

## HOST PARASITE INTERACTION

**Aim: To acquaint the students with host parasite-interaction**

### Host Pathogen Interactions

- After the pathogen enters the host until the disease symptoms appear, a series of interactions between host and pathogen take place.
- The disease symptoms may be considered as the signs of reaction of the host to infection by the pathogen.
- The severity of the symptoms varies depending upon the capacities of each of them to supersede the other.
- In that process, which may last for a few hours to many days, different biochemical reactions become involved.
- The establishment of the pathogen or colonization of the host by the pathogen after its entry initiates the symptoms.
- The **hypersensitive reaction** of the host is a mechanism to prevent such colonization by the pathogen.
- Many pathogens which cause disease in plants are highly specific in their nutritional requirements.
- They must reach the proper site inside the host tissue to obtain the required nutrients.
- The chances of a pathogen establishing itself in a host depend on:
  - its entering the suitable host
  - its reaching the proper location within the host tissue so that the required nutrients are obtainable
- A vascular pathogen may not establish itself inside a host if it is confined to sub-stomatal region.
- Similarly, wood rotting basidiomycetous fungus may not find food for its growth if it enters the leaf tissues.
- Tissue preference of parasites has been, for a long time, attributed to specific nutrients present in that particular tissue.
- Recent evidence indicates that tissue preference is solely influenced by inhibitory substances (**prohibitins**) present in the tissue.

### Post Entry Stages

- The subsequent stages, after the pathogen has entered a susceptible host which is pre-disposed to the disease, are of two kinds:
  - The pathogen may kill the host tissue in advance, drawing nutrients from the dead cells.
  - The pathogen and host may develop an harmonious relationship or association in which neither is killed. The pathogen absorbs nutrients from the living host cells.
- Another biological relationship which exists in nature in the case of root nodule bacteria and legume plants is **sympiosis**, whereby two biological systems derive benefit from each other.

### Effects of pathogens on photosynthesis

- The pathogen directly affects the photosynthetic capacity of the host.
- In many cases the pathogen causes chlorosis of the tissues, indicating through inhibition of certain enzyme activities.
- The Hill reaction, in which water is split into oxygen and hydrogen atoms, which in turn, is coupled with the production of adenosine triphosphate (ATP) through photosynthetic phosphorylation is adversely affected.
- Apparently the enzymes of CO<sub>2</sub> fixation in photosynthesis are not affected, but the glycolic acid oxidases are affected, resulting in a reduction in protein synthesis.
- Because of the reduction in photosynthetic activity, other chain reactions in the plant result, causing increased water loss, wilting and reduction of plant vigour.

### Effects of pathogens on respiration

- In general, plants infected by most pathogens react with an immediate increase in the respiratory rate.
- This increase is mostly non-specific, as even mechanical injuries to certain tissues also cause it.
- In incompatible and destructive host-pathogen relationships, the respiratory rate is high in the early stages of infection, whereas in compatible host-pathogen relationship, as in obligate parasitism, there is little change in the respiratory rate.
- In the destructive host-pathogen relationship, the pathogen destroys the normal physiological balance, the **Pasteur effect**, (which implies that in the presence of oxygen the fermentative degradation of carbohydrates is reduced and the energy release is more) is abolished, and hence, the respiratory efficiency is also reduced.

### **Effect of pathogens on Embden-Meyerhof-Parnas pathway**

- There is an accumulation of carbohydrates, mostly starch, in the infected tissues, coupled with a shift from the Embden-Meyerhof-Parnas pathway to the pentose-phosphate cycle which causes the accumulation of reduced nicotinamide adenine dinucleotide phosphate (NADPH<sub>2</sub>).
- The pathogen causes tissue disintegration of the host, which is accomplished by the activity of several oxidative enzymes such as peroxidase, phenol oxidase and ascorbic acid oxidase, linked up with the oxidation of NADPH<sub>2</sub> to NADP.
- The hypersensitive reaction in a plant that results in necrosis also causes a shift in the respiratory pathway from the Embden- Meyerhof-Parnas to the pentose phosphate system.
- There is also an increase in the oxidative enzyme activity to cause an enhancement of oxygen uptake. If certain oxidative enzyme inhibitors are produced in the host-pathogen system, the resistance of the host plant is reduced, and in the absence of oxidative enzyme inhibitors, the resistance of the host is increased.

### **Pathogenesis related proteins**

- Infection by pathogens also interferes with the host nucleic acid and protein metabolism, especially enzymes.
- Most conspicuous changes are in peroxidase, ascorbic acid oxidase, cytochrome oxidase, phenol oxidase, etc.
- In addition, several proteins accumulate which were first called **pathogenesis related proteins** and are now referred to as **stress proteins** as they accumulate in response to physical and biological stresses.
- Both in virus infected tissues and galls, nucleic acids especially RNA accumulate.
- Hypertrophy of host cells, accompanied by increases in the size of nuclei has been demonstrated.
- There is redirection of protein synthesis towards accumulation of proteins of the pathogen, or a reduction in the protein and nitrogen level of the host, especially towards the degenerative stages of disease.
- The synthesis of virus protein markedly affects the host protein metabolism. In hypersensitive reaction, however there is no great change in the total protein content or nitrogen level in the affected tissues.

### **Phytoalexins**

- The phenol metabolism of infected plants is profoundly altered.

- In incompatible host-pathogen combinations, there is more rapid accumulation of phenolic substances than in compatible combination.
- Certain phenolics and other aromatic substances with antimicrobial properties, named **phytoalexins**, are produced post-infectionally in host tissues, and this is believed to be a response to infection directed towards imparting resistance in the host.
- This response is greater in incompatible host-pathogen combination than in the compatible one.
- Production of phytoalexins may also be induced in the host in non-pathological conditions, such as physical stress and chemical stimuli.
- The production of leaf spots, necrosis and other types of lesions on the host are correlated with enhanced phenol oxidase activity. Oxidation of phenolic substances causes the accumulation of melanin pigments in the infection site, resulting in discoloration.

### **Growth regulators**

- Due to pathological condition of the plant, the growth regulatory mechanism is upset and depends largely on the nature of host –plant relationship.
- **Auxin** level often increases in the host tissue as a result of infection by fungi and bacteria.
- This may be due to increased auxin synthesis by the host and /or pathogen, or the suppression of activity of enzymes indole acetic acid oxidase which degrades the auxins under normal conditions.
- **Gibberellins** or gibberellin-like substances have been found in many plants and are also produced by a few fungi.
- The auxin activity in meristematic tissues of plants is believed to be controlled by gibberellins.
- Infection of plants with some pathogens alters the level of ***gibberellin-like substances*** which result in the hyper-elongation of the plant.
- One such typical example is the Bakane disease of rice plants caused by *Fusarium moniliforme* (teleomorph: *Gibberella fujikuroi*).
- Dwarfism or stunting in some host-pathogen interactions is caused by a reduction in the gibberellins.
- **Kinetin (cytokinin)**, 6-furfuryl aminopurine, an essential hormone, is involved in cell division in plant tissues.
- IAA and cytokinin act in a linked manner, the former responsible for cell expansion and the latter for cell division.

- In the absence of IAA, however, kinetin seems to be functionless.
- From plants and microorganisms, cytokinin has been isolated and characterized.
- It is a precursor of tRNA. Certain plant pathogens interfere with normal cytokinin metabolism in plants causing gall formation, senescence, fasciations, yellowing of leaves, green island formation, etc. which could be reversed by the exogenous application of kinetin.
- **Ethylene**, another well known growth regulator in plants plays a prominent role in causing epinasty, leaf yellowing and senescence in diseased plants.
- Many plant pathogens favour accumulation of high levels of ethylene in host tissues which contribute to symptom development like leaf drop, e.g., due to *Diplocarpon rosae*, the black spot of rose pathogen.