ROLE OF ENZYME IN PLANT DISEASES

Introduction

In the development of plant diseases, there is constant interaction between host and pathogen. In the interaction, the pathogens try to establish themselves in the host and the subsequent development of disease involves a chemical interaction between host and pathogen. Ultimately, some biochemical substances like enzymes, toxins, growth & regulators etc. are formed which cause disintegration of specific cell contents and death of host cells as well.

Disease in plants are not the direct result of infection. There leads to a chain of reaction through different stages of disease development like inoculation, penetration, infection incubation, symptom etc. Parasitism and disease ~~development~~ resistance run parallel and a tug of war is employed between host and pathogen. Enzymes, toxins, growth regulators, polysaccharides etc. are important chemicals ~~secreted~~ released by the pathogen. Enzyme are mainly employed by fungi during penetration.

Penetration is the entrance of the pathogen through direct or indirect methods i.e. wounds, natural opening and cuticular or epidermal surfaces. The actual method of penetration in fungi is of active or aggressive type. He the fungi apply mechanical force, enzymes, toxins etc. for their entrance whenever there is some resistance.

While the penetration tube is passing through the cuticle, it usually attains its smallest diameter and appears thread like, following that the diameter often increases. After cuticle, the penetration tube now meets with the epidermal wall, consisting mainly of cellulose and secretes enzyme which softens the cell wall. From here, it passes deeper into host tissues by the enzymatic activities.

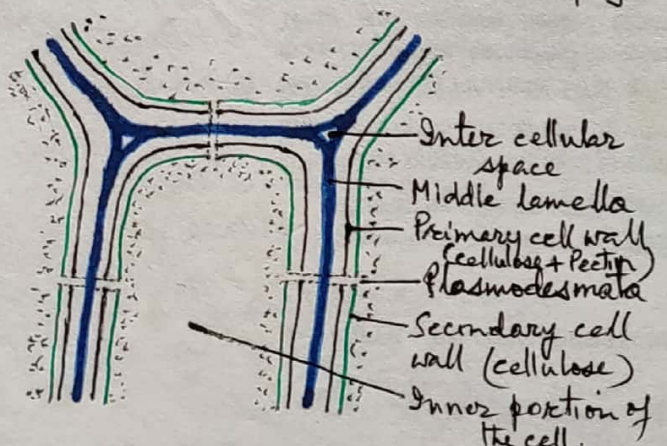


Fig. Schematic representation of structure & composition of cell wall.

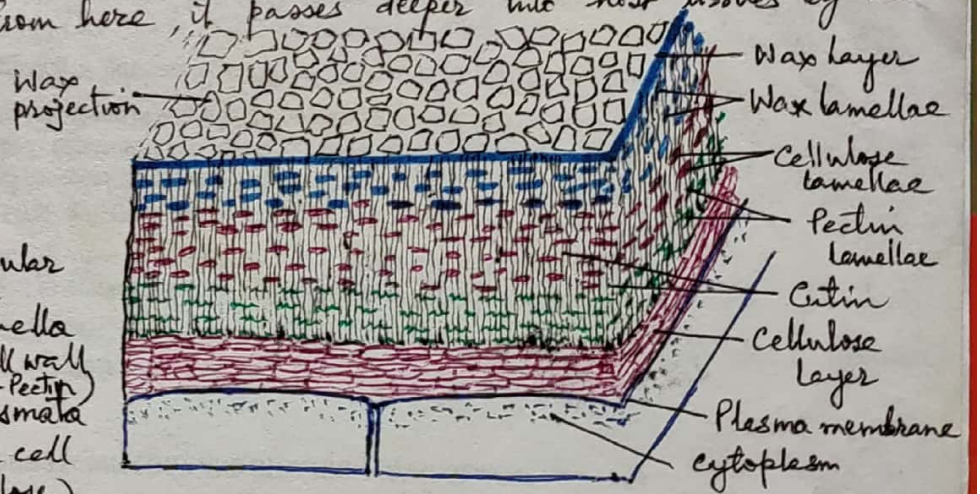


Fig. Schematic representation of the structure and composition of cuticle & cell wall of a foliar epidermal cell.

Thus the entire process seems to be first mechanical in early stage and chemical in later phase, a contact stimulus, followed by chemotropism. With the direct contact of penetration tubes with host tissue and their enzymatic activities, the contents undergoes rapid degradation, plasmolysis of cytoplasm and nucleus sets in causing ultimate injury and disintegration of invaded tissue.

In the next stage of disease development i.e. infection, pathogens release number of biochemically active substances such as enzymes, toxins, growth regulators etc. which affects number of structural integrity or physiological process of the host.

During infection, cell contents such as protein, starch, lipids and nucleic acids are degraded. In response to these, hosts produce variety of defence mechanism. Host releases enzymes during post-infection (incubation) stage. Other biochemicals are also released which inactivate pathogenic activities.

Physiological plant pathology was studied first by De Bary (1886) working on extra-cellular enzymes of Sclerotinia liberiana. He recorded two types of action - dissolution of middle lamella and killing of the host cells. Later R.L. Jones (1905) reported production of pectic enzymes by soft rot bacteria.

William Brown (1917) of London, demonstrated involvement of pectic enzyme - Protopectinase in maceration of tissue by Botrytis cinerea. In contrast to the observation of De Bary, he could not separate maceration and killing factors.

Biochemicals found in cell wall and cell contents of the host.

To understand the role of enzymes released by pathogen, one should also study the organization of the wall and cell wall components. Many species of host have a layer of wax which is followed by cuticle impregnated with wax. Cutin gradually decreases with the depth of epidermis and is replaced by pectin.

Pectin is found in a homogenous layer. Subsequently, pectin layer is replaced by cellulose. Cellulose layer may also contain some amount of protein towards innerside.

(A) At penetration stage

a/ Cuticular wax

It is the long chain of even numbers of primary alcohols, acids and their esters, secondary alcohols, paraffins, hydrocarbons etc. No pathogen can degrade it enzymatically. Only mechanical entry is possible.

b/ Cutin

It is chemically poly-ester of hydroxylated monocarboxylic acids (1-4β N-Acetyl glucosamine).

Some pathogenic fungi produces cutinase which breaks cutin by hydrolysis. The extent of distribution of cutinases among pathogens and their role in pathogenesis is yet to be - illucidated. In general, cutin is regarded only as mechanically penetrative. Cutin is chemically similar to suberin.

Heinen (1960) and Linskens et al. (1965) have reported two cutin degrading enzymes produced by ~~Rossmi~~ Penicillium spinulosum. These enzymes are cutin esterase and carboxy-cutin peroxidase (Carboxy Cutin esterase). A suberin degrading enzyme has been reported produced by Asmillarea mellea.

c/ Pectic substances

It forms the middle lamella and fills the inter-micellar spaces of cellulose fibrils. The different pectic substances are Pectic acid, Pectin and Protopectin.

Pectic acid :- It is the linear polymer of D-Galacturonic acid residue joined laterally by α 1-4 glycosidic linkage. It occurs as calcium and magnesium pectates, chiefly in the middle lamella.

Pectin :- It is methyl ester of pectic acid in which 75% or more carboxyl groups are esterified.

Protopectin :- It is similar to pectin but has very long pectin chains consisting of over 1000-2000 units (200 units of pectin). Pectic substances are degraded by pectolytic enzymes which are pectic ~~mg~~ methyl esterase, glycosidases, pectic lyases and wall modifying enzymes.

Enzymes involved

Pectic enzymes involved in the degradation of pectic substances are as follows :-

1. Pectic Methyl Esterase (PME)

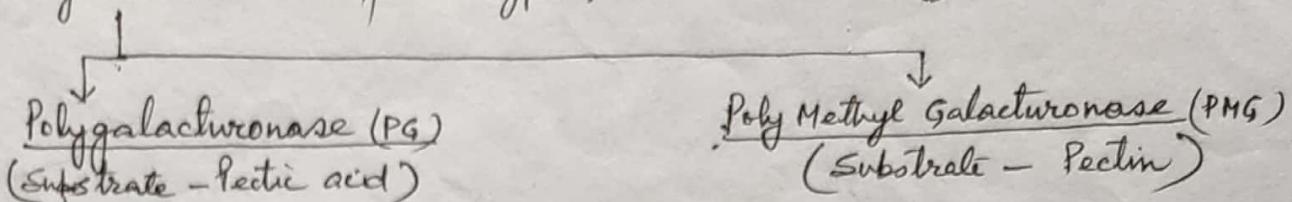
It removes methyl group from pectin and pectinic acid through hydrolysis and places carboxyl group which forms thiopectic acid. This may be neutralized by Ca or Mg to form pectates which is supposed to be a shafter material. PME is widely found among higher plants and much lesser extent in the micro-organisms.

2. Glycosidases

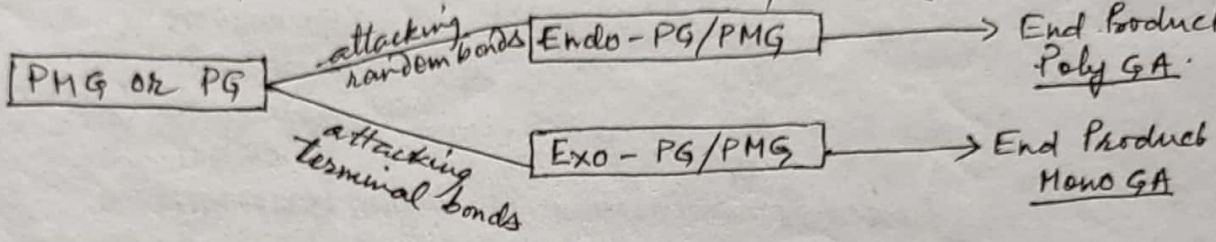
These enzymes act upon pectin and pectate chain by catalyzing the hydrolysis of α 1-4 glycoside bond ultimately ~~[illegible]~~

releasing D-Galacturonic acid or Polygalacturonoids.

Glycosidases are of two types :—



Depending upon site of attack (cleavage) and type of end product, both PG and PMG are of two types :—



3. Pectic Lyases (PL)

It was first reported by Albersheim et al. (1960). Earlier it was known as Transeliminases. They act on pectin or pectic acid and help in cleavage of bonds involving removal of the proton of C-5 of one residue resulting into formation of an unsaturated bond between C-4 and C-5.

On the basis of substrate, lyases are Pectic Acid Lyase (PAL) or Pectin Lyase (PL). These are further divided into ENDO- or EXO-, depending upon random and terminal cleavage respectively.

The pectic degrading enzymes (all types included) are produced by germinating spores in many diseases and probably act in association with other pathogen metabolites which assist in the penetration of the germ tubes of the pathogens. Pectin degradation results in the weaking of the cell walls and tissue maceration, thereby helping the germ tubes in inter- and/or intra-cellular invasion of the host tissue.

4. Wall Modifying Enzymes (WME)

Kaar and Albersheim (1970) reported this enzyme from Pectinol R-10, a commercial preperation from Aspergillus niger. Removal of this enzyme results in failure of other enzyme action.