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Ralstonia solanacearum the Causal Agent of Ginger Bacterial Wilt - A Review

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A B S T R A C T

Bacterial wilt caused by *Ralstonia solanacearum* is a serious threat for ginger production worldwide. Among different biovars of it, biovar III race 4 found to be most destructive. This study was focused on bacterial wilt of ginger with special reference to pathogen biology, mode of infection, epidemiology, disease symptoms, host pathogen interaction and its economic importance.

Introduction

Ginger (*Zingiber officinale* Roscoe) an herbaceous perennial plant mainly used as spice and flavor agent for food. Due to the presence of volatile oils consisting of Zingerone, shogaols and gingerols, ginger got its characteristic fragrance and flavor. Ginger rhizome which is the consumed portion is the horizontal stem of the plant that sends out the roots. Due to the attack of various pathogenic diseases of viral, bacterial, fungal and nematode origin, yield of the ginger reduced drastically. Among the various diseases, Soft rot, Bacterial wilt, Phyllosticta leaf spot, Mosaic and Chlorotic flecks are important. Bacterial wilt is one f the most destructive disease of ginger caused by *Ralstonia solanacearum* previously known as *Pseudomonas solanacearum* (16) is the causal agent of bacterial wilt which belongs to the Proteobacteria, βsubdivision, *Ralstonia* group and the genus *Ralstonia* reported from all the ginger growing countries. Bacterial wilt of ginger, also known as "ginger blast" or "Mahali"/ "green wilt" caused by *Ralstonia*

pseudosolanacearum (10) formerly *Ralstonia solanacearum.* Due to wide host range and long term survival ability in soil, it is very difficult to manage the disease completely. A brief knowledge of pathogen, its mode of infection, host range, pathogen survival, disease symptoms, host pathogen interaction is needed to generate a suitable management strategy.

Causal organism

Ralstonia solanacearum grouped into Kingdom: Bacteria, Phlum: Proteobacteria, Class: Beta proteobacteria, Order: Burkholderiales, Family: Burkholderiacaea, Genus: *Ralstonia*, Species: *Solanacearum* (24). Erwin F. Smith first described R. solanacearum in 1896 as *Bacillus solanacearum* and transferred it under genus *Pseudomonas* in 1914. (Fates and Impacts of the Genetically Modified Plant Growth-Promoting Bacterium *Pseudomonas fluorescens*, 1Lotta Jäderlund). Again it has been transferred to *Burkholderia*, first, then to Ralstonia (25). Thomas reported bacterial wilt of ginger in 1941 for the first time from India.(1). *R. solanacearum* is a Gram negative, aerobic non-spore forming, non-capsulated, and nitrate-reducing, ammonia-forming, aerobic, rod-shaped bacterium with polar flagella. (26). As per the host ranges and geographic distributions *Ralstonia solanacearum (RS)* grouped into five races.

Race 1 attacks Bananas, Race 2 ornamental plants, Race 3 potato, Race 4 Ginger and Race 5 mulberries (8). Different biovars of *Ralstonia solanacearum* is named on the base of biochemical tests. Based on the ability to use or oxidize several hexose alcohols and disaccharides *R. solanacearum* is classified into five biovars, biovar I, II, III, IV and V.(5,23). Differentiation of *R.solanacearum* into 5 different biovars based in their ability to produce acid from three disaccharides (maltose, lactose and cellobiose) and oxidize three hexose alcohols (Mannitol, Sorbitol and Dulcitol) (5,15,36). From various reports by Hayward (1964), Heet *et al*., (1 983) and Kumar and Sharma (2004), biovar III oxidizes both disaccharides and hexose alcohols where as biovar I oxidize hexose alcohols but not disaccharides, biovar II oxidizes only disaccharides and biovar IV oxidizes only alcohols.(3,11,12). As per Pegg and Moffett both biovar III and IV of *Ralstonia solanacearum* can attack ginger but in Australia biovar IV were highly virulent and destructive, it may be due to cool climate of Australia.(37).Under Indian climatic condition when biovar III has inoculated artificially in stem rapid wilt in ginger has been observed within 5 to 7 days after infection and 7 to 10 days under soil inoculation of pathogen. (4). Cook 1989 used Restriction fragment length polymorphism (RFLP) to study the relationship of *R. solanacearum* strains representing the three races and five biovars. As per the physiological properties they share a common character such as chemo organotrophic nutrition, aerobic metabolism, absence of fermentation, absence of photosynthesis, inability to fix nitrogen and capacity for growth on a large amount of organic substrates. *Ralstonia* species bear similar phylogeny and chemotaxonomic properties but they are different in pathogenicity, host relationship and other phenotypic properties.(9)

Disease cycle and epidemiology

Being a soil borne pathogen *Ralstonia solanacearum* infects roots by invading and multiplying in the xylem vessels. After penetrating the xylem vessels the bacteria produces certain enzymes like lytic enzymes, the extra cellular polysaccharides, endoglucanases and endopolygalacturonases which help in colonization of the bacteria

further causing the rot and disintegration of the tissue which helps in rapid wilting of infected plants (13,38). The disease is severe in hot humid Southern states as well as in cold high altitude Eastern Himalayan state of Sikkim with a temperature ranges from 28 - 30^0 C and 7 -22 ^{0} C respectively.(35)As per Stevenson et.al.2001 optimum temperature for growth of the pathogen is 28 – 30^0 C except certain races, race 3 strains which are pathogenic to potato are able to grow at lower temperature.(7)In India biovar 3 is more virulent and frequent than biovar 4 due to its vast adaptability towards the varying environmental conditions and adjustability towards the different soil edafic factors. Although *Ralstonia slanacearum* is a soil borne vascular pathogen, but its survival ability is less in soil and it can survive for a longer period in roots of alternate hosts, undecayed infected plant tissues, volunteers tubers from precedent crops or in deeper layer of soil where they do not confront the antagonism from others soil microorganisms (17). It got the ability to colonize in the rhizospheres of non-host plants (17). Infected rhizome serve as the primary source of the pathogen spread.(34)In field secondary spread occurs through rain splashes and run off water.(34)Pathogen further spread easily through irrigation water, contaminated tools, farm implements, planting materials movement etc.(9) There is high correlation exist between nematode and *Ralstonia* population. Wounds created by nematode feeding on roots serve as entry for the bacteria, thus presence of root knot nematode and level of bacterial infection are highly correlated with each other.(27)

Disease symptoms and signs

Different disease symptoms develops after the colonization of *Ralstonia solanacearum* in the plant vascular system. “Green wilt”, in this, symptom occurs early in the disease cycle and yellowing of leaf start. Leaves of infected green ginger roll and curl due to water stress caused by bacteria blocking the water conducting vascular system of the ginger stems. Yellowing of leaves followed by necrotic brown(39). This type of similar yellowing symptom also occur in another disease, known as *Fusarium* yellows caused by the fungus *Fusarium oxysporum* f. sp. *zingiberi*, however in *Fusarial* wilt rapid wilt don’t occur, which normally found in bacterial wilt. (6). Plant dries very rapidly and the foliage becomes yellow brown in 3-4 days. Infected shoots which become soft and completely rotted break off easily from the underground rhizome at the collar region and infected rhizome shows grayish brown discoloration with water soaked central part which becomes soft and rot in advance stage.

Symptoms also include downward curling bending of leaves due to loss of turgidity, growth of adventitious roots in the stems and development of narrow dark stripes in the infected vascular bundles beneath the epidermis due to multiplication of the bacteria in the vascular system that clog the vessels(20,30). The most prominent symptom which distinguish bacterial wilt from other diseases is the exudation of slimy creamy bacterial ooze on the surface of a cut made in the rhizome or on the above ground stem of an infected plant when suspended in a glass or beaker of water.

Genetic based characterization of *Ralstonia solanacearum*

Molecular based identification of the pathogen is very useful which based on the selective amplification of the 16S ribosomal rRNA gene through Polymerase Chain Reaction (PCR) where the presence of *R. solanacearum* is confirmed by the appearance of a band of targeted amplified DNA on an electrophoresis gel. (14) According to

Restriction Fragment Length Polymorphism (RFLP) there are two major geographical origins of the strains, American origin consisting of biovars 1 and 2 and Asian origin consisting of biovars 3,4 and 5,which is correlate to the finding of Cook *et al.,* (1989) who assessed the diversity of the pathogen using hypersensitive response and pathogenicity (hrp) genes as probes(2). Kumar *et al.,* (2004) studied that the diversity of *R. solanacearum* causing bacterial wilt on ginger and other hosts in India, the use of REP-PCR (Repetitive Extragenic Palindromic Polymerize chain reaction) and RFLP-PCR (Restriction Fragment Length polymorphism-polymerize chain reaction)as molecular tool could cluster the highly pathogenic isolate in a cluster at 100% similarity coefficient in conformity with their host origin and biovar (4).

Host Pathogen Interaction Mechanisms

Genin *et al*.(1992) studied that the virulence of the pathogen *R. solanacearum* on host species and induction of hypersensitive response in nonhosts is influenced by *hrp* (hypersensitive response and pathogenicity) gene cluster (31). The *hrpB* gene encodes for components of the type III secretion pathway(TTSP) which play a key role in pathogenesis of many bacterial pathogens of plants and animals(32). Arlat *et al.,* 1992 found that inactivation of one of the more than 20 *hrp* genes cease the ability of the pathogen to cause disease and multiply in susceptible plants, as well as loss of the ability to cause hypersensitive defense response in resistant plants(33).In *Ralstonia solanacearum* type III secretion requires the production of an Hrp pilus. The *hrpY* gene encodes this structural protein (31). *HrpB* and the *TTS* genes were induced in response to physical contact of bacteria with plant ceIls or cell wall fragments.

Economic importance

Due to its wide distribution in tropical, subtropical and temperate regions of the world *Ralstonia solanacearum* causes severe wilt in many crops(11,12).Assefa *et al.,* 2015 and Henok and Kurabachew during 2016 reviewed that *Ralstonia solanacearum* cause severe yield losses on different crops in different parts of the globe. For instance, 50-100% of potato in Kenya, 88% of tomato in Uganda, 70% of potato in India, 95% of tobacco in South Carolina and 100% on Pepper in Ethiopia. Tariku et.al.(2016) also reported that a severe outbreak of ginger wilt disease identified as bacterial wilt caused by *R. solanacearum* with disease incidence of 80-100% in Ethiopia which is similar to the report done by Habetwold *et al*., (2015) who reported 80-100% incidence of bacterial wilt in ginger growing areas(21,22,28,29).In field the range of disease is from 10-40% but it can also destroy the crop completely(19).In China prevalence of bacterial wilt reduces the yield by 20-30%.(18).

In conclusion due to its destructive nature Bacterial wilt considered as the major constraint for the cultivation of ginger. Among different biovars, biovar III is found most destructive in India. Among different races, race 4 attacks ginger. Hypersensitive response and pathogenicity (hrp) gene is required for virulence on host species and induction of hypersensitive response in nonhosts. Disease is both soil and water borne and survives between crops on infected seed rhizome. The severity of Ralstonia is influenced by different factors related to environment and soil. Among the different symptoms and signs of the disease rapid wilting and oozing are most prominent. Disease caused by *Ralstonia solanacearum* which is distributed throughout the world and causes loss in different countries that varies from 10-100%.

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Emend the descriptions of *Ralstonia solanacearum* and *Ralstonia syzygii* and reclassify current *R. syzygii* strains as *Ralstonia syzygii* subsp. *syzygii* subsp. nov., *R. solanacearum phylotype* IV strains as *Ralstonia syzygii* subsp. *indonesiensis* subsp. nov., banana blood disease bacterium strains as *Ralstonia syzygii* subsp. *Celebesensis* subsp. nov. and *R. solanacearum* phylotype I and III strains as *Ralstonia pseudosolanacearum* sp. nov. Int J Syst Evol Microbiol 64:3087–3103.

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