

Supplemental Table S2. The functions of components in nutrient signaling pathways in pathogenic and beneficial fungi.

Gene	Fungi	Description	References
Sucrose non-fermenting 1 (SNF1) protein kinase signaling pathway			
<i>Hxk1</i>	<i>F. verticillioides</i>	<i>HXK1</i> regulated carbon catabolism, sporulation, and pathogenesis.	[1]
	<i>F. graminearum</i>	The deletion led to inhibited vegetative growth and conidiation, whereas the overexpression led to increased deoxynivalenol production.	[2]
	<i>Botrytis cinerea</i>	The deletion mutants of <i>HXK1</i> were defective in growth and virulence.	[3]
<i>Snf1</i>	<i>M. oryzae</i>	The knock-out mutants displayed a defect in growth, conidial germination, appressorium formation, pathogenicity and sporulation.	[4]
	<i>F. oxysporum</i>	The disruption led to defects in the suppression of cell wall-degrading enzymes, utilization of certain carbon sources, and virulence.	[5]
	<i>V. dahliae</i>	The disruption of <i>VdSNF1</i> led to defects in growth, virulence, and colonization.	[6]
	<i>Pestalotiopsis microspora</i>	The deletion mutants contributed to defects in vegetative growth, pigmentation, conidia, cell wall integrity.	[7]
	<i>Alternaria alternata</i>	The deletion mutants were defective in aerial mycelium growth, conidia, and germination with smaller lesions on detached citrus leaves, and stronger tolerance to some stressors.	[8]
<i>Gal83</i>	<i>M. oryzae</i>	The mutants of <i>ΔMosnf4</i> , <i>ΔMosak1</i> , <i>ΔMosnf1</i> , <i>ΔMosip2</i> , and <i>ΔMosak1ΔMots3</i> , displayed distinct defects in lipid mobilization, sporulation, conidial germination, and appressorium formation.	[9]
<i>Sak1</i>			
<i>Snf4</i>			
<i>Reg1</i>	<i>B. cinerea</i>	The gene knock-out mutant of <i>bcreg1</i> kept the ability to penetration but were defective in pathogenicity and conidia formation.	[10]
<i>Ssn6</i>	<i>U. maydis</i>	The homologue of <i>Ssn6</i> , <i>Sql1</i> alleles in a certain form induced the fungal filamentous growth which can be reversed by exogenous cAMP.	[11]
<i>Tup1</i>	<i>U. maydis</i>	The deletion of the <i>Umtup1</i> led to defects in the mating, filamentation and virulence.	[12]
	<i>M. oryzae</i>	The disruption of <i>MoTUPI</i> suppressed vegetative growth and conidiogenesis, and led to the formation of appressorium-like structures that cannot penetrate into the host cells.	[13]
cAMP-dependent protein kinase A (cAMP-PKA) signaling pathway			
<i>Cdc25</i>	<i>C. higginsianum</i>	The deletion of <i>ChCDC25</i> led to defects in vegetative growth, conidiation, conidium germination, appressorium formation, stress chemicals tolerance, and virulence on <i>Arabidopsis</i> leaves.	[14]
	<i>U. maydis</i>	The deletion mutants of CDC25-like gene <i>sql2</i> were defective in pathogenic development, whereas the overexpression mutants of <i>sql2</i> were shown an enhanced filamentous growth that cannot be repressed by exogenous cAMP.	[15]
<i>Ras1</i>	<i>M. oryzae</i>	The overactive <i>MoRas2</i> resulted in improper activation of these two cascades, leading to abnormal appressorium formation without surface attachment.	[16]
	<i>C. orbiculare</i>	The disrupted mutants of <i>coras2</i> were defective in conidial germination and pathogenesis to the cucumber cotyledons.	[17]
<i>Ira1</i>		CoIra1 and CoRas2 colocalized in a vesicle-like structure in the appressorium, and the mutants of <i>coira1</i> had a defective in infection morphogenesis on cucumber plants.	
<i>Gpa3</i>	<i>M. grisea</i>	The disruption of Gα subunit genes <i>magB</i> restrained vegetative growth, conidiation, appressorium formation, infection, mating, and pathogenicity.	[18]

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<i>Gpa3</i>	<i>U. maydis</i>	The disruption of <i>gpa3</i> led to the loss of ability to respond to pheromone and defect in mating, whereas the active allele of <i>gpa3</i> resulted in mating without pheromone.	[19]
		The exogenous cAMP was comparable to pheromone to stimulate pheromone gene expression, and rescue the sterility and mutant morphology of <i>gpa3</i> deletion strains.	[20]
	<i>S. scitamineum</i>	Gpa3, adenyl cyclase UAC1 and PKA catalytic subunit ADR1 are essential for proper mating, filamentation, and virulence. These genes can regulate the expression of genes encoding ROS (reactive oxygen species) catabolic enzymes in <i>S. scitamineum</i> .	[21]
	<i>Umbilicaria muhlenbergii</i>	The deletion of Ga subunits <i>UmGPA3</i> displayed little effects on pseudoththal growth, the <i>ΔUmGpa3</i> mutant had defects in yeast-to-pseudoththal transition. The dominant active mutations of <i>UmGPA3</i> led to improper pseudoththal growth and disruption of the symbiotic interaction with <i>Trebouxia jamesii</i> .	[22]
	<i>M. oryzae</i>	Rgs7 couples with Ga subunit MagA to sense external signals and activate cAMP signaling required for appressorium function.	[23]
<i>Cyr1</i>	<i>S. sclerotiorum</i>	deletion of adenylate cyclase <i>sac1</i> gene led to low cAMP levels, more microconidia, attenuated virulence and slow growth rate.	[24]
	<i>C. higginsianum</i>	The mutants lacking of adenylate cyclase ChAC and PKA catalytic subunits ChPKA1 displayed distinct decrease in hyphal growth, conidiation, appressorial formation, pathogenicity, and stress tolerance to cell wall inhibitors.	[25]
<i>Bcy1</i>	<i>M. circinelloides</i>	The four PKA regulatory subunits encoding gene <i>pkaR1</i> , <i>pkaR2</i> , <i>pkaR3</i> , and <i>pkaR4</i> with different expression levels were shown with different roles in growth, germination, cell volume, sporulation, and differentiation in each isoform mutants.	[26,27]
	<i>C. lagenarium</i>	The breakdown of glycogen and lipid was very rapid in PKA regulatory subunit <i>mac1 sum1-99</i> mutant, whereas the dissolution was delayed significantly in catalytic subunit <i>cpkA</i> mutant.	[28]
		The mutants of RPK1, showed high PKA activity that suppressed vegetative growth, conidiation, and appressorium function.	[29]
		The knockout mutants of the adenylate cyclase CAC1 and PKA catalytic subunit CPK1 germinated poorly, and form appressoria which are nonfunctional that leads to the infection defect on cucumber.	[30]
<i>Tpk1</i>	<i>M. grisea</i>	PKA catalytic subunit <i>cpkA</i> was required for appressorial penetration and pathogenesis.	[31,32]
	<i>N. crassa</i>	The catalytic subunit <i>pkac-1</i> mutants showed defects in aerial hyphae formation and conidiation.	[33]
	<i>V. dahliae</i>	The mutants lacking for PKA catalytic subunit genes displayed less ethylene biosynthesis and defects in conidia production.	[34]
	<i>F. oxysporum</i>	The mutants lacking for PKA catalytic subunit gene <i>FoCPKA</i> lost virulence and showed a decrease in vegetative growth, spore production, root penetration and vascular colonization on <i>A. thaliana</i> roots, and the distance between septas of <i>fockp1</i> mutants was longer than that of wild-type strain.	[35]
	<i>M. oryzae</i>	The PKA catalytic subunits genes <i>Cpk2</i> and <i>CpkA</i> shared largely redundant functions in regulation of hyphal growth, conidiation and appressorium formation, but also played different roles during pathogenesis and morphogenesis.	[36]

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<i>Pde2</i>	<i>M. oryzae</i>	The loss of <i>PDEH</i> enhanced conidiation and increased intracellular cAMP levels during vegetative growth, leading to precocious appressorial development, loss of surface dependency and decrease of colonization.	[37]	
		The disruption of <i>PDEH</i> can partially rescue the abnormal phenotype of G alpha subunit encoding gene <i>ΔmagB</i> mutant and PKA catalytic subunit encoding gene <i>Δpka1</i> mutant.	[38].	
		<i>MoPdeH</i> can function upstream of the MAP kinase pathway to regulate the cell wall integrity (CWI), and also can mediate the crosstalk between cAMP pathway and osmotic sensing high osmolarity glycerol (HOG) pathway.	[39]	
<i>Yak1</i>	<i>M. oryzae</i>	The disruption of <i>MoYAK1</i> led to defects in germination, aerial hyphal formation, conidiation, glycogen and lipid metabolism, turgor generation, penetration and cell wall integrity.	[40]	
	<i>B. cinerea</i>	The deletion of <i>Yak1</i> resulted in lower pathogenicity, conidiation, sclerotium formation, and more sensitive to H ₂ O ₂ .	[41]	
Target of rapamycin (TOR) kinase signaling pathway				
<i>Gtr1</i>	<i>Candida albicans</i>	TOR can response to phosphate, and the overexpression of <i>Gtr1</i> suppresses TORC1 signaling defects.	[42]	
<i>Tip41</i>	<i>F. graminearum</i>	The FgFkbp12, FgPp2A, FgSit4, FgPpg1, FgTap42, FgTip41, FgAreA in TOR pathway formed a regulatory framework to regulating vegetative differentiation and virulence. FgTip41 regulated hyphal growth and virulence.	[43].	
<i>Tor2</i>		The deletion of <i>FgTor</i> may be lethal, as the mutants cannot be generated.	[43,44]	
		MoTor regulated the expression of <i>MoSNT2</i> , which stimulated the expression of autophagy genes <i>MoATG6/15/16/22</i> to regulate the conidiation, stress tolerance, cell wall integrity, and pathogenicity.	[45]	
<i>M. oryzae</i>	Asd4 regulated intracellular glutamine concentration to modulate the TOR inhibition of appressorium formation, and TOR signaling was activated in the <i>Δasd4</i> deletion mutants that fail to form appressoria.	[46]		
		Glucose can mediate TOR via the carbon-responsive Ampk β subunit-like protein, ABL1. The <i>ΔMoabl1</i> mutants produced few appressoria, and fail in autophagy, and this can be restored by inactivated TOR.	[47]	
<i>Sch9</i>	<i>F. graminearum</i>	FgSch9 interacted with FgMaf1, FgTor and FgHog1 to regulate fungal vegetative development, multiple stress responses and secondary metabolism.	[48].	
<i>Tap42</i>	<i>F. graminearum</i>	Antofine treatment led to the disruption of the FgRRD2-FgTap42 which was required for pathogenicity.	[49]	
<i>Rrd2</i>				
<i>Pph22/ Ppg1/ Sit4</i>	<i>F. graminearum</i>	The deletion mutants of <i>FgHLTF1</i> , whose expression was down-regulated by <i>FgPPG1</i> deletion or rapamycin treatment, showed defects in the vegetative growth, sexual reproduction, and virulence.	[50]	
		FgPpg1/Sit4 were need for inducing lipid droplets biogenesis which regulated the fungal vegetative growth, sexual development, and virulence via Nem1/Spo7-Pah1 cascade.	[51]	
	<i>M. oryzae</i>	MoPpe1, orthologue of Sit4/Ppe1 in yeast, interacted with MoSap1 under controlled by MoTap42 and MoTip41 to regulate the fungal autophagy, vegetative growth, appressorium function, infection, and rapamycin sensitivity.	[52,53]	

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Phosphate (PHO) signaling pathway			
Pho84	<i>G. margarita</i>	The knockdown of phosphate transceptor gene <i>GigmPT</i> (<i>Pho84</i> in yeast) by the host-induced gene silencing (HIGS) strategy led to a defect in arbuscule development.	[54]
Arg82	<i>Cryptococcus neoformans</i>	The mutants of <i>Arg1</i> , the orthologue of <i>Arg82</i> in yeast, displayed defects in the capsule production, cell wall organization, thermotolerance, cell separation, and mating filaments formation.	[55,56]
Pho81	<i>N. crassa</i>	In the mutant of <i>nuc-2</i> (<i>Pho81</i> in yeast), some genes involved in the initiation of mRNA translation were up-regulated.	[57]
	<i>V. dahliae</i>	The phosphate-starved <i>VdNUC-2</i> knockout mutants showed defects in radial growth, conidia production, and become more sensitive to hydrogen peroxide stress that cannot be restored by phosphate supply.	[58]
	<i>C. neoformans</i>	The inositol pyrophosphate IP ₇ promoted virulence, and destruction of IP ₇ -Pho81 or repression of Pho81 causes a defect in the virulence.	[59]
Pho80	<i>A. fumigatus</i>	The <i>PHO80</i> mutant displayed a polar growth defect and the increases of vacuolar accumulation, acid phosphatase activity and phosphate concentration.	[60]
	<i>S. indica</i>	The expression level of <i>SiPho80</i> was upregulated to maintain phosphate homeostasis under high phosphate levels, and <i>SiPho80</i> can restore the fungal tolerance to salt and heavy metal (e.g. copper, zinc, cobalt) in yeast <i>Δpho80</i> mutant.	[61]
Pho85	<i>U. maydis</i>	The mutants of <i>Cdk5/Pho85</i> displayed defects in the morphogenesis and polar growth required for virulence.	[62,63]
Pho4	<i>N. crassa</i>	The subcellular location of NUC-1 (<i>Pho4</i> in yeast) is important for regulating gene expression	[64]
Ino80	<i>Trichoderma virens</i>	IPA-1, a protein belongs to the SNF2 family, played an important role in plant disease resistance, and the deletion of <i>IPA-1</i> led to the diminished antibiotic activity of plant against <i>Rhizoctonia solani</i> .	[65]
Common targets and relevant components			
14-3-3	<i>F. mosseae</i>	<i>Fm201, Ri14-3-3</i> and <i>RiBMH2</i> restored the lethal yeast <i>bmh1 bmh2</i> double mutant. The gene silencing of both <i>Ri14-3-3</i> and <i>RiBMH2</i> impaired the arbuscule formation.	[66]
	<i>R. irregularis</i>		
Rim15	<i>Cochliobolus heterostrophus</i>	Deletion of <i>RIM15</i> affected the fungal pigmentation and vegetative growth.	[67]
Atg8	<i>M. grisea</i>	The <i>ΔMgATG8</i> mutants were defective in the autophagy, and failed in conidial cell death which is necessary for infection and pathogenicity.	[68]
	<i>F. graminearum</i>	The <i>ΔFgatg8</i> deletion mutants were defective in autophagy required for sporulation and sexual reproduction, and displayed the aerial hyphal collapse with more lipid droplets.	[69]
		The degradation of Gcn5 induced by rapamycin can suppress the cellular relocalization and acetylation of Atg8, leading to autophagy.	[70]
Rho1	<i>U. maydis</i>	The Rho1 controlled cell separation and polarity, and it interacted with the high-affinity ammonium transporter Ump2 to control filamentation which was required for fungal development and pathogenesis.	[71,72]

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