Preliminary Study on the Molecular Mechanism of Agricultural Meteorological Factors Influencing Rice Blast Occurrence - Postprint

Authors: Liu Tianhua, Jiaojiao Bai, Dongping Lü

Date: 2017-11-06T00:00:00+00:00

Abstract

Rice is a crucial food crop in China. Rice blast disease, caused by the ascomycete fungus *Magnaporthe oryzae*, represents one of the major limiting factors in rice production, inflicting substantial economic losses annually. Following the completion of whole-genome sequencing initiatives for both rice and *Magnaporthe oryzae*, the interaction mechanisms between rice and the blast pathogen have become increasingly elucidated. Rice resistance to *Magnaporthe oryzae* primarily derives from the effective blockage of pathogen invasion through its intrinsic innate immune system, whereas the pathogen can suppress rice basal immunity to cause disease in susceptible cultivars. The outbreak and epidemic of rice blast disease necessitate three essential conditions: susceptible rice varieties, pathogenic populations of *Magnaporthe oryzae*, and meteorological factors conducive to pathogenesis. Consequently, meteorological factors constitute a critical element in controlling the outbreak and epidemic of rice blast disease. This review synthesizes recent research findings, summarizing the interaction mechanisms between rice and *Magnaporthe oryzae* with respect to the fungal infection process, disease development and characteristics, and rice disease resistance mechanisms. Additionally, it analyzes the impacts of meteorological factors including temperature, light, and humidity on fungal pathogenicity and rice disease resistance, and preliminarily explores the molecular mechanisms by which these factors influence the outbreak and epidemic of rice blast disease, aiming to identify optimal strategies for preventing and controlling the occurrence and development of rice blast disease, thereby providing theoretical foundation and guidance for rational management of this disease.

Full Text

A Preliminary Study on the Molecular Mechanisms by Which Agro-Meteorological Factors Influence Rice Blast Occurrence

LIU Tianhua[1],[2],†, BAI Jiaojiao[1],[2],†, LYU Dongping[1],

[1]Center for Agricultural Resources Research, Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, Shijiazhuang 050022, China
[2]University of Chinese Academy of Sciences, Beijing 100049, China

Abstract

Rice is a staple food crop in China. Rice blast, caused by the ascomycete fungus *Magnaporthe oryzae*, represents one of the most significant constraints on rice production, causing substantial economic losses annually. With the completion of whole-genome sequencing of both rice and *M. oryzae*, the molecular mechanisms underlying their interactions have become increasingly clear. Rice resistance to blast primarily depends on its innate immune system effectively preventing pathogen invasion, while the pathogen can suppress this basal immunity to cause disease in susceptible varieties. The outbreak and epidemic spread of rice blast require three essential conditions: susceptible rice cultivars, virulent pathogen populations, and favorable meteorological conditions. Thus, meteorological factors play a crucial role in controlling blast epidemics. This review synthesizes recent research findings on rice-*M. oryzae* interactions, covering the infection process, disease development patterns, and resistance mechanisms. We analyze how meteorological factors—including temperature, light, and humidity—affect pathogen virulence and host resistance, and explore the molecular mechanisms through which these factors influence blast outbreaks. Our goal is to identify optimal strategies for blast prevention and control, providing a theoretical foundation for rational disease management.

Keywords: Rice blast; *Magnaporthe oryzae*; Meteorological factors; Molecular mechanism; Disease resistance mechanism

---

Rice blast is one of the most devastating diseases of rice (*Oryza sativa*), occurring in both northern and southern rice-growing regions of China. Under severe conditions, it can reduce rice yields by approximately 40–50% [1]. For instance, a blast outbreak in Bengbu, Anhui Province in 2014 caused extensive yield losses in the 'Liangyou 0293' hybrid rice, with some fields experiencing complete crop failure [2]. The causal agent, *Magnaporthe oryzae*, is a hemibiotrophic filamentous ascomycete that can infect dozens of plant species, including economically important cereals such as wheat and rice [3]. The whole-genome sequence of *M. oryzae* was completed as early as 2005 [4], revealing a genome of approximately 40 Mb encoding about 11,000 genes [5].

Like other crop diseases, the outbreak and epidemic spread of rice blast require three essential conditions: susceptible rice varieties, virulent pathogen populations, and favorable environmental and meteorological conditions [6]. Meteorological factors thus play a critical role in disease development. Suitable environmental conditions can trigger blast epidemics, whereas even when virulent pathogens contact susceptible rice plants, disease may not occur if meteorological conditions are unfavorable [7].

In recent years, research on rice-*M. oryzae* interactions has advanced considerably. However, studies on the molecular mechanisms by which meteorological factors such as light, temperature, and humidity affect pathogen virulence and rice resistance are still in their infancy. A comprehensive understanding of how meteorological factors influence disease development and the underlying mechanisms is essential for guiding effective blast control strategies.

2. Disease Cycle and Characteristics of Rice Blast

Rice blast primarily occurs during the seedling, tillering, and heading stages. Based on the infected plant part, blast can be classified as leaf blast, node blast, panicle blast, or neck blast [16]. The disease cycle comprises four stages: transmission, penetration, incubation, and secondary infection [8]. After airborne conidia land on rice leaves, they germinate and form appressoria that penetrate the cuticle and invade epidermal cells within approximately 6 hours. Following penetration, the disease enters an incubation period whose duration depends on ambient temperature. At 24–25 °C, the incubation period lasts about 5–6 days, though it is longer for panicle blast. Rice cultivar and leaf age also affect incubation length [17]. After incubation, lesions develop on infected leaves. Under favorable temperature and humidity conditions, these lesions produce massive numbers of conidia that detach and spread via air currents to other plants, initiating new infection cycles [18].

3. Mechanisms of Rice Resistance to *M. oryzae*

The infection process can be divided into five steps: (1) spore adhesion and germination, (2) germ tube formation, (3) appressorium development, (4) penetration peg formation, and (5) hyphal growth within host tissue [8]. Specifically, airborne conidia land on rice leaves and secrete mucilage at their tips for firm attachment. When temperature reaches 25–28 °C and relative humidity exceeds 90%, conidia germinate and produce germ tubes. As germ tubes elongate, they swell and differentiate into appressoria induced by hydrophobic leaf surface signals [9–11]. During appressorium maturation, massive accumulation of glycerol solutes generates turgor pressure up to 8 MPa, which is converted into mechanical force to drive the penetration peg through the rice epidermis into mesophyll cells [12–13]. A melanin layer between the appressorium cell wall and membrane is crucial for turgor generation. Melanin-deficient *M. oryzae* mutants cannot generate sufficient turgor and thus fail to form penetration pegs, losing pathogenicity [12–13]. After the penetration peg enters mesophyll cells, it ab-

sorbs nutrients and develops primary and secondary hyphae. Secondary hyphae spread to neighboring cells through plasmodesmata [14], and necrotic lesions become visible on leaf surfaces approximately 72 hours post-infection [15].

During co-evolution with pathogens, plants have evolved a two-layered innate immune system: PTI (pathogen-associated molecular pattern-triggered immunity) and ETI (effector-triggered immunity). Upon infection, plant pattern recognition receptors (PRRs) on the cell surface recognize pathogen-associated molecular patterns (PAMPs), activating PTI to defend against invasion. Some pathogens secrete effector proteins to suppress PTI and cause disease, while plants have evolved resistance proteins that recognize specific effectors to activate ETI, often culminating in hypersensitive response (HR) characterized by programmed cell death (PCD) at infection sites [19].

Rice employs both PTI and ETI against *M. oryzae*. Fungal PAMPs include endopolygalacturonases, xylanases, ergosterol, glucans, and chitin. Rice PRRs include receptor-like proteins (RLPs) and receptor-like kinases (RLKs) such as CEBiP, LYP4, LYP6, CERK1, OsBAK1, and OsFLS2. During infection, these PRRs recognize *M. oryzae* PAMPs and transduce signals to activate PTI responses, including reactive oxygen species production, pathogenesis-related (PR) gene expression, and phytoalexin synthesis. Through co-evolution, *M. oryzae* secretes effectors to suppress rice PTI, while rice resistance proteins directly or indirectly recognize these effectors to activate ETI, triggering HR-PCD that restricts fungal growth [19].

To date, over 100 blast resistance genes have been mapped in rice, with 22 cloned. Most encode NBS-LRR proteins containing nucleotide-binding sites (NBS) and leucine-rich repeat (LRR) domains, including *Pi2*, *Pi9*, *Pi36*, *Pi37*, *Pikm*, *Pit*, *Pib*, *Pi-ta*, and *Piz-t* [20]. Additionally, nutrient elements such as silicon affect rice blast resistance. Field experiments showed that silicon application increases leaf silicification and reduces blast incidence [21]. Liu et al. [22] found that exogenous silicon dioxide enhances resistance in both resistant and susceptible rice varieties by reducing fungal hyphae on leaf surfaces. Transmission electron microscopy revealed that mineralized silicon deposits surround hyphae, inhibiting their growth and invasion of mesophyll cells [22].

Using resistant varieties is the most economical and effective blast control measure [20]. Recent molecular breeding efforts have produced several highly resistant rice cultivars [23–24]. However, *M. oryzae* evolves rapidly, generating new physiological races that can overcome host resistance. Moreover, physiological races show regional diversity [25]; a variety resistant in one region may become susceptible in another due to differences in pathogen races and meteorological conditions. Therefore, understanding how meteorological factors affect disease development and their underlying mechanisms is crucial for effective blast management.

4. Molecular Mechanisms of Meteorological Factor Effects on Rice Blast

4.1. Temperature Effects Temperature is among the most important meteorological factors influencing rice blast. Ambient temperature affects *M. oryzae* growth, development, and virulence, as well as rice disease resistance.

4.1.1. Temperature Effects on *M. oryzae* Temperature influences all stages of *M. oryzae* infection. Although many fungal physiological activities can occur across a broad temperature range (e.g., hyphal growth from 8–37 °C), optimal temperatures for hyphal growth, conidiation, germ tube elongation, and appressorium formation are approximately 25–28 °C. When average daily temperatures fall within 24–28 °C, *M. oryzae* thrives and blast epidemics are likely. Conversely, temperatures outside this range inhibit hyphal growth and impair spore development, reducing pathogenicity. At temperatures above 35 °C or below 15 °C, *M. oryzae* grows slowly and develops poorly, resulting in low disease incidence [26]. Nevertheless, the fungus can maintain physiological activity under temperature extremes by synthesizing protective proteins. During hot summers, *M. oryzae* produces heat shock proteins such as MoSf1, which binds to protein kinase Pmk1 to activate the MAPK cascade and enhance thermotolerance [27]. In cold winters, the fungus overwinters as conidia and mycelia on rice straw, requiring cold adaptation for survival. The apoplast-localized adenylate kinase MoAK may be involved in cold adaptation, as its expression increases significantly at low temperatures [28].

Despite these insights into temperature adaptation, the molecular mechanisms underlying differential pathogenicity at various temperatures remain poorly understood. Fundamental studies on temperature-dependent transcriptomic and proteomic changes, particularly regarding effector expression, are still lacking.

4.1.2. Temperature Effects on Rice Disease Resistance Ambient temperature also affects rice disease resistance. Rice, a warm-season crop originating from tropical and subtropical regions, is highly sensitive to low-temperature stress, especially during seedling and booting stages. Prolonged cold stress reduces photosynthetic rate and root water uptake, causing stomatal closure, leaf wilting, and significantly decreased blast resistance [29]. Cold damage is particularly severe in northern Chinese rice-growing regions, though the direct molecular mechanisms linking low temperature to impaired immunity remain unclear. Zhang et al. [30] identified a cold-inducible antifreeze protein gene, XM_{483093}, whose overexpression enhances both freezing tolerance and blast resistance. Transgenic rice overexpressing XM_{483093} show fewer lesions and stronger PR gene expression after *M. oryzae* infection [30]. However, the relationship between cold tolerance and disease resistance is complex. Recent work revealed that the rice WRKY transcription factor OsWRKY76 has dual and opposing functions: overexpression enhances cold tolerance but suppresses PR gene expression and phytoalexin synthesis, significantly reducing blast resistance

[31].

Notably, major breakthroughs have elucidated rice temperature-sensing mechanisms. The COLD1 gene, encoding a G-protein signaling regulator, was identified as a key cold sensor. Upon cold stress, COLD1 interacts with the G-protein α subunit to activate Ca^{2+} channels, triggering cold tolerance responses [32]. Whether COLD1-mediated cold tolerance also affects immune responses under low temperature warrants further investigation. Similarly, the OgTT1 gene, encoding a 26S proteasome $ $2 subunit, was identified as a major QTL for thermotolerance in tropical African rice. The OgTT1 protein enhances proteasomal degradation of ubiquitinated substrates under high temperature, clearing toxic denatured proteins and protecting plant cells [33]. With increasing global climate change and frequent extreme heat events, the impact of high temperature on rice blast resistance and the role of OgTT1 require further study.

4.1.3. Temperature Effects on Plant-Pathogen Interactions Few studies have addressed temperature effects on rice-*M. oryzae* interactions specifically. However, recent work on *Arabidopsis thaliana* and *Pseudomonas syringae* reveals dynamic temperature-dependent interactions. Unlike animals with constant body temperatures, plants and microbes experience daily temperature fluctuations. Low temperatures promote bacterial effector secretion, while higher temperatures accelerate bacterial proliferation and PAMP synthesis. Plants have evolved corresponding defense strategies: at relatively low temperatures (10–23 °C), they preferentially activate ETI, whereas at higher temperatures (23–32 °C), they activate PTI [34]. How such temperature dynamics affect *M. oryzae* effector secretion, PAMP production, and the balance between rice PTI and ETI remains to be investigated.

4.2. Light Effects Light is a crucial environmental factor for both rice and *M. oryzae*, significantly influencing blast epidemics.

4.2.1. Light Effects on *M. oryzae* Light exerts a suppressive effect on rice blast development (light-dependent disease suppression), while a dark period after fungal contact with rice is critical for disease initiation [35]. Light inhibits hyphal growth, which proceeds fastest in darkness and slowest under continuous illumination [36]. Weak light promotes conidiation, while light intensity affects germ tube length—stronger light produces shorter germ tubes, leading to malformed appressoria and reduced infection rates [37]. Even weak light suppresses spore detachment from lesions [35]. Blue light inhibits *M. oryzae* growth and development, and both blue and red light suppress conidial release. The blue-light receptor gene *MGWC-1* mediates these effects; its mutation reduces light-dependent disease suppression [5]. Transcriptome analysis of dark-regulated genes revealed that melanin synthesis genes are differentially expressed during light-dark transitions. Since melanin accumulation in appressoria is essential for turgor generation and invasion [38], dark-induced expression of melanin-related genes promotes appressorium maturation and infection [37].

Paradoxically, conidiation requires light stimulation. The *Twilight* (*TWL*) gene mediates light-regulated conidiation: at night, TWL protein remains acetylated in the cytoplasm, but after dawn, light signals trigger its deacetylation and phosphorylation via Snf1 kinase, leading to nuclear translocation and activation of transcription factor TBF5 to induce conidiation [39].

4.2.2. Light Effects on Rice Disease Resistance Light also affects rice blast resistance. Weak light reduces photosynthetic efficiency and metabolic activity, weakening plants and increasing disease susceptibility [40]. Consequently, consecutive rainy days—characterized by high humidity, low light, and temperature fluctuations—create conditions highly favorable for blast epidemics. Some rice genes are regulated by both light and *M. oryzae* infection. For example, *OsATX* is upregulated by both light and blast infection [41], while *RML1* expression increases during infection but decreases under continuous light [42]. Although the functions of these genes in blast resistance remain unclear, they illustrate the complexity of light-mediated effects on rice immunity.

4.3. Humidity Effects Air humidity is another critical meteorological factor affecting blast epidemics. High humidity promotes conidiation and spore germination. Zhao [36] found that conidiation requires relative humidity above 90%, with higher humidity accelerating spore production. When humidity exceeds 90%, a water film forms on leaf surfaces, facilitating conidial germination, increasing infection rates, shortening incubation periods, and promoting lesion development and sporulation. Consequently, blast epidemics occur only under high humidity conditions [36].

Recent studies on *Arabidopsis* infected with *P. syringae* revealed that disease development requires both suppression of host defenses and high-humidity dependency. The bacterial effectors HopM1 and AvrE mediate this humidity dependence [43]. While high-humidity dependency is well-known in rice blast, the underlying molecular mechanisms remain obscure. Whether specific *M. oryzae* effectors mediate humidity-dependent pathogenesis requires further investigation.

Conclusion

Rice blast outbreaks are closely associated with temperature, humidity, and light conditions. Effective blast management requires not only planting resistant varieties but also implementing preventive measures based on meteorological forecasts, particularly during prolonged rainy, foggy, or dewy periods.

Although recent research has provided preliminary insights into how meteorological factors affect *M. oryzae* pathogenicity, rice resistance, and disease development, our understanding remains limited. In-depth studies on the molecular mechanisms through which meteorological factors influence blast epidemics, and application of these findings to molecular breeding and integrated disease

management, are crucial for ensuring stable rice production and national food security.

References

[1] Li B, Deng Y B, Yan X H, et al. Identification, genetic analysis and gene mapping of a rice blast resistance gene in japonica rice[J]. *Acta Agronomica Sinica*, 2014, 40(1): 54−62

[2] Sohu Business. Longping High-Tech' s Super Rice Liangyou 0293: From Favorite to Abandonment on Ten Thousand Mu[EB/OL]. [2015-04-10]. http://business.sohu.com/20150410/n411044347.shtml

[3] Yu J. Functional analysis of three Rho1 interacting proteins in *Magnaporth oryzae*[D]. Fuzhou: Fujian Agriculture and Forestry University, 2011

[4] Dean R A, Talbot N J, Ebbole D J, et al. The genome sequence of the rice blast fungus *Magnaporthe grisea*[J]. *Nature*, 2005, 434(7036): 980−986

[5] Chen J S, Zheng S Q, Zheng W, et al. Prediction for secreted proteins from *Magnaporthe grisea* genome[J]. *Scientia Agricultura Sinica*, 2006, 39(12): 2474−2482

[6] Zhang J Y, Xie G Q, Han Z L. Analyzing condition of rice blast happened in Yunnan Province in 2008[J]. *Yunnan Agricultural Science and Technology*, 2009(1): 50−51

[7] Li J H. Influence of agricultural meteorological factors on rice blast and preventive measures[J]. *Modern Agricultural Science and Technology*, 2014(21): 223−224

[8] Galhano R, Talbot N J. The biology of blast: Understanding how *Magnaporthe oryzae* invades rice plants[J]. *Fungal Biology Reviews*, 2011, 25(1): 61−67

[9] Veneault-Fourrey C, Barooah M, Egan M, et al. Autophagic fungal cell death is necessary for infection by the rice blast fungus[J]. *Science*, 2006, 312(5773): 580−583

[10] Talbot N J. On the trail of a cereal killer: Exploring the biology of *Magnaporthe grisea*[J]. *Annual Review of Microbiology*, 2003, 57: 177−202

[11] Ebbole D J. *Magnaporthe* as a model for understanding host-pathogen interactions[J]. *Annual Review of Phytopathology*, 2007, 45: 437−456

[12] Chumley F G, Valent B. Genetic analysis of melanin-deficient, non-pathogenic mutants of *Magnaporthe grisea*[J]. *Molecular Plant-Microbe Interactions*, 1990, 3: 135−143

[13] De Jong J C, McCormack B J, Smirnoff N, et al. Glycerol generates turgor in rice blast[J]. *Nature*, 1997, 389(6648): 244−245

[14] Wilson R A, Talbot N J. Under pressure: Investigating the biology of plant infection by *Magnaporthe oryzae*[J]. *Nature Reviews Microbiology*, 2009, 7(3): 185−195

[15] Kankanala P, Czymmek K, Valent B. Roles for rice membrane dynamics and plasmodesmata during biotrophic invasion by the blast fungus[J]. *The*

Plant Cell, 2007, 19(2): 706−724
[16] Li C H. The occurrence and prevention of rice blast[J]. *Shaanxi Agricultural Sciences*, 2011(5): 127−128
[17] Tan J L. The causes of rice blast and comprehensive prevention[J]. *Agricultural Technology Service*, 2014, 31(8): 32, 34
[18] Jinnong. Rice blast introduction and prevention[EB/OL]. [2007-7-17]. http://www.jinnong.cc/technology/sk10655.shtml
[19] Dangl J L, Dietrich R A, Richberg M H. Death don' t have no mercy: Cell death programs in plant-microbe interactions[J]. *Plant Cell*, 1996, 8(10): 1793-1807
[20] He F, Zhang H, Liu J L, et al. Recent advances in understanding the innate immune mechanisms and developing new disease resistance breeding strategies against the rice blast fungus *Magnaporthe oryzae* in rice[J]. *Hereditas*, 2014, 36(8): 756−765
[21] Tang X, Zheng Y, Tang L, et al. Effects of nitrogen and silicon nutrition on rice blast occurrence under intercropping with different type varieties[J]. *Chinese Journal of Rice Science*, 2006, 20(6): 663−666
[22] Liu J B, Gao C, Gao J, et al. Effects of silicon on the ultrastructure of *Oryzae sativa* leaves infected by *Magnaporthe grisea*[J]. *Journal of South China Agricultural University*, 2012, 33(1): 40−43
[23] Li J, Huang D Y. Present status of molecular breeding for blast resistance[J]. *Journal of Anhui Agricultural Sciences*, 2007, 35(35): 11413−11415
[24] Xu W W, Wang X, Huang Y X, et al. Advances in molecular markers of blast resistance genes and marker-assisted breeding in rice[J]. *Jiangsu Journal of Agricultural Sciences*, 2013, 29(4): 898−906
[25] Zhang J H, Sun H L, Liu Y D C, et al. A physiologic race on *Pyricularia oryzae* in Heilongjiang[J]. *Plant Protection*, 2009, 35(3): 137−140
[26] Zhang R Y. Rice blast disease characteristics and control methods[J]. *Friends of the Farmers for Getting Rich*, 2015(1): 61
[27] Li G T, Zhou X Y, Kong L G, et al. MoSfl1 is important for virulence and heat tolerance in *Magnaporthe oryzae*[J]. *PLoS One*, 2011, 6(5): e19951
[28] Li J, Jia B L, Liang X L, et al. An adenosine kinase in apoplastic location is involved in *Magnaporthe oryzae* cold acclimation[J]. *Journal of Basic Microbiology*, 2014, 54(4): 269−277
[29] Zhang X Y. Temperature on rice growth in different periods of analysis[J]. *Hunan Agricultural Machinery*, 2010, 37(11): 229−230
[30] Zhang S H, Wei Y, Pan H Y. Transgenic rice plants expressing a novel antifreeze glycopeptide possess resistance to cold and disease[J]. *Zeitschrift für Naturforschung C*, 2007, 62(7/8): 543−551
[31] Yokotani N, Sato Y, Tanabe S, et al. WRKY76 is a rice transcriptional repressor playing opposite roles in blast disease resistance and cold stress tolerance[J]. *Journal of Experimental Botany*, 2013, 64(16): 5085−5097
[32] Ma Y, Dai X Y, Xu Y Y, et al. COLD1 confers chilling tolerance in rice[J]. *Cell*, 2015, 160(6): 1209−1221
[33] Li X M, Chao D Y, Wu Y, et al. Natural alleles of a proteasome $ $2 subunit gene contribute to thermotolerance and adaptation of African rice[J].

Nature Genetics, 2015, 47(7): 827−833
[34] Cheng C, Gao X Q, Feng B M, et al. Plant immune response to pathogens differs with changing temperatures[J]. *Nature Communications*, 2013, 4: 2530
[35] Lee K, Singh P, Chung W C, et al. Light regulation of asexual development in the rice blast fungus, *Magnaporthe oryzae*[J]. *Fungal Genetics and Biology*, 2006, 43(10): 694–706
[36] Zhao Z J. Prevalence category of rice blast in main producing areas of Heilongjiang reclamation area and study forecast model[D]. Daqing: Heilongjiang Bayi Agricultural University, 2013
[37] Lin F C, Li D B. Factors affecting on conidium germination and its appressorium formation of *Magnaporthe grisea*[J]. *Chinese Journal of Rice Science*, 2001, 15(4): 291−297
[38] Howard R J, Valent B. Breaking and entering: Host penetration by the fungal rice blast pathogen *Magnaporthe grisea*[J]. *Annual Review of Microbiology*, 1996, 50(1): 491–512
[39] Deng Y Z, Qu Z W, Naqvi N I. Twilight, a novel circadian-regulated gene, integrates phototropism with nutrient and redox homeostasis during fungal development[J]. *PLoS Pathogens*, 2015, 11(6): e1004972
[40] Du Y X, Ji X, Zhang J, et al. Research progress on the impacts of low light intensity on rice growth and development[J]. *Chinese Journal of Eco-Agriculture*, 2013, 21(11): 1307–1317
[41] Agrawal G K, Rakwal R, Jwa N S, et al. Characterization of a novel rice gene *OsATX* and modulation of its expression by components of the stress signalling pathways[J]. *Physiologia Plantarum*, 2002, 116(1): 87–95
[42] Wang R, Hong G F, Han B. Transcript abundance of *rml1*, encoding a putative GT1-like factor in rice, is up-regulated by *Magnaporthe grisea* and down-regulated by light[J]. *Gene*, 2004, 423: 105–115
[43] He S Y. New phenomena beyond immune suppression revealed in the disease-susceptibility-reconstitution experiments[C]//The 4th International Conference on Biotic Plant Interactions. Nanjing, 2015

Note: Figure translations are in progress. See original paper for figures.