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Fungal pathogenicity of plants: Molecular approach

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ABSTRACT

Fungi are eukaryotic, carbon-heterotrophic microorganisms. Plants, like humans and other animals, also get sick, exhibit disease symptoms, and die. What are the determinants of fungal pathogenicity towards plants? A number of fungal mechanisms and molecules have been shown to contribute to fungal pathogenicity or virulence, understood as the capacity to cause damage in a host, in absolute or relative terms. Among them, cell wall degrading proteins, inhibitory proteins and toxins are included. Small secreted proteins and Pheromone also play important and even decisive roles in these processes. This is a short review makes an overview and summarizes the contribution of the most recent knowledge of molecules helping in pathogenesis of fungal biology.

Keywords: Disease symptoms, Fungal pathogenicity, Toxins, Small secreted proteins and Pheromone.

INTRODUCTION

Fungi are eukaryotic, carbon-heterotrophic microorganisms. To satisfy their need for organic nutrients, most fungal species live a saprophytic lifestyle. It has been estimated that the fungal kingdom contains more than 1.5 million species, but only around 100,000 have so far been described, with yeast, mold, and mushroom being the most familiar [1].

The interaction between and plant phytopathogenic fungi are complex. Virulence is a complex interrelationship between the infecting organism and the host. Pathogenesis involves interaction (and sometimes modification) of factors on both sides. This is particularly true of fungal

pathogenesis. Fungi are important pathogens of plants cause more significant yield losses than bacteria or viruses. Numerous fungi are devastating to human and plant pathogens that are a serious threat to agricultural industry and human health [2, 3]. Plants, like humans and other animals, also get sick, exhibit disease symptoms, and die. Plant diseases are caused by environmental stress, genetic or physiological disorders and infectious agents including viroids, viruses, bacteria and fungi. Inspite of strong efforts to develop and introduce new fungicides and resistant plant varieties, losses due to fungal diseases especially in agriculture are a growing stimulus for basic research in this field. A small minority, however, has acquired the capability to develop on living plants, often causing disease in the host.

The factors influencing the interaction of pathogenic fungi and their hosts have been a major research topic in the fungal community in recent years. Microbiologists have been attracted to this field of research because of the need for identification of the agents causing infectious diseases in economically important crops. These detailed investigations have been fuelled by the necessity to develop new strategies for the control of these economically highly important organisms. To be a successful pathogen, a fungus has to pass through a well-defined series of physical and biochemical steps which together constitute the disease cycle. What are the determinants of fungal pathogenicity towards plants? Active defence mechanisms may be countered by fungi in several different ways, including suppression of particular signal transduction or gene expression processes in plant cells, protection against antifungal compounds or enzymes, or, in the case of necrotrophic pathogens, induction of host cell death. Until the rapid rise of opportunistic fungal infections in humans, pathogenicity mechanisms in plant pathogens were better understood than those in animal pathogens. Pathogenesis involves the interaction of two partners with input from the environment, a concept described as the "disease triangle" in plant pathology. A more recent concept developed for animal pathogens is the "damage-response" framework which emphasizes that the outcome of an interaction is determined by the amount of damage incurred by the host. [4]In plant–fungus interactions, establishing a successful infection requires intricate signal exchanges at the plant surface and the intercellular space interface [4].

A number of fungal mechanisms and molecules have been shown to contribute to fungal pathogenicity or virulence, understood as the capacity to cause damage in a host, in absolute or relative terms. Among them, cell wall degrading proteins, inhibitory proteins [5], and enzymes involved in the synthesis of toxins [6, 7, 8, 9] are included.

The mechanisms of fungal pathogenesis are much less-well understood than are those of bacterial pathogens. This review work makes an overview and summarizes the contribution of the most recent knowledge of molecules helping in pathogenesis of fungi biology.

Molecular signals:

Because of the complex nature of the host-fungus interaction, there are few factors that are absolutely required for fungal virulence. However, some properties are frequently associated with pathogenesis across the fungal kingdom, and some have been found to be important for specific pathogens. In the early phases of infection, reception and transduction of external signals play a key role in triggering developmental and morphogenetic processes preceding penetration

of the host epidermis [10, 11, 12]. The role of signal transduction in pathogenesis has been studied investigated in phytopathogens fungi in particular, the involvement of heterotrimeric G proteins and MAPK signaling pathway [13, 14].

Signal transduction, morphogenesis and manipulation of the host plant are facilitated through a diversity of extracellular vector molecules and morphogenic proteins. Such molecules are secreted into the intercellular interface between the pathogen and the plant or delivered inside the host cell [15].

Toxins elicitors' importance in host defenses:

Plant pathogenic fungi utilize multiple strategies for infection of host plants. Pathogen-produced factors, called elicitors [16], that condition defence responses in plants are not usually thought of as 'toxins' when their roles in pathogenesis are considered.

The toxic activity of certain elicitors is extremely specific, i.e. an elicitor may affect only a single genotype of a single plant species. Among them is the production of toxins. Toxins produced by plant pathogenic fungi differ in structure as well as in their role in disease and mode of action [17]. Toxins play diverse roles in disease, from impacting symptom expression and disease progress to being absolutely required for pathogenesis. Some toxins are directly toxic, killing cells and allowing for infection of dead cells. Others interfere with induction of defense responses or induce programmed cell death-mediated defense responses in order to generate necrosis required for pathogenesis [18]. All of the identified perylenequinone toxins are produced by members of the Ascomycota, the largest phylum within the fungal kingdom. The similarity of the fungal perylenequinone structureto hypericin led to investigations of the fungal compounds as photosensitizers [19, 20] play diverse roles as defense compounds in plants, pathogenesis determinants in fungi, and as molecules responsible for photomovement of protozoans [21].

Proteins role:

Small secreted protein scan play important and even decisive roles in these processes. Here, I only consider proteins of less than 200 amino acid residues, which excludes hydrolases that are involved in cell wall breakdown and/or nutrient acquisition. Various approaches have led to the identification of small, secreted proteins or their genes from plant pathogenic fungi. Different methods follow for finding proteins where as [22] the most straightforward of these is isolation of the protein from the extracellular fluids of infected plant tissue, followed by protein sequencing by Edman degradation or tandem mass spectrometry. In this way, Avr4, Avr4E, Avr9 and five Ecps (''Extracellular proteins'') from *Cladosporium fulvum* were identified, as well as Six1 (''Secreted in Xylem 1'') from *Fusarium oxysporum*, and two peptides from *Uromycesvignae.* CgDN3 protein from *Colletotrichum gloeosporioides* required for pathogenicity on *Stylosanthes* [23]. ToxA belongs to *Pyrenophora tritici-repentis* required for pathogenicity on wheat [24, 25, 26, 27].

Small proteins secreted by plant pathogenic fungi in their hosts have been implicated in disease symptom development. The discovery of 12 distinct gene clusters comprising nearly 20% of the secreted proteins of *U. maydis*, and the finding that deletion of entire clusters affects virulence in

five cases support the importance of extracellular proteins and indicates that focusing on secreted proteins promises to be instrumental in increasing our understanding of fungal disease strategies. *Rhynchosporium secalis*, a pathogen of barley, produces a family of proteins called NIP (necrosis-inducing proteins), which causes nonspecific necrosis in barley as well and plants via stimulation of plasma membrane ATPase [28].One of these proteins (NIP1) also causes accumulation of pathogenesis-related proteins associated with plants resistance to pathogens [29].

Pheromone role:

The dimorphic maize (Zea mays) smut fungus *Ustilago maydis* is amenable to molecular genetics and cell biological methods and thus became an excellent model system for fungal plant pathogenicity [30, 31, 32, 33]. Pathogenic development is initiated by amating reaction that involves two compatible haploid yeast-like cells, which recognize each other at the plant surface through a pheromone (mfa1/2)/pheromone receptor (Pra1/2) system [34].

CONCLUSION

All these approaches will reveal an enormous amount of information on the molecules and strategies necessary for pathogenesis. Still have to find how many pathogenicity mechanisms does a fungus have? Knowledge of the pathogenic determining and that of virulence factors [35, 36] is crucial for designing effective crop protection strategies, including the development of resistant plant genotypes through classical plant breeding [37] or genetic engineering [38], fungicides [39] , or the use of biological control strategies [40].

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REFERENCES

[1] D. L .Hawksworth, *Mycological Research.*, **1991**, 95 (6), 641–655,
[2] V.H . Tournas, *Crit Rev Microbiol.*, **2005**, 31(1), 33-44.
[3] R.P. Tuori, T.J. Wolpert , L.M. Ciuffetti. *Mol. Plant-Microbe Interact.*, **2000**, 13, 456–464.
[4] G. San-Blas, L.R. Travassos, B.C. Fries, D.L. Goldman, A., Casadevall, A.K. Carmona, T.F. Barros, R. Puccia, M.K. Hostetter, S.G.Shanks, **2000**, *Med Mycol.*, 38 (Suppl 1), 79-86.
[5] M. Hahn , K. Mendgen. Signal and nutrient exchange at biotrophic plant–fungus interfaces, *Curr Opin Plant Biol.*, **2001**, 4, 322–327
[6] A. Casadevall ,L. A. Pirofsk, *Nat. Rev. Microbiol*, **2003** , 1, 17-24.
[7] H. P. Van Esse, J. W. Van't Klooster , M. D. Bolton, *Plant Cell.*, **2008**, 20 (7), 1948–1963.
[8] T. L. Friesen, J. D. Faris, P. S.Solomon , R. P. Oliver, *Cellular Microbiology.*, **2008**, 10 (7):1421–1428.
[9] X. Gao , M. V. Kolomiets, *Toxin Reviews.*, **2009**, 28 (2-3), 79–88.
[10] K. S. Kim, J. Y. Min, M. B. Dickman, *Molecular Plant-Microbe Interactions.*, 2008, 21 (5), 605–612.

[11] C. B. Lawrence, T. K. Mitchell, K. D. Craven, Y. Cho, R. A. Cramer, K. H. Kim, *Plant Pathology Journal.*, **2008**, 24(2), 101–111,
[12] J.A Lucas, *Plant Pathol.*, **2004** , 53, 679–691.
[13] N.D. Read, L.J. Kellock, T.J. Collins, A.M. Gundlach, *Planta.*, **1997**, 202, 163–170.
[14] S.L. Tucker, N.J. Talbot, *Annu Rev Phytopathol.*, **2001**, 39, 385–417.
[15] M. Bolker, *Fungal Gent Biol.*, **1998**, 25, 143-156.
[16] N .Lee, C.A. D'Souza, J.W. Kronstad , *Annu Rev Phytopathol.*, **2003**, 41, 399-427.
[17] N.T Keen, *Annual. Rev.Genet.*, **1990**, 24, 447-463.
[18] H.W. Knoche, J.P. Duvick, (Pegg, G.F. and Ayres, P.G., Eds.), **1987**, pp. 158–191. Cambridge University Press, Cambridge.
[19] T.J. Wolpert, L.D. Dunkle, L.M. Ciuffetti. *Annu. Rev. Phytopathol.*, **2002**, 40, 251–285.
[20] M.E Daub, *Phytopathology.*, **1982**, 72, 370–374.
[21] S. Yamazaki, A.Okube, Y. Akiyama, K. Fuwa, *Agric. Biol. Chem.*, **1975**, 39, 287–288.
[22] S. Boddi, C. Comparini, R. Calamassi, L. Pazzagli, G. Cappugi, A. Scala, *Plant J.*, **2004**, 24, 275–283.
[23] S. Stephenson, J. Hatfield, A.G. Rusu, D.J. Maclean, J.M. Manners, Mol. Plant- *Microbe Interact*., **2000**, 13, 929–941.
[24] G.M. Ballance, L. Lamari, C.C. Bernier, *Physiol. Mol. Plant Pathol.*, **1989**, 35, 203–213.
[25] L. Lamari, G.M. Ballance, N.P. Orolaza, R. Kowatsch, *Phytopathology.*, **1995**, 85, 333–338.
[26] L.M. Ciuffetti, R.P .Tuori, J.M. Gaventa, *Plant Cell.*, **1997**, 9, 135–144.
[27] J.R.Heitz, K.R. Downum, Eds., Light-Activated Pest Control. ACS Symposium Series, Vol. 616, **1995**, American Chemical Society, Washington, DC.
[28] L. Wevelsiep, E Rupping, W. Knogge , *Plnat Physiol.*, **1993**, 101, 293-301.
[29] M., Rohe, A. Gierlich, H. Hermann, M. Hahn, B. Schmidt, S. Rosahl, W. Knogge, *EMBO J.*, **1995**, 14, 4168-4177.
[30] M. Bolker, *Microbiology*., **2001**, 147, 1395–1401.
[31] A.D. Martinez-Espinoza, M.D. Garcia-Pedrajas, S.E. Gold, *Fungal Genet. Biol*., **2002**, 35, 1–20.
[32] R. Kahmann, J. Kamper, *New Phytol.*, **2004**, 164, 31–42.
[33] M.D. Garcia-Pedrajas, S.E. Gold, *Adv. Appl. Microbiol*., **2004**, 56, 263–290.
[34] M., Bolker, M. Urban, R. Kahmann, *Cell.*, **1992**, 68, 441–450.
[35] B. Pariaud, V. Ravigné, F. Halkett, H. Goyeau, J. Carlier, C. Lannou, *Plant Pathology.*, **2009**, 58(3), 409–424.
[36] D. Parker, M. Beckmann, H. Zubair, *Plant Journal*., **2009**, 59(5), 723–737.
[37] S . McCouch, , *PLoS Biology.*, **2004**, 2(10), Article 347,
[38] Y. Yang, H., Zhang, G. Li, W. Li, X. Wang, F. Song, *Plant Biotechnology Journal*., **2009**, 7(8): 763–777.
[39] B. S. Kim, B. K. Hwang, *Journal of Phytopathology.*, **2007**, 155 (11-12), 641–653,
[40] G. Berg. *Applied Microbiology and Biotechnology.* **2009**, 84(1), 11–18.