Agric. Rev., 25 (1) : 40 - 48, 2004

PHYTOPHTHORA NICOTIANAE VAR. *PARASITICA*, A VERSATILE FUNGUS CAUSING BUCKEYE FRUIT ROT OF TOMATO - A REVIEW

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ABSTRACT

Buckeye fruit rot is a major limiting factor in tomato cultivation. The disease is caused by *Phytophthora nicotianae* var. *parasitica*, which has generally been reported to infect only green fruits. However, at places, it has been reported to infect other plant parts too. Information in this review, has been presented on distribution, losses, symptomatology, etiology, infection, host range and perpetuation of this disease. Management practices, including cultural, biological and chemical have been described in detail.

Tomato (*Lycopersicon esculentum* Mill.), a member of solanaceae family, is an important commercial vegetable grown throughout the world. It is native to Central and South America and tomato is consumed as salad, ketchup, sauce, soup, pickle etc. Ripened fruits are good source of ascorbic acid, vitamin A, organic acid and minerals like potassium and sodium. The crop occupies an area of 2.85 million hectare with 77.54 million metric tones production (FAO, 1998) throughout the world. In India, the tomato covers an area of 0.35 million hectares with an annual production of 5.30 million metric tones (FAO, 1998). The crop is generally grown during the winter months (Oct.-April) in the plains of India and summers (April-Oct.) in hilly regions of the country. High humidity and warm temperature during summer crop favour development of various diseases. Of these, buckeye fruit rot of unripe tomato fruits is of serious concern to the small and marginal hill farmers. During rainy season, the disease appears in epiphytotic proportion and takes away a big chunk of production. In most of the localities buckeye fruit rot is confined to green fruits. Elsewhere, *Phtophthora parasitica* has also been reported to infect other plant parts also. This paper reviews the information available on *Phytophthora parasitica* occurring on tomato in general and buckeye fruit rot in particular.

Distribution, Importance and Losses

The disease is prevalent in almost all the tomato growing localities of the world such as California (Tompkins and Tucker, 1941), West Indies (Baker, 1939), Rhodesia (Hopkins, 1945), China (Wei and Cheo, 1946), Malaya (McIntosh, 1951), Mississippi (Welch, 1949), Germany (Gunther and Grummer, 1958), Sardinia (Marras, 1964), Sweden (Anderson, 1987) and England, (Bewley, 1922). The first occurrence of the disease was reported by Sherbakoff (1917) from Florida. In India, the disease, as buckeye rot, was reported for the first time from Solan, Himachal Pradesh by Jain *et al.*. (1961), though a fruit rot of similar nature was described by Ramakrishnan and Soumini (1947) from Coimbatore. Subsequently, the disease was reported from Bombay (Rao, 1966), Haryana (Srivastava, 1981), Punjab (Sharma and Singh, 1992) and other parts of the country. The disease is highly destructive and causes huge losses. Sherbakoff (1917) observed fifteen per cent fruit rot in the field and ten per cent in transit. The disease has been reported to cause forty per cent damage to fruits in Indiana (Kendrick, 1923). Heavy losses to green fruits due to this disease has also been reported from Denmark (Gram *et al.*, 1927) and Eastern Trans Vaal (Wager, 1935). Jain *et al.* (1961) reported 35-40 per

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cent losses to tomato fruits in Himachal Pradesh against only five per cent losses at Berthiervill Montreal (Lavallee, 1941). Sharma (1971) observed up to 90 per cent incidence under high humidity and good rainfall conditions in Himachal Pradesh.

Nomenclature

The first occurrence of tomato fruit rot was noted by Sherbakoff in 1915 at Goulds, Florida and named it as "Buckeye rot of tomato fruit" (Sherbakoff, 1917). The term originated from tomato buyers in Florida. Sherbakoff took up this name because it described very well the most striking feature of the lesion on affected fruits with broad zonations resembling the eye of a large animal.

Manifestation

Initially typical buckeye rot symptoms were reported to occur only on immature green fruits (Sherbakoff, 1917) but Reddick (1920) described an isolate of *Phytophthora parasitica* causing typical damping off of seedlings, stem girdling, fruit rot and foliage blight and he named the disease as stem girdle. Subsequently, it was reported by workers from different localities/countries that damping off of seedlings, collar rot, stem canker, and blossom blight, symptoms are also induced by *Phytophthora parasitica* in tomato (Bewely, 1922; Taylor, 1924; Samuel, 1930; Goidanich, 1936; Conners, 1937; Richardson, 1941; Singh and Srivastava, 1953; Sharma, 1971; Raicu and Stan, 1973). Root rot of tomato caused by *P. parasitica* has also been reported (Fulton and Fulton, 1951). Walker (1952) mentioned that the pathogen does not affect foliage and in this respect the disease is distinct from late blight, though it may cause damping off symptoms. Tomato roots, stems and fruits are attacked by *P. n.* var. *parasitica* but under natural conditions leaves are not infected (Raicu and Stan, 1973). In Himachal Pradesh, the disease symptoms occur only on immature green fruits. Other plant parts are not attacked, though, Sharma (1971) found that pathogen causes damping off, collar rot, stem canker and blossom blight under artificial inoculation conditions. The most typical symptoms are first observed on lower immature green fruits, which are either on or near to the soil level. The lesions have dark brown center surrounded by advancing zonations with water soaked appearance (Richardson, 1941). These enlarge rapidly and within 3-4 days whole of the fruit surface turns dark brown and feels soft on touch. The internal flesh shows discolouration without any rotting on the skin, which peels off easily. White flocculent superficial growth of the fungus consisting of sporangia develops profusely on the diseased fruits in warm and humid weather (Ramakrishnan and Soumini, 1947). Later, these fruits may drop down. With inroad by saprophytic microorganisms, infected fruits decompose fast. Mature green fruits are also infected by the pathogen, but, those turning yellow are less susceptible. In addition to zonated fruit spots, symptoms are observed without zonation also (Sherbakoff, 1917). Obrero and Aragaki (1965) reported that though green, mature and fully red fruits also rotted readily due to *P. parasitica* infection but those turning pink at the time of inoculation were resistant.

The Cause

Depending upon the symptoms produced several *Phytophthora* spp. have been reported to cause this disease. Sherbakoff (1917) described the causal fungus as *P. terrestria*, which was later on reported to be a synonym of *P. parasitica* (Tucker, 1931). Kreutzer and Bryant (1944) reported *P. capsici* as the incitant of tomato fruit rot in Colorado. The association of other *Phytophthora* species such as *P. drechsleri, P. pamivora, P. cryptogea, P. maxicana* etc. has also been observed (Hotson & Hortge 1923; Tompkin & Tucker, 1941; Simonds and Kreutzer, 1944, Thomas

Table 1. Symptoms developed by different *Phytophthora* spp. on different plant parts of tomato

Symptoms	Species associated	Location(s)	Reference(s)
Buckeye rot of tomato fruit	*P. terrestria*	Florida	Sherbakoff, 1917
Damping off and foot rot	*P. cryptogea*	Hertfordshire New Zealand	Bewley, 1922 Taylor, 1924
Stem girdle	*P. parasitica*	Canada, Ontario	Conners, 1937 Reddick, 1920
Buckeye rot	*P. drechsleri* *P. capsici*	California	Tompkins and Tucker, 1941
Buckeye fruit rot	*P. parasitica*	Cheshunt	Williams, 1941
Damping off and foot rot	*P. cryptogea*	Cheshunt South Australia	Green, 1941; Williams *et al.*, 1943, 1946 Samuel, 1930
Stem rot	*P. parasitica*	Hertfordshire and Hampshire	Williams and Sheard, 1943
Buckeye rot	*P. parasitica terrestria*	Tennessee	Felix, 1948
Root rot	*P. cryptogea* *P. parasitica*	Kent	Glasscok and Dermott 1950; Fulton and Fulton, 1951
Brown rot	*P. parasitica*	East Trans Vaal Germany	Wager, 1935; Gunther and Grummer, 1958
Root and crown rot	*P. n. var parasitica* *P. capsici*	Davis, California	Satour and Butler 1966, 1967
Seedling rot	*P. novar parasitica*	Cuba	Perez *et al.*, 1989a
Collar rot	*P. parasitica*	Italy	Goidanich, 1936; Sarejanni, 1936
Fruit rot	*P. pamivora*	India Coimbatore	Ramakrishnan and Soumini, 1947
Buckeye rot	*P. n. var parasitica*	India, H.P.	Jain *et al.*, 1961

et al., 1947). The comprehensive list of different types of symptoms produced and *Phytophthora* spp. associated is given in the Table 1.

In California, *P. drechsleri* and *P. capsici* proved pathogenic to unwounded fruits with incubation periods of 11 and 7 days, respectively (Tompkins and Tucker, 1941). Williams (1941) reported the association of *P. parasitica* with buckeye fruit rot. Richardson (1941) noted damping off and stem, fruit, leaves and root rot being caused by *P. parasitica.* Buckeye rot of green tomato fruits has consistantly been ascribed to *P. parasitica* by investigators in various countries Bewley (1922) suspected involvement of *P. cryptogea* (Tompkins and Tucker, 1941). *Phytophthora parasitica terrestria* has been reported to cause buckeye rot of tomato at Tennessee Agricultural Experiment Station (Felix, 1948). Weststeijn (1973) attributed tomato root and fruit rot to *P. nicotianae var. nicotianae.* However, latest and widely accepted name of the fungus responsible for buckeye fruit rot is *P. nicotianae* (Breda de Haan) var. *parasitica* (Dastur) Water House (Singh, 1985). The morphological and cultural characteristics of the fungus are well described by Richardson (1941), Rao (1984), Kouyeas (1953), Sharma, (1971), Sharma *et al.* (1976), Sharma and Sharma (1982, 1987), Shyam and Dodan (1991), Dodan and Shyam (1994) and the other monographs on *Phytophthora* (Erwin *et al.*,1983; Holliday, 1989).

Host Range

The pathogen is associated with

damping off, root rot, crown rot, stem canker, and fruit rot of different hosts, including, beans, maize, brinjal, chillies or peppers and numerous ornamental and wild plants (Singh, 1985). Isolates show difference in their virulences. Richardson (1941), on artificial inoculation, noted a wide range of hosts almost all belonging to the family solanaceae. In inoculation tests, yellow crookneck and zucchini pumpkin and *Cucurbita pepo* var. *condensa* were found susceptible to *P. parasitica* and *P. capsici,* whereas turnip to *P. drechsleri* (Tompkins and Tucker, 1941). Hickman (1958) reported that *P. parasitica* parasitizes plants in 72 genera and in 42 families of flowering plants. Plants such as *Euphorbia hypericifolia, Physalis minima, Mirabilis Jalapa, Commelina obliqua, Tagetes minor, Ipomea muricata, Galinsoga parviflora, Cynoglossum wallichi* were also susceptible to the pathogen (Sharma, 1971).

Penetration of host tissues

Primary infection occurs through the zoospores under optimum moisture and temperature regimes. Simonds and Kreutzer (1944) noted that after the establishment of contact between zoospores and host surface, the zoospores start germinating within 3 hours by germ tube and give rise to appressoria on the surface of the fruits. Infection hyphae develop from the appressoria, which penetrate the cuticle. Rosenbaum (1920) noted that a maximum period of 24 hours is necessary for the zoospores of *P. parasitica* to be in contact with the tomato fruit to produce infection and after 72 hours the lesions were macroscopically evident. Invasion takes place downward as well as radially. The hyphae invading intracellularly are smooth with granular protoplasm, branching profusely in the walled parenchymatous cells of the host. These penetrate deep into the fruit tissue and attack seeds covering them with the mycelium on all sides. The fungus also penetrates the seed coat and develops inter as well as intracellularly in the endosperm of the seed. The mycelium agglomerate in the epidermal layer of the affected portion of the fruit, push through the epidermis and cuticle by rupturing it and under humid conditions grow on the fruit surface and produce a crop of sporangia on simple branched sporangiophores. Sporangia are not produced inside the host tissues. These are variable in size and shape depending upon relative humidity (Sharma, 1971). Invasion also occurs through the stem end, but lesions develop slowly in this region (Walker, 1952).

Perpetuation of the pathogen

Richardson (1941) found that fungus could remain active in soil atleast for a year without the support of a susceptible host. Alcock (1931) doubted the seed borne nature of the fungus. Sharma (1971), however, confirmed the seed borne nature of the pathogen and reported that mycelium was found intermingled in the seed hair, penetrated into the seed coat and advanced toward the ovule and remained active for more than one and half years. He also indicated the role of some collateral/weed hosts in the perpetuation of the fungus. He further suggested that fungus remained viable for more than two years under ordinary field conditions in the soil and a depth of 10 cm and wet soil were more favourable for its perennation. Anderson (1987) analysed the seeds from diseased fruits and ruled out the possibility of its being seed borne once again. The disease is mainly soil borne and pathogen survives in soil for many years through its resting structures (Singh, 1985).

Epidemiology and Forecasting

The pathogen perpetuates in soil through its oospores and chlamydospores. Rosenbaum (1920) found fruits touching the ground more severely infected. An abundant supply of moisture, preferably free water in the infested soils, is essential for producing an epiphytotic of the disease. Kendrick (1923)

believed that abundant soil moisture was an important factor in the causation of the disease. Wager (1935) indicated the role of rainy weather in the development of the disease. Richardson (1941) demonstrated that high atmospheric temperature and relative humidity as favourable for infection. The pathogen causes severe damage only during wet weather (Baker, 1939). The disease is favoured by high temperature, humidity and contact of fruits with moist soil or irrigation water (Tompkins and Tucker, 1941). Further the fruit age has also been shown to be a factor in disease development. Small or full sized green fruits as well as mature fruits are readily infected by *P. parasitica* (Critopoulus, 1954). Inoculum surviving in soil is splashed from the soil by rain/irrigation water (Williams *et al.*, 1946). The susceptibility of the fruits also varies with amount of inoculum reaching to the fruit surface (Dodan and Shyam, 1996). After infection, zoospores are produced on fruits and carried by wind, water and other agencies to other fruits (Singh, 1985).Maximum fruit infection occurs at a temperature range of 20-25°C, RH > 60%, high rainfall and high soil moisture conditions (Threja *et al.*, 1989). Obrero and Aragaki (1965) reported optimum temperature for infection between 24-31°C. Sharma et *al.*, (1979) concluded that the disease does not occur at or below 20°C, but 22-25°C supplemented with slight rainfall (10 mm) will result in disease appearance after 4 days. The disease is not serious above 25°C as the sporangia germinate directly by germ tube rather than zoospores, thus reducing the amount of inoculum (Sharma *et al.*, 1979). Frequent irrigations, at 4-day interval, enhance the disease development as it, results in production of zoospores more rapidly and abundantly (Hoy *et al.*, 1984).

The role of various factors including height of the plants, total acidity, ascorbic acid, and nitrogen content of fruits and cuticular thickness of fruit towards resistance has been studied. Though significant negative correlation between average height of plants for a given variety to disease incidence was noticed yet the regression analysis showed that none of the factors mentioned above independently had any effect on incidence (Sharma *et al.*, 1975). Higher doses of nitrogen gave more yields but significantly higher disease incidence also, whereas increased levels of P suppressed it with K levels, generally, having little effect on yield or disease incidence (Sharma and Sohi, 1983).

Management

Integrated disease management practices, which include use of cultural, biological, chemical and resistance-breeding measures, effectively manage soil borne diseases.

(i) Cultural Practices: Use of cultural measures in the present day agriculture is of immense value. Addition of soybean residues to infested soil suppressed the activities of the pathogen (Richardson, 1941). Williams and Sheard (1943) recommended destruction of diseased fruits and keeping the plants trimmed to avoid moist and stagnant air conditions. Plants should be staked or tied to frames to prevent contact with the soil (Wager, 1935; Ramakrishnan and Saumini, 1947; Welch, 1949). Wider spacing 4-6 x 1-1.5 feet should be adopted in unstaked commercial plantings to control septoria leaf spot, early leaf blight and buckeye rot (Loest, 1948). The avoidance of poorly drained areas in the field also reduces the disease severity. The practice of staking is the most effective mean of control (Walker, 1952). Bryant & Kreutzer (1945) suggested setting of the plants on ridges in irrigated soils under conditions where staking was not feasible. Mulching and spraying the mulch with fungicide also reduced the disease (Welch, 1949). Foliage and fruits have to be removed upto 30 cm from ground level to control the disease (Sharma *et al.*, 1977b). The efficacy

of fungicide application increased by 27 per cent in staked plants (Bhardwaj *et al.*, 1995). Mulching with pine needles reduced the disease incidence and increased yield (Shyam and Gupta, 1996b) because it avoids splashing of soil inoculum by raindrops. Combination of fungicide sprays, clipping lower leaves, weeding, application of polythene mulch to obstruct dispersal of soil borne inoculum of the pathogen and removal of affected fruits increased yield and reduced disease incidence (Dodan *et al.*, 1994).

(ii) Chemical Control: Chemicals are unavoidable means of plant disease control, which form the protective covering on the host surface, or eradicate the established infections thereby reducing the production of secondary inoculum, and ultimately restricting the pace of progress of the disease. Various systemic and non-systemic fungicides have been evaluated and recommended for the control of buckeye fruit rot by various workers. Ramakrishnan and Soumini advocated spray of Bordeaux mixture as early as 1947. Chemicals such as tribasic copper sulphate (Fulton, 1954), Dithane M-45, basamid, Solasan, Vapam and ditrapex (Raicu and Stan, 1973; Raicu *et al.*, 1974), Difolatan, copper oxychloride (Sokhi and Sohi, 1975) have been recommended for its control.

An integrated approach consisting of spray of Dithane M-45 or Bordeaux mixture or captafol alongwith staking and removal of foliage and fruits upto 30 cm was found to be most effective and economical (Sharma *et al.*, 1977a, b; Thapa and Sharma, 1978; Das and Mohanty, 1987; Sharma, 1992). Further the sequential sprays of captafol, mancozeb and copper oxychloride after 40, 55 and 70 days of transplanting increased yield by 50.5 per cent by reducing the incidence of *Alternaria solani, A. alternata, P. n. var. parasitica* and *Septoria lycopersici* (Bhardwaj, 1991). Verma *et al.* (1994) while testing Dithane M-45 and Ridomil MZ-72 against P. *n. var. parasitica* and *P. infestans* on different tomato cultivars found that cultivar Roma gave highest yield of quality fruits with Ridomil MZ-72 and best seed yield with Dithane M-45. A combination of metalaxyl + mancozeb was most efficacious in checking the magnitude of buckeye fruit rot and Alternaria leaf spots (Shyam and Gupta 1996a). Pine needle mulch and Ridomil MZ sprays on mulch and foliage reduced the buckeye fruit rot incidence (Shyam and Gupta 1996b ; Gupta *et al.*, 1998).

(iii) Resistance Breeding: Lines showing resistant reaction under natural epiphytotic condition succumb to inoculations under laboratory conditions. The resistance is available only in small-fruited cherry type lines, which get eliminated to dilute in advance generations in the process of breeding for desirable fruit size. Fruits of 11 tomato varieties proved highly resistant to natural and artificial infections of *P. parasitica terrestria* and apparent segregates of Sherbakoff's 57 x Oxheart were most promising with respect to fruit size and resistance to buckeye fruit rot (Felix, 1948). Falix (1948) further noted that all *Lycopersicon* spp. Tested, including, L. *esculentum, L. glandulosum, L. cerasiforme and L. peruvianum* were susceptible to the disease (Felix, 1953). Obrero and Aragaki (1965) reported that hybrid N-64 (STEP 390 x HES 6578) was highly resistant when tested under in *vitro* conditions. They further observed that resistant varieties required more zoospores and longer incubation period for infection than susceptible ones. According to Sharma *et al.*, (1976) the factors contributing to buckeye rot resistance were plant height, ascorbic acid content, total acidity and total nitrogen content of green fruits. Polyphenol oxidase activity was higher in resistant varieties than in susceptible ones (Sharma and Bhardwaj, 1977). Tomato fruits are known to produce antifungal compounds such as

lycopene whose quantity vary with cultivars and fruit size. Smaller the fruit size, lower the lycopene content and stronger the reaction (Trique, 1977). Rattan and Saini (1979a,b) indicated that resistance to fruit rot is controlled by dominant genes and ascorbic acid content but Fageria (1994) observed it to be polygenically controlled. Various biochemical parameters such as total phenols, orthodihydroxy phenols, ascorbic acid, amino acids, peroxidase and polyphenol oxidase activities were found negatively associated with buckeye rot while sugars exhibited a positive association (Singh and Jamwal, 1996). Fruit rot resistant lines EC 129603, EC130035 and *Lycopersicon pimpinellifolium* contained higher levels of phenolics and phenol oxidizing enzymes than the susceptible Cv Solan Gola (Singh *et al.*, 1997) Cultivar Rossol was found to be resistant to buckeye fruit rot in laboratory as well as in field evaluation studies (Perez *et al.*, 1986, 1989a,b). Threja and Srivastava (1989) graded lines such as EC 129171/Pl-2, EC128964, EC129166, EC72901, EC122962-1 and Hybrid 10 as resistant. Kohli *et al.*, (1995, a, b) exposed huge tomato germplasm to natural epiphytotics and found that none of the varieties/lines was free from the disease though the lines EC 174023, EC 174041 and Sel 16 had good fruit size and tolerance to the disease. They also observed that resistance to disease is confined to small cherry fruited lines having fruit weight lesser than 20 g (Kohli *et al.*, 1996). Kumar *et al.*, (1997) categorized accessions LA 1428 and LA 2205 as resistant to fruit rot and LA 1308 and LA 2631 as moderately resistant to fruit rot. Small-fruited line L. *esculentum var.ceresiforme* does possess resistance against the disease (Dodan *et al.*, 1995; Fageria *et al.*, 1998).

(iv) Biocontrol: Information on the control of buckeye fruit rot through biological means is lacking but a few reports do exist against *P. parasitica* causing damping off in tomato. *Trichoderma koningii, Penicillium patulum* and cultural filtrate of *Streptomyces aurantiacus, S. longissimus and S. griseus* were observed to be antagonistic to the pathogen (Katser, 1938; Williams *et al.*, 1949, Tsintsadze and Tsilosani, 1973). Tomatp *cv.* Earlymech plants inoculated with *Glomus mosseae* increased plant resistance to *P. nicotianae var parasitica* infection (Trotta *et al.*, 1996) Further studies on the use of these antagonistic microorganisms for the management of buckeye fruit rot are required.

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