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Article

Infection Mechanism of Pathogenic Exduate by Soil-Borne Fungal Pathogens : A Review

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The processes to determine the composition, dynamics, and activity of infection mechanisms by the rhizosphere microflora have attracted the interest of scientists from multiple disciplines although considerable progress of the infection pathways and plant-pathogen interactions by soil borne fungal pathogens have been made. Soilborne pathogens are confined within a three-dimensional matrix of mineral soil particles, pores, organic matter in various stages of decomposition and a biological component. Among the physical and chemical properties of soils soil texture and matric water potential may be the two most important factors that determine spread exudates by soil borne fungal pathogens, based on the size of the soil pores. Pathogenic invasion of plant roots involves complex molecular mechanisms which occur in the diffuse interface between the root and the soil created by root exudates. The initial infection by soilborne pathogens can be caused by enzymes which breakdown cell wall layers to penetrate the plant cell wall for the fungus. However, the fate and mobility of the exudates are less well understood. Therefore, it needs to develop methods to control disease caused by enzymes produced by the soilborne pathogens by verifying many other possible pathways and mechanisms of infection processes occurring in soils.

Key words: Infection Mechanism, Pathogenic Exduate, Soil-Borne Diseases

Introduction

Disease suppressive soils have been recognized for over 100 years and the mechanisms by which disease suppression is brought about has been the subject of study for nearly four decades. Soils suppressive to soilborne plant diseases have been defined as those in which disease development is minimal even in the presence of a virulent pathogen and susceptible plant host (Bruehl, 1987).

Soils which contain diverse communities of microscopic organisms are capable of damaging plants. The organisms to be plant pathogens which are highly plant specific interaction between a soil organism in soils include fungi, bacteria, viruses, nematodes and protozoa which adversely affect plant growth and health as major influential force (Lynch, 1990; Raaijmakers, 2001).

Maintenance of plant health is a vital component of all crop production systems. Intensive production in agriculture increases the frequencies for diseases to develop compared with undisturbed natural ecosystems. Historically, soil-borne fungal plant pathogens have proven difficult to manage. Disease management in reducing or controlling pathogen populations or pathogen activity in soil has relied, for the most part, upon crop rotation or cultivation practices (Reeleder, 2001). However, pesticides have not been effective nor has plant resistance been available in many cases .

There are four main groups of plant pathogens (Agrios, 2005). More than 8,000 species of fungi among approximately 100,000 species of fungi described in the literature, living symbiotically on or in the roots of many plants, are known to cause diseases of plants and most plants are susceptible to some fungal pathogens (Agrios, 1997). Among those plant pathogens, only fungi (true fungi and oomycetes) and nematodes are major players that adversely affect plant growth and health in the soil whereas only a few groups of bacteria which require a wound or natural opening to penetrate into the plant and cause infection are considered to be soilborne because nonspore forming bacteria cannot survive well in soil for long periods (Brown et al., 1995; Campbell, 1996).

The soilborne diseases caused by fungal pathogens which persist in the soil matrix as a reservoir of inoculum

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of the pathogens and in residues on the soil surface may not be noticed until the above-ground (foliar) parts of the plant are affected severely showing symptoms such as stunting, wilting, chlorosis and death (Agrios, 1997). However, these diseases which can survive for long periods in the absence of the normal crop host are often very difficult to diagnose accurately.

Considerable progress has been made in recent years in the development of our understanding of the nature of plant-pathogen interactions. Much of the effort has focused on interactions that conform to the classical 'gene-for-gene' interactions originally proposed by Flor about 50 years ago, and has centred on foliar diseases (Ellis et al., 2000). However, the lack of knowledge is likely to be due in part to the difficulty of investigating interactions that occur in the soil, and also because of the genetic intractability of many root-infecting organisms. This overview attempts to highlight some key aspects of resistance mechanisms that are relevant to the control of soilborne pathogens.

Understanding the processes that determine the composition, dynamics, and activity of the rhizosphere microflora has attracted the interest of scientists from multiple disciplines (Raaijmakers et al., 2009). In this article, we reviewed the properties and secretion pathway of pathogenic exduate that causes diseases by soil-borne fungal pathogens which are the most important soilborne pathogens. Specific attention is given to mechanisms, both offensive and defensive, involved in the interactions between soilborne pathogens and plants in soil system.

Key features of soilborne plant pathogens Soil-borne pathogens are among the more important components of the soil flora in agro-ecosystems. In most agricultural ecosystems, soilborne plant pathogens which can be a major limitation in the production of marketable yields are adapted to grow and survive in the bulk soil, but the rhizosphere where microflora and microfauna, can interact with the pathogen and establishes a parasitic relationship is the infection court (Bruehl, 1987).

Plants mount resistance to pathogens using a variety of mechanisms that can target specific or multiple pathogens. These mechanisms include the production of antimicrobial metabolites, inactivation of pathogen-derived toxins and lytic enzymes, and triggering of host-defense responses by pathogen-or host-derived elicitors (Okubara and Paulitz, 2005). Interactions between roots and certain non-pathogenic soil-borne microbes can trigger a systemic resistance to root and foliar pathogens in the host (Fig. 1), which is known as induced systemic resistance (ISR) (Chen et al.,

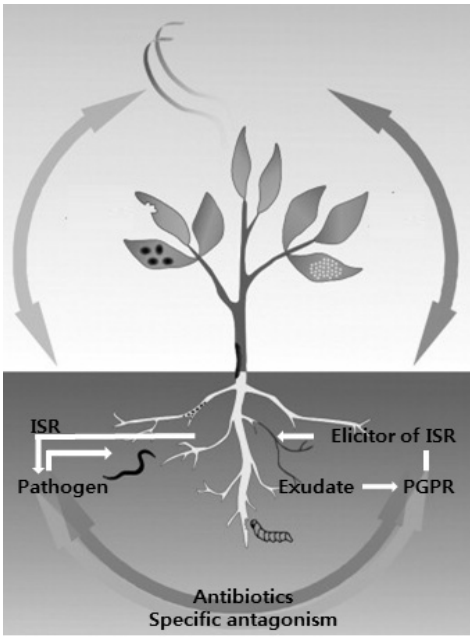

Fig. 1. Interactions between pathogens and root in soil through many of biotic and abiotic signals which are still unknown. ISR(induced systemic resistance) and PGPR(Plant growth promoting rhizosphere).

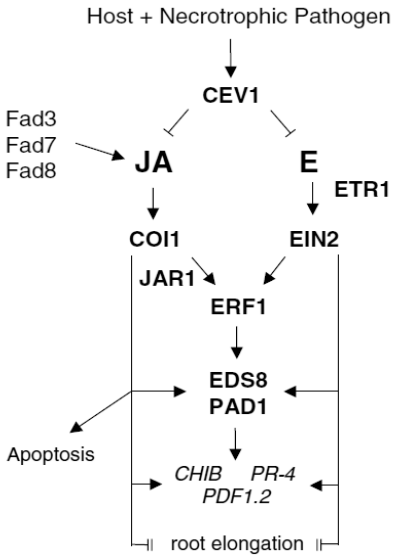

Fig. 2. Proposed jasmonate (JA)-and ethylene (E)-mediated host responses to necrotrophic pathogens. Fatty acid desaturases (Fad3 Fad7 Fad8) and cellulose synthase (Cev1) are required for the wild type effects of JA and E on tolerance to such pathogens. Increased flux through either the JA or E pathway enhances pathogen tolerance, while modulating root growth and morphology. (After Okubara and Paulitz, 2005).

1999; Pieterse and van Loon, 1999).

For leaf-pathogen interactions only a small number of defense pathways and resistance mechanisms have been reported so far in roots (Fig. 2). In response to challenge by necrotrophic fungal pathogens, roots typically exhibit the plant growth regulators jasmonic acid (JA) and ethylene (E)-dependent defenses (Devoto and Turner, 2003; Turner et al., 2002; Wang et al., 2002)

Root pathogens and the infection process Soilborne pathogens are confined within the soil, a three-dimensional matrix of mineral soil particles, pores, organic matter in various stages of decomposition, and a biological component. Thus, the spread of soilborne pathogens over time and space is more limited. Soil texture and matric soil water potential are probably the two most important factors that determine spread, based on the size of the soil pores (Raaijmakers et al., 2009). Monocyclic diseases considered to be caused by soil-borne pathogens with the limited travel distance of inoculum in soils are not much plant-to-plant spread in a single season whereas polycyclic foliar diseases spread from plant to plant by wind or rain in an exponential fashion in a single season.

The most significant pathogens of the roots of crops are either fungi or filamentous bacteria of the genus Streptomyces (Loria et al., 2003). Fungi are eukaryotic, filamentous, multicellular, heterotrophic organisms that produce a network of hyphae called the mycelium and absorb nutrients from the surrounding substrate (Alexopoulos et al., 1996). Almost all soilborne fungi are necrotrophic, meaning they kill host tissue with enzymes, peptide elicitors, or toxins in advance of the hyphae and do not require a living cell to obtain nutrients (Raaijmakers et al., 2009). Necrotrophic root rotting soil-borne pathogenic fungi which has a thick-walled resistant spore or structure can survive in the soil in a dormant, quiescent state, when environmental conditions are not suitable for growth, or when the host is not present. They must also withstand microbial degradation and lysis, parasitism and predation, constituting an important trophic level in the soil ecosystem (Okubara and Paulitz, 2005). Most necrotrophic pathogens with a wide host range are generalists. The biotrophic root pathogens with narrow host ranges, occurring on the above ground and portions of the plants, are few and require a living cell to obtain nutrients.

Pathogens can also enter roots at points damaged by soil animals or other disturbances, as well as attachment of the fungus to the root. Judelson and Blanco (2005) proposed course of infection by Phytophthora infestans (Fig. 3). As shown in Fig. 3, the fungal hyphae or zoospore contacts the surface of the root, there may be a recognition event on the part of both the fungus and plant.

Most soilborne fungi attack young, juvenile roots as opposed to secondary woody roots. A specialized group of pathogens that cause wilt diseases (e.g. Fusarium oxysporum, Verticillium dahliae) can penetrate through the endodermis into the vascular tissue and move up the

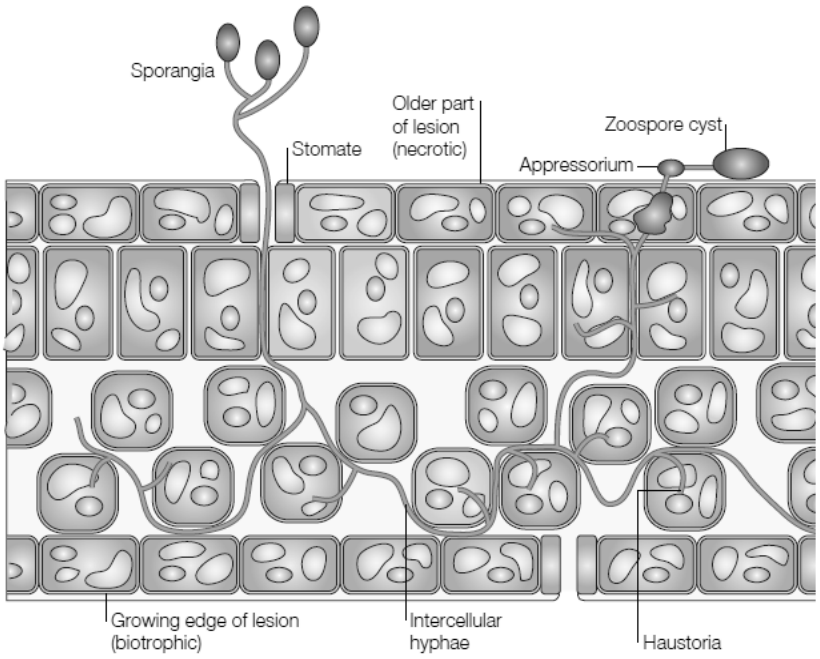

Fig. 3. Course of infection by *Phytophthora infestans* (Judelson and Blanco, 2005).

xylem to above-ground parts of the plant, impeding the flow of water. A plant may react to the presence of a pathogen by producing chemical or physical barriers that prevent entry or confine potential pathogens to a small part of the root (Okubara and Paulitz, 2005). Pathogenic invasion of plant roots involves complex molecular mechanisms which occur in the diffuse interface between the root and the soil created by root exudates and sloughed off root cells (Judelson and Blanco. 2005). The fungal pathogens block the movement of water in the xylem by producing mycelia, spores, and high-molecular-weight polysaccharides in the xylem vessels, while degrading plant cell walls and releasing pectic substances and other polymers that can clog the vascular system and reduce its water-transport efficiency to the leaves (Agrios, 2005).

The initial infection by soilborne pathogens that produce enzymes such as pectinases and other pectic enzymes; hemicellulases, cellulases, and proteinases that dissolve cell wall layers can be the stimulus for the fungus to penetrate the plant cell wall, resulting in allowing the hyphae to enter. Hemicelluloses and pectins in the cell wall are modified and broken down by a variety of enzymes produced by the soilborne pathogens, with multiple enzymes working cooperatively (Cantu et al., 2008). Glucanases and related enzymes hydrolyze the backbone of hemicelluloses. Xylosidases and related enzymes remove the side branches from xyloglucan (particularly xyloglucan fragments, or oligomers). Transglycosylases cut and join hemicelluloses together (Rose and Bennett, 1999).

The interactions and mechanisms of resistance to necrotrophic root pathogens are less well understood although a number of pathogenic and parasitic organisms can attack roots (Okubara and Paulitz, 2005). Fungi, having mechanisms of chemotaxis and chemotropism and sensing root exudates such as sugars, amino acids, organic acids and fatty acids, can move or grow in response to gradients of these compounds (Deacon and Donaldson, 1993; Donaldson and Deacon, 1993; Ruttledge and Nelson, 1997; Tyler, 2002). Electrostatic charge may also be an important sensory stimulus for swimming zoospores (Van West et al., 2002). Electrical fields with variable polarity between plant species, contributing to the specificity of electrotax, is become established within the rhizosphere due to normal ion flux across the surface membranes of plant roots (van West, et. al., 2002).

Chemoattraction is augmented by electrotaxis which functions as a mechanism for directing zoospores to roots. Regardless of pathogens, zoospores from Phytophthora species and other oomycetes such as Pythium were attracted by nonspecific chemo-attractants such as amino acids plants exude (van West, et. al., 2002). Plants also emit specific attractants including the isoflavones daidzen and genistein. Such attractants form concentration gradients from both root tips and wound sites, which presumably represent optimal sites for infection (Morris et al., 1992). Auto-attraction (or autoaggregation), occurring in response to calcium released by encysting zoospores, is a phenomenon in which zoospores move towards each other, resulting in which might increase the frequency of successful infections. However, one study has suggested that the autoattractant is species-specific (Tyler, 2002; Reid et al., 1995).

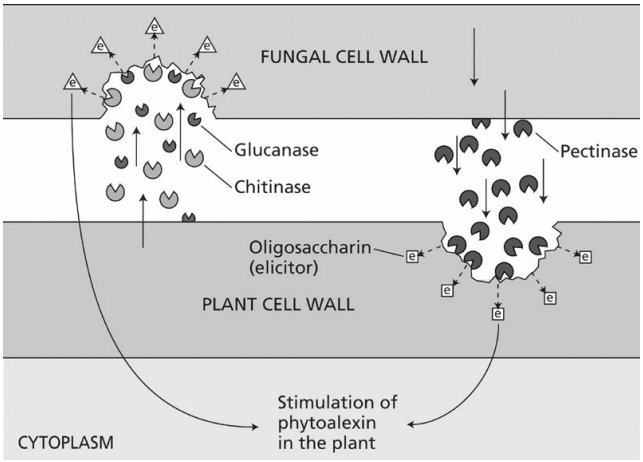

Fig. 4. Scheme for the production of oligosaccharins during fungal or oomycete invasion of plant cells. Enzymes secreted by the plant, such as chitinase and glucanase, attack the fungal or oomycete wall, releasing oligosaccharins that elicit the production of defense compounds (phytoalexins) in the plant. (After Brett and Waldron, 1996).

The hypersensitive response and apoptotic cell death, two oxidative processes that limit biotrophic pathogens, generally act to exacerbate disease symptoms induced by necrotrophic organisms. When plant cells are wounded or treated with certain low molecular weight elicitors, they activate a defense response that results in the production of high concentrations of hydrogen peroxide, superoxide radicals, and other active oxygen species in the cell wall. This "oxidative burst" is part of a defense response against invading pathogens (Brisson et al., 1994; Otte et al., 2001). Degradation of cell walls can result in the production of biologically active fragments, called oligosaccharins, that may be involved in natural developmental responses and in defense responses. Some of the reported physiological and developmental effects of oligosaccharins include stimulation of phytoalexin synthesis, oxidative bursts, ethylene synthesis, membrane depolarization, changes in cytoplasmic calcium, induced synthesis of pathogen-related proteins such as chitinase and glucanase, other systemic and local "wound" signals, and alterations in the growth and morphogenesis of isolated tissue samples.

Although pathogenesis-related proteins can be expressed in roots during pathogen challenge, salicylic acid has not been implicated in root-mediated interactions (Okubara and Paulitz, 2005). Jasmonic acid and ethylene have been found to mediate parallel as well as synergistic pathways that confer partial tolerance to necrotrophic pathogens, as well as induced systemic resistance to root and foliar pathogens.

Discussion

Soilborne pathogens are confined within the soil and the spread of soilborne pathogens over time and space is more limited. Soils suppressive to soilborne plant diseases have been defined as those in which disease development is minimal even in the presence of a virulent pathogen and susceptible plant host. Soil texture and water (matric) potential are probably the two most important factors that determine spread, based on the size of the soil pores. The initial infection by soilborne pathogens that produce enzymes such as pectinases and other pectic enzymes; hemicellulases, cellulases, and proteinases that dissolve cell wall layers can be the stimulus for the fungus to penetrate the plant cell wall. Pathogens can also enter roots at points damaged by soil animals or other disturbances, as well as attachment of the fungus to the root. Plants also emit specific attractants. Electrical fields with variable polarity between plant species, contributing to the specificity of electrotax, is become established within the rhizosphere due to normal ion flux across the surface membranes of plant roots. In general the biocontrol of soilborne disease relied on the control of soilborne pathogens although there are many other pathways and mechanisms to cause disease by enzymes produced by the pathogens. Therefore, we need to develop the methods to control these enzymes to cause diseases instead of controlling soilborne pathogens by identifying the fate and mobility of the enzymes released into the rhizosphere.

References

Agrios, G.N. 1997. Plant pathology (4th Ed.). Academic Press, California.

Agrios, G.N. 2005. Plant pathology, 5th Ed.. Elsevier, New York.

Alexopoulos, C.J., C.W. Mims, and M. Blackwell. 1996. Introductory Mycology. 4th Ed., John Wiley and Sons Inc., USA.

Brett, C.T. and K.W. Waldron. 1996. Physiology and Biochemistry of Plant Cell Walls. Springer. p.31-276.

Brisson, A., A. Olofsson, P. Ringler, M. Schmutz, and S. Stoylova. 1994. Two-dimensional crystallization of proteins on planar lipid films and structure determination by electron crystallography. Biol. Cell. 80:221-228.

Brown, D.J.F., W.M. Robertson, and D.L. Trudgill. 1995. Transmission of viruses by plant nematodes. Ann. Rev. Phytopathol. 33:223- 249.

Bruehl, G.W. 1987 Soilborne plant pathogens. Macmillan, NY.

Campbell, R.N. 1996. Fungal transmission of plant viruses. Ann. Rev. Phytopathol. 34:87-108.

Chen, C., R.R. Bélanger, N. Benhamou, and T.C. Paulitz. 1999. Role of salicylic acid in systemic resistance induced by Pseudomonas spp. against Pythium aphanidermatum in cucumber roots. Eur. J. Plant Pathol. 105:477-486.

Cantu, D., A.R. Vicente, J.M. Labavitch, A.B. Bennett1, and L.T. Ann. 2008. Powell1Strangers in the matrix: plant cell walls and pathogen susceptibility. Trends Plant Sci. 13:11.

Deacon, J.W. and S.P. Donaldson. 1993. Molecular recognition in the homing responses of zoosporic fungi, with special reference to Pythium and Phytophthora. Mycol. Res. 97:1153 -1171.

Devoto, A. and J.G. Turner. 2003. Regulation of jasmonate-mediated plant responses in Arabidopsis Ann. Bot. 92:329-337.

Donaldson, S.P. and J.W. Deacon. 1993. Effects of amino acids and sugars on zoospore taxis, encystment and cyst germination in Pythium aphanidermatum (Edson) Fitzp., P. Catenulatum Matthews and P. dissotocum Drechs. New Phytol. 123:289-295.

Ellis, R.J., T.M.T., Wison, M.J. Bailey. 2000. Identification of conserved traits in fluorescent pseudomonads with antifungal

activity. Environ. Microbiol. 2:274-284.
Judelson, H.S. and B.H Blanco. 2005. The spores of phytophthora: weapons of the plant destroyer. Nat. Rev. Microbiol. 3:47-58.
Loria, R., J. Coombs, M. Yoshida, J. Kers, and R. Bukhalid. 2003. A paucity of bacterial root diseases: Streptomyces. Physiol. Mol. Plant Pathol. 62:65-72.
Lynch, J. 1990. The rhizosphere. Wiley, London, UK. p458.
Morris, P.F. and E.W.B. Ward. 1992. Chemoattraction of zoospores of the soybean pathogen Phytophthora sojae by isoflavones. Physiol. Mol. Plant Pathol. 40:17-22.
Okubara, P.A. and T.C. Paulitz. 2005. Root defense responses to fungal pathogens: A molecular perspective. Plant and Soil. 274:215-226.
Otte, S., W. Belden, M. Heidtman, J. Liu, O. Jensen, and C. Barlowe. 2001. Erv41p and Erv46p. New components of COPII vesicles involved in transport between the ER and Golgi complex. J. Cell Biol. 152:503-518.
Pieterse, C.M.J. and van L.L. Loon. 1999. Salicylic acid-independent plant defence pathways. Trends Plant Sci. 4:52 -58.
Raaijmakers, J.M. 2001. Rhizosphere and rhizosphere competence. In: Maloy OC, Murray TD (eds) Encyclopedia of plant pathology. Wiley, USA, pp.859-860.
Raaijmakers, J.M., T.C. Paulitz, C. Steinberg, and C. Alabouvette, Y.M. Loccoz. 2009. The rhizosphere: a playground and battlefield for soilborne pathogens and beneficial microorganisms Plant Soil. 321:341-361.
Reeleder, R.D. and W. Hickey. 2001. A protocol for the extraction of DNA from thick-walled fungal spores residing in soil. Can. J. Plant Pathol. 23: 205.
Reid, B., B.M. Morris, and N.A.R. Gow. 1995. Calcium-dependent, genus-specific, autoaggregation of zoospores of phytopathogenic fungi. Exp. Mycol. 19:202-213.
Rose, J.C. and A.B. Bennett. 1999. Cooperative disassembly of the cellulose- xyloglucan network of plant cell walls: Parallels between cell expansion and fruit ripening. Trends in Plant Sci. 4:176-183.
Ruttledge, T.R. and E.B. Nelson. 1997 Extracted fatty acids from Gossypium hirsutum stimulatory to the seed-rotting fungus, Pythium ultimum. Phytochemistry 46:77-82.
Turner, J.G., C. Ellis, and A. Devoto. 2002. The jasmonate signal pathway. Plant Cell. 14:S153-S164.
Tyler, B.M. 2002. Molecular basis of recognition between Phytophthora pathogens and their hosts. Annu. Rev. Phytopathol. 40:137-167.
Wang, K.L.C., H. Li, and J.R.Ecker. 2002. Ethylene biosynthesis and signaling networks. Plant Cell 14:S131-S151.
West, P. van, B.M. Morris, B. Reid, A.A. Appiah, M.C. Osborne, T.A. Campbell, S.J. Shepherd, and N.A.R. Gow. 2002. Oomycete plant pathogens use electric fields to target roots. Mol. Plant Microbe Interact. 15:790-798.