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GENERAL INTRODUCTION AND REVIEW OF LITERATURE
The national economy of India mainly depends on its agricultural output. Though, we have made a considerable progress in various fields of life including agriculture and industries, yet we have to go a long way towards attaining the target to commensurate with population growth. Our agriculture and fruit industries suffer great losses due to post-harvest spoilage of their produce by pathogenic attacks. The colossal losses are further enhanced by the tropical conditions prevailing in India, which favour the parasitism of majority of pathogens. Chenuku and Thakur (1968) surveyed the Delhi market and reported 10.68% losses in apples, 31.45% in bananas, 17.73% in mangoes, 24.02% in potatoes and 19.32% in tomatoes. The losses in cucurbitis and brinjals reported by Chaudhary (1968), due to pathogenic attacks in storage varied from 8.15 to 10.15% and 13.96 to 26.62% in Jabalpur and Bombay markets respectively.

The microorganisms are mainly responsible for the causation of disease in fruits and vegetables during transit, storage and many times in field also. Initial understanding of disease came from the efforts of pioneer workers, like De-Bary (1866), Ward (1888), Jones (1909) and Brown (1915). Since then, a large number of workers like Horsfull (1959), Bateman (1963), Wood (1967), Albersheim et al. (1969), Dimond
(1971) and Byrd and Cutting (1971) have greatly contributed to the advancement of the knowledge of mechanisms of disease development. Horsfall and Dimond (1959) defined disease as a "malfunctioning process caused by continuous irritation, of course, these processes must result in some suffering and hence, disease is a pathological process". Agrios (1969) defined disease in plants as any disturbance brought by environmental factor interfering with normal physiology of the plant. He also involved chemical concept to define disease as "the sum of normal chemical reaction that are inhibited and of the abnormal chemical reactions induced inside the cells, and the tissue of the plant as result of the irritation brought about by the causal agent. It is now clearly understood that the disease is an expression of host and parasite interactions.

Interestingly, the pathogen appears to exist in the form of several strains. Its isolates are obtained from various hosts and they have a great deal of morphological, physiological and pathological variations.

The capacity of a pathogen in the invasion and expression of diseased state in the host is known as "pathogenicity". The interaction between host and parasite results in a number of sequential events. The pathogen, by virtue of its ability, gradually invades deep into the host tissues and simultaneously starts obliterating the normal metabolic pattern of the host.
Finally, with full establishment of parasite, the disease symptoms appear on the host. These symptoms include hypertrophy, hypotrophy, necrosis, curling, chlorosis and mottling, etc. The physiological state of the host is rendered weak by the pathogen and many-a-times it leads to its death. All these sequential events during pathogenesis have been lucidly discussed by Brown (1936), and Agrios (1969). Agrios (1969) described the process of pathogenicity mainly in the following steps:

Inoculation — In this process the pathogen comes in direct contact with the host.

Penetration — In this step the pathogen enters into the host through natural openings like stomata, lenticels, nectaries, etc. or through injury/wound.

Infection — In this, invading pathogen establishes contact with the susceptible host tissues and obtain nutrition from them.

After a gap of some time host expresses a number of reactions which are known as "symptoms". The time lapse between inoculation and expression of symptoms by the host is known as "incubation period". The environmental factors
play an important role in the disease development. A number of workers, on the experimental basis, emphasized that relative humidity, temperature, various types of injuries and nutritional factors have a marked influence on the development and incidence of various plant diseases. De-Bary (1886) showed that when the mycelium of Sclerotinia libertiana was placed on the intact surface of the host with nutrient medium infection readily took place. White and Baker (1954) found that Erysiphe graminis var. hordei could penetrate the host tissue even without the supply of external nutrients. Inoculum of certain fungi like Alternaria tenuis, Botryodiplodia theobromae and B. ananassae invariably require injury as a prerequisite (Tandon and Ghosh, 1962; Tandon and Bhargava, 1952; and Williamson and Tandon, 1966). Baker and Heal (1932) and Ramsey (1951) recorded that the lenticular regions on the apple surface are the major sites of entry by Penicillium expansum. Brown (1922) found that exosmosis of nutrients from the host tissues facilitated the infection. Lauritzen et al. (1925) also studied the effect of temperature and humidity on the infection and decay of sweet potatoes caused by Rhizoctonia sp. Studies carried out by Tandon and Tandon (1948), Tandon and Bhargava (1958) and Agrwala and Sharma (1968) established that certain pathogenic forms can penetrate only through specific sites like lenticels, stomata, calyx and stalk portion or intact surface of host. Brown
(1936), Gaumann (1950), Dickanson (1960) and Wood (1967) extensively reviewed this problem. Choudhry (1957) observed that 90% humidity and 26°C temperature were most suitable for the disease development in chillies. Tandon and Ghosh (1962) reported in pear fruit-rot caused by *Alternaria tenuis* that temperature played an important role in disease development. Mishra and Singh (1962) worked on banana anthracnose with special reference to temperature and relative humidity. Pierson (1966) also studied the effect of temperature on the growth of *Rhizopus stolonifer*. According to Wood (1967) humidity and nutritional factors have some relation in disease development caused by microorganisms. Singh and Prasad (1967) studied the epidemiology of anthracnose of *Dioscorea alata*. Chand et al. (1968) studied the epidemiology and control of bitter rot of apple caused by *Gloeosporium fracticenum*. Shrivastava and Tandon (1968) studied the influence of temperature on *Botryodiplodia* rot of citrus and sapodilla. *Botryodiplodia* rot of guava was studied by Shrivastava and Tandon (1969). Ali (1970), Rai (1971) and Rai S. (1971) studied the role of temperature, relative humidity and injury in disease development of *Musambi*, Papaya and Chillies respectively. Mehta et al. (1975) made detailed investigation on the pathogenicity of *Alternaria tenuis* and *Alternaria solani* responsible for the rot of tomato fruits, and concluded that relative humidity, temperature and age of culture
influenced the disease development. Third (1977) got similar results while working with Clathrodiium rot of apple fruits. Atri (1979) studied the effect of age of culture, temperature and relative humidity in relation to pear (Pyrus pyrifolia) rot caused by Botryodiplodia theobromae. Saxena (1982) has studied brinjal fruit rot disease caused by Rhizopus nodosus and Phytophthora nicotianae.

Diseases of plants mainly 'rots' are generally caused by certain enzymes secreted by the pathogen into the host or by host pathogen interactions. These enzymes decompose the cell walls and the pathogen goes inside and utilizes the nutrition of host cells by killing them. The normal function of the host is often disturbed by the pathogen by producing toxins (Luke and Wheeler, 1955; Leal et al., 1966; and Wood, 1972). The host also reacts against invading pathogen by its own defence mechanisms which involve the production of fungitoxic substances including phytoelaxines and phenolics resisting against the disease development.

The concept of cell wall degrading enzymes being involved in the host pathogen interactions was given by Brown (1915) and was supported by Tribe (1955), Fusteley (1957), Adam and McAllan (1956), Albersheim and Killias (1962), Mandle and Reese (1965), Bateman and Millar (1966), Dimond (1970), Wood (1970), Hall and Wood (1973), Mullen and Bateman (1975) and Skrekantia (1976).
The structure of cell wall is very complex and is considered to be of two parts — a thin primary cell wall and a thick secondary cell wall (Albersheim, 1965). Primary cell wall is the product of protoplasm, whereas the secondary thick wall is the transformation of primary cell wall after the cells stop growing. Biological investigations reveal that in the higher plants primary cell wall is made up of two phases (Northcole, 1958). The microfibril component of all green plants is composed of cellulase, characterized by long chains of β-1,4 linked glucose residue consisting of 8000 to 12000 units (Marx-Figini, 1964). The glucose chains bound each other by hydrogen bonds. The matrix of primary cell wall consists of polysaccharides with little amount of protein, pectin-polysaccharide and hemicellulose which are the major non-cellulosic polysaccharides found in plant cell walls. A large number of acids and natural polymers have been found in the pectic polysaccharide extraction. The acidic fraction consists of α-1-4 galacturonic acid residue in which two residues are interpressed (Aspinali et al., 1968).

According to Northcole (1972) water is supposed to be a very important component of cell wall matrix. He has given four important functions of it in the cell wall. These are as follows:

(a) as a structural component concerned with the regulation of polysaccharide gel viscosity.
(b) as a wetting agent governing the formation of hydrogen bonds.

(c) as a lattice complex.

(d) the water content of the wall obviously has a direct effect on cell wall permeability and it allows the presence of calcium ions which may form salt with acidic cell wall constituents.

Lignin is also one of the important components of cell wall matrix, and it is found in secondary thickening. It is soluble and aromatic with higher molecular weight it is formed/enzymatic degradation and subsequent polymerization of coumaryl coniferyl and sinapyl alcohols (Freudenberg, 1968).

Pectolytic enzymes are most important amongst the cell wall degrading enzymes. In the light of modern researches, the appearance of these enzymes during the process of pathogenesis is considered to be immediately related to the disintegration of structural components (cellulose, hemicellulose and pectic substances). Pectic substances in middle lamella and polygalacturonase rich compound in the matrix of the cell wall are obviously degraded during pathogenesis. These enzymes and tissue macerating enzymes involved in pathogenesis are produced by many phytopathogenic
fungi. They have an important role in the pathological investigations and therefore have been widely studied in vitro and in vivo in relation to various plant diseases. Many reviews related to pectic enzymes in relation to disease development have appeared in the past two decades (Husain and Kelman, 1957; Demain and Phaff, 1957; Sadasivan and Subramanian, 1963; Bateman and Miller, 1966; and Albersheim et al., 1969).

Pectic substances:

The pectic substances are linear chains of galacturonic acid units linked in $\alpha$-1-4 glycosidic linkage. Carboxyl groups in the chain are either not esterified or esterified to different degrees with methanol. On this basis, they are classified as follows:

Pectic acid: Here carboxyl groups are not esterified by methyl ester (-OCH$_3$) group. They form salts with polyvalent cations like Ca$^{++}$ and Mg$^{++}$ which are known as pectates.

Pectinic acid: These are the substances in which up to 75% carboxyl groups are methylated. They form the pectinates with mono- and poly-valent cations.

Pectins: When methylation of carboxyl group is beyond 75% they are known as pectins.
Protopectin: The insoluble native pectin material consisting of 1000-2000 galacturonic acid units is called protopectin. It differs from pectins, primarily in chain size, which consists of about 200 units and in the degree of methylation. A number of pectic enzymes are known to be involved in the breakdown of various types of pectic compounds.

On the basis of following criteria, Bateman and Miller (1966) have classified various pectic enzymes into pectic glycosidases and lyases:

(1) Mechanism with which $\alpha \leftarrow 1,4$ glycosidic bond is splitted, i.e., hydrolytic or transeliminative cleavage.

(2) Enzyme preference for Cl substrate that is pectin or pectic acid.

(3) Position of cleavage in the chain, i.e., terminal (exo type) or at random (endo type).

Classification of pectic glycosidases and lyases:

$\Delta A 7$ Hydrolytic cleavage of $\alpha \leftarrow 1,4$ glycosidic bonds of pectic substances.

(1) Random mechanism of hydrolysis —
(a) Pectin attacked in preference to pectic acid (endo PMG) endo polymethyl galacturonase.

(b) Pectic acid attacked in preference to pectin (endo PG) endo polygalacturonase.

(2) Terminal mechanism of hydrolysis —

(a) Pectin attacked in preference to pectic acid (exo PMG) exo polymethyl galacturonase.

(b) Pectic acid attacked in preference to pectin (exo PG) exo polygalacturonase.

\[ B \text{ Transeliminative cleavage of the } \alpha-1,4 \text{ glycosidic bonds of pectic substances.} \]

(1) Random mechanism of transeliminative degradation —

(a) Pectin attacked in preference to pectic acid (endo PMTE) endo pectin methyl transeliminase.

(b) Pectic acid attacked in preference to pectin (endo PGTE) endo polygalacturonase transeliminase.

(2) Terminal mechanism of transeliminative degradation —

(a) Pectin attacked in preference to pectic acid (exo PMTE) exo pectin methyl transeliminase.

(b) Pectic acid attacked in preference to pectin (exo PGTE) exo polygalacturonase transeliminase.
Therefore, 8 different types of enzymes may participate in the breakdown of various pectic substances:

1. Exo PG
2. Endo PG
3. Exo PGTE
4. Endo PGTE
5. Exo PMG
6. Endo PMG
7. Exo PMTE
8. Endo PMTE

In plant cell wall, cellulase forms an integral part. The cellulolytic process involved in the disease development consists of two independent enzymes (Reese et al., 1950). First is $C_1$ enzyme which attacks untreated cellulase fibre and second is $C_x$ enzyme which further degrades cellulase. The site of action of $C_1$ is most probably at the cross linkage between adjacent $\alpha-1,4$ glucosidic linkage of glucose anhydrous chains released by the action of $C_1$ enzyme. $C_x$ enzyme usually breaks the cellulase chain randomly. However, sometimes acts terminally on the substrate, as a result of which cellobiose is formed (a disaccharide and glucose units are released). Liu and King (1967) have proposed that in usual sense $C_1$ cannot be considered as an enzyme. It may be a protein linked with native cellulase by hydrogen bond more
tightly than cellulase hydrogen bond itself. Selby and Maitland (1967) reported that \( C_1 \) enzyme does not attack soluble cellulose such as CMC but \( C_1 \) and \( C_x \) both are needed for degradation of native cellulase.

Wood (1967) proposed a scheme for degradation of cellulase by cellulolytic enzymes:

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\begin{array}{c}
\text{NATIVE CELLULOSE} \\
\downarrow \text{C}_1 \text{ Enzyme} \\
\text{Linear Chain} \\
\downarrow \text{C}_x \text{ Enzyme} \\
\text{Cellobiose} \\
\downarrow \text{C}_x \text{ or } \beta-\text{Glucosidase} \\
\text{Glucose}
\end{array}
\]
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Pectolytic enzymes have a great phytopathological significance. Majority of fungal pathogens have been reported to produce pectic enzymes \textit{in vitro} as well as \textit{in vivo} and extensive work has been carried out on various aspects of these enzymes.
De-Bary (1886) was the first who demonstrated the secretion of protopectinase by Sclerotinia and reported a positive role of these enzymes in pathogenesis. Ward (1888) confirmed De-Bary's conclusion that enzyme takes part in tissue maceration. Brown (1915) reported the presence of extracellular pectin degrading enzyme in pathogenic fungi. He showed in Botryotis cinerea that enzyme preparation was present in both culture and in young mycelia. Jones (1909) observed that pectolytic enzymes secreted by soft rotting bacteria, were responsible for the breakdown of its infected tissue. After having a solid background a number of workers started work on these enzymes. Harter and Weiner (1921) observed 11 species of Rhizopus and found that pectinase was produced by R. tritici when grown on vegetative decoction or on media having pectin. He also reported that both the pathogenic as well as saprophytic Rhizopus species could produce this enzyme. Menon (1934), while working on Fusarium sp., Pythium debaryanum, Phytophthora erythrospextica, Glomerella cingulata and Sclerotinia sp., reported the influence of natural media on production of pectic enzymes. Waggoner and Dimond (1955) found that Fusarium oxysporum f. lycopersici produced polygalacturonase and pectin methyl esterases on pectin media. White and Fabian (1953) demonstrated that Alternaria humicola and Cladosporium isolated from black berry could not produce pectinase enzyme whereas Fusarium sp.
Pullularia sp., Botrytis cinerea and Penicillium sp. isolated from the same host showed a good deal of activity of pectolytic enzymes. The production and properties of pectic enzymes of Fusarium moniliformae and Rhizoctonia solani have been discussed by Singh and Wood (1956) and Deshpande (1961) respectively. Oxford (1944) showed the pectinase production in vivo by Pseudomonas sp. Phaff (1947) showed that Penicillium chrysogenum produced extracellular enzyme and polygalacturonase and methyl esterases were adaptive products. Elarosi (1958) reported that Rhizoctonia solani and Fusarium solani, the potato rot pathogens have synergistic relation due to their pectic enzymes. Bateman and Beer (1965) reported the secretion of polygalacturonase along with oxalic acid in the bean hypocotyl infected with Sclerotium rolfsii. Papavizas and Ayers (1966) reported the presence of polygalacturonase transeliminase enzyme in Fusarium oxysporum and F. solani. Phytophthora cinamomii could secrete PG and PME in the invaded host tissues (Hall and Zentmyer, 1967). Colonge et al. (1969) investigated PME and PG activities in appreciable amount in case of Sclerotinia fructigenia and Phytophthora palmivora.

Cellulolytic enzymes also have been found to play a significant role in some plant diseases. A great deal of information on microbial degradation of cellulase can be
obtained in the comprehensive reviews published by Siu (1951), Siu and Reese (1953), Wood (1960) and Gascoigne (1966). A claim on the inductive culture of *Sclerotium rolfsii* was made by Husain (1958), Bateman (1969) further carried out detailed study on extracellular secretion of cellulase by the same organism. Bateman (1969) demonstrated that the culture filtrate of *S. rolfsii* and water extract of lesion induced by this pathogen on bean hypocotyls contained a cellulase system (C\text{\textsubscript{x}} type) that exhibited optimum activity at 4 pH on carboxymethyl cellulase (CMC). It was considered that cellulase system has significant role in cellulose decomposition and thus in the causation of rots especially at the late stage of disease development. Shrivastava et al. (1959), Strider and Winstead (1961) and Bateman (1964) have observed the active participation of cellulase in various fruits under pathogenesis.

Production of pectolytic enzymes in culture medium is found to be influenced by a number of factors. Cole (1956) while studying the role of pectic enzymes, reported that the media which contain glucose and ammonium-tarterate gave active enzyme preparation. Singh and Wood (1956) confirmed the findings of Cole (1956) by showing that *Fusarium moniliforme* secreted macerating enzyme when certain natural extract, i.e., pectin substances or galacturonic acid were added to the liquid media.
The production of cellulase enzyme in culture media has also been demonstrated by Husain and Rich (1958), Somkuti et al. (1969), Dube and Gour (1975) and Vance et al. (1980). Different synthetic and semisynthetic media have been used for the production of cellulase by Rhizoctonia solani (Ali, 1970), Alternaria solani and Alternaria tenuis (Mehta, 1973), and Botryodiplodia theobromae (Mishra, 1978). Muthal and Saksena (1973) have also confirmed that C\text{X} enzyme was also produced in culture medium devoid of cellulose source while investigating an exo-cellular complex of Fusarium dimerum, a causal organism of Luffa aegyptiaca fruits. They reached to a conclusion that C\text{X} enzyme production was a constitutive property of the test organism. Wood (1960) reported that cellulose did not play a significant role in disease development but it certainly attacked the cellulase of the microfibrils in the later stage of soft rot diseases. Husain (1958) reported the production of cellulase enzyme by Sclerotium rolfsii in the culture media and suggested its probable role in the pathogenesis. However, cellulolytic enzyme produced in culture media by Colletotrichum homoides (Schmittemmer, 1960) and Fusarium oxysporum f. callistephe (Horst, 1965) did not show any correlation with their pathogenic potentialities. Barker and Walker (1962) observed a direct relationship of cellulolytic enzyme to the pathogenicity of Fallecuria filamentososa. Kelman and Cowling (1965) also observed a good deal of correlation
between cellulose secretion and pathogenic potentialities. There are a large number of evidences indicating the active involvement of cellulase in the infection process especially in the rot disease (Bateman, 1963a; Spalding, 1963; Muthuswami, et al., 1973; Pearson, 1974; and Rai and Dhawan, 1976).

Glucose was found to be inhibitory for the secretion of PG by Typhula idahoensis (Malanax and Huber, 1970) and Ceratocystis ulmi (Biehn and Dimond, 1970). Repression of PG enzyme in Fusarium oxysporum has been found to be caused by sugar and sugar-alcohols (Patil and Dimond, 1967). Gupta (1960-62) studied the protopectinase production by Fusarium orthoceros var. Ciceri on natural and synthetic media. Gupta (1956) reported that Glucose, Fructose and Mannose were favourable for the synthesis of pectolytic enzymes. Waggoner and Dimond (1955) have observed that the production of PG by Fusarium oxysporum f. lycopersici in culture was repressed by the presence of glucose in the medium. Similar results of PG repression by glucose were observed by Horton and Keen (1966) in case of Pyrenochaeta terrestris. It was found that the disease development was retarded and enzyme accumulation was not found when glucose level was increased by spraying infected plant with a solution of glucose (Patil and Dimond, 1968) and it was further observed that addition
of glucose to cut stem of *F. oxysporum f. lycopersici* infected tomato plant reduced the disease development and PG enzyme activity. In *E. coli*, β-galactosidase is induced by disaccharide lactose (Jacob and Monod, 1961). These disaccharides could not cause high specific effects for the induction/repression of polysaccharide-degrading enzyme synthesis by pathogen (Albersheim *et al.*, 1969).

Baldwin (1970), however, reported that *Xanthomonas malvacearum* could produce PME, PG or PGTE on any of the carbon source, viz., glycerine, pectin, sodium polypectate or galacturonic acid. The pectic substances were, however, found to be good carbon sources for the production of pectolytic enzymes by *Xanthomonas malvacearum* (Abo-El-Dohab, 1964), *Rhizopus stolonifer* (Spalding, 1963 and Trescott and Tampion, 1974), *Sclerotium rolfsii* (Bateman and Beer, 1965) and *Sclerotinia sclerotiorum* (Lumsdon and Roberta, 1970). Biehn and Dimond (1970) while working with *Ceratocystis ulmi* showed very poor production of PG in the pectin enriched media.

Bisen and Agrawal (1980) and Kamal and Wood (1956) reported that ammonium nitrogen was better than nitrate nitrogen for the production of pectolytic enzyme, by *Aspergillus niger* and *Verticillium dahlia.* Bateman (1966) worked on extracellular enzyme complex of *Fusarium solani*, f. *phaseoli*
and found that it contained a fraction of which brought about a transeliminative degradation of pectic substances.

Hancock and Millar (1965a) have done a lot of work on alfalfa pathogens, *Colletotrichum trifoli*, *Aschchyts imperfecta* and *Stemphylium botryosum* causing anthracnose, spring black stem leaf spot disease respectively. He concluded that all these organisms produce cellulase during pathogenesis. Ali (1970), Mehta (1973), Mishra (1978) and Saxena (1982) observed the effect of culture media on production of cellulase by *Rhizoctonia solani*, *Alternaria* sp. and *Botryodiplodia theobromae*.

Carbon and nitrogen sources caused considerable effects on the synthesis of cellulolytic enzyme in some fungi, viz., *Myrothecium verrucaria* (Hulme and Stranks, 1970), *Pyricularia orizae* (Hirayama et al., 1980) and *Sporocystophaga mixococccides* (Vance et al., 1980). Cellulase formation was found to be inhibited by glucose. Hosien et al. (1979) observed the effect of nitrogen source on the production of cellulase by *Fusarium moniliformae*. Husain (1958) reported the production of cellulase enzyme by *Sclerotium rolfsii* in the culture media and suggested its probable role in the pathogenesis. Baker and Walker (1962) observed that direct relationship of cellulolytic enzyme to the pathogenesis of *Pellicularia filamentosa*. Kelman and Cowling (1965) also found good deal of correlation between cellulase secretion and pathogenic
potentialities in case of *Pseudomonas solanacearum*. A large number of workers like Bateman (1963a), Muthuswami et al. (1973), Spalding (1963), Pearson (1974) and Rai and Dhawan (1976) have indicated the active involvement of cellulase in the infection process especially in rot diseases.

Pectolytic and cellulolytic enzymes are often affected by different factors like hydrogen ion concentration and temperature. Ayer et al. (1966) observed that maximum activities of PG and lyase occurred at pH 5.0 and 8.0 respectively, and they also found out that Ca$^{++}$ ion were inhibitory in the former case and stimulatory in the later case. Pectic enzymes have commonly been found to be active in the acidic conditions. The pH ranging from 3.5 to 5.0 appears to be optimum for the pectolytic enzyme produced by *Botrytis cinerea* (Tribe, 1955), and *Sclerotinia sclerotiorum* (Lumsden and Roberta, 1970). But on the other side *Bacterium aridum* (Tribe, 1955) observed maximum PG activity of pH 8.6, while PG and PMG of *Aphanomyces euteiches* were most active at pH 6.7 and 5.0 respectively.

Ayers (1966) worked on different isolates of *Fusarium oxysporum* and *F. solani*. He reported that PG enzyme which was produced by both the species of *Fusarium* was adaptive in nature and activity was high between pH 9.4 to 9.9.

Cellulase is also active in acidic range of pH. Verma and Verma (1962) observed pH 4.6 to 5.0 as the optimum pH.
value for the activity of cellulase secreted by *Curvularia lunata*. Jenson (1971) observed maximum cellulase activity between 4.0 to 5.0 in case of *Stereum gausapatum*. In case of *Alternaria solani*, A. *tenuis* (Mehta, 1973) and in case of mango isolate of *Botryodiplodia theobromae* (Mishra, 1978), optimum pH range was 4.0 to 5.0.

Temperature also plays an important role in production and activity of cell wall degrading enzymes. Temperature ranging between 30 to 40°C was optimum for the activity of pectolytic enzyme secreted by *Rhizopus stolonifer* (Trescott and Tampion, 1974).

The maximum cellulase activity was found in the range between 30 to 50°C in *Stereum gausapatum* (Jensen, 1971) and *Botryodiplodia theobromae* (Mishra, 1978). Mandel and Reese (1963) found 70°C as optimum temperature for the activity of cellulase secreted by *Trichoderma viridae*.

Association of pectic enzymes with the tissue maceration has been investigated by many workers including Sato (1968), Bateman (1968), Albersheim et al. (1969), Bateman and Millar (1966), McCledon (1964), Zaidin and Colindin (1964) and Bush and Conder (1968, 1970). The studies of last few years showed that cellulolytic enzyme have little or no significance of tissue maceration. Synergistic action of pectolytic and
cellulolytic enzyme have been given by a number of workers (Spalding, 1963; Bateman and Millar, 1966; Cole and Wood, 1970; Keen and Friend, 1968).

A number of chemical substances including fungicides, phenolics, antibiotics and plant growth regulators may also cause strong regulatory effects on the production of pectolytic and cellulolytic enzyme by fungal pathogens. The fungicides are a heterogeneous group of organic compounds which are usually unrelated chemically. They often have nothing more in common than just being fungicides, and even the mode of their action against the fungal pathogens may be quite different (Sisler and Cox, 1960). Foote et al. (1949) and Horsfall (1956) suggested that in vitro activity of such fungitoxicants for inhibitory respiratory enzymes, might be used for selecting effective fungicides. The effect of fungicides on the production and activity of various cell wall degrading enzymes have been studied by Byrde et al. (1956). MacMillan and Vauge (1964), Malcolm et al. (1969), Gupta and Prasad (1968), Pratt and McIntyre (1971) and Mehta (1973).

Amongst the various organic compounds, phenolic substances have attracted greater attention with regard to their effects on pectic enzymes. Many phenolic compounds particularly their oxidation products have been found to act as potent,
non-specific enzyme inhibitors (Byrde et al., 1959; Byrde, 1963; and Sanderson, 1965). Cole (1956) and Wood (1961) have reported that enzymes of Sclerotinia fructigena are inhibited by leucanthocyanine of high molecular weight specially when oxidised. Patil and Dimond (1969) observed that PG of Verticillium albo-astrum could be inhibited by chlorogenic acid, caffic acid and by potato phenols. Singh and Chand (1969) concluded that in culture media phenolic compounds do not seem to have appreciably inhibitory effect on the PG secretion of Gloeosporium fructigenum. Be Miller et al. (1969) have also reported that certain phenolic substances including caffic acid and ferulic acid, instead of inhibiting pectic enzymes, caused an increase in their activity. Thus, the above views clearly demonstrate that the pectic enzymes are greatly affected by various phenolic compounds.

A large number of plant growth regulators are known to control the synthesis of various enzymes in higher plants (Glasziou, 1969). Many of these plant growth regulators have also been found to be involved in several plant diseases caused by fungal pathogens (Sequeira, 1963). Albersheim (1963) had reported that endo-transeliminase could be inhibited by IAA (Indole acetic acid) but according to Bull (1968), it was an error in spectrophotometry. Influence of plant growth regulators on production of pectolytic and cellulolytic enzymes
was shown by Jermyn (1952), Vyas (1971), Mehta (1973), Mishra (1978) and Saxena (1982).

Effect of antibiotics has also been investigated in certain cases (Whiffen, 1948; Dekker, 1955; Brown and Elizabeth, 1957; Crowdy et al., 1959; Grover and Moore, 1963, Abo-El-Dohab, 1964; and Goel and Mehrotra, 1973, 1974).

The effects of fungicides, phenolics plant growth regulators and antibiotics on the production of pectolytic enzymes in vitro were studied by adding desired concentration of these substances in the basal culture (Czapek's dox) medium. The results of these experiments showing different degrees of control of pectic and cellulolytic enzymes might be due to repression of enzyme-synthesis caused by various effector substances in the culture. Though the control of enzyme activity after production by means of stimulation or inhibition of enzyme activity may also be brought about by some of these substances. But it is difficult to conclude whether the control of pectic enzyme activity was brought about by the effect on the synthesis of these enzymes or the regulatory effects were on the activity of these enzymes after their production. Thus it may be thought desirable to study the effect of all these substances in the enzyme reaction mixture instead of adding them in culture medium.

The literature on inhibition of cellulase has been
thoroughly reviewed by Mandel and Reese (1965). But many of these studies have been carried out by taking out active cellulase preparations from the culture filtrate of various fungal species and then the effects of various organic substances including unknown complex plant products on the activity of cellulase have been investigated by adding these substances in reaction mixture used for enzyme assay. Keeping this view in mind the study of effect of different substances including fungicides, phenolics, plant growth regulators and antibiotics and activity of pectolytic and cellulolytic enzymes.

A large amount of plant produce faces high degree of bio-degradation leading to the colossal spoilage which ultimately causes not only considerable loss to our national economy but also cuts short our food supply.

A significant bulk of the loss is caused by fungal diseases which takes place during storage, handling and transit. Fungi have been a real curse for man for centuries. It has received the attention of the scientists just in the last century and during the past two decades adequate knowledge and thorough understanding of the disease development from the viewpoint of host-parasite-environmental interactions have been gained and feasible control measures have also been recommended to the growers. Yet much remains to be done.
A constantly increasing number of investigations is underway to find out suitable remedies against these diseases.

This involves a thorough understanding of epidemiology, the host-parasite relationship, the annual cycling of the pathogen, and the degree of natural resistance in host plants.

In recent years plant pathologists have ingeniously developed a variety of successful methods in the eradication of plant diseases both in the fields as well as under post harvest conditions. The use of several chemotherapeutic agents like fungicides, phenolics antibiotics, volatile compounds, etc. particularly in the post-harvest control of disease have become popular to a great extent since their treatments are cheaper, less phytotoxic and highly inhibitory to pathogenic forms responsible for causing the damage to fruits and other valuable plants or their produce.

Keeping this view in mind, the present problem was undertaken to study the pathogenicity of these diseases under different conditions of temperature and age. Production of pectolytic and cellulolytic enzymes in different culture media and in different stages of pathogenesis was investigated to understand the process of cell wall degradation. Another aspect of these enzymes, i.e., their activity was also studied under different pH and temperature values. Effects of various chemical substances, viz., carbohydrates, native carbon sources,
amino acids, fungicides, phenolic compounds, growth regulators and antibiotics were also observed on the production as well as activity of these enzymes so as to give a line for future workers on control measures, as some of them may be used as strong inhibitors of the growth of pathogen and can check the disease at least in part.
HOST

Trichosanthes dioica (Parwal)

Sanskrit : Putulika
Hindi and Punjabi: Parwal
Bengali : Potol
Gujrati and Oriya: Potol
Telugu : Kommupotla
Tamil : Kombu
Kannad : Kaadupadavala
Malayalam : Potolom

A dioecious climber, with a perennial root stalk, found wild in the plains of North India from Punjab to Assam; it is also extensively cultivated all over the warmer regions of India, particularly in Madhya Pradesh, Uttar Pradesh, Bihar, West Bengal and Assam for its fruits. Leaves cordate or ovate-oblong; flower dioecious: male peduncles paired, both flowered; female solitary; fruits globose, oblong, smooth 5-12 cm x 2-6 cm not striped or striped; stripes light green on the young fruits; seeds globose.

Parthenocarpic development of fruit can be induced by spraying growth regulators like NAA (100 ppm) at the time of opening of female powers which generally occurs between 19.00 and 20.00 hrs, such are smaller and lighter, but the amount
of pulp is reported to be almost the same as in the unsprayed fruits.

This plant requires humid and hot climate. Depending on the area of cultivation, early planting is done during February-April and late planting from May to July. In northeastern states, it is grown as a summer crop. In western U.P., Delhi, Haryana and Punjab, it can be grown as a rainy season crop.

The fruits are commonly used as a vegetable in India. They are pickled and are also used in confectionery. The fruits are believed to be particularly suitable as food for convalescents. The vegetable is easily digestible and it is said to be diuretic and laxative. It is prescribed for patient suffering from the disorders of circulatory systems.

The fruit (edible matter, 95%) has the following composition: Moisture, 92.0%; Protein, 2.0%; Fat, 0.3%; Fibre, 3.0%; other carbohydrates, 2.2% and mineral matter, 0.5%. Chemically, it has calcium, 30.0; oxalic acid, 7.0; phosphorus, total, 40.0; phytin, 8.0; iron total, 1.7; ionizable, 0.5; magnesium, 9.0; sodium, 2.6; potassium 83.0; copper, 0.11; sulphur, 17.0; chlorine, 4.0; thiamine, 0.05; riboflavin, 0.06; nicotinic acid, 0.5; and vitamin C, 29.0 mg/100 g; and carotene 153 ug/100 g of edible matter. Iodine
and fluorine contents are 0.66 and 2.1 ppm on dry edible matter. A trace of 5-hydroxytryptamine has also been detected (Wealth of India, Vol. X).

The leaves are also eaten as a vegetable, and have the following composition: moisture, 80.5; protein, 5.4; fat, 1.1; fibre, 4.2; other carbohydrates, 5.8; and mineral matter, 3.0%; calcium, 53% and phosphorus 73 mg/100 g.

**Disease:**

This plant is attacked by serious disease - *F. oxysporum*. The infection by this starts in mid-region and spreads throughout the fruit. Brownish spots appear which, later on, coalesce and cover large portion of the fruit. There is no exudation on the surface of the fruit but it loses turgidity, becomes yellowish and later on brown. Internal tissue disintegrates, turn into a light brown, viscoid fluid and later on dries up, rendering the central portion hollow; the infected fruits emit bad odour.
HOST

*Solanum melongena* Linn.

<table>
<thead>
<tr>
<th>Language</th>
<th>Common Name</th>
</tr>
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<tbody>
<tr>
<td>Sanskrit</td>
<td>Vartaku vatigama</td>
</tr>
<tr>
<td>Hindi</td>
<td>Baigan, Bhata</td>
</tr>
<tr>
<td>Bengali</td>
<td>Begun, Kuli-begun</td>
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<td>Marathi</td>
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<td>Ringni, Vengni</td>
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<td>Telugu</td>
<td>Chirivanga</td>
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<tr>
<td>Tamil</td>
<td>Kathirikai</td>
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</tbody>
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A herbaceous prickly or sometimes unarmed, perennial, 0.6 - 2.4 m tall, cultivated throughout India as an annual for its edible fruits. Leaves ovate, sinuate or lobed; flower blue, in small clusters of 2-5; berries large ellipsoid or elongate, in various shades of white, yellow or dark purple, 2.5 - 25 cm long, glabrous, with thick calyx; seeds many, discoid.

It is difficult to fix the ancestry of cultivated egg plants but hybrid-vigour and continuous selection must have played an important role in the evolution and development of the various cultivated types of *S. melongena* which is adapted to a wide range of climatic conditions.

The genus *Solanum* is predominantly central and South
American and most of the species originated there, but the brinjal plant is probably a native of South Asia and even Arabia. From the study of ancient records it appears that the plant was native to India and was also first cultivated in this country; later, its cultivation spread through Iran or Egypt and other North African countries and to Turkey and the Balkans. In China, its cultivation has been known for the last 1,500 years. The origin of *S. melongena* in the Indo-Burmese region with a large number of types distributed all over the world suggested a parallel evolution of the various types of cultivated egg plants (*Wealth of India, Vol. 19*).

There are four main botanical varieties: (i) *incanum* syn. *S. incanum*, *S. coagulans*; (ii) var. *melongena* syn. *S. melongena* var. *esculenta*; (iii) var. *depressum*; and (iv) var. *serpentinum* syn. *S. serpentinum*. Of these, var. *incanum* bears bitter, usually non-edible fruit but the other three varieties and their hybrids are cultivated and bear different types of edible fruits.

*S. melongena* var. *incanum* is a medium-sized, prickly, perennial shrub with blue flowers and yellow ovoid or globose berries var. *melongena* includes the common eggplant with large, pendent, ovoid, oblong or ovoidal berries, 5.30 cm long shining, purple, white yellowish or striped.
Commercial types of eggplant are based on the colour and shape of fruits which may be oval, globose, pear-shaped or even cylindrical. As already mentioned, the colour of fruits ranges from white to deep purple or almost black and there are regional preference for colour. Purple brinjal are most popular in the northern parts of the country, while the long and green types are preferred in Bihar and Mysore and the round and green in Orissa.

Brinjal is a warm season crop and is very susceptible to frost, round types being less susceptible to frost than the long ones. It is grown almost throughout the year in the plains but on the hills only during summer, the crop extends up to September. Deep, fine, rich loam (pH 6) with proper drainage is most suitable. In clayey soil, the plant remains stunted and bears small fruits. The growth is luxurient in soils rich in organic matter and the plant bears more fruits.

Besides being esteemed as a vegetable, brinjals are consumed in variety of ways. The value of brinjal is enhanced as a vegetable during autumn when other vegetables are scarce. They are eaten when approaching ripeness and are a fairly good source of calcium, phosphorus, iron and Vitamin B.

Roots of brinjal plants are credited in the indigenous medicine as antiasthmatic and general stimulant. In Guiana, their juice is employed to cure otitis and toothache. Roots
are pounded and applied to ulcers in the nose.

Leaves are said to possess sialagogue and narcotic properties and are used in cholera, bronchitis, dysuria and asthma. Brinjal are recommended in liver complaints.

The seeds are used as a stimulant but are apt to lead to dyspepsia and constipation.

Brinjal is recorded to stimulate the intrahepatic metabolism of cholestrol. Both leaf and fruit, fresh are dry produce a marked drop in blood cholestrol level. The decholesterolizing action is attributed to the presence of magnesium and potassium salts in the plant tissues. Experimental results, however, have not been confirmed by clinical trials. Aqueous extracts of fruits inhibit choline esterase activity of human plasma. Extracts of the plant inhibit the growth of several types of bacteria; the pulp of the fruit is more effective than the juice. Dried fruit is reported to contain a goitrogenic principle.

Chemical composition of the edible portion of fruits (all except stalk and calyx) gave the following values: moisture, 92.7; protein, 1.4; fat, 0.3; minerals, 0.3; fibre, 1.3; and other carbohydrates, 4.0 g/100 g. The mineral constituents present are (mg/100 g edible matter), Ca, 18; Mg, 16; P, 47; (Phytin P, 3); Fe, 0.9 (ionisable Fe, 0.8); Na, 3; K, 200; Cu, 0.17; S, 44; and Cl, 52. Small quantities
of magnesium (2.4 mg/100 g) and iodine (7 µg/kg) are reported to be present. The vitamins present are vitamin A, 124 IU; thiamine, 0.04 mg; riboflavin, 0.1 mg; nicotinic acid, 0.9 mg; vitamin C, 12 mg; and choline, 52 mg/100 g of edible matter.

Brinjal contains 14.19% protein (dry wt. basis) of high biological value (71%) and digestibility coefficient (75%). It contains the following essential amino acids (g/g of N); arginine, 0.21; histidine, 0.11; lysine, 0.10; tryptophane, 0.06; phenylalanine, 0.27; methionine, 0.06; threonine, 0.23; leucine, 0.36; isoleucine, 0.32; and valine, 0.37. The sugars present in brinjal are sucrose, glucose and fructose (Wealth of India, Vol. IX).

Diseases of Brinjal:

About 20 diseases caused by various fungi have been reported to damage the crop. In several cases, the fungal infection is carried through seed. A serious leaf blight and fruit rot is caused by *Phomopsis vaxans* in Western India. Once established, the disease causes complete rotting of fruit. Damping-off of the seedling is caused in nurseries by *Rhizoctonia solani*. A blight caused by *Myrothecium roridum* is serious disease of brinjal crop in Orissa. *Phytophthora parasitica, P. palmivora* and *P. coloacasiae* cause brown water soaked patches on stem and fruit. Leaf spot of brinjal caused
by *Alternaria tenuis* and *A. melongena* and *Cercospora* spp. Other important disease of brinjal are root and foot rot (*Rhizoctonia solani* and *Fusarium* spp.) and sclerotial disease (*Sclerotium* and *Pellicularia* spp.).

The round green coloured brinjal fruit is attached by *F. moniliformae*. The infection by this pathogen starts from any region and covers the entire surface and for complete rotting it takes about 8 to 90 days. Fruit loses turgidity, becomes brown, and later becomes dark brown. Internal tissue disintegrates, turns dark brown and whole fruit becomes very pulpy.
PATHOGENS

Fusarium oxysporum:

This fungus has thick mycelium, unseptate, branched, measuring 1.2 to 2.3 μ diameter. Conidiophores flask-shaped, conidia slightly curved in the middle measuring 0.8 to 1.2 x 11.4 μ diameter; mycelium is vinaceous buff on the surface and livid vinaceous on the reverse. In the Petri plate, the isolates grow to 60 mm in diameter within 5 days. The species has been confirmed by Dr. C. Booth of the CMI, Kew, England (Letter No. H 825/81/ Y12 dated Nov. 4th, 1981).

Fusarium moniliforme:

This fungus has thin mycelium, unseptate branched, measuring 1.8 to 1 μ diameter. Conidia slightly elongated than that of F. oxysporum, conidia 0.5 - 2 x 0.8 - 3.1 μ diameter. Mycelium is rosy vinaceous on the surface and honey colour on the reverse. Inside the Petri plates the fungus grows to 90 mm in diameter within 12 days. The species has been confirmed by Dr. C. Booth of the CMI, Kew, England (Letter No. H 825/81/Y12 dated Nov. 4th, 1981).