Acta Phytopathologica et Entomologica Hungarica 38 (3–4), pp. 227–236 (2003)

Role of Hydrogen Peroxide in Symptom Expression of Barley Susceptible and Resistant to Powdery Mildew

Y. M. HAFEZ and Z. KIRÁLY

Plant Protection Institute, Hungarian Academy of Sciences,
Budapest, Hungary

Three genotypes of barley (cultivar Ingrid) expressing the genes *Mlo* (susceptible), *Mla12* (resistant with HR symptoms) and *mlo5* (resistant without HR) in relation to infection by race A6 of *Blumeria graminis* f. sp. *hordei* have been sprayed with a solution of H_2O_2 after establishment of infection (2–3 days after inoculation). Under the influence of H_2O_2 , leaves of the susceptible *Mlo* and *mlo5*-resistant plants exhibited HR-type symptoms with tissue necroses. The *Mla12*-resistant genotype produced HR earlier and the number of necrotic lesions increased, as compared to untreated control leaves. Treatment with H_2O_2 before establishment of infection (one day after inoculation), resulted in all the three genotypes in inhibition of the pathogen and symptomless response. It was possible to reverse the inhibitory as well as the HR-producing actions of H_2O_2 with injection of leaves with a combination of superoxide dismutase (SOD) and catalase (CAT) before treatment with H_2O_2.

It is suggested that the hypothetical negative regulation of HR-associated resistance in susceptible plants carrying the gene *Mlo* as well as in barley displaying HR-independent resistance and carrying the gene *mlo5,* could be associated with the limited production of H_2O_2 in infected plants. Supplying H_2O_2 to barley leaves that are either susceptible or display HR-independent resistance after establishment of infection, releases the negative regulation of symptoms of HR-associated resistance. This action of H_2O_2 is sensitive to antioxidant enzymes, such as SOD and CAT.

Keywords: Powdery mildew, hydrogen peroxide, antioxidants, catalase, superoxide dismutase, hypersensitive response.

It is known for a long time that resistance of barley to infection of powdery mildew (*Blumeria graminis* f. sp. *hordei* DC., Speer) may or may not be associated with the hypersensitive response (HR). As shown previously by several workers, host cultivars carrying the gene *Mla12* express a typical HR-associated race-specific resistance. However, cultivars having the gene *mlo5* exhibit race-nonspecific (general) resistance which is not associated with HR (Jørgensen, 1988; Hückelhoven et al., 1999).

We have shown earlier that the HR is a consequence, not the cause, of plant resistance to fungal pathogens (Király et al., 1972). Recently, several laboratories (Yu et al., 1998; Bendahmane et al., 1999; Cole et al., 2001; Schoelz et al., 2003) demonstrated that HR and resistance are two separate phenomena also in the cases of viral and bacterial infections. Furthermore, several investigations pointed out that plant tissue necrotization, including the HR phenomenon, is associated with the generation of reactive oxygen species (ROS) (Doke, 1985; Király et al., 1993; Baker and Orlandi, 1995; Barna et al., 2003; Hückelhoven and Kogel, 2003). A few publications also referred to the *in vitro* as well as *in vivo* sensitivity of plant pathogens to the action of ROS (Tzeng and DeVay, 1993; Király et al., 1993; Ouf et al., 1993; Wu et al., 1997; Király and El-Zahaby, 2000; El-Zahaby et al., 2004).

0238–1249/2003/$ 20.00

We raised the question, what is indeed the role of the ROS hydrogen peroxide (H_2O_2) in symptom expression of powdery mildew resistant barley plants carrying two resistance genes, *Mla12* and *mlo5,* which determine HR-type and general (race non-specific) resistance, respectively.

Materials and Methods

Plant material

Susceptible barley (*Hordeum vulgare* L.) cultivar Ingrid, carrying the gene *Mlo* and incompatible resistant Ingrid carrying the genes *mlo5* or *Mla12* were used. Seeds of the above-mentioned cultivars were sown in plastic pots (10 or 15 cm diameter) containing a mixture of soil:peat:sand (2:1:1, v/v/v) and grown under greenhouse conditions at 18–23 °C, with 16 hours photoperiod per day using supplemental light by mercury lamps (HQL) with a light intensity of 160 mE m^{-2} s^{-1} and 75–80% relative humidity.

Powdery mildew pathogen

Barley powdery mildew, *Blumeria graminis* f. sp. *hordei* DC. (Speer) race A6 kindly supplied by the Plant Pathology Department of the Justus Liebig University, Giessen, Germany, was maintained on susceptible barley seedlings in the greenhouse. Conidia of the pathogen were powdered onto leaves of susceptible and resistant barley.

Application of H_2O_2 to intact and detached barley leaves

Leaves of 8–10 day-old-seedlings were inoculated with the pathogen. Some intact inoculated leaves were detached at different time periods after inoculation. Either intact or detached leaves were sprayed with 20–500 mM H_2O_2 one, two and three days after inoculation. The water solution of H_2O_2 contained 0.5% Tween. Leaves which were treated with H_2O_2 only or infected only with the pathogen served as controls.

Application of superoxide dismutase (SOD) and catalase (CAT)

Barley intact and detached leaves infected with the powdery mildew fungus were injected with a water solution which contained 5000 units catalase (CAT) (E.C.1.11.1.6) and 2500 units superoxide dismutase (SOD) (E.C.1.15.1.1) / ml. After evaporation of water leaves were sprayed with H_2O_2.

Results

Compatible (susceptible) host/pathogen combination

Infection of barley cultivar Ingrid with race A6 of powdery mildew resulted in a susceptible reaction of the host, e. g. production of fungal mycelia and conidia. We have

shown previously (Király and El-Zahaby, 2000; El-Zahaby et al., 2003) that by treating barley leaves with chemicals which produce ROS, symptom expression of susceptible cultivars profoundly changed and the host exhibited HR-type necrotic spots, similar to the resistant reaction.

In this study we applied H_2O_2 as a spray, 1, 2 and 3 days after inoculation on intact as well as on detached leaves. When we sprayed barley leaves with a H_2O_2 solution one day after inoculation, no symptoms of susceptibility or resistance were expressed at all, because the pathogen was supposedly killed or inhibited before establishment of infection. One hundred mM solution of H_2O_2 was effective in the case of intact, attached leaves and 50 mM H_2O_2 was inhibitory in the case of detached leaves. Leaf detachment was carried out immediately after inoculation with the pathogen. However, if we treated attached leaves with 100 mM H_2O_2 or detached leaves with 50 mM H_2O_2 2 or 3 days after inoculation, we induced HR-type tissue necrotization characteristic of resistant cultivar/avirulent pathogenic race combinations *(Fig. 1).* Spraying leaves only with H_2O_2 did not cause any symptoms.

The HR-type necrotic symptoms caused by leaf treatments with H_2O_2 were fully reversed (inhibited) when we injected a mixture of SOD and CAT into infected leaves before spraying leaves with H_2O_2 *(Fig. 1).* This experiment demonstrated that the resistance reaction with HR-type symptoms was indeed induced by the combined action of infection and a certain amount of H_2O_2.

Resistant cultivar carrying the gene Mla12 *(incompatible combination)*

This type of host/pathogen combination expresses HR-type resistance to some races of the fungus (e.g. race A6). When we sprayed attached leaves of cultivar Ingrid *Mla12* with H_2O_2 as early as one day after inoculation, the concentration of 100 mM H_2O_2 inhibited the development of hypersensitive necrosis (HR) and the resistant plant remained symptomless because the powdery mildew pathogen was supposedly killed before establishment of infection. Similar suppression of HR-type symptoms was experienced with detached leaves when we applied 50 mM H_2O_2 *(Fig. 2C).* Leaf detachment was carried out immediately after inoculation.

On the other hand, when we sprayed attached barley leaves with 50 mM H_2O_2 2–3 days after inoculation, tissue necrotization was stimulated: the number of leaf necroses increased, as compared to the control infected leaves. Treatment of detached leaves with 20 mM H_2O_2 2 days after inoculation also stimulated the appearance of HR which occurred 1–2 days earlier than in control infected leaves. The number of necroses also increased *(Fig. 3C).* Similar result has been obtained with another incompatible combination: *Mlg*/A6 (not shown) which is characterized by penetration resistance caused by cell wall apposition.

Stimulation of HR caused by H_2O_2-treatments was reversed by injection of a mixture of SOD and CAT into infected leaves before the H_2O_2-treatments *(Fig. 3D).* Injection of antioxidants into leaves which were treated with H_2O_2 very early (one day) after inoculation when HR necroses were inhibited, also resulted in reversal of the action of H_2O_2, namely infected leaves produced regular HR, similar to the control infected leaves *(Fig.*

2D). It was concluded that the antioxidant capacity of these compounds protected leaves from the effects of H_2O_2 on symptom expression.

It seems reasonable to suppose that the action of H_2O_2 was required for the stimulated HR-producing ability of infection when we applied H_2O_2-treatment 2–3 days after inoculation (after establishment), however, it was required for inhibiting the pathogen and the production of HR when H_2O_2-treatment was applied very early (one day) after inoculation.

Resistant cultivar carrying the gene mlo5 *(race non-specific resistance)*

It is known (Jørgensen, 1988) that barley cultivars carrying the gene *mlo5* exert resistance to most of the pathogenic races of *Blumeria graminis* f. sp. *hordei.* In other words, these plants express a general, race non-specific resistance which is not associated with the HR.

If we sprayed attached barley leaves with 100 mM H_2O_2 one day after inoculation, there was no change in host reaction. In other words, no symptoms were detected in leaves. However, treatment with H_2O_2 2 or 3 days after inoculation induced HR-type necroses in infected leaves *(Fig. 4C).* Necroses were induced with 100 or 50 mM H_2O_2 in case of attached and detached leaves, respectively. Detachment of leaves was carried out one day after inoculation. Treatments only with H_2O_2 did not induce any symptoms in barley leaves.

It was also demonstrated in these experiments that injection of leaves with antioxidants (SOD plus CAT) reversed the HR-inducing ability of H_2O_2-treatments in both attached and detached inoculated leaves *(Fig. 4D),* indicating that a certain amount of H_2O_2 is required for the production of HR-type necroses. This experiment supports the hypothesis that the capacity to develop HR is suppressed in susceptible as well as in the *mlo5* resistant cultivars (Büschges et al., 1997; Shirasu and Schulze-Lefert, 2000; Hückelhoven et al., 2003).

Discussion

It is evident from previous experiments (see introduction) that the HR-type tissue necrotization may or may not be associated with resistance of plants to infections and the HR could be a consequence, not the cause, of disease resistance.

Fig. 1. Induction of HR in a susceptible combination of barley cultivar Ingrid *(Mlo)* with race A6 of powdery mildew. (A): Infected leaves with powdery mildew symptoms. (B): Uninfected leaves treated with 50 mM H_2O_2. (C): Infected and H_2O_2-treated leaves with symptoms of HR. Treatment was applied after establishment of infection (2 days after inoculation). (D): Infected and H_2O_2-treated leaves injected with a mixture of SOD and CAT. Symptoms of HR were reversed to the original symptoms of susceptibility ▶

Fig. 2. Inhibition of HR in a resistant combination of barley cultivar Ingrid *(Mla12)* with race A6 of powdery mildew. (A): Infected leaves exhibiting HR. (B): Uninfected leaves treated with 50 mM H_2O_2. (C): Symptomless infected leaves treated with H_2O_2 before establishment of infection (one day after inoculation). (D): Infected and H_2O_2-treated leaves injected with SOD and CAT. The original necrotic symptoms of HR were restored ▶

Fig. 1. A B C D

Fig. 2. A B C D

Fig. 3. A B C D

Fig. 4. A B C D

Baulcombe and co-workers (Bendahmane et al., 1999) claim that "extreme resistance" to viral infections, which is not associated with HR, is the consequence of an early resistance mechanism in which arrest of viral replication is very effective. In other words, the time is too short for the development of HR (tissue necrotization), thus, resistance to the virus overrides the induction of HR. However, if the development of resistance needs a longer time period, infection is able to induce HR in the resistant host. Furthermore, if resistance develops very late, systemic spread of the virus occurs first and only subsequently can the "resistant" host develop HR. Whether or not ROS have a role in virus resistance, remained an unresolved question. Another example of HR-independent resistance is the gene *Cf-9*-encoded tomato resistance to *Cladosporium fulvum.* If there is sustained production of the elicitor of the pathogen in the *Cf-9* resistant plant, HR-independent resistance is changed to HR-associated-type of resistance (Hammond-Kosack et al., 1995).

It was demonstrated in several laboratories that the presence of ROS in mildew resistant barley plants may be associated with the hypersensitive response (Thordal-Christensen et al., 1997; Hückelhoven and Kogel, 2003; Hückelhoven et al., 1999; Király and El-Zahaby, 2000; El-Zahaby et al., 2003). However, regulatory compounds may modify the action of ROS. Several publications (Levine et al., 1994; El-Zahaby et al., 1995; Foyer et al., 1997) referred to the possible role of antioxidants in balancing the action of ROS in powdery mildew-infected barley and other host/pathogen combinations. In susceptible barley leaves antioxidant reactions are induced that inhibit tissue necrotization and suppress lipid peroxidation. However, these antioxidative processes are less efficiently activated in resistant leaves which may result in necrotization (HR). Our results show that H_2O_2 plays a pivotal role in inducing HR as well as pathogen arrest in different barley/powdery mildew combinations.

In a compatible host/pathogen combination (cultivar Ingrid/race A6 of the fungus) H_2O_2 applied to the infected leaves 2–3 days after inoculation caused an HR-type resistance characteristic of resistant cultivars *(Fig. 1).* Earlier we have shown that ROS-producing chemicals, such as the riboflavin-methionine mixture with illumination and the xanthine-xanthine oxidase mixture can cause similar HR-type symptoms in infected susceptible barley (Király and El-Zahaby, 2000; El-Zahaby et al., 2003). In this study we demonstrate that H_2O_2 is responsible for host tissue necrotization and 100 mM or 50 mM H_2O_2 applied as a spray is necessary for the induction of HR in attached and detached susceptible leaves. Hydrogen peroxide was also responsible for pathogen arrest because

◀ Fig. 3. Stimulation of HR in the resistant cultivar Ingrid *(Mla12)* infected with race A6 of powdery mildew. (A): Infected leaves exhibiting HR. (B): Uninfected leaves treated with 20 mM H_2O_2. (C): Stimulated necrotization in infected and H_2O_2-treated leaves. Treatment was applied 2 days after inoculation (after establishment of infection). (D): Stimulation of HR was reversed in infected and H_2O_2-treated leaves which were injected with SOD + CAT

◀ Fig. 4. Induction of HR in a resistant cultivar displaying HR-independent resistance (Ingrid carrying the gene *mlo5*). (A): Infected symptomless leaves. (B): Uninfected leaves treated with 50 mM H_2O_2. (C): Induced HR with the stimulated necrotization in infected and H_2O_2-treated leaves. Treatment was applied after establishment of infection (2 days after inoculation). (D): The stimulated HR was reversed in infected and H_2O_2-treated leaves which were injected with SOD + CAT

an early (one day after inoculation) application of H_2O_2 inhibited appearance of any symptoms in leaves. Furthermore, the role of H_2O_2 in inducing HR-type resistance in a susceptible barley cultivar was substantiated by another result: we were able to reverse the action of H_2O_2 when we injected a combination of SOD and CAT solution into infected leaves before treatment of leaves with H_2O_2. As a result, host susceptibility was restored and the fungus produced mycelia and conidia in those leaves *(Fig. 1).*

In a resistant host which produces typical HR (cultivar Ingrid carrying the gene *Mla12* and infected with race A6), treatment of leaves with H_2O_2 resulted in a stimulated development of HR-type necroses and HR appeared earlier than in the untreated and detached infected leaves *(Fig. 3).* As in case of the susceptible host, an early (one day after inoculation) application of the H_2O_2-spray induced arrest of the pathogen and consequently, no symptoms appeared *(Fig. 2).* The antioxidant action of SOD and CAT protected leaves from effects of H_2O_2 on symptom expression *(Figs 2 and 3).*

The race non-specific resistance of cultivar Ingrid carrying the gene *mlo5* against race A6 resulted in a symptomless resistance which was not associated with the HR. However, HR was produced as a consequence of spraying leaves with H_2O_2 2–3 days after inoculation *(Fig. 4).* Leaf treatments only with H_2O_2 did not cause any symptoms, as was experienced in all of our experiments. Antioxidants (SOD and CAT) prevented development of HR, indicating that a certain amount of H_2O_2 is necessary for the induction of necrotization which is associated with HR.

It would seem that H_2O_2 which is produced after infection as a result of the oxidative burst, exerts a dual role in symptom expression and resistance: it may arrest pathogen growth (resistance) and induces HR-associated host symptoms. In other words, increased amounts and/or sustained production of H_2O_2 can convert susceptibility to resistance and HR-independent to HR-associated resistance. When we applied H_2O_2 to leaves before establishment of infection (one day after inoculation), pathogen arrest occurred early and neither susceptible nor HR-type symptoms were expressed. However, appearance of HR (necrotization) or stimulation of the HR symptoms occurred if H_2O_2 was applied to infected leaves after establishment of infection (2–3 days after inoculation).

It is important to note that in relation to other host/pathogen interactions several investigators called the attention to the action of high doses of H_2O_2 in resistance and the role of low doses in signalling stimulated antioxidant actions (Doke, 1985; Levine et al., 1994; Gechev et al., 2002). It remains to be seen whether in our powdery mildew-infected barley plants an artificial supply of H_2O_2 influences the natural antioxidant capacity of the host? This would balance the harmful effects of ROS and expression of symptoms.

Several research groups hypothesized that the ability for resistance, which is associated with HR, is suppressed in barley cultivars susceptible to powdery mildew infection. These plants express the gene *Mlo* which may function as a negative regulator of plant tissue necrotization. It was also claimed that the *mlo5* resistance, which is not associated with HR, could be the consequence of another type of negative regulation of HR where the pathogen arrest (resistance) is uncoupled from HR (Büschges et al., 1997; Shirasu and Schulze-Lefert, 2000; Hüchelhoven et al., 2003).

One can suppose that the negative regulation of host tissue necrotization in both susceptible and *mlo5*-resistant barley plants is in relation to the limited production of H_2O_2. Artificial supply of this compound, as a spray to infected leaves, can induce HR-associated resistance in both susceptible and *mlo5*-resistant cultivars. Release of this negative control by H_2O_2 can be reversed by injecting tissues with antioxidants, such as SOD and CAT. It is not known at present whether H_2O_2 can influence the natural antioxidant capacity of leaves, as was shown earlier in naturally infected barley plants (El-Zahaby et al., 1995).

Literature

Baker, C. J. and Orlandi, E. W. (1995): Active oxygen and plant pathogenesis. Annu. Rev. Phytopathol. 33, 299–321.

Barna, B., Fodor, J., Pogány, M. and Király, Z. (2003): Role of reactive oxygen species and antioxidants in plant disease resistance. Pest Manag. Sci. 59, 459–464.

Bendahmane, A., Kanyuka, K. and Baulcombe, D. C. (1999): The Rx gene from potato controls separate virus resistance and death responses. Plant Cell 11, 781–791.

Büschges, R., Hollricher, K., Panstruga, R., Simons, G., Wolter, M., Frijters, A., van Daelen, R., van der Lee, T., Diergaarde, P., Groenendijk, J., Topsch, S., Vos, P., Salamini, F. and Schulze-Lefert, P. (1997): The barley *Mlo* gene: a novel control element of plant pathogen resistance. Cell 88, 695–705.

Cole, A. B., Király, L., Ross, K. and Schoelz, J. E. (2001): Uncoupling resistance from cell death in the hypersensitive response of *Nicotiana* species to *Cauliflower mosaic virus* infection. Mol. Plant-Microbe Interact. 14, 31–41.

Doke, N. (1985): NADPH-dependent $O_2^{\cdot -}$ generation in membrane fraction isolated from wounded potato tubers inoculated with *Phytophthora infestans.* Physiol. Plant Pathol. 27, 311–322.

El-Zahaby, H. M., Gullner, G. and Király, Z. (1995): Effect of powdery mildew infection of barley on the ascorbate-glutathione cycle and other antioxidants in different host-pathogen interactions. Phytopathology 85, 1225–1230.

El-Zahaby, H. M., Hafez, Y. M. and Király, Z. (2004): Effect of reactive oxygen species on plant pathogens in planta and on disease symptoms. (In press).

Foyer, Ch., Lopez-Delgado, H., Dat, J. F. and Scott, I. M. (1997): Hydrogen peroxide and glutathione-associated mechanisms of acclimatory stress tolerance and signalling. Physiol. Plant. 100, 241–254.

Gechev, T. I., Gadjev, F., Van Breusegem, D., Inzé, S., Dukiandjiev, V., Toneva, I. and Minkov, I. (2002): Hydrogen peroxide protects tobacco from oxidative stress by inducing a set of antioxidant enzymes. Cell. Mol. Life Sci. 59, 708–714.

Hammond-Kosack, K. E., Staskawics, B. J., Jones, J. D. G. and Baulcombe, D. C. (1995): Functional expression of a fungal avirulence gene from a modified potato virus X genome. Mol. Plant-Microbe Interact. 8, 181–185.

Hückelhoven, R. and Kogel, K.-H. (2003): Reactive oxygen intermediates in plant-microbe interactions: Who is who in powdery mildew resistance? Planta 216, 891–902.

Hückelhoven, R., Dechert, C. and Kogel, K.-H. (2003): Overexpression of barley BAX inhibitor 1 induces breakdown of *mlo*-mediated penetration resistance of *Blumeria graminis.* Proc. Natl. Acad. Sci. USA 100, 5555–5560.

Hückelhoven, R., Fodor, J., Preis, C. and Kogel, K.-H. (1999): Hypersensitive cell death and papilla formation in barley attacked by the powdery mildew fungus are associated with H_2O_2 but not with salicylic acid accumulation. Plant Physiol. 119, 1251–1260.

Jørgensen, J. H. (1988): Genetic-analysis of barley mutants with modifications of powdery mildew resistance gene Ml–a12. Genome 30, 129–132.

Király, Z., Barna, B. and Érsek, T. (1972): Hypersensitivity as a consequence, not the cause, of plant resistance to infection. Nature 239, 215–219.

Király, Z. and El-Zahaby, H. M. (2000): Effect of recative oxygen species on rust and powdery mildew pathogens and on symptoms. Acta Pytopathol. Entomol. Hung. 35, 239–240.

Király, Z., El-Zahaby, H., Galal, A., Abdou, S., Ádám, A., Barna, B. and Klement, Z. (1993): Effect on oxy free radicals on plant pathogenic bacteria and fungi and on some plant diseases. In: Gy. Mózsik, I. Emerit, J. Fehér, B. Matkovics and Á. Vincze (eds): Oxygen Free Radicals and Scavengers in the Natural Sciences. Akadémiai Kiadó, Budapest, pp. 9–19.

Levine, A., Tenhaken, R., Dixon, R. and Lamb, C. (1994): H_2O_2 from the oxidative burst orchestrates the plant hypersensitive disease resistance response. Cell 79, 583–593.

Ouf, M. F., Gazar, A. A., Shehata, Z. A., Abdou, El-S., Király, Z. and Barna, B. (1993): The effect of superoxide anion on germination and infectivity of wheat stem rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and Henn.) uredospores. Cereal Res. Commun. 21, 31–37.

Schoelz, J. E., Palanichelvam, K., Cole, A. B., Király, L. and Cawly, J. (2003): Dissecting the avirulence and resistance components that comprise the hypersensitive response to Cauliflower mosaic virus in Nicotiana. In: G. Stacey and N. T. Keen (eds): Plant-Microbe Interact. Vol. 6. APS Press, St. Paul, Minn., USA. pp. 259–283.

Shirasu, K. and Schulze-Lefert, P. (2000): Regulators of cell death in disease resistance. Plant Molec. Biol. 44, 371–385.

Thordal-Christensen, H., Zhang, Z., Wie, Y. and Collinge, D. B. (1997): Subcellular localization of H_2O_2 in plants. H_2O_2 accumulation papillae and hypersensitive response during the barley-powdery mildew interaction. Plant J. 11, 1187–1194.

Tzeng, D. D. and DeVay, J. E. (1993): Role of oxygen radicals in plant disease development. In: J. H. Andrews, I. C. Tommerup (eds): Advances in Plant Pathology. Vol 10., Academic Press, London, pp. 1–34.

Wu, G. S., Shortt, B. J., Lawrence, E. B., Leon, J., Fitzsimmons, K. C., Levine, E. B., Raskin, I. and Shah, D. M. (1997): Activation of host defense mechanisms by elevated production of H_2O_2 in transgenic plants. Plant Physiol. 115, 427–435.

Yu, I. C., Parker, J. and Bent, A. F. (1998): Gene-for-gene disease resistance without the hypersensitive response in Arabidopsis dndl mutant. Proc. Natl. Acad. Sci. USA 95, 7819–7824.

Acknowledgement

This work was supported by the Hungarian Research Grant (OTKA) Nos. Ts040835, T037341.