B·DEBATE International Center for Scientific Debate BARCELONA

PIERRE DE WIT CV

SPEAKER AT:

THE DEATH OF PLANT CELLS.

FROM PROTEASES TO FIELD APPLICATIONS

October, 2nd and 3rd , 2013, Barcelona

Pierre De Wit, Laboratory of Phytopathology, Wageningen University, Wageningen, The Netherlands

Pierre De Wit received his PhD at Wageningen University studying the pathosystem Cladosporium fulvum-tomato. After his PhD he did spend a sabbatical year in the USA at the department of Phytopathology of KSU, Lexington Kentucky, USA. In 1982 he became assistant, professor at the Laboratory of Phytopathology of Wageningen University, associate professor in 1986 and full professor and head of the Laboratory in 1990. His research is focused on fungal effectors from C. fulvum and related Dothideomycete fungi like Mycospaerella fijiensis and Dothistroma septosporum, and effector-triggered immunity responses in tomato and tobacco. His group discovered the biological functions of several fungal effectors, including Avr2, a cysteine protease inhibitor, and Avr4, a chitin-binding protein that also induced Cf-2 and Cf-4-mediated hypersensitive responses, respectively. Recently, he discovered effectors with enzymatic functions including a-tomatinase and endo-arabinase. In 1999 he became member of the Royal Netherlands Academy of Sciences and in 2008 he became Royal Netherlands Academy Professor. Pierre de Wit received the Noel T. Keen award and the Emile Christian Hansen award for his his pioneering work on fungal effectors.

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The Role of Plant and Fungal proteases in the Pathosystem Cladosporium fulvum-tomato

The tomato pathogen Cladosporium fulvum is a biotroph that secretes multiple effectors in the apoplast of its host plant during infection. One of them is a cysteine protease inhibitor, Avr2, that interacts with cysteine-protease Rcr3 and triggers Cf-2-dependent defense responses including a hypersensitive response. Rcr3 is guarded by the Cf-2 resistance protein. The Cf-2 protein also mediates disease resistance to the pathogenic root nematode Globodera rostochiensis. The Cf-2-mediated nematode resistance is triggered by the venom allergen-like effector protein, Gr-VAP1,that is secreted by G. rostochiensis and perturbs Rcr3. Interaction of both fungal Avr2 and nematode Gr-VAP1 with Rcr3 from Solanum pimpinellifolium (pim) triggers a Cf-2-mediated hypersensitive response. In the absence of Cf-2, Rcr3 increases susceptibility of tomato plants to G. rostochiensis, thus showing its role as a virulence target. Monitoring of host proteins like Rcr3 targeted by multiple pathogens broadens the spectrum of disease resistances mediated by single resistance genes like Cf-2. C. fulvum also secretes chitin-binding proteins like Avr4 and Ecp6, of which the first protects fungi against the deleterious effects of plant chitinases and the second scavenges chitin fragments preventing them from eliciting chitin receptor-mediated basal defense. Recently, we found evidence that plant pathogenic fungi also secrete protease that can inactivate chitinases by removing their chitin-binding domains. This might be the last line of defense in case the first two are not sufficient. The discovery of a novel effector Avr5, a pattern-associated molecular pattern, an endo-arabinase, and an exoglucanase releasing a damage-associated molecular pattern will alsobe briefly discussed.

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