PLANT GENES AS SOURCES TO IMPROVE PATHOGEN RESISTANCE OF CULTIVATED PLANTS

Zsófia TÓTH – Erzsébet KISS

Szent István University, Institute of Genetics and Biotechnology, 1. Páter Károly Street, 2100, Gödöllő, Hungary
E-mail: Toth.Zsofia@mkk.szie.hu, Kiss.Erzsebet@mkk.szie.hu

Abstract: Diseases cause large problem in agriculture that the growers usually try to overcome by chemical control. Unfortunately, the amount of applied chemicals increases in every year all over the world. Since the chemicals have numerous negative side-effects, the alternative techniques are welcome in crop production. Plenty of studies demonstrated that resistance genes regulate plant defense response to pathogen attack and infection. Beside the induced expression of resistance genes the disease responsiveness may also depend on plant susceptibility genes, presence in host of which is required for success of invasion. Breeding plant varieties integrating resistance genes or inhibiting the function of susceptibility genes significantly increase the disease resistance. In this manner the amount of pesticides applied for pathogen control can be reduced and crop production may turn into a much more environmentally friendly process.

Keywords: pathogen response, signaling, resistance genes, susceptibility genes, plant breeding

Introduction

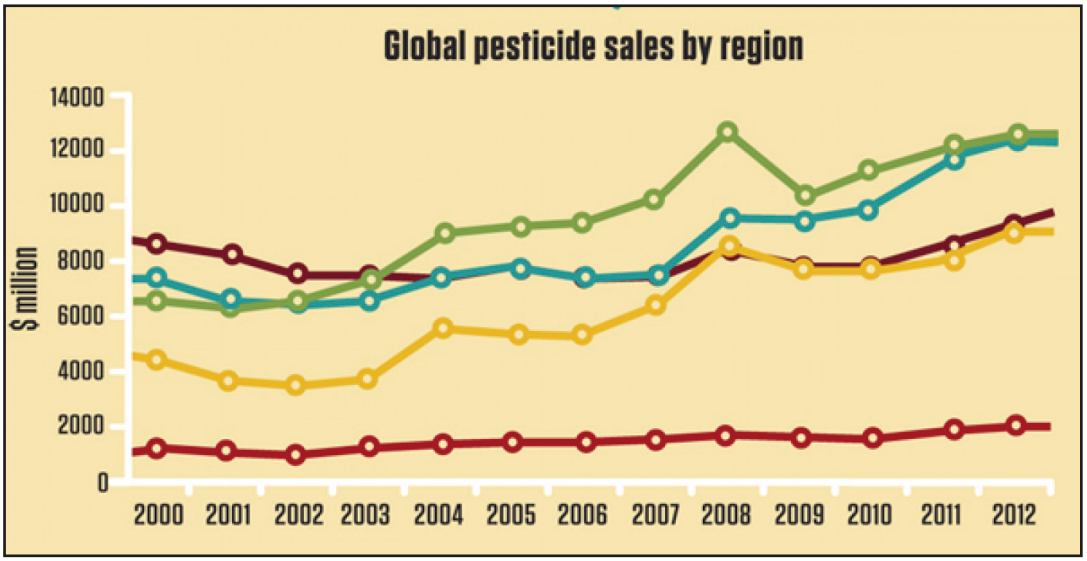
Although numerous studies quest the genetic and molecular background of diseases and already a lot of results have been born, the pathogenic organisms still cause serious problems in agriculture. The growers apply chemicals on their fields to control the invasion of pathogens. The fairly effective - but because of the necessity of repeated spraying - very expensive chemicals require skilled labour with stringent regulatory restrictions. Although this process significantly increases the prime cost of crop production, the amount of chemicals applied to control pest increases every year (Figure 1). The world used approximately 5.2 billion pounds of pesticides per year in the last decade, but it still raised in the subsequent years (FAOSTAT). Mostly, herbicides are used all over the world, but the insecticide and fungicide consumption is also high in some regions due the impact of climate conditions, such as high humidity. Beside the primary high cost, the application of chemicals has high risk of potentially harmful impact on the environment, ecosystems and consumers. The toxicity of some chemicals increases the number of morbidity cases in all over the world. The human health hazards range from acute dangers to serious chronic diseases, such as cancer, endocrine disruption. Therefore studies aspiring development of new alternative techniques to control diseases are exceptionally conducive. Many chemical-free opportunities are to limit disease spread, such as agrotechnical methods, integrated pest management or application of resistant/tolerant varieties for cultivation. These techniques are excellent scopes to grow crops environmentally friendly, and together may be sufficient to fight against diseases without yield loss. Breeding new varieties with integration of resistance genes is an admitted technique using nowadays the marker assisted selection (MAS). By MAS the breeding period may be limited to two years, which is a large progress compared to the traditional techniques. Studies discovering new genes, which relate to defense or promote immune response support the new plant breeding purposes.

Regulation of plant defense during host-pathogen interaction

Plants are subjects of numerous challenges during their lifecycle. Since they are immobile organisms, cannot escape from attackers, the plants were forced to develop complex defense system to survive at a higher rate. During the long host-pathogen co-evolution, plants configured specific prevention

DOI: 10.18380/SZIE.COLUM.2015.1.37.

Figure 1. Global pesticides sales by region of the world (Source: The Washington Post, August 18. 2013).



mechanisms, such as strengthened cell wall, or production of antimicrobial compounds. Once the pathogen passes these tricks, and enters the plant cell, the plant recognizes the pathogen-secreted effector proteins by its 'receptors'. These 'receptors' are encoded by resistance *(R)* genes and while the effectors proteins by the pathogenic *avr* genes. The mutual recognition is called the 'gene-for-gene' interaction, first described in 1942 (Flor, 1942, 1971). If these two proteins interact, the reaction induces a complex signaling cascade which may end with systemic acquired resistance (SAR), or with programmed cell death, the hypersensitive reaction (HR) (Király et al., 1972; Fodor et al., 1997). Both responds are mediated via salicylic acid signaling and significantly limit the pathogen growth; HR totally inhibits infection. This type of immune response is called the effector-triggered immunity (ETI), which based on the specific gene-for-gene interaction. If this specificity does not exist, the plant still may recognize its aggressor by the pathogen- or microbe-associated molecular pattern (PAMP, MAMP). The specific molecular pattern may be the bacterial flagellins, lipopolysaccharides, nucleic acids (e.g. viral and bacterial DNA/RNA), peptidoglycans, fungal chitins or glucans. These molecules can be percieved by pattern recognition receptors (PRR) of the plant and the detection induces the PAMP-triggered immunity (PTI), a form of basal defense. During PTI process mainly those genes are activated, which are involved in the biosynthesis of antimicrobial compounds, promote pathogenic cell wall degradation, regulate plant cell wall fortification or stomatal closure. Some of the signaling components of PTI overlap with the ones of ETI, but somehow the PTI-triggered defense reaction is not as effective to control pathogen spread, as the HR or SAR is. Additionally to PTI the plant cell detects microbial compounds released by the injured microorganisms. For example cucumber hypocotyl recognized the α-1,4-linked oligomers of galacturonic acid and oligo-β-glucans released from damaged *Phytophthora megasperma* f. sp. *glycinea* cell wall triggering hydrogen-peroxide production (Svalheim and Robertsen, 1993). This phenomenon is called Damage Associated Molecular Pattern (DAMP), similar mark of pathogen presence as PAMP/MAMP and provokes DAMP-triggered immunity (DTI).

The susceptibility of the plant to diseases is not always an obligatory result of host immunity failure. Earlier studies demonstrated that susceptibility of many plant species rather depends on host compatibility factors, than early response of PTI or *R* genes. Numerous genes are identified to play a role in advancing

DOI: 10.18380/SZIE.COLUM.2015.1.37.

pathogen proliferation, especially of biotrophic fungi, which require cooperation of host compatibility factors for their invasion. The genes impairing prepenetration requirements (enable the pathogen to enter the plant cell) or fulfilling postpenetration necessaries are termed as susceptibility *(S)* genes.

Difference between necrotrophic and biotrophic pathogens

Fungi and oomycetes produce spores, which germinate on plant surface and develop fungal hypha. The fungus may enter the plant cell through natural openings, or may punch the cell wall using their appressoria and form the haustoria for feeding and effector transmission (Yi and Valent, 2013). Early response to fungi may associate with papilla formation and cell death along with accumulation of H_2O_2 (Huckelhoven et al., 1999). Contrarily the bacteria are unable to breach cell wall, therefore these organisms resort to the natural openings, such as wounds or stomata. Bacteria often form the type III or type IV secretion system to translocate their elicitor molecules, which may induce early response of plant defense (Ott et al., 2006; Klement et al., 2003; Bozsó et al., 2005) Based on lifestyle of the pathogen we discriminate necrotrophic and biotrophic organisms. The necrotrophic pathogens degrade their host and utilize nutrients from the rotten tissues. In contrast, the biotrophic pathogens survive only on live plants, therefore they are able to manipulate plant metabolism to retard senescence of the infected tissue ('green island' effect) (Bushnell and Allen, 1962). The hemibiotroph organisms are considered as necrotrophs, but have an initial biotrophic lifestyle of the early stage of infection. Especially the hemibiotroph *Colletotrichum graminicola* fungus induces green island effect, but it also globally accelerates senescence in aging maize leaves in order to utilize them as carbon sink (Behr et al., 2010). Since the lifestyle differs to a great extent, the immune response is also distinct in many aspects between bio-, hemibio- and necrotrophs. For example the gene-for-gene interaction is assumed to occur only in biotrophs; in necrotrophs the receptor-like protein kinases (RLKs) mediated PTI induces the response (Llorente et al., 2005). The plant defense signaling components also differ between the infection styles; the salicylic acid (SA)-amediated signal transduction is active usually in biotroph attack, but the ethylene (ET)/jasmonic acid (JA) signals regulate on the effect of necrotroph infection mostly. The hydrogen-peroxide and other reactive oxygen intermediates (ROI) may also provide signaling function during the infection; these components are found to promote the HR, often called oxidative burst. The H_2O_2 is the most stable ROI that may induce defense gene-expression, activate phytoalexin production during biotic stress. It was also reported that this molecule inhibit biotroph growth, but mostly benefit necrotroph invasion (Thordal-Christensen et al., 1997). However, the NADPH oxidase gene, *RBOHD* (for respiratory burst oxidase homolog D) was found to regulate HR in *Arabidopsis-Alternaria* pathosystem specifically in the infected single cells and not in neighboring ones (Pogany et al., 2009). Additionally externally applied H2O2 inhibited replication of Tobacco mosaic virus (TMV), therefore early accumulation of ROI promotes resistance to TMV (Bacsó et al., 2011).

Mutation analysis is a prominent approach for identifying the components of the signaling pathways. Many recessive mutations result in a constitutive defense response, such as the *acd2* (accelerated cell death), *cim3* (constitutive immunity), *cpr1-1* (constitutive expressor of pathogenesis-related *(PR)* genes), *edr1* (enhanced disease resistance) and *lsd* (lesion stimulating disease) inactivation does. However other mutations compromise the defense response; the mutants providing SA-signaling deficiency are the *npr* (non-expressor of *PR* genes)/*nim* (non-inducible immunity), *sai* (SA insensitive); *pad4* (phytoalexin deficient),

eds1 (enhanced disease susceptibility); the mutant with ET-signaling deficiency is the *ein* (ethylene insensitive); with JA-signaling deficiency is the *coi1* (coronatine-insensitive). All of these mutant plants displayed increased susceptibility to pathogen infections, meaning that these signaling pathways are also components of defense reaction (Yang et al., 1997; Brodersen et al., 2002; Wang et al., 2002; Glazebrook, 2005; Katsir et al., 2008). Functional screens on these mutants provide better insight into the role of defense genes in signaling pathways.

Genes that regulate plant responses

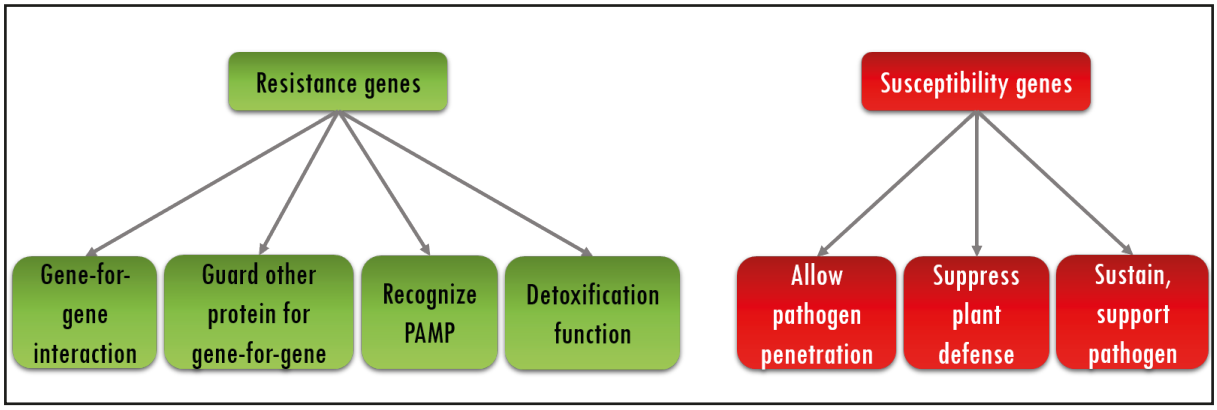
To trigger the immune response the pathogen needs to be recognized by R proteins. The *R* genes are usually dominant alleles and may be categorized into four groups based on their function: (i) *R* genes, which are direct targets in the gene-for-gene interaction; (ii) the genes that support the target in this interaction; (iii) the genes that recognize the PAMPs; (iv) the genes that have detoxification function (Figure 2). The *R* genes regulate robust resistance against a specific pathogen or a pathogen race. However this robustness is maintained as long as a new virulent strain does not try to infect the plant. Beside the *R* gene mediated resistance the Quantitative Disease Resistance (QDR) loci are encoded by multiple genes (quantitative trait loci/QTL), therefore these are more effective traits to evade pathogens. The selection pressure on pathogen races is lower, therefore the QDR provides more durable resistance to diseases than *R* genes (Parlevliet, 2002). Discovery of *R* genes or QDR-s and then integrating them into susceptible variety is an efficient procedure supported nowadays by MAS.

In contrast to the dominant resistance genes, the susceptibility genes increase resistance if they lose their function; therefore these genes are beneficial to enhance pathogen tolerance only in recessive form. The susceptibility genes may be categorized into three groups based on the timing of pathogen support (i) the genes, which allow accommodation of the attacker; (ii) the genes, which suppress defense response; (iii) and the genes, which aid the pathogen to be supplied by nutrients/ water (Figure 2).

Plant resistance genes

As the first key element of PTI, the pattern recognition receptors (PRR) detect the PAMP, DAMP or MAMP of pathogens. The first discovered PRR was the *Xa21* gene in *Oryza sativa*, which encodes both extracellular LRR (leucine-rich repeat) and transmembrane protein kinase (Song et al., 1995). This protein complex is responsible for recognition of Ax21 (activator of Xa21 triggered immunity) peptide of bacterium *Xanthomonas oryzae* pv. *oryzae* (Lee et al., 2009). After detection, *Xa21* induces intracellular defense response. In plant breeding integration of a single locus of this gene resulted increased resistance to several bacterial blight isolates (Wang et

Figure 2. Function based categories of resistance and susceptibility genes.



DOI: 10.18380/SZIE.COLUM.2015.1.37.

al., 1996; Zhai et al., 2002). As an example for fungal PAMP, the chitin of the pathogen cell wall can be recognized by the CEBiP transmembrane protein and induces immunity in rice (Zipfel and Robatzek, 2010). Similarly to this, the Elongation Factor Tu (EF-Tu) bacterial peptide can be detected by the plant EF-Tu receptor (EFR). *EFR* belongs to the same subfamily as the leucine-rich repeat-receptor-like protein kinase (LRR-RLK), the FLS2 (flagellin-sensitive 2), which was also found to regulate defense responses (Zipfel et al., 2006). Insertion of the *EFR* gene into the wheat genome enhanced resistance to bacterial diseases (Schoonbeek et al., 2015). FLS2 protein was found to be ET-dependent, and to be integrated into the plasma membrane. FLS2 bound to the bacterial flagellin 22-amino-acid epitope (Flg22) at the early stage of bacterial infection. This interaction with Flg22 regulated FLS2 to associate with BAK1, another LRR-receptor-like kinase. The FLS2-BAK1 complex then activated *BIK1 (Botrytis-induced kinase 1)* gene, which triggered the mitogen activated protein kinase (MAPK) cascade to govern the defense response (Veronese et al., 2006; Nicaise et al., 2009; Lu et al., 2010). *BIK1* was found to positively manage defense against necrotrophs, but repressed the response to the virulent biotrophic bacteria *Pseudomonas syringa*e pv *tomato* (Veronese et al., 2006). Additionally, the FLS2 physically associated also to the resistance proteins RPM1, RPS2 and RPS5, which all regulate in ETI (Qi et al., 2011). This fact proves that PTI and ETI signaling components overlap (Thomma et. al., 2011).

The *R* genes, which encode proteins for 'gene-for-gene' interaction usually belong the *NBS-LRR* superfamily. These genes code for a central nucleotide binding site (NBS) and leucine-rich repeat (LRR) domain at the C-terminal. These proteins are categorized into two groups based on the N-terminal domain: (i) the TIR (Toll/Interleukin-1 receptor-like domain) group genes and (ii) the non-TIR or Coiled-coil (CC) NBS-LRRs (Gururania et al., 2012). NBS-LRR proteins play a role in regulation of the effector triggered immunity. They may bind directly to the pathogen effector or guard other protein for completion the interaction in order to induce defense response (DeYoung and Innes, 2006). For example the tomato *Bs4* TIR-NBS-LRR complex detected directly the AvrBs4 effector protein of *Xanthomonas campestris* pv. *vesicatoria*. *Bs4* represents high homology to the tobacco *N* and potato *Y-1* resistance genes (Schornack et al., 2004). The barley *Mla1* and *Mla6* genes were found to be active in powdery mildew (PM) infection in barley. These genes code CC-NBS-LRR proteins; especially the *Mla6* activated *RAR1* and *SGT1* resistance genes for induction of immunity against PM (Shen et al., 2003). Furthermore the *RAR1* was required for activation of the tobacco *N* gene against Tobacco Mosaic Virus too (Liu et al., 2002). The Pi-ta CC-NBS-LRR protein directly interacted with the rice blast fungus AVR-Pita effector. Only a single amino acid change in the protein altered resistance trait to susceptibility (Bryan et al., 2000). The *RPW8* gene, found in *Arabidopsis*, code an N-terminal transmembrane protein and a coiled coil domain. The *RPW8.1* and *RPW8.2* regulated defense response through the SA-mediated signaling, and associated with *PAD4*, *EDS5*, *NPR1* and *SGT1b* defense genes for activation PM resistance and HR. These *RPW8* genes are independent of *COI1*- and *EIN2*-mediated signaling pathways. However the *edr1* mutation, which repress the SA-signaling, lowered the activity of *RPW8.1* and *RPW8.2* and programed cell death too (Xiao et al., 2005). The tomato *Cf* genes has been used for decades to improve resistance in crop plants. These genes code extra-cytoplasmic LRRs and C-terminal membrane anchor (Jones et al., 1994). The *Cf-4* gene, following the interaction with Avr4 effector of *Cladosporium fulvum*, triggered ETI and HR in tomato. Interestingly, *Cf-4* also recognized homologous cognate effector

DOI: 10.18380/SZIE.COLUM.2015.1.37.

proteins secreted by multiple pathogen species (Stergiopoulos et al., 2010). Most of the *R* genes encoding NBS-LRR proteins are putatively targeted in cytoplasma. However the *Arabidopsis thaliana RRS1-R* gene coding a TIR-NBS-LRR complex harbors a nuclear localization signal and a WRKY-type DNA binding domain at the C-terminal extension. Several hypothesis are, how this protein activates defense against the wilt *(Ralstonia solanacearum)*: (i) the PopP2 effector contains also a nuclear localization signal, in this way the interaction with RRS1-R is achieved inside the nucleus; or (ii) the RRS1-R is located in the cytoplasm in inactive form, and following the interaction the complex of RRS1-R-PopP2 is transmitted together into the nucleus (Lahaye, 2002; Deslandes et al., 2003). As an example for guarding other protein, the tomato Pto intracellular Ser/Thr protein kinase activated ETI along with guidance of Prf TIR-NBS-LRR protein. Pto interacted directly with AvrPto or AvrPtoB elicitors secreted by the bacteria *P. syringae* pv. *tomato* (Oh and Martin, 2011). Discovery of *R* genes increases the evidence that resistance proteins rather guard a few host protein for effector recognition, than direct contact with pathogen secreted proteins. Based on the guard model a single R protein may be able to interact with multiple effectors and other R genes are transcribed in order to guide their interaction and trigger immunity (Zhang et al., 2013).

Host selective toxins (HSTs) are effective weapons of necrotrophs to kill the plants. These phytotoxins generate necrotic lesions in plant tissues and forward colonization of the pathogen. Phytoalexins are then accumulated in order to evade the toxic effect of HSTs. For example the camalexin phytoalexin accumulated in response to *Alternaria brassiscicola* infection in *Arabidopsis*. (Saga et al., 2012). Furthermore camalexin also increased resistance against *Botrytis cinerea* and *Leptosphaeria maculans* (Bohman et al., 2004). Another gene family with antimicrobial activity are the defensins, the small cysteine-rich molecules. These compounds are able to inhibit the virulence of microorganisms directly by alteration of the fungal membrane permeability, or may enhance plant innate immunity by triggering programmed cell death (Aerts et al., 2008; Hegedüs and Marx, 2013). These genes are widespread among the plants, insects and mammals, therefore they probably have common ancestral origin. The plant defensin proteins inhibit colonization of a broad range of filamentous ascomycetes, such as *Fusarium graminearum*, *B. cinerea*, or *A. brassicicola* (de Zelicourt et al., 2007; Stotz et al., 2009; Sagaram et al., 2011). Especially the *PDF1.2* plant defensin gene was found to be JA-dependent; the JA signaling-deficient mutant depressed the *PDF1.2* expression and represented high susceptibility to necrotrophic fungus (Veronese et al., 2004). *PDF1.2* probably depends on *BIK1*-mediated pathway also, because induction of the gene was significantly lower in the *bik1* mutant in response to pathogen infection, than in wild genotype (Veronese et al., 2006). Plant are able to express genes, which directly deactivate hazardous compounds of the pathogen. As an example a specific detoxification gene found in maize, the *Hm1* encoding HC toxin reductase neutralized the cyclic tetrapeptide toxin produced by *Colchiobolus carbonum* (Johal and Briggs, 1992).

Quantitative disease resistance loci

The quantitative resistance loci (QRLs) provide broad-spectrum and long-lasting resistance against microorganisms. More than hundred resistance loci are found in wheat against *Fusarium* head blight (FHB) already (Buerstmayr et al., 2009). In a breeding experiment 19 QTL-Near Isogenic Line pairs (with *Fusarium* resistance locus) developed by microsatellite markers showed significant resistance to head blight (Pumphrey et al., 2007). In 'Arina' wheat cultivar the resistance QTL to FHB was localized on chromosome 4D, which results in stable resistivity against

DOI: 10.18380/SZIE.COLUM.2015.1.37.

the fungus (Draeger et al., 2007), while according to another study using the GK Mini Manó/Frontana DH population the FHB resistance loci of 'Frontana' are located on chromosomes1B, 2D, 3B, 5A, 5B and 6B (Szabó-Hevér et al., 2014). Recombinant Inbred Lines (RIL) were developed and used to test tolerance to barley leaf rust *(Puccinia hordei)* by crossing the susceptible 'L94' and resistant 'Vada' varieties. The lines contained six QRLs *(Rphq1-Rphq6)*, which were found to be responsible for partial resistance against *P. hordei* (Qi et al., 1998). The head smut is a serious disease of maize, the *qHSR1* QRL provides resistance against it without any change in other agronomic traits (Zhao et al., 2012). The *Sr36/Pm6* gene cluster resulted in significant resistance in wheat cultivars against stem rust (Purnhauser et al., 2011). Studies of resistance to powdery mildew in cultivated grapevine demonstrated that single dominant genes (*REN1*, *RUN1*) regulate responses to evade pathogenic infection (Molnár, 2007; Hoffmann et al., 2008; Kozma et al., 2009; Katula-Debreceni et al., 2010; Li et al., 2013). Especially, the *REN1* was found to co-segregate with the NBS-LRR gene cluster in 'Dzhandhzal kara' and 'Kishmish vatkana' resistant *Vitis vinifera* varieties (Coleman et al., 2009). Adaptation of QDRs along with *R* genes may maintain a broad range defense system for susceptible plants.

Plant susceptibility genes

Pathogens enter the plant cell by punching the cell wall, or intrude through wounds and leaf stomata. These entry processes may also be achieved by the assistance of plant. Many genes are identified to be required in the host for compliance of infection. If that certain gene is not carried by the plant, the pathogen may not be able to infect it. The first discovered *S* gene was identified in barley, called the *Mildew Resistance Locus O*, *MLO* (Jørgensen, 1976). The MLO seven transmembrane protein is integrated into the cell membrane and supports development of haustoria of filamentous biotrophs. This *S* factor requires Ca^{2+} and calmodulin to suppress defense responses (Ayliffe and Lagudah, 2004), and is independent from JA/ET or SA-mediated signaling (Consonni et al., 2006). The loss of function *mlo* resulted an interaction between syntaxin, SNARE (Ror2) and SNAP (HvSNAP34) proteins and promoted the membrane vesicle fusion (Ayliffe and Lagudah, 2004). This vesicle trafficking increased resistance to *Blumeria graminis* fsp. *hordei* powdery mildew and other pathogens resembling a 'non-host resistance' trait (Humphry et al., 2006). Similarly to MLO the BAX inhibitor-1 (BI-1) protein compose a six or seven transmembrane complex, it allows the penetration of *B. graminis*, and additionally it suppresses programmed cell death (Eichmann et al., 2004; Eichmann et al., 2010). Interestingly the overexpression of this gene restored the PM penetration success in *mlo* mutants, as well as *MLO* overexpression in *bi-1* mutants (Huckelhoven et al., 2003). BI-1 protein belongs to the Lifeguard protein family, in which the members were found to negatively regulate the cell death too (Hu et al., 2009). The modulation of the cell surface may also limit the invasion of the attacking organism. The decreased very-long-chain aldehyde levels of *glossy11* maize mutant leaf surface inhibited PM spore germination (Hansjakob et al., 2011). Similarly the *irg1* and *ram2* mutations altered the *Medicago* leaf cuticle layer, therefore it became resistant to several pathogens (Uppalapati et al., 2012; Wang et al., 2012).

Expansins are used by plant for cell wall growth and stretch. The expansin *EXLA2* provide susceptibility to *B. cinerea* and *A. brassicicola*, probably by enabling pathogen entry. Mutation in *EXLA2* resulted in an additional side-effect; it increased hypersensitivity to abiotic stresses (Abuqamar et al., 2013). The cellulose synthase-like *(CSLA9)* gene was required for *Agrobacterium* attachment to the plant root surface, suggesting that *CSLA9* is an essential

DOI: 10.18380/SZIE.COLUM.2015.1.37.

cue for host recognition (Zhu et al., 2003). The *AtCLCd* chloride channel encoding gene repressed the Flg22-triggered immunity in *Arabidopsis*. The T-DNA insertion 'knock-out' mutants represented enhanced response to Flg22, and increased resistance to virulent strain of the bacterial pathogen *Pseudomonas syringae* pv. *tomato* DC3000 (Guo et al., 2014). The small G proteins genes (RAC/ROP) also regulate vesicle trafficking; among them the *HvRAC1*, *HvRAC3*, and *HvROP6* encode susceptibility factors in barley. Overexpression of these genes increased the sensitivity to PM infection to a greater extent (Schultheiss et al., 2002; Pathuri et al., 2008). The orthologs identified in rice *(OsRAC4, OsRAC5, OsRACB)* acted as compatibility factors also in response to the adapted fungus, *Magnaporthe oryzae* (Jung et al., 2006; Chen et al., 2010a). However the *HvRAC1* provided resistance against the non-adapted *M. oryzae* in barley (Pathuri et al., 2008), which indicates specificity of the gene to the attacker. A thiopurine methyltransferase (ubiquitin-conjugating enzyme), and an ADP ribosylation factor-GTPase-activating protein (ARF-GAP) acted as candidates of *Blumeria graminis* f. sp. *hordei* effector molecule (Schmidt et al., 2014). Schmidt and co-workers suggests that the *ARF-GAP* vesicle trafficking genes are conserved targets of mildew effectors. Taken together, the genes mediating cytoskeleton rearrangements and vesicle trafficking *(MLO, LFG, BI-1, ROP, RAC)* are responsible for sensitivity to adapted but resistance to non-adapted fungi (van Schie and Takken, 2014).

Pathogens may sustain infection by inhibition of defense signaling or response. The SA signaling obtains key role in defense system against biotrophs, therefore enhancing this pathway increases resistance. The SA 3-hydroxylase enzyme degrades SA by conversion it into 2,3-DHBA in *Arabidopsis*. The mutation in gene encoding this enzyme resulted in SA accumulation and increased tolerance against *P. syringae* (Zhang et al., 2013). SA signaling is escalated in response to biotrophs, but not to necrotrophs. The bHLH3/13/14/17 (basic loop-helix-loop) transcription factors found to suppress JA signaling; the quadruple knock-out mutant expressively increased innate JA and resistance to *B. cinerea*. However due to the antagonistic relation (Robert-Seilaniantz et al., 2011), in these plants the JA signaling was intensified along with repression of SA pathway, therefore the appearance of susceptibility to biotrophic pathogen (Song et al., 2013). Mutation in cellulose synthase genes activated the JA and ET mediated defense responses, and enhanced resistance against pathogens (Ellis and Turner, 2001; Ellis et al., 2002; Hernandez-Blanco et al., 2007). Interestingly, in this case the decreased cellulose content triggered the immune response. This is probably in association with accumulation of oligogalacturonides (cellulose precursors), which mimic DAMP and trigger DTI (van Schie and Takken, 2014). Similarly to this, although the increased callose content benefit resistance, *pmr4* callose synthesis inhibited mutant also showed decreased susceptibility to PM species. The accumulated oligosaccharides - which are able to induce the DTI - can be the explanation for this (Nishimura et al., 2003). The *PMR4* down-regulation generated a total resistance to the adapted, but not to the non-adapted fungi (Jacobs et al., 2003; Huibers et al., 2013).

Sequentially, after recognition of the pathogen, the phosphorylation-mediated MAP kinase cascade activates the response to biotic stress. Therefore the molecules inactivating the cascade components can be considered as susceptibility factors too. The MAPK phosphatases (MKPs) dephosphorylate the cascade components, in this way abolish its function. The *MKP1* and *MKP2* loss of function mutant provoked decreased susceptibility to virulent *Ralstonia* and *Pseudomonas* bacteria (Bartels et al., 2009; Lumbreras et al., 2010; Anderson et al., 2011). On the contrary, some MAPKs repress PTI; the *MPK4* of soybean and *MAPK5* of rice reduced activity, therefore

DOI: 10.18380/SZIE.COLUM.2015.1.37.

the effectiveness of PTI. The mutation in these genes resulted in increased resistance to several pathogens (Xiong and Yang, 2003; Liu et al., 2011). The *enhanced disease resistance 1 (EDR1)* locus encodes a putative MAPKK kinase, which was found to negatively regulate the SA-mediated responses in *Arabidopsis* (Frye et al., 2001). However it also depends on the ethylene signal, since the *ein* mutation altered the expression of *EDR1* in response to senescence. The EDR1 probably acts in a cross-talk between ET and SA-mediated pathway operating in cell death and ageing (Tang et al., 2005). Followed the activation of MAPKs-mediated cascade, the transcription factors (TFs) actuate the defense reaction. The mostly active TFs during infection are the WRKY transcription factors, which were found to positively or negatively regulate defense. Especially the rice *WRRKY45-2* gene acted as susceptibility factor against *X. oryzae*, but the homolog gene, which differs only in a few amino acids positively regulated defense against the same pathogen (Tao et al., 2009). The *Arabidopsis AtWRKY18/40/60* regulatory genes played a role in tempering the SA-mediated defense pathway. Double or triple mutants increased resistance to biotrophic *P. syringae* and susceptibility to *B. cinerea* compared to wild-type plant (Xu et al., 2006). Another transcription factor, the TaNAC21/22 was found to negatively regulate defense against stripe rust, *Puccinia striiformis* f. sp. *tritici*. This *NAC* gene was the target of tae-miR164 microRNA, and this miRNA was found in earlier studies to regulate defense responses (Feng et al., 2014). The calcium-/calmodulin- and lipid-binding proteins also suppress the defense reaction in host plants. The SR1 calmodulin-binding transcription factor repressed the immunity by directly binding to the *EDS1*, *NDR1* and *EIN3* promoters (Du et al., 2009; Nie et al., 2012). The lipids act as signaling molecules and are required for ETI and HR (Andersson et al., 2006). The lesion mimic mutant, *acd11* had limited sphingosine (a sphingolipid) transfer protein content increasing the cellular SA level and resistance to biotrophs. Similar function was found with the sphingolipid fatty acid hydroxylase gene, the *AtFAH1/2* (Brodersen et al., 2002; Konig et al., 2012). The SA-mediated defense response is suppressed by fatty acid desaturase (FAD7). In the *fad7* mutant the basal SA level did not show alteration, but in response to aphid attack, the SA accumulated along with enhanced defense (Avila et al., 2012).

Once the pathogen passes the plant defense barriers, the plant is forced to sustain the attacker. Additionally these organisms are able to manipulate plant metabolism to fulfill their nutritional needs and to facilitate their replication and spread. Maintenance involves the modification of host sugar transport; the cell membrane localized sugar transporters (SWEET11 and SWEET13) were forced by *X. oryzae* to transfer more sugar into the intercellular region. Mutation in these genes abolished *X. oryzae* proliferation (Chen et al., 2010b). SWEET11 associated with copper transporter, COPT1, which was also required for susceptibility to *X. oryzae* (Yuan et al., 2010). The alcohol dehydrogenase gene *(ADH)* was up-regulated by PM in barley, however the mutation in *ADH* inhibited PM proliferation (Pathuri et al., 2011). Lipids may also be utilized by the pathogens; the maize *lox3* lipoxygenase mutant plant became full resistant to three different fungus genus. The *lox3* inactivation also blocked the toxin production of *Fusarium* (Gao et al., 2007). The reason of the increased resistance to biotrophs may be explained by the repressed JA synthesis in mutant plant. Therefore inhibition of lipoxygenase activity depress JA synthesis and SA signaling can be enhanced along with defense against biotrophs (Gao et al., 2009).

Hypertrophy and endoreduplication of plant cell also benefit pathogens maintenance. The increased cell size extends the nutrient or water content; the endoreduplication results in multiplication of chromosomes override

overall metabolism of the host. *Xanthomonas* infection induced cell size enlargement in pepper by triggering bHLH transcription factor Upa20 activity via its AvrBs3 effector (Kay et al., 2007). The genes *PMR5* and *PMR6* encode pectate-lyases, which play a role in completion the accommodation of PM haustorium. The presence of these genes is required at later stage of infection and they are independent of SA-mediated signaling (Vogel et al., 2002; 2004). Additionally recent study represented that these genes were influenced by the pathogen to modulate ploidy level of mesophyll cells under the infected, haustorium containing epidermal cells. Therefore the metabolic capacity could be enhanced at the site of infection (Chandran et al., 2010; 2013). Intriguingly, although in *pmr5* and *pmr6* mutants the penetration efficiency was not repressed, the fungus developed less hyphae, conidiophores, and conidia (Vogel et al., 2002; 2004). Substantial study observed that cell cycle regulatory genes were up-regulated in mesophyll cells at infection sites: some cyclin-dependent kinases (CDKs), CDK inhibitors and the *MYB3R4* transcription factor, known to be regulator of G2/M transition. The mutation in *MYB3R4* and in *PUX2* (plant ubiquitin regulatory X domain-containing protein 2) abolished the endorduplication along with weakened *Golovinomyces orontii* colonization (Chandran et al., 2010). The plant susceptibility factors may support pathogen replication as the eIF4E cap-binding and eIF4G scaffold protein do (Kawaguchi and Bailey-Serres, 2002). These proteins are part of the host translation initiation complex and are forced by potyviruses to translate viral RNA too. The viral RNA 5' end is covered by the viral VPg protein, which interacts with the plant eIF4E (Wittmann et al., 1997). Additionally eIF4E also acted as susceptibility factor of melon tombusvirus and *Arabidopsis* bromovirus (Yoshii et al., 2004; Nieto et al., 2006). Isoforms and mutations of eIF4E and eIF4G were found in many plant species that abolish susceptibility to potyviruses (Wang and Krishnaswamy, 2012).

Using single marker association and linkage disequlibrium analysis a powdery mildew susceptibility locus (*Sen1*) was identified in 'Chardonnay' grape (*Vitis vinifera* L.), which can be applied for negative selection in breeding programs (Barba et al., 2014)

Since the *S* genes mostly possess primary function, disabling the S factor may result in negative side-effects beside the desired decrease in susceptibility. In several cases the mutation increases resistance to biotrophs along with susceptibility to necrotrophs (Veronese et al., 2006; Lai et al., 2008) and vice versa (Flors et al., 2007; Mang et al., 2009). It is likely the result of the antagonistic relation between SA- and JA/ET-mediated signaling and their regulating elements. Along with the enhanced susceptibility to other pathogens, other phenotypic changes may also occur, such as stunted plants, susceptibility to abiotic stresses (Bessire et al., 2007; Tang et al., 2007). Therefore monitoring the pleiotropic effect mediated by inactivation of an *S* gene is highly recommended. If the new trait was tested in other plant species, it may act totally differently in the desired genetic background. Additionally the new variety should also be evaluated in field conditions, because interaction with beneficial microbes or resistance-breaking pathogens may alter the performance (van Schie and Takken, 2014). As an example, the *mlo* and *eIF4E*-based resistance have been used for many decades (Cook, 1961; Jørgensen, 1992; Moury and Verdin, 2012). Although the *mlo* provided penetration block it did not result in any resistance-breaking pathogen strain, the *eIF4E* raised new resistance-breaking potyviruses with mutation in viral protein VPg (Masuta et al., 1999).

Perspectives, breeding strategies

Overexpression of *R* genes, or T-DNA knockout of *S* genes requires genetic transformation of the

DOI: 10.18380/SZIE.COLUM.2015.1.37.

plant, which is usually done by *Agrobacterium* or the gene-gun. The current GMO regulation in the European Union discourages the application of transgenic plants in agriculture. The GMO legislation of EU declares that the evaluation of crops is based on the method used for breeding, instead of agronomic value of the new variety. The regulation allows MAS method for searching and integration of *R* genes or QDRs into susceptible variety. However in some cases the QDRs provide only partial resistance, in turn the *R* genes are race-specific. The monogenic, *S* gene-based defense covers more durable resistance to multiple diseases. Especially the obligate biotrophs depend strongly on function of host factor, such as the biosynthesis of essential metabolites, therefore the inactivation of these compatibility factors disables pathogen invasion. The recent inventions provide good solutions for inactivation of the specific susceptibility gene without T-DNA insertion. The transcription activator-like (TAL) effector nucleases of pathogens may be customized to introduce mutation at designated location of the *S* gene (Bogdanove, 2014). Ironically the pathogen virulence factor can now be modified to apply against them. Since the breeding technology develops exponentially, the recent GMO principle, as well as the regulation of the applied techniques should be under debate (Hartung and Schiemann, 2014).

Acknowledgement

This study was financially supported by the *KTIA_AIK_12-1-2012-0012* and the Research Centre of Excellence- *9878/2015/FEKUT* Grant of the Hungarian Ministry of Education.

References

Abuqamar S., Ajeb S., Sham A., Enan M. R., Iratni R. (2013). A mutation in the *expansin-like A2* gene enhances resistance to necrotrophic fungi and hypersensitivity to abiotic stress in *Arabidopsis thaliana*. Mol Plant Pathol. **14:** 8. 813-827. DOI: http://dx.doi.org/10.1111/mpp.12049

Aerts A. M., Francois I. E., Cammue B. P., Thevissen K. (2008). The mode of antifungal action of plant, insect and human defensins. Cell Mol Life Sci. **65:** 13. 2069-2079. DOI: http://dx.doi.org/10.1007/s00018-008-8035-0

Anderson J. C., Bartels S., Gonzalez Besteiro M. A., Shahollari B., Ulm R., Peck S. C. (2011). *Arabidopsis* MAP Kinase Phosphatase 1 (AtMKP1) negatively regulates MPK6-mediated PAMP responses and resistance against bacteria. Plant J. **67:** 2. 258-268. DOI: http://dx.doi.org/10.1111/j.1365-313x.2011.04588.x

Andersson M. X., Kourtchenko O., Dangl J. L., Mackey D., Ellerstrom M. (2006). Phospholipase-dependent signalling during the AvrRpm1- and AvrRpt2-induced disease resistance responses in *Arabidopsis thaliana*. Plant J. **47:** 6. 947-959. DOI: http://dx.doi.org/10.1111/j.1365-313x.2006.02844.x

Avila C. A., Arevalo-Soliz L. M., Jia L., Navarre D. A., Chen Z., Howe G. A., Meng Q. W., Smith J. E., Goggin F. L. (2012). Loss of function of *FATTY ACID DESATURASE7* in tomato enhances basal aphid resistance in a salicylate-dependent manner. Plant Physiol. **158:** 4. 2028-2041. DOI: http://dx.doi.org/10.1104/pp.111.191262

Ayliffe M. A., Lagudah E. S. (2004). Molecular genetics of disease resistance in cereals. Annals of Botany. **94:** 6. 765-773. DOI: http://dx.doi.org/10.1093/aob/mch207

Bacsó R., Hafez Y., Király Z., Király L. (2011). Inhibition of virus replication and symptom expression by reactive oxygen species in tobacco infected with Tobacco mosaic virus. Acta Phytopathologica et Entomologica Hungarica. **46:** 1. 1-10. DOI: http://dx.doi.org/10.1556/aphyt.46.2011.1.1

Barba P., Cadle-Davidson L., Harriman J., Glaubitz J. C., Brooks S., Hyma K., Reisch B. (2014). Grapevine powdery mildew resistance and susceptibility loci identified on a high-resolution SNP map. Theoretaical and Applied Genetics **127:** 73-84. DOI: http://dx.doi.org/10.1007/s00122-013-2202-x

Bartels S., Anderson J. C., González Besteiro M. A., Carreri A., Hirt H., Buchala A., Métraux J. P., Peck S. C., Ulm R. (2009). *MAP KINASE PHOSPHATASE1* and *PROTEIN TYROSINE PHOSPHATASE1* are repressors of salicylic acid synthesis and *SNC1*-mediated responses in *Arabidopsis*. Plant Cell. **21:** 9. 2884-2897. DOI: http://dx.doi.org/10.1105/tpc.109.067678

Behr M., Humbeck K., Hause G., Deising H. B., Wirsel S. G. (2010). The hemibiotroph *Colletotrichum graminicola* locally induces photosynthetically active green islands but globally accelerates senescence on aging maize leaves. Mol Plant Microbe Interact. **23:** 7. 879-892. DOI: http://dx.doi.org/10.1094/mpmi-23-7-0879

Bessire M., Chassot C., Jacquat A. C., Humphry M., Borel S., Petetot J. M., Metraux J. P., Nawrath C. (2007). A permeable cuticle in *Arabidopsis* leads to a strong resistance to *Botrytis cinerea*. EMBO J. **26:** 8. 2158-2168. DOI: http://dx.doi.org/10.1038/sj.emboj.7601658

Bogdanove A. J. (2014). Principles and applications of TAL effectors for plant physiology and metabolism. Curr Opin Plant Biol. **19:** 99-104. DOI: http://dx.doi.org/10.1016/j.pbi.2014.05.007

Bohman S., Staal J., Thomma B. P., Wang M., Dixelius C. (2004). Characterisation of an *Arabidopsis-Leptosphaeria* maculans pathosystem: resistance partially requires camalexin biosynthesis and is independent of salicylic acid, ethylene and jasmonic acid signalling. Plant J. **37:** 1. 9-20. DOI: http://dx.doi.org/10.1046/j.1365-313x.2003.01927.x

Bozsó Z., Ott P. G., Szatmari A., Czelleng A., Varga G., Besenyei E., Sárdi É., Bányai É., Klement Z. (2005) Early detection of bacterium-induced basal resistance in tobacco leaves with diaminobenzidine and dichlorofluorescein diacetate .J Phytopathol. **153:** 10. 596-607. DOI: http://dx.doi.org/10.1111/j.1439-0434.2005.01026.x

Brodersen P., Petersen M., Pike H. M., Olszak B., Skov S., Ødum N., Jørgensen L. B., Brown R. E., Mundy J. (2002). Knockout of *Arabidopsis ACCELERATED-CELL-DEATH11* encoding a sphingosine transfer protein causes activation of programmed cell death and defense. Genes Dev. **16:** 4. 490-502. DOI: http://dx.doi.org/10.1101/gad.218202

Bryan G. T., Wu K. S., Farrall L., Jia Y., Hershey H. P., McAdams S. A., Faulk K. N., Donaldson G. K., Tarchini R., Valent B. (2000). A single amino acid difference distinguishes resistant and susceptible alleles of the rice blast resistance gene *Pi-ta*. Plant Cell. **12:** 11. 2033-2046. DOI: http://dx.doi.org/10.2307/3871103

Buerstmayr H., Ban T., Andreson J. A. (2009). QTL mapping and marker-assisted selection for *Fusarium* head blight resistance in wheat. Plant Breeding. **128:** 1. 1-26. DOI: http://dx.doi.org/10.1111/j.1439-0523.2008.01550.x

Bushnell W. R., Allen P. J. (1962). Induction of disease symptoms in barley by powdery mildew. Plant Physiol. **37:** 1. 50-59. DOI: http://dx.doi.org/10.1104/pp.37.1.50

Chandran D., Inada N., Hather G., Kleindt C. K., Wildermuth M. C. (2010). Laser microdissection of *Arabidopsis* cells at the powdery mildew infection site reveals site-specific processes and regulators. Proc Natl Acad Sci U S A. **107:** 1. 460-465. DOI: http://dx.doi.org/10.1073/pnas.0912492107

Chandran D., Rickert J., Cherk C., Dotson B. R., Wildermuth M. C. (2013). Host cell ploidy underlying the fungal feeding site is a determinant of powdery mildew growth and reproduction. Mol Plant Microbe Interact. **26:** 5. 537-545. DOI: http://dx.doi.org/10.1094/mpmi-10-12-0254-r

Chen L., Shiotani K., Togashi T., Miki D., Aoyama M., Wong H. L., Kawasaki T., Shimamoto K. (2010a). Analysis of the Rac/Rop small GTPase family in rice: expression, subcellular localization and role in disease resistance. Plant Cell Physiol. **51:** 4. 585-595. DOI: http://dx.doi.org/10.1038/nature09606

Chen L. Q., Hou B. H., Lalonde S., Takanaga H., Hartung M. L., Qu X. Q., Guo W. J., Kim J. G., Underwood W., Chaudhuri B., Chermak D., Antony G., White F. F., Somerville S. C., Mudgett M. B., Frommer W. B. (2010b). Sugar transporters for intercellular exchange and nutrition of pathogens. Nature. **468:** 7323. 527-532. DOI: http://dx.doi.org/10.1038/nature09606

 DOI: 10.18380/SZIE.COLUM.2015.1.37.

Coleman C., Copetti D., Cipriani G., Hoffmann S., Kozma P., Kovács L., Morgante M., Testolin R., Di Gaspero G. (2009) The powdery mildew resistance gene *REN1* co-segregates with an *NBS-LRR* gene cluster in two Central Asian grapevines. BMC Genet. **10:** 89. DOI: http://dx.doi.org/10.1186/1471-2156-10-89

Consonni C., Humphry M. E., Hartmann H. A., Livaja M., Durner J., Westphal L., Vogel J., Lipka V., Kemmerling B., Schulze-Lefert P., Somerville S. C., Panstruga R. (2006). Conserved requirement for a plant host cell protein in powdery mildew pathogenesis. Nat Genet. **38:** 6. 716-720. DOI: http://dx.doi.org/10.1038/ng1806

Cook A. A. (1961). A mutation for resistance to potato virus Y in pepper. Phytopathology. **51:** 550-552.

de Zelicourt A., Letousey P., Thoiron S., Campion C., Simoneau P., Elmorjani K., Marion D., Simier P., Delavault P. (2007). Ha-DEF1, a sunflower defensin, induces cell death in *Orobanche* parasitic plants. Planta. **226:** 3. 591-600. DOI: http://dx.doi.org/10.1007/s00425-007-0507-1

Deslandes L., Olivier J., Peeters N., Feng D. X., Khounlotham M., Boucher C., Somssich I., Genin S., Marco Y. (2003). Physical interaction between RRS1-R, a protein conferring resistance to bacterial wilt, and PopP2, a type III effector targeted to the plant nucleus. Proc Natl Acad Sci U S A. **100:** 13. 8024-8029. DOI: http://dx.doi.org/10.1073/pnas.1230660100

DeYoung B. J., Innes R. W. (2006). Plant NBS-LRR proteins in pathogen sensing and host defense. Nature Immunology. **7:** 12. 1243-1249. DOI: http://dx.doi.org/10.1038/ni1410

Draeger R., Gosman N., Steed A., Chandler E., Thomsett M., Srinivasachary, Schondelmaier J., Buerstmayr H., Lemmens M., Schmolke M., Mesterházy Á., Nicholson P. (2007) Identification of QTLs for resistance to *Fusarium* head blight, DON accumulation and associated traits in the winter wheat variety Arina. Theor Appl Genet. **115:** 5. 617-625. DOI: http://dx.doi.org/10.1007/s00122-007-0592-3

Du L., Ali G. S., Simons K. A., Hou J., Yang T., Reddy A. S., Poovaiah B. W. (2009). Ca(2+)/ calmodulin regulates salicylic-acid-mediated plant immunity. Nature. **457:** 7233. 1154-1158. DOI: http://dx.doi.org/10.1038/nature07612

Eichmann R., Bischof M., Weis C., Shaw J., Lacomme C., Schweizer P., Duchkov D., Hensel G., Kumlehn J., Huckelhoven R. (2010). *BAX INHIBITOR-1* is required for full susceptibility of barley to powdery mildew. Mol Plant Microbe Interact. **23:** 9. 1217-1227. DOI: http://dx.doi.org/10.1094/mpmi-23-9-1217

Eichmann R., Schultheiss H., Kogel K. H., Huckelhoven R. (2004). The barley apoptosis suppressor homologue *BAX inhibitor-1* compromises nonhost penetration resistance of barley to the inappropriate pathogen *Blumeria graminis* f. sp. *tritici*. Mol Plant Microbe Interact. **17:** 5. 484-490. DOI: http://dx.doi.org/10.1094/mpmi.2004.17.5.484

Ellis C., Karafyllidis I., Wasternack C., Turner J. G. (2002). The *Arabidopsis* mutant *cev1* links cell wall signaling to jasmonate and ethylene responses. Plant Cell. **14:** 7. 1557-1566. DOI: http://dx.doi.org/10.1105/tpc.002022

Ellis C., Turner J. G. (2001). The *Arabidopsis* mutant *cev1* has constitutively active jasmonate and ethylene signal pathways and enhanced resistance to pathogens. Plant Cell. **13:** 5. 1025-1033. DOI: http://dx.doi.org/10.1105/tpc.13.5.1025

Feng H., Duan X., Zhang Q., Li X., Wang B., Huang L., Wang X., Kang Z. (2014). The target gene of *tae-miR164*, a novel NAC transcription factor from the NAM subfamily, negatively regulates resistance of wheat to stripe rust. Mol Plant Pathol. **15:** 3. 284-296. DOI: http://dx.doi.org/10.1111/mpp.12089

Flor H. H. (1942). Inheritance of pathogenicity in *Melampsora lini*. Phytopath. 653-669.

Flor H. H. (1971). Current status of the gene-for-gene concept. Annu Rev Phytopathol. **9:** 275–296. DOI: http://dx.doi.org/10.1146/annurev.py.09.090171.001423

Flors V., Leyva Mde L., Vicedo B., Finiti I., Real M. D., Garcia-Agustin P., Bennett A. B., Gonzalez-Bosch C. (2007). Absence of the endo-beta-1,4-glucanases *Cel1* and *Cel2* reduces susceptibility to *Botrytis cinerea* in tomato. Plant J. **52:** 6. 1027-1040. DOI: http://dx.doi.org/10.1111/j.1365-313x.2007.03299.x

Fodor J., Gullner G., Ádám A. L., Barna B., Kömives T., Király Z. (1997) Local and systemic responses of antioxidants to tobacco mosaic virus infection and to salicylic acid in tobacco (role in systemic acquired resistance). Plant Physiol. **114:** 4. 1443-1451.

Frye C. A., Tang D., Innes R. W. (2001). Negative regulation of defense responses in plants by a conserved MAPKK kinase. Proc Natl Acad Sci U S A. **98:** 1. 373-378. DOI: http://dx.doi.org/10.1073/pnas.98.1.373

Gao X., Brodhagen M., Isakeit T., Brown S. H., Gobel C., Betran J., Feussner I., Keller N. P., Kolomiets M. V. (2009). Inactivation of the lipoxygenase *ZmLOX3* increases susceptibility of maize to *Aspergillus* spp. Mol Plant Microbe Interact. **22:** 2. 222-231. DOI: http://dx.doi.org/10.1094/mpmi-22-2-0222

Gao X., Shim W-B., Göbel C., Kunze S., Feussner I., Meeley R., Balint-Kurti P., Kolomiets M. (2007). Disruption of a maize 9-lipoxygenase results in increased resistance to fungal pathogens and reduced levels of contamination with mycotoxin fumonisin. Mol Plant Microbe Interact. **20:** 8. 922-933. DOI: http://dx.doi.org/10.1094/mpmi-20-8-0922

Glazebrook J. (2005). Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. Annu Rev Phytopathol. **43:** 205-227. DOI: http://dx.doi.org/10.1146/annurev.phyto.43.040204.135923

Guo W., Zuo Z., Cheng X., Sun J., Li H., Li L., Qiu J-L. (2014). The chloride channel family gene *CLCd* negatively regulates pathogen-associated molecular pattern (PAMP)-triggered immunity in *Arabidopsis*. J Exp Bot. **65:** 4. 1205-1215. DOI: http://dx.doi.org/10.1093/jxb/ert484

Gururania M. A., Venkatesh J., Upadhyaya C. P., Nookaraju A., Pandey S. K., Park S. W. (2012). Plant disease resistance genes: Current status and future directions. Physiological and Molecular Plant Pathology **78:**51–65.

Hansjakob A., Riederer M., Hildebrandt U. (2011). Wax matters: abscence of very-long-chain aldehydes from the leaf cuticular wax of the *glossy11* mutant of maize compromises the prepenetration proccesses of *Blumria graminis*. Plant Pathology. **60:** 6. 1151-1161. DOI: http://dx.doi.org/10.1111/j.1365-3059.2011.02467.x

Hartung F., Schiemann J. (2014). Precise plant breeding using new genome editing techniques: opportunities, safety and regulation in the EU. Plant J. **78:** 5. 742-752. DOI: http://dx.doi.org/10.1111/tpj.12413

Hegedüs N., Marx F. (2013). Antifungal proteins: More than antimicrobials? Fungal Biol Rev. **26:** 4. 132-145. DOI: http://dx.doi.org/10.1016/j.fbr.2012.07.002

Hernandez-Blanco C., Feng D. X., Hu J., Sanchez-Vallet A., Deslandes L., Llorente F., Berrocal-Lobo M., Keller H., Barlet X., Sanchez-Rodriguez C., Anderson L. K., Somerville S., Marco Y., Molina A. (2007). Impairment of cellulose synthases required for *Arabidopsis* secondary cell wall formation enhances disease resistance. Plant Cell. **19:** 3. 890-903. DOI: http://dx.doi.org/10.1105/tpc.106.048058

Hoffmann S., Di Gaspero G., Kovacs L., Howard S., Kiss E., Galbacs Z., Testolin R., Kozma P. (2008) Resistance to *5* in the grapevine 'Kishmish vatkana' is controlled by a single locus through restriction of hyphal growth. Theor Appl Genet **116:** 427-438. DOI: http://dx.doi.org/10.1007/s00122-007-0680-4

Hu L., Smith T. F., Goldberger G. (2009). *LFG*: a candidate apoptosis regulatory gene family. Apoptosis. **14:** 11. 1255-1265. DOI: http://dx.doi.org/10.1007/s10495-009-0402-2

Huckelhoven R., Dechert C., Kogel K. H. (2003). Overexpression of barley *BAX inhibitor 1* induces breakdown of mlo-mediated penetration resistance to *Blumeria graminis*. Proc Natl Acad Sci U S A. **100:** 9. 5555-5560. DOI: http://dx.doi.org/10.1073/pnas.0931464100

Huckelhoven R., Fodor J., Preis C., Kogel K. H. (1999) Hypersensitive cell death and papilla formation in barley attacked by the powdery mildew fungus are associated with hydrogen peroxide but not with salicylic acid accumulation. Plant Physiol. **119:** 4. 1251-1260. DOI: http://dx.doi.org/10.1104/pp.119.4.1251

Huibers R. P., Loonen A. E., Gao D., Van den Ackerveken G., Visser R. G., Bai Y. (2013). Powdery mildew resistance in tomato by impairment of *SlPMR4* and *SlDMR1*. PLoS One. **8:** 6. e67467. DOI: http://dx.doi.org/10.1371/journal.pone.0067467

 DOI: 10.18380/SZIE.COLUM.2015.1.37.

Humphry M., Consonni C., Panstruga R. (2006). *Mlo*-based powdery mildew immunity: silver bullet or simply non-host resistance? Mol Plant Pathol. **7:** 6. 605-610. DOI: http://dx.doi.org/10.1111/j.1364-3703.2006.00362.x

Jacobs A. K., Lipka V., Burton R. A., Panstruga R., Strizhov N., Schulze-Lefert P., Fincher G. B. (2003). An *Arabidopsis* callose synthase, *GSL5*, is required for wound and papillary callose formation. Plant Cell. **15:** 11. 2503-2513. DOI: http://dx.doi.org/10.1105/tpc.016097

Johal G. S., Briggs S. P. (1992). Reductase activity encoded by the *HM1* disease resistance gene in maize. Science. **258:** 5184. 985-987. DOI: http://dx.doi.org/10.1126/science.1359642

Jones D. A., Thomas C. M., Hammond-Kosack K. E., Balint-Kurti P. J., Jones J. D. (1994). Isolation of the tomato *Cf-9* gene for resistance to *Cladosporium fulvum* by transposon tagging. Science. **266:** 5186. 789-793. DOI: http://dx.doi.org/10.1126/science.7973631

Jung Y. H., Agrawal G. K., Rakwal R., Kim J. A., Lee M. O., Choi P. G., Kim Y. J., Kim M. J., Shibato J., Kim S. H., Iwahashi H., Jwa N. S. (2006). Functional characterization of *OsRacB* GTPase--a potentially negative regulator of basal disease resistance in rice. Plant Physiol Biochem. **44:** 1. 68-77. DOI: http://dx.doi.org/10.1016/j.plaphy.2005.12.001

Jørgensen J. H. (1992). Discovery, characterization and exploitation of *Mlo* powdery mildew resistance in barley. Euphytica. **63:** 1. 141-152. DOI: http://dx.doi.org/10.1007/bf00023919

Jørgensen J. H. (1976). Identification of powdery mildew resistant barley mutants and their allelic relationship. Barley Genetics III Karl Thiemig. 446-455.

Katula-Debreceni D., Lencsés A. K., Szőke A., Veres A., Hoffmann S., Kozma P., Kovács L. G., Heszky L., Kiss E. (2010) Marker-assisted selection for two dominant powdery mildew resistance genes introgressed into a hybrid grape population. Scientia Horticulturae. **126:** 4. 448–453. DOI: http://dx.doi.org/10.1016/j.scienta.2010.08.012

Katsir L., Schilmiller A. L., Staswick P. E., He S. Y., Howe G. A. (2008). *COI1* is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. Proc Natl Acad Sci U S A. **105:** 19. 7100-7105. DOI: http://dx.doi.org/10.1073/pnas.0802332105

Kay S., Hahn S., Marois E., Hause G., Bonas U. (2007). A bacterial effector acts as a plant transcription factor and induces a cell size regulator. Science. **318:** 5850. 648-651. DOI: http://dx.doi.org/10.1126/science.1144956

Kawaguchi R., Bailey-Serres J. (2002). Regulation of translational initiation in plants. Curr Opin Plant Biol. **5:** 5. 460-465. DOI: http://dx.doi.org/10.1016/s1369-5266(02)00290-x

Király Z., Barna B., Érsek T. (1972) Hypersensitivity as a consequence, not the cause, of plant resistance to infection. Nature. **239:** 456-458. DOI: http://dx.doi.org/10.1038/239456a0

Klement Z., Bozsó Z., Kecskés M. L., Besenyei E., Czelleng A., Ott P.G. (2003) Local early induced resistance of plants as the first line of defence against bacteria. Pest Manag. Sci. **59:** 4. 465-474. DOI: http://dx.doi.org/10.1002/ps.694

Konig S., Feussner K., Schwarz M., Kaever A., Iven T., Landesfeind M., Ternes P., Karlovsky P., Lipka V., Feussner I. (2012). *Arabidopsis* mutants of sphingolipid fatty acid alpha-hydroxylases accumulate ceramides and salicylates. New Phytol. **196:** 4. 1086-1097. DOI: http://dx.doi.org/10.1111/j.1469-8137.2012.04351.x

Kozma P., Kiss E., Hoffmann S., Galbács Z., Tula D. (2009) Using the powdery mildew resistant *Muscadinia rotundifolia* and *Vitis vinifera* 'Kishmish vatkana' for breeding new cultivars. Acta Horticulturae. **827:** 559-564.

Lahaye T. (2002). The *Arabidopsis RRS1-R* disease resistance gene – uncovering the plant's nucleus as the new battlefield of plant defense? Trends in Plant Science. **7:** 10. 425-427. Doi: http://dx.doi.org/10.1016/s1360-1385(02)02334-8

Lai Z., Vinod K., Zheng Z., Fan B., Chen Z. (2008). Roles of *Arabidopsis* WRKY3 and WRKY4 transcription factors in plant responses to pathogens. BMC Plant Biol. **8:** 68. DOI: http://dx.doi.org/10.1186/1471-2229-8-68

Lee S. W., Han S. W., Sririyanum M., Park C. J., Seo Y. S., Ronald P. C. (2009). A type I-secreted, sulfated peptide triggers *XA21*-mediated innate immunity. Science. **326:** 5954. 850-853. DOI: http://dx.doi.org/10.1126/science.1173438

Li C., Erwin A., Pap D., Coleman C., Higgins A. D., Kiss E., Kozma P., Hoffmann S., Ramming D. W., Kovács L. G. (2013) Selection for *Run1-Ren1* dihybrid grapevines using microsatellite markers. Am. J. Enol. Vitic. **64:** 1. 152-155. DOI: http://dx.doi.org/10.1186/1471-2229-8-68

Liu J. Z., Horstman H. D., Braun E., Graham M. A., Zhang C., Navarre D., Qiu W. L., Lee Y., Nettleton D., Hill J. H., Whitham S. A. (2011). Soybean homologs of MPK4 negatively regulate defense responses and positively regulate growth and development. Plant Physiol. **157:** 3. 1363-1378. DOI: http://dx.doi.org/10.1104/pp.111.185686

Liu Y., Schiff M., Marathe R., Dinesh-Kumar S. P. (2002). Tobacco *Rar1*, *EDS1* and *NPR1*/*NIM1* like genes are required for *N*-mediated resistance to tobacco mosaic virus. Plant J. **30:** 4. 415-429. DOI: http://dx.doi.org/10.1046/j.1365-313x.2002.01297.x

Llorente F., Alonso-Blanco C., Sanchez-Rodriguez C., Jorda L., Molina A. (2005). ERECTA receptor-like kinase and heterotrimeric G protein from *Arabidopsis* are required for resistance to the necrotrophic fungus *Plectosphaerella cucumerina*. Plant J. **43:** 2. 165-180. DOI: http://dx.doi.org/10.1111/j.1365-313x.2005.02440.x

Lu D., Wu S., Gao X., Zhang Y., Shan L., He P. (2010). A receptor-like cytoplasmic kinase, BIK1, associates with a flagellin receptor complex to initiate plant innate immunity. Proc Natl Acad Sci U S A. **107:** 1. 496-501. DOI: http://dx.doi.org/10.1073/pnas.0909705107

Lumbreras V., Vilela B., Irar S., Sole M., Capellades M., Valls M., Coca M., Pages M. (2010). MAPK phosphatase MKP2 mediates disease responses in *Arabidopsis* and functionally interacts with MPK3 and MPK6. Plant J. **63:** 6. 1017-1030. DOI: http://dx.doi.org/10.1111/j.1365-313x.2010.04297.x

Mang H. G., Laluk K. A., Parsons E. P., Kosma D. K., Cooper B. R., Park H. C., AbuQamar S., Boccongelli C., Miyazaki S., Consiglio F., Chilosi G., Bohnert H. J., Bressan R. A., Mengiste T., Jenks M. A. (2009). The *Arabidopsis RESURRECTION1* gene regulates a novel antagonistic interaction in plant defense to biotrophs and necrotrophs. Plant Physiol. **151:** 1. 290-305. DOI: http://dx.doi.org/10.1104/pp.109.142158

Masuta C., Nishimura M., Morishita H., Hataya T. (1999). A single amino acid change in viral genome-associated protein of potato virus y correlates with resistance breaking in 'virgin a mutant' tobacco. Phytopathology. **89:** 2. 118-123. DOI: http://dx.doi.org/10.1094/phyto.1999.89.2.118

Molnár S., Galbács Z., Halász G., Hoffmann S., Kiss E., Kozma P., Veres A., Galli Z., Szőke A., Heszky L., (2007) Marker assisted selection (MAS) for powdery mildew resistance in a grapevine hybrid family. Vitis. **46:** 4. 212-213.

Moury B., Verdin E. (2012). Viruses of pepper crops in the Mediterranean basin: a remarkable stasis. Adv Virus Res. **84:** 127-162. DOI: http://dx.doi.org/10.1016/b978-0-12-394314-9.00004-x

Nicaise V., Roux M., Zipfel C. (2009). Recent advances in PAMP-triggered immunity against bacteria: pattern recognition receptors watch over and raise the alarm. Plant Physiol. **150:** 4. 1638-1647. DOi: http://dx.doi.org/10.1104/pp.109.139709

Nie H., Zhao C., Wu G., Wu Y., Chen Y., Tang D. (2012). SR1, a calmodulin-binding transcription factor, modulates plant defense and ethylene-induced senescence by directly regulating *NDR1* and *EIN3*. Plant Physiol. **158:** 4. 1847-1859. DOI: http://dx.doi.org/10.1104/pp.111.192310

 DOI: 10.18380/SZIE.COLUM.2015.1.37.

Nieto C., Morales M., Orieda G., Clepet C., Monfort A., Sturbois B., Puigdomènech P., Pitrat M., Caboche M., Dogimont C., Garcia-Mas J., Aranda M. A., Bendahmane A. (2006). An *eIF4E* allele confers resistance to an uncapped and non-polyadenylated RNA virus in melon. Plant J. **48:** 3. 452-462. DOI: http://dx.doi.org/10.1111/j.1365-313x.2006.02885.x

Nishimura M. T., Stein M., Hou B. H., Vogel J. P., Edwards H., Somerville S. C. (2003). Loss of a callose synthase results in salicylic acid-dependent disease resistance. Science. **301:** 5635. 969-972. DOI: http://dx.doi.org/10.1126/science.1086716

Oh C. S., Martin G. B. (2011). Effector-triggered immunity mediated by the Pto kinase. Trends Plant Sci. **16:** 3. 132-140. DOI: http://dx.doi.org/10.1016/j.tplants.2010.11.001

Ott P. G., Varga G. J., Szatmári Á., Bozsó Z., Klement É., Medzihradszky K. F., Besenyei E., Czelleng A., Klement Z. (2006) Novel extracellular chitinases rapidly and specifically induced by general bacterial elicitors and suppressed by virulent bacteria as a marker of early basal resistance in tobacco. Mol Plant Microbe Interact **19:** 161-172 DOI: http://dx.doi.org/10.1094/mpmi-19-0161

Parlevliet J. (2002). Durability of resistance against fungal, bacterial and viral pathogens; present situation. Euphytica **124:** 2. 147-156.

Pathuri I. P., Reitberger I. E., Huckelhoven R., Proels R. K. (2011). *Alcohol dehydrogenase 1* of barley modulates susceptibility to the parasitic fungus *Blumeria graminis* f.sp. *hordei*. J Exp Bot. **62:** 10. 3449-3457. DOI: http://dx.doi.org/10.1093/jxb/err017

Pathuri I. P., Zellerhoff N., Schaffrath U., Hensel G., Kumlehn J., Kogel K. H., Eichmann R., Huckelhoven R. (2008). Constitutively activated barley *ROPs* modulate epidermal cell size, defense reactions and interactions with fungal leaf pathogens. Plant Cell Rep. **27:** 12. 1877-1887. DOI: http://dx.doi.org/10.1007/s00299-008-0607-9

Pogány M., von Rad U., Grün S., Dongó A., Pintye A., Simoneau P., Bahnweg G., Kiss L., Barna B., Durner J. (2009) Dual roles of reactive oxygen species and NADPH oxidase RBOHD in an *Arabidopsis-Alternaria* pathosystem. Plant Physiol. **151:** 3. 1459-1475. DOI: http://dx.doi.org/10.1104/pp.109.141994

Pumphrey M. O., Bernardo R., Anderson J. A. (2007). Validating the QTL for *Fusarium* head blight resistance in near-isogenic wheat lines developed from breeding populations. Crop Sci. **47:** 1. 200-206. DOI: http://dx.doi.org/10.2135/cropsci2006.03.0206

Purnhauser L., Bóna L., Láng L. (2011) Occurrence of 1BL.1RS wheat-rye chromosome translocation and of *Sr36/P*. Euphytica. **179:** 2. 287-295. DOI: http://dx.doi.org/10.1007/s10681-010-0312-y

Qi X., Niks R. E., Stam P., Lindhout P. (1998). Identification of QTLs for partial resistance to leaf rust *(Puccinia hordei)* in barley. Theoretical and Applied Genetics. **96:** 8. 1205-1215. DOI: http://dx.doi.org/10.1007/s001220050858

Qi Y., Tsuda K., Glazebrook J., Katagiri F. (2011). Physical association of pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) immune receptors in *Arabidopsis*. Mol Plant Pathol. **12:** 7. 702-708. DOI: http://dx.doi.org/10.1111/j.1364-3703.2010.00704.x

Robert-Seilaniantz A., Grant M., Jones J. D. (2011). Hormone crosstalk in plant disease and defense: more than just jasmonate-salicylate antagonism. Annu Rev Phytopathol. **49:** 317-343. DOI: http://dx.doi.org/10.1146/annurev-phyto-073009-114447

Saga H., Ogawa T., Kai K., Suzuki H., Ogata Y., Sakurai N., Shibata D., Ohta D. (2012). Identification and characterization of *ANAC042*, a transcription factor family gene involved in the regulation of camalexin biosynthesis in *Arabidopsis*. Mol Plant Microbe Interact. **25:** 5. 684-696. DOI: http://dx.doi.org/10.1094/mpmi-09-11-0244

Sagaram U. S., Pandurangi R., Kaur J., Smith T. J., Shah D. M. (2011). Structure-activity determinants in antifungal plant defensins *MsDef1* and *MtDef4* with different modes of action against *Fusarium graminearum*. PLoS One. **6:** 4. e18550. DOI: http://dx.doi.org/10.1371/journal.pone.0018550

Schmidt S. M., Kuhn H., Micali C., Liller C., Kwaaitaal M., Panstruga R. (2014). Interaction of a *Blumeria graminis* f. sp. *hordei* effector candidate with a barley ARF-GAP suggests that host vesicle trafficking is a fungal pathogenicity target. Mol Plant Pathol. **15:** 6. 535-549. DOI: http://dx.doi.org/10.1111/mpp.12110

Schoonbeek H. J., Wang H. H., Stefanato F. L., Craze M., Bowden S., Wallington E., Zipfel C., Ridout C. J. (2015). *Arabidopsis* EF-Tu receptor enhances bacterial disease resistance in transgenic wheat. New Phytol. **206:** 2. 606-613. DOI: http://dx.doi.org/10.1111/nph.13356

Schornack S., Ballvora A., Gurlebeck D., Peart J., Baulcombe D., Ganal M., Baker B., Bonas U., Lahaye T. (2004). The tomato resistance protein Bs4 is a predicted non-nuclear TIR-NB-LRR protein that mediates defense responses to severely truncated derivatives of *AvrBs4* and overexpressed *AvrBs3*. Plant J. **37:** 1. 46-60. DOI: http://dx.doi.org/10.1046/j.1365-313x.2003.01937.x

Schultheiss H., Dechert C., Kogel K. H., Huckelhoven R. (2002). A small GTP-binding host protein is required for entry of powdery mildew fungus into epidermal cells of barley. Plant Physiol. **128:** 4. 1447-1454. DOI: http://dx.doi.org/10.1104/pp.010805

Shen Q. H., Zhou F., Bieri S., Haizel T., Shirasu K., Schulze-Lefert P. (2003). Recognition specificity and *RAR1/SGT1* dependence in barley *Mla* disease resistance genes to the powdery mildew fungus. Plant Cell. **15:** 3. 732-744. DOI: http://dx.doi.org/10.1105/tpc.009258

Song S., Qi T., Fan M., Zhang X., Gao H., Huang H., Wu D., Guo H., Xie D. (2013). The bHLH subgroup IIId factors negatively regulate jasmonate-mediated plant defense and development. PLoS Genet. **9:** 7. e1003653. DOI: http://dx.doi.org/10.1371/journal.pgen.1003653

Song W. Y., Wang G. L., Chen L. L., Kim H. S., Pi L. Y., Holsten T., Gardner J., Wang B., Zhai W. X., Zhu L. H., Fauquet C., Ronald P. (1995). A receptor kinase-like protein encoded by the rice disease resistance gene, *Xa21*. Science. **271:** 5243. 1804-1806. DOI: http://dx.doi.org/10.1126/science.270.5243.1804

Stergiopoulos I., van den Burg H. A., Okmen B., Beenen H. G., van Liere S., Kema G. H., de Wit P. J. (2010). Tomato Cf resistance proteins mediate recognition of cognate homologous effectors from fungi pathogenic on dicots and monocots. Proc Natl Acad Sci U S A. **107:** 16. 7610-7615. DOI: http://dx.doi.org/10.1073/pnas.1002910107

Stotz H. U., Spence B., Wang Y. (2009). A defensin from tomato with dual function in defense and development. Plant Mol Biol. **71:** 1-2.:131-143. DOI: http://dx.doi.org/10.1007/s11103-009-9512-z

Svalheim Ø., Robertsen B. (1993). Elicitation of H_2O_2-production in cucumber hypocotyl segments by oligo-1,4-α-D-galacturonides and an oligo-β-glucan preparation from cell walls of *Phythophthora megasperma* f. sp. *glycinea*. Physiologia Plantarum. **88:** 4. 675-681. DOI: http://dx.doi.org/10.1111/j.1399-3054.1993.tb01388.x

Szabó-Hevér Á., Lehoczki-Krsjak S., Varga M., Purnhauser L., Pauk J., Lantos C., Mesterházy Á. (2014). Differential influence of QTL linked to Fusarium head blight, Fusarium-damaged kernel, deoxynivalenol contents and associated morphological traits in a Frontana-derived wheat population. Euphytica. **47:** 8. 732-738. DOI: http://dx.doi.org/10.1007/s10681-014-1124-2

Tang D., Christiansen K. M., Innes R. W. (2005). Regulation of plant disease resistance, stress responses, cell death, and ethylene signaling in *Arabidopsis* by the EDR1 protein kinase. Plant Physiol. **138:** 2. 1018-1026. DOI: http://dx.doi.org/10.1104/pp.105.060400

Tang D., Simonich M. T., Innes R. W. (2007). Mutations in *LACS2*, a long-chain acyl-coenzyme A synthetase, enhance susceptibility to avirulent *Pseudomonas syringae* but confer resistance to *Botrytis cinerea* in *Arabidopsis*. Plant Physiol. **144:** 2. 1093-1103. DOI: http://dx.doi.org/10.1104/pp.106.094318

Tao Z., Liu H., Qiu D., Zhou Y., Li X., Xu C., Wang S. (2009). A pair of allelic *WRKY* genes play opposite roles in rice-bacteria interactions. Plant Physiol. **151:** 2. 936-948. DOI: http://dx.doi.org/10.1104/pp.109.145623

 DOI: 10.18380/SZIE.COLUM.2015.1.37.

Thomma B. P., Nürnberger T., Joosten M. H. (2011). Of PAMPs and effectors: the blurred PTI-ETI dichotomy. Plant Cell. **23:** 1. 4-15. DOI: http://dx.doi.org/10.1105/tpc.110.082602

Thordal-Christensen H., Zhang Z., Wei Y., Collinge D. B. (1997). Subcellular localization of H_2O_2 in plants. H_2O_2 accumulation in papillae and hypersensitive response during the barley—powdery mildew interaction. The Plant Journal. **11:** 6. 1187-1194. DOI: http://dx.doi.org/10.1046/j.1365-313x.1997.11061187.x

Uppalapati S. R., Ishiga Y., Doraiswamy V., Bedair M., Mittal S., Chen J., Nakashima J., Tang Y., Tadege M., Ratet P., Chen R., Schultheiss H., Mysore K. S. (2012). Loss of abaxial leaf epicuticular wax in *Medicago truncatula irg1/palm1* mutants results in reduced spore differentiation of an thracnose and nonhost rust pathogens. Plant Cell. **24:** 1. 353-370. DOI: http://dx.doi.org/10.1105/tpc.111.093104

van Schie C. C., Takken F. L. (2014). Susceptibility genes 101: how to be a good host. Annu Rev Phytopathol. **52:** 551-581. DOI: http://dx.doi.org/10.1146/annurev-phyto-102313-045854

Veronese P., Chen X., Bluhm B., Salmeron J., Dietrich R., Mengiste T. (2004). The *BOS* loci of *Arabidopsis* are required for resistance to *Botrytis cinerea* infection. Plant J. **40:** 4. 558-574. DOI: http://dx.doi.org/10.1111/j.1365-313x.2004.02232.x

Veronese P., Nakagami H., Bluhm B., Abuqamar S., Chen X., Salmeron J., Dietrich R. A., Hirt H., Mengiste T. (2006). The membrane-anchored *BOTRYTIS-INDUCED KINASE1* plays distinct roles in *Arabidopsis* resistance to necrotrophic and biotrophic pathogens. Plant Cell. **18:** 1. 257-273. DOI: http://dx.doi.org/10.1105/tpc.105.035576

Vogel J. P., Raab T. K., Schiff C., Somerville S. C. (2002). *PMR6*, a pectate lyase-like gene required for powdery mildew susceptibility in *Arabidopsis*. Plant Cell. **14:** 9. 2095-2106. DOI: http://dx.doi.org/10.1105/tpc.003509

Vogel J. P., Raab T. K., Somerville C. R., Somerville S. C. (2004). Mutations in *PMR5* result in powdery mildew resistance and altered cell wall composition. Plant J. **40:** 6. 968-978. DOI: http://dx.doi.org/10.1111/j.1365-313x.2004.02264.x

Wang A., Krishnaswamy S. (2012). Eucaryotic translation initiation factor 4E-mediated recessive resistance to plant viruses and its utility in crop improvement. Mol Plant Pathol. **13:** 7. 795-803. DOI: http://dx.doi.org/10.1111/j.1364-3703.2012.00791.x

Wang E., Schornack S., Marsh J. F., Gobbato E., Schwessinger B., Eastmond P., Schultze M., Kamoun S., Oldroyd G. E. (2012). A common signaling process that promotes mycorrhizal and oomycete colonization of plants. Curr Biol. **22:** 23. 2242-2246. DOI: http://dx.doi.org/10.1016/j.cub.2012.09.043

Wang G. L., Song W. Y., Ruan D. L., Sideris S., Ronald P. C. (1996). The cloned gene, *Xa21*, confers resistance to multiple *Xanthomonas oryzae* pv. *oryzae* isolates in transgenic plants. Mol Plant Microbe Interact. **9:** 9. 850-855. DOI: http://dx.doi.org/10.1094/mpmi-9-0850

Wang K. L., Li H., Ecker J. R. (2002). Ethylene biosynthesis and signaling networks. Plant Cell. **14:** 131-151.

Wittmann S., Chatel H., Fortin M. G., Laliberte J. F. (1997). Interaction of the viral protein genome linked of turnip mosaic potyvirus with the translational eukaryotic initiation factor (iso) 4E of *Arabidopsis thaliana* using the yeast two-hybrid system. Virology. **234:** 1. 84-92. DOI: http://dx.doi.org/10.1006/viro.1997.8634

Xiao S., Calis O., Patrick E., Zhang G., Charoenwattana P., Muskett P., Parker J. E., Turner J. G. (2005). The atypical resistance gene, *RPW8*, recruits components of basal defence for powdery mildew resistance in *Arabidopsis*. Plant J. **42:** 1. 95-110. DOI: http://dx.doi.org/10.1111/j.1365-313x.2005.02356.x

Xiong L., Yang Y. (2003). Disease resistance and abiotic stress tolerance in rice are inversely modulated by an abscisic acid-inducible mitogen-activated protein kinase. Plant Cell. **15:** 3. 745-759. DOI: http://dx.doi.org/10.1105/tpc.008714

Xu X, Chen C, Fan B, Chen Z. 2006. Physical and functional interactions between pathogen-induced *Arabidopsis* WRKY18, WRKY40, and WRKY60 transcription factors. Plant Cell. **18:** 5. 1310-1326. DOI: http://dx.doi.org/10.1105/tpc.105.037523

Yang Y., Shah J., Klessig D. F. (1997). Signal perception and transduction in plant defense responses. Genes Dev. **11:** 13. 1621-1639. DOI: http://dx.doi.org/10.1101/gad.11.13.1621

Yi M., Valent B. (2013). Communication between filamentous pathogens and plants at the biotrophic interface. Annu Rev Phytopathol. **51:** 587-611. DOI: http://dx.doi.org/10.1146/annurev-phyto-081211-172916

Yoshii M., Nishikiori M., Tomita K.,Yoshioka N., Kozuka R., Naito S., Ishikawa M. (2004). The *Arabidopsis* cucumovirus multiplication 1 and 2 loci encode translation initiation factors 4E and 4G. J Virol. **78:** 12. 6102-6111. DOI: http://dx.doi.org/10.1128/jvi.78.12.6102-6111.2004

Yuan M., Chu Z., Li X., Xu C., Wang S. (2010). The bacterial pathogen *Xanthomonas oryzae* overcomes rice defenses by regulating host copper redistribution. Plant Cell. **22:** 9. 3164-3176. DOI: http://dx.doi.org/10.1105/tpc.110.078022

Zhai W., Wang W., Zhou Y., Li X., Zheng X., Zhang Q., Wang G., Zhu L. (2002). Breeding bacterial blight-resistant hybrid rice with the cloned bacterial blight resistance gene *Xa21*. Molecular Breeding. **8:** 4. 285-293.

Zhang K., Halitschke R., Yin C., Liu C. J., Gan S. S. (2013a). Salicylic acid 3-hydroxylase regulates *Arabidopsis* leaf longevity by mediating salicylic acid catabolism. Proc Natl Acad Sci U S A. **110:** 36. 14807-14812. DOI: http://dx.doi.org/10.1073/pnas.1302702110

Zhang Y., Lubberstedt T., Xu M. (2013b). The genetic and molecular basis of plant resistance to pathogens. J Genet Genomics. **40:** 1. 23-35. DOI: http://dx.doi.org/10.1016/j.jgg.2012.11.003

Zhao X., Tan G., Xing Y., Wei L., Chao Q., Zuo W., Lübberstedt T., Xu M. (2012). Marker-assisted introgression of *qHSR1* to improve maize resistance to head smut. Molecular Breeding. **30:** 2. 1077-1088. DOI: http://dx.doi.org/10.1007/s11032-011-9694-3

Zhu Y., Nam J., Carpita N. C., Matthysse A. G., Gelvin S. B. (2003). *Agrobacterium*-mediated root transformation is inhibited by mutation of an *Arabidopsis* cellulose synthase-like gene. Plant Physiol. **133:** 3. 1000-1010. DOI: http://dx.doi.org/10.1104/pp.103.030726

Zipfel C., Kunze G., Chinchilla D., Caniard A., Jones J. D., Boller T., Felix G. (2006). Perception of the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. Cell. **125:** 4. 749-760. DOI: http://dx.doi.org/10.1016/j.cell.2006.03.037

Zipfel C., Robatzek S. (2010). Pathogen-associated molecular pattern-triggered immunity: veni, vidi...? Plant Physiol. **154:** 2. 551-554. DOI: http://dx.doi.org/10.1104/pp.110.161547

 DOI: 10.18380/SZIE.COLUM.2015.1.37.