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A Toolbox for Managing Blast and Sheath Blight Diseases of Rice in the United States of America

Yulin Jia, Melissa H. Jia, Xueyan Wang and Haijun Zhao

Abstract

Rice blast disease caused by the fungus *Magnaporthe oryzae* and rice sheath blight disease caused by the fungus *Rhizoctonia solani* are two major hurdles for stable rice production worldwide. Presently, fungicides are still needed to manage these two devastating fungal pathogens. After two decades of research efforts, a toolbox has been assembled with the following components: (1) insight into pathogen genomic identity and pathogen avirulence (AVR) genes that can be used to enhance plant breeding; (2) new mapping populations and germplasm and genetic stocks that can be used as starting materials to identify effective host resistance (R) genes; (3) user-friendly disease evaluation methods that can be used to accelerate the identification and utilization of R genes; (4) validated effective R genes that are readily available for improving genetic resistance; (5) host genetic markers that can be used to accelerate the development of new resistant germplasms/cultivars; and (6) an improved understanding of resistance mechanisms that can facilitate the engineering of resistance in commercial varieties. Appropriate employment of these tools in breeding and crop protection will reduce production costs and create an environmentally benign, sustainable rice production system.

Keywords: resource, resistance gene, avirulence gene, interaction, innate immunity

1. Introduction

In the twentieth century, researchers around the globe focused on studying plant pathogens to develop effective pesticides and cultural practices. Since the late twentieth century, this focus has shifted to identifying resistant resources, effective resistance (R) genes, and deploying them in precision agricultural systems. Rice has been grown in the United States for over 300 years and is concentrated in the Southern US, including the states of Arkansas, Mississippi, Missouri, Louisiana, Texas, and California. Among them, Arkansas is located in south-central USA at ~35° N latitude, 92° W longitude and produces ~50% of the total rice production in the USA. The total annual acreage of rice in the USA is presently about 1.5 million hectares, producing about 2% of the total world rice production. Rice is being consumed domestically and/or utilized as by-products. Recently, more rice is being consumed domestically, but the majority of rice produced in the USA is exported. As a result, the USA is one of the top exporting countries in the international market. Rice production in the USA has evolved to a highly mechanized, flood intensive irrigated system with the use of airplanes, tractors, computers, lasers, fertilizers,

and pesticides at its disposal. Yield per hectare is currently about 7.5 tons/hectare [1] and has been one of the top breeding priorities. Rice breeding programs in the USA are associated with private companies such as Rice Tech Inc., BASF, and major state university agriculture experiment stations, consisting of one or more rice breeders, pathologists, and other scientists. Additionally, the USDA Agriculture Research Service (ARS) has conducted research in Stuttgart, Arkansas, since 1931 [2]. Soon after the establishment of the USDA, ARS, Dale Bumpers National Rice Research Center (DB NRRC, 1998), the molecular plant pathology program has been performing translational research to tackle the major constraints of rice production.

1.1 Rice blast disease

One of the major constraints for rice production in the USA is rice blast. Blast disease of rice is caused by the filamentous fungus *Magnaporthe oryzae*

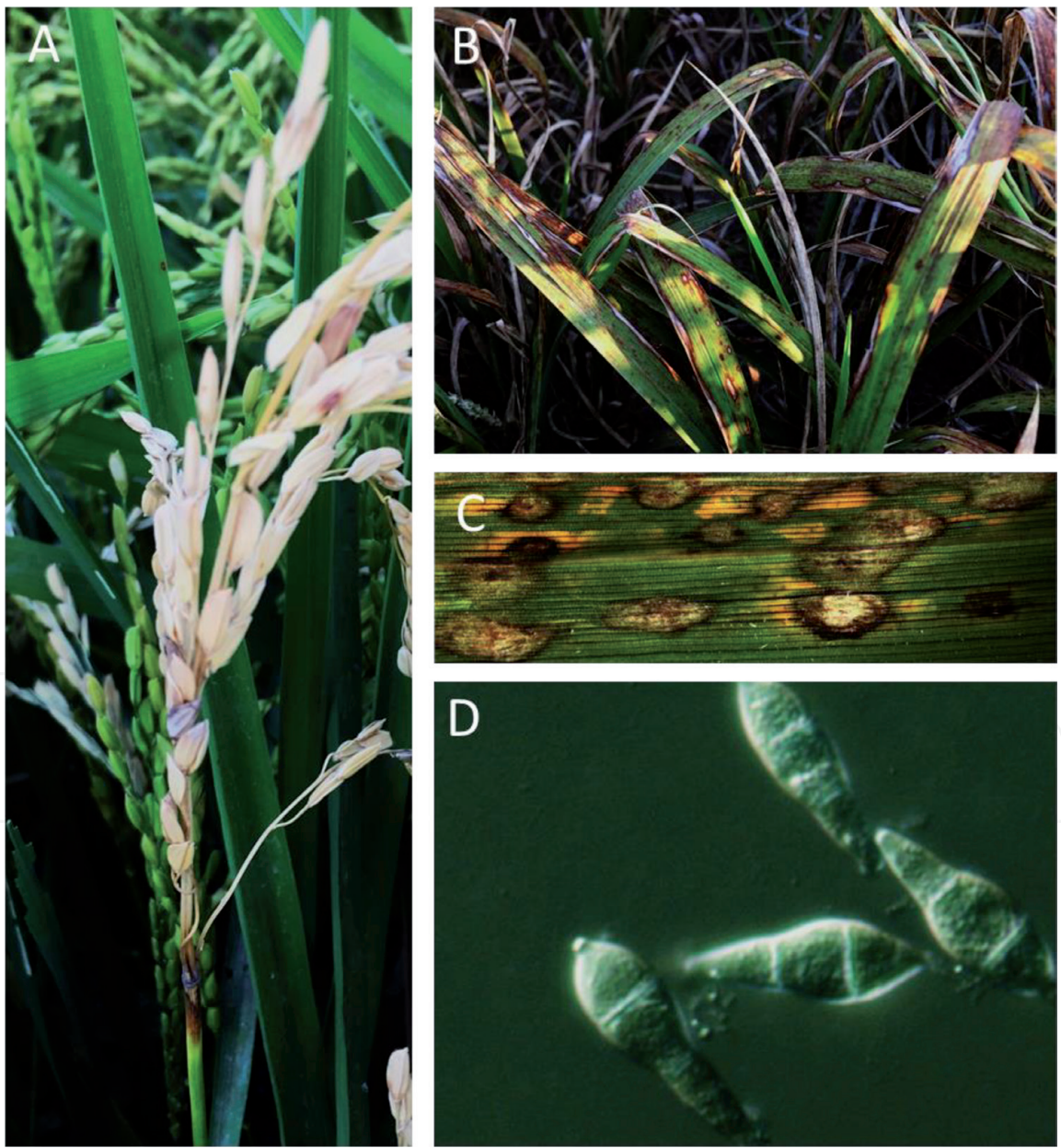


Figure 1. Photographs showing symptoms of leaf and panicle blast and asexual spores of rice blast fungus. (A) Panicle damage caused by blast; (B) severe blast lesions on rice seedlings affecting rice seedling establishment; (C) blast lesions on a rice leaf after diseased leaf from a field was placed in a petri dish with a prewetted filter paper for 24 h; and (D) four asexual spores of the rice blast fungus [9]. The pictures were taken either with an iPhone, with a dissecting microscope, or with a Nikon eclipse microscope.

(synonymous with *Pyricularia oryzae*) which belongs to the *M. grisea* species complex. The *M. grisea* species complex is known to infect a wide range of monocots causing numerous diseases. However, infection of *M. oryzae* is highly specific to its host—rice (*Oryza sativa*) [3]. The infection of a *M. oryzae* isolate to an alternative species was only demonstrated under greenhouse conditions [4]. *M. oryzae* is a polycyclic pathogen that can reproduce 3–5 generations during a single crop season depending on geographic regions [5]. *M. oryzae* can survive in debris and seeds from previous crop seasons, and the fungi carrying debris and seeds are the primary sources of inoculum for blast epidemics [6–8]. Infection of *M. oryzae* starts with asexual conidia. The conidia germinate within a few hours after attachment and penetrate the host cells. Visible symptoms on rice leaves can be seen as early as 5 days after initial contact. A single blast lesion can produce thousands of conidia within a week and these conidia can spread to another rice plant through air, dew/water, and physical contact. Each conidium is capable of causing the loss of a single rice panicle (Figure 1).

1.2 Sheath blight disease

The soil-borne, necrotrophic *Rhizoctonia solani* species have a wide range of host plant species. The anastomosis group AG1-IA of *R. solani* infects rice and causes sheath blight disease. *R. solani* is a monocyclic fungus. The life cycle of *R. solani* begins with mycelia growth from sclerotia soon after attachment onto rice seedlings/plants. The mycelia then move upward along the sheaths and leaves of rice plants, ultimately resulting in damages on the sheaths, leaves, and grains. The life cycle ends with the formation of overwintering structures, sclerotia on the sheaths, leaves, seeds, and in soils [10] (Figure 2).

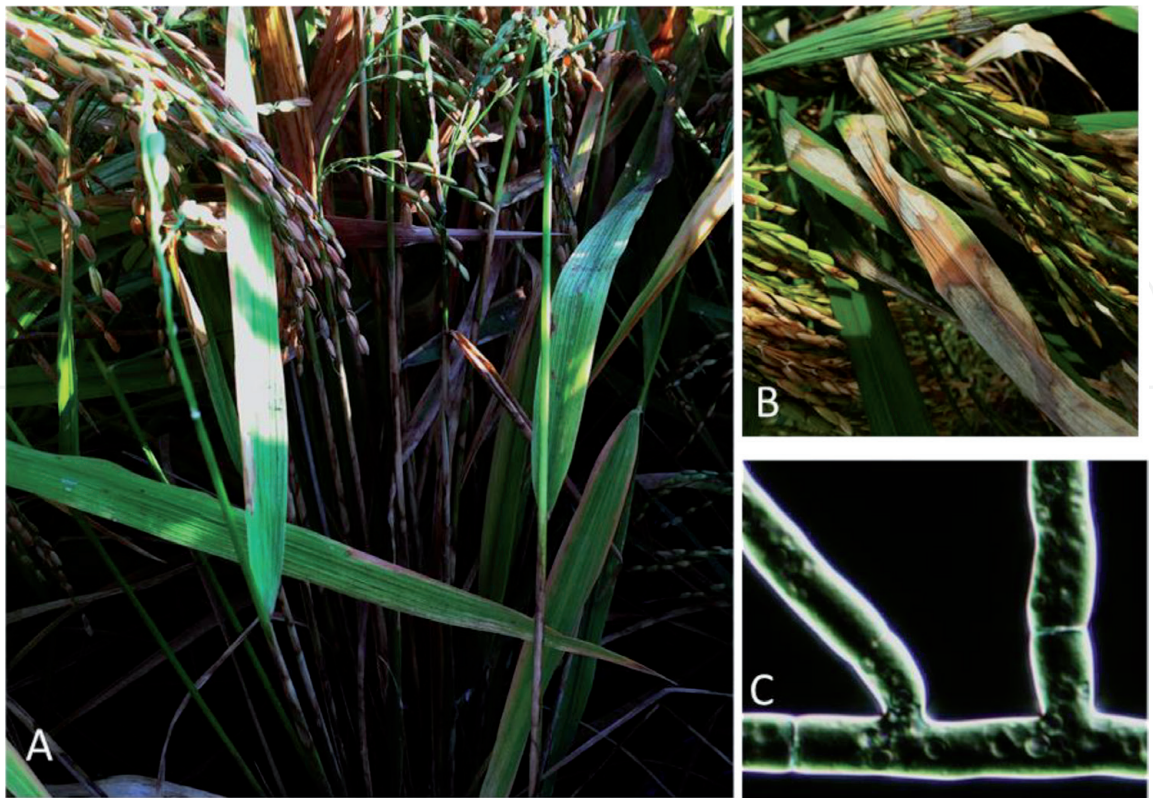


Figure 2. Photographs showing sheath blight disease on the sheaths, leaves, and grains (A and B) and young mycelia sheath blight fungus with 45 and 90° angles (C). Pictures were taken with an iPhone or with a Nikon eclipse microscope.

1.3 The epidemics, climate, and damages

In the Southern US, rice blast disease can be found annually and occasionally results in significant crop damages. However, sheath blight disease occurs more often than blast disease partially due to high-density cultivation. An extended dew period and light are known to stimulate sporulation of *M. oryzae*. Light rain is known to keep plant surfaces wet and create near 100% relative humidity, helping the attachment and penetration of the conidia of *M. oryzae*. High humid conditions also favor the growth, infection, and spread of *R. solani* to other leaves and other plants [10]. In California, there is no rain during the rice-growing season. As a result, significant yield loss due to blast has not been reported [11]. Sheath blight disease has not been reported in California either despite a phenotypically similar disease, the aggregate sheath spot of rice caused by *Rhizoctonia oryzae-sativae* [12], commonly occurring. Presently, substantial fungicides have been used to prevent crop losses of these fungal diseases in the USA.

2. Pathogen genomic identity and pathogen avirulence (AVR) genes

Knowledge of pathogen populations is important to identify effective *R* genes and develop long-lasting strategies to prevent crop loss due to diseases. DNA fingerprint based on MGR586, mitochondrial DNA Restriction Fragment Length Polymorphism (RFLP), mating type, vegetative compatibility, virulence, DNA sequencing, simple sequence repeat (SSR) markers, and avirulence (AVR) gene analyses have been interchangeably used to characterize *M. oryzae* populations [13–20]. The genetic identity of blast populations evaluated by SSR is not significantly different among rice production areas in the Southern US. The identity, however, is significantly different over the past 6 decades [19], suggesting that the environmental dynamics overtime such as weather, deployed rice varieties, and soil fertility in these years may play important roles in shaping the genetic identity of blast fungi. The pathogenicity of blast races (isolates) has been routinely evaluated with the international rice differential system since 1960s [20]. The most commonly found blast races are IB1, IB17, IB49, and IC17 while IA1, IA37, IA65, IA69, IA113, IB21, IB25, IB37, IB41, IC1, IC9, IE1k, IG1, and IH1 are the least commonly found blast races in the Southern US, whereas in California, IG1 is the only predominant blast race [19]. Similar blast races to those in the Southern US were also found in the winter nursery for the Southern US rice breeders in Puerto Rico [21].

The fungi purified from sheath blight-like diseased samples were evaluated with DNA markers, anastomosis grouping, speed of *in vitro* growth, and infection assays with detached leaf and microchamber assays [22]. All sheath blight-causing agents in 102 rice samples were determined to be *R. solani* with a diagnostic DNA marker derived from a ribosomal DNA internal transcribed spacer. Anastomosis grouping tests were conducted in cooperation with Dr. Craig Rothrock's lab (Department of Plant Pathology, University of Arkansas, Fayetteville, Arkansas, USA). A total of 13 testers, namely, (ID A1 1-4, AG-B1); (ID521, AG-9); (ID CI, AG-8); (ID1529, AG-7); (NTA3-1, AG-6); (ID ST6-1, AG-5); (ID AH-1, AG-4); (ID W14 L, AG-3); (ID RI-64, AG2-2); (ID F56 L, AG2-1); (ID M43, AG1-1C); (ID Cs-Ka, AG1-IA); and (ID SFBV-1, AG1-IB), from different hosts were used. All the 102 isolates were determined to be IG1-IA. Three groups—fast growing (such as RR0321-4, RR0319-8, RR0101-1); intermediate growing (such as RR0305, RR0316-1); and slow growing (such as RR0316-1, RR0140-1, RR0141-1)—were identified by measuring the growth of each isolate in a nutrient-supported petri dish. The speeds of growth were found to be closely correlated with the lengths of disease

lesions in the detached leaves of two rice varieties, suggesting that the fast-growing isolates were more virulent than those of slow-growing isolates [22]. These characterized isolates have been used to identify genetic resistance and molecular studies ever since.

3. Mapping populations and improved rice germplasm and genetic stocks

3.1 Mapping populations

Rice germplasms with different *R* resources is a prerequisite for developing improved rice varieties with *R* genes providing overlapped resistance to various blast races (isolates). In the Southern US, tropical japonica rice varieties are mainly grown, whereas in the state of California, temperate japonica rice varieties are grown. Major resistant resources to *M. oryzae* in the Southern US are mainly from indica rice varieties such as Tetep, Te Qing, and Zhe733. Complete resistant resources to *R. solani* have not been identified; however, moderate resistance from rice germplasms such as Jasmine 85 has been identified. These resistant resources were used to develop mapping populations and adaptive germplasms through single seed descend and doubled haploid breeding strategies (Figure 3 and Table 1).

3.2 Improved rice germplasms and genetic stocks

Germplasms with improved resistance to both blast and sheath blight diseases are helpful for rice breeders to develop new rice cultivars [34]. Four rice germplasms, LJRIL103 (PI 660982), LJRIL158 (PI 660983), LJRIL186 (PI 660984), and LJRIL220 (PI 660985), with resistance to both blast and sheath blight diseases were



Figure 3.
Photograph showing a view of the rice research plots of USDA ARS DBNRRC and the University of Arkansas Rice Research Center, Stuttgart, Arkansas, USA, 2016. Most rice resources and mapping populations were advanced in similar field plots. The picture was taken with a drone in 2016.

Name of genetic sources	Plant identification	Key information	Number	Year of release	Reference
C/M doubled haploid	GSOR 200001–200325	Sheath blight resistance	325	2006	[23]
Early/Katy mapping population	GSOR 100361–100600	Blast resistance	240	2007	[24]
K/Z mapping population	GSOR100001– 100355	Molecular map/blast resistance	355	2007	[25]
SB5 mapping population	GSOR 101601–102,174	Blast and sheath blight resistance	574	2009	[26]
Katy//M202 backcrossing lines	GSOR 102501–102544	Blast resistance	42	2012	[27]
Weedy red rice mapping population 1	GSOR 303101–303287	Blast	187	2015	[28]
Weedy red rice mapping population 2	GSOR 303301–303536	Blast and sheath blight resistance	236	2015	[28]
USDA core collection	GSOR 310001–311795	Blast resistance	1795	2015	[29–33]

Table 1.
List of major genetic resources for blast and sheath blight resistance in the USA. Most of the rice germplasms are available at USDA-GSOR (www.ars.usda.gov/GSOR).

identified. They were identified from 800 progenies of a cross between US-adapted rice germplasm Lemont with Jasmine 85 [26]. These germplasms contain suitable agronomic traits in addition to the aromatic nature of LJRIL103, LJRIL158, and LJRIL186. Disease resistance and aromatic genes were tagged with DNA makers to ensure their incorporations.

Loss-of-function mutants can help identify the functionality of the corresponding wild-type allele [35]. For example, lesion mimic mutants (LMMs) with a phenotype resembling hypersensitive cell death without pathogen attack are useful for studying the molecular basis of plant innate immunity. A rice LMM was identified from the rice cultivar Katy after treatment with fast neutrons [36]. The severe lesion mimic phenotype of LMM1 can be induced by blast pathogens and water-related stress, respectively (M.S. Jia and Y. Jia, unpublished data). LMM1 has an enhanced resistance to both blast and sheath blight disease [36]. Genetic analysis suggests that a single recessive gene is responsible for the lesion mimic phenotype in LMM1. Further characterization of the underlying gene in LMM1 will help elucidate the mechanisms of plant innate immunity and abiotic stress responses.

The abovementioned mapping populations, characterized rice germplasms and genetic stocks, are now being used to map and clone *R* genes to both rice blast and sheath blight disease and develop DNA markers for marker-assisted breeding [37].

4. User-friendly disease evaluation methods

In the Southern US, genetic resistance to *M. oryzae* was investigated by Drs. Atkins, Johnston, and Marchetti [20, 38, 39]. Analyses of disease reactions to

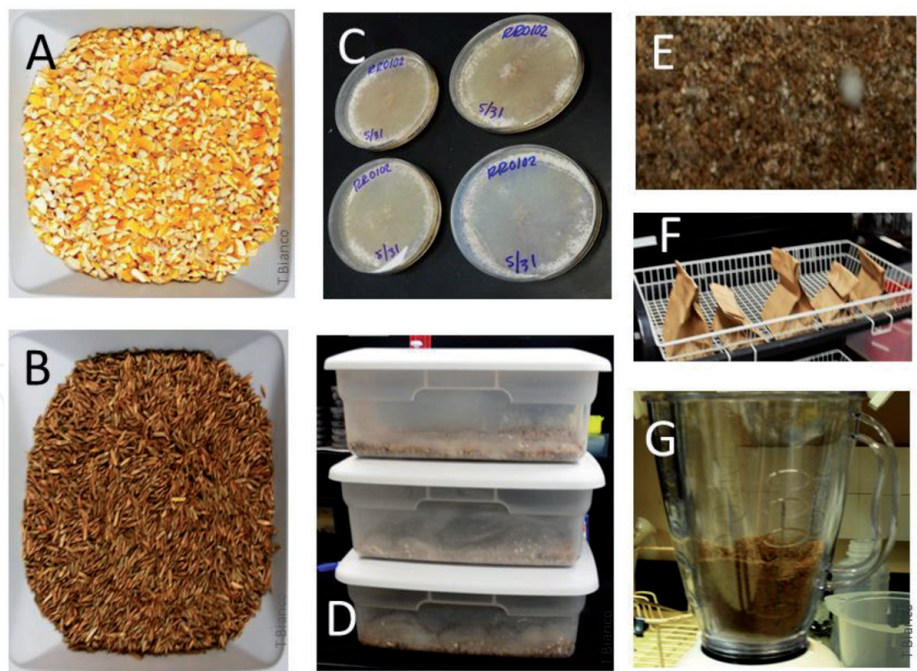


Figure 4. Photographic presentation of the massive production of the sheath blight inoculant for field evaluation. Step 1: mixing A (corn chips) and B (rye) in a 2:1 weight ratio, adding water, and autoclaving twice. Step 2: growing mycelia in petri dishes containing PDA media until the appearance of white sclerotia (C). Step 3: mixing mycelia from C with a mixture of A and B from step 1, and incubating in a sterilized plastic or metal container for 3–5 days until the appearance of white sclerotia (D and E). Step 4: air drying mycelia and sclerotia in brown bags at 24°C with a fan (F). Step 5: grinding mycelia with a grinder (G) before inoculating plants under field conditions.

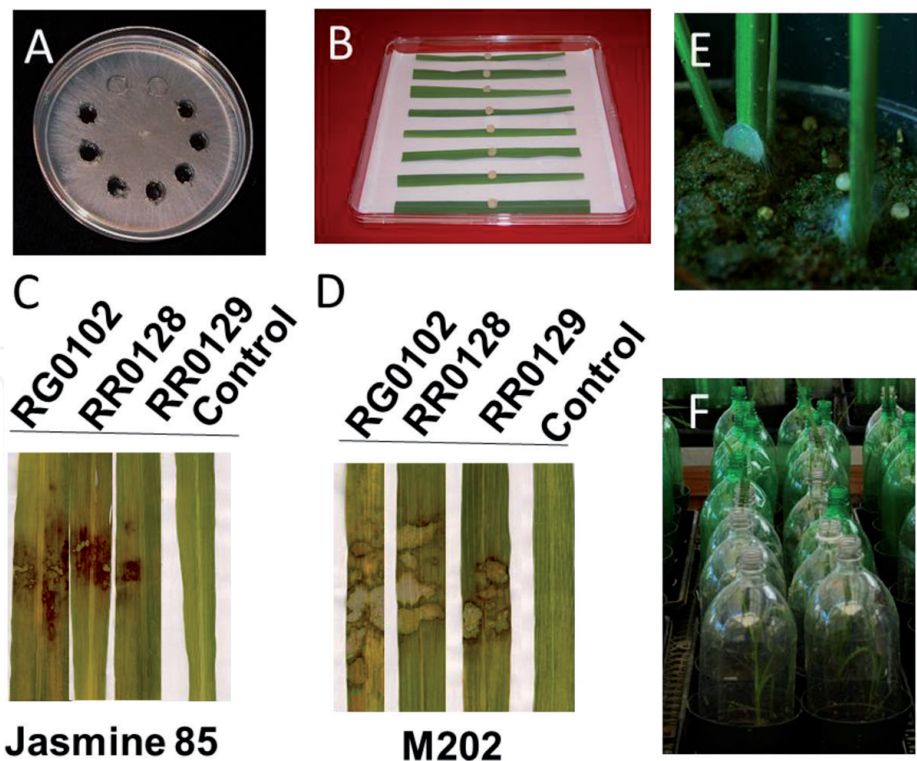


Figure 5. Photographic presentation of two controlled sheath blight evaluation methods. (1) Detached leaf method: mycelia grown on PDA media (A), and PDA plugs removed from a were placed onto detached leaves (6–12 cm in length) (B) at 24°C for 3 days. Symptoms of detached leaves from rice varieties jasmine 85 and M202 after inoculations with three *R solani* isolates versus the control PDA without pathogens (C and D). (2) Soft-drink bottle method: PDA plugs from A were placed onto the bottom of sheaths (E) and covered with 2-L soft-drink bottles (F) for 3–5 days until stable symptoms appeared. Length of lesions was measured for both methods as the severity of disease reactions.

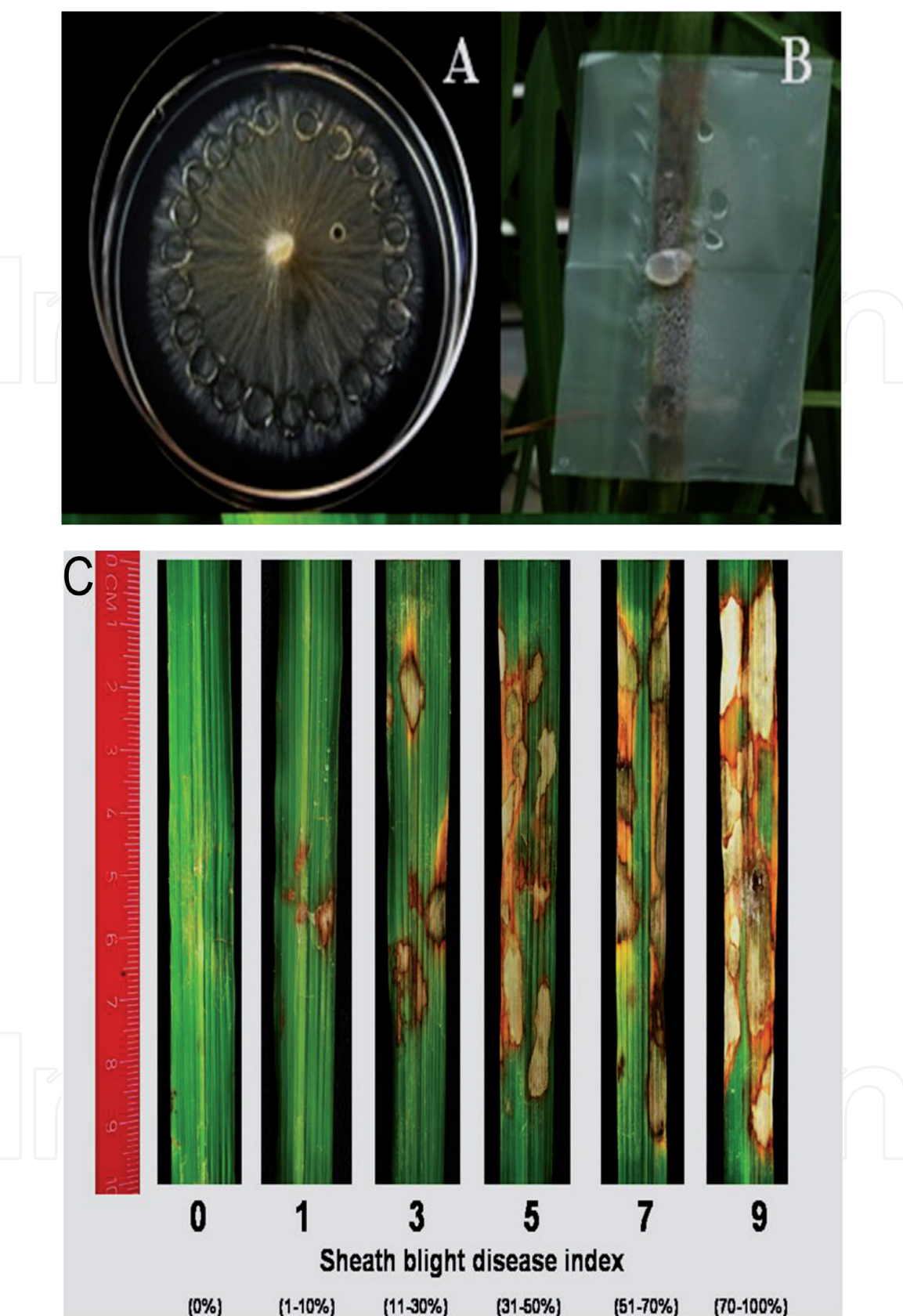


Figure 6. Parafilm method for sheath blight disease evaluation: a PDA containing mycelia (A) in a petri dish containing mycelia after 3 days of culturing at 30°C was removed and covered with parafilm and wrapped onto the second youngest leaf for 3–5 days (B) until stable symptoms appeared. A rating scale based on visual length and area of symptoms was assigned as indicated, with 0 representing immunity and 9 representing extreme susceptibility (C).

M. oryzae have been performed under field conditions where complex biotic and abiotic factors impacting the inheritance of resistance were encountered resulting in inconsistencies of disease reactions. In 1999, Dr. Marchetti and his

colleagues demonstrated that disease reactions under an upland blast nursery were reliable to identify *R* genes among breeding lines [40]. Under greenhouse conditions, the phenotypes of rice to *M. oryzae* are categorized as 0–5 where 0 represents complete immunity, 1 represents hypersensitive cell death showing tiny brown spots, 2 represents infected lesions without mycelia, and, for susceptible reactions, 3–5 exhibit different sizes of lesions with visible mycelia coincident with different levels of resistance [32]. Phenotypes evaluated under the upland rice blast nursery were verified with 200 individuals of a mapping population under greenhouse conditions at DBNRRC [41]. Since then, the greenhouse methods have been used to determine the inheritance and genetic mechanisms of blast resistance [32, 41, 42]. In 2015, several IRRI monogenic lines generously donated by IRRI were added to further identify blast *R* genes under greenhouse conditions [43].

The early evaluation of sheath blight relied on replicated field plot experiments with fungal mycelia grown in corn chips or rye (**Figure 4**).

Disease reactions were scored by visually rating the disease severity on the sheaths and leaves of whole plants. The results of the evaluations are useful for mapping *R* genes. As an alternative, greenhouse methods such as detached leaf, soft-drink bottles, and parafilm methods were developed to validate and verify the function of *R* genes (**Figures 5 and 6**). These greenhouse methods are being used routinely for initial *R* gene discovery because they use less time, labor, land, and fertilizer.

5. Effective *R* genes

5.1 Effective major *R* genes

A total of 14 known major blast *R* genes have been used in the USA since 1960s. **Table 2** lists their chromosomal locations, representing germplasms, DNA markers to monitor respective *R* genes, and the avirulent and virulent races of these selected rice germplasms (**Table 2**). Based on field observations, most blast *R* genes are dominant whereas a single haplotype of *R* gene is effective for resistance. Among them, six dominant blast *R* genes *Pia*, *Piks*, *Pi66(t)*, *Pikh*, *Pikm*, and *Pi43(t)/Pi1*, and one recessive *R* gene *pid* were on chromosome 11. Comprehensively, one was found on chromosome 2, two on chromosome 6, one on chromosome 8, one on chromosome 9, and two dominants on chromosome 12. Three of the dominant *R* genes, *Pi9*, *Pi42(t)*, and *Pi43(t)*, provide resistance to all races, while *Pita2/Ptr* is effective to all races except IE1k.

The genetic markers linked or derived from the cloned *R* genes were developed to predict resistance function and to monitor the existence of each of the *R* genes [31–33, 44–52]. Differential blast races were identified (**Table 2**) and have been used to validate their predicted resistance efficacies.

5.2 Effective minor *R* genes

Distinct phenotyping variation of rice after infection via *M. oryzae* in different rice germplasms and in the same germplasm at different growth stages under greenhouse [53] and field conditions are also referred as dilatory, partial, field, and adult resistance interchangeably [54]. A total of 11 blast *R* quantitative trait loci (QTLs) responsible for a phenotypic variation ranging from 5.17 to 26.53% were identified with different blast races under greenhouse conditions [55] (**Table 3**) and verified with different blast isolates/races [56]. Using the same method, four additional blast *R* QTLs were identified from different rice germplasms [57].

Chr.	Name of R gene	Selected germplasm	Marker	Name of blast races		Reference
				Avirulence	Virulence	
2	<i>Pi-b</i>	Saber, Te-Qing	RM208, Pib dom	IB1, IB45, IH1, IG1, IC17, IE1, IE1k	IB49, IB54	[31]
6	<i>Piz(t)</i>	Zenith	RM527, AP4791, AP5659-1, AP5659-5	IH1, IG1, IC17, IE1k	IA45, IB1, IB49, IB54, IB33	[32]
6	<i>Pi9</i>	IR9660- 48-1-1-2 (GSOR310687)	KS6/KS28	IA45, IB1, IB49, IB54, IB45, IH1, IG1, IC17, IE1, IE1k		[33]
8	<i>Pi42(t)</i>	Zhe733	RM72	IA45, IB1, IB49, IB54, IB45, IH1, IG1, IC17, IE1, IE1k		[44]
9	<i>Pii</i>	Dawn		IH1		[39]
11	<i>Pia</i>	Bluebonnet		IB1		[39]
11	<i>Pikh</i>	Lebonnet	RM224	IB45, IB54, IH1, IG1	IB49	[45]
	<i>Piks</i>	M2354	E/P RM224	IB54	IA45, IB49, IB33, IB45, IH1, IG1, IC17, IE1, IE1k	[46]
11	<i>Pi66(t)</i>	DGWG		IB54	IB45, IC17, IG1, IH1	[47]
11	<i>Pikm</i>	Tsuyuake	Q/P RM224	IB45, IB54, IH1, IG1	IC17	[46]
11	<i>Pi43(t)/ Pi1</i>	Zhe733	RM1233	IA45, IB1, IB49, IB54, IB45, IH1, IG1, IC17, IE1, IE1k		[44]
11	<i>Pid</i>	Lebonnet		IB1	IA45, IB49, IB54, IB45, IH1, IG1, IC17, IE1, IE1k	[45]
12	<i>Pita</i>	Katy	YL100/YL102, YL155/YL87	IB49, IC17	IE1k	[29, 48–50]
12	<i>Ptr (Pita2)</i>	Katy	HJ16–12	IA45,IB1, IB49, IB54, IB45, IH1, IG1, IC17, IE1	IE1k	[51, 52]

Table 2.
DNA markers and resistance efficacies of deployed blast R genes in the USA since 1960 (Figure 7).

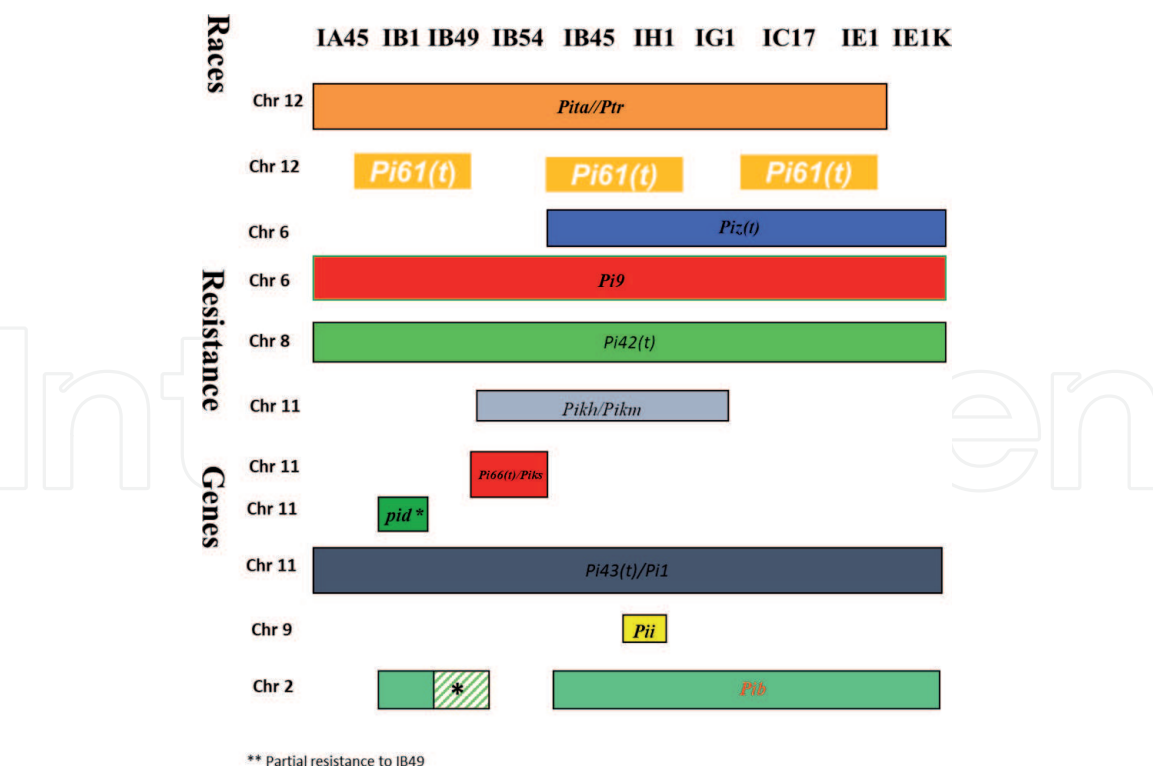


Figure 7.
Graphic presentation of resistance spectra of blast R genes in the USA. The common races, name of R genes, and chromosomal locations are indicated.

QTL	Chr.	Blast race	Marker interval	Nearest marker locus (physical location in MB)	Phenotypic variation (%)	Nearest major R genes
<i>qBLAST3</i>	3	IB45	RM251–RM338	RM282 (12.4)	5.17	
<i>qBLAST8.1</i>	8	IB49	RM6863–RM72	RM1148 (4.0)	6.69	<i>Pi36</i>
<i>qBLAST8.2</i>	8	IC17	RM310–RM72	RM72 (6.8)	7.22	
<i>qBLAST9.1</i>	9	IB54	RM257–RM108	RM257 (17.7)	4.64	
<i>qBLAST9.2</i>	9	IC17	RM257–RM107	RM108 (17.9)	7.62	NBS-LRR
<i>qBLAST9.3</i>	9	IC17	RM107–RM245	RM215 (21.2)	4.49	
<i>qBLAST11</i>	11	IB45	RM206–RM224	RM224 (27.8)	26.53	<i>Pikm/Pik</i>
		IB54	RM206–RM224	RM224 (27.8)	19.6	
<i>qBLAST12.1</i>	12	IB1	RM6998–OSM89	OSM89 (7.9)	5.44	<i>Pi-ta/Ptra</i>
<i>qBLAST12.2</i>		IB49	RM247–RM277	OSM89 (7.9)	9.7	
		ID1	RM247–RM277	OSM89 (7.9)	10.18	

Chr. indicates chromosome, MB indicates megabase pair, NBS-LRR indicates the protein with nucleotide-binding sites—leucine-rich repeat domain is often encoded by the R gene.

Table 3.
List of minor resistance genes to rice blast disease with indicated nearby major R genes and NBS-LRR proteins [55, 56].

Thus far, major sheath blight *R* genes have not been identified. However, the major sheath blight *R* QTL *qShB9-2* responsible for 24.3–27.2% of phenotypic variation using microchamber and mist chamber assays, respectively, and other nine minor *R* QTLs to sheath blight were also identified [58, 59]. These sheath blight *R* QTLs were verified with replicated field plot experiments in multiple locations [60]. This demonstrated that there exist useful genetic factors that can be used for breeding. DNA markers linked to these *R* QTLs can not only be used to pyramid resistance into new rice varieties via marker-assisted breeding but can also be used to clone and characterize genes underlying these *R* QTLs.

6. Resistance effectiveness

M. oryzae is a hemi-biotrophic organism with an extended period of biotrophic invasion that forced the evolution of robust major blast *R* genes in host. The resistance mediated by major blast *R* genes follows the gene-for-gene model where the *R* genes in rice detect the corresponding *AVR* genes in *M. oryzae* in triggering resistance responses [61]. The existence of *AVR-Pita1* in US blast populations suggest that *AVR-Pita1* may play an important role in fitness and pathogenicity. Ironically, what is needed for pathogens to survive also makes the pathogen less virulent and fit. This never-ending booming-and-busting cycle of host-pathogen interactions presents a unique opportunity to develop durable resistance. In the Southern US, after the blast epidemics in 1980s, a blast-resistant rice variety Katy was released in 1990 [62]. Katy contains a cluster of major *R* genes at the *Pi-ta* locus from the landrace indica variety Tetep and *Piks* from tropic variety Newbonnet [41]. Further analysis of Katy revealed that there are three linked blast *R* genes, *Pi-ta* and *Pi-ta2/Ptr* genes near the centromere of rice chromosome 12. *Pi-ta* is a classical *R* gene with NBS-LRR [63] and *Ptr*, which is allelic to *Pi-ta2*, encodes a predicted protein with four armadillo repeats [52]. *Ptr* was shown to confer resistance to a wide range of blast races except for IE1k and help *Pi-ta* with unknown mechanisms [52]. To date, a handful of rice varieties with the *Pita*, *Pita2/Ptr* cluster in a linkage block including Katy, Drew, Madison, Kaybonnet, Cybonnet, Banks, Ahrent, Catahoula, and Templeton have been released in the Southern US since 1990 [64–66]. Amei and colleagues showed that the *Pi-ta* gene has been bred into cultivated species of rice for decades [67]. The counter resistance from the pathogen usually occurs after breeders release a new resistant rice variety [68]. One of the counter resistance strategies of *M. oryzae* is to alter the structural integrity and expression of the *AVR* genes. The blast races (isolates) with partial, complete deletions, point mutations altering amino acids, and transposon insertions at the *AVR-Pita1* locus have been found in commercial rice fields in the Southern US since the release of *Pi-ta* [16–18]. The resistance mediated by the *Pi-ta/Pi-ta2/Ptr* gene cluster has been stable for over two decades. Consistently, most blast populations were found to carry *AVR-Pita1* [16–18] that verified the durability of resistance mediated by *Pi-ta/Pi-ta2/Ptr*. The observed resistance durability could be due to the lack of deployment of rice cultivars with the *Pi-ta/Pi-ta2/Ptr* genes to force the loss of *AVR-Pita1*. This is consistent with the fact that limited *Pi-ta/Pi-ta2/Ptr* containing rice varieties have been grown due to moderate yield advantages compared to other rice varieties lacking the genes since their releases [69]. Alternatively, it is also fully possible that *AVR-Pita1* is important for the survival of *M. oryzae* with unknown mechanisms.

7. Summary

In the USA, any rice cultivar with one or two major blast *R* genes will continue to be effective to prevent rice blast disease. On the other hand, a combination of major

R QTLs, suitable plant architecture, and growth rate should be considered to prevent sheath blight disease. A defense gene expression and cell reaction study suggested that strong resistance responses mediated by *Pi-ta* could be initiated as early as 24 h after pathogen inoculation [70]. However, the molecular mechanisms underlying *Pi-ta* or *Ptr*-mediated disease resistance pathways [71], the interactions between major blast R genes and R QTL [74], the role of micro RNA/long noncoding RNA in rice disease resistance [72–75], and the relation of resistance versus productivity are still largely unclear [69]. Therefore, a clear understanding of the abovementioned plant innate immunity systems will be required for engineering resistance via genome editing. The lack of robust major R genes to *R. solani* may be due to the saprophytic nature of *R. solani* where the pathogen feed on the dead tissue of rice plants. Comparative analysis of defense genes in different hosts of *R. solani* may help identify useful R genes [76]. The genome of *R. solani* is mosaic and the draft sequence of *R. solani* IG1-IA genome is readily available [77]. Moving forward, the completion of whole genome sequencing will be the next urgent step to identify clues to manage *R. solani*. In brief, continued identification and characterization of R genes will be essential to safeguard rice crops. Ultimately, fungicides will be significantly reduced to prevent rice blast and sheath blight diseases in the future.

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Author details

Yulin Jia^{1*}, Melissa H. Jia¹, Xueyan Wang^{1,2} and Haijun Zhao¹

¹ USDA ARS, Dale Bumpers National Rice Research Center, Stuttgart, Arkansas, USA

² University of Arkansas Rice Research and Extension Center, Stuttgart, Arkansas, USA

*Address all correspondence to: yulin.jia@ars.usda.gov

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