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Host-Pathogen Dynamics of *Puccinia graminis* in Small Grains and Grasses

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Abstract: -

Stem rust is a significant disease affecting wheat, barley, oats, rye, and several key grasses such as timothy, tall fescue, and perennial ryegrass. The causal agent, *Puccinia graminis*, is essentially an obligate biotrophic fungus. While it can be grown under artificial conditions, the process is difficult growth is slow, and successive culturing often leads to abnormal ploidy levels and a loss of infectivity. *P. graminis* follows a heteroecious life cycle, requiring two distinct hosts to complete its development. It produces five different spore stages during its asexual cycle on grass hosts, while its sexual cycle begins with resting spores and completes on an alternate host, typically barberry (*Berberis* spp.). Although *Berberis* species exhibit limited variation in resistance or susceptibility, small grain crops and their respective *P. graminis* pathotypes show complex gene-for-gene interactions, with diverse resistance and susceptibility traits matched by corresponding virulence or avirulence factors in the pathogen.

ECONOMIC IMPORTANCE:

Stem rust was once considered the most devastating disease of wheat across nearly all wheat-producing regions worldwide. Historical records, including references in the Bible, describe cereal rust and smut epidemics as divine punishment, and archaeological evidence of stem rust-infected wheat has been found in Bronze Age Israel. The Roman festival of Robigalia, initiated by Numa Pompilius, was held to protect cereal crops through offerings to rust deities. However, Aristotle and Theophrastus later linked rust outbreaks to warm, wet weather conditions. Stem rust thrives in warm climates and can cause severe losses across vast areas. Even healthy-looking wheat fields just weeks before harvest can be destroyed if spores are carried in from heavily infected regions, leading to

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rapid disease escalation. The infection disrupts nutrient transport to grain heads, resulting in shriveled kernels. Additionally, weakened stems are more prone to lodging, compounding the yield loss. Major 20th-century epidemics in Europe occurred in 1932 and 1951, with Scandinavian wheat yield losses reaching 9–33% in 1951, and losses of 5–20% recorded in eastern and central Europe in 1932. In Australia, outbreaks were intermittent and mostly in warmer regions like Queensland and northern New South Wales, but a widespread epidemic occurred in southern states in 1974. In India, stem rust mainly affects the warmer southern regions and rarely causes major losses in the northwestern wheat belt, except during unusually warm winters. In China, it is a problem mainly in the spring wheat areas of the north and Inner Mongolia, with severe epidemics in 1948, 1951, 1952, and 1956 due to favorable weather. In the United States, stem rust historically impacted spring wheat in the northern Great Plains. Between 1920 and 1960, yield losses exceeded 10% in eight years and topped 20% in five of those. In 1935, the worst outbreak saw more than half the spring wheat crop in North Dakota and Minnesota lost. Although no major epidemics have occurred in the U.S. or Canada since 1974, the fungus remains a threat. Today's success in managing stem rust relies on continuous breeding efforts to stack race-specific resistance genes in new wheat varieties. Additionally, the eradication of barberry the alternate host in Europe and North America has played a critical role by reducing genetic diversity and slowing the emergence of new virulent *P. graminis* strains.

LIFE CYCLE

Puccinia graminis, the causal agent of stem rust, evolved as a full-cycle, heteroecious rust fungus, requiring two distinct hosts to complete its life cycle. In temperate regions, it produces thick-walled, two-celled teliospores on grass hosts near the end of their growing season. Initially dikaryotic (containing two haploid nuclei), teliospores undergo nuclear fusion (karyogamy) as they mature. Unlike many other fungal spores, teliospores remain attached to their stalks within the pustule and do not disperse. Instead, they overwinter in infected plant debris and germinate in the spring, coinciding with bud break and new leaf growth on the alternate hosts *Berberis* or *Mahonia* species. During dormancy, meiosis is initiated after karyogamy but halts at the diplonema stage. In spring, the teliospore gives rise to a promycelium (or basidium), where meiosis resumes and results in four haploid nuclei, each separated by septa. Each cell forms a sterigma from which a haploid basidiospore develops. Mitosis later produces two identical haploid nuclei in each mature basidiospore. These basidiospores are released

and carried by wind to infect *Berberis* species, especially *B. vulgaris*. Older barberry leaves become resistant to infection, likely due to thicker cuticles that impede germ tube penetration. Upon successful infection, flask-shaped structures called pycnia form on the upper leaf surface. Within pycnia, small haploid pycniospores are produced and exuded in sugary nectar, which attracts insects and facilitates spore movement. Pycniospores function as male gametes, while the flexuous hyphae extending from pycnia act as female structures. Two mating types (+ and –), regulated by a single gene, enable sexual reproduction when compatible pycniospores and flexuous hyphae interact. A mating signal protein complex in the nectar of the opposite mating type triggers a surface cap on pycniospores, allowing recognition and fusion. Once fusion occurs, the pycniospore nucleus travels through the hyphae to the protoaecium, establishing a dikaryotic state. Growth resumes, and cup-shaped aecia form beneath the pycnium. These structures break through the lower epidermis, releasing dikaryotic aeciospores that can infect cereal hosts, but not barberry. Upon infecting grasses like wheat, aeciospores form dense hyphal mats beneath the epidermis. These mats produce sporophores that generate dikaryotic urediniospores, which emerge as pustules (uredinia). Urediniospores, dispersed by wind, repeatedly infect grass tissues, primarily stems and leaf sheaths. Eventually, uredinia stop producing urediniospores and instead form teliospores, becoming telia. In regions like the central U.S., where barberry has been mostly eradicated, *P. graminis* survives year-round in the uredinial stage. It moves from winter wheat in the southern Great Plains to spring wheat in the north, with volunteer wheat serving as a "green bridge" to maintain the fungus between seasons. A similar cycle sustains the pathogen in Australia and India. In Europe, where stem rust is no longer a significant issue, the pathogen's ability to survive harsh winters or dry summers remains unclear.

INFECTION PROCESS

Most studies on the infection mechanisms of *Puccinia graminis* have concentrated on the uredinial stage, as this is the most economically damaging phase of the fungus's life cycle. Infection by *P. graminis* begins when urediniospores land on a moist surface of cereal or grass plants and germinate. A germ tube emerges from the spore and typically grows perpendicular to the long axis of epidermal cells, an orientation that increases the likelihood of encountering a stoma. For effective growth and orientation, the germ tube must remain closely attached to the plant's waxy cuticle. Once it reaches a stoma, the germ tube halts elongation and forms an

appressorium directly over the stomatal opening. At this stage, the two nuclei that migrated from the urediniospore into the germ tube move into the appressorium, where they divide by mitosis and are then separated from the rest of the germ tube by a septum. Germination and appressorium formation typically take place at night when dew is present. Growth temporarily pauses after the appressorium forms and resumes at dawn likely due to changes in photosynthesis and stomatal CO_2 concentration rather than direct light effects. Notably, Yirgou and Caldwell (1968) demonstrated that high CO_2 levels inhibit penetration, while penetration occurs effectively in CO_2-free air regardless of light conditions. Penetration also fails on non-photosynthetic albino or etiolated wheat in normal CO_2 levels. When conditions are favorable, a slender penetration peg emerges from the underside of the appressorium and enters the plant through the stomatal opening, reaching the substomatal cavity. There, it forms an elongated substomatal vesicle where a second synchronized round of mitosis occurs. From each end of this vesicle, infection hyphae grow, each receiving a pair of nuclei. Upon contacting a host cell, the infection hypha develops a haustorial mother cell, which is separated from the hypha by a septum and typically contains 2–4 nuclei. The haustorial mother cell forms a narrow penetration peg that enters the host cell wall using enzymatic degradation and mechanical pressure. This cell has a uniquely structured wall with two additional layers absent in standard intercellular hyphae. Once inside the host cell wall, the haustorial peg expands to form a haustorium within the host's periplasmic space. While this structure is developing, the infection hypha can resume growth and initiate the formation of additional haustoria in nearby cells. This process may continue until the energy reserves of the original urediniospore are depleted. At this point, the fungus relies entirely on nutrient uptake via the haustoria to sustain development and avoid triggering host defenses. Molecular studies support the critical role of haustoria in nutrient acquisition. In *Uromyces fabae* (broad bean rust), genes encoding an amino acid transporter and a hexose transporter are specifically activated in haustoria. Additionally, Struck et al. (1996) reported high levels of H^+-ATPase activity in haustorial membranes, essential for active transport of nutrients. These findings align with earlier observations by Jäger and Reisener (1969), who noted that *P. graminis* f. sp. *tritici* relies primarily on host-derived amino acids and has limited capacity for synthesizing them independently.

EVOLUTION AND SUBSPECIFIC VARIATION

Leppik (1959) hypothesized that the ancestral forms of *Puccinia graminis* likely evolved on plants from the Berberidaceae family in central Asia. As grassland ecosystems expanded throughout the northern hemisphere during the Tertiary period, these rust fungi spread along with them. Some rust fungi that originally infected Berberidaceae species later shifted their telial stage to grasses growing nearby. This host switch and subsequent divergence of *P. graminis* likely occurred after the Berberidaceae family had diversified into its modern genera, but before *Mahonia* and *Berberis* became genetically distinct. The current host range of the telial stage supports the idea of a central Asian origin; about 90% of its grass hosts belong to the Festucoideae subfamily, which is predominantly distributed in the northern hemisphere. This also implies that the Poaceae family had already divided into subfamilies before Festucoideae species were exposed to rust originating from barberry. With the rise of agriculture, humans played a major role in spreading *P. graminis* from Asia across the Mediterranean into Europe, Africa, the Americas, and ultimately Australia. Over time, *P. graminis* was divided into distinct forms based on host specificity and morphological features, particularly the size and shape of urediniospores. Initially, six formae speciales were recognized: *tritici* (wheat), *secalis* (rye), *avenae* (oat), *agrostidis* (*Agrostis* spp.), *poae* (bluegrass), and *airae* (*Aira caespitosa*), with more added as new host-specific forms were identified (e.g., *epigaei* for *Calamagrostis* spp.). The closely related species *P. phlei-pratensis*, primarily infecting timothy grass, was sometimes treated as another forma specialis of *P. graminis*. Urban (1967) further classified *P. graminis* in Europe into two subspecies based on urediniospore morphology: *ssp. graminis* and *ssp. graminicola*. Within *ssp. graminis*, two varieties were defined: *var. graminis* and *var. stakmanii*. Urediniospores of *ssp. graminis* are generally long-ellipsoidal to subcylindrical and 1.5–1.8 times larger than the broadly ellipsoidal to ovoid spores of *ssp. graminicola*. Among the two varieties, *var. graminis* produces slightly larger spores than *var. stakmanii*. The wheat stem rust fungus falls under *P. graminis ssp. graminis var. graminis*, while oat and rye stem rusts are categorized as *var. stakmanii*. Most rusts on festucoid grasses are grouped under *ssp. graminicola*, considered the ancestral lineage from which cereal-infecting forms evolved.

Despite these classifications, the genetic basis for differences in spore morphology remains unclear, and some findings challenge the validity of these subspecific divisions. For instance, Johnson (1949) observed high levels of fertility in

hybrid crosses between wheat and rye stem rust fungi (*ssp. graminis var. graminis* × *var. stakmanii*) and between oat stem rust and rusts of *Agrostis* or *Poa* (*var. stakmanii* × *ssp. graminicola*). However, crosses between other combinations—such as wheat and oat or rye and oat stem rusts—exhibited little to no fertility, indicating reproductive barriers even within what had been grouped into the same subspecies or variety. Molecular studies have also questioned traditional classifications. Zambino and Szabo (1993) found identical internal transcribed spacer (ITS) rDNA sequences in isolates of *ff. spp. avenae*, *dactylis*, *lolii*, and *poae*, while these differed from those in *ff. spp. tritici* and *secalis*, suggesting closer genetic relationships across traditionally separated subspecies and more distant relationships within the same variety. Additional evidence of reproductive incompatibility was observed in F1 hybrids between wheat and oat stem rust fungi: infected barberry plants developed pycnia without nectar, and no aecia formed, although a few urediniospores and teliospores eventually appeared in aging pycnial infections. Similar disruptions occurred in hybrids between rye and oat stem rust fungi, both placed in *var. stakmanii*. Johnson (1949) concluded that natural hybridization is limited by two main factors: genetic incompatibility leading to sterility between distantly related forms, and reduced fitness in hybrid progeny, which were often less pathogenic than the more virulent parent on a given host.

CONCLUSION

Due to its high destructiveness and the critical economic value of its cereal hosts, *Puccinia graminis* ranks among the most intensively researched plant pathogens. Extensive global research has produced thousands of publications addressing its ecology, epidemiology, and interactions with host plants, particularly wheat. Despite this wealth of knowledge, many aspects of how this fungus causes disease remain unclear. The infection process of *P. graminis* is complex and developmentally regulated, involving eight distinct structures. While it's known that leaf surface features and CO_2 levels can stimulate the formation of early infection structures, it's still uncertain whether additional plant- or fungus-derived signals are involved.

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