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Review

Molecular and genetic aspects of controlling the soilborne necrotrophic pathogens *Rhizoctonia* and *Pythium*

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ABSTRACT

The soilborne necrotrophic pathogens *Rhizoctonia* and *Pythium* infect a wide range of crops in the US and worldwide. These pathogens pose challenges to growers because the diseases they cause are not adequately controlled by fungicides, rotation or, for many hosts, natural genetic resistance. Although a combination of management practices are likely to be required for control of *Rhizoctonia* and *Pythium*, genetic resistance remains a key missing component. This review discusses the recent deployment of introduced genes and genome-based information for control of *Rhizoctonia*, with emphasis on three pathosystems: *Rhizoctonia solani* AG8 and wheat, *R. solani* AG1-IA and rice, and *R. solani* AG3 or AG4 and potato. Molecular mechanisms underlying disease suppression will be addressed, if appropriate. Although less is known about genes and factors suppressive to *Pythium*, pathogen genomics and biological control studies are providing useful leads to effectors and antifungal factors. Prospects for resistance to *Rhizoctonia* and *Pythium* spp. will continue to improve with growing knowledge of pathogenicity strategies, host defense gene action relative to the pathogen infection process, and the role of environmental factors on pathogen–host interactions.

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Contents

1. Introduction

The primary objective of this review is to summarize recent molecular knowledge about enhanced host resistance to the soilborne necrotrophic pathogens *Rhizoctonia solani* Kühn and *Pythium ultimum* Trow. Both of these pathogens cause yield-limiting diseases of seedlings that are poorly controlled by existing management practices. The hosts wheat (*Triticum aestivum* L.) and rice (*Oryza sativa* L.) have been the focus of recent genomic and genetic approaches for enhanced resistance to *Rhizoctonia*

Abbreviations: E, ethylene; ESI-qTOF MS, electrospray ionization-quadrupole time-of-flight mass spectrometry; FT-ICR/MS, Fourier transform-ion cyclotron resonance/mass spectrometry; JA, jasmonic acid; SNP, single-nucleotide polymorphism; QTL, quantitative trait loci.

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spp.; fewer but equally important advances in *Solanum tuberosum* L. (potato)-*Rhizoctonia* interactions also are included. Together, these hosts represent three of the four major staple crops of the world. Knowledge about enhancing resistance against *Pythium* spp. in these crops is more limited. However, as a globally occurring soilborne pathogen, it is given consideration here. Finally, challenges in the development of resistance against *Rhizoctonia* and *Pythium* spp. are discussed.

Soilborne necrotrophic pathogens such as *Rhizoctonia* and *Pythium* are difficult to control, owing to their longevity in the soil, ability to outgrow or evade plant defenses and the logistics, cost and efficacy of fungicide applications. In many cases, the pathogens cause disease on more than one host species, confounding rotation measures. Resistance to fungicides continues to be a concern [1], and there are no available effective sources of native genetic resistance to these pathogens in rice, wheat or potato [2–7]. Annual losses to the wheat and barley industries due to soilborne fungal pathogens amount to over $100 million in the state of Washington, and $ billions worldwide. The rice industry is estimated to sustain up to 20% yield loss in India and 50% in Asia [8] solely due to sheath blight, caused by *R. solani*. The $4.0 billion US potato industry of Washington and Idaho sustains estimated annual losses of 19–30% due to Rhizoctonia damping off, stem and root canker and black scurf of tubers [9]. Calculated losses in the US and worldwide were conservatively estimated at $320 million in 2012. Based on global potato production totals of $49.7 billion in 2011 (FAOSTAT, http://faostat.fao.org/), about $1 billion in annual losses was sustained by the world potato market. Molecular and genetic approaches offer means of improving disease resistance, a sustainable resource, against *Rhizoctonia* and *Pythium*, and of realizing some of the yield potential lost to these pathogens in the US and worldwide.

1.1. Pathogens and disease symptoms

R. solani anastomosis group 8 (AG8) is the principal causal agent of Rhizoctonia root rot and bare patch of wheat and barley. The diseases were first reported in Australia in the 1920s and 1930s (reviewed in 10). The pathogen primarily affects seedlings, and has a broad host range, attacking both monocot and dicot crops. Root girdling and breakage impair the support and uptake functions of the root, and severe root loss can result in yield loss or plant death [10]. In the field, plants exhibit patches of stunting or seedling loss, especially in no-till production systems [10–12]. The pathogen survives in surface residue, and, in the Pacific Northwest, below ground as infectious propagules in the form of thick-walled monilioid hyphae or as condensed hyphal masses called sclerotia. *Rhizoctonia oryzae* Ryker & Gooch (teleomorph *Waitea circinata* Warcup & Talbot) often coincides with *R. solani* AG8 in the Pacific Northwest [13]. Pre-emergence damping-off, or failure of the seedling to emerge from the soil due to necrosis of emerging radicles and death of germinating seedlings, has been observed with severe infections of *R. oryzae* Ryker & Gooch [14,15]. *R. oryzae* also causes uneven stand height in cereals [16] and is pathogenic to pea (*Pisum sativum* L.), chickpea (*Cicer arietinum* L.), lentil (*Lens culinaris* Medik) and canola [17,18].

R. solani AG1-IA, the causal agent of sheath blight of rice, has become a problem in the production of semi-dwarf rice cultivars at sown at high density [19]. The sheath blight pathogen also has a broad host-range, infecting maize (*Zea mays* L.), soybean (*Glycine max* [L.] Merr.) and other Fabaceae [20]. Infectious propagules persist in the soil as sclerotia that spread to above-ground organs during flooding [21]. Infected rice plants become stunted, and necrotic lesions develop on the leaf sheath, blade and culm [19].

R. solani AG3 and AG4 cause symptoms on and yield losses of potato [9]. Necrotic lesions form on roots, stolons and underground stems, causing damping-off, and root and stem canker [22,23]. Sclerotia that form on the surface of young tubers cause black scurf, problematic for the potato seed industry [24].

Pythium diseases of wheat, rice and potato are caused by multiple species, and all three crops are generally susceptible. *Pythium* spp. are able to survive in the soil as thick-walled oospores [25] in the absence of host residue, making them particularly long-lived in the field. Pythium root rot and damping-off of wheat seedlings are associated with *P. ultimum* and *P. irregulare* group I Buisman, two of the most virulent species to small-grain cereals in the Pacific Northwest, USA [26]. The absence of visible lesions on infected roots, and the characteristic loss of fine roots and root hairs that only can be observed with a microscope, make the disease difficult to diagnose. However, pruning of seminal roots and reduced length of the first true leaf are observable in wheat seedlings. In the field, plants show stunting, reduced emergence, and overall reduction in seedling vigor [26]. Rice is infected by *P. irregulare*, *P. arrhenomanes* and *P. graminicola*, which result in uneven stand height [27,28]. Potato tubers undergo postharvest damage by multiple species of *Pythium*, causal agents of Pythium leak disease. Pathogens enter through wounds, and induce watery, blackened necrotic zones inside the tuber. *P. ultimum* and *P. aphanidermatum* are the primary causal agents in cool and warm temperatures, respectively [29]. In addition, *Pythium sylvaticum*, also a wheat pathogen [26], causes seedling damping-off, root stunting and a type of dry rot of tubers [29]. Infected seed pieces fail to emerge or are delayed in emergence, resulting in poor stands.

1.2. Modes of host penetration

Members of the genus *Rhizoctonia* are true fungi, whereas *Pythium* spp. are Oomycetes, more closely related to the brown algae than to the fungi [30–32]. Nevertheless common elements in hyphal morphology, pathogen ingress and nutrient acquisition are shared between necrotrophic fungi and Oomycetes. *Rhizoctonia*, *Pythium* and other necrotrophic plant pathogens acquire nutrients from dying or dead cells of host plants; however, these pathogens likely have a very brief biotrophic phase during which they recognize specific hosts and initiate parasitic relationships. Hyphae of *Rhizoctonia* spp. grow in close association with host surfaces, especially along junctions between epidermal cells, forming branches that can give rise to hyphal aggregates known as infection cushions [33,34]. *R. solani* that infect aerial portions of the plant, including *R. solani* AG1-IA on rice and *R. solani* AG3 on potato sprouts [35,36], gain entry into rice and potato tissues *via* infection cushions or lobate appressoria that penetrate the cuticle, or *via* stomata or wounds [22,33,34,37,38]. Hyphae grow both inter- and intracellularly through the tissues of most host species [39]. Host cell death is exacerbated by toxins [21] and by cutinases, chitinases and other cell wall-degrading enzymes [38,39]. Root-infecting *R. solani* also have been found to produce infection cushions, as documented for *Gossypium hirsutum* L. (cotton) and *Phaseolus lunatus* L. (lima bean) [37]. On cereal roots, hyphae of *R. solani* AG8 penetrate the root epidermis and cause browning, necrosis and death of the outer cortical cells, and leave the stele intact [10]. Host necrotic death can spread across the root diameter, resulting in breakage.

Seeds and young roots of germinating plants also are susceptible to attack by *Pythium*, which respond to host root exudates [25]. *Pythium* zoospores adhere to host tissue *via* pathogen-derived glycoproteins and host root mucilage. The zoospores become encased (encysted) in plant cell wall polysaccharides; the cysts produce germ tubes that infect host tissues within minutes to hours, depending upon the host cultivar and pathogen [25]. On wheat roots, *P. ultimum* causes loss of lateral roots and root hairs without visible necrotic lesions, indicating a different tissue specificity and mode of infection compared to *R. solani* AG8.

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