Early blight (Alternaria solani) etiology, morphology, epidemiology and management of tomato: Review article

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Abstract

Alternaria solani is a disease that causes early blight in tomatoes, as well as potato and other Solanaceae family crops. Due to early blight up to 79% yield loss reported in different countries. A. solani cause severe infection in tomato crop and infection occur in all the part of plant stem, leaf, and fruit. It may cause damping off, stem canker, fruit rot, and collar rot, among other symptoms. The pathogen causes a dark lesion with a yellow halo and a concentric ring-like symptom. Pathogen septate and branched mycelium and conidiophores produce muriform conidia it may singly or in a chain. The pathogen can survive in the form of mycelium and conidia in the soil and dry and warm weather, 25-30°C temperature required for growth of pathogen. Crop rotation, removal of contaminated plant waste, and the use of chemicals such as Bavistin, Score, Sanit, kavach, Isignia, Antracol, and others to manage early blight of tomatoes are also effective ways to handle the disease.

Keywords: early blight, Alternaria solani, potato dextrose agar, fungicide, bioagent

Introduction

According to Pritesh et al., 2011, Solanaceae family crop Lycopersicon esculentum Mill (tomato) is largely grown vegetable on the world. It is the most widely cultivated tropical vegetable crop on the planet according to Hadian et al., 2011. The attack of various bacterial, viral, and fungal diseases on tomato production resulted in a huge loss of production (Devi et al. 2017). Roy et al. and Rex et al. in 2019, Verma et al. in 2018 all are reported few common diseases of tomatoes such as early blight, wilt, late blight, damping off, bacterial wilt and TMV.

The pathogenic fungus Alternaria solani causes the most devastating disease, early blight and it causes significant losses in both quantity and quality of fruit yield (Tomazoni et al. 2016; S. Perveen et al. 2019). The pathogen can attack on all part of crop leaf, stem, fruit and all stage of crop growth during Rabi and Kharif season. Early blight has caused yield losses of up to 79 percent in India, the United States, Nigeria, and Canada (Sherf and MacNab, 1986; Datar and Mayee, 1981; Basu, 1974b; Gwary and Nahunnaro, 1998; Praveen, 2019). “Collar rot causes seedling losses in the field that range from 20% to 40%” (Sherf and MacNab, 1986). Disease formation takes place at 8°–32°C temperature with saturated humidity and under free moisture.

Different Alternaria species caused early blight (Kumar et al., 2008). At first, Alternaria solani was the most common source of EB. (Varma et al., 2007; Radhajeyalakshmi et al., 2009; Gomes et al., 2010; Al Hussaen, 2012; Bessadat et al., 2017) (83, 53, 24, 4, 8). “However, early blight on tomatoes is now considered to be caused by organisms such as A. alternata, A. grandis, and A. tomatophila” (Simmons 2000, 2007; Adhikari et al. 2017; Bessadat et al., 2017). “Early blight disease caused by A. tomatophila on tomato crops in Brazil” (Rodrigues et al. 2010). In Russia, A. tomatophila and A. solani were also discovered in early blight cases (Gannibal et al. 2014)

Alternaria solani involvement in early tomato blight has been recorded in Punjab, Rajasthan, Haryana, Uttar Pradesh, Jammu and Kashmir, and Madhya Pradesh in India (Deora et al. 2004; Loganathan et al. 2016). In Punjab, India, researchers discovered the highest disease severity in the district Tapa and the lowest disease severity of 8.2% in the Babakala District (Abhinandan et al. 2004).
Early blight can cause damping off, collar rot, stem cankers, leaf blight, and fruit rot, among other symptoms, at any stage of plant development. On the oldest leaves, the symptoms begin as small brownish to black lesions that grow to larger brownish to black lesions. Dark lesions appear in a circular pattern on the fruit which may cover a large area. On the stem, lesions are dark, sunken, and enlarge in a concentric pattern.

For the characteristics study of Alternaria solani and the mycelium growth, the pathogen was isolated in sterile glass Petri plates using an autoclave and incubated at 27±2 °C temperature on PDA as a medium.

Crop rotation and transplants with pathogen-free plant are two disease-control strategies [Madden et al. (1978); Sherf and MacNab (1986)] [40, 70]. Fungicide therapies are the most efficient prevention mechanisms in general, but they are not economically feasible in all parts of the world, and they may not be effective in epidemic-prone weather (Herriot et al., 1986) [27]. The disease is managed by using resistant cultivars (Madden et al., 1978; Shittenberg et al., 1995; Keinath et al., 1996) [40, 71, 33]. “Cultural, biological, chemical application, resistant varieties, and pathogen-free plant material are all options used for handling EB in tomato crops” (Sarfraz et al. reported in 2018) [60].

**Symptomatology**

The *Alternaria solani* infects of plants above ground, and the symptoms are known by a variety of names (Sherf and MacNab, 1986) [70]. This disease can harm all of the tomato's aerial components, including the stem, leaf, and fruit, at any stage of development (Blancard, 2012) [9]. Starting with the leaf and spreading to the stem and fruit, the pathogen will infect each and every part of the plant (Johnson et al., 2018) [30]. *A. solani* infection can cause collar rot, damping off, stem canker, and fruit rot if it is serious (Walker JC, 1952) [60]. Lesions on young tomato seedlings can totally girdle the stem, resulting in “collar rot,” which can cause decreased plant vigour or death (Gleason and Edmonds, 2006; Kemmitt, 2012) [23]. Stem cankers are a term used in older literature to describe collar rot and stem lesions (Barksdale and Stoner 1977) [6].

Premature defoliation is caused by *Alternaria solani*, which weakens plants and exposes fruit to sunscald damage (Sherf and MacNab, 1986) [70]. *Alternaria solani* is more likely to attack semi-ripe fruits than ripe fruits (Mehta et al., 1975) [42]. Fruits that are infected drop before reaching maturity, and those that do achieve maturity become unmarketable (Chaerani and Voorrips, 2006) [59]. Lesions on young tomato seedlings can totally girdle the stem, resulting in “collar rot,” which can cause decreased plant vigour or death (Gleason and Edmonds, 2006; Kemmitt, 2012) [23]. Stem cankers are a term used in older literature to describe collar rot and stem lesions (Barksdale and Stoner 1977) [6].

If the plants get older, the fungus begins on the older leaves and grows upward (Sherf and MacNab, 1986) [70]. The first symptoms that appear on leaves start with small black to brown lesions which are 1 to 2 mm in size and that enlarge and form concentric rings and that frequently surrounded by a yellow halo under ideal conditions. Dark pigmented concentric rings are common in lesions larger than 10 mm in diameter. Early blight is characterised by this so-called “bullseye” style lesion (Kemmitt, G., 2002) [34]. It has a dark, slightly sunken appearance that expands into a concentric ring formation on the plant's stem. Sunken stem lesions are most often present above the soil surface level on the stem (Mc Govern, 2011) [42]. A concentric ring developed on the fruit's attachment point between the stem and the fruit (Vorrips & Chaerani, 2006) [59]. The advanced lesion is dark brown in colour and coated in a black velvety mass of fungal spore.

**Etiology**

*Alternaria solani* is the cause of early tomato blight (Ell. And Mart). The fungus (*Alternaria solani*), which belongs to the Imperfecti (Deuteromycotina), genus *Alternaria*, class Hyphomycetes, and order Hyphales, was first described by Ellis and Martin (Agrios, 2005) [3]. The mycelium of *A. solani* are hyaline, separte and branched and letter on it become dark colour. No sexual reproduction and no sexual spore are present or germinate. It’s a large-spored fungus with simple conidiophores on which single conidia are produced (Neergaard, 1945) [46]. Muriform and beaked conidia are produce by conidiophore of *Alternaria solani* according to Neergaard (1945) [46] and Ellis and Gibson (1975) [20] with horizontal and vertical septation. Size of conidia is 12 -20 x 120 – 296 µm and there are 9-11 transvers septa on it. *Alternaria solani* has dark coloured melanized cells (Rotem, 1994) [62]. Conidia in the form of a club and it can be produced singly or in a chain. In the media, isolates on potato dextrose agar and other media contain yellowish to reddish diffusible pigments.

The morphology of *Alternaria solani* colonies varies, but they are usually effusive, greyish brown to black in colour, and have a cottony, felty, or velvety texture (Ellis & Gibson 1975) [20]. *Alternaria solani* cells are multinucleate, but the number of nuclei in different organs varies. Following nuclear separation of hyphal cells, multiple septation occurs, resulting in the division of elongated tip cells into several multinucleate cells (Alexander and King, 1969) [35]. Beaks range in length from half to double that of the conidium, 5–9 µm in diameter, straight or flexuous, separte, filiform and hyaline to pale brown (Ellis & Martin 1882; Rao 1964; Rao 1969) [19, 56, 57].

**Ecology and Spread**

Dry and warm weather with temperature 25 -30 °C of and high relative humidity more than 60% are required. Infection is more common during the rainy season. Early blight is possible under stress conditions involving a shortage of nitrogen in tomato crops (Soltanpour and Harrison, 1974) [74]. Areas with higher humidity, rainfall, and temperature have been reported to have early blight disease (Sahu et al., 2014) [66]. In the presence of free moisture and at temperatures of 28-30 °C, conidia germinate in around 40 minutes (Kemmitt, G., 2002) [34]. The pathogen can be found in soil and contaminated plant debris, as well as on Solanaceous weeds in the form of mycelium and conidia.

The dark pigmentation of the mycelium increases its tolerance of fungal spore. The morphology of *Alternaria solani* colonies varies, but they are usually effusive, greyish brown to black in colour, and have a cottony, felty, or velvety texture (Ellis & Gibson 1975) [20]. *Alternaria solani* cells are multinucleate, but the number of nuclei in different organs varies. Following nuclear separation of hyphal cells, multiple septation occurs, resulting in the division of elongated tip cells into several multinucleate cells (Alexander and King, 1969) [35]. Beaks range in length from half to double that of the conidium, 5–9 µm in diameter, straight or flexuous, separte, filiform and hyaline to pale brown (Ellis & Martin 1882; Rao 1964; Rao 1969) [19, 56, 57].
Disease development and Disease cycle

The fungus can be found in soil, alternative hosts, seed and plant debris in the form of mycelia or conidia, which can be used as primary inoculum sources during the winter. Conidia have a thick cell wall that aids the fungus in adapting to changing environmental conditions (Foolad et al., 2008) [21]. Germination Conidia in the presence of moisture at temperatures ranging from 8 to 32 degrees Celsius in cool and humid conditions (Jones, 1991; Kemmitt, 2002) [31, 34]. After effective conidia germination, which results in the formation of with one or even more germ tubes that enter the host through aspersion or wounds, stomata are formed by rising hyphae (Perez and Martinez, 1999; Agrios, 2005) [3]. The temperature must be Between 10° and 25° C for fungal hyphae to successfully penetrate the host plant (Sherf and MacNab, 1986) [30]. The pathogen releases a variety of enzymes that destroy cell wall of host, as well as a variety of toxins which kill cells of the host and make nutrients accessible to the pathogen released from of the host cells (Rotem, 1994) [62]. Depending on environmental factors, cultivar susceptibility, and leaf age, lesions appear 2–3 days after infection, and spores appear 3–5 days later (Jones, 1991; Sherf and MacNab, 1986) [31, 30]. In general, spore production necessitates a long period of wetness, but spores can also be formed in a cycle of dry and wet conditions. Conidiophores develop during rainy nights, and after a period of sunshine and dryness, spores appear 3 days after infection, and spores appear 3–5 days later (Jones, 1991; Sherf and MacNab, 1986) [31, 30]. The pathogen releases a variety of enzymes that destroy cell wall of host, as well as a variety of toxins which kill cells of the host and make nutrients accessible to the pathogen released from the host cells (Rotem, 1994) [62].

Since resistant cultivars may increase the duration between fungicide sprays while also preserving disease control, they could be the most cost-effective control strategy (Keinath et al. (1996); Madden et al. (1978); Shitienberg et al. (1995)) [33, 40, 71]. Chowdappa et al. said in 2013 [14], “the Rhizobacteriav bacillus subtilis is a plant growth promoter that improved systemic resistance in Tomato by producing peroxidase, polyphenol oxidase and superoxide dismutase, such type of growth hormone and defence-related enzymes.” Plants develop a systemic resistance to the toxin produced by pathogens. Various toxins were isolated from the pathogen by Suvarnalatha Devi et al., (2010) [18]. To defend itself from the invasion of Alternaria solani, researchers looked into the higher levels of phenol and peroxidase activity in infected plants (Shahbazi et al., 2010) [69]. Antifungal activity was also observed in extracts of many plants, such as Cinnamomum zeylanicum, Glycyrrhiza glabra, Hemidesmus indicus, Inula racemosa, Ferula foetida, Saussurea lappa, Syzygium aromaticum and Rubia cordifolia against the Alternaria solani pathogen levels ranging from medium to high (Yeole et al. 2014) [87]. In lab conditions, extracts of Eucalyptus camaldulensis, Azadirachta indica, Parthenium hysterophorus, and Datura stramonium reduced early blight (Raza et al. 2016) [58]. Similarly, Lantana camara has an influence on tomato early blight decline (Kumar and Bannwal 2016) [38]. Biological controls have also been reported as an alternate approach for the management of EB in many research papers. In tomato, Pseudomonas fluorescens, Pseudomonas aeruginosa, Pseudomonas putida, and Pseudomonas cepacia caused less disease severity and increased fruit weights than the control (Joseph et al. 2001) [32]. In addition, fungi such as Trichoderma viride (Sarkar et al. 2016) [68] and Trichoderma harzianum (Chowdappa et al., 2013) [14] have been shown to be effective in the treatment of tomato early blight diseases. In general, fungicide therapies are the most effective prevention methods, and they’re not cost-effective in all places of the globe, and they will not be effective in epidemic-prone weather (Herriot et al., 1986) [27]. Fungicides are
applied for the first time 1–2 days after transplantation, and then must be applied every 7–10 days for successful control, raising manufacturing costs and polluting the environment (Foolad et al. 2008; Kemmitt, G., 2002) [21, 34]. When disease pressure is high, fungicides often fail to function (Foolad et al. 2008) [21].

For controlling the disease, mancozeb and carbendazim were superior (Bais et al.). The fungicides tebuconazole and difenoconazole were the most effective in controlling tomato early blight (Rani S. 2017). Use of fungicide and their controlling efficacy are like Antracol (Propineb 70%) control 68.71%, Bavistin (Carbendazim 75%) control 72.30%, Score (Difenconazole 25%) control 74.89%, Sanit (Metiram 70%) control 64.12%, Kavach (Clorothalonil 75%) control 48.22% and Isignia (Pyraclostrobin 20%) control 60% [Kumar et al. (2017); Sahu et al. (2013); Chohan et al. (2015); Neesha et al. (2015) and Soni et al. (2015) ] [37, 66, 13, 47, 75]. Thiram (75%) was found to be the most effective at 5 thousand parts per million, with Thiram (TMTD 80%) and Arasan (50%) being the most effective at 10 thousand parts per million (Sahni and Singh, 1967) [64].

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