REVIEW OF LITERATURE
HISTORY AND DISTRIBUTION OF THE DISEASE

Bacterial leaf spot due to *Xanthomonas vesicatoria* (Doudge) Dowson was first observed in the USA on chilies in 1912 and later on tomato in 1917 (Gardner and Kendrick, 1921). The disease is now known to occur in all countries of Asia. Some early country reports are Pakistan (Akhtar et al., 1985), Brazil (Reifsneider et al., 1985), Italy (Buonamico and Stravato, 1992) and Indonesia (Vos and Broekhuysen, 1998). In India, the disease was said to have been noticed for the first time on chilies at the Agricultural college farm, Poona in August, 1948. The bacterial organism was identical with *Xanthomonas vesicatoria* causing bacterial leaf spot of pepper and fruit canker of tomato in Georgia and Indiana respectively. It is inferred that the disease was introduced into India along with imported seeds of chilies and tomato from America where it incites a severe disease on chilies and tomato (Patel et al., 1950).

2.1 Symptomatology of the Disease:

The disease occurs in four fairly distinct phases (Nayudu and Walker, 1960) as water soaked lesions which gradually enlarge and extend from lower to upper leaf surfaces. Lesions then turn brown in the centre, with a narrow pale yellow halo. The leaf tissue between the lesions becomes chlorotic and leaves show decided epinasty. Infected leaves turn brown, become dry, leathery and drop off.
Symptoms of the disease can be found on all apical parts of the chilli plant including cotyledons, leaves, leaf petioles, young stems and fruit stalks (Shekhawat and Chakravarti, 1977) Buonanno et al., (1999) reported that a bacterial leaf spot is also caused by Pseudomonas syringae pv. syringae and also by Xanthomonas campestris pv. vesicatoria

2.2. Nomenclature and classification of the pathogen

Prior to 1976, fluorescent Pseudomonads causing lesions on leaves and Xanthomonads constituted the largest groups of named species of phytopathogenic bacteria. Over 90% of Xanthomonads included of which 120 species were grouped as pathovars with the type species Xanthomonas campestris. Since 1980, there have been few pathovars that have been elevated to the rank of species. Recent proposals elevate several pathovars of Xanthomonas to species rank and to rename them (Gardan et al., 1992, Vauterin et al., 1993 and 1995) Based on the DNA similarity groups, Vauterin et al., (1995) proposed a major reclassification scheme, which include DNA similarity data on over 180 Xanthomonad strains with almost 800 pairwise comparison. Their results showed that the type species Xanthomonas campestris pv. campestris has DNA similarity values of only 26% or less with over 20 named pathovars, of which vesicatoria is also included
Taxonomic change in 1976 (Dye et al., 1980, Sneath et al., 1992, Hayward 1993) proposed naming *Xanthomonas vesicatoria* (Dodge) Dowson as *Xanthomonas campestris pv vesicatoria* V濑sa et al., (1995) more recently proposed changing the name to *Xanthomonas axonopodis pv vesicatoria* from *X c pv vesicatoria*.

*X vesicatoria* strains from pepper fields were identified based on morphology, growth on Yeast Dextrose Calcium carbonate Agar medium, biochemical and nutritional tests, hypersensitivity reaction on tobacco, FAME (Fatty Acid Methyl Ester) analysis and pathogenicity on capsicum and tomato. They also were characterized on the criteria of reaction on capsicum and tomato Xcv race differential lines, ability to degrade starch and pectin, composition of fatty acids, reaction to a panel of monoclonal antibodies (MAB’s XVI, XV5, XV6, XV8, XV10, XV15, XV21, XV30) and sensitivity to copper and streptomycin, respectively. Of the strains tested, one strain isolated from capsicum didn’t cause a hypersensitive response on any of the three capsicum race differential lines and it was designated as race 6. This strain was highly aggressive on the *X c pv vesicatoria* susceptible variety and was pathogenic on tomato cultivars. This race of Xcv is capable of overcoming all three of the known sources of resistance in capsicum to this pathogen. The existence of genetically distinct subgroups leads to the conclusion that genetic variation may already occur in the strains of *X vesicatoria* in different geographic areas (Chung et al., 1996).
Gonclaves and Rosato (1997) proposed that of *X. c. pv. vesicatoria* should be classified into eight groups using Cook and Stall's, method for classification. They classified isolates based on pathogenicity on capsicum, hypersensitivity on tobacco, auxotrophy, growth curve and extracellular enzyme (amylase, cellulase, pectinase, polygalacturonase and protease) production into five groups of which Group I is non-pathogenic on both compatible hosts (tomato and capsicum), Group II presented auxotrophy for a component of yeast extract and was white in colour, Group III did not produce pectinase, Group IV was pathogenic only on capsicum and Group V were similar to Group IV but lacked pectinase activity.

RAPD was more sensitive than ribotyping, allowing for the detection of differences among very similar isolates and generating "fingerprints" that are useful for strain typing and disease diagnosis (Ferreira et al., 1997).

Sun et al., (1999) was first researcher in India to identify races using the internationally accepted differential host plants of the nineteen bacterial strains isolated from pepper (*Capsicum annuum*) and tomato, these strains were identified as Xcv PT race 1 and the other 16 isolates, with a wide distribution, belonged to Xcv race 3.

According to recent surveys, race 6 has become the most predominant race. Although the pathogen is highly mutable and many bacterial spot races are being discovered, genetic resistance continues to be very important and offers significant promise in combating this disease (Subramanya, 1999).
Jones et al. (2000) reported that within the group of Xanthomonads pathogenic on pepper and tomato, four distinct phenotypic groups exist of which three form distinct genomic species. These include 1 X a pv vesicatoria (A and C group) On the basis of phenotypic and genotypic differences between the A and C group strains, the C strains should by considered as a subspecies within X a pv vesicatoria, 2 Xanthomonas vesicatoria (B group), 3 Xanthomonas gardneri (D group) Y c pv vesicatoria is highly mutable and there are presently 11 races (0 to 10) of the pathogen that have been described However, the distribution of most of the newly described races (1 to 11) has not yet been determined

2.3. Survival of the pathogen.

Krupka and Crossan (1956) reported that the pathogen could survive at least nine months in infected leaf tissue on or in the soil This was confirmed by Lewis and Brown (1961), who postulated that the outbreaks might be related to the survival of the organism within tissues of surface sterilized seeds

According to Peterson (1963), the bacterium survived in the woody and leaf tissue but did not survive in soil alone for more than two weeks It overwintered in dead tomato stems

Crossan and Moichat (1964) recovered the pathogen from the vascular and cortical tissues of the secondary roots, tap root, lower and upper stem, peduncles, ovarian tissues, and seeds. The infected tissues were not necessarily discoloured.
Jones et al. (1986) recovered the pathogen from infected crop residue 6 months after placing it in the field in Dec 1981 and 1982 in Florida. When diseased tissue from spring crops (January-May) was placed in the field in May and June, the pathogen was recovered after three months and 6 weeks respectively. The pathogen survived on crop residue in the soil, on volunteer tomato plants and on tomato and pepper (Capsicum) seed.

2.4. Influence of environment on disease development:

The disease occurs in different climatic and geographical areas of the world. Temperature plays an important role. High humidity provides a condition for the bacterium to exude from diseased tissues. Though rainfall and number of rainy days have no direct influence on lesion development, it aids in dispersion and invasion by the pathogen through flooding of nurseries and chilli crop fields. In addition, rainfall causes a lower temperature as a result of reduced sunshine, thus furthering infection in warm places. Typhoons, accompanied by heavy rains, cause faster bacterial spread and hence severity of the disease. Variations in yearly rainfall pattern considerably have a significant influence on the incidence and spread of the disease. Davis and Helmos (1958) reported that the longer the plants were kept at 100% R.H. prior to inoculation, the greater was their susceptibility.
Nayudu and Walker (1960) reported that the disease developed more rapidly at a continuous 24°C, the temperature favouring good vegetative growth of the host plant. Night temperatures of 24°C and 28°C, regardless of day temperatures, were found to be favourable for disease development. A night temperature of 16°C suppressed the disease regardless of day temperature.

Morton (1966) revealed that the symptoms were maximum at 22.5-25°C on capsicum and 25-27°C on tomato excised leaves suspended in a constant temperature bath. Incubation of similar leaves under variable conditions in the glasshouse resulted in a more rapid appearance and more severe symptoms. Higher relative humidity before inoculation results in greater susceptibility.

Oragvelidze (1976) reported that the optimum condition for growth of the pathogen was 30°C, 100% relative humidity and pH 7.0 to 7.5.

Shekhawat and Chakiavetti (1976) reported that plants up to 45-50 days old were more susceptible to the pathogen in pot culture tests. They found that maximum disease development in the field occurred in July, August and September months when there was a R.H. of 96% and temperatures ranging from 22-24°C. Spattering rains are the chief means of local dissemination of the bacterium from the ooze developing on affected plant parts. After heavy rains, when plants are soaked and infected with the pathogen, the disease becomes prominent.
Disease severity was higher in pot experiments than in the field (Akhtar et al., 1986). Long periods of high RH caused high Capsicum annuum yield losses due to infection by \( Y \) \( c \) \( pv \) vesicatoria in Bhat, India (Sinha and Sinha, 1991). Carro et al., (1996) reported that severe disease can prevail under favourable environmental conditions even with low initial inoculum.

2.5. Host species specificity:

Plant species specificity is considered one of the two types of pathogen specificity to determine the host range, the other being cultivar specificity which determines the cultivar range within a given host species.

Dye (1964) isolated a bacterium from tomato fruit lesions in various areas and from leaf spots on Nicandra physaloides growing as a weed in an Auckland tomato crop which was identified as the pathogen.

Laub and Stall (1967) suspected solanaceous weeds such as night shade (Solanum nigrum) and ground Cherry (Physalis minima) were hosts of the pathogen in Florida.

Raj and Moniz (1975) reported that the pathogen isolated from Datura innoxia with leaf spot symptoms was Xanthomonas vesicatoria in India. The pathogen was recorded on Physalis peruviana in India causing a bacterial leaf spot (Kishun et al., 1977).
Sohl and Sohl (1979) reported *Argemone mexicana* and *Tinospora cordifolia* (Wild) Miers as new hosts which were probably playing an important role in infected chili plants on the Hessaraghatta farm, Bangalore.

2.6. Disease physiology:

2.6.a. Physiology of the pathogen

Xanthomonads constitute an economic group amongst the phytopathogenic bacteria. Several workers have studied different *Xanthomonas* species for their nutritional requirements with respect to carbon (Starr, 1946; Fang et al., 1957, Magasanik, 1957, Watanabe, 1963b, 1966, Kotasthane et al., 1965, Reddy et al., 1979) nitrogen (Patel et al., 1951, Nayudu and Walker, 1961a, Nayudu, 1963, Kotasthane et al., 1965, Reddy et al., 1972, Mista and Thapilyal, 1977, Krishna rao and Nayudu, 1978b) and vitamin (Watanabe, 1966) sources. Bruggeman et al., (1998) noted that the bacterium preferentially consumes glucose and fructose formed during sterilization of the medium but not sucrose, while the osmolality from unhydrolysed sucrose assists xanthan biosynthesis.

A number of plant pathogenic bacteria are known to produce protease in culture filtrates. Shekhawat and Sivacisava (1968) reported that *X. c. pv oryzae* produces proteolytic enzymes in culture. Some pathogens have been shown to produce protease during pathogenesis (Reddy et al., 1971)
2.6.b. Physiology of the host-pathogen interaction:

The survey of literature indicates that there are very few studies on the physiology of *Xanthomonas* species infected plants (Patel and Walker, 1963, Easwaran, 1973, Padmanabhan *et al.*, 1974, Verma and Singh, 1974, Marimuthu and Kandaswamy, 1980, 1983, Nema, 1989, 1991). Very few studies have been conducted on the physiology of *X. a pv. vesicatoria* infected chilli plants. Metabolic alterations in the host plant take place after invasion of the pathogen into the host tissue. Infection of plant tissues immediately evokes a complex array of physiological and biochemical responses in cells adjacent to the disrupted cell layer. Cell membranes, cytosol and cell wall are the three main parts of the host plant affected during host-pathogen interactions.

Generation of ethylene, increase in reactive oxygen species like catalase and peroxidase, induction of protease inhibitors, chitinase, glucanase, acid peroxidases, and defense related enzymes like polyphenoloxidases and phenylalanine ammonia lyase are some of the changes that take place in the cytosol of infected tissue. Gabriel and Pirone (1967) studied a causal relationship between permeability changes and the typical wilting of Tabasco pepper plants (*Capsicum frutescens* L) infected with tobacco etch virus. Cook and Stall (1968) reported that maximum loss of electrolytes occurred within a few hours after the bacterial population in the leaves became sufficient to induce symptoms.
Infection accelerates the activation of some enzymes. Regarding enhanced consumption of carbohydrates, enzymes like amylases are found to be activated in diseased tissues (Tanaka and Akai, 1962). Starch accumulation is directly associated with the regulation of ADP-glucose phosphorylase, an enzyme active in diseased tissues, by a change in concentrations during the infection process (Urtnami, 1971).

Buonanno and Kumar (1995) carried out studies on the active oxygen producing and scavenging enzymes like lipoxygenases, peroxidases, superoxide dismutase, and catalase activities in pepper leaves infected with the pathogen and reported that the changes observed, uncontrolled production of active oxygen species, is hypothesized at advanced stages of infection.

Cellulase encoding genes have been cloned from a number of genera of phytopathogenic bacteria including Xanthomonas and Pseudomonas (Daniels et al., 1987). The endo-1,4-glucanases of cellulases of higher plants are cell wall-associated enzymes believed to function in cell wall changes associated with the diverse process of organ abscission, and cell elongation (Harpster et al., 1997). Protease may play a major role in disease progression. Increased levels of protease production was evident during pathogenesis in the fruit of chillies infected by Colletotrichum capsici (Hilda et al., 1996).
Sammanne et al. (1991) reported trans-cinnamate, 4-hyroxylase activities doubled in Phytophthora capsici resistant C. annuum cultivars and susceptible cultivars following infection. Fleuriel and Macheix, (1991) reported that the origin and causes of browning (effect of diseases) is due to the enzymatic (peroxidases and PPO's) oxidation and phenolic compounds involved in the browning reaction. Solorzano et al. (1996) confirmed the relationship that exists between the enzymes and resistant mechanisms in tomato plants. The inverse relationship between the evolution of capsaicinoids (produced by cinnamic acid pathway) and peroxidase activity might indicate that this enzyme is involved in capsaicinoid degradation in fruits of chillies (Contreras Padilla and Yahia, 1998).

Farkas and Kiraly (1962) reported that quinones and other oxidized phenols inhibit microorganisms and their enzymes. These reactions are correlated with defense mechanism of infected plants. Peroxidase catalyzes, the oxidation of a number of phenols (Thompson, 1964). It's activity is more noticeably increased in diseased tissues (Jennings et al., 1969). Oku (1964) and Reddy and Sundhar (1975b) reported that the lesions by X. translucens f. sp. oryzicola are thought to be due to formation of melanoid pigments.

2.7 Phytoalexins:

Despite numerous reports on the formation of phytoalexins and their biosynthesis in many host-pathogen combinations (Ingham, 1972; Purkayastha,
1973), little attention has been paid to this aspect in chillies. The phytoalexins are non-specific and it was distinguishable in resistant cultivars but not in susceptible ones (Uehara, 1962)

The hypersensitive and phytoalexin compound, capsidiol, plays a role in the mode of action of fosetyl-Al (Aluminum tris (o-ethyl phosphonate)) protection of C. annuum fruit segments from Phytophthora nicotianaec var parasitica decay (Guest, 1984)

Chawla et al., (1992) reported that culture filtrates of Colletotrichum capsici elicited production of the phytoalexin, capsidiol, in chilli fruits. Elicitor activity was destroyed when the partially purified preparation was treated with sodium-m-periodate but remained unaffected when subjected to proteinase, RNase and DNase, suggesting a polysaccharide nature.

Browning characteristic of the hypersensitive reaction and phytoalexin accumulation were induced by arachidonic acid in potato cv. Kennebec tubers. Phytoalexin accumulation was not affected by phenylthiourea (an inhibitor of PPO's that inhibits browning of tissue in old plants but not in young plants) indicating that this aspect of the hypersensitive response is not necessarily related to the browning of tissue. (Castoria et al., 1995)

Romeio and Kimura (1997) reported that cell components like lipopolysaccharide (LPS), exopolysaccharide (EPS), capsular glycoproteins and
the cell envelope extracted and purified from cells of compatible and
incompatible isolates of the pathogen, when infiltrated into leaves of *Capsicum*,
all induced protection against a compatible isolate but only EPS and the cell
envelope elicited phytoalexins. They concluded that the resistance mechanisms
other than phytoalexins may exist in *Capsicum* leaves and that these appear to be
more effective protectants than phytoalexins.

Studies of Garcia and Gómez (1999) on elicitation of phytoalexins in
pepper (*Capsicum annuum*) seedlings with arachidonic acid resulted in different
defense responses. Transcriptional stimulation of the pepper phytoalexin
biosynthesis related gene 5-epi aristolochene synthase was demonstrated and a
partial cDNA of the 1-aminocyclopropane-1-carboxylate (ACC) oxidase gene
(CA-ACCO) was also isolated. Southern blot analysis revealed three to four
members of the CA-ACCO gene family.

2.8 Toxins:

Infection results in the inability to regulate plant metabolism. Toxic
compounds produced by some pathogens that bring on death of invaded cells as
well as surrounding cells are related either to specific or non-specific
pathogenicity. Physiological changes in the infected host could be due to
pathogen-produced toxic metabolites. These toxins have been characterized as
host specific and host non-specific toxins (Patil, 1974; Rudolph, 1976, Yoder,
1980; Mitchell and Durbin, 1981)
The vivotoxin concept has not been wholly accepted (Graniti, 1972) High molecular weight polysaccharides were reported to be toxins (Angadi, 1978).

Scheffer and Pringle (1961) and Yoder (1980) divided the substances produced by the pathogen and involved in disease as primary determinants – essential for pathogenicity or pathogenicity factor(s) These are largely the "host specific" plant toxins but may also be non-specific plant toxins. Loss of ability to produce these toxins leads to loss of pathogenicity Secondary determinants not required for pathogenicity or virulence can contribute to virulence or pathogenicity.

Phytotoxins, microbial toxins and animal toxins are the three groups of toxins classified based on their origin. Based on its specificity in toxic action, phytotoxins are broadly classified into host-specific and non-specific phytotoxins. Host specific toxins are toxic to their respective susceptible host and have little or no effect on non-host or resistant plants (Pringle and Scheffer, 1964). Most of these are low molecular weight compounds produced by plant pathogens and are known to be involved in about fourteen plant diseases (Glechust and Grogen, 1976, Kohimoto et al., 1979, Scheffer and Yoder, 1972, Yoder, 1980). Production of non-specific toxins, phytotoxic to hosts as well as non-hosts of the pathogen, is not necessarily related to virulence of pathogenic isolates (Pegg, 1981). Most of the 150 characterized fungal and bacterial phytotoxins affect respiration, membrane permeability and cell metabolism at different concentrations in a non-specific way.
More specifically an individual phytotoxin may cause a symptom such as chlorosis or wilting, affecting chlorophyll synthesis or water conduction, respectively.

**Bacterial Phytotoxins**

Water soaking, wilting, chlorosis and necrosis are suggestive of the involvement of toxin(s). Toxins produced by plant pathogenic bacteria which result in these symptoms are described below.

**a. Water soaking toxins**

"Water soaking" is a general symptom of many bacterial and fungal diseases. Involvement of toxins in inducing water soaking was suggested by Strobel (1977) Water soaking is necessary for bacterial movement through intercellular spaces (Keen and Williams, 1971) or for bacterial multiplication and not a secondary symptom (El-Banoby and Rudolph, 1981). Keen and Williams (1971) showed that lipomucopolysaccharide (LMP) from culture filtrates of *Pseudomonas syringae pv lachrymans* induced water soaking at 5000 ppm when infiltrated into cucumber leaves. The LMP forms a complex with proteins to render the plasma membrane more permeable to water. Chowdhury and Verma (1980) found that the EPS from *X c pv malvacearum* produced persistent water soaking in leaves of susceptible cotton plants.
b. Chlorosis-inducing toxins

Chlorosis inducing bacterial phytotoxins are generally low molecular weight amino acids or peptide derivatives which act by inhibiting specific host enzymes (Patil, 1974) *Pseudomonas syringae pv tabaci* the casual organism of the wild fire disease of tobacco (with a necrotic spot surrounded by a yellow halo as its characteristic symptom) produces wildfire toxin which induces chlorosis non-specifically in its host and non-host plants by inhibiting glutamine synthetase and releasing ammonia. Accumulation of ammonia was responsible for the chlorotic symptoms (Turner, 1981).

Crosthwaite and Sheen (1979) reported that *P. syringae pv tabaci* toxin inhibited photosynthetic CO₂ fixation by inhibiting ribulose 1,5-bisphosphate carboxylase activity of fraction I proteins of tobacco cultivars but did not inhibit peroxidase or PPO activities. *Pseudomonas sp*, causal agent of chlorotic leaf spot of timothy (*Phleum pratense*), produces a toxin with two identical analogs that are in the toxin of *P. syringae pv tabaci* (Taylor and Durbin, 1973).

Phaseotoxin (Patil, 1974) and phaseolotoxin (Mitchell, 1976) are the two toxins produced by *P. syringae pv phaseolicola* which induce chlorosis. In addition to these, an analog of 2-seine phaseolotoxin was also identified (Mitchell and Parsons, 1977).
Mitchell (1982) reported that *P. s. pv. atropurpurea, P. s. pv. glycinea, P. s. pv. maculicola* and *P. s. pv. moriyrinorum* produce a chlorosis inducing coronatine, a tricyclic molecule containing an unusual cyclic amino acid (Ichihara *et al.*, 1977). Glycotoxin produced by *P. s. pv. glycinea* is a chlorosis inducing toxin and was subsequently established to be identical to coronatine (Mitchell and Yang, 1978).


c. *Necrosis inducing toxins*.

Syringomycin (SR) and syringotoxins (ST) are two wide spectrum antibiotics and phytotoxins produced by *P. s. pv. syringae* that cause necrosis of plant tissues (Gross *et al.*, 1977, Gonzalez *et al.*, 1981).

Ricketsia like bacteria which cause Pierce's disease of grapevines was reported to produce phytotoxins in culture which induced scalding and necrosis of leaf margins (Lee *et al.*, 1982).

**Xanthomonas spp. Toxins:**

**Xanthomonas spp** produce high molecular weight polysaccharides (Xanthan) associated with the extracellular slime. Some of these compounds have been identified as toxins (Misaki *et al.*, 1962; Sutton and Williams, 1970; Patil,

*X campestris* (Sutton and Williams, 1970), *X oryzae* (Misaki et al, 1962, Egawa et al, 1968, Kuo et al, 1970, Fan and Kuo, 1972; Purushothaman and Prasad, 1972), *X phaseoli* (Corey and Starr, 1957, Leach et al, 1957) and *X vesicatoria* (Cook and Stall, 1969) were a few phytopathogenic species of *Xanthomonas* producing phytotoxins which result in wilting and necroses. The volatile products produced by *X c pv vesicatoria* might be associated with the necrotic effect.

2.9. Disease management:

Seed is the most important source of primary infection.

Application of tribasic copper sulphate or Bordeaux mixture gave good disease control (Crossan and Morehart, 1964) Spraying with Bordeaux mixture (3-3-50), Dithane Z-78 (0.1%) and streptomycin (200 ppm) at 15 day intervals between July and August was reported to give good control of the disease in chilli (according to Nafde and Patel as quoted by Rangaswami and Rajagopalan, 1973).

Control of *X. campestris pv vesicatoria* was best when manzeh and zinc sulphate were added to a copper hydroxide spray applied at low spray pressure (2.1 kg/cm²) twice a week (Kucharek et al, 1986).
Jones and Kelley, (1995) reported that copper-mancozeb and gentian violet reduced disease severity but no yield differences were observed in the pepper or tomato trials with any of the treatments.

Applications of copper hydroxide and mancozeb significantly reduced the epiphytic population of *X. campestris pv. vesicatoria* on pepper leaves and slowed the spread of the disease (Bernal and Berger, 1996).

Campbell *et al.*, (1997) reported Kocide 2000 (copper hydroxide) and Mankocide (copper hydroxide) as two novel chemicals which gave good control of the bacterial leaf spot disease of pepper (*Capsicum*) and tomato caused by *X. axonopodis pv. vesicatoria* (*X. vesicatoria*).

Antimicrobial metabolite production was tested by determining the antagonistic activity of *B. subtilis* against *X. vesicatoria*. Bacteria of the genus *Bacillus* have been among the most outstanding microorganisms for the production of antibiotics and have been used for control of many plant pathogens (Tejeda *et al.*, 1998).

Park *et al.*, (1999) suggested that the integration of microbial antagonists and fungicides may be an efficient way to reduce fungicide sprays whilst maintaining reliable disease controls.

The current status of biological control of plant diseases is related to the use of microorganisms and agriculture could not have begun without the benefit of naturally occurring microbes. In crop crops, modern biological control is mainly aimed at the introduction of microorganisms.
Scope of the present investigation:

The present study includes investigations on various aspects of \( X \ a \ pv \ vesicatoria \), the causal agent of leaf spot disease of chillies. Since its very first report at the Agricultural college farm, Poona in 1948, this disease has become a serious problem in different parts of India. It is likely to pose an additional threat to chilli cultivation in South Indian states like Andhra Pradesh, Tamil Nadu and Karnataka, where chilli is the primary commercial crop. The present investigation was undertaken because of the potential economic importance of bacterial leaf spot of chilli.

An overall survey of the literature revealed that there are few studies on the pathogen \( X \ a \ pv \ vesicatoria \) and the physiology of infected leaves. Although there are some reports on the physiology of the host-pathogen interaction, very little was found to explain the mechanism of symptom expression. The present investigation was undertaken to study the pathophysiology of \( X \ a \ pv \ vesicatoria \) infected chilli plants.

The main objective of the present study was to investigate mechanisms of symptom production that might provide insight into the host-parasite interaction.
Objectives:

1. To study the nutritional and biochemical characters of the bacterial pathogen, *Xanthomonas axonopodis pv vesicatoria*, the incitant of leaf spot disease of chillies

2. To determine the host-range survival of the pathogen

3. To study the physiological and biochemical alternations in the pathogen and in the host during disease development

4. To study the gene products (PR-proteins) expressed during host-pathogen interaction

5. To find out the effective anti-bacterial controls (chemicals and botanicals) of bacterial leaf spot disease caused by *X a pv vesicatoria*