Recovery Plan

For

Wheat Blast

Caused by

Magnaporthe oryzae Triticum pathotype

June 4, 2013

This recovery plan is one of several disease-specific documents produced as part of the National Plant Disease Recovery System (NPDRS) called for in Homeland Security Presidential Directive Number 9 (HSPD-9). The purpose of the NPDRS is to ensure that the tools, infrastructure, communication networks, and capacity required to mitigate the impacts of high consequence plant disease outbreaks can maintain a reasonable level of crop production.

Each disease-specific plan is intended to provide a brief primer on the disease, assess the status of critical recovery components, and identify disease management research, Extension, and education needs. These documents are not intended to be stand-alone documents that can address all of the many and varied aspects of plant disease outbreak and all of the decisions that must be made and actions taken to achieve effective response and recovery. They are, however, documents that will help USDA guide further efforts directed toward plant disease recovery.

Terms Used

Magnaporthe oryzae is divided into host-specialized populations including the *M. oryzae Oryza* pathotype causing rice blast in the U.S. and worldwide; the *M. oryzae Triticum* pathotype causing wheat blast disease in South America; and the *M. oryzae Lolium* pathotype causing Gray Leaf Spot (GLS) disease of the turf grasses perennial ryegrass and tall fescue, and the forage/cover crop annual ryegrass in the U.S. and Japan. For clarity in this recovery plan, the rice pathogens will be referred to as 'rice isolates', the wheat pathogens from South American will be referred to as 'wheat isolates', and the ryegrass pathogens from the United States and Japan will be referred to as 'ryegrass isolates'.

Executive Summary

Wheat Blast is a serious disease of wheat causing yield failures and significant economic losses during epidemic years. The disease was first identified in 1985 in Paraná State of Brazil and has since spread to important wheat-producing regions of Brazil, Bolivia and Paraguay. The disease has been reported on wheat in Northeastern Argentina and on barley in Uruguay. Although the disease occurs sporadically depending on weather conditions, it is now considered a major threat to wheat production in major wheat regions. The pathogen, *Magnaporthe oryzae Triticum* pathotype, can infect all above-ground parts of the wheat plant, but head blast, resembling Fusarium head blight in appearance, is the most common symptom in the field. Infected spikes become bleached and produce small shriveled seed or none at all. The risk for introduction of wheat blast strains from South America is high due to the seed-borne nature of the fungus and the increased trade and travel between the U.S. and countries where the disease occurs. Few resistance genes have been identified for wheat blast. Fungicides are not effective in controlling wheat head blast if warm, rainy weather occurs during the heading stage. Therefore, controlling the wheat planting date so that heading does not correspond to rainy periods constitutes a major management strategy in South America.

The fungus *M. oryzae* occurs as a series of host-specialized pathogen populations also including the pathotypes responsible for rice blast disease and Gray Leaf Spot disease (ryegrass isolates) on turf grasses. Both of these diseases already occur in the U.S. Rice isolates are genetically distinct from wheat isolates and are unlikely to cross over to infect wheat in the field. In contrast, U.S. ryegrass isolates are genetically closely-related to wheat isolates from South America, and recent evidence confirms that some of these native isolates are already able to infect wheat. Rice blast has been well-studied as a model system for fungal pathogenesis of plants. Although rice breeders have identified more than a hundred blast resistance genes in rice, durable resistance to rice blast has not been achieved due to the extreme potential for variation in the fungus. Wheat blast disease resembles rice blast in many respects, but there are also major differences that will impact control strategies.

Research priorities for wheat blast include identifying rapidly deployable resistant wheat varieties together with effective fungicide treatments. Effective resistance must be confirmed by

rigorous field tests in areas of South America where the disease is endemic, the pathogen population is diverse and conditions favor disease. Longer term, there is a critical need for identification of effective resistance genes and molecular markers to move these genes into new wheat varieties. It is critical to optimize fungicide treatments for field control of the disease and for seed treatments to prevent pathogen spread through contaminated seed. Validation of PCR-based diagnostics for detection of the pathogen and differentiating the South American wheat strains from native ryegrass strains is high priority. There is a need to understand the epidemiology of the wheat blast pathogen in South America and to determine risk from native U.S. ryegrass strains. Development of a wheat blast forecasting model will inform wheat stakeholders when environmental conditions favor disease development. Extension and education priorities are also critical because most stakeholders involved in wheat production are currently unaware of wheat blast disease. It is critical to develop workshops, short courses and Extension publications to educate stakeholders about the risk from wheat blast, how to differentiate wheat blast from Fusarium head blight and other diseases that resemble wheat blast, and how to respond in the event wheat blast occurs. It is important to incorporate blast surveillance into ongoing wheat disease monitoring networks such as the developing iPIPE, and to dovetail outreach efforts with NCERA-184 and WERA-97 to avoid duplication of effort and promote inter-group cooperation and activities.

Wheat Blast or Brusone do Trigo

(caused by *Magnaporthe oryzae Triticum* pathotype)

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I. Introduction

Wheat blast, caused by the *Magnaporthe oryzae Triticum* pathotype (wheat isolates), was first reported in the State of Paraná in Brazil in 1985. This fungus has since become a major pathogen within Brazil, Bolivia and Paraguay, and it has been reported in northeastern Argentina (Goulart et al., 2007; Kohli et al., 2011; Urashima et al., 2009). The disease also occurs on triticale, barley and black oats (Kohli et al., 2011; Urashima et al., 2004). The pathogen can infect all above-ground parts of the wheat plant, but damage in the field comes mainly from head (spike) blast,

Figure 1. Left: Wheat blast (also known as Brusone do Trigo) in wheat fields near Londrina, Paraná in August 2009. The scale of synchronous head infection without previous leaf blast symptoms raises the major question of inoculum source for these very large wheat fields. Photo from Andreas von Tiedemann (Georg-August University Goettingen, Germany) & Etienne Duveiller (CIMMYT Global Wheat Program, Mexico). **Middle and right:** Field view and infected wheat heads in Mato Grosso do Sul, Brazil in May, 2012. Photos from Jose Mauricio Fernandes (Embrapa, Brazil).

which produces shriveled seeds or totally prevents grain filling. Symptoms closely resemble and could easily be mistaken for Fusarium head blight. As with rice blast, epidemics causing severe crop losses are sporadic, depending on warm weather with high humidity (Urashima et al., 1993). In South America, severe wheat blast epidemics (1987, 1997, 2002, 2009) may be correlated with occurrence of the El Niño weather pattern (Mauricio Fernandes, Mohan Kohli, personal communications). Yield losses to this disease range from low, when the weather doesn't favor disease, to as high as 100% when conditions favor disease. Effective resistance is generally lacking for the wheat blast disease and fungicide treatments are unreliable when weather favors disease. The widespread, extremely damaging wheat blast outbreak in the fall of 2009 led to organization of the 1st International Wheat Blast Workshop in Brazil in May, 2010 (http://blog.cimmyt.org/?p=3707). A major outcome of this workshop was the establishment of the International Wheat Blast Consortium to promote disease control research and facilitate sharing of fungal strains and wheat varieties among international researchers.

Wheat isolates from South America are closely related to U.S. ryegrass isolates: Wheat blast disease is caused by the haploid, ascomyceteous fungus *Magnaporthe oryzae* (Anamorph *Pyricularia oryzae*) (Couch et al., 2005), which is associated with extensive host specialization at both the host species level. *M. oryzae* forms a distinct species from *M. grisea*, which infects crabgrass and other *Digitaria* species. *M. oryzae* is divided into crop-adapted populations that are specialized for infecting rice (O*ryza* pathotype), wheat (*Triticum* pathotype), perennial and annual ryegrass (*Lolium* pathotype), finger millet (*Eleusine* pathotype), foxtail millet (*Setaria* pathotype) and many others

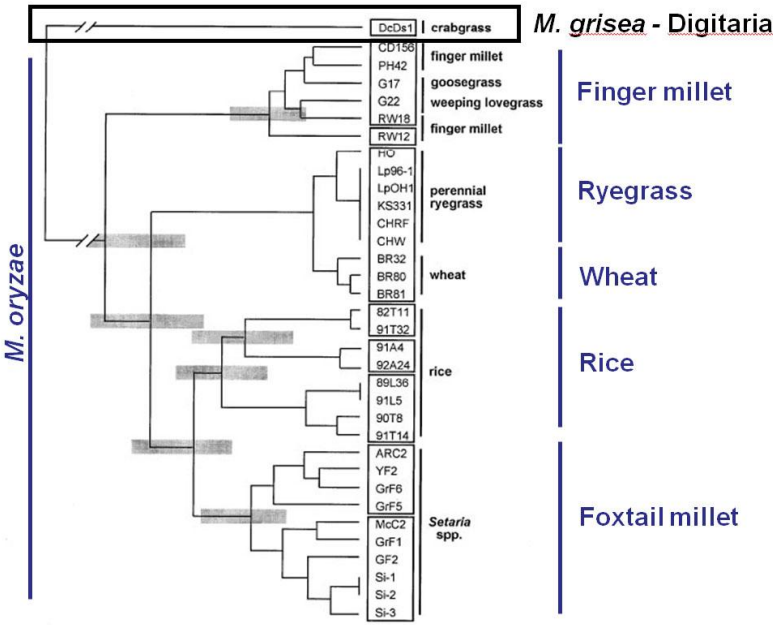

Figure 2. A phenogram showing the relative relatedness of different host specific forms of *M. oryzae*. (Farman, 2002)

(Farman, 2002; Kato et al., 2000; Tosa et al., 2004). DNA fingerprint analyses with various genes and transposable element sequences have determined relationships among different *M. oryzae* populations (Figure 2). Strains from the different populations are interfertile and only differ by a small number of genes that control host specificity (Tosa et al., 2006; Valent and Khang, 2010). Rice isolates differ from wheat isolates in containing a distinct set of transposable elements, but wheat and ryegrass isolates share the same set of transposable elements and are extremely closely related (Farman, 2002; Tosa et al., 2004; Viji et al., 2001). Characterization of ryegrass isolates in Japan, the other country where GLS has been reported, identified two distinct sub-groups based on fingerprint patterns, mating ability and pathogenicity. One sub-group resembled the U.S. ryegrass isolates in being highly aggressive against *Lolium* species even at 20 C, and having moderate sexual fertility (fertile only as males) (Farman, 2002; Tosa et al., 2004). The other sub-group showed intermediate levels of aggressiveness toward ryegrass and wheat and high levels of sexual fertility (isolates were hermaphroditic). Members of this second subgroup may represent an ancestor to the more specialized wheat and ryegrass populations (Tosa et al., 2004). Genome sequencing is blurring the line between the wheat- and ryegrass-adapted populations. This close genetic relatedness increases the likelihood for ryegrass strains to evolve into aggressive wheat pathogens.

In the U.S., GLS was reported for the first time on forage annual ryegrass in 1971 in Mississippi and Louisiana (Bain et al., 1972; Carver et al., 1972), on perennial ryegrass in 1992 in Pennsylvania (Landschoot and Hoyland, 1992), and on tall grass fescue in 1996 in North Carolina (Tredway et al., 2005). GLS disease on perennial ryegrass has now spread to Indiana, (Latin and Harmon, 2004), Illinois (Pedersen et al., 2000), Kentucky (Williams et al., 2001), Ohio, West Virginia, Virginia, Tennessee, and North Carolina (Harmon and Latin, 2003), Connecticut, Rhode Island (Schumann and Jackson, 1999), California, Nevada, and Utah (Wong, 2006). It occurs infrequently in Iowa, Nebraska, and Kansas, and has not been confirmed in environmental northern Midwestern states such as Michigan, Wisconsin, and Minnesota (Latin and Harmon, 2004). Farman (Farman, 2002) proposed that climate is a major factor preventing wheat blast epidemics in the U.S. In regions conducive to blast diseases, *M. oryzae* does not show up on ubiquitous and highly susceptible weeds such as crabgrass and foxtails, or on forage and turf grasses until July or August. Winter wheat grown in these areas is typically harvested in

mid- to late-June, before the peak period for blast disease epidemics. Likewise, spring wheat is grown in U.S. regions where the climate historically has not been conducive to *M. oryzae* diseases. For example, the GLS fungus appears unable to survive harsh winters in North Central Indiana (Harmon and Latin, 2005). Nevertheless, increasing global temperatures will undoubtedly increase the potential for overwintering and earlier development of *M. oryzae* diseases coincident with winter wheat production. Also, conditions in spring wheat regions may approach the warm, humid environments that promote wheat blast epidemics.

Isolation of a ryegrass strain from wheat in the U.S.: In May of 2011, *M. oryzae* was isolated from a single severely-blasted wheat head found in a wheat test plot at the University of Kentucky Research and Education Center in west KY (Figure 3). Subsequent analysis showed that the strain isolated from the wheat head in Kentucky, hereafter referred to as the 'Kentucky strain' did not appear to be a wheat isolate imported from South America (http://news.ca.uky.edu/article/uk-researchers-find-important-new-disease). Instead, the Kentucky strain was identified as a native U.S. ryegrass isolate (Farman, 2002; Tredway et al., 2003; Viji and Gnanamanickam, 1998). By comparative analysis of sequenced whole genomes, the Kentucky strain was more similar to native strains isolated from U.S. annual ryegrass than to wheat isolates from S.A. (M. Farman, K. Pedley and B. Valent, unpublished results). Wheat pathogenicity of the Kentucky strain and other ryegrass isolates was compared to South American wheat isolates by inoculation in Biosafety-Level 3 containment greenhouses in Fort Detrick, Maryland (Gary Peterson, unpublished results). In these greenhouse inoculations, the Kentucky strain and a subset of other native ryegrass strains were as aggressive on wheat as the South American wheat isolates assayed. This supports previous reports that some, but not all, U.S. ryegrass isolates infect wheat in the greenhouse (Tredway et al., 2005; Viji et al., 2001). Finding *M. oryzae* infections in May was unusual, since *M. oryzae* does not normally show up on susceptible weeds, forage and turf grasses until July or August (M. Farman, personal observations). Blast has not been identified a second time on wheat in the U.S. But this

Figure 3. Blasted wheat head found in a University of Kentucky test plot, 220 miles west of the main campus, in May, 2011.

incident indicates that native *M. oryzae* strains capable of infecting wheat are already present in the U.S.

II. Symptoms, Disease Cycle and other Pathogen Biology

Symptoms: The wheat blast pathogen can infect all above-ground parts of the wheat plant, but head infection is the most common symptom in the field (Figure 4a,b). Often, infected heads become bleached and either produce shriveled seeds or no seed at all (Figure 4b). Highly susceptible cultivars can be infected at the seedling stage (Figure 4c). Wheat leaf lesions in the field vary from small dark brown spots without light centers (Type 1), round or eye-shaped lesions with small light centers (Type 2), continuing through increasingly larger, eye-shaped lesions with light tan centers and dark brown margins (Type 5 lesions). Sporulating lesions appear gray from the color of the spores (Figure 4c). The potential for individual lesions to sporulate depends on the relative area of the light tan region (the sporulating region) inside the darker brown margins. A Type 5 leaf lesion on rice produces tens of thousands of spores per

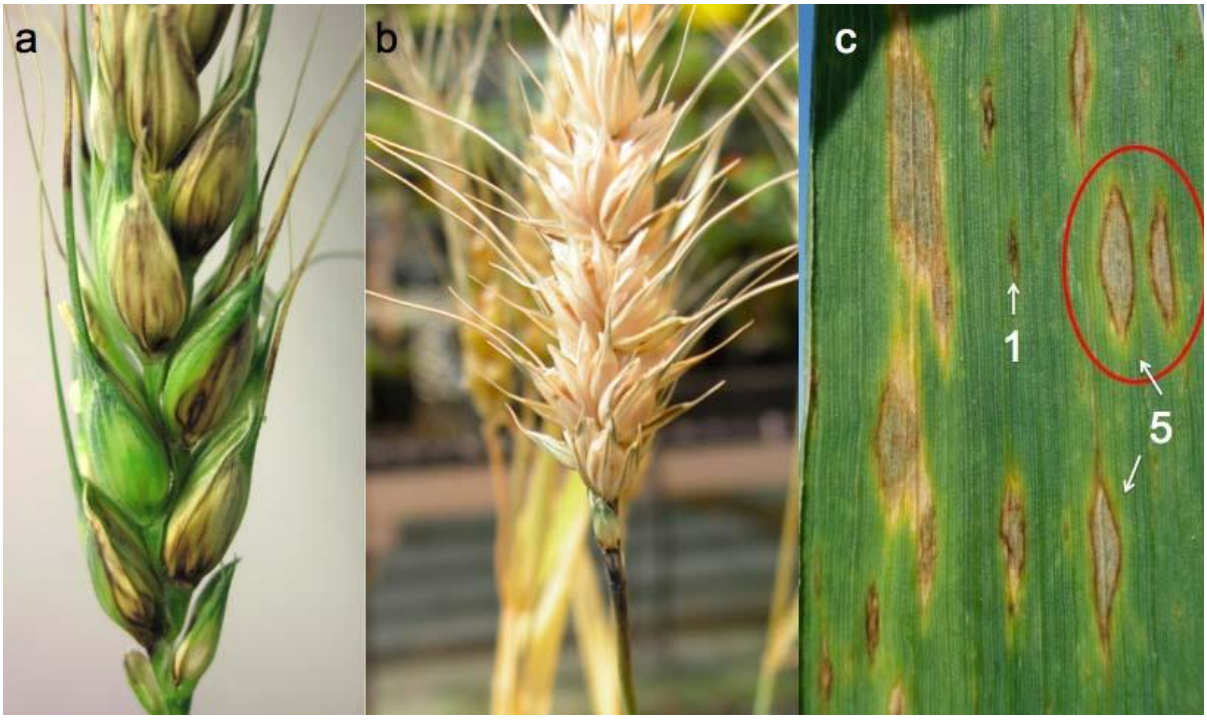

Figure 4. (a) Blast infection of wheat spiklets. Photo from Christian Cruz. (b) Head symptoms showing blackened rachis with entire wheat head bleached. Photo from Gary Peterson. (c) Sporulating wheat leaf lesions from the field in Bolivia. Lesion types 1 and 5 are labeled. The other lesions show intermediate types between 1 to 5. Photo from Guillermo Barea, Norfield SRL, Bolivia.

night over a 20-day period (Kato, 1974). Uniformly dark brown Type 1 lesions represent an enlarged resistance reaction and fail to produce spores. The predominance of wheat head blast symptoms without leaf lesions in the field represents a major difference between wheat blast and rice blast. For rice, leaf blast is a major symptom together with leaf collar blast, stem node blast, neck blast and panicle blast, and sporulation from leaves is assumed to provide inoculum for panicles. For rice, the youngest (still expanding) leaves are more susceptible than mature leaves.

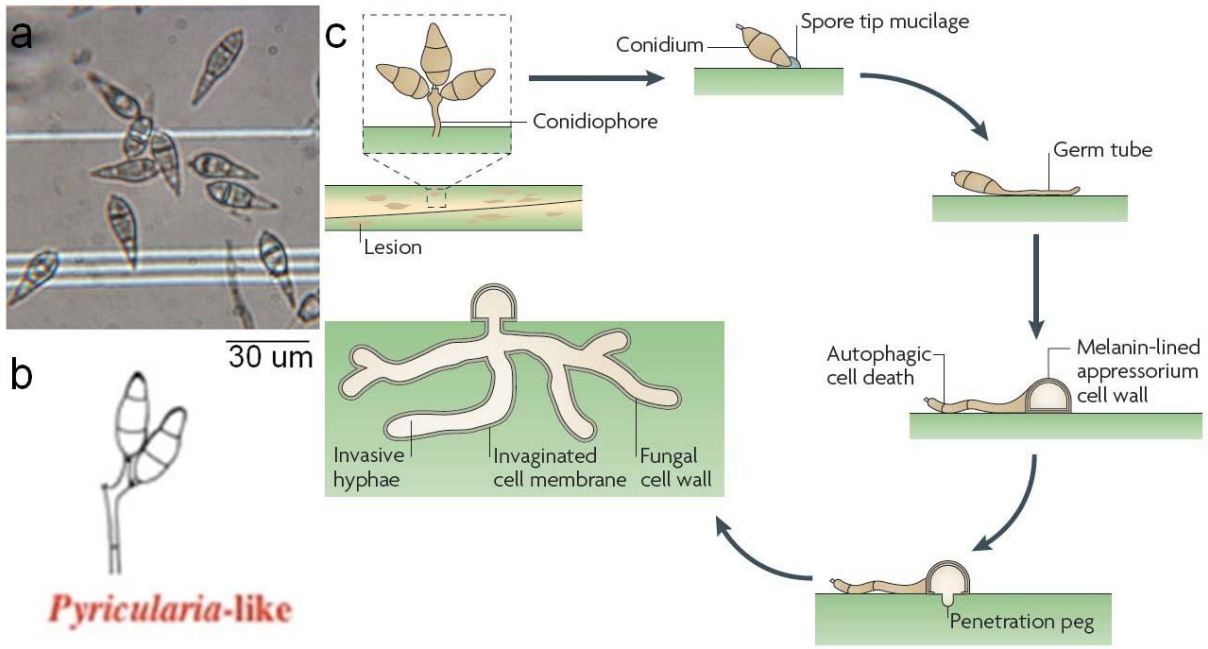

Figure 5. Rice Blast Disease Cycle: Hyaline to pale gray, pyriform, asexual conidia (a) are borne sympodially (b) on aerial conidiophores. (c) After landing on the host surface, the conidium grows as a specialized germ tube that senses the plant surface and differentiates a specialized cell, a melanized appressorium, which generates and uses tremendous turgor pressure (80-times atmospheric pressure) to puncture the outer plant surface and gain access to host tissue. Pressure build-up requires the appressorium to be immersed in water on the leaf surface. The contents of each conidium are redeployed by a process of autophagy, in which the spore empties out and the entire contents are moved into the appressorium before it becomes sealed off and a penetration peg punctures the plant surface. After penetration, the fungus forms specialized biotrophic invasive hyphae that successively colonize living rice cells for 4-5 days before sporulating macroscopic lesions appear. Figure 5c was reproduced from Wilson and Talbot (2009).

The Blast Disease Cycle: The disease cycle is well-studied for rice isolates (Valent and Khang, 2010; Wang and Valent, 2009; Wilson and Talbot, 2009), but wheat and ryegrass isolates appear to execute the same disease cycle (Tufan et al., 2009). The hallmark of this disease is the

diagnostic pyriform conidia that gave rise to the anamorph species name *Pyricularia*. Each conidium has three cells (Figure 5), with a single nucleus per cell. All nuclei in a conidium are derived through mitosis from a single nucleus, and consequently, a fungal strain can be purified by isolation of a single spore. The melanin layer in the appressorium is essential for this cell to build-up the very high pressure required to puncture the outer plant surface and gain access to host tissue (Figure 5c). This accounts for the special class of fungicides, the melanin biosynthesis inhibitors (tricyclazole, pyroquilon, phthalide, and carpropamid), which are specific for controlling rice blast disease. Pressure build-up requires immersion of the appressorium in water, explaining in part the requirement for extended periods of rain or dew for this disease. Conidia appear packed with nutrients that are redeployed to power appressorial mediated host entry. After penetration, the fungus forms specialized biotrophic invasive hyphae (Figure 5c) that successively colonize living rice cells without visible symptoms for the first 4 days. Sporulating macroscopic lesions appear after 4 days in rice (Valent and Khang, 2010). Water is also required for release of conidia from conidiophores to reinitiate the disease cycle.

***The sexual cycle, ascospores and microconidia*:** The *M. oryzae* sexual cycle has not been observed in nature on any host, but some strains undergo sexual crosses in the laboratory (Yaegashi and Udagawa, 1978). Wheat blast isolates show high levels of sexual fertility, functioning as hermaphrodites and crossing to produce abundant viable ascospores (Urashima et al., 1993). This level of fertility in the laboratory raises the possibility that the wheat pathogen may undergo sexual recombination in the field. This is in contrast to the infertility of most rice and ryegrass isolates and the predominantly clonal asexual populations of these pathogens in the field.

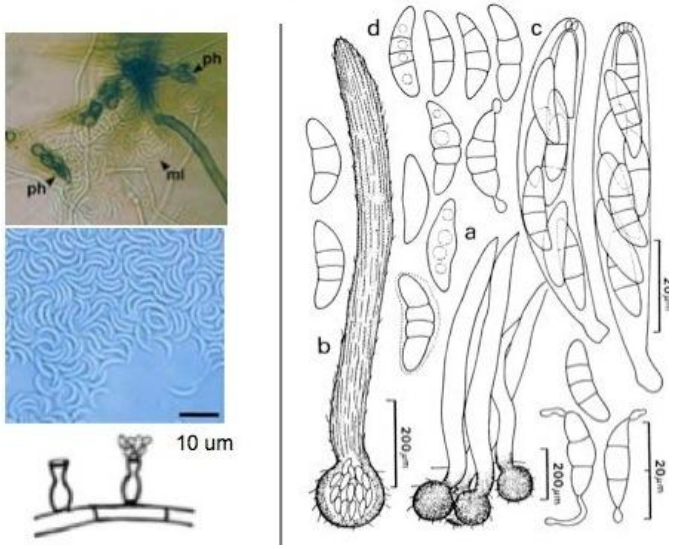

Figure 6. (Left) Sexually fertile strains produce microconidia from phialides. (Reproduced from Chuma et al., 2009). (Right) Perithecia (a,b), asci (c) and ascospores (d). (Reproduced from Yaegashi and Udagawa, 1978).

M. oryzae is a heterothallic Ascomycete, class Pyrenomycete. Fully fertile strains are self-sterile hermaphrodites (functioning both as females and males), with compatibility for mating governed by alternate alleles of the mating type locus *MAT1*. Two additional spore forms, ascospores and microconidia, are produced by sexually fertile strains. Ascospores are produced in unordered asci within perithecia with long necks (Figure 6 right) (Yaegashi and Udagawa, 1978). Ascospores are hyaline and crescent-shaped, with four cells, each containing a single mitotically-derived nucleus. Perithecia produce asci within 2 to 3 weeks when strains of opposite mating type are incubated on oatmeal agar in the light at ~20 °C. Mature asci are extruded through the perithecial necks and ascospores are released into a viscous liquid. Ascospores produce appressoria for plant penetration. Sexually fertile isolates also produce small, crescent-shaped microconidia, 6 micrometers in length and 0.7 micrometers in width (Figure 6 left), which are hypothesized to function as spermatia. Microconidia are produced from phialides, and conditions under which they germinate are not known (Chuma et al., 2009).

Potential for root pathogenicity and microsclerotia: The ability of sexually fertile *M. oryzae* strains to produce microconidia from phialides is consistent with the fungus's close taxonomic relatedness to the phialide-producing root pathogens, *Magnaporthe poae* and *Gaeumannomyces graminis* (Sesma and Osbourn, 2004; Zhang et al., 2011). In laboratory studies, rice isolates can infect roots through similar processes used by these classical root pathogens, by forming dark runner hyphae on the root surface, hyphopodia for root penetration, bulbous invasive hyphae inside root cells and microsclerotia-like structures (Sesma and Osbourn, 2004). It has been reported that the rice blast fungus can produce sclerotia *in vitro* and that conidia emerging out of the sclerotia can cause leaf blast (Gangopadhyay and Row, 1986). Sclerotia are compact mycelial masses that survive unfavorable environmental conditions for long periods. It is not known if this laboratory-defined biology has relevance to wheat blast disease epidemiology.

III. Inoculum Source and Spread

Based on rice blast, it is assumed that conidia are the main means for spread of the wheat blast fungus. Blast conidia appear adapted for rapid penetration of host plants and not for persistence outside the host (Figure 5c). Extensive laboratory studies on appressorium formation have shown

that conidia lose ability to produce appressoria after several days at room temperature. After soaking for at least a half hour, drying of conidia from both rice and wheat isolates blocks their ability to germinate and form appressoria (Kato, 1974; Cruz and Bockus, unpublished results). It is unclear what this means for the field biology of the fungus, in which water both releases spores from conidiophores and initiates their germination. For rice blast, the youngest expanding leaves are most susceptible, and new rice leaves emerge around the time developing lesions sporulate. For rice blast, most spores have been detected in the rice canopy, about one third the height of the plants, although spores have been detected kilometers away from fields (Kato, 1974). A study of fungal isolates from triticale indicated that conidia travel at least 1000 meters from the inoculum source (Urashima et al, 2007).

For rice blast disease, sporulating leaf lesions are presumed to be the inoculum source for neck and panicle blast. The lack of lesions on wheat leaves before occurrence of head blast raises the question of the source of the inoculum causing simultaneous infections over such extensive areas (Figure 1). Conidia might originate from any or all of the following three sources.

Seed: *M. oryzae* is known to be seed-borne (Greer and Webster, 2001; Urashima et al., 2004), indicating the potential for disease to spread to new geographic locations through movement of infected seed. Polymerase chain reaction (PCR) has been successfully applied to the detection of *M. oryzae* in rice seeds (Chadha and Gopalakrishna, 2006). Inoculations with a fluorescent rice isolate, labeled with the jellyfish green fluorescent protein, showed that the fungus was primarily located in the seed coats of infested seed and that spores were produced soon after these seeds germinated (Faivre-Rampant et al., 2012). Fungus from seeds infected coleoptiles and primary roots and produced mycelium that colonized primary leaves and secondary roots. Faivre-Rampant (2012) also reported that infested seeds were produced after inoculation of rice plants at the ripening stage, the heading stage, or even after inoculation of fully-developed flag leaves before heading. Seedlings from infested seeds of a highly susceptible rice variety often died and served as inoculum for healthy neighboring plants (Faivre-Rampant et al., 2012). Although seed transmission of wheat blast has been established (Urashima et al., 2004), it is not clear what role this plays in the epidemiology of wheat head blast since the fungus is often not identified on leaves or culms soon after planting. It has been suggested that *M. oryzae* is capable of endophytic

growth in rice plants (Marcel et al., 2010), which would provide a conidium-independent route to head infection, but this has not been proven.

Crop Residue: Infected leaf debris and stubble are considered to be a source for seasonal carryover of *M. oryzae* pathotypes causing rice blast and GLS. It is reported that *M. oryzae* does not survive in stubble at the colder temperatures in Northern Indiana (Harmon and Latin, 2005), suggesting poor winter survival. No published peer-reviewed research was found concerning the survival of the wheat blast pathotype in infected plant debris.

Secondary Hosts: A common assumption in South America seems to be that conidia responsible for wheat infection come from blast lesions on the weeds surrounding wheat fields. The potential for cross-infectivity of the different host species-adapted forms of *M. oryzae* is currently disputed. Some artificial inoculation experiments have indicated that wheat is susceptible to isolates from weed species such as Alexandergrass (*Brachiaria plantaginea*), Sourgrass (*Digitaria insularis*), crabgrass (*D. sanguinalis*), goosegrass (*Eleusine indica*), fountain grass (*Pennisetum setaseum*), *Rhynchelytrum roseum*, and knotroot foxtail (*Setaria geniculata*) (Tredway et al., 2005; Urashima et al., 2004; Viji et al., 2001). In other studies, fungal isolates collected in nature belong to the pathotype specialized for the host species from which they were collected (Couch et al., 2005; Tredway et al., 2005), indicating that cross infection is rare in nature. Blast disease assays are very sensitive to general plant health, fertilizer regime, etc, and care must be taken in interpretation of artificial inoculation results. Greenhouse/growth chamber inoculations suggest that wheat isolates are pathogenic to barley, common millet, corn, oat, rye, sorghum, and triticale (Urashima et al., 1993; Urashima et al., 2004). Black oats (*Avena strigosa*) and foxtail millet (*Setaria italica*) are components of the crop rotation system in the wheat blast endemic region and may serve as secondary hosts (Kohli et al., 2011). However, the widespread, synchronous development of blasted wheat heads in large production fields argues against spores from secondary hosts as the source of inoculum.

IV. Environmental Conditions Favoring Wheat Blast Disease

Like rice blast, wheat blast is a sporadic disease, with the most extensive damage occurring during warm, wet years (Urashima et al., 1993). Wheat blast epidemics are reported to follow

several days of continuous rains and temperatures from 18-25 C during flowering, followed by hot, sunny and humid days (Kohli et al., 2011). Controlled chamber studies indicate that the minimum temperature for infection is 10 C and the maximum is 32 C, with optimum between 25 and 30 C (Cardoso et al., 2008). Results were almost identical for the GLS pathogen (Moss and Trevanthan, 1987). High relative humidity is required for sporulation (Alves and Fernandes et al. 2006) and the rice blast pathogen has similar requirements (Kato, 1974,). Rain and/or heavy dew are required for appressorium function and spore release, and high humidity promotes lesion expansion. Rice blast and GLS are favored by high nitrogen fertilization (Williams et al., 2001), and rice blast is favored by aerobic soils and by drought stress. Although unknowns surround the effects of global climate change, one report suggests that rising CO_2 levels will enhance rice blast disease (Kobayashi et al., 2006). It is currently not known if wheat blast disease will respond to these environmental factors in the same way as rice blast. Preliminary climate-based risk maps for wheat blast are in progress (R. Magarey, C. Cruz, J. Stack unpublished data).

V. Resistance

Preemptive identification of elite U.S. wheat varieties with resistance to wheat blast would enable rapid deployment of resistant varieties in response to an outbreak of the disease. Wheat blast sensitivity has been determined for >498 US wheat varieties. Winter wheat varieties (418) and spring wheat varieties (80) have been screened for blast resistance in greenhouse and growth chamber assays performed in Biosafety Level 3 (BSL-3) laboratories in Maryland and Kansas (Bockus et al., 2012; Cruz et al., 2012; unpublished results from Peterson et al. and Cruz et al.). These varieties were screened using at least one Brazilian *M. oryzae* strain (T-25) that was isolated in 1988. Many varieties were inoculated with additional Brazilian wheat isolates from the late 1980s, and with Bolivian wheat isolates from 2011 and 2012. Seedling inoculation was not always predictive of head

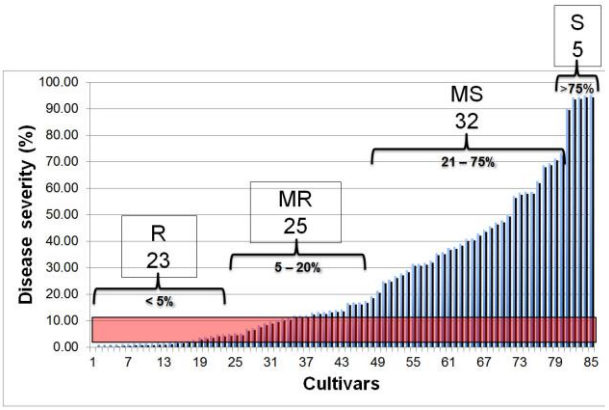

Figure 7. Head inoculation assays under BSL-3 containment growth chamber/greenhouse conditions show a continuum of symptoms, taken from Cruz et al, 2012.

susceptibility so resistance was assessed directly by head inoculations (Cruz et al., 2012; Urashima et al., 2009). Infection was scored based on percentage of blighted wheat spikelets. Using these strains, head blast susceptibility among these varieties occurs as a continuum from highly susceptible (>75% blighted spikelets) to highly resistant (<5% blighted spikelets) (Figure 7). Three *Aegilops tauschii* accessions also show high levels of resistance (Bockus et al., 2012). Some U.S. varieties that are currently widely grown are highly susceptible to the tested wheat blast isolates. Additionally, the newer (2011 and 2012) wheat isolates are more aggressive than the Brazilian wheat isolates from the 1980's and generally cause greater disease on all cultivars, even on those that display high levels of resistance to the earlier isolates.

Experience in South America shows that wheat varieties showing high levels of resistance to a limited number of isolates in greenhouse/growth chamber conditions may not show resistance against natural field populations (Kohli et al., 2011), and it is critical to confirm identified resistance in U.S. wheat by field tests in South America. Field tests of U.S. wheat varieties showing <10% spike infection are underway in two locations in Bolivia, near Santa Cruz and in an apparent wheat blast hot spot in Quirusillas. Field tests are planned at multiple locations in Brazil. Additionally, a USDA-ARS Specific Cooperative Agreement project has been critical for establishment of an irrigated field test site in Paraguay (Project # 1920-22000-041-07, Field evaluation of wheat blast of U.S. wheat germplasm in Paraguay, Gary Peterson, Foreign Disease-Weed Science in Ft. Detrick).

Longer term, there is a critical need for identification of effective wheat blast resistance genes for breeders to include in new wheat varieties. Two resistance genes to wheat blast, *Rmg2* on chromosome 7A and *Rmg3* on chromosome 6B, have been identified in wheat variety Thatcher (Zhan et al., 2008). Varieties derived from the advanced CIMMYT line Milan appear to contain high levels of resistance throughout the area of endemic disease (Kohli et al., 2011). Varieties with this resistance source are now being widely deployed and it remains to be seen how long this resistance will remain effective.

It is important to understand if wheat blast will develop the complicated race structure characteristic of rice blast, for which hundreds of races have been identified based on reaction to

over a hundred major resistance genes identified in rice (Valent and Khang, 2010; Wang and Valent, 2009). Race specificity for wheat isolates has been reported through studies of wheat leaf blast in Brazil (Urashima et al., 2004). Specifically, inoculation of 72 wheat isolates from Mato Grosso do Sul and Paraná on 20 Brazilian wheat varieties in greenhouse assays identified 54 distinct virulence patterns, which suggests cultivar-specific resistance in wheat blast (Urashima et al., 2004). Among spring wheat cultivars tested for wheat blast resistance in Brazil, (Cruz et al., 2012; Urashima et al., 2004), a few showed seedling-stage resistance to a subset of wheat isolates but no variety has been found to be resistant to all isolates. Differential wheat varieties have not been established for wheat blast. Varieties that display seedling-stage resistance are often less susceptible at the heading stage, but no wheat variety has been identified that displays high-level, broad-spectrum resistance to head infection.

VI. Monitoring and Detection

A factor complicating rapid identification of wheat blast is that wheat spikes infected by blast resemble those infected by *Fusarium* spp. (cause of Fusarium head blight, FHB), a widely-spread disease in the U.S. In areas that have FHB, the similar symptoms of the two diseases may render the wheat blast unnoticed when it first appears in the field, unless wheat producers and Extension specialists are trained to anticipate and recognize the disease. In particular, training and close-examination will be required for first-responders to differentiate the FHB-type bleaching, which shows traces of orange to pink spore masses, from wheat blast-type bleaching, which is more creamy but with gray *M. oryzae* spore masses at the base of the bleached section. If microscopic examination of infected tissue does not reveal the gray pyriform spores that are diagnostic for wheat blast, incubation of diseased tissue under high humidity conditions should induce sporulation. Note that recovering the blast fungus from leaf lesions that have passed their sporulation potential can be difficult. Also, the blast fungus is hard to recover from field tissue if the tissue is too wet. Lesions incubated with free water standing on their surface will typically only yield saprophytic contaminants.

Diagnostics Development and Validation: It is critical to develop PCR tests for four diagnostic purposes: 1) confirmation of the wheat blast pathogen in suspect diseased wheat samples; 2) detection of the wheat blast pathogen in seed lots to prevent disease introduction and movement

between U.S. wheat regions; 3) detection of wheat blast in asymptomatic tissues and in spore trap experiments for in-field disease monitoring and epidemiology studies; 4) determining the origin (native or exotic) of the pathogen population if a wheat blast outbreak occurs, as this will affect how research and regulatory communities respond.

PCR has already been used to detect *M. oryzae* in infected perennial ryegrass (Harmon et al., 2003). Development of PCR-based diagnostic tools for identifying wheat isolates and differentiating them from ryegrass isolates is underway (Kerry Pedley, Jim Stack et al.). Sensitive, initial screening could be performed using the repetitive MoTeR transposon (Farman, 2007) – a high copy number DNA sequence that is largely restricted to wheat and ryegrass isolates. No wheat isolate-specific repeats have been identified (Mark Farman, personal communication). Positive samples would then be re-screened using a single/low copy marker(s) capable of distinguishing between wheat and ryegrass isolates. Genome sequencing is proving a cost-effective way to genotype and determine the origin of particular *M. oryzae* strains (M. Farman, K. Pedley and B. Valent, unpublished results).

Development of a Quantitative Pathway Risk Assessment for the Triticum pathotype of M. oryzae will highlight possible routes of entry and establishment in order to focus monitoring efforts (C. Cruz, R.D. Magarey, G. Fowler and J. Stack, unpublished results). Although the U.S. is one of the largest producers and exporters of wheat globally, it also imports wheat from Brazil as part of the international grain trade. Since wheat isolates are seed-borne, contaminated grain may be an important source of inoculum for introduction. The goal of this study is to estimate the probability of wheat isolate entry into the U.S. associated with the importation of wheat grain from two wheat blast endemic areas in Brazil: Paraná and Rio Grande do Sul. To make decisions regarding trade policy, it is important to understand the risk associated with this pathogen. Model results may be used to inform regulatory policy for U.S. wheat imports from at-risk countries as well as to develop preparedness plans for early detection and effective mitigation.

VII. Current Status of Control and Response

Complete, broad-spectrum resistance has not been identified for wheat head blast. Resistant and moderately resistant U.S. winter and spring wheat varieties have been identified by inoculation

with a small number of wheat isolates tested in BSL-3 biocontainment facilities in Maryland and Kansas. Field tests on some of these varieties are underway with natural pathogen populations in Bolivia and field tests are also planned in Brazil and Paraguay. In South America, mixtures of triazoles (tebuconazole and metconazole) and strobilurins have been used effectively to control head blast in moderately resistant wheat varieties (Kohli et al., 2011). However, fungicide applications have not provided effective control with susceptible wheat varieties in disease-conducive environments (Goulart, 2005, Urashima et al., 2009). This is assumed to be due to challenges with fungicide application (application to vertical heads is highly variable and inconsistent) that also impact control of other head diseases such as FHB. However, it is important to know if fungicides are not working well because of poor activity of the active ingredient, improper timing of application, incomplete application, or some or all of the above.

Controlling planting dates is an important aspect of disease management in South America. Planting of the winter crop before April 15 is forbidden in Bolivia (April 10 in Brazil) (Mehta et al., 1992), since the early planting dates more likely correspond to wet, blast-conducive periods during heading. These weather conditions are important for infection and disease development during the heading and grain-fill period. Deep plowing of infected plant residues and elimination of possible alternate hosts such as gramineous weeds have been recommended in Brazil. These practices are not likely to be acceptable or feasible in the U.S. for a variety of reasons.

VIII. USDA Pathogen Permits

Due to the threat this disease poses to U.S. wheat production, wheat isolates from South America must be stored and worked on only under Biosafety Level-3 containment conditions. Currently, USDA-APHIS has granted approval for strain acquisition, storage, and research to groups with appropriate BSL-3 facilities in the USDA-FDWSU (Ft. Detrick, MD), and at Kansas State University (Manhattan, KS). A permanent collection of strains from South America is maintained at the FDWSU by Gary Peterson. Strain acquisition, storage, and research with native ryegrass isolates is not regulated.

IX. Economic Impact and Compensation

Wheat adds an estimated $30 billion to the U.S. economy. Globally, wheat accounts for one-fifth of all calories consumed and is the most important source of food protein worldwide. The economic importance of this disease derives from the fact that the fungus can reduce yield and the quality of the grain (Goulart, 2005). Infected grains are usually small, wrinkled, deformed, and have low-test weight. The highest yield losses occur when infections begin during flowering or grain formation. Reported yield losses in Brazil on susceptible cultivars vary from 10.5 up to 100% (Goulart and Paiva, 2000; Goulart et al., 1992).

X. Mitigation and Disease Control

Resistance: Preemptive deployment of elite U.S. varieties with effective resistance to wheat blast would go a long way toward preventing disease establishment. Therefore, screening current varieties for resistance is an important first step to enable planting of resistant varieties in regions where the disease has occurred and surrounding areas. Longer term, it is critical for breeders to be able to incorporate effective resistance into new varieties. Currently, the time needed to identify and validate genetic traits such as wheat blast resistance may take 3-10 years and to integrate these traits into a market-ready wheat variety, usually takes another 7-14 years. Clearly, with current warming trends, it is important that we prepare for wheat blast through timely investment in research into methods of detection, monitoring and control, as well as education of personnel in every sector of the wheat industry.

Fungicides: Fungicides are partially effective against wheat head blast and are considered only optimally effective when applied to wheat cultivars that are at least moderately blast resistant. Because the wheat blast fungus is seed-borne, another management strategy is seed treatment with fungicides. In Bolivia, Benlate® and Carbendazim+Thiram® have given excellent control of the pathogen at a rate of 200 ml per each 100 kg of seed. Seed treatment is a potential way to reduce the spread of the pathogen from region to region, and to reduce the level of initial inoculum.

It has been reported that seed treatments do not impact head blast occurrence in the field due to the length of time between planting and head emergence (Kohli et al., 2011). However, this

needs to be further explored and confirmed. Although seed treatments do not appear effective in protecting wheat planted in an area where the disease occurs, a number of studies on the efficacy of fungicidal seed treatments for controlling wheat blast were completed by three different research groups and published in Brazil (Goulart and Paiva, 1991; Igarashi, 1990; Igarashi and Oliveira, 1992; Lasca et al., 2001). A summation of these studies showed excellent control of seed-borne infection using iprodione (50g ai/100 kg seed) plus thiram (159g ai/100 kg seed), carboxin (22.5g ai/100kg seed) plus prochloraz (82.5g ai/100kg seed), carbendazim (52.5g ai/100kg seed) plus iprodione (26.2g ai/100kg seed), carbendazim (50g ai/100kg seed) plus mancozeb (160g ai/100kg seed) and triflumizole (45g ai/100kg seed) plus thiophanate methyl (135g ai/100kg seed). Most of the fungicides from these published studies are not labeled for use on wheat in the U.S. New tests are needed to evaluate choices of fungicides to control wheat blast for seed importation so they are labeled for use on wheat in the U.S. or eligible for a section 22(ee). The following seed treatment fungicides (ai) are labeled on wheat: Dividend (difenoconazole), Dynasty (azoxystrobin) and Maxim (fludioxonil) from Syngenta; Raxil (tebuconazole), Rancona or Rancona Pinnacle (ipconazole), Proceed (prothioconazole + tebuconazole), Prevail (carboxin + PCNB), Vitavax (carboxin), Baytan (triadimenol) and (thiabendazole) from Bayer; and Stamina (pyraclostrobin + triticonazole) and Charter (triticonazole) from BASF. Because private and public breeders conduct winter increases of valuable U.S. wheat germplasm in Argentina these studies may define useful pre-shipment treatments. A useful resource for potential fungicide seed treatments is: (http://msuextension.org/publications/AgandNaturalResources/MT199608AG.pdf).

XI. Experts and Infrastructure

Research projects on wheat blast are active at the USDA/ARS Bio-Safety Level 3 Plant Pathogen Containment facilities in the Foreign Disease-Weed Science Unit in Ft. Detrick, MD. (http://www.ars.usda.gov/research/projects/projects.htm?ACCN_NO=411489). These projects are "EMERGING FOREIGN FUNGAL PLANT PATHOGENS: DETECTION, BIOLOGY, AND INTERACTIONS WITH HOST PLANTS" (Project Number: 1920-22000-041-00). Research Project: INTERNATIONAL WHEAT BLAST CONSORTIUM; Project Number: 1920-22000-041-04, Project Type: Nonfunded Cooperative Agreement. Research Project: WHOLE GENOME SEQUENCING OF *MAGNAPORTHE ORYZAE* ISOLATES AND GENE

EXPRESSION ANALYSIS OF INFECTED WHEAT; Project Number: 1920-22000-041-08, Project Type: Specific Cooperative Agreement.

Wheat blast research was previously supported by a competitive grant from the USDA Plant Biosecurity Program (#2009-55605-05201) entitled "GENOME-ENABLED DIAGNOSIS OF THE WHEAT BLAST PATHOGEN AND IDENTIFICATION OF RESISTANCE RESOURCES." This project involved researchers from Kansas State University and the USDA ARS NAA, Foreign Disease-Weed Science Research Unit, Fort Detrick, MD (http://www.k-state.edu/wheatblast/). This research is continuing in an expanded form through a competitive grant from USDA AFRI-Fungal Pathogens Integrated Project (#2013-68004-20378), entitled "NOVEL STRATEGIES FOR MANAGING BLAST DISEASES ON RICE AND WHEAT." B. Valent serves as Project Director together with 18 Co-PIs including collaborators from Brazil, Bolivia and Paraguay.

The following individuals have expertise on wheat blast disease:

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XII. Research, Extension, and Education Priorities

Research Priorities:

1. **Identify host resistance to wheat blast.**

a) Confirm identified blast resistance in U.S. spring and winter wheat varieties through field tests in blast prone regions of South America.

b) Identify broadly effective wheat blast resistance genes from resistant germplasm and develop molecular markers to incorporate this resistance into U.S. wheat varieties.

c) Perform genetic analysis to determine if resistance is due to major genes or quantitative trait loci.

d) Understand potential for race structure in the wheat pathogen population and the potential for resistance genes to be broken by evolution of new races.

2. **Validate, refine, and deploy PCR-based diagnostic protocols for detection of wheat blast in seed lots and asymptomatic tissues.**

3. **Optimize fungicide treatments for field control and seed treatments for prevention.**

4. **Understand wheat blast epidemiology in South America.**

a) Identify the inoculum source for wheat blast in the field. This information is critical for developing blast forecasting models and management guidelines.

b) Use spore trap technology to determine the aerobiology of conidia.

c) Determine optimum conditions for *Magnaporthe* survival particularly in response to freeze/thaw cycles.

d) Determine the genetic structure of the S. American wheat pathogen population(s). Specifically, determine if sexual reproduction occurs in the field and how this contributes to disease development.

e) Determine if *M. oryzae* grows endophytically in wheat plants and if this contributes to wheat disease epidemiology.

f) Collect disease progress, host and microclimate data during outbreak and non-outbreak years to determine the rate of epidemic development and better understand the relationship between the disease development on the leaves and the head.

5. **Determine the risk to U.S. wheat posed by native ryegrass strains.**

a) Monitor additional ryegrass strains for population structure and ability to infect wheat.

b) Monitor the status of blast disease in annual and perennial ryegrass in wheat-growing regions of the U.S.

c) Monitor the growing use annual ryegrass as a cover crop in the U.S. (http://ryegrasscovercrop.com/) and potential impact towards a native origin for wheat blast.

6. **Develop and validate a wheat blast disease forecast model in order to prioritize monitoring efforts to regions and times when the threat of disease occurrence is high.** By identifying areas at high risk for disease, the models also help guide efforts to monitor wheat and ryegrass populations in the U.S.

7. **Study impact of global warming on enhancing wheat blast risk.**

Extension Priorities: Education in the following areas will help wheat producers, agricultural professionals, decision makers and other professionals to rapidly identify wheat blast and respond to control the disease.

1. **Train farmers, plant disease diagnosticians, Extension specialists (agronomists, plant pathologists), and other agricultural professionals to identify and differentiate wheat blast from Fusarium head blight and other diseases causing similar symptoms on wheat.**

2. **Train Extension specialists, crop consultants, farm advisors/educators and growers to use prediction models to evaluate the risk of blast epidemics and modify management strategies.** Training should incorporate possible increased risk of wheat blast due to global warming/climate change.

3. **Incorporate wheat blast surveillance into ongoing wheat disease monitoring networks, perhaps by incorporating efforts into the developing iPiPE.**

a) Recommend inclusion of susceptible winter and spring wheat trap plants in breeder plots in U.S. areas deemed likely for establishment of wheat blast in the U.S. (central and south eastern US?)

b) Provide access to a list of highly susceptible entries from diverse regions of the U.S.

c) Monitor barley for occurrence of blast.

4. **Educate growers and agriculture professionals about gray leaf spot and blast on annual ryegrass and the potential threat to wheat production.**

Education Priorities:

1. **Develop and host wheat blast workshops and short courses for wheat stakeholders including farmers, plant disease diagnosticians, Extension specialists and research plant pathologists, other agricultural professionals, national, regional and state commodity leaders, and decision makers.** Plant diagnosticians should be engaged through the National Plant Diagnostic Network.

2. **Develop and disseminate Extension publications on identification and management of wheat blast.**

3. Engage print and electronic media outlets to encourage the widespread dissemination of accurate information on wheat blast to the public at large.

4. Dovetail outreach efforts with NCERA184 and WERA97 to avoid duplication of effort and promote inter-group cooperation and activities.

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NOTE: Wheat blast specific references are shown in bold.

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Web Resources

Wheat Blast Web Page

American Phytopathological Society

National Plant Diagnostic Network

Great Plains Diagnostic Network

National Association of Wheat Growers

National Wheat Improvement Committee

USDA National Plant Disease Recovery System

Small Grain Seed Treatment Guide, from Montana State University