pathogens will actively enter hosts in which they cannot multiply.
 47. The greater rate of increase of pathogens than

their hosts is necessarily associated with the greater rate of reproduction of small than large organisms.

48. Soil and plant debris are major reservoirs of plant pathogens.

E. Predisposition

49. Resistance to a specific pathogen is more common than susceptibility.

50. Plants may increase or decrease in susceptibility with age.

51. Conditions which favor plant growth commonly favor disease.

52. Tillage is a major predisposing agent for plant disease.

53. The predisposing effect of tillage is greater with wild than with cultivated plants.

54. Short periods of high temperature may predispose plants to pathogenic and nonpathogenic organisms.

55. High nitrogen fertilization commonly favors disease.

56. High potassium fertilization commonly reduces disease.

57. Several minor elements may be decisive for specific disease situations.

58. Diseases are more abundant in tropical than in temperate climates, even on the same crop.

F. Epidemiology

59. Pathogens increase more rapidly in monoculture than in mixed stands of plant species, or with rotation of crops.

60. Damage due to disease is usually less in cross-fertilized than in self-fertilized crops.

61. The optimum temperature for most diseases is about 25 C.

62. Most fungal and bacterial pathogens require free moisture for the initiation of infection.

63. The major sources of free water are rain, dew, guttation water, and irrigation water.

64. Alternating light and darkness is usually necessary for normal disease development.

65. The diurnal periodicities of pathogens are adapted to the diurnal periodicities of the environment and of their hosts.

66. The effect of environment is most critical during penetration of the host.

67. Host exudates commonly favor pathogens.

68. Disease increases as the logarithm of the inoculum.

69. Metabolites of microorganisms in normal soil prevent the invasion of that soil by plant pathogens, unless the pathogen is aided by added nutrients.

70. An endemic pathogen is less virulent on native than on introduced host species.

71. Pathogens are typically more tolerant of extremes of heat and cold than are their hosts.

72. Soil-borne diseases show less seasonal variability than airborne diseases.

73. There is little relation between the biotic potential of the pathogen and the severity of contrasting types of diseases.

74. Disease epidemics can be forecast from the incidence of the pathogen and the weather.

75. The number of diseases of a crop increases with increased production of that crop.

G. Control

76. All knowledge of factors affecting disease has potential value in control.

77. There is always a differential response of host and pathogen to environment.

78. Control of disease is usually directed at inhibition of the pathogen.

79. Control practices may be classified under exclusion, eradication, protection, immunization, and therapy.

80. Genetic resistance is usually due to toxic chemicals formed after entry of the pathogen.

81. Genetic resistance is inherited by Mendelian laws.

82. For each gene for resistance in the host there is a corresponding gene for virulence in the pathogen.

83. Wound healing is a major mechanism of disease resistance.

84. Parasitic fungi usually have a lower temperature of inactivation than nonparasitic fungi.

85. Chemical control depends on a differential effect of a fungicide on host and pathogen.

86. Treatments which are therapeutic to one pathogen may predispose to another.

87. Closely related pathogens may protect against each other.

88. Resistance may be constitutional or acquired.

89. Protective measures become less effective with increase in the inoculum potential.

90. The superficial logic of quarantines is offset by the difficulty of administration.

91. A large number of unrelated chemicals have potential and practical value in disease control.

92. *In vitro* toxicity to the pathogen is a useful but unreliable index of the value of a chemical for disease control.

93. Biological controls show more promise for soil-borne than for airborne pathogens.

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