



**FACULTY OF AGRICULTURAL SCIENCES & ALLIED INDUSTRIES**

## LECTURE 17

### PLANT DISEASE EPIDEMIOLOGY

- Epidemiology deals with the outbreaks and spread of diseases in a population.
- It is the study of rate of multiplication of a pathogen which determines its capacity to spread a disease in a plant population.
- It is the most important part of the study of plant diseases from practical point of view.
- Epidemiologically, the diseases have been described as

#### **i) Simple interest diseases**

#### **ii) Compound interest diseases.**

##### **Simple interest diseases**

- In these diseases, the rate of increase is mathematically analogous to the simple interest in money.
- There is only one generation of the pathogen in the life of the infected crop.
- The primary inoculum is seed or soil borne and secondary infection is rare.
- All the infections noticed in the field are from the pre-existing inoculum in the soil.
- Most important example is loose smut of wheat (*U. tritici*), where the inoculum is internally seed borne or is carried in the seed resulting in the infected ears in the season showing black powdery loose mass in place of grains.
- In wilt and root rot diseases, the primary inoculum is important and there are very remote chances of secondary spread, even if the pathogen sporulates, due to the soil barriers and other factors.

##### **Compound interest diseases**

- In these diseases, the rate of increase is mathematically analogous to compound interest in money.
- The pathogen produces spores at a very rapid rate which are disseminated by external agencies like air, thereby infecting other plants.
- The incubation and sporulation period is also very short.
- The infection cycle is repeated many times during the cropping season.
- Secondary inoculum plays a vital role in the development of epidemics in such diseases.
- The prevailing environmental conditions play an important role in such diseases.
- Wheat rust, late blight of potato and apple scab are some diseases of this type.

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### Mathematical model of disease spread

- Vander Plank in 1963 in his historical book "Plant Diseases- Epidemics and Control" suggested a model based on the infection rate 'r'.
- It is the rate at which the population of the pathogen increases. The 'r' is on average estimated from successive estimates of population of pathogen as proportion 'X' of the infected plants (in case of systemic diseases) or of infected susceptible tissues in case of local lesion diseases.
- The equation for describing the epidemic is:
- $X = X_0 e^{rt}$

Where X = proportion of disease at any time

$X_0$  = the amount of critical inoculum

r = average infection rate

t = time during which infection occurred

- The value of e in the equation is the base of natural logarithm = 1 + 1/n
- We are dealing here with an exponential function and the basic assumption here is that at a given time the rate of disease increase is proportional to the amount of disease at that moment. This assumption is true at the beginning where plenty of tissue is there to be infected. As the disease progresses, the amount of susceptible tissue left for infection declines and the rate of increase is determined by the amount of susceptible tissue left i.e. 1-x, but not the one which is present (x). The infection rate 'r' is very important and used to compare epidemics of diseases at different localities/cultivars /fungicide treatments. The comparative 'r' is derived by taking log and transposing:
  - $\log e^x = \log e^{x_0} + rt$
  - $rt = \log e^x - \log e^{x_0}$
  - $r = \frac{1}{t} \log e^{x/x_0}$
  - t
- This 'r' can be assumed at any time during the epidemic but its use is simpler in early stages of development of the epidemics which is an important stage. For comparing two epidemics in cultivars or otherwise, 2 readings at the starting point i.e. t<sub>1</sub> (x<sub>1</sub>) and the other time i.e. t<sub>2</sub> (x<sub>2</sub>) are must.
  - Average infection rate in them calculated as:
    - $r = \frac{1}{t_2 - t_1} \log e \frac{x_2}{x_1}$
    - t<sub>2</sub>-t<sub>1</sub> x<sub>1</sub>
  - When the disease has reached very high intensity, the formula for calculating 'r' is more complicated because the factor (1-x) is introduced. In that case:
    - $R = \frac{1}{T_2 - T_1} \log e \frac{x_2 (1 - x_1)}{x_1 (1 - x_2)}$
    - T<sub>2</sub>-t<sub>1</sub> x<sub>1</sub> (1-x)

### Slow and rapid epiphytotics

- The form an epidemic can take is governed by
- the nature of pathogen

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- its host
- the weather which works as a reference in the battle between the two.
- At one extreme are the epidemics which develop slowly (tardive) and at the other end are those which develop rapidly (explosive).
- Many intermediate types may also occur.
- Slow epiphytotics occur among populations of perennial, long lived plants such as fruit trees.
- The causal organisms are mostly systemic to varying extents e.g. Dutch elm disease or chestnut blight.
- Rapid epidemics are chiefly caused by non-systemic pathogens with high multiplication/ reproduction rate which have several generations within a short time.
- Mostly annual crops are affected by such epiphytotics.
- They are more affected by environments than the slow epiphytotics.
- The increase of disease is rapid rising to distinct peak in short time, then showing sharp decline as the weather turns unfavorable, host become resistant due to maturity etc.

### Analysis of epidemics

- Epidemic is a system i.e. an inter-locking complex of processes characterized by many reciprocal cause- effect pathways.
- It is a structural and functional phenomenon having two or more separable components and some interaction between these components.
- However, each component of the system can be studied separately.
- Analysis of a system with 2 or a few components is easy but that with too many variable and non variable components with their own components and all interacting with each other is a tedious and time consuming job.
- Epidemics interact with other sub eco-systems of agro-ecosystem making it more complex.
- In such a complex scenario, it is rather difficult to pin point the individual effect of any of the parameters say temperature or rainfall or any cultural practice on development of epidemics and its overall contribution.
- However, there are some diseases in which the role of some critical parameters like temperature, leaf wetness and rainfall or RH has been studied.

### Elements of epidemics

- The disease epidemics develop as a result of the timely combination of the same elements that result in plant disease:
  - susceptible host plants,
  - a virulent pathogen,
  - favourable environmental conditions over a relatively long period of time.
- Humans may unwittingly help initiate and develop epidemics through some of their activities, e.g., by tipping or pruning plants in wet weather.
- Humans may also stop the initiation and development of epidemics by using appropriate control measures under situations favourable for epidemics.

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- The chance of an epidemic increases when the susceptibility of the host and virulence of the pathogen are greater, as the environmental conditions approach the optimum level for pathogen growth, reproduction, and spread, and as the duration of all favourable combinations is prolonged or repeated.

### The Disease Pyramid

- To describe the interaction of the components of plant disease epidemics, the disease triangle can be expanded to include time and humans factors.
- The specific point in time at which a particular event in disease development occurs and the length of time during which the event takes place affect the amount of disease.
- The interaction of four components can be visualized as a tetrahedron, or pyramid, in which each plane represents one of the components. This figure is referred to as the disease tetrahedron or disease pyramid.
- The effect of time on disease development becomes apparent when one considers the importance of the time of year, the duration and frequency of favourable temperature and rains, the time of appearance of the vector, the duration of the infection cycle of a particular disease, and so on.
- If the four components of the disease tetrahedron could be quantified, the volume of the tetrahedron would be proportional to the amount of disease on a plant or in a plant population.

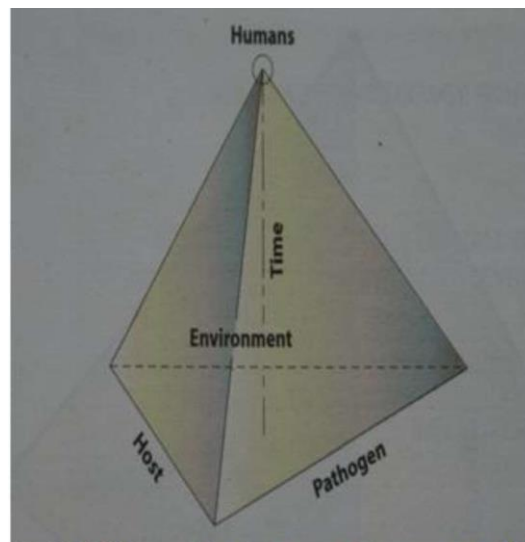


Fig.23.1 Disease tetrahedron or disease pyramid.

### Effect of Humans

- Disease development in cultivated plants is also influenced greatly by a fifth component: humans.
- Humans affect the kind of plants grown in a given area, the degree of plant resistance, the numbers planted, time of planting and density of the plants.

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- By the resistance of the particular plants they cultivate, humans also determine which pathogens and pathogen races will predominate.
- By their cultural practices, and by the chemical and biological controls they may use, humans affect the amount of primary and secondary inoculum available to attack plants.
- Humans also modify the effect of environment on disease development by delaying or speeding up planting or harvesting, by planting in raised beds by protecting plant surfaces with chemicals before rains, by regulating the humidity in produce storage areas, and so on.
- The timing of human activities in growing and protecting plants may affect various combinations of these components to a considerable degree, thereby affecting the amount of disease in individual plants and in plant populations greatly.
- The human component has sometimes been used in place of the component “time” in the disease tetrahedron, but it should be considered a distinct fifth component that influences the development of plant disease directly and indirectly.