

Plant disease epidemiology – Meaning and importance, difference between simple and compound interest diseases – Factors affecting plant disease epidemics – host, pathogen, environment and time factor

Epidemiology or epiphytology is the study of the outbreak of disease, its course, intensity, cause and effects and the various factors governing it. Based on the occurrence and geographical distribution they are classified as follows:

Endemic or Enphytotic

When a disease is more or less constantly occurring year after year in a moderate to severe form in a country or locality then it is called as endemic disease. eg: wart disease of potato (*Synchytrium endobioticum*) is endemic in Darjeeling, citrus canker (*Xanthomonas axonopodis* pv *citri*) in Asia and sorghum rust (*Puccinia purpurea*).

Epidemic or Epiphytotic

It is a sudden outbreak of a disease periodically over a widespread area in a devastatingly severe form causing severe losses or complete destruction. This is constantly present in a locality but it assumes severe form only on occasions. This is because of the occurrence of favorable environment responsible for the rapid development of disease. eg: wheat stem rust (*Puccinia graminis tritici*) and powdery mildew (*Erysiphe graminis* var *tritici*), late blight of potato (*Phytophthora infestans*), red rot of sugar cane (*Colletotrichum falcatum*), downy mildew of grapevine (*Plasmophora viticola*) and rice blast (*Pyricularia oryzae*).

Certain disease are endemic in one area and become epidemic in another area. Eg: Citrus canker is endemic in Asia but epidemic in the introduced place, Florida (U.S.A). The downy mildew of corn is a endemic disease in India but became epidemic in the Philippines.

Pandemic

When an epidemic disease spreads over continents or subcontinents and involves mass mortality it is considered as pandemic. The outbreak of black stem rust of wheat in India during 1947 is best example for a pandemic disease.

Sporadic

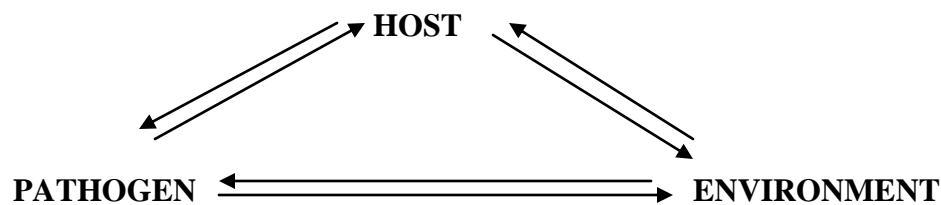
Diseases which occur at irregular intervals over limited areas or locations are called sporadic. They occur relatively in few instances. Eg: Fusarium wilt of cotton (*Fusarium*

oxysporum f sp. vasiinfectum) grain smut of sorghum (*Sporisorium sorghi*) and loose smut of wheat (*Ustilago nuda*).

An epidemic may cause widespread and mass destruction of crop in a short time or may persist for long periods depending upon the three following factors responsible for the disease:

1. Host
2. Pathogen and
3. Environment

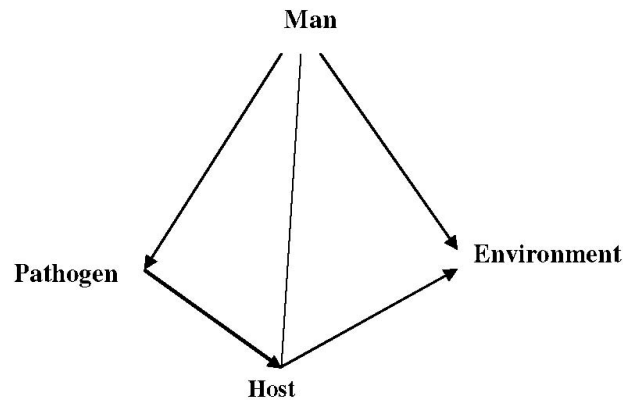
Environment flow chart



Pathogen

A course of epidemic in nature differs with the nature of the host, the pathogen and the environment. In arecanut the Koleroga fungus, *Phytophthora arecae* become destructive during monsoon period (July-Sep) and wanes away with rising temperatures and dry conditions. The above disease once again become destructive during rainy season. This type of epidemic is known as seasonal epidemic or annual epidemic. Outbreak of *Phytophthora* wilt of betelvine occurs during rainy season in South India. In temperate zone peach leaf curl and apple scab follow the similar course.

Epidemics caused as a result of introduction of new pathogens in the locality hither to free from them, appear in two phases viz., destructive phase and innocent phase (due to biologic equilibrium reached between new comer pathogen and the original inhabitant). The well known epidemics of late blight of potato in Europe and blast disease of rice in South East Asia, powdery mildew and downy mildew of grapevine in Europe, leaf rust of coffee in Sri Lanka and anthracnose of grapevine in India are examples of this category. In the above diseases the pathogens after taking heavy toll of the crops have settled down.



Factors governing epidemic or essential conditions for an epidemic

A disease is sometimes sporadic and assumes epidemic proportions under special circumstances. The essential conditions for an epiphytotic or the factors governing epidemics can be grouped under the three heads.

1. Nature of host
2. Nature of the pathogen and
3. Environment

An epidemic can only result from the cumulative effects of all the three factors mentioned above, acting simultaneously. Few pathogens are capable of assuming epiphytotic conditions while others are sporadic. The former group consists of late blight of potato, blast of rice, downy mildew diseases and rust diseases.

Host	Pathogen	Environment
Susceptibility of the host	Introduction of a new pathogen	Temperature
Aggregation and distribution of susceptible hosts	Presence of aggressive strain of the pathogen	Moisture and humidity
Introduction of new hosts	High birth rate of the pathogen	Rainfall
Introduction of	Low death rate of the pathogen	Light and

new collateral or alternate pathogen host		shade
	Easy and rapid dispersal of the pathogen Wind Adaptability of the pathogen	Wind

A. Host Factors

1. Susceptibility of the host

Plants have ability to combat disease which manifests itself as susceptibility or resistance. Plants are predisposed to the attack depending on their nature, environment and stage of growth. Presence of susceptible varieties in an area may act as one of the causes of epidemic. For example, late maturing varieties of groundnut are more susceptible to early leaf spot (*Cercospora arachidicola*) and late leaf spot (*Phaeoisariopsis*) than the early maturing varieties. Similarly late maturing varieties of wheat are susceptible to loose smut (*Ustilago nuda tritici*) than the early maturing varieties. Early sown sugarcane varieties of sugarcane are more susceptible to leaf rust in Deccan canals in Bombay area than the late sown varieties.

Wheat plant becomes susceptible to black rust (*Puccinia graminis tritici*) at the boot stage but is resistant when young. Susceptibility of rice plants to blast disease (*Pyricularia oryzae*) increases with application of heavy doses of nitrogenous fertilizers. Cottons plants are susceptible to *Fusarium* wilt (*F.oxysporum f.sp. vasinfectum*) at soil temperatures of 26 to 28°C, brinjal to *Verticillium* wilt *Verticillium dahliae* at 20°C. But crop plants are resistant to these soil-borne diseases at relatively lower or higher temperatures. Under the above conditions, the pathogen multiplies faster, cause infection and effectively uses its propagules for quick secondary spread causing epidemic.

2. Aggregation and distribution of susceptible hosts

Abundance of susceptible hosts in an area is one of the major causes of the spread of epidemics. Continuous cultivation of susceptible variety or varieties in an area, that too in a large contiguous area help in the build up of inoculum and improve the chances of epidemics. Under the above conditions the pathogen increases the rate of multiplication of its propagules and repeats the disease cycles in a short span. Wheat cultivation area in the U.S.A and Canada and

rice cultivation area in East Asian countries are exposed to a greater danger of epidemics by wheat black rust and rice blast respectively.

Destructive epidemic of early and late leaf spots of groundnut in Bombay area (Gujarat and Maharashtra States) during 1912-1913 was mainly the result of cultivation of local varieties in a larger area. Panama wilt (*Fusarium oxysporum* f.sp. *vasinfectum*) susceptible table variety, 'Son' in banana was responsible for the destructive epidemic in parts of Bombay area (Gujarat and Maharashtra) during 1936 – 1940 Countrywide cultivation of red rot (*Colletotrichum falcatum*) susceptible sugarcane varieties (local varieties like *Pundy*, *Khajuria* etc.,) practically made their cultivation impossible in Bombay area.

3. Introduction of new host (s)

Disease proneness in the host is induced by environment and other factors. The host is liable to vigorous attack and successful infection by the pathogen. A resistant or moderately resistant variety may become susceptible or highly susceptible. A susceptible variety may become highly susceptible when conditions favouring proneness are existing and cause severe damage. Under the above conditions the pathogen multiplies faster, cause infection and produces more propagules for secondary spread. Introduction of an exotic cotton variety (C4 (Cambodia) caused outbreak of bacterial blight (*Xanthomonas axonopodis* pv. *malvacearum*) and grey mildew (*Septoacylindrium gossypii*) in local variety, Deviraj, grown in Maharashtra area in India.

4. Introduction of new collateral or alternate hosts

Alternate hosts are those plants on which the heteroecious pathogens pass part of their life cycles. Similarly, collateral hosts are some wild plants in which the pathogen survives when primary host is not available. Both alternate and collateral hosts are important in building up the primary inoculum to the next crop. They determine the course and intensity of an epidemic.

Grass hosts (collateral hosts) of *Sclerospora sacchari*, *S. philippinensis* (downy mildews), *Pyricularia oryzae* (rice blast), *Ustilago scitaminea* (sugarcane smut) may produce abundant inoculum which aid in building up of epidemics. Outbreak of heteroecious blister rust of pine (*Cronartium ribicola*) in Europe and the U.S.A happened due to import or introduction of *Pinus strobus* from the USA.

B. Pathogenic Factors

1. Introduction of new pathogen

Some pathogens, epidemic in certain area, may become quite aggressive and outbreak as epidemic when introduced to new area. For example late blight of potato caused by *Phytophthora infestans* was epidemic in South America. This disease became epidemic when the infected tubers were introduced in Europe (in 1843-45). Fire blight (*Erwinia amylovora* in North America is endemic. Fire blight spread to Pacific coast fruit-growing areas of the U.S.A in 1884 and subsequently it reached Canada. It reached New Zealand in 1919 and it appeared in England in 1957. The mode of introduction had been through fruit boxes. Coffee rust (*Hemileia vastatrix*) is indigenous in Ethiopia, where *Coffea arabica* is native. The disease spread to Sri Lanka in 1869, India in 1870, Sumatra in 1876, Java in 1878 and the Philippines in 1889. It also spread from Kenya to the Congo by 1918 and reached the Cameroons. From 1950 onwards, it spread to the remainder of West Africa.

The mode of long distance transport of *H. vastatrix* is wind. Spores have been trapped at up to 1000 m above sea level up to 150 m from infected sites. Dutch elm diseases (*Ceratocystis ulmi*) first reported in 1919 in Holland, spread throughout Europe and reached Great Britain in 1927. It was introduced to the eastern United States on elm logs imported from Europe.

2. Presence of aggressive strain of the pathogen

All the strains of a pathogen are not aggressive. Only the aggressive strains are capable of causing infectious diseases which spread as epidemic. They are characterised by rapid cycle of infection and causing successful infection in new hosts. Rapid cycle of infection is essential for successful infection and it happens only by aggressive strain of the pathogen. e.g., *Puccinia graminis tritici* (wheat black rust) in India, stripe rust, bunt and loose smut of wheat in the U.S.A. and Europe. The possibility of outbreak of epidemics increases with the number of physiologic forms or pathogenic strains of the pathogen present in a locality.

3. High birth rate of the pathogen

Pathogen with high reproductive capacity and capable of rapid dissemination over wide areas mostly cause epidemics. The fungal members causing powdery mildews, downy mildews, rusts, blasts, blights etc., produce enormous amount of spores. These spores are easily dispersed by using water or insects and cause infections to new plants. The high degree of fecundity and

the enormous amounts of inoculum produced by some common plant pathogens are given in table.

Fecundity rates of plant pathogens

Sl. No	Pathogen	Extent of fecundity
	Wheat stem rust (<i>Puccinia graminis tritici</i>)	Twenty five trillion uredospores in one hectare of wheat crops.
2	Wheat stem rust (<i>Puccinia graminis tritici</i>)	64,000 million aeciospores from aecial cups in a single barberry bush
3	Cedar rust of apple	Two billion teliospores in a single gall.
4.	A corn plant infected with downy mildew	225 million sporangia in one night
5.	A. grapevine infected by downy mildew	32,000 sporangia per sq.cm
6.	Bunt of wheat	6 to 12 million smut spores in a single kernel
7.	Smut of corn	125,000 billion smut spores in one hectare
8.	Chestnut blight	150,000,000 spores in a single spore horn
9.	<i>Fomes applanatus</i>	5,460 billion spores in a single fruiting body

4. Low death rate

Epiphytotics may also be caused by low death rate diseases. These diseases are caused by agents of systemic nature which are protected by plant tissues. As they are protected by plant tissues the chances of high mortality is reduced to the minimum. In these diseases the chief source for accumulation of inoculum for epiphytotics is the diseased plant organ used for vegetative propagation (corms, setts, tubers, etc.). Here the buildup of epidemics is comparatively low compared to high birth rate diseases. When a particular area is planted and covered with diseased planting material the chances of occurrence of epiphytotics are very high. e.g., virus and phytoplasma diseases in crops propagated through vegetative plant parts.

5. Easy and rapid dispersal of the pathogen

The ability of the pathogen to cause epidemic depends both on the high birth rate and dispersal. The propagules of the pathogen produced should be dispersed for development of an epidemic. It may happen by external agencies like wind, water, insects, mites and nematodes. Fungal spores / conidia are minute and light and resistant to adverse conditions. Fungal spores are mostly disseminated by wind. Bacteria are mostly disseminated by water or insects. Virus and phytoplasma diseases are mostly transmitted by insects, mites or nematodes. Epidemics are determined by the velocity of wind, direction of wind, moisture, relative humidity, temperature, presence and number of vectors and their rate of reproduction.

6. Adaptability of the pathogen

Pathogens have the capacity to adapt to adverse conditions. Fungi produce different types of spores like oospores, ascospores and smut spores (chlamydospores) which help in tiding over adverse conditions. Bacteria survive in diseased plant parts. Viruses and phytoplasmas live in collateral hosts or insect vectors in the absence of the suitable crop hosts.

C. Environmental Factors

The environmental conditions such as temperature, relative humidity, rainfall, duration and intensity of light, etc. play very important role in causing epidemics. These are actually the deciding factors and influence almost all the stages of disease cycle. Favourable environmental conditions are needed for sporulation, liberation of spores, dissemination of pathogen, germination, infection and establishment of pathogen in the host.

For example, persistent optimum temperature and moisture are needed for spore germination and entry of germ tube in the host. Similarly optimum temperature, moisture, light and specific nutrition is required for the development of the disease and sporulation of pathogen. Compound interest diseases and simple interest diseases The terms compound interest and simple interest are for explaining rate of increase of pathogen. These terms were introduced by Van der Plank in 1963 in the book 'Plant Diseases-Epidemics and Control'. Based on the mode of multiplication of pathogen, the diseases are classified of two types:

1. Simple interest diseases
2. Compound interest diseases

1. Simple interest diseases

In simple interest diseases the increase is mathematically analogous to simple interest in money. There is only one generation of the pathogen in the life of the crop. The primary inoculum is seed-borne or soil-borne. The secondary infection rarely occurs during the crop season. That is, the pathogens do not spread from plant to plant in one growing season. Simple interest diseases are caused by seed-or soil-borne smuts, like loose smut of wheat, covered smut of barley and soil borne fungi which attack roots, like wilt (*Fusarium oxysporum*) and root rot (*Rhizoctonia* spp.) diseases.

Most of the smuts infect the seedlings, grow along with the growth of the plant and produce spores in the inflorescence on maturity of the crop. There is no secondary spread from the smutted heads. These smut diseases are mostly systemic in nature. They do not produce propagules external to the host during the active season of the crop. Dispersal of propagules of these fungi is restricted by existing climatic and biotic conditions.

2. Compound interest diseases

In compound interest diseases the rate of increase is mathematically analogous to compound interest in money. The pathogen produces enormous amount of spores at a very rapid rate. These spores are disseminated rapidly by wind and infect the other plants. Both the inoculation and sporulation period are short so that the pathogen spreads from plant to plant during the same growing season. New crop of spores is produced, disseminated and the cycle is repeated fast. Thus more generations of the pathogen are produced in the life of a crop. e.g., late blight of potato, powdery mildews and rust diseases. If we consider wheat stem rust caused by *Puccinia graminis tritici* as an example, the fungus produces uredospores in very large numbers (50,000 to 4,00,000 uredospores per uredosorus).

These spores are spread by wind and infect other plants. Each of the freshly infected wheat plant produces uredopustules within 5 to 7 days at 24°C. Thus within a week of appearance of the first pustule in the crop several thousand new pustules are formed which could repeat the process within a week. If the climatic conditions of about 24°C temperature and relative humidity remain for only few weeks, the entire crop is severely affected by the disease.

Course of epidemic

The course of epidemic follows two distinct phases viz.,

- i. Progressively destructive phase and

ii. the decline phase

i. Progressively destructive phase

Some epidemics develop slowly (tardive) while others develop rapidly. Slow epidemics (or epiphytotics) usually occur among population caused by systemic pathogens. The pathogen multiplies slowly following the characters of simple interest disease. They belong to low death rate category and have less incubation period and sporulation period. However, the rapid epiphytotics are greatly influenced by environmental factors.

ii. Decline phase

During early stage, an epidemic spreads vigorously causing diseases in new hosts. After development of a saturation stage it shows a decline by itself. No epidemics may be due to non-availability of susceptibles non-availability of susceptible stages of the crop, unfavourable weather conditions and reduction in aggressiveness of the pathogens. Generally the hosts are prone to the disease at a specific developing stage. Once this stage is crossed in a plant its proneness to infections is reduced or completely lost. Under the conditions the epidemic declines. The decline in the epidemic may also be due to unfavourable weather conditions for disease development. As a result future spread of the disease will be checked and the epidemic will decline. Wheat crop in Northern India usually gets the attack of rusts in January to March.

Epidemics develop during these months. Although the plant remains prone to attack afterwards also, further development of the disease is checked because of rise in temperature which is favourable for the pathogen. Due to the above mentioned and other causes, the aggressiveness of the pathogen may be reduced. When all susceptible individuals are destroyed by the pathogen, it may try to parasitize the remaining resistant individuals of the same species. In these adverse conditions, the pathogen may lose its power of successful infection, its reproduction may slow down and the pathogen becomes less aggressive.

Slow and rapid epiphytotics

The form of epidemic is decided by the nature of the pathogen, host and the weather. Epidemic may develop slowly and is called ‘tardive’. Epidemic which develops rapidly is called ‘explosive’. In between these intermediate forms of epidemic may occur.

i. Slow epiphytotics

Slow epiphytotics occur among perennial (tree) populations. Infected host survives for several years before dying. Most of the characters of a simple interest disease are found in slow

epiphytotics. The causal agent is mostly systemic. The pathogen multiplies slowly. Their movement from plant to plant is much slower. They are low death rate pathogen. In slow epiphytotics, crop sanitation is the best method. e.g., Swollen shoot of cocoa.

This disease spreads very slowly from tree to tree and still less from one garden to another garden. For instance, the incidence of 31 % swollen shoot increased to 75 % over a period of 2.5 years. As stated by Van der Plank (1959) the rate of multiplication of a systemic disease of trees is about ten fold a year whereas it is 10,000 fold in respect of herbaceous plants and it is of higher

rates for local lesion pathogens e.g., late blight of potato, wheat stem rust, etc.,

ii. Rapid epiphytotics

Rapid epiphytotics occur among annual crops. It is caused by non-systemic pathogens with high birth rate. Several generations of the pathogen is produced within a short time. Rapid epiphytotics are largely governed by environmental factors compared to slow epiphytotics. Disease increase is rapid and the disease rises to a peak in short time and then show sharp decline when the weather turns unfavourable or when the host becomes resistant due to maturity or due to restricted dispersal of propagules of pathogen. e.g., apple scab. This type of epiphytotic is controlled by protective spraying or dusting with chemicals.