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Research Article

SEED-BORNE BACTERIAL DISEASES OF TOMATO (*LYCOPERSICON ESCULENTUM* MILL.) AND THEIR CONTROL MEASURES: A REVIEW

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ABSTRACT
Tomato *(Lycopersicon esculentum* Mill.) belongs to family Solanaceae. In this article vast information is collected regarding various types of bacterial diseases associated with the plants and how to cure and manage these diseases. Main diseases undertaken for the study are Canker, Leaf Spot and Wilt.

INTRODUCTION

The tomato crop suffers from a large number of diseases caused by fungi, viruses, mycoplasma, nematode and bacteria. Here a brief account of bacterial diseases given below:

Bacterial diseases

The symptoms of common bacterial diseases and their possible pathogens are: wilting of leaves (*Clavibacter michiganensis* sub sp. *michiganensis, Pseudomonas corrugata, P. solanacearum*), wilting of whole plant *(P. solanacearum, P. corrugata),* irregular yellow-brown necrotic areas on leaves (*C. m.* sub sp. *michiganensis, Xanthomonas campestris* pv. *vesicatoria),* scorching of leaves, cankering and yellow discoloration of vascular system (*C. m.* subsp. *michiganensis, X. c.* pv. *vesicatoria),* brown discolouration of vascular system and production of adventitious roots (*P. solanacearum, P. corrugata),* pith necrosis (*P. corrugata*), marbling of young green fruits (*C. m.* sub sp. *michiganensis*), spotting of fruits (*X. c*. pv. *vesicatoria, C. m* sub sp. *michiganensis, P. syringae* pv. *tomato*), leaf and stem spotting (*X. c.* pv. *vesicatoria, P. syringae* pv. *tomato, P. chichorii, P. marginalis, P. viridiflava)* and stem necrosis (*P. marginalis, P. viridiflava).*

Some important bacterial diseases of tomato are: bacterial canker (*Clavibacter michiganensis* sub sp. *michiganensis* (Smith) Davis *et al., Xanthomonas campestris* pv. *vesicatoria)* and bacterial wilt (*Pseudomonas solanacearum* Smith) (Singh, 1999).

Many of the above mentioned and other bacteria have been reported to be seed- borne (Bradbury, 1986). These are: *Agrobacterium rhizogens, A. rubi, A. tumifaciens, Aplanobacter dissimutans, Bacillus fructodestruens, B. leguminiperdus, B. lycopersici, B. tubifex, B. lycopersici vitiati, Erwinia carotovora* subsp. *atroseptica, E. c.* subsp. *carotovora, E. chrysanthemi* pv. *dianthicola, E. c.* pv. *zeae, E. rhapontici, Pseudomonas briosii, P. cepacia, P. cichorii, P. hemmiana, P. lycopersici, P. marginalis* pv. *marginalis, P. syringae* pv. *atrofaciens, P. s.* pv. *gracae, P. s.* pv. *japonica, P. s.* pv. *mellea, P. s*. pv. *savastanoi, P. s*. pv. *syringae, P. s.* pv. *tabaci, P. viridiflava, P. viridilivida, Rhodococcus fascians, Xanthomonas campestris* pv. *physalidicola* and *X. c.* pv. *raphani.* Richardson (1990) listed various diseases namely: bacterial canker, grand rapid disease (*Clavibacter michiganensis*), ring rot of potato (*C. michiganensis* ssp. *sepedonicus*), fruit spot (*P. gardeneri*), wilt (*Pseudomonas solanacearum*), speak, bacterial leaf spot (*P. syringae* pv. *tomato*) and bacterial spot and black spot (*X. campestris* pv. *vesicatoria*) to be seed-borne.

Bacterial Canker: The disease was first described by Erwin F. Smith on tomato growing in greenhouses at Grand Rapids, Michigan and called it Grand Rapids disease but later named it bacterial canker. After 1926 it was reported from New York, New Jersey to California and in several southern states. It caused losses in Canada, most European countries, Israel, Morocco, Kenya, South Africa, Australia and New Zealand (Walker, 1976). It is a serious disease throughout the world in favourable conditions. At 31-83% systemic infection of seedlings, up to 46 per cent loss in yield may occur (Chang, Ries and Pataky, 1992).

Causal organism: Bacrerial canker is caused by *Clavibacter michiganensis* sub sp. *michiganensis* (Smith) Davis *et al.* (*Cmm*). The bacterial cells are wedge-shaped, coccoid and curved or straight rods. On nutrient agar, the colonies are smooth but become butyrous (butter like) with prolonged sub culturing, pale yellow becoming deeper yellow, opaque and glistening.

The semi-selective media MB_1M and TAM has been reported to be more efficient for detection of *Cmm* from ground seeds in phosphate buffer than other media detecting 10^3 c.f.u./ml of *Cmm*. Detection of *Cmm* in contaminated tomato seed samples on the semi-selective medium MB_1M culture medium showed

reproducibility (Valarini, 1995).The pathogen was detected in tomato seeds based on rapid screening of seed lots using indirect immunofluorescence staining (IF) followed by dilution plating of IF positive seed lots (Franken, Kamminga, Snijders, Zovwen, Vander and Birnbaum, 1993). A sensitive and specific ELISA based on polyclonal antiserum was developed for detection of *Cmm* in tomato breeding material and seeds. The most suitable parts of a tomato plant for reliable detection was the lower stem region (Kramer and Griesbach, 1995). Scortichini (1997) reported three cultivars- Arletta, Uragano and Sofia of tomato in unheated greenhouses of a nursery at Italy to be susceptible for bacterial canker and with disease incidence of 15-20%. The bacterial colonies were identified on the basis of biochemical and pathogenicity test after comparison with SDS–PAGE, whole-cell protein profiles of the isolates with type- strains of the subspecies.

The bacterium is soil inhabitant and remains in seed coat. The seed coat was attached to cotyledons and moved above ground after germination. Invasion of the cotyledons through stomata was the locus of infection. The organism, after getting established in the phloem invaded cortex and caused formation of lysogenous cavities (Singh, 1999, Bose *et al.* 2004).

Symptomatology: It causes temporary or permanent wilting of leaflets. The symptoms are light streaks beginning at the junction of leaf petiole and stem, extending down the internodes and up to the petiole. At later stage, longitudinal canker-like openings appeared on stem, petiole and midrib resulting in stunting of plants (Bose *et al.* 2004). In secondary spread, invasion of cotyledons, leaves and stem takes place through stomata, resulting in small, light coloured spots, which soon become surrounded by a white halo. Blood (1933) described the penetration through stomata in cotyledons and leaves of young seedlings. However, the pathogen penetrated through wound of roots, stem and fruits or through natural openings. Bryan (1930) reported that the pathogen confined largely to phloem and vessels. Pine and Kontoxis (1955) showed that systemic advance of the bacterium was chiefly in the spiral vessels, from the vessels bacterium invaded xylem parenchyma, phloem pith and cortex where lysogenous cavities developed. When such cavities are formed in the cortex, open cankers eventually appeared on fruits, blister-like exterior lesions may occur and the discolouration extends to the placenta and the seeds (Bose *et al.* 2004).

The bacterium was shown to initiate seed-borne infection in the cultivars “Punjab Chuhara” and “Pusa Ruby” when fruits were inoculated and seeds extracted, sown in the field then infected seedlings developed with marked symptoms (Singh and Shrivastava, 1990). Almedia, Malavlta and Robbs (1996) from Brazil reported the systemic infection and seed transmission of the bacteria. They recovered the pathogen from the infected leaves and made artificial inoculations and studied morphological, cultural, physiological and biochemical characteristics. The density of *C. michiganensis* sub sp. *michiganensis* required to cause bacterial canker of tomato was > 10^6 c.f.u./g tomato leaves. No disease was observed at a density of <10^3-10^5 c.f.u./g. The disease was more severe in an open field than in a field sheltered from rain by a plastic film. It was concluded that the multiplication and dissemination of the pathogen increased in wet weather and that cultivation under plastic film helped in controlling the disease (Bose *et al.* 2004).

Seed transmission, over winter survival and dispersal of pathogen on processing tomato and its spreading on alternate hosts and non- host plants have been evaluated with rifampicin tolerent strain *Cmm* (Rif^+) (Chang, Ries and Pataky, 1992). According to them the bacterium was transmitted at low rate from seed to transplanting by sowing infested seed in the green houses and transplanting to a production field. The population of Rif^+ detected on seed harvested from systemically infected plants ranged from about 10^2 to 10^5 cfu/g. Seed survival of Rif^+ *Cmm* associated with infested tomato debris was greater for debris on the soil surface than for debris that was buried. The population of Rif^+ *Cmm* on alternative hosts and non-host plants fluctuated from 0 to C. 10^9 cfu /g fresh weight on solanaceous plants (*Capsicum annuum, Datura stramonuim, Lycopersicon esculentum, Nicotiana tabacum, Solanum melangena, S. nigrum, S. tubersoum*) and from 0 to C. 10^3 cfu /g. fresh weight on non-solanaceous weeds (*Amaranthus retroflexus, Chenopodium album* and *Xanthium saccharatum*). The symptoms of secondary infection, marginal scorch of leaflets or bird’s eye symptoms on fruits were not observed on tomato. Rif^+ *Cmm* spread from foci of infection, but symptoms of secondary infection occurred earlier and were more severe on the susceptible cultivars. The pathogen enters or penetrates through trichomes in canker of tomato (Mehrotra and Aggrawal, 2003).

Ueno, Teraoka, Hosakawa and Watanabe (1994) stated that pathogen produced phytotoxic glycopeptides, which caused wilt of tomato cuttings. They studied the effect of crude toxins on callus. In tomato, cuttings

treated with unheated and heated (120°C, 5 min) toxin, the transpiration rate decreased to 20 to 30%. They suggested about heat labile component such as proteins may be associated in the toxin activity, in addition to the heat stable component as polysaccharide.

Survival and dissemination of the pathogen: Kendrick and Walker (1948), Grofan and Kendrick (1953) reported that bacterium survived from season to season in or on seeds, diseased plant debris in soil (up to 4 years) and on some solanaceous weeds such as *Solanum nigrum.*

The disease was influenced by soil and air factors. The optimum soil and air temperature for disease development was about 28°C, minimum temperature 16°C or maximum temperature was 36°C. Incubation period and severity of bacterial canker are influenced by temperature, plant age, inoculum concentration and cultivars (Chang, Ries and Pataky, 1992).

Gitaitis, Beaver and Voloudakis (1991) reported that bacterium was also carried by seedling which exhibit no symptoms even after surface sterilization due infection the bacterium could found in the tissues. Chang, Ries and Pataky (1991, 1992) reported that such seedlings are the major source of dissemination of the bacterium over long distance in an area. In the field dissemination is from open cankers and is mainly carried by raindrop splashes and pathogen becomes established in the phloem. It is the only vascular bacterial organism that is known to migrate through phloem eventually. It invades the cortex causing cavities, which may break open canker on the surface. The temperature (25-30°C) reached inside the greenhouses and seed transmission of the bacterium was thought to be the factors that caused the epidemic (Scortichini, 1997).

Disease management: Blood (1933, 1937) showed that at the time of processing of seed extraction high hydrogen ion concentration or high acidity eradicate the bacterium and also by soaking the seeds in acetic acid at the time of fermentation.

Soaking of tomato seeds for 25 min in 0.25% falisom Nassbeize (Phenyl mercury acetate) at 52°C effectively controlled *Cmm* and *Pseudomonas syringae* pv. *tomato.* Soaking in phenyl mercury acetate or 3% perhydrol without heat treatment were less effective (Bogatsevska, Vitanov and Boneva, 1989). In green house trails on possible control agents, disease incidence was greatly reduced by application of a new antibiotic NTC named (Bose *et al.* 2004). The pathogen was controlled from naturally infected tomato seeds that were soaked in 0.6 M HCl for 5 hrs in 0.25 or 0.50% acidified cupric acetate (ACA) for 20 min or in water at 52°C for 20 min or at 56° for 30 min (Bogatsevsta, Vitanav and Boneva, 1989). However, only HCl treatment and treatment with ACA (0.25%) at 52°C for 20 min was sufficient to eliminate saprophytes. In the blotter test all individual treatments that eliminated pathogen except water at 56°C significantly ($P = 0.05$) reduced germination. In contrast only the HCl treatment significantly reduced the germination of seeds planted in steam-sterilized soil (UC soil mix) and seeds treated with hot water (52°C for 20 min) germinated at a greater rate than the untreated control (Fatami, Schaad and Bolkan, 1991).

From Canada, it was reported that the soaking of *Cmm* infected (83%) tomato seeds in 0.1M HCl or 0.05% O-hydroxydiphenyl for 1 hr reduced seed infection to <1% and field incidence of primary canker to <2% as compared with 24% in infected check plots. This improved the seed germination also.

The seed treatment with hot (50°C) solution of 0.6% sodium hypochlorite for 15 min reduced seed infection to zero. The primary canker incidence was 4.5% but field incidence of primary canker was 10% compared with 55% in the infected check plots. There was no seed in harvested fruits of HCl or o-hydroxyphenoyl treatment plots in 1989 compared to 97% in the infected check plots (Dhanvantri, 1989, Dhanvantri and Brown, 1993).

Naumann (1980) recommended a 4-year crop rotation, plant hygiene and soil sterilization for controlling the disease. Under laboratory conditions good production of tomato plants in hydroponic culture was obtained by acidification of the nutrient solution to pH 3 for 12 hrs once a week.

Soil solarization (approx. 6 weeks of soil mulching with transparent polythene sheets) significantly reduced disease incidence throughout the cropping season (Bose *et al.* 2004).

Bacterial Wilt: *Disease incidence:* Bacterial wilt, caused by *Ralstonia* (=*Pseudomonas*) *solanacearum* (Smith) Yabuuchi *et al.* is one of the most serious disease in tropical, sub-tropical and some warm temperate and humid regions of the world. The disease was also called "southern bacterial wilt" that was reported from Japan, Ceylon, Kenya, Hawaii, Congo, Greece, North Australia, South Africa including India (Rangaswami and Mahadevan, 2002, Gopalakrishnan, 2004).

Bacterial wilt of tomato incited by race biovar III or *Ralstonia solanacerum* (syn. *Pseudomonas solanacearum)* is the most important disease in zone II of Himachal Pradesh.

In India, the disease poses a constant threat to tomato in Karantaka, Madhya Pradesh, Marathwada region of Maharastra and West Bengal. The loss in yield may vary between 10.8 and 90.6 per cent depending on the stage at which infection occurs and the environmental conditions (Kisun, 1987). Maximum loss occurred during summer when crop was infected within 60 days of planting.

Detection: Rajeshwari, Shylaja, Krishnappa, Shetty, Mortensen and Mathur (1998) used ELISA for detection of *Ralstonia solanacearum* in tomato. The bacterium was detected by using serological methods from soil and plant material.

Disease symptomatology and epidmiology: Kelman (1953), Buddenhagen and Kelman (1964) and Walker (1976) observed that vascular system in tomato changed brown to black and development of adventitious roots including stem.

Initially, lower leaves droop and wilting of plant occurs. Wilting, stunting, yellowing of the foliage and finally collapse of entire plant are important symptoms of the disease. The vascular bundles turned brown, slimy bacterial material exuded from the bundles when the stem is cross-sectioned. In humid weather due to high moisture, sudden drooping of plants takes place from any point without yellowing and rotting of stem (Walker, 1976, Bose *et al.* 2004, Sharma and Agrawal, 2010).

The pathogen is soil-borne, persisting for long periods in soil. From the soil, the pathogen invaded the host plant mainly through natural openings and wounds. In roots, pathogen attack at point where secondary roots emerged, mechanical injury due to root knot and spiral nematodes aid in root penetration (Katsura and Uemura, 1963, Libman and Sequeira, 1964, Walker, 1976).

Winstead and Walker (1952), Kelman and Cowling (1965) reported that the pathogen produced PME (Polymethyl esterase), PG (Polyglacturonase) and cellulase in culture. Husain and Kelman (1957, 1958) reported that wilting was due to accumulation of extra cellular polysaccharide (EPS) produced as slime in the vessels, resulting in moment of water in them. The pathogen migrated first in large xylem vessels in stem later progressed in inter cellular spaces of cortex and pith causing lysogenous cavities. Invasion in a single lateral, bundle in petiole caused drooping (epinasty) while invasion of all bundles caused wilt. When vessels were invaded adventitious roots developed in the tissue just outside of the invaded bundles (Walker, 1976).

The seed infection also resulted in wilting of adult plants. In 6 cultivars, seed from healthy plants obtained from a wilt infested field showed no evidence of the pathogen. No relationship was found in between the per cent seed infection and plant mortality (Singh, 1994). Singh (1999) reported that the high moisture and nematode infection favoured the disease. The toxin responsible for causing wilt was isolated from *Pseudomonas solanacearum* race 1 biovar III by alcohol precipitation and ion exchange chromatography. Characterization of toxin revealed the presence of glucose, mannose and polypeptides. Cell lines of 5 wilt susceptible tomato varieties growing on modified MS media, were treated with purified toxin (EC 50). Resistant plants had a significant increased in rishitin production followed toxin and pathogen treatments. The toxin induced physical damage of membrane system in susceptible cell (Rath and Addy, 1977, Bsoe *et al.* 2004).

Disease management: The hot water treatment at 50°C for 25 minutes was found effective in controlling the wilt. An antibioic validomycin A was tested against *Pseudomonas solanacearum* for its efficacy in controlling tomato bacterial wilt. In culture medium that containing trehalose as the sole carbohydrate @ 50µg/ml, inhibited growth of bacteria to rates similar to that of bacteria in media without carbohydrate for 7 days after inoculation. The antibiotic gave excellent control of wilt in green house, pot testes when directly injected into plant stem as well foliar spray @ 250µg/ml 5 days before and 2 days after inoculation had reduced disease by 47.4% by 4 weeks after inoculation. In tomato stem at 0 and 5 cm above the soil line, the bacterial population in the non-treated plot reached 3.84×10^{10} c.f.u./g fresh weights, whereas in VM-A (500µg/ml) treated plot reached 2.13×10^{9}c.f.u./g fresh weights. The VM-A delays the appearance of disease symptoms (Ishikawa, Fujimori and Matsura, 1996). Application of super phosphate increases the disease incidence while nitrogen fertilizers supress it (Singh, 1999).

Tomato crops sprayed with 200 ppm streptomycin sulphate (oxytetracycline) or agromin at 4 and 7 days intervals gave high degree of control of the pathogen (Mazumdar, 1998).

A soil amendment comprising of bagasse, rice husk powder, oyster shell powder, urea, KNO_3, Ca $(H_2PO_4)_2$ + $CaSO_4$, CaO, $Na_2B_4O_7$, $ZnSO_4$, mineral ash and chicken manure for controlling bacterial wilt in the field was recommended. Soil amendments with soybean flour, rice husk + urea along with recommended dose of fertilizers also proved beneficial (Bose *et al.* 2004).

Soil drenching with a mixture of asafoetida, turmeric powder and water (ATW) after transplanting of tomato plants was effective for disease control in the field. Spraying 5% ATW also inhibited growth of *R. solanacearum* in *in vitro* (Bora, 1995).

The wilt pathogens showed complete reduction in population when the bacterium was cultured in acidified nutrient solution (pH 3.5-4.0) and suggested that control of wilt might be possible in acidified nutrient solutions (Bose *et al.* 2004).

Arwiyanto and Goto (1994) tested the bacteriocinogenic *Sterlizia* strain (Sr10) of *Pseudomonas solanacearum* to check its ability to suppress the development of bacterial wilt in green house. The spontaneous avirulent mutant of strain Str-10 protected tomato plant from bacterial wilt. The protection was more pronounced when the temperature was low (18-25°C) and lost at high (30-37°C) temperature. The strain Str-10 op type was maintained in the rhizosphere and rhizoplane. The promising protection due to Str-10 op was obtained when the population ratio of the challenge strain Str-10 op type was smaller than 1.The ability of Str-10 type in suppressing the wilt presumed to be derived from induced resistance of host plant.

The antimicrobial activity spectra against the indicator bacteria and fungi varied greatly with the species and strains of pseudomonades. The bacterial strain ATCC 7700 of *Pseudomonas aeruginosa* was highly antagonistic *in vitro* to *R. solanacearum.* The soaking of tomato plantlet roots and seeds at time of transplanting infected with *R. solanacearum* by *P. aeruginosa* strain ATCC 7700 increased the survival of seedlings in soil. It was supposed that due to some antibodies the pathogen was killed. It was proved by treating the seeds with killed cells of *P. aeruginosa* (Furuya, Yamasaki, Nishioka, Shiraish, Iiyama and Matsuyama, 1997).

Antagonistic bacteria like *Bacillus subtilis* (B1); *Pseudomonas* sp. (106, 115) and *P. cepacia* (*Burkholderia cepacia*) had a high inhibitory effect against *R. solanacearum* both in the laboratory and the green house. Thus, these were the bacterium promising for the biological control of *R. solanacearum.* Pot experiment, were conducted under green house conditions to determine the combined effect of host resistance, antagonists *viz*. *Pseudomonas fluorescens* and *Bacillus cereus* and soil amendments ($NaNO_2$ and KNO_3) against the pathogen (Kumar and Sood, 2002). At high temperature loss of resistance had been reported by Mew and Ho (1977) and Rema Devi and Menon (1980).

Kapoor, Sugha and Singh (1991) screened 62 exotic and Indian lines/varieties of tomato for resistance of bacterial wilt. Arka Souran has moderate resistance variability among the isolates of the pathogen and weather parameters especially temperature determine stability of resistance.

Crop rotation with cruciferous vegetables and maize, okra, cowpea or resistant tomato cultivars were helpful in suppressing the disease and reduce the incidence of pathogen (Bose *et al.* 2004).

Bacterial Leaf Spot*:* Kritzman (1989) described a method for detecting *Pseudomonas syringe* pv. *tomato, P. corrugata, Xanthomonas campestris* pv. *vesicatoria* and *Corynebacterium michiganense* (*Clavibacter michiganensis* sub sp. *michiganensis*, *Cmm*) to a level of 10 c.f.u./g tomato seed.

Sijam, Chang and Gitaitis (1991) from Griffin, GA., USA developed an agar medium for the isolation and identification of *Xanthomonas campestris* pv. vesicatoria *from* the tomato seeds. The pathogen or pathovars of *Xcv* showed a clear ring after 1-2 day around their colonies after inoculation on CKTM medium. The recovery of *X. c*. pv. *vesicatoria* from tomato seeds ranged from 17.7 to 100% on CKTM compared with 6.3 to 44.4% on Tween-B medium. In addition to recovery of *X. c*. pv. *vesicatoria* on CKTM medium was qualitatively superior to that of Tween- B medium, with a greater reduction of contaminating micro flora.

X. c. pv. *vesicatoria,* the causal agent of bacterial spots on tomato and pepper spreaded very fast in warm and humid environment (pathogen also listed in A2 quarantine list). The bacteria showed spot on seedlings in European countries, defoliation in young plants and spots on leaf, stem and fruits in older plants. The leaf spot disease is also caused by *Cmm* and *P. syringae* pv. *tomato* in tomato.

The bacterium was identified and confirmed by immunofluroscence techniques confirmed by central science laboratory, New York (USA) with analysis of fatty acid profile. Other bacterial species that were identified as C*mm* and *P. syringae* pv. *tomato* (Ravnikar, Demsar and Dreo, 2001).

Black, Seal, Zakia, Nono-Womdium and Swai (2001) reported from Tanzania that in rainy season during 1997 and 1998 bacterial spot was found in most of the mainland vegetable regions of northern and southern high lands. In fields of tomato 59 and 50 field surveys during 1997 and 1998 respectively showed bacterial spot and disease incidence, which varied greatly between years and fields (from <5% to >90%). The pathogen was confirmed in 5 to 26 samples of farmers saved seeds by pathogenicity and biochemical tests.

For the control of *Xanthomonas campestris* pv. *vesicatoria* in tomato sodium hypochlorite, chlorohydric acid, sodium hydrochloride and the combination of sodium hypochlorite and chlorohydric acid at 2% and 3% were used in artificially inoculated seeds of the tomato variety L-72-1. The soaking of seeds in sodium hypochlorite or the combination of sodium hypochlorite and chlorohydric acid for 20 min gave the best disease control (Hernadez and Medina, 1991).

Ward and O´Garro (1992) reported that 240 strains tested for sensitivity to bactericides 61, 64 and 74% were resistant to copper, zinc and streptomycin respectively from Barbados, West Indies. The leaf spot disease was controlled by using zineb @ 0.2% and sprayed in interval of 7-10 days (Beach, 1950, Borders, 1951, Boelema and Gelderblom, 1956, Nene and Thapliyal, 1997).

Shukla and Gupta (2004) applied several chemicals for controlling of bacterial spot disease of tomato. They concluded that streptomycin sulphate (100 ppm) was found most effective resulting in 3.8 cm diametric inhibition zone and 11.24 cm^2 inhibition area followed by combination of streptomycin sulphate (10ppm) plus copper oxychloride (1000 ppm) which differed significantly from streptomycin sulphate alone exhibiting 3.4 cm and 8.96 cm^2 diametric zone and inhibition area, respectively.

Sterile aqueous garlic extract (10, 20 and 30 g/100ml) was toxic to *X. campestris* pv. *vesicatoria* isolated from tomato seeds. The maximum inhibition zone on PDA was observed after adding benzene, chloroform, ethyl acetate and methanol- ethyl acetate in garlic extract. After soaking of tomato seeds in culture suspension of the pathogen and treated with aqueous garlic extract @ 30g/100ml (w/v), 15-35 day after sowing plants had an 85% decrease in number of leaf spots as compared to the control (Mangamma and Sreeramulu, 1991).

Bacterial Speck and Leaf Spot of Tomato: Karaca and Demir (1998) from Turkey isolated *Pseudomonas syringae* pv. *tomato*, *Corynebacterium michiganensis* pv. *michiganensis* (*Clavibacter michiganensis* subsp. *michiganensis*) and *Xanthomanas campestris* pv. *vesicatoria* from tomato.

A survey was carried out to identifiy survival niches of phytopathogens in Sao Paulo, Brazil in soil with irrigated tomato and *Pseudomonas syringae* pv. *tomato* was isolated these tomato seeds (Valarini and Spadotto, 1995). *Amaranthus deflexus* and *Emilia sonchifolia* were identified as host for *X. c.* pv. *phaseoli*. They reported that presence of alternate hosts perpetuates the survival of the pathogen (Valarini and Spadotto, 1995).

Seeds of tomato and several weed species were inoculated with Rif-Nal mutant of *P. s*. pv. *tomato* (PST-M), *P. s.* pv. *syringae* (PSS-M) and *P. viridiflava* (PV-M) by vaccum infiltration and soaking methods and stored in paper bags under lab conditions. PST-M was not detected after 2 months. PST-M was detected only in seeds of buckhorn plantain (*Plantago lanceolata)* 2 months after inoculation. PV-M survived in seeds of buckhorn plantain for 4 months and jimson weed *(Datura stramonium)* for 6 months, suggesting the possibility of the natural survival on seeds of certain weed species. The transmission of PST-M, PSS-M and PV-M from contaminated seeds to emerging seedlings was not demonstrated. When all host-sampling time periods were considered collectively, the vaccum infiltration procedure was significantly more efficient method for seed inoculation with PV-M than the soaking procedure (Mariano and McCarter, 1992).

Kritzman and Ovadia (1989) from Israel reported that when tomato seeds were dipped in a specially developed solution (PL-488) for 1 h at 45°C *Pseudomonas syringae* pv. *tomato, P. corrugata, Xanthomonas campestris* pv. *vesicatoria* and *Corynebacterium michiganensis* (*Clavibacter michiganensis*) were all controlled. The ratio of seeds/solution in the thermo-statistically controlled bath circulator was 1: 4. No phytotoxic effects were observed in relation to seeds germination or vigour.

In a study for the controlling of *Pseudomonas syringe* pv. *tomato* (PST), *P. corrugata* (PC), *Xanthomonas campestris* pv. *vesicatoria* (XCV) and *Clavibacter michiganensis* sub sp. *michiganensis* (CMM) tomato seeds were immersed at a ratio of 1:4 (w/v) seeds : chemicals containing cupric acetate, acetic acid, pentachloronitrobenzene, 5-ethoxy-3 (tricholoromethyl)-1,2,4-thiadiazole and triton X-100 for 1 h at 45 ± 0.1°C. PST, XCV and CMM were almost eradicated after immerson of seeds for 30 min at 25°C where as XCV was controlled after 1 h treatment at 45°C. The treatment did not affect germination or seedling vigour (Kritzman, 1993).

Catara and Bella (1990) from Italy reported that in sicily infection of bacterial speck caused by *Pseudomonas syringae* pv. *tomato* was quite common under plastic houses and induced severe loss. The environmental conditions of small plastic house and the reduced use of copper compounds were involved in the outbreak of the disease.

The tomato pith necrosis had been detected in green house and field in Israel since 1981. The symptoms include pith necrosis, brown black discolouration of the vascular system and collapse of stem. *Pseudomonas corrugata* was consistently isolated from stem, petioles and fruits pedicles of infected tomato plants. The pathogen was also isolated from 2 important seed lots of a hybrid cultivars which was the first indication that *P. corrugata* might be seed-borne (Zutra, 1989).

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