Possible questions (question bank) in Plant Disease Epidemiology

For MSc students, Dept. of Plant Protection

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1. When a plant disease is become epidemic?
2. What we mean by Disease rating?
3. What are the purposes of disease assessments?
4. What is disease incidence and disease severity? Which one is more reliable? Explain why?
5. What is disease survey? What is the purpose of surveys?
6. What are other activities that interrelated to disease assessment?
7. What does it mean by Traditional methods of disease assessment and nowadays methods for disease assessments?
8. What is loss assessment? Why we do these assessments?
9. What are the factors that should be considered during sampling for disease assessments?
10. What affects the accuracy of sampling for disease?
11. What is the connection between the growth stage of the crop or plant at the time of the assessment?
12. Mention why simple interest diseases (monocyclic or polyetic) may well require fewer assessments than compound interest or polycyclic diseases?
13. Why in monocyclic pathogens the amount of disease at the end of the season is proportional to the initial inoculum but, with polycyclic pathogens, the relationship is less direct?
14. Mention what affects the timing and frequency of disease assessment?

15. What are the equations of Disease incidence and disease severity? Are they considered direct or indirect and/or qualitative or quantitative methods?
16. Why researchers say that the Disease incidence is a binary variable?
17. Mention the type of diseases that are suitable with disease incidence and those suitable with disease severity?
18. What are the role of fungal colonies in disease measurements? Give an example of plant disease that is colony dependent measurement.
19. What is Disease prevalence (occurrence)? How it can be calculated?
20. What are the disease assessment keys? Which of them is more appropriate?
21. Mention why percentage scales are more advantages than some other scales?
22. What are the possible operator errors during visual assessments?
23. What is disease index (DI)? Write down how it can be calculated?
24. What are the Direct qualitative methods? Name a plant disease that can be assessed using this type of method.
25. What does it mean by Indirect disease assessment methods? Give an example.
26. What are the Indirect disease assessment methods for air and/or rain-borne fungal spores?
27. Explain why the data from the visual assessment of plant disease severity do not correlate with the amount of fungal biomass colonizing host tissue, which leads to inaccurate disease-yield loss relationships?
28. What is Remote sensing? Is it considered a direct or indirect method of measuring diseases?
29. Explain why verifying remote sensing data should be confirmed by actual visits to the target crop is important?
30. What are the effects of lesion position on the losses? Explain with examples
31. What does it mean by empirical yield loss models? Mention TWO categories of these type of models

32. What is single-point or critical-point models? What is multiple-point models? What is the main difference between them?
33. What are integral models for loss assessments? Give an example
34. What is Area Under The Disease Progress Curve (AUDPC)? For which purpose the descriptor is used for?
35. What we mean by generalized or non-linear models? Why they called non-linear?
36. What is synoptic or multivariate statistical models used for yield loss assessments due to plant diseases?
37. On what bases does the expert systems and geographic information systems (GIS) can depend on and by which the losses could be measured?
38. Explain how the diversity of fungal spores affect plant disease epidemiology?
39. Explain the effect of the spores being actively discharged or passively discharged on the epidemiology.
40. How epidemiological diseases occur in soil inhabiting plant pathogens? explain with examples
41. It is well known that airborne spores are most effect so far for disease epidemiology, explain how this works?
42. Why zoospores are highly susceptible to disruption by surface-active agents (surfactants)? How to connect this phenomenon to a potential basis for disease control?
43. What are produce soap-like compounds (saponins)? Give examples of plants that produce such products and mention how they affect to lyse zoospores?
44. Mention how the use of surfactants such as rhamnolipids, or even crude extracts of saponin-containing tissues such as oat roots, could provide disease control in hydroponic glasshouse-cropping systems where zoosporic fungi can cause serious diseases?

45. Mention what are the features that affect long-distance dispersal
46. of airborne spores?
47. Mention the reasons that make the hyaline (colorless), thin-walled conidia of *Blumeria graminis* (cereal powdery mildew) or the wind-borne sporangia of *Phytophthora infestans* (potato blight) remain viable for only a short time on bright, cloudless days, whereas the pigmented uredospores of rust fungi (e.g. *Puccinia graminis*) and conidia of *Cladosporium* can remain viable for days or even weeks in air.
48. What are wind gusts? Mention the importance of gusts in the removal of conidia.
49. Mention on what the rate at which spores settle onto surfaces depend on? Write the equation and its components.
50. Explain on what factors the disease patterns are resulted from?
51. What are the effects of natural events on the long-distance transport of spores?
52. What does it mean by water dispersal of pathogen inocula? Give examples to explain?
53. What is disease gradient? What causes of disease gradients? What are the types of disease gradients?
54. Explain the gradients of monocyclic or polycyclic diseases in crops
55. Mention the measurement of disease component which are used for measuring disease gradients.
56. Explain why for most diseases, only some of measurements of disease component are appropriate?
57. What is inoculum source and inoculum destination? How can we classify the sources of inoculum with giving examples?
58. What is pathogen population? What are the Components of *Blumeria graminis* (cause of cereal powdery mildew) population?

59. What are the difficulties in defining the population of root-infecting fungi? Explain with examples
60. Would it be possible to measure pathogen population indirectly? Explain how with examples?
61. What does it mean by the size of population unit? On what is depend on? Explain with examples
62. Mention why the study of pathogen population dynamics will rarely be possible in isolation from the host population dynamics?
63. What are the effects of time scale on measuring pathogen populations? Then mention the effect of short generation time scale and long generation time scale on measuring the population.
64. What is latent period? Explain how the generation time correspond to the latent period?
65. Define:
    a. Pathocron
    b. Phyllocron
    c. Threshold theorem
    d. Quasiperiodic
    e. Chaotic pattern
    f. …
    g. …
66. Compare between Sclerotinia sclerotiorum (cause of rot diseases of vegetables) and Phytophthora infestans (potato late blight) in their population change, epidemics, and number of generations per season.
67. It is necessary to have a simultaneous study of host and pathogen populations when studying epidemics?
68. From where the changes in population size may come from?

69. What we mean by changes in the stage-structure of a pathogen? Explain with giving examples
70. In which cases the birth, death, immigration, and emigration, need to be considered?
71. Mention how the growth rate of a pathogen population become density-dependent and/or density independent?
72. What is the role of measuring the total count of new units infected (R_o)? explain how this works in the short-term change with a fixed host population?
73. What are the principles of threshold theorem? Does it releted to *Ro*?
74. Explain why any disease cannot increase in a crop unless *Ro* is greater than 1?
75. Mention through what main ways the past state of a population is likely to influence its present growing conditions?
76. What are the direct and indirect linkages between host and pathogens? explain by giving examples
77. To think about the long-term dynamics of diseases, you have to know several ways about pathogens life cycle and development, what are these ways?
78. It is possible that a pathogen will distinct theoretically but in reality this is impossible, explain why?
79. Through what density-dependent factors a pathogen population may altered?
80. What is competition? Mention through what the competition is activated? give different examples of competition of pathogens?
81. What is The Competitive Exclusion Principle (CEP)? How it works, explain?
82. Mention the effects of genetic resistance or susceptibility and co-evolution of host and pathogen on plant disease epidemics.