The Process of Early Blight Disease Development in Tomato

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Abstract
Early blight (EB) caused by several species of *Alterneria* and is the most drastic and devastating disease of tomato. Annual economic yield losses due to EB have been estimated about 79%. *Alternaria* are known only to reproduce asexually, but a highly-virulent isolate has the potential to overcome existing resistance genes. Currently, cultural practices and fungicide applications are employed for the management of EB due to the lack of strong resistant cultivars. This present review will deal with the current understanding of causal agents of EB of tomato, disease development and disease cycle.
Keywords: Early blight, *Alternaria solani*, *Solanum lycopersicum*, fungicides

Introduction
About (35±78%) decrease in yield have been observed in the countries like USA, Australia, Israel, the UK and India, (Datar and Mayee, 1982). The isolation of this necrotrophic pathogen from tomato plants were first reported in Greece (Saregiannis, 1936). However the severity of early blight disease increases during 1995-1998, that leads to complete defoliation of tomato plants, leads to heavy yield losses (*Solanum lycopersicum* L. (Peralta *et al*., 2005). As Tomato forms an important and richest source of vitamin A and C and an adequate amount of the antioxidant i.e. lycopene is produced that protects the body against cancer and heart disease (Bohm, Edge and Truscott, 2012). Tomato plants growing in regions with heavy rainfall, high moisture content and fairly high temperatures are more prone to *Alternaria infection*. Apart from the early blight symptoms on leaves, *Alternaria solani* causes collar rot, lesions on stem and fruit rot symptoms in tomato plants. Various effective control measures were taken to prevent this necrotrophic fungal infection (Early blight) in tomato plants by successful crop rotation and effective use of fungicides and use of resistant / disease free varieties (Madden *et al*., 1978; Sherf and MacNab, 1986).

Pathogen
The necrotrophic fungi (*Alternaria solani*) were first described by Ellis & Martin belongs to the fungi Imperfecti (Deuteromycotina), genus *Alternaria*, class Hyphomycetes and order Hyphales (Agrios, 2005). *A. solani*, *A. alternata* and *A. brassicicola* are the economically important plant pathogens that belong to genus *Alternaria*. It is the large spored fungal group bearing simple conidiophores on to which are born separate conidia singly (Neergaard, 1945). Alterneria possesses transverse and longitudinally septate conidia, the cells are multinucleated. Dark colored melanized cells are present in *Alterneria solani* (Rotem, 1994) that protect the fungus against harsh environmental conditions, providing resistance to microbes and hydrolytic enzymes. Many species of Alternaria are significant causes of necrotrophic diseases of crops. No sexual stages have been documented for most of the species including *A. solani.* Club-shaped conidia may be produced either individually or in a chain, depending on the species.

Disease development and disease cycle
The fungus makes its survival in contaminated seeds and in infected crop debris. For disease development in crops, infected debris and contaminated seeds serve as primary source for inoculum production. Numerous spores are

produced on the host tissue that is infected with fungus under wet and humid conditions. Spore diffusion from plant to plant takes place by wind and rain splashing. Warm temperatures and extended periods of leaf wetness favor the disease development in infected crops. Under stress conditions involving the lack of nitrogen to tomato crops, there are successful chances of early blight (Soltanpour and Harrison, 1974).

Disease cycle of *A. solani* on its host plant successfully begins under free moisture/humid conditions at a wide range of temperatures. After successful germination of conidia resulting in the formation of one or more germ tubes, that penetrate the host by means of aspersoria or enter through wounds, stomata by means of growing hyphae (Perez and Martinez, 1999; Agrios, 2005). Successful penetration of fungal hyphae into the host plant, the optimum temperature between 10° and 25°C is required (Sherf and MacNab, 1986).Various enzymes are released by the pathogen that degrade the host cell wall and number of toxins are produced that kill the host cells and makes nutrients available to the pathogen released from the host cells (Rotem, 1994). Infection lesions become visible after 2–3 days of infection and after 3–5 days spore production occurs, this relatively short disease cycle supports the polycyclic infection of Alternaria solani (Sherf and MacNab 1986), and three different stages are included in the life cycle of *A. solani* including soil, seed and air borne stages **(Figure 1).** Fungus is adapted to adverse environmental conditions by the thick wall of conidia (Foolad and Merk, 2008). Main hosts of *A. solani* are the crops belonging to Solanaceae family viz. tomato, potato, eggplant, and pepper (Neergaard 1945; Ellis and Gibson 1975).

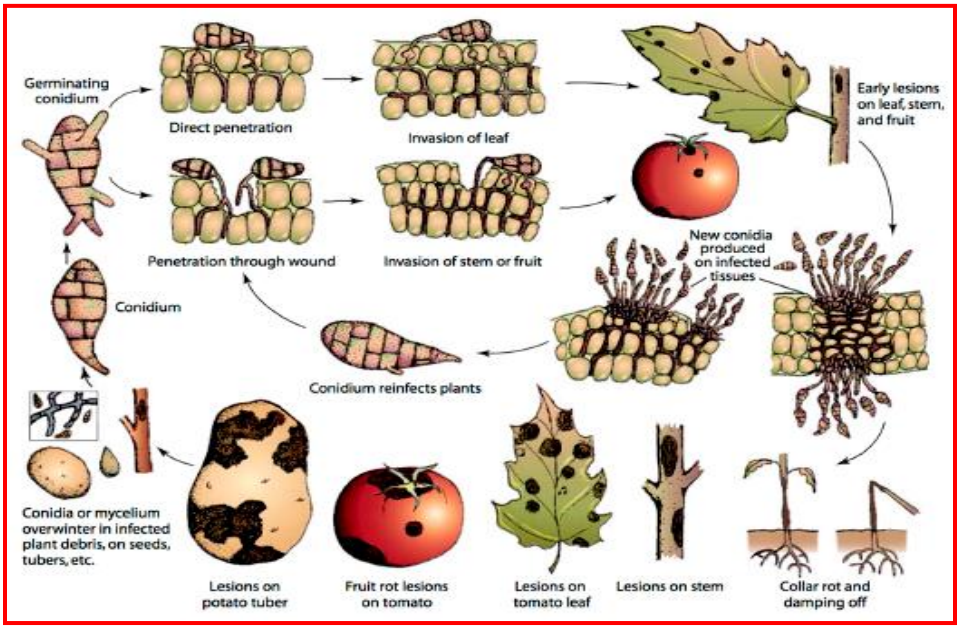

Figure 1. Infection process and disease development in tomato caused by caused by *Alternaria solani* (Agrios, 2005)

Disease symptoms

A. solani, cause severe infection to all above ground parts of plants and various names have been given for the different symptoms, which often leads to confusion (Sherf and MacNab 1986). Symptoms of *A. solani* infection on foliage is termed as early blight (EB), on fruits as fruit rot, on stems of seedlings as collar rot, and those on stems of adult plants as stem lesions (Walker, 1952). Older leaves are first infected and infection spread upward as the plants become older (Sherf and MacNab 1986). Initially, small dark and necrotic lesions appear on the older leaves and the lesions enlarge, form concentric rings that are often surrounded by a yellowish zone. Premature defoliation is caused by *A. solani* that weakens the plants and set the fruit to injury from sunscald (Sherf and MacNab 1986). Partial girdling of the stems of seedlings occurs and when the stem is completely girdled by the lesion can cause death of the plant. Susceptibility to *A. solani* of semi ripe fruits is more than ripened fruits (Mehta *et al*., 1975). The infected fruits fall drop before attaining maturity and the fruits became unmarketable that reaches to maturity (Chaerani and Voorrips, 2006).

Effect of leaf wetness duration

4. Effect of leaf wetness duration

Leaf wetness is having profound effect on the infection process in tomato plants. Susceptible tomato plants with fully expanded leaves were used for inoculation and sprayed with a conidial suspension (eight or nine fully expanded leaves). Wetness is maintained immediately and inoculated plants are placed in the controlled-environment chamber. After two weeks of inoculation, Necrotic lesions on the leaves seem to be visible. *A. solani* causes defoliation in inoculated plants. Inoculum concentration, leaf wetness duration and susceptible plant variety effects the early blight development in tomato plants.

It is important to reduce the chances of *A. solani* in tomato by using some fungicides that must be applied when environmental conditions favor their successful treatments **(Table 1).**

Table 1. List of some common fungicides used for the treatment of early blight in tomato (FRAC, 2018).

Active ingredient	Common name	Comments
Penthiopyrad	Fontelis	Very good
Boscalid	Endura, Lance WDG	Very good
Pyraclostrobin	Cabrio	Very good, but common insensitivity
Fenamidone	Reason	Very good, but common insensitivity
Azoxystrobin	Quadris	Very good, but common insensitivity
Cymoxanil and Famoxadone	Tanos	Good, but common insensitivity
Pyrimethanil	Scala	Good
Difenoconazole and Cyprodinil	Inspire Super	Good
Mancozeb and Zoxamide	Gavel	Good
Difenoconazole and Mandipropamid	Revus Top	Good
Cyprodinil and Fludioxonil	Switch	Good
Chlorothalonil	Bravo, Echo, Equus	Fair
Copper (copper hydroxide, copper oxychloride)	Kocide 2000, Champ Formula 2, Nu-Cop 50DF, C-O-C-S WDG	Fair

Concluding remarks

A wealth of information on the tomato and *A. solani* interaction is available. However, some important aspects need further attention. No conclusive evidence is available so far concerning the existence of physiological races. This should be studied using homozygous tester lines and isolates.

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